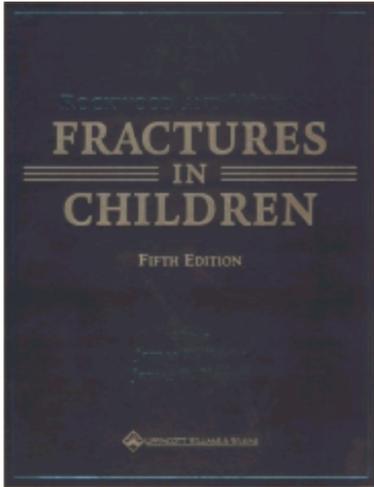


**Rockwood and Wilkins' Fractures in Children, 5th edition:** by James H., Md. Beaty (Editor), James R., Md. Kasser (Editor), Charles A. Rockwood By Lippincott Williams & Wilkins Publishers (September 30, 2001)



By OkDoKeY

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# Rockwood and Wilkins' Fractures in Children

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## PREFACE

The amount of published information about fractures in skeletally immature patients has grown exponentially over the past 5 years, and a number of areas of controversy have been further defined by advocates on both sides of the issues. We have tried to ensure that this edition reflects the current consensus and presents all relevant information so that readers can make informed decisions about fracture management. One of our goals in this edition of *Fractures in Children* was to decrease the overall size of the book, while increasing the amount of information on the critical areas of operative techniques and management of complications while retaining pertinent information about anatomy, etiology, diagnosis, and treatment options. The format of the book, including layout and illustrations, has been updated to be more "user-friendly," and a number of new contributors have been added, each of whom brings their own expertise and experience to the text. As with earlier editions, our purpose is to provide a text that will be useful to medical students, residents, and practicing orthopaedists and will ultimately help ensure that children with musculoskeletal injuries receive the best possible treatment.

*James H. Beaty, M.D.*  
*James R. Kasser, M.D.*

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In addition, a number of people at each of our institutions were instrumental in the hands-on production of this text. From the Campbell Foundation in Memphis, Kay Daugherty (Editorial), Barry Burns (Graphics), and Joan Crowson (Library) provided invaluable assistance. From Boston, Theresa Medua (Secretarial) and Alison Clapp (Library).

To those most personally affected by our undertaking of this project—our families—we express heartfelt thanks for their understanding and support during the countless nights and weekends that were consumed by our work on the book. Our wives, Terry Beaty and Candace Kasser, deserve special recognition for always believing we could do this and for supporting us through the long months of production.

## DEDICATION

We dedicate this edition of *Fractures in Children* to Charles Rockwood and Kaye Wilkins who began this extraordinary text almost 20 years ago. The addition to the legendary *Rockwood and Green's Fractures in Adults* of a volume focusing specifically on children's fractures expanded the scope of information and provided a much needed compendium on the treatment of fractures involving the immature skeleton. Had these two men not had the foresight and fortitude to embark on that first edition and the dedication to produce subsequent editions, those who treat musculoskeletal injuries in children would not have had this valuable resource. We have been inspired by their example and hope to help continue the tradition they began. In honor of their tremendous contributions, this volume, and all subsequent editions, will be titled *Rockwood and Wilkins' Fractures in Children*.

# THE PRESENT STATUS OF CHILDREN'S FRACTURES

KAYE E. WILKINS  
ALARIC J. AROOJIS

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In the past generation there have been many changes in how fractures in children are handled. This has been the result of many factors. First, there has been a drastic change in the dissemination of information regarding the management of children's fractures. Second, there has been a change in the philosophy of how fractures are treated, with more emphasis on operative management. Third, there has been a change in the incidence of fractures in the United States and Europe. Fourth, and finally, in a modern North American environment, there have been changes in the etiology of fractures in children. Each of these factors will be discussed as a separate section in this chapter. The first two factors will be discussed briefly. The third and fourth factors will be discussed in more detail.

The whole goal in studying the incidence of children's fractures is to develop preventative strategies. The experience of others in this aspect will be discussed in the fifth section of this chapter.

## THE INFORMATIONAL CHANGES

### Single-Authored Texts

At the turn of the century the major fracture texts were authored by single individuals who used their own personal experience as their major reference source. In the more popular single-authored texts by Stimson (10), Scudder (9), and Cotton (3), the trend was to discuss both adult and children's fractures in the same sections on a geographical basis; for example, fractures about the elbow. This single-authored text concept continued into the late 1950s and early 1960s, with the most popular texts of that period being those by Bohler (2), Key and Conwell (4), and Watson-Jones (11).

### Multiauthored Texts

With the explosion of orthopaedic knowledge and the development of regional anatomic orthopaedic specialization, it became impossible for one author to produce a fracture text that was all-encompassing. Thus began the trend toward multiauthored fracture texts with two to three editors. The first to start this trend in fracture texts in North America were Charles Rockwood and David Green, who produced the first edition of their multiauthored textbook *Fractures* in 1975 (7). A year later, Wilson had revised Watson-Jones' text with some multiple authors (12). In this text, Chapter 17, authored by Anthony Catterall, focused on children's fractures. The first edition of *Fractures* by Rockwood and Green did not include children's fractures.

### Exclusive Children's Fracture Texts

In North America, one of the pioneers in fracture treatment in children was Walter P. Blount, who in 1955 was the first to author a textbook devoted exclusively to children (1). His philosophy of nonoperative management set the standard for treating children's fractures for more than a generation. Almost 20 years later, Mercer Rang authored another textbook devoted exclusively to children's fractures (6). His book has served as a standard reference for the treatment of children's fractures to this day.

### Multiauthored Children's Fracture Texts

Again, it soon became impossible for one person to author a textbook on children's fractures that covered each fracture type and its treatment in sufficient detail to satisfy the demands of the present-day orthopaedic surgeon. Thus evolved the multiauthored text on children's fractures. The first edition of a textbook of this type was added as volume III to the two volumes of *Fractures in Adults*, edited by Rockwood and Green in 1984 (8). This multiauthored concept of textbooks on children's fractures has been followed by many other excellent texts. These multiauthored textbooks have been well received; they are now in subsequent editions. These present-day textbooks now serve as a single encompassing reference source for the physician who treats children's fractures.

### Emergence of Pediatric Orthopaedics

#### Consolidation of Information

#### Pediatric Orthopaedic Journals

In the past, information regarding the management of children's fractures was interspersed in journals and meetings dealing with general orthopaedics. In 1981, *The Journal of Pediatric Orthopedics* was established by Lynn Staheli and Robert Hensinger, and it contained articles devoted exclusively to orthopaedic conditions seen in pediatric patients. It is interesting to note that the first article in their first volume was related to trauma, "The Surgical Treatment of Partial Closure of the Growth

Plate” by Anders Langenskiold (5). Part B of *The Journal of Pediatric Orthopedics* was established in 1992 to allow the expansion of the number of articles from non-North American authors.

### ***Pediatric Fracture Courses***

The recognition of the uniqueness of children's fractures has stimulated the production of a number of courses organized world-wide devoted exclusively to the management of children's trauma (see [Appendix A](#)). Thus, there has been a concentration of both spoken and written information regarding children's fractures in the modern orthopaedic community.

## **CHANGES IN THE PHILOSOPHY OF TREATING FRACTURES IN CHILDREN**

### **Blount's Nonoperative Axioms**

Dr. Walter Blount, in his textbook *Fractures in Children*, emphasized that because of growth, children's fractures have a great potential to remodel (1). In fact, he outlined the rules of remodeling as to what amount of angulation would be accepted in children's fractures. However, he was very opposed to operative intervention. This was especially true in his opinion of intramedullary fixation of femoral shaft fractures in children:

“The operation is unnecessary, however and as such must be condemned. It introduces the hazard of an unnecessary anesthetic, unnecessary exposure of bone ends, and trauma to the entire marrow cavity of the femur. There is no reason for doing it” (1).

### **Complications with Operative Intervention**

It was his experience that nonunions often followed open reductions in children. He went on to delineate the serious other complications that he had seen with open reduction, including death from sepsis. His comment was, “One postoperative osteomyelitis in a lifetime is enough to cure a surgeon of a casual attitude toward open reduction.”

This aversion to operative management of children's fractures set the tone for a generation of orthopaedic surgeons.

### **Trend Toward Minimal Invasion**

Certainly, prior to the mid-1950s, when Blount published his textbook (1), operative procedures usually required large incisions and extensive dissection. In the 45 years since the publication of his book, there has been a trend toward more operative intervention with good results. However, the surgery performed is minimally invasive, and fixation is often only temporary.

### **Factors Creating a Trend Toward Operative Intervention**

This trend toward more operative intervention has been the result of four factors: (a) improvement in technology; (b) rapid healing allowing minimal and temporary fixation; (c) the financial and social pressures to limit the hospitalization of children; and (d) the expectation by the public for a perfect outcome in every case.

#### ***Improvement in Technology***

The use of the image intensifier has greatly improved the ease with which fractures can be reduced and stabilized internally with percutaneous methods. Other technical advances such as computed tomography (CT), magnetic resonance imaging (MRI), and sonographic imaging have expanded the ability to better define the fracture patterns. The use of powered instruments and cannulated screws has improved the accuracy of applying fixation devices via percutaneous techniques.

#### ***Rapid Healing***

Because children heal and remodel rapidly, in many cases the fixation devices need to be utilized for only a short period of time. Children tolerate all types of casts well for short periods of time, which allows a minimally stabilized fracture to be immobilized until there is sufficient internal callous to supplement the limited internal fixation.

#### ***Minimal Hospitalization***

The rising costs of hospitalization have created a trend to mobilize children to an outpatient setting as soon as possible. This has been reinforced by the fact that in two thirds of the families in the United States both parents are wage earners.

Cox and Clarke, in evaluating the fracture management in their hospital in Southampton, England, found a high incidence of secondary hospital treatment for fractures initially managed nonoperatively (15). There was a 12% readmission rate to correct late displacement of fractures of the radius and distal humerus. In addition, 24% of their internal fixation procedures were to salvage unacceptable results of nonoperative management. It was their conclusion that more selective initial operative intervention in radial and distal humeral fractures could decrease the incidence of costly readmissions to the hospital.

There are both social and financial pressures to mobilize the child early. The trend now is to temporarily surgically stabilize these fractures so that the patient can be discharged early.

#### ***The Perfect Result***

Modern parents have become very sophisticated and now expect a perfect outcome for their child. They inspect the x-rays, question the alignment, and expect the alignment to be perfect or anatomic.

A common statement made by the patient's father is, “He has tremendous potential to be a great athlete.” These pressures often direct the treating physician toward operative intervention to obtain a perfect alignment.

### **Changes from Previous Editions**

The trend toward the establishment of surgical intervention can be seen in the changes in the previous editions of this textbook. In the first edition (19), very little mention was made regarding intramedullary fixation of either femoral or radial and ulnar shaft fractures. There was an extensive discussion of methods of traction for femoral shaft fractures and supracondylar fractures. In the fourth edition (20), the reverse was true. There was considerable discussion of intramedullary fixation and very little mention regarding traction techniques.

### **Phases in Development of Operative Techniques**

Often, a new procedure is proposed and becomes widely used. Initially, there is a wave of enthusiasm for the benefits of the procedure. However, with more widespread use of a procedure, problems become more apparent and thus there often are modifications of the original technique. Thus, it takes a period of time before the technique becomes relatively complication free.

### **Specific Problems with Operative Techniques**

Some of the specific problems that have occurred over the years are listed as follows:

1. Ulnar nerve injury with mediolateral pin fixation of supracondylar fractures ( [16](#))
2. High refracture rate with external fixation of femoral shaft fractures ( [18](#))
3. Avascular necrosis of the femoral head following use of interlocking intramedullary nails ( [13,17](#))

As will be mentioned in the following chapters of this textbook dealing with the specific fractures, there have been recent changes in the operative technique or postoperative management to minimize the development of these problems.

### **Nonoperative Techniques Need To Be Maintained**

Unfortunately, with this emphasis on operative management, the fact that most children's fractures can be managed by nonoperative techniques becomes obscured. As a result, many of the recent orthopaedic trainees are not developing good nonoperative technical skills.

Two recent articles have demonstrated improved results of treating children's fractures by focusing on improvements of prior nonoperative methods. Chess and co-workers ([14](#)) have shown that when properly applied, a well-molded short arm cast provides just as good a result as a long arm cast in treating displaced fractures of the distal radial metaphysis. The key to success in using a short arm cast is in a careful molding of the cast at the fractures site so there is a proper cast index of 0.7 or less. Walker and Rang ([21](#)) recently revised the concept of treating unstable fractures of the shafts of the radius and ulna with a long arm cast with the elbow in extension. This has resulted in a lower remanipulation rate.

Continued focus on developing and maintaining nonoperative skills such as appropriate cast application and proper moulding techniques needs to be constantly reemphasized as the mainstay of treating children's fractures.

## **THE PRESENT STATUS OF THE INCIDENCE OF FRACTURES IN CHILDREN**

The incidence of children's fractures is extremely variable. It can vary with the child's age, the season of the year, cultural and environmental climates, and the hour of the day, to name just a few factors. As a culture changes from a primarily rural to an urban setting, the injury patterns may change as well. It is important to develop a general picture of how, when, and why fractures occur in children.

### **Incidence of Fractures**

#### ***Healing Processes***

Early reviews primarily developed a knowledge base of fracture healing in children. Walking's 1934 review demonstrated that children's fractures heal differently and included such concepts as the overgrowth of long bones after fracture and the ability of children's fractures to remodel significant angular deformities ( [56](#)). In 1941, Beekman and Sullivan published an extensive review of the incidence of children's fractures ( [24](#)). Their pioneering work—still quoted today—included a study of 2,094 long bone fractures seen over a 10-year period at Bellevue Hospital in New York City. The major purpose of their study was to develop basic principles for treating children's fractures.

In 1954, two major reports, one by Hanlon and Estes ([36](#)) and the other by Lichtenberg ([47](#)), confirmed the findings of the previous studies with regard to the general incidence of children's long bone fractures and their ability to heal and readily remodel. These initial reviews were mainly statistical analyses and did not delve deeply into the true epidemiology of children's fractures. In 1965, Wong explored the effect of cultural factors on the incidence of fractures by comparing Indian, Malay, and Swedish children ([58](#)). In the 1970s, two other studies, one by Iqbal ([34](#)) and another by Reed ([50](#)), added more statistics regarding the incidence of the various long bone fractures.

#### ***Preventive Programs***

Landin's 1983 report on 8,682 fractures established a trend in reviewing the incidence of children's fractures ( [41](#)). He reviewed the data on all fractures in children that occurred in Malmo, Sweden, over 30 years, and examined the factors affecting the incidence of children's fractures. His study remains a landmark on this subject. By studying two populations, 30 years apart, he evaluated whether fracture patterns were changing, and if so, the reasons for such changes. His initial goal was to establish data for preventive programs, so he focused on fractures that produced clean, concise, concrete data.

In 1997 Landin updated his work, reemphasizing the statistics from his previous publication ( [40](#)). He felt that the twofold increase in fracture rate during the 30 years from 1950 to 1979 in Malmo was due mainly to an increased participation in sports. In 1999, in cooperation with Tiderius and Duppe, Landin ( [55](#)) studied the incidence in the same age group again in Malmo and found that the incident rate had actually declined by 9% in the years 1993 to 1994. The only exception was an increase of distal forearm fractures in girls, which he attributed to their increased participation in sporting events.

Cheng and Shen, in their 1993 study from Hong Kong, also set out to define the problems of children's fractures by separating the incidences into age groups ( [28](#)). They tried to gather epidemiologic data on which to build preventive programs. In 1999 this study was expanded to include almost 6,500 fractures in children 16 and younger over a 10-year period ([27](#)). The fracture patterns changed little over those 10 years. What did change was the increased incidence of closed reduction and percutaneous pin fixation of fractures, with a corresponding decrease in open reductions. There was also a marked decrease in the hospital stay of their patients.

In short, the goals for studying children's fractures have changed over the past 60 years. Originally, the goal was to identify the most common bones fractured and how they heal. The goals of present studies are to gather data in an attempt to decrease the incidence of fractures by establishing preventive programs.

### **Defining the Incidence of Fractures**

#### ***Variations***

##### ***Cultural Differences***

When one looks at the incidence of specific fractures within a well-defined group of children, the data are usually concise. However, when trying to obtain the global or general incidence of injury or fracture patterns for all types of children, there may be problems. For instance, Cheng and Shen studied children in Hong Kong who lived in confined high-rise apartments ([28](#)). Their risk of exposure to injury differed from the study by Reed of children living in the rural environment of Winnipeg, Canada ([50](#)). Two separate reviews by Laffoy ([39](#)) and Westfelt ([57](#)) have found that children in a poor social environment (as defined by a lower social class or by dependence on public assistance) had an increased incidence of accidents. In England, children from single-parent families have been found to have higher accident and infection rates ([31](#)).

Thus, in domestic settings where many people are on public assistance or where there is a higher incidence of disruption of family structure, social rather than physical factors may be more of an influence on the incidence of injuries.

##### ***Climatic Differences***

The climate may be a strong factor as well. Children in colder climates, with ice and snow, are exposed to risks different from those of children living in warmer climates. The exposure time to outdoor activities may be greater for children who live in warmer climates. For example, the incidence of chronic overuse elbow injuries in young baseball players (little league elbow) is far greater in the southern United States than in more northern communities. This is simply because there is greater playing or exposure time.

### **Difficulties in Comparing Fracture Studies**

## Defining Age Groups

Another problem with comparing studies is the definition of pediatric age groups. Some use 12 years as a cutoff age; others extend it to 16 or 20.

## Inpatient Versus Outpatient Studies

Some studies report only fracture victims admitted to a hospital, which loads them toward the more serious injuries.

## Anatomic Location

Reports vary in the precision of their defined types of fracture patterns. In the older series, reports were only of the long bone involved, such as the radius. More recent series have emphasized a more specific location, separating the radius, for example, into physeal, distal, shaft, and proximal fracture types.

Thus, in trying to define the exact incidence of fractures, it is difficult to compare series because of cultural, environmental, and age differences. In this section, these differences were considered in grouping the results and producing average figures. These data are presented in an attempt to provide a reasonable and accurate reflection of the overall incidence of injuries and fractures in all children.

## Frequency of Childhood Fractures

In Landin's series from Malmo, Sweden, the chance of a child sustaining a fracture during childhood (birth to age 16) was 42% for boys and 27% for girls (41). When considered on an annual basis, 2.1% of all the children (2.6% for boys, 1.7% for girls) sustained at least one fracture each year. These figures were for all fracture types and included those treated on an inpatient basis and an outpatient basis. The overall chance of fracture per year was 1.6% for both girls and boys in a study from England of both outpatients and inpatients by Worlock and Stower (59).

The chance of a child sustaining a fracture severe enough to require inpatient treatment during the first 16 years of life is 6.8% (28). Thus, on an annual basis, 0.43% of the children in an average community will be admitted for a fracture-related problem during the year.

In a series of 23,915 patients seen at four major hospitals for injury-related complaints, 4,265 (17.8%) had fractures (26,32,33,57). Thus, close to 20% of the patients who present to hospitals with injuries have a fracture.

It is interesting to note that in a follow-up study by Tiderius, Landin, and Duppe (55) in the years 1993 and 1994, 13 years after the termination of the original 30-year study by Landin (41), there was almost a 10% decrease in the incidence of fractures in the 0- to 16-year age group. They attributed this to less physical activity on the part of modern-day children coupled with better protective sports equipment and increased traffic safety (e.g., stronger cars and use of auto restraint systems). The overall incidence of children's fractures is summarized in Table 1-1.

Percentage of children sustaining at least one fracture from 0 to 16 years of age: boys, 42%; girls, 27%  
Percentage of children sustaining a fracture in 1 year: 1.6%–2.1%  
Percentage of children who are hospitalized because of a fracture: during entire childhood (0–16 years), 6.8%; each year, 0.43%  
Percentage of patients with injuries (all types) who have fractures: 17.8%

Data from references 26, 28, 32, 33, 42, 57, and 59.

TABLE 1-1. OVERALL FREQUENCY OF FRACTURES

## Age Groups

### Correlation With Incidence of Injuries

Starting with birth and extending to age 12, all the major series that segregated patients by age group have demonstrated a linear increase in the annual incidence of fractures with age (Fig. 1-1) (27,28,34,41,59). There seems to be a peak at 12 years, with some decrease until age 16, probably related to a significant decrease in the incidence of fractures in girls over age 12. The percentage of injured boys as compared with girls continues to increase in the older age groups.

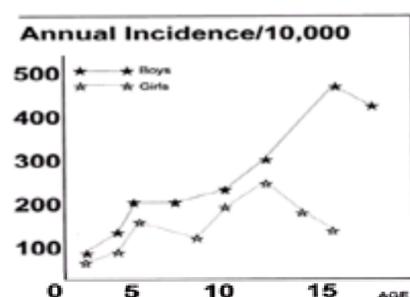
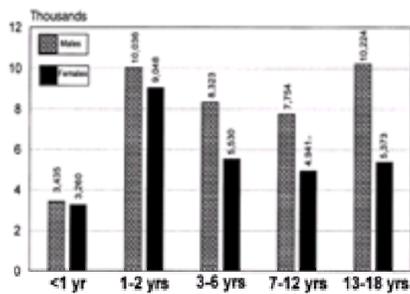


FIGURE 1-1. Incidence of fractures by age. Boys peak at 15 years. Girls peak earlier, at 12 years and then decline. [Reprinted from Landin LA. Fracture patterns in children. *Acta Orthop Scand* 1983;54(suppl 202):13, with permission.]

These fracture statistics differ slightly from the incidence of overall injuries: the incidence of injuries peaks early, at ages 1 to 2 years (Fig. 1-2) (39). Although there is a high incidence of injuries in children ages 1 to 2, the incidence of fractures is low. Most injuries in children of this age are nonorthopaedic entities such as head injuries, lacerations, and abrasions. In fact, the incidence of lacerations in both sexes peaks at this age (51).



**FIGURE 1-2.** Injuries per 100,000 children per year. Estimated U.S. injury rates in children by age and sex, 1978. (Reprinted from Rivara FP, Bergman AB, LoGerfo JP, et al. Epidemiology of childhood injuries. *Am J Dis Child* 1982;136:503; with permission.)

### Trauma

In 1962, Kempe and associates (37) called attention to the high incidence of fractures and other injuries in young children that were due to nonaccidental trauma. They termed these injuries part of the battered child syndrome. Akbarnia and colleagues later defined the specific fracture patterns seen in victims of child abuse (22). Not all fractures in the first year of life can be attributed to abuse, however. In a review of fractures occurring in the first year of life, McClelland and Heiple found that fully 44% were from documented accidental and nonabusive etiologies (47). They also noted that 23% of these patients had a generalized condition that predisposed them to fractures. Thus, although nonaccidental trauma remains the leading cause of fractures during the first year of life, other constitutional conditions may predispose children to fractures from accidental causes. The high incidence of fractures from nonaccidental trauma extends to age 3 (38).

### Gender

The male predominance of injury and fracture victims has been discussed (Fig. 1-1 and Fig. 1-2, Table 1-1). For all age groups, the overall ratio of boys to girls who sustain a single fracture is 2.7:1 (28). In girls, fracture incidence peaks just before adolescence and then decreases during adolescence (28,41,50). In Cheng and co-workers' 10-year study from Hong Kong (27), the male incidence in the 12- to 16-year age group was 83%. The incidence of fractures in girls steadily declined from their peak in the 0- to 3-year age group.

In some areas there is little difference in the incidence of fractures between boys and girls. For example, during the first 2 years of life, the overall incidence of injuries and fractures in both genders is nearly equal. During these first 2 years, the injury rates for foreign body ingestion, poisons, and burns have no significant gender differences. With activities in which there is a male difference in participation, such as with sports equipment and bicycles, there is a marked increase in the incidence of injuries in boys (27,51).

The injury incidence may not be due to the rate of exposure alone; behavior may be a major factor. For example, one study found that the incidence of auto/pedestrian childhood injuries peaks in both sexes at ages 5 to 8 (53). When the total number of street crossings per day was studied, both sexes did so equally, but despite this equal exposure, boys had a higher number of injuries. Thus, the difference in the rate between the sexes begins to develop a male predominance when behaviors change. The difference in the injury rate between the genders may change in the future as more girls participate in activities with increased physical risk (27,51).

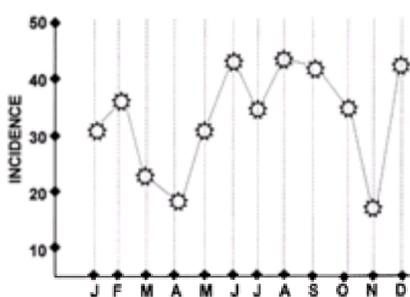
### Right Versus Left Frequency

In most series, the left upper extremity demonstrates a slight but significant predominance (27,28,32,34,41,51). The ratio of left to right overall averages 1.3:1. In some fractures, however, especially those of supracondylar bones, lateral condyles, and the distal radius, the incidence is far greater, increasing to as much as 2.3:1 for the lateral condyle. In the lower extremity, the incidence of injury on the right side is slightly increased (32,41).

The reasons for the predominance of the left upper extremity have been studied, but no definite answers have been found. Rohl (52) speculated that the right upper extremity is often being used actively during the injury, so the left assumes the role of protection. In a study examining the left-sided predominance in the upper extremity, Mortenson and Thonell (49) questioned patients and their parents on arrival to the emergency department about which arm was used for protection and the position of the fractured extremity at the time of the accident. They found two trends: regardless of handedness, the left arm was used more often to break the fall, and when exposed to trauma, the left arm was more likely to be fractured. The cause for this latter increased incidence in the left side was thought to be due to either the increased fragility or immature neuromuscular coordination of the nondominant extremity.

### Frequency by Season and Time of Day

Fractures are more common during the summer, when children are out of school and exposed to more vigorous physical activities. Four studies from the northern hemisphere have confirmed this summertime increase (Fig. 1-3) (27,28,52,59).



**FIGURE 1-3.** Distribution of children's fractures on a monthly basis. Note the general increase from May to October. (Reprinted from Reed MH. Fractures and dislocations of the extremities in children. *J Trauma* 1977;17:353; with permission.)

The most consistent climatic factor appears to be the number of hours of sunshine. Masterson and co-workers (46), in an eloquent study from Ireland, found a strong positive correlation between monthly sunshine hours and monthly fracture admissions. There was also a weak negative correlation with monthly rainfall. Overall, the average number of fractures in the summer was 2.5 times that in the winter. In days with more sunshine hours than average, the average fracture admission rate was 2.31 per day; on days with fewer sunshine hours than average, the admission rate was 1.07 per day.

In Sweden, the incidence of fractures in the summer had a bimodal pattern that seemed to be influenced by cultural traditions. In two large series of both accidents and fractures in Sweden by Westfelt (57) and Landin (41), the researchers noticed increases in May and September and significant decreases in June, July, and August. Both writers attributed this to the fact that children in their region left the cities to spend the summer in the countryside. Thus, the decrease in the overall fracture rate probably was due to a decrease in the number of children at risk remaining in the city.

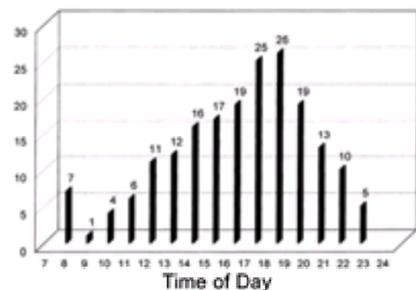
Masterson and co-workers (46) speculated that because the rate of growth increases during the summer, the number of physal fractures should also increase, because the physes would be weaker during this time. For example, the incidence of a slipped capital femoral epiphysis, which is related to physal weakness,

increases during the summer (23). However, Landin, in his study of more than 8,000 fractures of all types, found the overall seasonal incidence of physal injuries to be exactly the same as nonphysal injuries (41).

Age may affect the seasonal variation of fractures. In children ages 0 to 3, no seasonal variations are seen. The number of fractures in this age group was consistent throughout the year (38).

Thus, it appears that climate, especially in areas where there are definite seasonal variations, influences the incidence of fractures in all children, especially in the older children. However, in small children and infants, whose activities are not seasonally dependent, there appears to be no significant seasonal influence.

The time of day in which children are most active seems to correlate with the peak time for fracture occurrence. In Sweden, the incidence peaked between 2 and 3 P.M. (57). In a well-documented study from Texas by Shank and co-workers (54), the hourly incidence of fractures formed a well-defined bell curve peaking at about 6 P.M. (Fig. 1-4).



**FIGURE 1-4.** Incidence of children's fractures per time of day. There is an almost bell-shaped curve with a peak at around 6 P.M. (Reprinted from Shank LP, Bagg RJ, Wagnon J. Etiology of pediatric fractures: the fatigue factors in children's fractures. Presented at the National Conference on Pediatric Trauma, Indianapolis, 1992; with permission.)

### Long-Term Trends

#### Increase in Minor Trauma

Landin's study is the only one that has compared the changes over a significant time span: his data were collected over 30 years (41). He classified the degree of trauma as slight, moderate, or severe. The incidence of all trauma in both boys and girls increased significantly over the 30-year study period, but the incidence of severe trauma increased only slightly. The greatest increase was seen in the "slight" category. Landin attributed the increase in this category to the introduction of subsidized medical care. Because expense was not a factor, parents were more inclined in the later years of the study to seek medical attention for relatively minor complaints. Physicians, likewise, were more inclined to order x-rays. Thus, many of the minor injuries, such as torus fractures, which were often ignored in the earlier years, were seen more often at medical facilities during the later years.

Likewise, the overall incidence of fractures in Malmo, Sweden, (the same city as Landin's original study) (41) significantly decreased (10%) in the more recent years (55).

The one fracture type that exhibited a true increase over this period was that of the femoral shaft. This increase was thought to be influenced by new types of play activities and increased participation in sports.

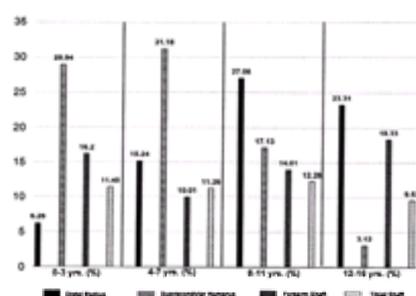
#### Increase in Child Abuse

The number of fractures due to nonaccidental causes (child abuse) has risen consistently in the past decades. In Kowal-Vern and associates' study of fractures in children ages 0 to 3 (38), the number of fractures due to abuse increased almost 150 times from 1984 to 1989. This increase was attributed to a combination of improved recognition, better social resources, and an increase in the number of cases of child abuse.

### Specific Fracture Incidences

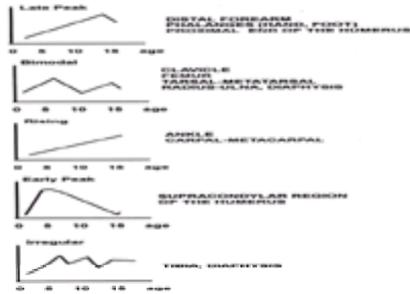
#### Age Factors

The anatomic areas most often fractured seem to be the same in the major series, but these rates change with age. For example, the supracondylar fracture of the humerus is most common in the first decade, with a peak at age 7. Fractures of the femur are most common in children ages 0 to 3. Fractures of the physis are more common just before skeletal maturity. This variation is best illustrated in Cheng and Shen's data (Fig. 1-5) (28).



**FIGURE 1-5.** The frequency of occurrence of the most common fracture areas in children. The frequency of each fracture pattern differs with the various age groups. The figures express the percentage of total fractures for that age group and represent boys and girls combined. (Reprinted from Cheng JC, Shen WY. Limb fracture pattern in different pediatric age groups: a study of 3,350 children. *J Orthop Trauma* 1993;7:17; with permission.)

Landin found a similar age variability and divided it into six distinct patterns (Fig. 1-6) (41). When he compared these variability patterns with the common etiologies, he found some correlation. For example, late-peak fractures (distal forearm, phalanges, proximal humerus) were closely correlated with sports and equipment etiologies. Bimodal pattern fractures (clavicle, femur, radioulnar, diaphyses) showed an early increase from lower energy trauma, then a late peak in incidence due to injury from high- or moderate-energy trauma. Early peak fractures (supracondylar humeral fractures are a classic example) were due mainly to falls from high levels.



**FIGURE 1-6.** Patterns of fracture: variations with age. The peak ages for the various fracture types occur in one of five patterns. [Reprinted from Landin LA. Fracture patterns in children. *Acta Orthop Scand* 1983;54(suppl 220):80; with permission.]

### Locations

Early reports of children's fractures lumped together the areas fractured, and fractures were reported only as to the long bone involved (e.g., radius, humerus, femur) (24,32,34,43,44,48). More recent reports have split fractures into the more specific areas of the long bone involved (e.g., the distal radius, the radial neck, the supracondylar area of the humerus) (28,34,41,50,59). This change in reporting—from the so-called “lumpers” to the “splitters”—has produced a more accurate picture of the true incidence of each specific fracture type.

### Single Bones

In children, fractures in the upper extremity are much more common than those in the lower extremity (32,34). Overall, the radius is the most commonly fractured long bone, followed by the humerus. In the lower extremity, the tibia is more commonly fractured than the femur (Table 1-2).

Bone	%
Radius	45.1
Humerus	18.4
Tibia	15.1
Clavicle	13.8
Femur	7.6

Data from references 24, 32, 34, 43, 44, and 48.

**TABLE 1-2. INCIDENCE OF FRACTURES IN LONG BONES**

### Specific Areas Fractured

In recent years, five reports produced by so-called splitters divided fracture types into many anatomic areas (28,34,41,50,59). In trying to reach a global consensus, the author has identified areas common to all the reports but has taken some liberties to do so. For example, distal radial metaphyseal and physeal fractures were combined to form the distal radius. Likewise, the carpals, metacarpals, and phalanges were combined to form the region of the hand and wrist. All the fractures around the elbow, from those of the radial neck to supracondylar fractures, were grouped as elbow fractures. This grouping allows comparison of the regional incidence of specific fracture types in children (Table 1-3).

Fracture	%
Distal radius and physis	23.3
Hand (carpals, metacarpals, and phalanges)	20.1
Elbow area (distal humerus and proximal radius and ulna)	12.0
Clavicle	6.4
Radius shaft	6.4
Tibia shaft	6.2
Foot (metatarsals and phalanges)	5.9
Ankle (distal tibia)	4.4
Femur (neck and shaft)	2.3
Humerus (proximal and shaft)	1.4
Other	11.6

Data from references 28, 34, 41, 50, and 59.

**TABLE 1-3. INCIDENCE OF SPECIFIC FRACTURE TYPES**

The individual reports agreed that the most common area fractured was the distal radius. The next most common area, however, varied from the hand in Landin's series (41) to the elbow (mainly supracondylar fractures) in Cheng and Shen's series (27,28).

### Physeal Injuries

The incidence of physeal injuries overall varied from 14.5% (29) to a high of 27.6% (45). To obtain an overall incidence of physeal fractures, six reports totaling 6,479 fractures in children were combined (25,29,45,48,50,59). In this group, 1,404 involved the physis, producing an average overall incidence of 21.7% for physeal fractures (Table 1-4).

Total fractures = 6,477  
 Number of physeal injuries = 1,404  
 Percentage of physeal injuries = 21.7%

Data from references 25, 29, 30, 45, 48, and 59.

#### TABLE 1-4. INCIDENCE OF PHYSEAL FRACTURES

##### *Open Fractures*

The overall incidence of open fractures in children is consistent. The data were combined from the four reports in which the incidence of open fractures was reported ([28,32,45,59](#)). The incidence in these reports varied from 1.5% to 2.6%. Combined, these reports represented a total of 8,367 fractures with 246 open fractures, resulting in an average incidence of 2.9% ([Table 1-5](#)).

Total number of fractures = 8,367

Total open fractures = 246

Percentage = 2.9%

---

Data from references 28, 32, 45, and 59.

#### TABLE 1-5. INCIDENCE OF OPEN FRACTURES

Regional trauma centers often see patients exposed to more severe trauma, so there may be a higher incidence of open fractures in these patients. The incidence of open fractures was 9% in a report of patients admitted to the trauma center of the Children's National Medical Center, Washington, D.C. ([26](#)).

##### *Multiple Fractures*

Multiple fractures in children are uncommon: the incidence ranges in the various series from 1.7% to as much as 9.7%. In four major reports totaling 5,262 patients, 192 patients had more than one fracture ([Table 1-6](#)) ([28,32,34,59](#)). The incidence in this multiple series was 3.6%.

Total fractures = 5,262

Total number of multiple fractures = 192

Percentage = 3.6%

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Data from references 28, 32, 45, and 58.

#### TABLE 1-6. INCIDENCE OF MULTIPLE FRACTURES

##### *Recurrent Fractures*

Children with generalized bone dysplasias, such as osteogenesis imperfecta and other metabolic diseases that produce osteopenia, are expected to have repeat fractures. In these patients, the etiology of these recurrent fractures is understandable and predictable. However, some children with normal osseous structures are prone to recurrent fractures, for reasons that remain unclear. The incidence of recurrent fractures in children is about 1% ([30](#)).

Landin and Nilsson ([42](#)) found that children who sustained fractures with relatively little trauma had a lower mineral content in their forearms, but they could not correlate this finding with children who had repeat fractures. Thus, in children who seem to be structurally normal, there does not appear to be a physical reason for their recurrent fractures.

##### *Repeat Fractures*

Failure to find a physical cause for repeat fractures shifts the focus to a psychological or social cause. The one common factor in accident repeaters has been a high incidence of dysfunctional families ([36](#)). In Sweden, Westfelt found that children who were accident repeaters came from "socially handicapped" families (i.e., those on public assistance or those with a caregiver who was an alcoholic) ([57](#)). Thus, recurrent fractures are probably due more to behavioral or social causes than physical causes. Landin in his follow-up article ([40](#)) followed children with repeat fractures (four or more) into adolescence and adulthood. He found these children had a significantly increased incidence of convictions for serious criminal offenses when compared with children with only one lifetime fracture.

#### ETIOLOGY OF FRACTURES

While studying the epidemiology of fractures, it is important to focus on the etiology of fractures and the settings in which they occur. In this age of computerized gizmos and the "need for speed," children are often the victims of the consumer market and unsafe products. Fractures do not occur in a vacuum, and well-researched studies that analyze the physical and social environment in which they occur are extremely valuable. It is a truism that children, unfortunately, learn more from experience than by education, so efforts should be made toward creating a safer environment for play and recreation. Studies that identify risky patterns of use or unsafe playground behavior go a long way in directing specific preventive health measures. Recommendations can be made to manufacturers regarding modification of a product, and education can be imparted to parents, school authorities, health-care professionals, physical trainers, and children.

##### *Broad Causes*

Broadly, fractures can occur due to three main causes: accidental trauma, nonaccidental injury (child abuse), and pathologic conditions. Because accidental trauma forms the largest etiologic group, it will be addressed in detail here. Nonaccidental trauma and fractures resulting from pathologic conditions will be addressed in later chapters of this book.

##### *Fractures Resulting from Accidental Trauma*

Accidental trauma can occur in a variety of settings, some often overlapping others. However, for purposes of simplicity, fractures can be considered to occur in the

following five environments: home environment; school environment; play and recreational activities; motor vehicle and road accidents; and uncommon causes such as ice cream truck, water tubing, and gunshot and missile injuries.

### **Home Environment**

Fractures sustained in the home environment are defined as those that occur in the house and surrounding vicinity. These generally occur in a fairly supervised environment and are mainly due to falls from furniture, stairs, fences, and trees.

### **Falls from Heights**

Falls can vary in severity from a simple fall while running, to a fall of great magnitude, such as from a third story window. In falling from heights, adults often land on their lower extremities, accounting for the high number of lower extremity fractures, especially the calcaneus. Children tend to fall head first, using the upper extremities to break the fall. This accounts for the larger number of skull and radial fractures in children. Femoral fractures are also more common in children falling from great heights. In contrast to adults, spinal fractures are rare in children who fall from great heights ( [61,84,97,100](#)).

In one study, children falling three stories or less all survived. Falls from the fifth or sixth floor resulted in a 50% mortality rate ( [61](#)).

### **Injury Rate Increases with Age**

Injuries also can occur from the unsupervised use of unsafe play equipment, such as trampolines and monkey bars, within the home environment and will be considered in detail later. The overall incidence of fractures occurring because of such play activity in the home environment increases with age. Only 15% occur in toddlers, but 56% occur during older years ( [110](#)).

### **Social Factors**

Interestingly, a Swedish study ( [109](#)) showed that an increased incidence of fractures in a home environment did not necessarily correlate with the physical attributes or poor safety precautions of the house. Rather, it implicated disruption of the family structure and presence of social handicaps (alcoholism, welfare recipients, etc.) as the true cause for the increase.

### **School Environment**

The supervised environments at school are generally safe, and the overall annual rate of injury (total percentage of children injured in a single year) in the school environment ranges from 2.8% to 9.2% ( [63,81,95,109](#)). True rates may be higher because of inaccurate reporting, especially of mild injuries. In one series, the official rate was 5.6%, but when the parents were closely questioned, the incidence of unreported, trivial injuries was as much as 15% ( [71](#)). The annual fracture rate of school injuries is low. Of all injuries sustained by children at school in a year, only 5% to 10% involved fractures ( [71,81,95](#)). In Worlock and Stower's series of children's fractures from England ( [110](#)), only 20% occurred at school. A large incidence of injuries (53%) occurring in school are related to athletics and sporting events ( [81](#)). These injuries are highest in the middle-school children. The peak time of day for injuries at school is in the morning, which differs from the injury patterns of children in general ( [81](#)).

### **Play and Recreational Activities**

#### **Playground**

Play is an essential element of a child's life. It enhances physical development and fosters social interaction. Noncompetitive sports and recreational activities are enjoyed by all children. Unfortunately, unsupervised or careless use of some play equipment can endanger life and limb. When Mott et al. ( [86](#)) studied the incidence and pattern of injuries to children using public playgrounds, they found that approximately 1% of children using playgrounds sustained injuries. Sixty-five percent of these children were injured by falling from equipment such as climbing frames, slides, swings, and monkey bars. They found that changing playground surfaces from concrete to more impact-absorbing surfaces such as bark reduced the incidence and severity of head injury but increased the tendency to long bone fractures (40%), bruises, and sprains.

In a study of injuries resulting from playground equipment, Waltzman et al. ( [106](#)) found that most injuries occurred in boys (56%) with a peak incidence in the summer months. Fractures accounted for 61% of these injuries, 90% of which involved the upper extremity and were sustained due to falls from playground equipment such as monkey bars and climbing frames. Younger children (1–4 years) were more likely to sustain fractures than older children. In their study, the surface below the equipment apparently did not influence the type or severity of fracture; with 30 of the 79 fractures occurring on "soft surfaces."

Similar observations were made in a study by Lillis and Jaffe ( [83](#)) in which upper extremity injuries, especially fractures, accounted for the majority of hospitalizations resulting from injuries on playground equipment. Older children sustained more injuries on climbing apparatus, whereas younger children sustained more injuries on slides.

#### **Newer Play Devices**

Other recreational activities enjoyed by children, such as bicycling, skating, skateboarding, and sledding, are an important cause of fractures and injuries in children. Several studies have analyzed the incidence and pattern of injuries arising from the unsupervised or careless use of this equipment and have suggested safety precautions and equipment modification to decrease the risk of injury. A disturbing trend is the rekindled enthusiasm toward the use of trampolines, skateboards, and in-line skates over the past decade, and several studies have highlighted their risks and dangers.

#### **Bicycle Injuries**

Bicycle injuries are a significant cause of mortality and morbidity for children ( [92](#)). Bicycle mishaps are the most common causes of serious head injury in children ( [108](#)). Boys in the 5- to 14-year age group are at greatest risk for bicycle injury (80%). Puranik et al. ( [92](#)) studied the profile of pediatric bicycle injuries in a sample of 211 children who were treated for bicycle-related injury at their trauma center over a 4-year period. They found that bicycle injuries accounted for 18% of all pediatric trauma patients. Bicycle/motor vehicle collisions caused 86% of injuries. Sixty-seven percent had head injuries and 29% sustained fractures. More than half of the incidents occurred on the weekend. Sixteen percent were injured by ejection from a bicycle after losing control, hitting a pothole, or colliding with a fixed object or another bicycle. Fractures mainly involved the lower extremity, upper extremity, skull, ribs, and pelvis in decreasing order of incidence.

**Helmet Use Low.** More importantly, the study detected that the use of safety helmets was disturbingly low (<2%). Other studies confirm the observation that less than 13% to 15% of children wear helmets while riding bicycles ( [72,93](#)). The Year 2000 Health Objectives call for helmet use by 50% of bicyclists ( [102](#)). Research has shown that legislation, combined with education and helmet subsidies, is the most effective strategy to increase use of safety helmets in child bicyclists ( [65](#)). As public awareness of both the severity and preventability of bicycle-related injuries grows, the goal of safer bicycling practices and lower injury rates can be achieved ( [92](#)).

**Injuries from Bicycle Parts.** Bicycle spokes and handle bars are also responsible for an increasing number of fractures and soft tissue injuries in children. D'Souza et al. ( [70](#)) and Segers et al. ( [94](#)) found that bicycle spoke injuries are typically sustained when the child's foot is caught in the spokes of the rotating wheel. Out of a total of 130 children with bicycle spoke injuries, 29 children sustained fractures of the tibia, fibula, or foot bone. Several had lacerations and soft tissue defects. D'Souza et al. ( [70](#)) suggested that a mesh cover to prevent the toes from entering between the spokes and a plastic shield to bridge the gap between the fork and horizontal upright can substantially decrease the incidence of these injuries.

#### **Skateboarding**

Skateboarding and in-line skating have experienced a renewed surge in popularity over the past two decades. With the increasing number of participants, high-tech equipment development and vigorous advertising, skateboard and skating injuries are expected to increase. Because the nature of skateboarding encompasses both

high speed and extreme maneuvers, high-energy trauma fractures and other injuries can occur, as highlighted by several studies (73,89,91). Studies have shown that skateboarding-related injuries are more severe and have more serious consequences than roller-skating or in-line skating injuries (89). In a study of skateboarding injuries, Fountain et al. (73) found that fractures of the upper or lower extremity accounted for 50% of all skateboarding injuries. Interestingly, more than one third of those injured sustained injuries within the first week of skateboarding. Most injuries occurred in preadolescent boys (75%) 10 to 16 years of age, and despite traffic legislation, 65% sustained injuries on public roads, footpaths, and parking lots. Several organizations have recommended safety guidelines and precautions such as use of helmets, knee and elbow pads, and wrist guards, but such regulations are seldom enforced.

### Roller Skates and In-Line Skates

In a study of in-line skate and roller skate injuries in childhood, Jerosch et al. (78) found that in a group of 1,036 skaters, 60% had sustained injuries. Eight percent of these were fractures, mostly involving the elbow, forearm, wrist, and fingers (78%). Less than 20% used protective devices, and most lacked knowledge of the basic techniques of skating, braking, and falling. In a larger study of 60,730 skating injuries in children, Powell and Tanz (91) found that 68% of the children were preadolescent boys with a mean age of 11.8 years. Fractures were the most common injury (65%), and two thirds of these involved the distal forearm. Two and a half percent required hospital admissions; 90% of these admissions were for a fracture. Similarly, Mitts and Hennrikus (85) found that 75% of in-line skating fractures in children occurred in the distal forearm as a result of falls on the outstretched hand. One in eight children sustained a fracture during the first attempt at the sport. The orthopaedic community has an obligation to educate the public on the need for wearing wrist guards when using in-line skates or roller skates.

### Trampoline-Related Injuries

Trampolines enjoyed increasing popularity in the 1990s and are a significant cause of morbidity in children. Several studies have noted a dramatic increase in the number of pediatric trampoline injuries (PTIs) during the past 10 years, rightfully deeming it as a "national epidemic" (75,98). Furnival et al. (75), in a retrospective study of PTIs over a 7-year period, found that the annual number of PTIs tripled between 1990 and 1997. In contrast to other recreational activities in which males constitute the population at risk, PTI patients were predominantly female, with a median age of 7 years. Nearly a third of the injuries resulted from falling off the trampoline. Fractures of the upper and lower extremities occurred in 45% and were more frequently associated with falls off the trampoline. In another excellent study on PTIs, Smith (98) found that there was virtually a 100% increase in injuries from 1990 to 1995, with an average of greater than 60,000 injuries per year. Younger children had a higher incidence of upper extremity fractures and other injuries. In a later study, Smith and Shields (99) came up with some interesting data. Fractures, especially involving the upper extremity, accounted for 35% of all injuries. Interestingly, more than 50% of the injuries occurred under direct adult supervision. More disturbingly, 73% of the parents were aware of the potential dangers of trampolines, and 96% of the injuries occurred in the home backyard. These researchers, along with others (75), rightly concluded that use of warning labels, public education, and even direct adult supervision were inadequate in preventing these injuries and have called for a total ban on the recreational, school, and competitive pediatric use of trampolines (57,99).

### Skiing Injuries

Skiing injuries are seasonal in nature and occur with outdoor winter recreational activity. In a study of major skiing injuries in children and adolescents, Shorter et al. (96) found greater than 90% of injured children to be boys 5 to 18 years of age. Sixty percent of the accidents occurred due to collisions with stationary objects such as trees, poles, and stakes. Most injuries occurred in the afternoon, among beginners, and in the first week of skiing season. Fractures accounted for one third of the total injuries sustained. The two main factors implicated in skiing injuries are excessive speed and loss of control; effective prevention efforts should target both of these factors.

### Snowboarding Injuries

Snowboarding runs a similar risk to skiing. Bladin et al. (62) found that approximately 60% of snowboarding injuries involved the lower limbs and occurred in novices. The most common injuries were sprains (53%) and fractures (26%). Compared with skiers, snowboarders had 2½ times as many fractures, particularly to the upper limb, as well as more ankle injuries such as sprains. The absence of ski poles and the fixed position of the feet on the snowboard mean that the upper limbs absorb the full impact of any fall.

### Motor Vehicle Accidents

This category includes injuries sustained by occupants of a motor vehicle and victims of vehicle-versus-pedestrian accidents.

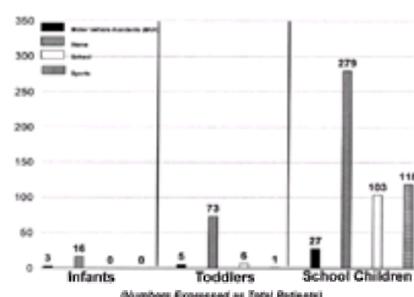
The injury patterns of children involved in motor vehicle accidents differ from those of adults. In all types of motor vehicle accidents for all ages, children constitute a little over 10% of the total number of patients injured (79,101). Of all the persons injured as motor vehicle occupants, only about 17% to 18% are children. Of the victims of vehicle-versus-pedestrian accidents, about 29% are children. Of the total number of children involved in motor vehicle accidents, 56.4% were vehicle-versus-pedestrian accidents, and 19.6% were vehicle-versus-bicycle accidents (69).

The fracture rate of children in motor vehicle accidents is less than that of adults. Of the total number of vehicle-versus-pedestrian accidents, about 22% of the children sustained fractures; 40% of the adults sustained fractures in the same type of accident. This has been attributed to the fact that children are more likely to "bounce" when hit (69).

Children are twice as likely as adults to sustain a femur fracture when struck by an automobile, but in adults tibia and knee injuries are more common in the same type of accident. This seems to be related to where the car's bumper strikes the victim (64,102). Motor vehicle accidents do produce a high proportion of spinal and pelvic injuries (64).

### Summary

The etiologic aspects of children's fractures are summarized in Fig. 1-7 and Table 1-7.



**FIGURE 1-7.** The incidence of fractures in children expressed as the four common etiologic categories. Most fractures occur at home. The numbers are expressed as total patients per each age category. (Reprinted from Worlock P, Stower M. Fracture patterns in Nottingham children. *J Pediatr Orthop* 1986;6:656; with permission.)

Home environment  
 Injuries  
 83% of all children's injuries  
 Fractures  
 37% of all children's fractures  
 School environment  
 Injuries  
 Overall rate, 2.8%–9.2% annually  
 53% related to athletic events  
 Peak age: middle-school group  
 Fractures  
 Occur in only 5%–10% of all school-related injuries  
 About 20% of all children's fractures  
 Motor vehicle accidents (MVA)  
 Injuries  
 Children only 10% of all MVAs  
 Of children's MVAs, only 17–18% were occupants; remainder were vehicle/pedestrian or vehicle/bicycle  
 Fractures  
 Higher incidence of femur fractures in vehicle pedestrian accidents in children  
 Children have a higher incidence of spinal and pelvic fractures with MVAs than with other mechanisms

**TABLE 1-7. SUMMARY OF ETIOLOGIC FACTORS IN CHILDREN'S FRACTURES**

## Less Common Etiologies

### *Ice Cream Truck*

Mubarak et al. (87) reported on ice cream truck–related accidents in which children, distracted by ice cream trucks, were struck by an oncoming vehicle, sustaining pelvic and lower limb fractures. The vision of oncoming drivers was often blocked by the large size of the ice cream truck parked by the curb.

### *Water Tubing*

Parmar et al. reported serious injuries sustained during water tubing (the pulling of an inner tube behind a power boat) (90).

### *Gunshot (Missile) Wounds: Definition*

Gunshot or missile wounds arise from objects projected into space by an explosive device. The missiles may be single or multiple. Single missiles vary from low-velocity handguns to high-velocity assault weapons. Multiple missiles can result from a shotgun blast or shrapnel from war weapons. Missile injuries represent open fractures with varying degrees of soft tissue injury. The incidence of gunshot wounds in children has become increasingly common in the United States (107).

### *Gunshot and Firearm Injuries*

In a sad reflection of the changing times and the newly pervasive gun culture, firearms are determined to be second only to motor vehicles as the leading cause of death in youths. In considering the prevalence of firearms in the United States, it has been estimated that there are about 200 million privately owned guns in the United States and that approximately 40% of U.S. households contain firearms of some type (66). The incidence of gunshot wounds in children has become increasingly common in the United States (107).

### **Etiology**

In two reports from inner-city hospitals in the United States in the 1990s, most injuries resulted from random violence to innocent bystanders; the prime example was “drive-by shootings” (101,107). Few were self-inflicted, either voluntarily or accidentally. In a 1976 report on patients in a relatively rural setting in Canada, almost all the missile injuries were accidental, having been caused by the patient or a close friend or relative (82).

In the urban setting, handguns and rifles are the most common weapons (101,104,107). In the rural setting, the most common weapon is a shotgun (82). The firepower of these weapons has changed over the years. In one urban hospital reporting gunshot wounds from 1973 to 1983, most of the injuries were from .32- or .38-caliber weapons; only 5% were high-caliber or high-velocity weapons (88). In a later study of gunshot wounds from the same institution from 1991 to 1994, the incidence of injuries from high-caliber and high-velocity weapons (e.g., .357 Magnum, AK-47, and other assault rifles) had increased to 35% (108).

In the urban setting, the victims' ages ranged from 1 to 17 years, and most of the injuries were in children 12 to 14 (88,101,104,107). In the rural setting, the patients were younger; the average age was 9 years (82).

Of 839 children sustaining gunshot wounds, 274 (32.6%) involved the extremities (88,101,104,107). Of the gunshot wounds that involved the extremities, 51.3% produced significant fractures (82,101,107). No single bone seemed to predominate, although a great majority of the fractures were distal to the elbow (88,101,104,107).

### **Complications**

The two most common complications were growth arrest and infection. Other complications include delayed union or malunion. Considering the magnitude of many of these injuries, the infection rate for extremity wounds was low (about 7.3%). This low rate was probably due to a vigorous and aggressive program of acute wound management (82,101,107). The type of missile did not seem to have any relation to the development of an infection (107).

In Letts and Miller's 1976 series, one sixth of the patients had some type of growth disturbance (82). In a third of their patients, the missile was only in close proximity to the physis. In a 1995 report by Washington and co-workers, the incidence of missiles affecting the physis was exactly the same (107). However, all were a result of a direct injury to the physis by the missile. None of their patients with growth arrest had proximity missile wounds. The higher incidence of growth abnormalities in the 1976 series was due to the larger number of shotgun and hunting rifle injuries, which dissipate more of their energy peripheral to the missile track.

In two of the studies in which patients were followed closely, all of the fractures ultimately healed (82,107).

### **Prevention**

In an excellent report in 1999, Freed et al. (74) analyzed the magnitude and implications of the increasing incidence of firearm-related injuries in children. They suggested a product-oriented approach, focusing on the gun, in an attempt to provide an efficient strategy of gun control and hence reduce the disturbing trend of firearm-related injuries and death among youths. Rather than modifying behavioral or environmental issues, which are more complex, they suggested focusing primarily on strategies that offset the accessibility and design of firearms. In brief, these strategies included the following:

Reducing the number of guns in the environment through restrictive legislation, gun buy-back programs, gun taxes, and physician counseling.

Modifying the design of guns to make them more child-proof and preventing unauthorized and unintended use.

### **Falls**

Falls can vary in severity from a simple fall while running, to a fall of great magnitude, such as from a third story window. Falls are often classified as the most common cause of injuries. Falls are more likely to be a cause of injury in the younger than in the older child. Falls in the home are usually associated with furniture or stairs; outside the home, most falls involve playground equipment (76,77).

In falling from great heights, adults often land on their lower extremities, accounting for the high number of lower extremity fractures, especially the calcaneus. Children tend to fall head first, using the upper extremities to break the fall. This accounts for the larger number of skull and radial fractures in children. Femoral

fractures are also more common in children falling from great heights. In contrast to adults, spinal fractures are rare in children who fall from great heights ([61,84,97,100](#)). In one study, children falling three stories or less all survived ([61](#)). Falls from the fifth or sixth floor resulted in a 50% mortality rate.

### **Intrinsic Causes**

#### **Nutrition**

In a study in Spain, a significant difference in the fracture rate was found when cities with a high calcium content in their water were compared with those with a lower calcium content. With all other factors being equal (e.g., fluoride content, socioeconomic background), children who lived in the cities with a lower calcium content had a higher fracture rate ([105](#)).

An increase in the consumption of carbonated beverages has been shown to produce an increased incidence of fractures in adolescents ([111](#)).

#### **Bone Density**

Bone density may be a factor, but the data are unclear. Landin and Nilsson ([80](#)) found that the mineral content of the forearms was lower in children who sustained fractures from mild trauma than in children who had never sustained fractures. It was not significantly different, however, in those sustaining fractures from severe trauma. This study used measurements of bone density of the cortical bone in the forearms. Cook and co-workers ([67](#)), using measurements of bone density obtained from trabecular bone in the spine and femoral neck, found no difference between children who had sustained fractures and those who had not.

#### **Premature Infants**

Fractures not related to birth trauma reportedly occur in 1% to 2% of low-birth-weight or premature infants during their stay in a neonatal intensive care unit ([60](#)). Using a combination of the clinical history, radiographic appearance, and laboratory data, these infants have been found to have evidence of bone loss from inadequate calcium and phosphorus intake. Correcting the metabolic status of these low-birth-weight infants, with special emphasis on calcium and phosphorus intake, appears to decrease the incidence of repeat fractures and to improve the radiographic appearance of their bony tissues. Once the metabolic abnormalities are corrected, this temporary deficiency seems to have no long-term effects. When premature infants were followed into later years, there was no difference in their fracture incidence when compared with that of children of normal birth weight ([68](#)).

## **PREVENTIVE PROGRAMS**

One of the major goals of studying the incidence of fractures is to identify problem areas; it is hoped that by targeting these areas, programs can be designed to decrease the risk factors.

#### **National Campaigns**

Several safety programs have been started by national organizations. The foremost is the American Academy of Pediatrics, which has committees on accident and poisoning prevention. This group has produced guidelines for athletics ([114](#)), playgrounds ([118,121](#)), trampolines ([112,113](#)), and skateboards ([115](#)). Recently, the American Academy of Orthopaedic Surgeons has produced a program designed to decrease the incidence of playground injuries. These programs offer background data and guidelines for various activities, but their effectiveness has not yet been fully studied.

#### **Local Community Participation**

To be effective, accident prevention programs require local participation and cooperation. They must be broad based, and they require considerable effort by members of the local community. In the United States, one effective program is the New York Health Department's "Kids Can't Fly" campaign, developed in response to the large number of injuries and deaths from children falling out of apartment house windows in the 1970s ([120](#)). This extensive program consisted of a good reporting system from hospital emergency rooms, with follow-up by public health personnel; a strong media campaign to educate the public; a door-to-door hazard identification program; and the distribution of low- or no-cost, easily installed window guards to families in high-rise apartments. The city required landlords to provide window guards in apartments where children 10 years or younger lived. The success of this program was demonstrated by a 50% decrease in reported falls after 3 years and a 96% decrease after 7 years ([116,120](#)).

Over the past 30 years, Sweden has developed broader based, community-oriented programs to decrease the incidence of all types of childhood injuries ([117](#)). The development of these pilot programs has been relatively easy in a country like Sweden because the population is homogeneous, the incidence of poverty is low, and the government is stable. The Swedish program had a three-pronged approach: injury surveillance and prevention research; establishment of a safer environment for children through legislative regulation; and a broad-based safety education campaign. These programs have produced positive results. Schelp demonstrated a 27% reduction in home accidents in the municipality of Falkoping only 3 years after the establishment of a community-wide campaign ([119](#)).

One of the most dramatic changes came in the prevention of drowning. In Sweden, 100 children drowned in 1954; by 1988, the number had decreased to 10 ([117](#)). In the same time period, there was only a 5% decrease in the number of childhood drownings in the United States. The decrease in drowning did not result from establishing new, sophisticated emergency medical services or intensive care units in hospitals; it came by teaching children to swim and promoting the use of life jackets on toddlers playing near the water. In addition, there was a public education campaign in the media locally and nationally.

Effective prevention programs require local community participation and education. All the articles, lectures, and pamphlets in the world cannot help unless local communities make the necessary changes to decrease accident risks.

## **APPENDIX A: FRACTURE COURSES**

1. The 1st Swedish International Seminar on "Treatment of Fractures in Children," Institute for Medical Postgraduate Education, Uppsala, Sweden, May 27–30, 1990
2. Seminario de Actualizacion, en "Fracturas del Niño," Madrid, Spain, October 29–30, 1994
3. Operative Management of Children's Fractures—An Interactive Course, The University of Texas Health Science Center, San Antonio, Texas, August 8–9, 1997
4. First Caribbean Children's Fracture Course, Port-au-Prince, Haiti, April 15–18, 1998
5. Third Seminario Internacional sobre Fracturas en el Niño, Madrid, Spain, October 29–30

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## **CHAPTER REFERENCES**

### **The Informational Changes**

1. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955.
2. Bohler L. *The treatment of fractures*. New York: Grune & Stratton, 1956.
3. Cotton FJ. *Dislocations and joint fractures*, 2nd ed. Philadelphia: WB Saunders, 1924.
4. Key JA, Conwell HE. *The management of fractures, dislocations & sprains*. St Louis: CV Mosby, 1951.
5. Langenskiold A. The surgical treatment of partial closure of the growth plate. *J Pediatr Orthop* 1981;1:3–17.
6. Rang M. *Children's fractures*. Philadelphia: JB Lippincott, 1974.
7. Rockwood CA Jr, Green DP, eds. *Fractures*. Vols. 1 and II. Philadelphia: JB Lippincott, 1975.
8. Rockwood CA Jr, Wilkins KE, King RE, eds. *Fractures in children*. Philadelphia: JB Lippincott, 1984.
9. Scudder CL. *The treatment of fractures*. Philadelphia: WB Saunders, 1904.

10. Stimson LA. *A practical treatise on fractures and dislocations*. New York: Lea Brothers, 1900.
11. Watson-Jones R. *Fractures and joint injuries*, 4th ed. Edinburgh, UK: E & S Livingstone, 1955.
12. Watson-Jones R, Caterall A. Fractures in children. In: Wilson JN, ed. *Fractures & joint injuries*. Edinburgh, UK: Churchill Livingstone, 1976:487.

#### Changes in the Philosophy of Treating Fractures in Children

13. Beaty JH, Austin SM, Warner WC, et al. Interlocking intramedullary nailing of femoral shaft fractures in adolescents: preliminary results and complications. *J Pediatr Orthop* 1994;14:178.
14. Chess DG, Hyndman JC, Leahey JL. Short-arm plaster for paediatric distal forearm fractures. *J Bone Joint Surg [Br]* 1987;69:506.
15. Cox PJ, Clarke NM. Improving the outcome of paediatric orthopaedic trauma: an audit of inpatient management in Southampton. *Ann R Coll Surg Eng*. 1997;79:441.
16. Lyons JP, Ashley E, Hoffer M. Ulnar nerve palsies after percutaneous cross pinning of supracondylar fractures in children's elbows. *J Pediatr Orthop* 1998;18:43.
17. Mileski RA, Garvin KL, Huurman WW. Avascular necrosis of the femoral head after closed intramedullary shortening in an adolescent. *J Pediatr Orthop* 1995;15:24.
18. Probe R, Londsey RW, Hadley NA, et al. Refracture of adolescent femoral shaft fractures: a complication of external fixation: a report of two cases. *J Pediatr Orthop* 1993;13:102.
19. Rockwood CA Jr, Wilkins KE, King RE, eds. *Fractures in children*. Philadelphia: JB Lippincott, 1984.
20. Rockwood CA Jr, Wilkins KE, Beaty JH, eds. *Fractures in children*, 4th ed. Vol III. Philadelphia: Lippincott-Raven, 1996.
21. Walker JL, Rang M. Forearm fractures in children: cast treatment with elbow extension. *J Bone Joint Surg [Br]* 1991;73:299.

#### The Present Status of the Incidence of Fractures in Children

22. Akbarnia B, Torg JS, Kirkpatrick J, et al. Manifestations of the battered-child syndrome. *J Bone Joint Surg [Am]* 1974;56:1159.
23. Andren L, Borgstrom KE. Seasonal variation of epiphysiolysis of the hip and possibility of causal factor. *Acta Orthop Scand* 1958;28:22.
24. Beekman F, Sullivan JE. Some observations on fractures of long bones in children. *Am J Surg* 1941;51:722.
25. Bisgard JD, Martenson L. Fractures in children. *Surg Gynecol Obstet* 1937;65:464.
26. Buckley SL, Gotschall C, Robertson W Jr, et al. The relationships of skeletal injuries with trauma score, injury severity score, length of hospital stay, hospital charges, and mortality in children admitted to a regional pediatric trauma center. *J Pediatr Orthop* 1994;14:449.
27. Cheng JC, Ng BK, Ying SY, et al. A 10-year study of the changes in the pattern and treatment of 6,493 fractures. *J Pediatr Orthop* 1999;19:344.
28. Cheng JC, Shen WY. Limb fracture pattern in different pediatric age groups: a study of 3350 children. *J Orthop Trauma* 1993;7:15.
29. Compere EL. Growth arrest in long bones as result of fractures that include the epiphysis. *JAMA* 1935;105:2140.
30. Dershowitz R. Is it of any practical value to identify accident-prone children? *Pediatrics* 1977;60:786.
31. Fleming DM, Charlton JR. Morbidity and health care utilization of children in households with one adult: comparative observational study. *BMJ* 1998;316:1572.
32. Hanlon CR, Estes WL. Fractures in childhood—a statistical analysis. *Am J Surg* 1954;87:312.
33. Hindmarsh J, Melin G, Melin KA. Accidents in childhood. *Acta Chir Scand* 1946;94:483.
34. Iqbal QM. Long-bone fractures among children in Malaysia. *Int Surg* 1975;59:410.
35. Izant RJ, Hubay CA. The annual injury of 15,000,000 children: a limited study of childhood accidental injury and death. *J Trauma* 1966;6:65.
36. Jones JG. The child accident repeater, a review. *Clin Pediatr* 1980;19:284.
37. Kempe CH, Silverman FN, Steele BF, et al. The battered-child syndrome. *JAMA* 1962;181:17.
38. Kowal-Vern A, Paxton TP, Ros SP, et al. Fractures in the under-3-year-old age cohort. *Clin Pediatr* 1992;31:653.
39. Laffoy M. Childhood accidents at home. *Ir Med J* 1997;90:26.
40. Landin LA. Epidemiology of children's fractures. *J Pediatr Orthop B* 1997;6:79.
41. Landin LA. Fracture patterns in children. *Acta Orthop Scand* 1983;54(suppl 202):1.
42. Landin LA, Nilsson BE. Bone mineral content in children with fractures. *Clin Orthop* 1983;178:292.
43. Lichtenberg RP. A study of 2,532 fractures in children. *Am J Surg* 1954;87:330.
44. Lopez AA, Rennie TF. A survey of accidents to children aged under 15 years seen at a district hospital in Sydney in one year. *Med J Aust* 1969;1:806.
45. Mann DC, Rajmaira S. Distribution of physeal and nonphyseal fractures in 2,650 long-bone fractures in children aged 0–16 years. *J Pediatr Orthop* 1990;10:713.
46. Masterson E, Borton D, O'Brien T. Victims of our climate. *Injury* 1993;24:247.
47. McClelland CQ, Heiple KG. Fractures in the first year of life: a diagnostic dilemma? *Am J Dis Child* 1982;136:26.
48. Mizuta T, Benson WM, Foster BK, et al. Statistical analysis of the incidence of physeal injuries. *J Pediatr Orthop* 1987;7:518.
49. Mortensson W, Thonell S. Left-side dominance of upper extremity fracture in children. *Acta Orthop Scand* 1991;62:154.
50. Reed MH. Fractures and dislocations of the extremities in children. *J Trauma* 1977;17:351.
51. Rivara FP, Bergman AB, LoGerfo JP, et al. Epidemiology of childhood injuries. II. Sex differences in injury rates. *Am J Dis Child* 1982;136:502.
52. Rohl L. On fractures through the radial condyle of the humerus in children. *Acta Chir Scand* 1952;104:74–80.
53. Routledge DA, Repett-Wright R, Howarth CI. The exposure of young children to accident risk as pedestrians. *Ergonomics* 1974;17:457.
54. Shank LP, Bagg RJ, Wagnon J. Etiology of pediatric fractures: the fatigue factors in children's fractures. Presented at National Conference on Pediatric Trauma, Indianapolis, 1992.
55. Tiderius CJ, Landin L, Duppe H. Decreasing incidence of fractures in children—an epidemiological analysis of 1673 fractures in Malmo, Sweden, 1993–1994. *Acta Orthop Scand* 1999;70:622.
56. Walking AA. End results of fractures of long bones in children. *Penn Med J* 1934:748.
57. Westfelt JARN. Environmental factors in childhood accidents: a prospective study in Goteborg, Sweden. *Acta Paediatr Scand* 1982;(suppl 291).
58. Wong PCN. A comparative epidemiologic study of fractures among Indian, Malay and Swedish children. *Med J Malaya* 1965;20:132.
59. Worlock P, Stower M. Fracture patterns in Nottingham children. *J Pediatr Orthop* 1986;6:656.

#### Etiology of Fractures

60. Amir J, Katz K, Grunebaum M, et al. Fractures in premature infants. *J Pediatr Orthop* 1988;8:41.
61. Barlow B, Neimirska M, Gandhi RP, et al. Ten years of experience with falls from a height in children. *J Pediatr Surg* 1983;18:509.
62. Bladin C, Giddings P, Robinson M. Australian snowboard injury data base study. A four-year prospective study. *Am J Sports Med* 1993;21:701.
63. Boyce WT, Boyce WT, Sprunger LW, et al. Epidemiology of injuries in a large, urban school district. *Pediatrics* 1984;74:342.
64. Buckley SL, Gotschall C, Robertson W Jr, et al. The relationships of skeletal injuries with trauma score, injury severity score, length of hospital stay, hospital charges, and mortality in children admitted to a regional pediatric trauma center. *J Pediatr Orthop* 1994;14:449.
65. Cameron M, Vulcan AP, Furich C, et al. Mandatory bicycle helmet use following a decade of helmet promotion in Victoria, Australia—an evaluation. *Accid Anal Prev* 1996;26:325.
66. Cook PJ, Ludwig J. *Guns in America*. Washington, DC: Police Foundation, 1996.
67. Cook SD, Harding AF, Morgan EL, et al. Association of bone mineral density and pediatric fractures. *J Pediatr Orthop* 1987;7:424.
68. Dahlenburg SL, Bishop NJ, Lucas A. Are preterm infants at risk for subsequent fracture? *Arch Dis Child* 1989;64:1384.
69. Derlet RW, Silva J Jr, Holcroft J. Pedestrian accidents: adult and pediatric injuries. *J Emerg Med* 1989;7:5.
70. D'Souza LG, Hynes DE, McManus F, et al. The bicycle spoke injury: an avoidable accident? *Foot Ankle Int* 1996;17:170.
71. Feldman W, Woodward CA, Hodgson C, et al. Prospective study of school injuries: incidence, types, related factors and initial management. *Can Med Assoc J* 1983;129:1279.
72. Finvers KA, Strother RT, Mohtadi N. The effect of bicycling helmets in preventing significant bicycle-related injuries in children. *Clin J Sport Med* 1996;6:102.
73. Fountain JL, Meyers MC. Skateboarding injuries. *Sports Med* 1996;22:360.
74. Freed LH, Vernick JS, Hargarten SW. Prevention of firearm-related injuries and deaths among youth. A product-oriented approach. *Pediatr Clin North Am* 1998;45:427.
75. Furnival RA, Street KA, Schunk JE. Too many pediatric trampoline injuries. *Pediatrics* 1999;103:57.
76. Gallagher SS, Finison K, Guyer B, et al. The incidence of injuries among 87,000 Massachusetts children and adolescents: results of the 1980-81 statewide childhood injury prevention program surveillance system. *Am J Pub Health* 1984;74:1340.
77. Garrettsen LK, Gallagher SS. Falls in children and youth. *Pediatr Clin North Am* 1985;32:153.
78. Jerosch J, Heidjann J, Thorwesten L, et al. Injury patterns in acceptance of passive and active injury prophylaxis for inline skating. *Knee Surg Sports Traumatol Arthrosc* 1998;6:44.
79. Landin LA. Fracture patterns in children. *Acta Orthop Scand* 1983;64(suppl 202):1.
80. Landin LA, Nilsson BE. Bone mineral content in children with fractures. *Clin Orthop* 1983;178:292.
81. Lenaway DD, Ambler AG, Beaudoin DE. The epidemiology of school-related injuries: new perspectives. *Am J Prev Med* 1992;8:193.
82. Letts RM, Miller D. Gunshot wounds of the extremities in children. *J Trauma* 1976;16:807.
83. Lillis KA, Jaffe DM. Playground injuries in children. *Pediatr Emerg Care* 1997;13:149.
84. Meller JL, Shermeta DW. Falls in urban children: a problem revisited. *Am J Dis Child* 1987;141:1271.
85. Mitts KG, Hennrikus WL. In-line skating fractures in children. *J Pediatr Orthop* 1996;16:640.
86. Mott A, Evans R, Rolfe K, et al. Patterns of injuries to children on public playgrounds. *Arch Dis Child* 1994;71:328.
87. Mubarak SJ, Lavernia C, Silva PD. Ice-cream truck-related injuries to children. *J Pediatr Orthop* 1998;18:46.
88. Ordog GJ, Prakash A, Wasserberger J, et al. Pediatric gunshot wounds. *J Trauma* 1987;27:1272.
89. Osberg JS, Schneps SE, Di Scala C, Li G. Skateboarding: more dangerous than roller skating or in-line skating. *Arch Pediatr Adolesc Med* 1998;152:985.
90. Parmar P, Letts M, Jarvis J. Injuries caused by water tubing. *J Pediatr Orthop* 1998;18:49.

91. Powell EC, Tanz RR. In-line skate and rollerskate injuries in childhood. *Pediatr Emerg Care* 1996;12:259.
92. Puranik S, Long J, Coffman S. Profile of pediatric bicycle injuries. *South Med J* 1998;91:1033.
93. Rogers GB. Bicycle helmet use patterns among children. *Pediatrics* 1996;97:166.
94. Segers MJM, Wink D, Clevers GJ. Bicycle-spoke injuries: a prospective study. *Injury* 1997;28:267.
95. Sheps SB, Evans GD. Epidemiology of school injuries: a 2-year experience in a municipal health department. *Pediatrics* 1987;79:69.
96. Shorter NA, Jensen PE, Harmon BJ, et al. Skiing injuries in children and adolescents. *J Trauma* 1996;40:997.
97. Sieben RL, Leavitt JD, French JH. Falls as childhood accidents: an increasing urban risk. *Pediatrics* 1971;47:886.
98. Smith GA. Injuries to children in the united states related to trampolines, 1990-1995: a national epidemic. *Pediatrics* 1998;101:406.
99. Smith GA, Shields BJ. Trampoline-related injuries to children. *Arch Pediatr Adolesc Med* 1998;152:694.
100. Smith MD, Burrington JD, Woolf AD. Injuries in children sustained in free falls: an analysis of 66 cases. *J Trauma* 1975;15:987.
101. Stucky W, Loder RT. Extremity gunshot wounds in children. *J Pediatr Orthop* 1991;11:64.
102. Topoleski T, Schlesinger I, Wexler LM, et al. Motor vehicle injuries in pediatric trauma patients. Presented at the American Academy of Orthopaedic Surgeons Annual Meeting, Orlando, 1995.
103. US Public Health Service. *Healthy People 2000: national health promotion and disease prevention objectives*. Washington, DC: DHSS Publication no. PH58 90-50212, 1990.
104. Valentine J, Blocker S, Chang JHT. Gunshot injuries in children. *J Trauma* 24:952, 1984.
105. Verd VS, Dominguez SJ, Gonzalez QM, et al. Association between calcium content of drinking water and fractures in children. *An Esp Pediatr* 1992;37:461.
106. Waltzman ML, Shannon M, Bowen AP, et al. Monkey bar injuries: complications of play. *Pediatrics* 1999;103:58.
107. Washington ER, Lee WA, Ross WA Jr. Gunshot wounds to the extremities in children and adolescents. *Orthop Clin North Am* 1995;26:19.
108. Weiss B. Bicycle-related head injuries. *Clin Sports Med* 1999;13:99.
109. Westfelt JARN. Environmental factors in childhood accidents: a prospective study in Goteborg, Sweden. *Acta Paediatr Scand* 1982;(suppl 291).
110. Worlock P, Stower M. Fracture patterns in Nottingham children. *J Pediatr Orthop* 1986;6:656.
111. Wyshak G, Frisch RE. Carbonated beverages, dietary calcium, the dietary calcium/phosphorus ratio, and bone fractures in girls and boys. *J Adolesc Health* 1994;15:210.

#### Preventive Programs

112. American Academy of Pediatrics, Committee on Accident and Poison Prevention: trampolines. Evanston, Illinois, September 1977.
113. American Academy of Pediatrics, Committee on Accident and Poison Prevention: trampolines II. *Pediatrics* 1981;67:438.
114. American Academy of Pediatrics, Committee on Pediatric Aspects of Physical Fitness, Recreation and Sports: competitive athletics for children of elementary school age. *Pediatrics* 1981;67:928.
115. American Academy of Pediatrics, Committee on Accident and Poison Prevention: skateboard injuries. *Pediatrics* 1989;6:1070-1071.
116. Barlow B, Neimirska M, Gandhi RP, et al. Ten years of experience with falls from a height in children. *J Pediatr Surg* 1983;18:509.
117. Bergman AB, Rivara FP. Sweden's experience in reducing childhood injuries. *Pediatrics* 1991;88:69.
118. Reichelderfer TE, Overback A, Greensher J. Unsafe playgrounds. *Pediatrics* 1979;64:962.
119. Schelp L. The role of organizations in community participation—prevention of accidental injuries in a rural Swedish municipality. *Soc Sci Med* 1988;26:1087.
120. Spiegel CN, Lindaman FC. Children can't fly: a program to prevent childhood morbidity and mortality from window falls. *Am J Dis Child* 1977;67:1143.
121. Werner P. Playground injuries and voluntary product standards for home and public playgrounds. *Pediatrics* 1982;69:18.

## THE BIOLOGIC ASPECTS OF CHILDREN'S FRACTURES

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### THE IMMATURE SKELETON

Compared with the relatively static, mature bone of adults, the changing structure and function, both physiologic and biomechanical, of immature bones make them susceptible to different patterns of failure. Even the types of fracture patterns within a given bone demonstrate temporal (chronobiologic) variations that may be correlated with progressive anatomic changes affecting the epiphysis, physis, metaphysis, and diaphysis at macroscopic and microscopic levels.

Skeletal trauma accounts for 10% to 15% of all childhood injuries ([60,128,129,131,171](#)). Fractures of the immature skeleton differ from those of the mature skeleton ([6,128,129](#)). Fractures in children are more common and are more likely to occur after seemingly insignificant trauma. Fractures may involve the various growth mechanisms: Physeal disruptions make up about 15% of all skeletal injuries in children ([128,129,131,132,157](#)). Damage involving specific growth regions, such as the physis or epiphyseal ossification center, may lead to acute or chronic growth disturbances ([127,128,166,190](#)). The physis is constantly changing, both with active longitudinal and latitudinal (diametric) growth and in mechanical relation to other components. Physeal fracture patterns vary with the extent of chondro-osseous maturation. Salter-Harris type I injuries are common in infants, and types II, III, and IV become more common as the secondary ossification center enlarges and physeal undulations develop. Joint injuries, dislocations, and ligamentous disruptions are much less common in children; it is more likely that one of the contiguous physes will be damaged. Changing trabecular and cortical structures affect metaphyseal and diaphyseal fracture patterns, and the variable size of the secondary ossification center affects susceptibility to physeal and epiphyseal injuries.

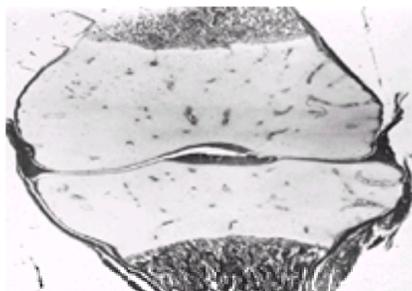
The options of treatments available for the treatment of skeletal injuries in children are expanding. Most notable is the introduction of growth factors, such as the bone morphogenic proteins (BMPs), for the induction of bone formation either in non-healing defects or for bone fusions. It has become necessary for the orthopaedic surgeon to have a good knowledge of the biological aspects of fracture repair. This chapter covers the basic biology of bone growth and fracture repair, including the roles of growth factors and the extracellular matrix.

### ANATOMIC REGIONS OF THE CHILD'S BONE

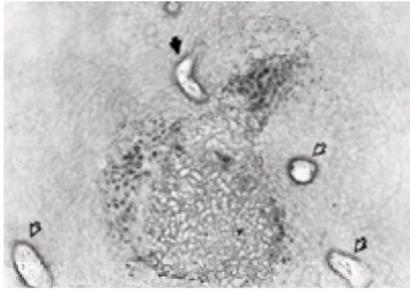
The major long bones of children can be divided into four distinct, constantly changing anatomic areas: the epiphysis, physis, metaphysis, and diaphysis ([86](#)). Each region is prone to certain patterns of injury; the intrinsic susceptibility changes with physiologic and biomechanical changes during postnatal development. The four regions originate and become modified as a result of the basic endochondral ossification process. Subsequently, they are supplemented by membranous bone formation along the metaphyseal and diaphyseal shafts. Finally, the regions are remodeled to create mature cortical and trabecular bone.

#### Epiphysis

At birth, each epiphysis (except the distal femur) consists of a completely cartilaginous structure at the end of each long bone ([Fig. 2-1](#)), the chondroepiphysis. The corresponding ossifying structure is the chondro-osseous epiphysis. At a time characteristic for each of these chondroepiphyses, a secondary center of ossification forms and gradually enlarges until the cartilaginous area has been almost completely replaced by bone at skeletal maturity. This chondro-osseous transformation is vascular-dependent ([Fig. 2-2](#)). Only articular cartilage remains at maturity.



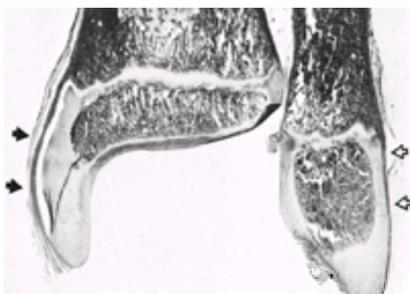
**FIGURE 2-1.** Chondroepiphyses of the distal femur and proximal tibia. These structures have an extensively developed vascular system (cartilage canals) before secondary ossification.



**FIGURE 2-2.** Early formation of the secondary ossification center within the epiphyseal cartilage. This usually occurs in a region well vascularized by cartilage canals (*open arrows*). One of the canals sends a branch into the hypertrophic cells (*solid arrow*), triggering the ossification process.

As the ossification center expands, it undergoes structural modifications. The region adjacent to the physis forms a distinct subchondral plate parallel to the metaphysis, creating the radiographically characteristic lucent physeal line. The appearance of the ossification centers differ in certain chondroepiphyses, a factor that must be considered when diagnosing fractures of these regions. The ossification center imparts increasing rigidity to the more resilient epiphyseal cartilage as the secondary osseous tissue expands ([176](#)).

The external surface of an epiphysis is composed of either articular cartilage or perichondrium ([Fig. 2-3](#)). Muscle fibers, tendons, and ligaments may attach directly to the perichondrium, which is densely contiguous with the underlying hyaline cartilage. The perichondrium contributes to the continued centrifugal enlargement of the epiphysis. It also blends imperceptibly into the periosteum. This perichondrial/periosteal tissue continuity contributes to the biomechanical strength of the epiphyseal/metaphyseal junction at the zone of Ranvier.



**FIGURE 2-3.** As the epiphysis matures, the ossification center expands and progressively follows the contours of the chondroepiphysis. The epiphyseal surface is either articular cartilage or perichondrium along the outer surfaces, as in the medial (*solid arrows*) and lateral (*open arrows*) malleoli.

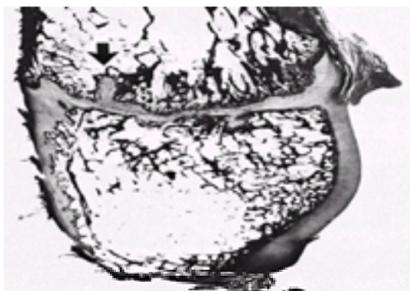
When the hyaline cartilage of the chondroepiphysis first forms, there are no easily demonstrable histologic differences between the cells of the joint surface and the rest of the epiphyseal cartilage. However, at some point, a finite cell population becomes stabilized and physiologically different from the remaining epiphyseal cartilage. McKibbin ([104](#)) established that these two cartilage types are different physiologically and, by implication, biochemically. If a contiguous core of articular and hyaline cartilage is removed, turned 180 degrees, and reinserted, the transposed hyaline cartilage eventually will form bone at the joint surface, whereas the transposed articular cartilage remains cartilaginous and becomes surrounded by the enlarging secondary ossification center. Normally, articular cartilage does not appear capable of calcification and ossification. As skeletal maturity is reached, a tide mark progressively develops as a demarcation between the articular and calcified epiphyseal hyaline cartilage.

An important aspect of McKibbin's experiment was an explanation of nonunion of certain fractures in which the fragment may be rotated, causing the articular surface to lie against metaphyseal and epiphyseal bone. Union is unlikely in such a situation because the articular surface is incapable of a reparative osteogenic response, an essential component of bone healing.

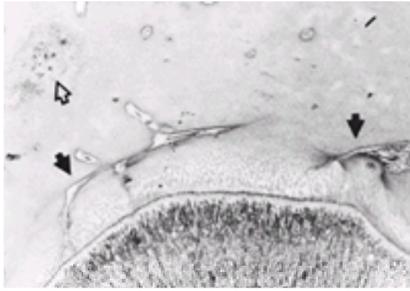
### Physis

The growth plate, or physis, is the essential structure adding bone through endochondral ossification ([121,126,130,166](#)). The primary function of the physis is rapid, integrated longitudinal and latitudinal growth. Injuries to this component are unique to skeletally immature patients.

Because the physeal cartilage remains radiolucent, except for the final stages of physiologic epiphysiodesis, its exact location must be inferred from the metaphyseal contour, which follows the physeal contour. The changing size of the secondary ossification center more effectively demarcates the physeal contour on the epiphyseal (germinal layer) side. As this center of ossification enlarges centrifugally to approach the physis, the originally spherical shape of the ossification center flattens and gradually develops a contour paralleling the metaphyseal contour. Similar contouring also occurs as the ossification center approaches the lateral and subarticular regions of the epiphysis ([Fig. 2-4](#)). The region of the ossification center juxtaposed to the physis forms a discrete subchondral bone plate that the essential epiphyseal blood vessels must penetrate to reach the physeal germinal zone ([Fig. 2-5](#)). Damage to this osseous plate in a fracture may cause localized physeal ischemia.

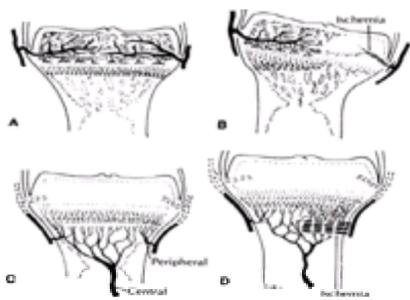


**FIGURE 2-4.** Distal fibula, showing the variably undulated physis, including a mammillary process (*arrow*). The physeal and epiphyseal cartilage turns proximally at the medial region (lappet formation) to participate in the formation of the distal tibiofibular articulation. Note the difference in the subarticular subchondral bone, which has formed a thick plate, compared with the thin, outer subchondral bone.

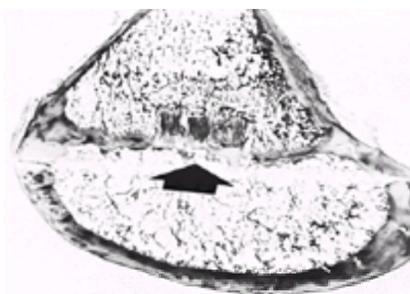


**FIGURE 2-5.** Epiphyseal circulation (*solid arrows*) in a toddler. These supply the germinal/dividing zones of the physis. The open arrow indicates the early ossification center. As this area enlarges, it will incorporate the epiphyseal vessels.

If a segment of the epiphyseal vasculature is compromised, whether temporarily or permanently, the zones of cellular growth associated with these particular vessels cannot undergo appropriate cell division. In contrast, unaffected regions of the physis continue longitudinal and latitudinal growth, leaving the affected region behind ([Fig. 2-6](#) and [Fig. 2-7](#)). The growth rates of the cells directly adjacent to the affected area are more mechanically compromised than cellular areas farther away. The differential rather than uniform growth results in an angular or longitudinal growth deformity, or both ([24,132](#)).



**FIGURE 2-6.** Patterns of response to ischemia of the epiphyseal (**A,B**) versus metaphyseal (**C,D**) circulatory systems. Metaphyseal ischemia is usually transient; epiphyseal ischemia is usually severe and permanent.



**FIGURE 2-7.** Histologic section showing an area of central ischemic growth arrest (*arrow*). The infarcted area of cartilage is left behind as the rest of the physis continues longitudinal growth.

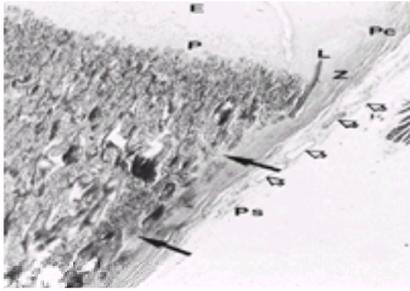
Interruption of the metaphyseal circulation has no effect on chondrogenesis within the germinal zone or the sequential cartilage maturation within the hypertrophic zone of the physis (see [Fig. 2-6](#)). However, the subsequent transformation of cartilage to bone (primary spongiosa) is blocked ([182](#)). This causes widening of the affected area, because more cartilage is added to the cell columns but none is replaced by invasive metaphyseal vessels and bone. Once the disrupted metaphyseal circulation is reestablished, this widened, calcified region of the physis is rapidly penetrated and ossified, returning the physis to its normal width. This is the mechanism seen in growth plate fractures and in fractures of the metaphysis. The metaphyseal blood supply is temporarily blocked by separation or impaction, and requires 3 to 4 weeks for restoration. If the circulatory compromise has been caused by a metaphyseal fracture, there also may be a temporary halt to bone formation in the transiently ischemic portion of the metaphysis. This leads to an apparent sclerosis when the bone is compared with the adjacent vascularized metaphysis, which undergoes a relative disuse osteoporosis. Compromise of the metaphyseal circulation has minimal, if any, effect on physal development, particularly when compared with the major detrimental effects of epiphyseal circulatory compromise.

The effects of physal ischemia have been studied extensively by Trueta and coworkers ([180,181,182](#) and [183](#)). Disrupting the epiphyseal circulation leads to either partial or complete cessation of growth. The central region seems more sensitive to ischemia than the periphery, which may have a variable capacity to recover through continued latitudinal growth ([112,125](#)). Ischemic compromise leads to different rates of growth across the affected physis and significant changes in physal contour ([19](#)). Some changes may be caused by venous stasis rather than arterial damage ([78](#)).

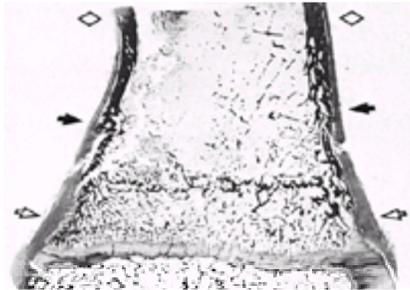
### Metaphysis

The metaphysis is a variably contoured flare at each end of the diaphysis. Its major characteristics are decreased thickness of the cortical bone and increased trabecular bone in the secondary spongiosa. Extensive endochondral modeling centrally and peripherally initially forms the primary spongiosa, which then is remodeled into the more mature secondary spongiosa, a process that involves osteoclastic and osteoblastic activity. The metaphyses exhibit considerable bone turnover compared with other regions of the bone, and this factor is responsible for the increased uptake of radionuclides in technetium 99m bone scans ([105](#)).

The metaphyseal cortex also changes with time. Compared with the confluent diaphysis, the metaphyseal cortex is thinner and is more porous (trabecular fenestration; [Fig. 2-8](#)). These cortical fenestrations contain fibrovascular soft tissue elements that connect the metaphyseal marrow spaces with the subperiosteal region. The metaphyseal cortex exhibits greater fenestration near the physis than in the diaphysis, with which it gradually blends as an increasingly thicker, dense bone ([Fig. 2-9](#)). As temporal longitudinal growth continues, cortical fenestration becomes a less dominant feature, and the overall width of the cortex increases, creating a greater morphologic transition between the juxtaphyseal and juxtadiaphyseal cortices. The metaphyseal region does not develop extensive secondary and tertiary haversian systems until the late stages of skeletal maturation. These microscopic anatomic changes appear to be directly correlated with changing fracture patterns and are the reason why torus (buckle) fractures are more likely to occur than complete metaphyseal or epiphyseal/physal fractures.



**FIGURE 2-8.** Cortical fenestration (*solid arrows*) of a metaphysis. Note the interdigitation of periosteal (Ps) tissue with the fenestrations. The periosteum blends into the periochondrium (Pc). Extensive vascularity is often present in this region (*open arrows*). (E, epiphysis; P, physis; Z, zone of Ranvier; L, ring of Lacroix.)



**FIGURE 2-9.** Section of distal tibia showing the transition (*solid arrows*) of cortical bone from the dense, remodeled diaphysis (diamonds) to the fenestrated metaphysis (*open arrows*). Note the progressive change from a relatively thin periosteum over the diaphysis to a much thicker one at the metaphysis.

Another microscopic anatomic variation in the metaphysis occurs at the junction of the primary spongiosa and the hypertrophic region of the physis. In most rapidly growing bones, the trabeculae tend to be longitudinally oriented. However, in shorter growing bones, such as the metacarpals and phalanges, trabecular formation is predominantly horizontal. As growth decelerates in adolescence, a similar horizontal orientation may be seen in the major long bones. These variations in trabecular orientation affect the responsiveness of metaphyseal and physeal regions to abnormal stress and predispose to certain fracture modes.

Although the periosteum is attached relatively loosely to the diaphysis, it is firmly fixed to the metaphysis because of the increasingly complex continuity of fibrous tissue through the metaphyseal fenestrations. Such intermingling of endosteal and interosseous fibrous tissues with the periosteal tissue imparts additional biomechanical strength to the region (170). The periosteum subsequently attaches densely into the peripheral physis, blending into the zone of Ranvier as well as the epiphyseal perichondrium. The fenestrated metaphyseal cortex extends to the physis as the thin osseous ring of Lacroix.

The metaphysis is the site of extensive osseous modeling and remodeling, both peripherally and centrally (Fig. 2-10). The metaphyseal cortex is fenestrated, modified trabecular bone on which the periosteum deposits membranous bone to thicken the cortex progressively. Similar endosteal bone formation occurs. As this metaphyseal region thickens, the trabecular bone is progressively invaded by diaphyseal osteon systems, not unlike osteons traversing the fracture site in primary bone healing. This converts peripheral trabecular (woven or fiber) bone to lamellar (osteonal) bone, which has different biomechanical capacities, and thus progressively transforms metaphyseal cortex into diaphyseal cortex as longitudinal growth continues. A torus (buckle) fracture is most likely to occur in a metaphyseal region with a trabecular, fenestrated, compressible cortex.



**FIGURE 2-10.** Extensive modeling and remodeling of the medial (M) versus the lateral (L) cortex of the distal femur may create irregularities that have been misinterpreted as fracture, stress fracture, infection, and tumor. Note the well-formed subchondral bone at the periphery of the epiphyseal ossification center.

As in the diaphysis, there are no significant direct muscle attachments to the metaphyseal bone. Instead, muscle fibers primarily blend into the periosteum. The medial distal femoral attachment of the adductor muscles is a significant exception. Because of extensive remodeling and insertion of muscle and tendon in this area, the bone often appears irregular and may be misinterpreted as showing chronic trauma (i.e., a stress fracture), infection, or a tumor.

#### ***Transverse Lines of Park and Harris***

Many bones exhibit transversely oriented, dense trabecular linear bone patterns within the metaphysis. These lines usually duplicate the contiguous physeal contour. They may appear after trauma, particularly when the child has been immobilized in bed (e.g., traction for femoral fracture), and they also may appear after generalized illnesses or even localized processes within the bone (e.g., osteomyelitis) (1,62,137,138). The lines result from a temporary slowdown of normal longitudinal growth after injury or illness, and they often are called Harris growth slowdown or arrest lines (Fig. 2-11). Because of the slowdown, the trabeculae of the primary spongiosa become more transversely than longitudinally oriented, creating a temporary thickening in the primary spongiosa adjacent to the physis. Once the normal longitudinal growth rate resumes, longitudinal trabecular orientation is restored. The thickened, transversely oriented osseous plate is left behind, to be gradually remodeled as primary spongiosa becomes secondary spongiosa.



**FIGURE 2-11.** Histologic section **(A)** and x-ray study **(B)** of a distal femur showing a typical Harris line (*arrows*). This formed during an acute illness and chemotherapy for leukemia. The child then resumed a more normal pattern of growth until her death from leukemia about 14 months later.

Usually, transverse lines are distributed relatively symmetrically through the skeleton and occupy identical sites in the corresponding bones on the two sides of the body. They are thickest in metaphyses that grow most rapidly, such as the distal femur and proximal tibia. In the metaphyses with slowest growth, they may not form at all, or they are exceedingly thin and lie at the very end of the shaft, directly under the provisional zone of calcification. These transverse lines parallel the contours of the physal provisional zone of calcification. When several transverse lines are present, they tend to be parallel. The lines nearest the end of the shaft ordinarily are the thickest and widest; lines away from the physes tend to be thinner and less distinct and are usually broken and irregular. As they eventually become part of the elongating diaphysis, they disappear completely with endosteal remodeling.

Park (137,138) found that temporary longitudinal growth arrest of a bone was a prerequisite for the formation of a transverse line. During this initial phase of growth stoppage or slowdown, a thin, transverse, osseous template was formed along the zone of proliferative cartilage. When longitudinal growth in the proliferating cartilage resumed (the recovery phase), the process became visible radiographically as a transverse line.

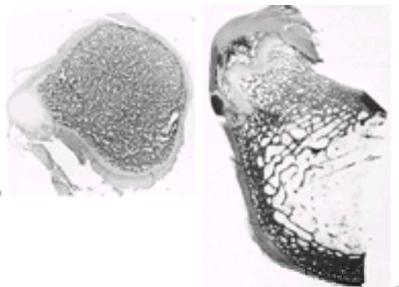
More rapidly growing bones are associated with longitudinally oriented trabeculae in the juxtaphyseal region; slower growing bones, particularly the proximal radius, metacarpals, metatarsals, and phalanges, normally have a greater amount of transversely oriented primary spongiosa (118), making transverse septa a normal finding. These particular bones do not have a sufficient difference in the orientation of trabeculae to manifest transverse lines on radiographs. However, if growth slows in the rapidly growing areas normally characterized by longitudinal orientation of trabeculae (e.g., distal femur), then more primary spongiosa bone is formed in a transverse orientation (127). This bone can be quite thick, and probably relates to the duration of the biologic stress. Once normal rates of longitudinal growth and trabecular orientation are reestablished, the transversely oriented septal, juxtaphyseal plate is a contrast to the preexisting longitudinally oriented trabeculae and appears on radiographs as a specific transverse line. As remodeling occurs, with migration of the epiphysis away from this region, and with conversion of primary spongiosa to secondary spongiosa, there is a gradual breakup of this transverse trabecular orientation.

#### Useful to Assess Growth After Injury

These biologic marker lines are important in analyzing the effects of a fracture on growth. They can be measured and the sides compared to corroborate femoral overgrowth after diaphyseal fracture and eccentric overgrowth medially after proximal tibial metaphyseal fracture. A line that converges toward a physis suggests localized growth damage that may result in an osseous bridge and the risk of angular deformity.

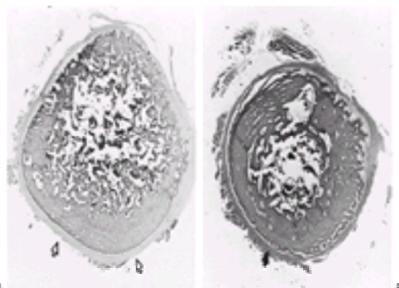
#### Diaphysis

The diaphysis constitutes the major portion of each long bone. It is principally a product of periosteal, membranous osseous tissue apposition on the original endochondral model. This leads to the gradual replacement of the endochondrally derived primary ossification center and primary spongiosa; the latter is replaced by secondary spongiosa in the metaphyseal region. At birth, the diaphysis is composed of laminar (fetal, woven) bone that characteristically lacks haversian systems. The neonatal femoral diaphysis appears to be the only area exhibiting any significant change from this fetal osseous state to a more mature bone with osteon systems (lamellar bone) before birth (Fig. 2-12).



**FIGURE 2-12.** Sections of the femur at the level of the lesser trochanter at birth **(A)** and age 7 years **(B)**. At birth, some cortical thickening and osteon remodeling is evident laterally; the rest of the cortex is irregular. By age 7 years, extensive thickening and remodeling of the cortex has taken place.

Periosteum-mediated, membranous, appositional bone formation with concomitant endosteal remodeling leads to enlargement of the overall diameter of the shaft, variably increased width of the diaphyseal cortices, and formation of the marrow cavity. Mature, lamellar bone with intrinsic but constantly remodeling osteonal patterns progressively becomes the dominant feature (Fig. 2-13).



**FIGURE 2-13.** Transverse sections of the tibial diaphysis in a neonate **(A)** and at age 2 years **(B)**. A thick periosteum is evident in **A** (*open arrows*), in association with a rapidly forming anterior cortex. At age 2 years, new subperiosteal (membranous) bone is being added to the cortex (*solid arrow*).

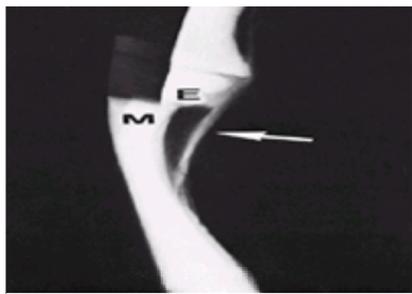
The developing diaphyseal bone in a neonate or young child is extremely vascular. When analyzed in cross section, it appears much less dense than the maturing bone of older children, adolescents, and adults. Subsequent growth leads to increased complexity of the haversian (osteonal) systems and the formation of increasing amounts of extracellular matrix, causing a relative decrease in cross-sectional porosity and an increase in hardness, factors that constantly change the child's susceptibility to different fracture patterns. Certain bones, especially the tibia, exhibit a significant decrease in vascularity as the bone matures; this factor affects the rate of healing and risk of nonunion.

The vascularity of the developing skeleton constantly changes. In experimental studies, significant chronobiologic changes in flow patterns were found in the developing canine tibia and femur ([89,90,105,106,161](#)). In particular, there was a dramatic decrease in tibial circulation with increasing skeletal maturation. This also occurs in humans, which helps to explain the increasing delay in fracture healing and the increased incidence of nonunion of the tibia in adolescents and adults. A poor vascular response could impair the early, crucial stages of callus formation.

Other researchers have suggested that adequate vascularity was a major factor in fracture healing, ([150,151,184,190,194](#)), but they did not consider chronobiologic changes in blood flow patterns.

### The Periosteum

A child's periosteum is thicker, is more readily elevated from the diaphyseal and metaphyseal bone, and exhibits greater osteogenic potential than that of an adult ([126](#)). The periosteum is loosely attached to much of the shaft of the bone, but it attaches densely into the physeal periphery (the zone of Ranvier; [Fig. 2-14](#)) through intricate collagen meshworks, thereby playing a role in fracture mechanics and treatment of growth mechanism injuries ([170](#)). The thicker, stronger, more biologically active periosteum affects fracture displacement, reduction, and the rate of subperiosteal callus formation. It also may serve as an effective internal restraint in closed reductions.



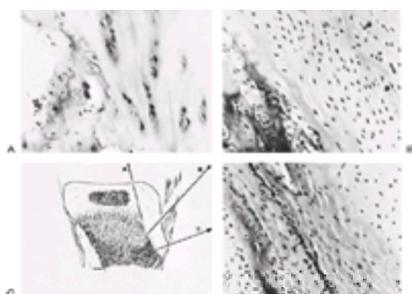
**FIGURE 2-14.** Simulated type 1 epiphyseal (E) displacement from the metaphysis (M). Note the thick periosteum (*arrow*) and its contiguity with the cartilage of the epiphysis (radiopaque here because of the cartilage and air contrast). In the body, however, the similar soft tissue radiodensities of cartilage, ligament, muscle, and so forth blend together, making them radiolucent.

Because of its contiguity with the underlying bone, the periosteum is usually injured to some extent in all fractures in children. However, because the periosteum more easily separates from the bone in children, there is much less likelihood of complete circumferential rupture. A significant portion of the periosteum usually remains intact on the concave (compression) side of an injury. This intact periosteal hinge or sleeve may lessen the extent of displacement of the fracture fragments, and it also can be used to assist in the reduction, because the intact portion contributes to the intrinsic stability. Because the periosteum allows some tissue continuity across the fracture, the subperiosteal new bone that it forms quickly, bridges the fracture gap and leads to more rapid long-term stability. The periosteum may be specifically damaged, with or without concomitant injury to the contiguous bone. Such avulsion injuries may lead to the formation of ectopic bone ([120](#)). In contrast, severe disruption of the periosteum, as in an open injury, may impair the fracture healing response. Complete loss of a bone segment, with the periosteal sleeve reasonably intact, may be followed by complete reformation of the missing bone ([16](#)).

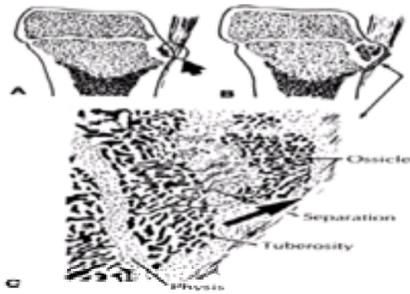
The periosteum, rather than the bone itself, serves as the origin for most muscle fibers along the metaphysis and diaphysis. This mechanism allows coordinated growth of bone and muscle units; this would be impossible if all the muscle tissue attached directly to the developing bone or cartilage. Exceptions include the attachment of muscle fibers near the linea aspera and into the medial distal femoral metaphysis. The latter pattern of direct metaphyseal osseous attachment may be associated with significant irregularity of cortical and trabecular bone. Radiographs of this area often are misinterpreted as showing a neoplastic, osteomyelitic, or traumatic response, even though they exhibit only a variation of skeletal development.

### Apophysis

Because of the differing histologic composition of the tibial tuberosity (fibrocartilage instead of columnar cartilage; [Fig. 2-15](#)), failure patterns differ from those in other physes. This area develops primarily as a tensile-responsive structure (i.e., an apophysis). However, the introduction of an osseous secondary ossification center initially in the distal tuberosity interposes osseous tissue, which tends to fail in tension and which may lead to avulsion of part of this ossification center ([Fig. 2-16](#)). Healing of the displaced fragment to the underlying undisplaced secondary center creates the symptomatic reactive overgrowth known as an Osgood-Schlatter lesion ([119,123](#)). Similarly, in adolescents, excessive tensile stress may avulse the entire tuberosity during the late stages of closure ([124](#)).



**FIGURE 2-15.** Histology of a typical apophysis, the tibial tuberosity (tubercle). **A:** Attenuated columnar cartilage adjacent to the main proximal tibial physis. **B:** Fibrocartilage and minimal hypertrophic matrix in the mid-tuberosity region. **C:** Fibrocartilage and membranous ossification in the distal end of the tuberosity.



**FIGURE 2-16.** Avulsion (tension) failure of the developing ossification center of an apophysis. The degree of displacement determines the likelihood of healing and the symptoms and size of the final lump, typical of an Osgood-Schlatter injury.

## THE MOLECULAR BONE

### Molecules of the Cartilage and Bone Matrices

#### The Cartilage Matrix

The cartilage matrix is synthesized by chondrocytes. The main constituents of the cartilaginous matrix are collagens (mainly type II) and proteoglycans. Although collagen type II provides structural strength, the proteoglycans have structural and regulatory effects. The structural effects of proteoglycans arise through binding to the collagen components and the water-binding properties that provides resilience to compression. Regulatory effects include growth factor interactions, cell matrix interactions, and regulation of collagen fibril size. Specific molecules expressed and their functions are listed in [Table 2-1](#).

Component	Site of Expression Within Physic and Proposed Functions
Collagens	
Collagen II (IIa)	Proteoglycan collagen of all cartilage
Collagen IX	Proliferative zone of the phys
Collagen X (short chain collagen)	Hypertrophic cartilage
Collagen XI (IIa)	Proliferative and hypertrophic zone of the phys
Proteoglycans	
Aggrecan	Throughout cartilage
Decorin (DS-PG2)	Within chondrocytes and the interterritorial capsules of the upper proliferative chondrocytes
Biglycan (DS-PG1)	Territorial capsules of the upper proliferative chondrocytes
Fibromodulin	
Matrix Gla protein	Cartilage

**TABLE 2-1. MATRIX MOLECULES OF CARTILAGE**

#### The Bone Matrix

Except for a small percentage of molecules from the circulation and preexistent matrices that may become entrapped, the bone matrix is almost entirely synthesized by osteoblasts. The composition of the bone matrix was outlined by Buckwalter and associates (20). Briefly, bone matrix is a composite material composed of an inorganic (mineral) portion and an organic portion. The composite structure provides physical strength and resilience to fracture. Bone with deficient inorganic mineral content is pliable, and bone with deficient organic content is brittle.

The composition of living bone is 60% to 70% inorganic components, 5% to 8% water, and the remainder is organic (76). The inorganic portion is mainly hydroxyapatite, with some carbonate and acid phosphate groups. It has also been suggested that bone crystals do not contain hydroxyl groups and should be termed apatite rather than hydroxyapatite (20). The organic portion is composed of collagen type I (90%) and noncollagenous proteins. The noncollagenous protein portion includes a number of proteins and proteoglycans that perform structural and regulatory functions. Actual molecules and functions are outlined in [Table 2-2](#) and in the following section.

Component	Proposed Functions
Collagens	
Collagen I	Imparts strength, site of initial mineralization
Collagen V	Provide the inner core of the collagen fibril (8, 46)
Collagen VI	Cell attachment
Collagen XII	Collagen fibril size
Proteoglycans	
Decorin (DS-PG2)	Collagen fibril size, TGF- $\beta$ activity (162, 163)
Biglycan (DS-PG1)	Collagen fibril assembly, TGF- $\beta$ activity (164, 195)
Fibromodulin	Collagen fibril diameter, binding of cells to matrix molecules (66)
Osteocalcin (bone Gla protein)	Binds hydroxyapatite (146, 387)
Matrix Gla protein	Controls mineralization (92, 146)
Osteonectin	Binds calcium (11)
Osteopontin	Cell attachment (102)

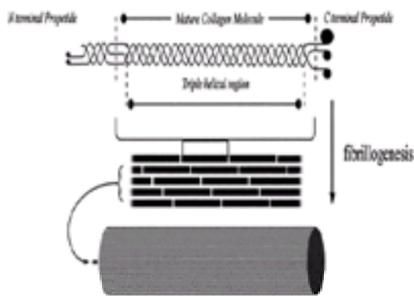
**TABLE 2-2. COMPOSITION OF BONE**

#### Matrix Constituents

Although it is not a complete list, the following provides an example of the major proteins found within bone and cartilage matrices.

#### Collagens

Collagens are a family of proteins coded by at least 19 distinct genes. Members are expressed in most tissues. Collagens have a triple helical region that arise from the repeated winding of three collagen molecules around a common axis. Collagens are synthesized as a propeptide that is often glycosylated. Collagen is secreted from cells and is processed in the extracellular space. The processed collagen forms into subunits that then undergo fibrillogenesis (Fig. 2-17). The fact that the final fiber is composed of many individual molecules accounts for the observed dominant negative mutations that can be observed within the collagen family (74). The incorporation of individual molecules that contain mutations that affect the packing of the peptides into the triple helix can disturb the structure of the whole fiber. The molecular structures that arise are in the form of fibrils or netlike structures. In reality, the multimeric fibers observed *in vivo* are often composed of a number of different collagens (5).



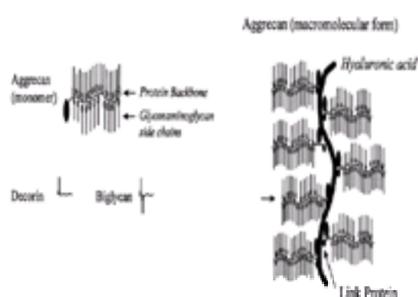
**FIGURE 2-17.** Collagens are synthesized as a propeptide that is often glycosylated (not shown). The collagen molecule has a triple helical region that arises from the repeated winding of three collagen molecules around a common axis. The processed collagen forms into subunits that then undergo fibrillogenesis.

Collagen type I is the main collagen found in bone and other tissues. It is composed of two  $\alpha 1(I)$  and one  $\alpha 2(I)$  polypeptides. The collagen type I fibers act as sites for initial mineralization and provide tensile strength to the bone. Mutations in the propeptides can cause a variety of phenotypes affecting mineralization and bone fragility, the most severe being osteogenesis imperfecta. In contrast, collagen type II is a triple helical molecule that is composed of three  $\alpha 1(II)$  polypeptides and is expressed within cartilage. It is the main fibril-forming collagen in cartilage. Mutations cause Langer-Saldino achondrogenesis and spondyloepiphyseal dysplasia congenita (27,45).

Other collagen types, such as V, IX and XI, associate with the collagen fibers. They may influence collagen diameters and interact with other matrix molecules. Mutations in types IX and XI can result in a number of clinical manifestations (134). Collagen type X is associated with the matrix of hypertrophic chondrocytes and is involved with the mineralization process (80,81,139). Mutation causes spondylometaphyseal dysplasia (74), but the deletion of the encoding gene results in mild changes (73,155).

### Proteoglycans

Proteoglycans are present in large amounts within all connective tissues. Proteoglycans are proteins that have either one or a number of polysaccharide chains linked to a protein core. The polysaccharide's glycosaminoglycan side chains are either heparin, heparin sulfate, chondroitin sulfate, dermatan sulfate, or keratan sulfate. The glycosaminoglycans differ in the composition of their constituent disaccharide structures. They can combine with other molecules within the matrix to form macromolecular structures (49) (Fig. 2-18).



**FIGURE 2-18.** Proteoglycans are proteins, which have either one or a number of polysaccharide (glycosaminoglycan) chains linked to a protein core. Aggrecan is present in cartilage and has the ability to form macromolecular structures with hyaluronic acid and link protein. Decorin and biglycan are present in bone and cartilage matrix.

Proteoglycans are a critical component of cartilage and bone (23,113,144). The proteoglycans present in the physis include large proteoglycans like aggrecan as well as smaller proteoglycans such as decorin, biglycan, and possibly, fibromodulin. Decorin and biglycan have side chains of dermatan sulfate, and betaglycan has chondroitin and heparin sulfate chains. Fibromodulin has side chains of keratan sulfate. The territorial capsules of the chondrocytes in the upper proliferative region of the physis stains for biglycan, the interterritorial matrix stains for decorin (7). These proteoglycans have a structural role but are also known to interact with growth factors (7,67,144).

### Other Noncollagenous Proteins

Osteocalcin is also known as bone Gla protein. It has three residues of gamma-carboxyglutamic acid that enable it to bind to hydroxyapatite. It is thought to play a role in mineralization of the bone matrix (146,147), but the exact mechanism and function are undetermined (35,63).

Osteonectin has the ability to bind calcium and collagen type I, and may enable the process of mineralization that is initiated on the collagen type I fibers (11).

Osteopontin is thought to be critically involved with the binding of osteoclasts (70,149), cells that degrade to the bone matrix (103).

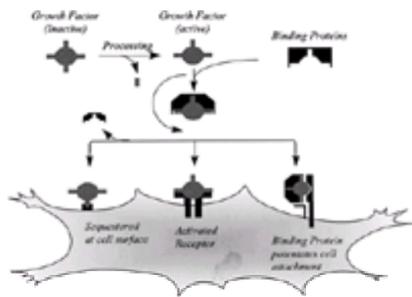
Matrix Gla protein is an inhibitor of calcification. The cartilage of mice lacking this protein undergoes spontaneous calcification (93).

### Growth Factors

Within an individual, cell-to-cell communication occurs between neighboring cells and between cells that are separated by an almost complete body length. Communication signals take the form of diffusible molecules which pass between the cells or by cell surface-bound receptor-ligand interactions (88,193). In addition, neighboring cells can pass information between one another via their gap junctions (48). These channels enable the passage of small molecules, including calcium ions, between neighboring cells. Calcium is a key second messenger that provokes a number of cellular events (110).

Hormones are a group of diverse molecules that are secreted by endocrine glands and are transported to their effect target tissues by body fluids. They coordinate body functions in complex organisms. Hormones can be in the form of amino acid derivatives (e.g., epinephrine) polypeptides (e.g., somatotropin or growth hormone), glycoproteins (e.g., follicle-stimulating hormone), steroids (e.g., testosterone), or fatty acids (e.g., prostaglandins).

Growth factors and hormones may circulate in a free form or be bound to carrier molecules or the extracellular matrix (136). The binding of growth factors and hormones to other molecules may result in inhibition of the degradation, delivery, and controlling of activity. Many growth factors, including the fibroblast growth factors, transforming growth factor- $\beta$  (TGF- $\beta$ ), and insulin-like growth factors, can be bound to the matrix. Cell activation usually requires the factors to bind to receptors on the cell surface, although a number of hydrophobic hormones pass directly through the outer membrane and bind to intracellular receptors (31,44,99,116) (Fig. 2-19).



**FIGURE 2-19.** The figure shows aspects of growth factor interactions. Any particular growth factor will possess only a subset of such interactions. Growth factors may require activation (e.g., TGF- $\beta$ ). Binding proteins may sequester or protect the growth factor. The binding protein may also potentiate the binding of the growth factor to the surface receptor (e.g., FGF and heparin). Cells may also sequester the growth factor at the cell surface.

A degree of redundancy often exists in that a gene knockout for one particular growth factor may result in only slight changes in the phenotype observed. A good example is the double mutant of BMP-5 and 7, which is lethal during embryonic development, but a null mutation in either one has little effect ( [169](#)).

### Fibroblast Growth Factors

The biologic effects of the fibroblast growth factors are widespread. Fibroblast growth factors are angiogenic and can influence mitosis and differentiation in many cell types. The receptors to these growth factors have been implicated in a number of skeletal deformities including Pfeiffer's syndrome (FGFR1), Crouzon's and Jackson-Weiss syndromes (FGFR2), and achondroplasia (FGFR3).

To date, the fibroblast growth factor family comprises at least nine members including acidic fibroblast growth factor (FGF-1), basic fibroblast growth factor (FGF-2) ([10,15,47,97,111,156,172,174,199](#)). Additional fibroblast growth factors exist that have far less homology. FGF-1 and FGF-2 are present in the extracellular matrix of bone ([64](#)).

The FGFs are also complicated by the presence of alternative forms of the specific forms of FGF-1 and FGF-2. FGF-1 is typically 140 amino acids in length, but larger forms of 160 and 154 amino acids have been identified ([27,43,53,61](#)). FGF-2 is normally translated as an 155 amino acid molecule, but through the use of alternative start codons, another three higher molecular weight forms have been identified.

The acidic and basic forms of FGFs are well conserved across species. Comparing the amino acid composition of FGF-1 and FGF-2 from different species, Hearn found a 92% sequence identity between human and bovine acidic fibroblast growth factor. Only 2/155 and 3/155 amino acids differ in human and bovine, and human and ovine, forms of basic fibroblast growth factor, respectively ([65](#)).

Six receptor molecules have been identified so far. FGF receptors can be divided into two groups by the relative affinity of the ligands to their receptors.

### Transforming Growth Factor

The TGF- $\beta$  superfamily is composed of more than 24 members ([68](#)). They are subdivided into families including TGF- $\beta$ , inhibin, decapentaplegic protein/vegetal hemisphere 1 (DPP/Vg1), and müllerian-inhibiting substance. Members of the TGF- $\beta$  and the DPP/Vg1 families have critical functions in the development of the skeleton, its growth and maintenance, and fracture repair. The bone morphogenic proteins (except for BMP-1) are members of the DPP/Vg1 family and are discussed in the next section.

All TGF- $\beta$  family members except TGF- $\beta$ 4 are synthesized as large precursor forms that are processed to active forms. The active form is either a heterodimer or homodimer. It is thought that the pro-region may either help in the folding of the proteins during synthesis or control activity. In the case of TGF- $\beta$ 1, the pro-region and a second glycoprotein can also bind to the active factor to form a latent complex. Members of the TGF- $\beta$  family are highly expressed in bone (TGF- $\beta$ 1, TGF- $\beta$ 2). Important in fracture repair, TGF- $\beta$ 1 and TGF- $\beta$ 2 are also released in large quantities during platelet activation.

Apart from the presence of the growth factor itself, the presence or absence of the latent complex controls the activity of TGF- $\beta$ 1. TGF- $\beta$  members can also be sequestered in the matrix. The active TGF- $\beta$ 1 complex can be released from the latent complex by extreme pH or by catalytic methods. This is particularly important in fracture repair and bone remodeling. The activation of latent TGF- $\beta$  is likely to be critical in the induction of fracture repair and osteoblast function.

The active TGF- $\beta$  molecules may also be bound and their activity controlled by a number of matrix molecules, including betaglycan and decorin ([100,197](#)). Alternatively, the active TGF- $\beta$  may bind to cellular receptors, of which there are at least nine. However, most of the actions are mediated through two receptors termed receptor 1 and 2. Receptors 1 and 2 are members of the serine/threonine kinase family ([100](#)). TGF- $\beta$  receptor type 3 is a membrane-bound proteoglycan termed betaglycan. Betaglycan is thought to act as a TGF- $\beta$  cell surface reservoir and is not involved with signal transduction itself. Betaglycan has the possibility of binding FGF through the heparin sulfate chains and may present TGF- $\beta$  in conjunction with FGF to the cell ([100](#)). TGF- $\beta$  also binds to the small proteoglycans: biglycan, decorin, and fibromodulin ([67](#)). The small proteoglycans bind TGF- $\beta$  through the leucine-rich repeats in their protein cores and are thought to sequester TGF- $\beta$  in the matrix. They also compete with betaglycan in binding TGF- $\beta$ . Decorin has the ability to negatively regulate the activity of TGF- $\beta$  ([13,160](#)).

### Bone Morphogenic Proteins

The bone morphogenic proteins and their orthopaedic relevance have recently been reviewed by Schmitt and colleagues ([160](#)). The bone morphogenic proteins (except BMP-1) represent a group of related growth factors that have critical roles in the cell proliferation and differentiation of a number of cell types including mesenchymal cells, chondrocytes, and osteoblasts ([28,82,83,186](#)). They have roles in embryo and fetal development, bone growth, and fracture repair. They also include a number of growth factors (BMP-2, BMP-7) (OP-1), which are being proposed for the treatment of fractures and the establishment of bone fusions.

BMPs exist as glycosylated dimers. Thirteen have been identified so far, but owing to sequence homology, only BMP-2 through 9 can be classed as members of the TGF- $\beta$  family. Particular BMPs produce ectopic cartilage or bone when implanted subcutaneously ([2,188](#)). Like the other growth factors discussed so far, the BMPs have a number of binding proteins both in the intracellular matrix and on the cell surface. A secreted glycoprotein termed noggin can bind and inactivate BMPs ([50](#)). Chordin is a similar protein that most likely has a similar function ([142](#)). It has been proposed that these proteins control BMP activity and may also serve as a mechanism for establishing gradients of BMPs across the embryo during development. Active BMPs bind to heterotetrameric serine/threonine kinase receptors. The nonactivated receptors exist as type 1 and 2 receptor proteins, the type 2 receptor autophosphorylates. Once the ligand binds, the two receptors are brought together and the receptor type 1 portion is phosphorylated. Only after the receptor type 1 is phosphorylated is a cellular response achieved. Intracellular activation is via the intracellular proteins termed SMADs (the humor equivalent of the MAD (mothers against decapentaplegic) protein), but other inhibitors can still come into play. Exposure of the cell to a number of other growth factors (including cer-1) can inhibit the activation of the cell by BMPs ([140,160](#)).

### Angiogenic Growth Factors

Angiogenic factors are growth factors that promote neovascularization. They are critical in fracture repair. The invasion of the metaphyseal vascular supply is crucial to endochondral ossification, and fracture repair does not occur without an adequate vascular supply. It is probably not by accident that a number of angiogenic factors such as TGF- $\beta$  and FGF-2 are sequestered in the bone matrix. Angiogenic factors act directly or indirectly on endothelial cells, promoting proliferation and migration of the cells into areas in which they are released. Angiogenic factors acting indirectly by recruiting macrophages monocytes, in turn, release their own direct-acting angiogenic factors ([165](#)).

Direct-acting angiogenic factors include platelet-derived endothelial growth factors (PDEGFs), TGF- $\beta$ , and FGF-2 to name but a few. Indirect acting angiogenic

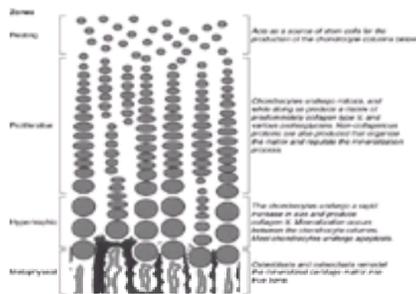
factors include TGF- $\beta$  and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ).

## MECHANISMS OF BONE GROWTH

Because bone is rigid, it cannot grow by internal expansion and bone growth is achieved by adding newly synthesized bone to existing bone by two mechanisms: *endochondral ossification* and *intramembranous ossification*. These mechanisms are named by the intermediate structures, that must be passed to form the bone. The production of any particular bone after initial differentiation may involve discrete, juxtaposed, or interspersed areas of each basic pattern. Endochondral-derived bones generally have membranous ossification by appositional bone growth from the periosteum. Similarly, membrane-derived bones may grow and elongate by an endochondral process ([126,130](#)).

### Endochondral Ossification

Endochondral ossification is the process by which bone forms via a cartilaginous intermediate. The physis best reflects this process. Physes are temporary cartilaginous tissue situated between the primary and secondary ossification centers of all long bones. From 9 to 10 weeks' gestational age to skeletal maturity at 15 to 17 years, they are responsible for the longitudinal growth of bone. The physis can be divided into at least three zones. The reserve zone is situated on the epiphyseal side and contains small, spherical cells randomly distributed throughout the zone. In the adjacent proliferative zone, chondrocytes undergo mitosis and are organized into columns running parallel to the axis of bone growth. Cells in the proliferative zone mature and eventually increase to 5 to 10 times their volume in the hypertrophic region. Matrix vesicles are also deposited within the longitudinal septa of the physis. Matrix vesicles are membrane-encapsulated structures that are thought to concentrate calcium and phosphate. Enzymes such as alkaline phosphatase convert organic phosphates to inorganic phosphate. The longitudinal septum around the terminal hypertrophic chondrocytes mineralizes, and this mineralized matrix forms the template for new bone deposition in the metaphysis ([Fig. 2-20](#)).



**FIGURE 2-20.** The figure shows the process of endochondral ossification within the physis. Although not as organized, endochondral ossification follows a similar pattern during fracture repair.

Associated with these changes in cellular arrangement and volume, the matrix in the physis also undergoes a continual modification in content. The two major macromolecules of cartilage matrix produced by the chondrocytes are the proteoglycans (predominantly aggrecan with lesser amounts of decorin, biglycan, and fibromodulin) and the collagens (types II, IX, X, and XI). The major change in physal proteoglycan structure occurs as chondrocytes organize into columns in the proliferative zone. Additional variation occurs in the hypertrophic region, where the glycosaminoglycan sulfation pattern demonstrates differences between the pericellular and extracellular spaces and the appearance of a unique collagen (type 10) is observed. The small proteoglycans—decorin, biglycan, and fibromodulin—are also differentially expressed across the physis, although detailed studies of these proteoglycans have not been done (see [Table 2-1](#)).

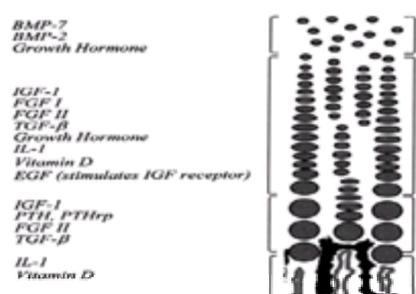
The cellular changes and associated matrix alterations are geared toward producing a microenvironment within the hypertrophic zone of the physis, which is conducive to matrix mineralization.

### Regulatory Mechanisms in the Physis

Hormones, for example, normal growth hormone, have a global effect on physal function throughout the body, but many growth factors act locally. The action of these growth factors have often been determined *in vitro* and *in vivo* and can often present conflicting results due to experimental design and models used. Most investigations have concentrated on the effect of single or, in some cases, two growth factors. However, it is likely that endochondral ossification is controlled by a large number of growth factors whose activity is controlled by a large number of binding proteins both within the matrix and on the chondrocyte surface. Cellular response is determined by parallel processing of the intracellular signals that are induced by a number of active growth factors binding to their specific receptors. Presented is an outline of the likely actions of a number of key growth factors on endochondral ossification. It is not complete, and the models will continue to change.

BMP-2 and 7 promote proliferation and matrix synthesis in undifferentiated chondrocytes ([40,84](#)). It is believed that once the chondrocytes start differentiating, the expression of noggin inhibits the continual outgrowth of the undifferentiated chondrocytes ([18](#)). The prechondrocytes may also respond to growth hormone ([117,133](#)). Once the chondrocyte has lost its resting phenotype, insulin like growth factor-1 (IGF-1) may act as a stimulator of proliferation and differentiation ([117,176](#)). EGF can augment IGF stimulation by increasing the expression of the IGF-1 receptor ([12](#)). Although the chondrocytes synthesize large quantities of matrix molecules, they also synthesize FGF-1, FGF-2, TGF- $\beta$ , and a number of the BMPs ([16,25,29](#)). These molecules can act in an autocrine manner, but many are sequestered into the newly forming cartilage matrix. FGF-2 in low doses is mitogenic for the chondrocytes ([94](#)); however, as occurs in achondroplasia, constant activation of FGF receptor (FGFR3) is inhibitory ([87,95](#)). FGF/heparin sulfate interaction is probable in the differentiation of the physal chondrocytes because the continuous exposure of FGF-2 inhibits chondrocyte differentiation *in vitro* and inhibitors of glycosaminoglycan sulfation (including heparin sulfate) restore the differentiation process. Additional sulfate permits glycosaminoglycan sulfation and returns the effect of FGF-2 ([30](#)).

Vitamin D metabolites and parathyroid hormone have roles in calcium mobilization within the body, but they also influence endochondral ossification. Parathyroid hormone and parathyroid hormone-related protein (PTHrP) can inhibit the maturation of chondrocytes. It is postulated that physal chondrocytes regulate the local production of PTHrP by secreting a protein (Indian Hedgehog). This protein stimulates the chondrocyte to produce PTHrP, which slows the maturation of proliferative chondrocytes to hypertrophic form ([85,187](#)). Expression of the mRNA for BMP-6 peaks before mineralization ([25](#)) ([Fig. 2-21](#)).



**FIGURE 2-21.** Growth factors that control or influence maturation and proliferation are shown. See the text for specific actions.

Although the chondrocytes of the physis will proliferate and form a cartilaginous matrix with only the epiphyseal vascular supply, the metaphyseal vessels are critical

for the mineralization process (183). Metaphyseal vascular invasion occurs at the hypertrophic–metaphyseal interface. The endothelial cells most likely invade as a consequence of angiogenic factors present in the matrix or secreted by the chondrocytes themselves. Both TGF- $\beta$  and FGFs are known to be angiogenic. It is interesting that an oversupply of FGF-2 infused into the physis induces vascular invasion from the metaphysis only; even if the FGF-2 is present at the epiphyseal side of the physis, the epiphyseal vessel will not invade (4). Although it is often stated that the metaphyseal vessels provide the necessary nutrients for the mineralization process, it is possible that they provide additional growth factors that initiate the mineralization process.

The vascular supply also brings osteoblasts, osteoclasts, and other cell types. The osteoclasts degrade the mineralized cartilage matrix while osteoblasts lay down new bone that is also rich in growth factors such as TGF- $\beta$ , FGF-2 and the BMPs.

### Membranous Ossification

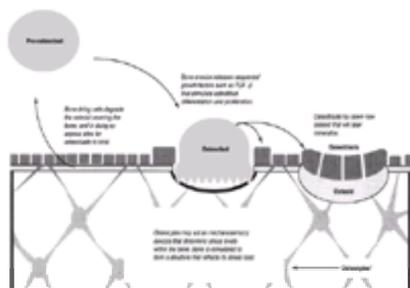
All axial and appendicular skeletal elements are involved in secondary membranous ossification. The diaphyseal cortex of developing tubular bone is progressively formed (modeled) by the periosteum and modified (remodeled) by the re-formation of osteons. This peripheral periosteal process of membrane-derived ossification is extensive and rapid in fracture healing in infants and young children. The replacement process also may be seen when portions of the developing metaphysis or diaphysis are removed for use as bone grafts.

Intramembranous ossification occurs when osteoprogenitor cells are formed from the overlying tissue. The osteoprogenitor cells continue to differentiate into osteoblasts, which produce a matrix that undergoes mineralization.

### Remodeling of Bones

The first bone to be laid down either from the physis or in the fracture callus is woven bone, which is remodeled to lamellar bone. Although cancellous bone can be remodeled and obtain its nutrients from the surface, cortical bone is remodeled into a complex structure of osteons that together form the cortical bone. Osteons are tubular structures that interconnect. They consist of layers of ordered lamellar bone around a central canal. The central canal contains blood vessels, lymphatics, and in some cases, nerves (20).

Bone is constantly remodeled by osteoclasts and osteoblasts. The bone is encapsulated by bone-lining cells that have the potential to become activated osteoblasts. The bone-lining cells, like osteocytes, have slender cellular processes that make contact with the osteocytes within the mineralized bone. Osteocytes are thought to arise from osteoblasts that have become entrapped during bone formation. It has been proposed that the bone-lining cells need to erode the osteoid that covers the underlying bone for osteoclasts to bind (107,108). Osteoclasts are bone-degrading cells that are produced from the hematopoietic pathway. On activation, they bind to the surface of the bone and secrete enzymes into the space beneath. The space is acidic and contains many proteolytic and bone degrading enzymes (98). The acidic pH and proteases are thought to release and activate the sequestered TGF- $\beta$ , resulting in the differentiation and activation of the bone-lining cells to osteoblasts and onto osteocytes (41,42,101). The osteoblasts then lay down new osteoid, and subsequent mineralization results in bone. Although it is usually accepted that the osteoblast activity and osteoclast activity are linked, discussion still exists about the signals that determine the equilibrium that is required to keep the bone density at functional levels. The osteocytes may be the mechanosensory system. Osteocytes also possess cellular processes that connect osteocytes to one another and to the bone-lining cells above (32) (Fig. 2-22). It is possible that the osteocytes are responsible for sensing bone stress; if undue stress is detected, they favor bone deposition, whereas if a lack of stress is detected, they favor bone resorption.



**FIGURE 2-22.** Osteoclasts and osteoblasts constantly remodel bone. Osteocytes exist within the bone. Bone-lining cells need to erode the osteoid that covers the underlying bone for osteoclasts to bind. Osteoclasts bind to the surface of the bone and secrete enzymes into the space beneath. The acidic pH and proteases are thought to release and activate the sequestered TGF- $\beta$  that results in the differentiation and activation of the pre-osteoblasts to osteoblasts. The osteoblasts then lay down new osteoid, and subsequent mineralization results in bone.

## FRACTURE REPAIR

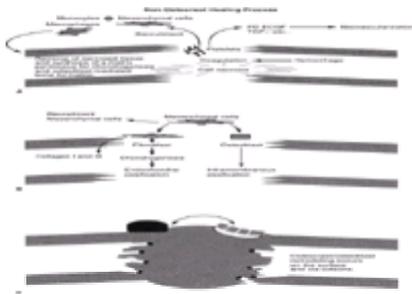
Injuries to the developing skeleton may involve osseous, fibrous, and cartilaginous tissues. Healing of these tissues differs, depending on both the type of tissue and the temporal maturation.

### Osseous Healing

The progressive changes of the normal process of osseous fracture healing, whether in the diaphysis, metaphysis, or epiphyseal ossification center, may be grouped conveniently into a series of phases that occur in a reasonably chronologic sequence (104,152,154). Several factors that influence bone healing can be identified from clinical observation as well as experimental work, and these factors must be taken into account when treating childhood fractures on a rational basis. Many experiments have been performed on animals, although because of differences in macroscopic and microscopic bone structure and skeletal homeostatic mechanisms, they may respond differently than skeletally immature humans (137,148,168,171,175,185,196). Furthermore, most experiments have been performed on skeletally mature animals, and such data are not always relevant to fracture healing in the developing skeleton. In addition, certain areas of the developing skeleton, particularly the physis and epiphyseal hyaline cartilage, probably do not heal by classic callus formation. In fact, when this type of osseous (callus) repair occurs in these cartilaginous regions, significant growth deformities may result owing to formation of an osseous bridge between the secondary ossification center and the metaphysis (see Chapter 5).

As in adults, there are three basic mechanisms of fracture repair: primary osteonal, secondary osteonal and nonosteonal. Primary osteonal fracture healing occurs when cortical bone is laid down without any intermediate, and therefore hardly any callus forms; it is only possible if cortical bone is repositioned and fixed in close proximity. Secondary osteonal union occurs if cortical bone is laid down between two segments of fractured cortical bone before callus formation. Nonosteonal union occurs through endosteal and periosteal callus formation (158).

Fracture repair in the immature skeleton can be divided into three closely integrated, but sequential, phases: the inflammatory phase, the reparative phase, and the remodeling phase (Fig. 2-23). In children, the remodeling phase is temporally much more extensive and physiologically more active (depending on the child's age) than the comparable phase in adults. The remodeling phase is further modified by the effects of the physis responding to changing joint reaction forces and biologic stresses to alter angular growth dynamics. This occurs even when the fracture is mid-diaphyseal.



**FIGURE 2-23.** The figure demonstrates the three phases of fracture repair **(A)** inflammatory phase, **(B)** reparative phase, and **(C)** remodeling phase. The inflammatory cells remove the debris from the fracture site and, together with the fibroblastic cells, develop the site into a matrix that will support the cells that enable new bone to be formed. The mesenchymal cells are recruited by the release of growth factors in the fracture site. The mesenchymal cells may differentiate into osteoblasts that produce bone in a membranous fashion. Alternately the mesenchymal cell may become chondrogenic and produce bone by the endochondral pathway. Remodeling begins with resorption of mechanically unnecessary, inefficient portions of the callus and the subsequent orientation of trabecular bone along the lines of stress.

## Cellular Response to Trauma

### Inflammatory Phase

Immediately after a fracture through any of the osseous portions of the developing skeleton (diaphysis, metaphysis, or epiphyseal ossification center), several cellular processes begin.

#### Hematoma Formation

Bleeding of the damaged periosteum, contiguous bone, and soft tissues starts the process of repair through the release of growth factors, cytokines, and prostaglandins. If the fracture is localized to the maturing diaphysis, there is bleeding from the haversian systems, as well as from the multiple small blood vessels of the microcirculatory systems of the endosteal and periosteal surfaces and contiguous soft tissue anastomoses ( 56). In the region of the metaphysis, this bleeding may be extensive because of the anastomotic ramifications of the peripheral and central metaphyseal vascular systems. A hematoma accumulates within the medullary canal at the fracture site, beneath the elevated periosteum, and extraperiosteally whenever the periosteum is disrupted during the fracture. In contrast to adults, the periosteum strips away easily from the underlying bone in children, allowing the fracture hematoma to dissect along the diaphysis and metaphysis; this is evident in the subsequent amount of new bone formation along the shaft.

However, the dense attachments of the periosteum into the zone of Ranvier limit subperiosteal hematoma formation to the metaphysis and diaphysis. Because the perichondrium is densely attached, this type of hemorrhagic response is uncharacteristic of the epiphyseal ossification center, thus limiting its contributions to callus formation and any intrinsic stabilization effect. Further, because of the partially or completely intracapsular nature of some epiphyses, propagation of a fracture into the joint allows decompression of some of the bleeding into the joint, again limiting the potential volume for eventual callus formation.

Coagulation and platelet activation stop the blood loss but also produce both inflammatory mediators and angiogenic factors. Endothelial cells respond and increase the vascular permeability, and allow the passage of leukocytes, monocytes, and macrophages into the fracture site. Neovascularization is also initiated. Angiogenic factors like platelet-derived growth factor (PDGF) and TGF- $\beta$ , also promote osteoblast recruitment and activation.

#### Local Necrosis

The blood supply is temporarily disrupted for a few millimeters on either side of the fracture, creating juxtaposed, avascular trabecular and cortical bone ( 55) and producing local necrosis. It is likely that the necrosis also results in the release of sequestered growth factors (e.g., IGF-1, TGF- $\beta$ , FGF-1, and FGF-2) from the bone. These growth factors may help in promoting differentiation of the surrounding mesenchymal cells into bone-forming cells.

The inflammatory cells remove the debris from the fracture site and, with the fibroblastic cells, develop the site into a matrix that will support the cells that enable new bone to be formed. This initial matrix often contains collagens type I, III, and V.

#### Organization of Hematoma

The initial cellular repair process involves organization of the fracture hematoma ( 39,55,62,69). Fibrovascular tissue replaces the clot with a matrix rich in collagens I, III, and V. This matrix allows chondrogenesis or intramembranous bone formation. Such mechanisms eventually lead to mineralization and the formation of the woven bone of the provisional (primary) callus. Initial invasion and cell division are around the damaged bone ends but proceed centrifugally away from the fracture site, thus placing the most mature repair process closest to the fracture site. However, bone formation occurs only in the presence of an intact, functional microvascular supply. If the vascular supply is deficient, then this modulation of cartilaginous to osseous tissue cannot readily occur.

### Reparative Phase

#### Cellular Organisation

The fracture hematoma is the area in which the early stages of healing occur ( 145). Osteogenic cells proliferate from the periosteum to form an external callus and, to a lesser extent, from the endosteum to form an internal callus. However, when the periosteum is severely disrupted, healing cells must differentiate from the ingrowth of undifferentiated mesenchymal cells throughout the hematoma. By 10 to 14 days in a child, the fracture callus consists of a thick, enveloping mass of peripheral osteogenic tissue that is beginning to be evident radiographically. This new bone is primarily woven (fiber) bone ( 101,114,150,151).

The next step in osseous fracture healing is cellular organization ( 33). During this stage, the circumferential tissues serve primarily as a fibrous scaffold over which cells migrate and orient to induce a stable repair. This pluripotential mesenchyme is theoretically capable of modulation into cartilage, bone, or fibrous tissue (54,57,135). The mesenchymal cells are recruited by the release of growth factors in the fracture site. Members of the BMP family, and possibly their inhibitors, are likely to be involved in the recruitment and differentiation of the mesenchymal cells. The mesenchymal cells may differentiate into osteoblasts that produce bone in a membranous fashion or may become chondrogenic and produce bone by the endochondral pathway. Both mechanisms usually are present in a fracture callus, and the degree to which each is present depends on the type of bone, age, degree of fixation, level of bone loss, and trauma. In children, because of the osteoblastic activity, the periosteum contributes significantly to new bone formation by accentuating the normal process of membranous ossification to supplement the cellular organization within the hematoma, which is going through a cartilaginous phase ( 58,59). The region around the fracture site thus repeats the process of endochondral ossification, in close juxtaposition to membranous ossification from the elevated periosteum. Similar processes occur within the medullary cavity. An integral part of the reparative process at this stage is microvascular invasion, which occurs very readily in children because of the state of vascularity within and without the bone and surrounding soft tissues (26). Vessels come from the periosteal region as well as from the nutrient artery and endosteal vessels.

Until this bone goes through the final stages of maturation, it is still biologically plastic and, if not protected, may gradually deform, especially in an active young child after early release from an immobilization device. Even in a cast, this plasticity may allow deformation from isometric muscle activity.

Clinical union is attained when the fracture site no longer moves and is not painful to attempts at manipulation, although it is by no means restored to its original strength at this time. With time, the primary callus is gradually replaced. This is enhanced in the child because appositional growth and increasing diameter envelop the original fracture region, the cartilage and woven bone have been replaced by mature, lamellar bone, and the fracture has consolidated and essentially returned to

most of its normal biologic standards and response to stress.

### **Remodeling Phase**

The last phase (remodeling) begins with resorption of mechanically unnecessary, inefficient portions of the callus and the subsequent orientation of trabecular bone along the lines of stress. The remodeling phase is the longest of the three phases and in children may continue until (and beyond) skeletal maturation in response to constantly changing stress patterns imposed by continued skeletal growth and development. Initially, new bone is laid down by both the fracture callus and the more extensive but confluent subperiosteal tissue. This bone is randomly oriented and cannot withstand all biologic stresses imposed on it. However, as the bone grows diametrically in the diaphyseal or metaphyseal regions, this new bone is gradually and increasingly incorporated into the preexisting cortical bone, aligned in accord with predominant stress patterns, and replaced by physiologic remodeling processes. The degree of remodeling and progressive replacement of fracture callus is greater in younger children, who have an immense capacity for growth and change.

The critical step between the reparative and remodeling phases is the establishment of an intact bony bridge between the fragments. Because this involves the joining of separated segments of hard tissue, the whole system must become immobile. Once the bridge has been established—provided that adequate, continued mechanical protection is given—subsequent biologic failure is unlikely. If the two or more fracture fragments remain connected by the periosteum or related material, as is likely in a child, it is easy to see how reparative activity could be conducted from one side to the other relatively easily and rapidly.

The intact bone must then readapt to functional demands. This is much easier in children, whose skeletons are actively and continually remodeling in response to stress, than in adults, who have more static skeletons. The processes of replacement and repair are continuous and concomitant in the normally developing skeleton. The mechanisms involved in fracture healing essentially are no different than most of the active maturational processes. These processes are much more active in children and are more active in the metaphysis than in the diaphysis.

The fracture remodeling process differs in cortical or cancellous bone. Both involve a process of simultaneous bone removal and replacement by the osteoclasts and osteoblasts through the accompanying blood vessels. In cancellous bone of the metaphysis or the endosteal surface of the diaphysis, the cells are never very far away from blood vessels, and the whole process of apposition and replacement may occur on the surface of the trabeculae. However, in compact bone, the more deeply placed cells require the presence of an adequately functioning perfusion system that must be replaced. This is a much longer sequence of events and is not a major method of bone repair in children, except when the fracture involves densely cortical regions such as the femoral or tibial shafts. McKibbin ( 104) presented an extensive discussion of this process, which is sometimes referred to as *primary bone union* because no intermediate cells are involved.

### **Physeal Healing Patterns**

The physis has a limited ability to repair; it primarily heals by increased endochondral bone and cartilage formation, and gradual reinvasion by the disrupted metaphyseal vessels to replace the temporarily widened physis eventually. Very little experimental work, mostly in rats, has been directed at assessing the posttraumatic cellular response patterns of the physis ( 17).

Depending on the level of cellular injury within the physis, *three types of chondro-osseous healing* may occur. First, when the fracture occurs through the cell columns, healing occurs primarily by continued, relatively rapid increases in the number of cells within the columns, causing moderate widening of the physis. Because there are some small epiphyseal vessels in this region, some damaged tissue may be resorbed early in the healing process. These vessels also exhibit a hyperemic response, increasing cellular proliferation rates, especially in the peripheral zone of Ranvier. The metaphyseal response parallels this, in that an increased rate of bone replacement of the hypertrophic chondrocyte also occurs. Once the level of fracture fibrosis and debris within the physis is encountered, the vessels rapidly invade to reach the rest of the maturing cell columns. These cellular response patterns lead to restoration of normal anatomy within 3 to 4 weeks ( 153).

Second, when the fracture occurs through the transition of hypertrophic cells to primary spongiosa (the most commonly involved cellular level), there may be marked separation, with the gap filled by hemorrhagic and fibroblastic tissue. This region may progressively form disorganized cartilaginous tissue, not unlike the initial, disorganized cartilaginous callus around a diaphyseal fracture. Meanwhile, cellular proliferation, cell column formation, hypertrophy, and calcification continue on the epiphyseal side of the disorganized callus, leading to widening of the physis. A vascular invasion of the remnants of hypertrophic, calcified cartilage also rapidly occurs on the metaphyseal side of the fracture. However, once metaphyseal vessel invasion reaches the disorganized cartilaginous callus, vascular-mediated bone replacement is temporarily slowed, because there is no pattern of cell columns to invade in an organized fashion. As the callus cartilage matures and calcifies, the metaphyseal vessels begin to invade and replace the cartilage with bone irregularly ( 21). This callus may be variably thick, depending on the degree of longitudinal and lateral displacement and periosteal continuity with the physeal periphery. The callus is replaced at different rates, and the invading metaphyseal vessels reach the normal cell columns, which have been maturing in a normal sequence but without osseous replacement. This widened physis is rapidly invaded by the vessels and replaced by primary spongiosa, and normal physeal width is progressively restored.

The callus in the subperiosteal region contributes to early stability. This region heals by vascular invasion of the callus to form trabecular bone between the original metaphyseal cortex and the subperiosteal membranous bone forming continuously external to the metaphyseal cartilaginous callus. These three microscopic bone regions progressively merge and remodel, making the region strong biomechanically. With further growth and remodeling, this coalescent bone is completely replaced. These initial cellular replacement processes in both metaphyseal and physeal regions probably take 3 to 6 weeks. However, remodeling may continue for months to years, and it enhances the capacity for spontaneous correction of many residual deformities.

Third, when the injury extends across all cell layers of the physis, the repair processes differ slightly. Fibrous tissue initially fills the gap between separated physeal components, whereas typical callus formation occurs in the contiguous metaphyseal spongiosa or epiphyseal ossification center. If large surfaces of nonossified epiphyseal cartilage also are involved, fibrous tissue initially forms in the intervening region. The reparative response shows irregular healing of the epiphyseal and physeal cartilage, with loss of normal cellular architecture. Within the central physeal regions, diametric expansion of cell columns is minimal, so closure of a large defect by physeal cartilage is unlikely. The gap will remain fibrous, but with the potential to ossify. Toward the physeal periphery, diametric expansion is more likely, but still may not lead to closure of large cartilage gaps by progressive replacement of fibrous tissue. This replacement process essentially requires the germinal and hypertrophic cell regions to diametrically expand by cell division, maturation, and matrix expansion. The intervening fibrous tissue may disappear through growth, but only if the gap is narrow. Because blood supply is minimal in this region, the fibrous tissue similarly is not well vascularized, and significant cell modulation, especially to osteoblastic tissue, is less likely in the short term. However, the larger the gap filled with fibrous tissue and the longer the time from fracture to skeletal maturity, the greater the likelihood of developing sufficient vascularity to commence an osteoblastic response and to form an osseous bridge. Further, in young children with minimal epiphyseal ossification, the blood supply to the physeal germinal region is not as well defined, whereas once the ossification center expands and forms a subchondral plate over the germinal region, microvasculature probably increases and the chances for vascularization and ossification of the fibrous region increase. This explains the delayed appearance of the osseous bridge.

If accurate anatomic reduction is performed, a thin gap should be present that should fill in with minimal fibrous tissue, allowing progressive replacement of the tissue by diametric expansion of the physis and contiguous epiphysis. However, if the fragment has been partially or completely devascularized by either the initial trauma or subsequent dissection to effect an open reduction, cellular growth and diametric and longitudinal expansion may not occur. This increases the chances of cellular disorganization, fibrosis, and eventual osteoblastic response. Failure to correct anatomic displacement, especially in Salter-Harris type 4 growth mechanism injuries, increases the possibility of apposition of the epiphyseal ossification center and metaphyseal one, and thereby enhances the risk of forming an osseous bridge between the two regions.

### **Remodeling of Bones in Children After Injury**

In a growing child, the normal process of bone remodeling in the diaphysis and metaphysis (particularly the latter) may realign initially malunited fragments, making absolutely accurate anatomic reduction less important than in a comparable injury in an adult. However, although some residual angular deformities undergo spontaneous correction, accurate anatomic reduction should be the goal whenever possible ( 51,122,129). Bone and cartilage generally remodel in response to normal stresses of body weight, muscle action, and joint reaction forces, as well as intrinsic control mechanisms such as the periosteum. The potential for spontaneous, complete correction is greater if the child is younger, the fracture site is closer to the physis, and there is relative alignment of the angulation in the normal plane of motion of the joint. This is particularly evident in fractures involving hinge joints such as the knee, ankle, elbow, or wrist, in which corrections are relatively rapid if the angulation is in the normal plane of motion. However, spontaneous correction of angular deformities is unlikely in other directions (relative to normal joint motion), such as a cubitus varus deformity following a supracondylar fracture of the humerus. Similarly, *rotational deformities usually do not correct spontaneously*.

## Growth Stimulation

Fractures may stimulate longitudinal growth by increasing the blood supply to the metaphysis, physis, and epiphysis, and at least on an experimental basis, by disrupting the periosteum and its physiologic restraint on the rates of longitudinal growth of the physes ( 34). Such increased growth may make the bone longer than it would have been without an injury (9,36,184). Eccentric overgrowth may also occur; this is particularly evident in tibia valgum following an incomplete fracture of the proximal tibial metaphysis.

## THE FUTURE OF FRACTURE REPAIR

Bone grafts contain bone growth factors that normally induce bone formation and have the appropriate osteoconductive matrix. Autogenic grafts also contain osteogenic cells. Bone grafts are effective, but there are difficulties in obtaining safe and reliable tissue. Although the mechanisms of fracture repair are not fully understood, the level of understanding has enabled key molecules to be targeted as therapeutic in controlling and promoting fracture repair. Filler compounds have been developed that either stimulate mesenchymal cells, leading to new bone formation (osteoinductive) or enable the bone-forming cells to infiltrate and incorporate into bone (osteoconductive).

Specific growth factors have been targeted for their ability to promote bone formation. Two growth factors (BMP-2 and Osteogenic Protein-1) (BMP-7) show great promise for their ability to promote fracture repair (83,84,90,96,173,192). A number of others, such as TGF- $\beta$ , IGF, PDGFs, and FGF-2, also may prove to be useful.

TGF- $\beta$  plays a major role in fracture repair by promoting proliferation and differentiation of the mesenchymal cells. Exogenous TGF- $\beta$  administration can initiate the repair process and callus formation in uninjured bone ( 75). The addition of TGF- $\beta$  to fractures promotes wound repair and results in a larger, stronger callus ( 75). It also may be of use in promoting repair in nonhealing bone defects. PDGF also increases callus size but does not improve the fracture mechanically ( 115). Growth hormone and IGF-1 have also been tested to determine their effects on fracture repair. Although growth hormone produces inconsistent results, the administration of IGF-1 increases intramembranous bone formation (3,179). The FGFs also increase the callus size and mineral content ( 72,77,189). It is possible that the effect of FGFs and of a number of the other growth factors is a result of the angiogenic properties of such growth factors. There are many reviews on the use of growth factors for fracture repair (37,38,91,178), and more research is required to establish the most useful factors and effective delivery devices ( 71). However, there is little doubt that in the near future, orthopaedic surgeons will be using growth factor-containing compounds to induce new bone formation and to improve fracture repair.

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## CHAPTER REFERENCES

1. Acheson RM. Effects of starvation, septicaemia and chronic illness on the growth cartilage plate and metaphysis of the immature rat. *J Anat* 1959;93:123–130.
2. Aono A, Hazama M, Notoya K, et al. Potent ectopic bone-inducing activity of bone morphogenetic protein-4/7 heterodimer. *Biochem Biophys Res Commun* 1995;210:670–677.
3. Bak B. Fracture healing and growth hormone. A biochemical study in the rat. *Dan Med Bull* 1993;40:519–536.
4. Baron J, Klein KO, Yanovski JA, et al. Induction of growth plate cartilage ossification by basic fibroblast growth factor. *Endocrinology*, 1994;135:2790–2793.
5. Bateman J, Lamande S, Ramshaw J. Collagen superfamily. Eds. In: Comper WD, ed. *Extracellular matrix*. Amsterdam: Harwood Academic Publishers, 1996;22–67.
6. Beckman F, Sullivan J. Some observations of fractures of long bones in the child. *Am J Surg* 1941;51:722–738.
7. Bianco P, Fisher LW, Young MF, et al. Expression and localization of the two small proteoglycans biglycan and decorin in developing human skeletal and non-skeletal tissues. *J Histochem Cytochem* 1990;38:1549–1563.
8. Birk DE, Fitch JM, Babiarz JP, et al. Collagen fibrillogenesis in vitro: interaction of types I and V collagen regulates fibril diameter. *J Cell Sci* 1990;95:649–657.
9. Bisgard JD. Longitudinal overgrowth of long bones with special references to fractures. *Surg Gynecol Obstet* 1936;62:823–835.
10. Böhlen P, Baird A, Esch F, et al. Isolation and partial molecular characterization of pituitary fibroblast growth factor. *Proc Natl Acad Sci U S A* 1984;81:5364–5368.
11. Bolander ME, Young MF, Fisher LW, et al. Osteonectin cDNA sequence reveals potential binding regions for calcium and hydroxyapatite and shows homologies with both a basement membrane protein (SPARC) and a serine proteinase inhibitor (ovomucoid). *Proc Natl Acad Sci U S A* 1998;85:2919–2923.
12. Bonassar LJ, Trippel SB. Interaction of epidermal growth factor and insulin-like growth factor-I in the regulation of growth plate chondrocytes. *Exp Cell Res* 1997;234:1–6.
13. Border WA, Noble NA, Yamamoto T, et al. Natural inhibitor of transforming growth factor-beta protects against scarring in experimental kidney disease. *Nature* 1992;360:361–364.
14. Borgi R, Butel J, Finidori G. La regenerescence diaphysaire d'un os long chez l'enfant. *Rev Chir Orthop* 1979;65:413–414.
15. Bovi PD, Curatola AM, Kern FG, et al. An oncogene isolated by transfection of Kaposi's sarcoma DNA encodes a growth factor that is a member of the FGF family. *Cell* 1987;50:729–737.
16. Boyan BD, Schwartz Z, Park Snyder S, et al. Latent transforming growth factor-beta is produced by chondrocytes and activated by extracellular matrix vesicles upon exposure to 1,25-(OH)<sub>2</sub>D<sub>3</sub>. *J Biol Chem* 1994;269:28374–28381.
17. Brashear HR Jr. Epiphyseal fractures—a microscopic study of the healing process in rats. *J Bone Joint Surg Am* 1959;41A:1055–1064.
18. Brunet LJ, McMahon JA, McMahon AP, Harland RM. Noggin, cartilage morphogenesis, and joint formation in the mammalian skeleton. *Science* 280:1455–1457.
19. Bucholz RW, Ogden JA. Patterns of ischemic necrosis of the proximal femur in nonoperatively treated congenital hip disease. In: Nelson CL ed. *The hip: proceedings of the hip society*, vol 6. St. Louis: CV Mosby, 1978:43–63.
20. Buckwalter JA, Glimcher MJ, Cooper RR, Recker R. Bone Biology. I: Structure, blood supply, cells, matrix, and mineralization. *Instr Course Lect* 1996;45:371–386.
21. Burger M, Sherman BS, Sobel AE. Observations on the influence of chondroitin sulphate on the rate of bone repair. *J Bone Joint Surg* 1962;44B:675–687.
22. Burgess WH, Mehlman T, Marshak DR, et al. Structural evidence that endothelial cell growth factor is the precursor of both endothelial cell growth factor and acidic fibroblast growth factor. *Proc Natl Acad Sci USA* 1986;83:7216–7220.
23. Byers S, van Rooden JC, Foster BK. Structural changes in the large proteoglycan, aggrecan, in different zones of the ovine growth plate. *Calcif Tissue Int* 1997;60:71–78.
24. Calandruccio RA, Gilmer WS. Proliferation, regeneration and repair of articular cartilage of immature animals. *J Bone Joint Surg* 1962;44A:431–455.
25. Carey DE, Liu X. Expression of bone morphogenetic protein-6 messenger RNA in bovine growth plate chondrocytes of different size. *J Bone Miner Res* 1995;10:401–405.
26. Chalmers J, Gray DH, Rush J. Observations on the induction of bone in soft tissues. *J Bone Joint Surg* 1975;57B:36–45.
27. Chan D, Taylor TKF, Cole WG. Characterization of an arginine 789 to cysteine substitution in alpha-1(II) collagen chains of a patient with spondyloepiphyseal dysplasia. *J Biol Chem* 1993;268:15238–15245.
28. Cheifetz S, Li IW, McCulloch CA, et al. Influence of osteogenic protein-1 (OP-1—P-7) and transforming growth factor-beta 1 on bone formation in vitro. *Connect Tissue Res* 1996;35:71–78.
29. Chintala SK, Miller RR, McDevitt CA. Basic fibroblast growth factor binds to heparan sulfate in the extracellular matrix of rat growth plate chondrocytes. *Arch Biochem Biophys* 1994;310:180–186.
30. Chintala SK, Miller RR, McDevitt CA. Role of heparan sulfate in the terminal differentiation of growth plate chondrocytes. *Arch Biochem Biophys* 1995;316:227–234.
31. Cohen S, Ushiro H, Stoscheck C, Chinkers M. A native 170,000 epidermal growth factor receptor-kinase complex from shed plasma membrane vesicles. *J Biol Chem* 1982;257:1523–1531.
32. Cowin SC, Moss Salentijn L, Moss ML. Candidates for the mechanosensory system in bone. *J Biomech Eng* 1991;113:191–197.
33. Crelin ES, White AA III, Panjabi M.M, Southwick WO. Microscopic Changes in Fractured Rabbit Tibias. *Conn Med* 1978;42:561–569.
34. Crilly RG. Longitudinal overgrowth of chicken radius. *J Anat* 1972;112:11–18.
35. Ducy P, Desbois C, Boyce B, et al. Increased bone formation in osteocalcin-deficient mice. *Nature* 1996;382(6590):448–452.
36. Edvardson P, Syversen SM. Overgrowth of the femur after fractures of the shaft in childhood. *J Bone Joint Surg* 1976;58B:339–346.
37. Einhorn TA, Trippel SB. Growth factor treatment of fractures. *Instr Course Lect* 1997;46:483–486.
38. Einhorn TA. Enhancement of fracture healing. *Instr Course Lect* 1996;45:401–416.
39. Ekeland A, Engesaeter LB, Langeland N. Influence of age on mechanical properties of healing fractures and intact bones in rats. *Acta Orthop Scand* 1982;53:5277.
40. Erickson DM, Harris SE, Dean DD, et al. Recombinant bone morphogenetic protein (BMP)-2 regulates costochondral growth plate chondrocytes and induces expression of BMP-2 and BMP-4 in a cell maturation-dependent manner. *J Orthop Res* 1997;15:371–380.
41. Erlebacher A, Derynck R. Increased expression of TGF-beta 2 in osteoblasts results in an osteoporosis-like phenotype. *J Cell Biol* 1996;132:195–210.
42. Erlebacher A, Filvaroff EH, Ye JQ, Derynck R. Osteoblastic responses to TGF-beta during bone remodeling. *Mol Biol Cell* 1998;9:1903–1918.
43. Esch F, Veno N, Baird A, et al. Primary structure of bovine brain acidic fibroblast growth factor (FGF). *Biochem Biophys Res Comm* 1985;133:554–562.

44. Evans RM. The steroid and thyroid hormone receptor superfamily. *Science* 1988;240:889–895.
45. Eyre DR, Upton MP, Shapiro FD, et al. Nonexpression of cartilage type II collagen in a case of Langer-Saldino achondrogenesis. *Am J Hum Genet* 1986;39:52–67.
46. Fichard A, Kleman, JP, Ruggiero F. Another look at collagen V and XI molecules. *Matrix Biol* 1994;14:515–531.
47. Finch PW, Rubin JS, Turu M, et al. Human KGF is FGF-related with properties of a paracrine effector of epithelial cell growth. *Science* 1989;245:752–755.
48. Finkbeiner S. Calcium waves in astrocytes-filling in the gaps. *Neuron* 1992;8:1101–1108
49. Fosang A, Hardingham T. Matrix proteoglycans. In: Comper WD, ed. *Extracellular matrix*. Amsterdam: Harwood Academic Publishers, 1996;200–229.
50. Gazzerri E, Gangji V, Canalis E. Bone morphogenetic proteins induce the expression of noggin, which limits their activity in cultured rat osteoblasts. *J Clin Invest* 1998;102:2106–2114.
51. Giberson RG, Ivins JC. Fractures of the distal part of the forearm in children: Correction of deformity by growth. *Minn Med* 1952;35:744.
52. Gibson G, Lin DL, Francki K, et al. Type X collagen is colocalized with a proteoglycan epitope to form distinct morphological structures in bovine growth cartilage. *Bone* 1996;19:307–315.
53. Gimenez-Gallego G, Rodkey J, Bennett C, et al. Brain-derived acidic fibroblast growth factor, complete amino acid sequence and homologies. *Science* 1985;230:1385–1388.
54. Girgis FG, Pritchard JJ. Experimental production of cartilage during the repair of fractures of the skull vault in rats. *J Bone Joint Surg* 1958;40B:274–281.
55. Goldhaber P. Osteogenic induction across millipore filters in vivo. *Science* 1966;133:2065–2067.
56. Gotham L. Vascular reactions in experimental fractures: microangiographic and radioisotope studies. *Acta Chir Scand* 1961;284(Suppl):1–34.
57. Haines RW. Cartilage canals. *J Anat* 1933;68:45–64.
58. Ham AW. A histological study of the early phase of bone repair. *J Bone Joint Surg* 1930;12:827–844.
59. Ham AW. *Histology*, 6th ed. Philadelphia: JB Lippincott, 1969.
60. Hanlon CR, Estes WL. Fractures in childhood-A statistical analysis. *Am J Surg* 1954;87:312–323.
61. Harper JW, Strydom DJ, Lobb RR. Human class 1 heparin-binding growth factor: structure and homology to bovine acidic brain fibroblast growth factor. *Biochemistry* 1986;25:4097–4103.
62. Harris HA. The growth of long bones in childhood with special reference to certain bony striations of the metaphysis and to the role of vitamins. *Arch Intern Med* 1926;38:785–806.
63. Hauschka PV, Lian JB, Cole DE, Gundberg CM. Osteocalcin and matrix Gla protein: vitamin K-dependent proteins in bone. *Physiol Rev* 1989 69:990–1047.
64. Hauschka PV, Mavrakos AE, Iafrafi MD, et al. Growth factors in bone matrix. Isolation of multiple types by affinity chromatography on heparin-Sepharose. *J Biol Chem* 1986;261:12665–12674.
65. Hearn MTW. Structure and function of the heparin-binding (fibroblast) growth factor family. *Baillères Clin Endocrinol Metab* 1991;5:571–593.
66. Hedbom E, Heinegard D. Binding of fibromodulin and decorin to separate sites on fibrillar collagens. *J Biol Chem* 1993;268:27307–27312.
67. Hildebrand A, Romaris M, Rasmussen LM, et al. Interaction of the small interstitial proteoglycans biglycan, decorin and fibromodulin with transforming growth factor beta. *Biochem J* 1994;302:527–534.
68. Hogan BL, Blessing M, Winnier GE, et al. Growth factors in development: the role of TGF-beta related polypeptide signalling molecules in embryogenesis. *Dev Suppl* 1994;53–60.
69. Hueter C. *Die formentenwicklung am skelet des menschlichen thorax*. Leipzig: FCW Vogt, 1865.
70. Hultenby K, Reinholdt FP, Heinegard D, et al. Osteopontin: a ligand for the alpha v beta 3 integrin of the osteoclast clear zone in osteopetrotic (ia/ia) rats. *Ann N Y Acad Sci* 1995;760:315–318.
71. Illi OE, Feldmann CP. Stimulation of fracture healing by local application of humoral factors integrated in biodegradable implants. *Eur J Pediatr Surg* 1998;8:251–255.
72. Inui K, Maeda M, Sano A, et al. Local application of basic fibroblast growth factor minipellet induces the healing of segmental bony defects in rabbits. *Calcif Tissue Int* 1998;63:490–495.
73. Jacenko O, Ito S, Olsen BR. Skeletal and hematopoietic defects in mice transgenic for collagen X. *Ann N Y Acad Sci* 1996;785:278–280.
74. Jacenko O, LuValle PA, Olsen BR. Spondylometaphyseal dysplasia in mice carrying a dominant negative mutation in a matrix protein specific for cartilage-to-bone transition. *Nature* 1993;365:56–61.
75. Joyce ME, Jingushi S, Scully SP, Bollander ME. Role of growth factors in fracture healing. *Prog Clin Biol Res* 1991;365:391–416.
76. Kaplan FS, Hayes WC, Keaveny TM, et al. Form and function of bone. In: Simon SR, ed. *Orthopaedic basic science: American Academy of Orthopaedic Surgeons*. Rosemont, IL: Port City Press, 1994:127–184.
77. Kato T, Kawaguchi H, Hanada K, et al. Single local injection of recombinant fibroblast growth factor-2 stimulates healing of segmental bone defects in rabbits. *J Orthop Res* 1998;16:654–659.
78. Keck SW, Kelly PJ. The effect of venous stasis on intra-osseous pressure and longitudinal bone growth in the dog. *J Bone Joint Surg* 1965;47A:539–544.
79. Keene DR, Oxford JT, Morris NP. Ultrastructural localization of collagen types II, IX, and XI in the growth plate of human rib and fetal bovine epiphyseal cartilage: type XI collagen is restricted to thin fibrils. *J Histochem Cytochem* 1995;43:967–979.
80. Kielty CM, Kwan AP, Holmes DF, et al. Type X collagen, a product of hypertrophic chondrocytes. *Biochem J* 1985;227:545–554.
81. Kirsch T, von der Mark K. Isolation of bovine type X collagen and immunolocalization in growth-plate cartilage. *Biochem J* 1990;265:453–459.
82. Klein Nulend J, Louwse RT, Heyligers IC, et al. Osteogenic protein (OP-1, BMP-7) stimulates cartilage differentiation of human and goat perichondrium tissue in vitro. *J Biomed Mater Res* 1998;40:614–620.
83. Klein Nulend J, Semeins CM, Mulder JW, et al. Stimulation of cartilage differentiation by osteogenic protein-1 in cultures of human perichondrium. *Tissue Eng* 1998;4:305–313.
84. Kleinulend J, Louwse RT, Heyligers IC, et al. Osteogenic protein (Op-1, Bmp-7) stimulates cartilage differentiation of human and goat perichondrium tissue in vitro. *J Biomed Mater Res* 1998;40:614–620.
85. Kronenberg HM, Lanske B, Kovacs CS, et al. Functional analysis of the PTH/PTHrP network of ligands and receptors. *Recent Prog Horm Res* 1998;53:283–301; discussion 301–303.
86. Lacroix P. The organization of bone. Philadelphia: Blakiston, 1951.
87. Legeai Mallet L, Benoist Lasselin C, Delezoide AL, et al. Fibroblast growth factor receptor 3 mutations promote apoptosis but do not alter chondrocyte proliferation in thanatophoric dysplasia. *J Biol Chem* 273:13007–13014.
88. Levi-Montalcini R, Hamburger V. Selective growth stimulating effects of mouse sarcoma on the sensory and sympathetic nervous system of the chick embryo. *J Exp Zool* 1951;116:321–362.
89. Light TR, McKinstry P, Schnitzer J, Ogden JA. Bone blood flow: regional variation with skeletal maturation. In: Arlet J, Ficat RP, Hungerford DS, eds. *Bone circulation*. Baltimore: Williams & Wilkins, 1984:XXX.
90. Lind M. Growth factor stimulation of bone healing. Effects on osteoblasts, osteomies, and implants fixation. *Acta Orthop Scand Suppl* 1998;283:2–37.
91. Lind M. Growth factors: possible new clinical tools. A review. *Acta Orthop Scand* 1996;67:407–417.
92. Lockwood R, Latta LL. Bone blood flow changes with diaphyseal fracture. *Trans Orthop Res Soc* 1980;5:158.
93. Luo G, Ducey P, McKee MD, et al. Spontaneous calcification of arteries and cartilage in mice lacking matrix GLA protein. *Nature* 1997;385:78–81.
94. Makower AM, Wroblewski J, Pawlowski A. Effects of IGF-I, rGH, FGF, EGF and NCS on DNA-synthesis, cell proliferation and morphology of chondrocytes isolated from rat rib growth cartilage. *Cell Biol Int* 1989;13:259–270.
95. Mancilla EE, De Luca F, Uyeda JA, et al. Effects of fibroblast growth factor-2 on longitudinal bone growth. *Endocrinology* 1998;139:2900–2904.
96. Margolin MD, Cogan AG, Taylor M, et al. Maxillary sinus augmentation in the non-human primate: a comparative radiographic and histologic study between recombinant human osteogenic protein-1 and natural bone mineral. *J Periodontol* 1998;69:911–919.
97. Marics I, Adelaide J, Raybaud F, et al. Characterization of the HST-related FGF.6 gene, a new member of the fibroblast growth factor gene family. *Oncogene* 1989;4:335–340.
98. Marks SC, Jr. Osteoclast biology: lessons from mammalian mutations. *Am J Med Genet* 1998;34:43–54.
99. Massague J, Weis Garcia F. Serine/threonine kinase receptors: mediators of transforming growth factor beta family signals. *Cancer Surv* 1996;27:41–64.
100. Massague J. Receptors for the TGF-beta family. *Cell* 1992;69:1067–1070.
101. Massague J. The transforming growth factor-b family, 1990;6:597–641
102. McClements P, Templeton RW, Pritchard JJ. Repair of a bone gap. *J Anat* 1961;95:616.
103. McKee MD, Nanci A. Osteopontin at mineralized tissue interfaces in bone, teeth, and osseointegrated implants: ultrastructural distribution and implications for mineralized tissue formation, turnover, and repair. *Microsc Res Tech* 1996;33:141–164.
104. McKibbin B. The biology of fracture healing in long bones. *J Bone Joint Surg* 1978;60B:150–162.
105. McKinstry P, Schnitzer JE, Light TR, et al. Relationship of 99mTC-MDP uptake to regional osseous circulation in skeletally immature and mature dogs. *Skeletal Radio* 1982;8:115–121.
106. McKinstry P, Schnitzer JE, Light TR, Ogden JA. Quantitation of regional chondro-osseous circulation in the maturing canine tibia and femur. *Am J Physiol* 1982;124:H365–H375.
107. Meikle MC, Bord S, Hembry RM, et al. Human osteoblasts in culture synthesize collagenase and other matrix metalloproteinases in response to osteotropic hormones and cytokines. *J Cell Sci* 1992;103:1093–1099.
108. Meikle MC, McGarrity AM, Thomson BM, Reynolds JJ. Bone-derived growth factors modulate collagenase and TIMP (tissue inhibitor of metalloproteinases) activity and type I collagen degradation by mouse calvarial osteoblasts. *Bone Miner* 1991;12:41–55.
109. Mizuta T, Benson WM, Foster BK, et al. Statistical analysis of the incidence of physeal injuries. *J Pediatr Orthop* 1987;7:518–523.
110. Moolenaar WH, Defize LHK, DeLaat SW. Calcium in the action of growth factors. Calcium and the Cell. *Ciba Found Symp* 1986;122:212–231.
111. Moore R, Casey G, Brooks S, et al. Sequence, topography and protein coding potential of mouse int-2: a putative oncogene activated by mouse mammary tumour virus. *EMBO J* 1986;5:919–924.
112. Morscher E. Posttraumatic zapfenepiphyse. *Arch Orthop Unfallchir* 1967;61:128–136.
113. Muir H. The chondrocyte, architect of cartilage. Biomechanics, structure, function and molecular biology of cartilage matrix macromolecules. *Bioessays* 1995;17:1039–1048.
114. Mulholland MC, Pritchard JJ. The fracture gap. *J Anat* 1959;93:590.
115. Nash TJ, Howlett CR, Martin C, et al. Effect of platelet-derived growth factor on tibial osteotomies in rabbits. *Bone* 1994;15:203–208.
116. Neufeld G, Gospodarowicz D. The identification and partial characterization of the fibroblast growth factor receptor of baby hamster kidney cells. *J Biol Chem* 1985;260:13860–13868.
117. Nilsson A, Ohlsson C, Isaksson OG, et al. Hormonal regulation of longitudinal bone growth. *Eur J Clin Nutr* 1994;48(Suppl):S150–S158; discussion S158–S160.
118. Ogden JA, Grogan DP, Light TR. Postnatal skeletal development and growth of musculoskeletal system. In: Albright JA, Brand RD, eds. *The scientific basis of orthopaedics*. New York: Appleton & Lange, 1987.
119. Ogden JA, Hempton R, Southwick WO. Development of the tibial tuberosity. *Anat Rec* 1975;182:431–446.
120. Ogden JA, Pals MJ, Murphy MJ, Bronson ML. Ectopic bone secondary to avulsion of the periosteum. *Skeletal Radio* 1979;4:124–128.
121. Ogden JA, Rosenberg LC. Defining the growth plate. In: Uthoff HK, Wiley JJ, eds. *Behavior of the growth plate*. New York: Raven Press, 1988.
122. Ogden JA, Southwick WO. Adequate reduction of fractures and dislocations. *Radiol Clin North Am* 1973;11:667–682.
123. Ogden JA, Southwick WO. Osgood-Schlatter's disease and the development of the tibial tuberosity. *Clin Orthop* 1976;116:180–189.
124. Ogden JA, Tross RB, Murphy MJ. Fractures of the tibial tuberosity in adolescents. *J Bone Joint Surg* 1980;62A:205–215.

125. Ogden JA. An anatomical and histological study of the factors affecting development and evolution of avascular necrosis in congenital dislocation of the hip. In: Harris WH ed. *The hip: proceedings of the Hip Society*, vol 2. St. Louis: CV Mosby, 1974:125–153.
126. Ogden JA. Chondro-osseous development and growth. In: Urist MR, eds. *Fundamental and clinical bone physiology*. Philadelphia: JB Lippincott, 1980.
127. Ogden JA. Growth slowdown and arrest lines. *J Pediatr Orthop* 1984;4:409–415.
128. Ogden JA. Injury to the immature skeleton. In: Touloukian R, eds. *Pediatric trauma*, 2nd ed. New York: John Wiley & Sons, 1990.
129. Ogden JA. *Skeletal injury in the child*, 2nd ed. Philadelphia: WB Saunders, 1990.
130. Ogden JA. The development and growth of the musculoskeletal system. In: Albright JA, Brand RA, eds. *The scientific basis of orthopaedics*. New York: Appleton-Century-Crofts, 1979.
131. Ogden JA. The role of orthopaedic Surgery in sports medicine. *Yale J Biol Med* 1980;53:281–288.
132. Ogden JA, Southwick WO. Electrical injury involving the immature skeleton. *Skeletal Radio*, 1981;6:187–192.
133. Ohlsson C, Nilsson A, Isaksson O, Lindahl A. Growth hormone induces multiplication of the slowly cycling germinal cells of the rat tibial growth plate. *Proc Natl Acad Sci U S A* 1992;89:9826–9830.
134. Olsen BR. Mutations in collagen genes resulting in metaphyseal and epiphyseal dysplasias. *Bone* 1995;17:45s–49s.
135. Owen M. The origin of bone cells. *Int Rev Cyto* 1970;28:213–238.
136. Partridge WM. Transport of protein-bound hormones into tissues in vivo. *Endocr Rev* 1981;2:102–123.
137. Park EA. Bone growth in health and disease. *Arch Dis Child* 29:269–281.
138. Park EA. The imprinting of nutritional disturbances on growing bone. *Pediatrics* 1964;33:815–862.
139. Paschalis EP, Jacenko O, Olsen B, et al. The role of type X collagen in endochondral ossification as deduced by Fourier transform infrared microscopy analysis. *Connect Tissue Res* 35:371–377.
140. Pearce J, Penny G, Rossant J. A mouse cerberus/Dan-related gene family. *Dev Biol* 1999;209:98–110.
141. Petit B, Ronziere MC, Hartmann DJ, Herbage D. Ultrastructural organization of type XI collagen in fetal bovine epiphyseal cartilage. *Histochemistry* 1993;100:231–239.
142. Piccolo S, Sasai Y, Lu B, De Robertis EM. Dorsal-ventral patterning in Xenopus: inhibition of ventral signals by direct binding of chordin to BMP-4. *Cell* 1996;86:589–598.
143. Poole AR, Matsui Y, Hinek A, Lee ER. Cartilage macromolecules and the calcification of cartilage matrix. *Anat Rec* 1989;224:167–179.
144. Poole AR, Webber C, Pidoux I, et al. Localization of a dermatan sulfate proteoglycan (DS-PGII) in cartilage and the presence of an immunologically related species in other tissues. *J Histochem Cytochem* 1986;34:619–625.
145. Potts WJ. The role of the hematoma in fracture healing. *Surg Gynecol Obstet* 1933;57:318–324.
146. Price PA. Gla-containing proteins of bone. *Connect Tissue Res* 1989;21:51–57.
147. Price PA, Williamson MK. Primary structure of bovine matrix Gla protein, a new vitamin K-dependent bone protein. *J Biol Chem* 1985;260:14971–14975.
148. Pritchard JJ, Ruzicka AJ. Comparison of fracture repair in the frog, lizard, and the rat. *J Anat* 1950;84:236–261.
149. Reinholt FP, Hultenby K, Oldberg A, Heinegard D. Osteopontin—a possible anchor of osteoclasts to bone. *Proc Natl Acad Sci U S A* 1990;87:4473–4475.
150. Rhinelander FW, Phillips RS, Steel WM, Bier JC. Microangiography and bone healing. II. Displaced closed fractures. *J Bone Joint Surg* 1968;50A:643–662.
151. Rhinelander FW. Tibial blood supply in relation to healing. *Clin Orthop* 1974;105:34–81.
152. Roche AF, Wainer H, Thissen D. *Skeletal maturity—the knee joint as a biological indicator*. New York: Plenum, 1975.
153. Rohlig H. Perlost und langenzwachstum. *Beitr Orthop Traumatol* 1966;13:603–606.
154. Rokhanen P, Statis P. The repair of experimental fractures during long-term anticoagulant treatment. *Acta Orthop Scand* 1964;35:21–38.
155. Rosati R, Horan GS, Pinerio GJ, et al. Normal long bone growth and development in type X collagen-null mice. *Nat Genet* 1994;8:129–35.
156. Rubin JS, Osada H, Finch PW, et al. Purification and characterization of a newly identified growth factor specific for epithelial cells. *Proc Natl Acad Sci U S A* 1989;86:802–806.
157. Ryoppy S. Injuries of the growing skeleton. *Ann Chir Gynaecol* 1972;61:3–10.
158. Sandberg MM, Aro HT, Vuorio EI. Gene expression during bone repair. *Clin Orthop* 1993;289:292–312.
159. Sandell LJ, Sugai JV, Trippel SB. Expression of collagens I, II, X, and XI and aggrecan mRNAs by bovine growth plate chondrocytes in situ. *J Orthop Res* 1994;12:1–14.
160. Schmitt J, Hwang K, Winn S, Hollinger J. Bone morphogenetic proteins: an update on basic biology and clinical relevance. *J Orthop Res* 1999;17:269–278.
161. Schnitzer JE, McKinstry P, Light TR, Ogden JA. Quantitation of regional osseous circulation in the maturing canine tibia and femur. *Surg Forum* 1980;31:509–511.
162. Schonherr E, Broszat M, Brandan E, et al. Decorin core protein fragment Leu155-Val260 interacts with TGF-beta but does not compete for decorin binding to type I collagen. *Arch Biochem Biophys* 1998;355:241–248.
163. Schonherr E, Hausser H, Beavan L, Kresse H. Decorin-type I collagen interaction. Presence of separate core protein-binding domains. *J Biol Chem* 1995;270:8877–8883
164. Schonherr E, Witsch Prehm P, et al. Interaction of biglycan with type I collagen. *J Biol Chem* 1995;270:2776–2783.
165. Schultz GS, Grant MB. Neovascular growth factors. *Eye* 1991;5:170–180.
166. Siffert RS. The effect of trauma to the epiphysis and growth plate. *Skeletal Radio*. 1977;2:21–30.
167. Siffert RS. The growth plate and its affections. *J Bone Joint Surg* 1966;48A:546–563.
168. Simkin A, Robin G. Fracture formation in differing collagen fiber patterns of compact bone. *J Biomech* 1974;7:183–188.
169. Solloway MJ, Robertson EJ. Early embryonic lethality in Bmp5;Bmp7 double mutant mice suggests functional redundancy within the 60A subgroup. *Development* 1999;126:1753–1768.
170. Speer D. Collagenous architecture of the growth plate and perichondral ossification groove. *J Bone Joint Surg* 1982;64A:399–407.
171. Streicher HJ. Bericht über 1500 kindliche und jugendliche frakturen. *Hefte Unfallchir* 1956;35:129.
172. Taira M, Yoshida T, Miyagawa K, et al. cDNA sequence of human transforming gene hst and identification of the coding sequence required for transforming activity. *Proc Natl Acad Sci U S A* 1987;84:2980–2984.
173. Takiguchi T, Kobayashi M, Suzuki R, et al. Recombinant human bone morphogenetic protein-2 stimulates osteoblast differentiation and suppresses matrix metalloproteinase-1 production in human bone cells isolated from mandibulae. *J Periodontol Res* 1998;33:476–485.
174. Thomas, KA, Rios-Candelore M, Fitzpatrick S. Purification and characterization of acidic fibroblast growth factor from bovine brain. *Proc Natl Acad Sci USA* 1984;81:357–361.
175. Tonna EA, Cronkite EP. Cellular response to fracture studied with tritiated thymidine. *J Bone Joint Surg* 1961;43A:352–362.
176. Treharne RW. Review of Wolff's law and its proposed means of operation. *Orthop Rev* 1981;10:35–44.
177. Trippel SB, Chernauek SD, Van Wyk JJ, et al. Demonstration of type I and type II somatomedin receptors on bovine growth plate chondrocytes. *J Orthop Res* 1988;6:817–26.
178. Trippel SB. Growth factors as therapeutic agents. *Instr Course Lect* 1997;46:473–476.
179. Trippel SB. Potential role of insulin-like growth factors in fracture healing. *Clin Orthop* 1998;355:S301–S313.
180. Trueta J, Cavadias AX. A study of the blood supply of the long bones. *Surg Gynecol Obstet* 1964;118:485–498.
181. Trueta J, Morgan JD. The vascular contribution to osteogenesis. *J Bone Joint Surg* 1960;42B:97–109.
182. Trueta J. *Studies of the development and decay of the human frame*. Philadelphia: W.B. Saunders, 1968.
183. Trueta, O, Amato, P. The vascular contribution to osteogenesis III. Changes in the growth cartilage caused by experimentally induced ischaemia. *J Bone Joint Surg* 1960;42B:571–587.
184. Tscherne H, Suren EG. Fehlstellungen, wachstumsstörungen und pseudoarthrosen nach kindlichen frakturen. *Langenbecks Arch Chir* 1976;342:299–304.
185. Uthoff HK, Rahn B. Healing patterns of metaphyseal fractures. *Trans Orthop Res Soc* 1981;6:40.
186. Volk SW, Luvalle P, Leask T, Leboy PS. A Bmp-responsive transcriptional region in the chicken type X collagen gene. *J Bone Miner Res* 1998;13:1521–1529.
187. Vortkamp A, Lee K, Lanske B, et al. Regulation of rate of cartilage differentiation by Indian hedgehog and PTH-related protein. *Science* 1996;273:613–622.
188. Wang EA, Rosen V, D'Alessandro JS, et al. Recombinant human bone morphogenetic protein induces bone formation. *Proc Natl Acad Sci U S A* 1990;87:2220–2224.
189. Wang JS. Basic fibroblast growth factor for stimulation of bone formation in osteoinductive or conductive implants. *Acta Orthop Scand Suppl* 1996;269:1–33.
190. Warrell E, Taylor JF. The effect of trauma on tibial growth. *J Bone Joint Surg* 1976;58B:375.
191. Weinman DT, Kelly PJ, Owen CA. Blood flow in bone distal to a femoral arteriovenous fistula in dogs. *J Bone Joint Surg* 1964;46A:1676–1682.
192. Whang K, Tsai DC, Nam EK, et al. Ectopic bone formation via rhBMP-2 delivery from porous bioabsorbable polymer scaffolds. *J Biomed Mater Res* 1998;42:491–499.
193. Wieser RJ, Janik-Schmitt B, Renaver D, et al. Contact-dependent inhibition of growth of normal diploid human fibroblasts by plasma membrane glycoproteins. *Biochimie* 1988;70:1661–1671.
194. Wray JB. Acute changes in femoral arterial blood flow after closed tibial fracture in dogs. *J Bone Joint Surg* 1964;46A:1262–1268.
195. Xu T, Bianco P, Fisher LW, et al. Targeted disruption of the biglycan gene leads to an osteoporosis-like phenotype in mice. *Nat Genet* 1998;20:78–82.
196. Yamagishi M, Toshimure Y. The biomechanics of fracture healing. *J Bone Joint Surg* 1955;37A:1035–1068.
197. Yamaguchi Y, Mann D, Ruoslahti E. Negative regulation of transforming growth factor-B by the proteoglycan decorin. *Nature* 1990;346:281–284.
198. Yamamoto N, Akiyama S, Katagiri T, et al. Smad1 and smad5 act downstream of intracellular signalings of BMP-2 that inhibits myogenic differentiation and induces osteoblast differentiation in C2C12 myoblasts. *Biochem Biophys Res Commun* 1997;238:574–580.
199. Zhan X, Bates B, Hu X, Goldfarb M. The human FGF-5 oncogene encodes a novel protein related to fibroblast growth factors. *Mol Cell Bio*. 1988;8:3487–3495.

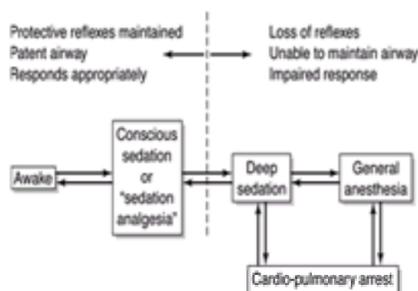
[Guidelines and Principles of Sedation in Children](#)[Definitions](#)[Monitoring](#)[Patient Assessment](#)[Oral Intake Precautions](#)[Hemodynamic Status](#)[Coexisting Nonmusculoskeletal Injuries](#)[Status of the Airway](#)[Treatment Facility](#)[Sedative Medications](#)[Nitrous Oxide](#)[Ketamine](#)[Benzodiazepines](#)[Opioids](#)[Chloral Hydrate](#)[Barbiturates](#)[Propofol](#)[Regional Anesthesia in the Child with a Musculoskeletal Injury](#)[Regional Anesthetic Agents](#)[Local Anesthetic Toxicity](#)[Intravenous Regional Anesthesia](#)[Local Infiltration Anesthesia: Hematoma Block](#)[Femoral Nerve Block](#)[Postoperative Analgesia in the Child with a Musculoskeletal Injury](#)[Postoperative Analgesia With Opioids](#)[Postoperative Analgesia With Nonsteroidal Antiinflammatory Drugs](#)[Postoperative Analgesia With Local Anesthetic Agents](#)[Treatment of Postoperative Nausea](#)[Chapter References](#)

Providing pain relief is one of the many important parts of the management of children's fractures. In addition, because having a fracture reduced is not only painful but also frightening to many children, providing young patients with adequate sedation and amnesia are additional welcome elements of good care. However, the correct use of any of the available medications for obtaining these goals must involve an appropriate understanding of proper dose, desired effects, and untoward side effects. The purpose of this chapter is to provide a thorough source of information regarding safe and effective analgesia and sedation for children with fractures. This chapter discusses the concept of sedation and its definitions, the various medications used to achieve the sedation state, and the various medications used to achieve analgesia, including both systemic medications and local anesthetics. Intravenous regional anesthesia (Bier Blocks), hematoma blocks, and femoral nerve blocks (for femur fractures) are discussed in depth. The management of postoperative pain is discussed, along with the treatment of the troublesome side effect of postoperative nausea. The author hopes that the orthopaedic practitioner will find this chapter of significant benefit, not only in the emergency room setting but also in the office and on the hospital ward.

## GUIDELINES AND PRINCIPLES OF SEDATION IN CHILDREN

### Definitions

The practitioner must recognize that sedation describes a continuum ranging from near wakefulness to complete loss of consciousness ( [Fig. 3-1](#)). Terms used to describe various stages along this continuum have included conscious sedation, deep sedation, and general anesthesia ( [63](#)).



**FIGURE 3-1.** Sedation and analgesia for procedures is a continuum. (Reproduced with permission from American Society of Anesthesiologists from ASA 1997 Annual Refresher Course Lectures. Sedation and Analgesia in Pediatric Patients for Procedures Outside the Operating Room. Richard F. Kaplan, M.D. No. 221)

Strictly speaking, the term conscious sedation means a pharmacologically controlled altered state of consciousness in which patients maintain their ability to respond purposefully to verbal commands. For nonverbal patients or young infants, conscious sedation implies the ability to respond purposefully to physical stimulation, not simply by reflex withdrawal to pain. Unfortunately, most physician and nursing personnel tend to use the term "conscious sedation" to mean anything short of a general anesthetic. For such reasons, the consensus of the 1996 report by the American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists ( [108](#)) is that the term conscious sedation, although in common usage, is imprecise. This report recommends replacing the term conscious sedation with the more descriptive term sedation/analgesia ( [Fig. 3-1](#)). Whatever the preferred term, the important point to recognize is that the safest level of sedation is that which permits purposeful response to verbal or physical stimulation. It is at this level of sedation that the risk of hypoventilation, apnea, or cardiovascular instability is minimal. Unfortunately, and realistically speaking, such relatively light levels of sedation are totally inadequate for the performance of a painful procedure such as the reduction of a fracture. Also the younger and less cooperative the patient, the less likely that so-called conscious sedation can realistically be achieved at all ( [92](#)). Therefore, it is very likely that for orthopaedic procedures, children may have to be sedated to levels at which they are not easily responsive to verbal stimulation, and as such, at increased risk for respiratory and cardiovascular compromise. Even in children in whom light levels of sedation (true conscious sedation) is possible, unintended oversedation may occur without warning. Oversedation may lead to (a) loss of the airway, (b) impaired protective reflexes leading to the possibility of aspiration of gastric contents, and (c) cardiopulmonary arrest ( [Fig. 3-1](#)). It is for these reasons that careful monitoring of sedated patients as prescribed in standard guidelines ( [108](#)) is absolutely imperative.

### Monitoring

Obviously, the purpose for monitoring sedated patients is to provide timely detection and correction of abnormalities in respiratory and cardiovascular function. The

monitoring process begins before the administration of any sedative medications. Monitoring continues unabated until the patient returns to baseline pre-sedation level of consciousness and is ready for discharge. Acceptable discharge criteria are noted later ( [Table 3-1](#)).

1. Cardiovascular function and airway patency are satisfactory and stable.
2. The patient is easily arousable, and protective reflexes are intact.
3. The patient can talk (if age appropriate).
4. The patient can sit up unaided (if age appropriate).
5. For a very young or handicapped child incapable of the usually expected responses, the pre-sedation level of responsiveness or a level as close as possible to the normal level for that child should be achieved.
6. The state of hydration is adequate.\*

\* Adequate hydration may be achieved with intravenous fluids. There is no specific requirement that children be able to tolerate oral fluids before discharge from a treatment facility. Children who are nauseated, or actively vomiting should be treated and observed until this problem resolves (see section on treatment of postoperative nausea).  
Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures. Pediatrics 1992;89:1110-1115, with permission.

### TABLE 3-1. RECOMMENDED DISCHARGE CRITERIA AFTER SEDATION

Of vital importance to the monitoring process is the presence of qualified personnel who are competent in the use of monitoring devices, and capable of recognizing the clinical signs of airway or hemodynamic instability. Although skill in at least pediatric basic life support is necessary ( [63,119](#)), training in pediatric advanced life support (PALS) is certainly desirable. In the author's opinion, this training should be considered absolutely necessary. The orthopaedic surgeon must always demand, and have available, skilled health professionals, either physicians or nurses, to assist in observing the patient and the monitors during procedures requiring medications that are known to depress respiratory or cardiovascular function. Having one person performing both the surgical procedure and monitoring the patient is a practice that should be strongly discouraged in all but the most desperate circumstances.

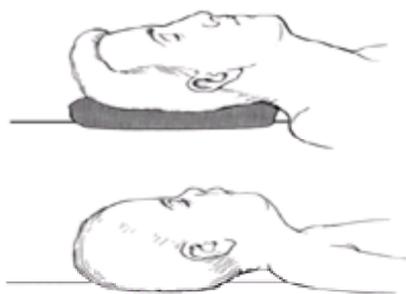
Oxygenation, ventilation, and circulation are the three parameters that require careful assessment. For the most part, monitoring temperature is usually of minimal importance. The major exceptions, of course, are children who arrive in the hospital either severely hypothermic or febrile.

Monitoring oxygenation requires continuous pulse oximetry and continual visual inspection of the patient. Note that the term continuous monitoring refers to a constant measurement undertaken for a period of time without interruption. Continual monitoring refers to an assessment taken at frequent regular intervals.

The value of pulse oximetry as an early detector of impending hypoxemia has been well demonstrated ( [34](#)). The problem with relying on visual inspection alone to determine adequacy of oxygenation is that cyanosis is both a late and variable sign of hypoxemia. Demonstrable cyanosis requires the presence of at least 5 g of desaturated hemoglobin per deciliter. Therefore, for example, a patient with a hemoglobin level of 10 g/dL would theoretically not even appear cyanotic until the oxygen saturation level (SpO<sub>2</sub>) plummets to 50%. For this same reason, a severely anemic patient may never develop visible cyanosis even at profound levels of hypoxemia. To add to a potentially confusing situation, the ambient light (especially fluorescent light) in many clinical environments may make any patient appear cyanotic ( [33](#)). Therefore, pulse oximetry is essential in all heavily sedated patients to detect abnormalities of oxygenation rapidly.

Note, however, that the pulse oximeter is not perfect. Factors that affect the accuracy of the pulse oximeter include patient movement ( [33](#)), direct bright light on the probe ( [21](#)), and malposition of the probe ( [9](#)). Correct probe placement, shielding the probe site from bright light, and gentle restraint of the monitoring site should improve the dependability of this all-important monitor.

Monitoring ventilation goes hand in hand with monitoring oxygenation. Monitoring ventilation requires close observation of the patient and either intermittent or continuous auscultation of breath sounds. A sedated child's head may flex forward easily, producing airway obstruction as the child begins to fall asleep ( [33](#)). Maintaining patients in the so-called sniffing position helps prevent airway obstruction ( [Fig. 3-2](#)). The sniffing position consists of elevating the patient's head with pads under the occiput, keeping the shoulders flat on the table, and extending the head at the atlanto-occipital joint ( [134](#)). Children younger than 3 years of age have a relatively large head in proportion to the size of their trunk and do not require padding under the occiput ( [35](#)). Along with continual assessment of the child's head position, any restraining devices should be checked and rechecked to ensure that they are not contributing to either airway obstruction or restriction of chest movement ( [63](#)).



**FIGURE 3-2.** The sniffing position. In an adult or in an older child, a folded sheet or towel under the occiput, plus moderate head extension at the atlanto-occipital joint, helps to maintain an open airway. In a child younger than 3 years of age, the relatively large head size in proportion to the trunk makes occipital padding unnecessary ( [35](#)).

Auscultation with the precordial stethoscope is valuable in the monitoring of both ventilation (breath sounds) and circulation (heart sounds). Its use is encouraged in the monitoring of deeply sedated patients ( [63](#)).

Monitoring circulation for most sedated children consists of intermittent determination of heart rate and blood pressure ( [63](#)). For children, normal values for heart rate and blood pressure vary with age ( [Table 3-2](#) and [Table 3-3](#)). A simple formula for calculating the normal systolic blood pressure and lower limit of normal for systolic blood pressure in children by age is worth memorizing ( [Table 3-4](#)). Electrocardiographic (ECG) monitoring is especially important for the child with an underlying history of a significant cardiac dysrhythmia or known ECG abnormality such as long QT syndrome, or history of Wolff-Parkinson-White syndrome. In the absence of monitor artifact, the pulse oximeter provides continuous assessment of heart rate. Deeply sedated children should have blood pressure and heart rate and respiratory rate measurements determined and recorded at least at 5-minute intervals ( [63](#)). For children under conscious sedation (sedation/analgesia), the frequency of vital sign determination is at the discretion of the physician ( [63](#)).

Age	Range (beats/min)
Newborn	110-150
1-11 months	80-150
2 years	85-125
4	75-115
6	65-110
8	60-110

Rasch DK, Webster DE. *Clinical manual of pediatric anesthesia*. New York: McGraw-Hill, 1994:16, with permission.

**TABLE 3-2. NORMAL VALUES FOR HEART RATE BY AGE**

Age	Blood Pressure (mm Hg)	
	Systolic	Diastolic
Full-term infant	60 (45)*	35
3-10 day	70-75 (50)	40
6 mo	95 (55)	45
4 yr	98	57
6 yr	110	60
8 yr	112	60
12 yr	115	65
16 yr	120	65

\* The numbers in parentheses refer to mean arterial blood pressure. Steward DJ. *Manual of pediatric anesthesia*. New York: Churchill-Livingstone, 1990:24, and Rasch DK, Webster DE. *Clinical Manual of Pediatric Anesthesia*. New York: McGraw-Hill, 1994:17, with permission.

**TABLE 3-3. NORMAL VALUES FOR BLOOD PRESSURE BY AGE**

$$80 + (2 \times \text{age in years}) = \text{normal systolic BP for age}$$

$$70 + (2 \times \text{age in years}) = \text{lower limit of normal systolic BP for age}$$

Rasch DK, Webster DE. *Clinical Manual of Pediatric Anesthesia*. New York: McGraw-Hill, 1994:197, with permission.

**TABLE 3-4. CALCULATION OF NORMAL BLOOD PRESSURE BY AGE**

It is important to emphasize again that monitoring the patient must continue until the patient meets preset discharge criteria ( [Table 3-1](#) (63)). Often, when a surgical procedure is over and patients are no longer being actively stimulated, unintentional deep sedation with resulting airway obstruction and apnea may occur. Therefore, it is essential to remain vigilant until the patient recovers completely from their sedative medications. Note that the time to recovery varies depending on the amount and type of sedative medication given, and this point should be taken into account when planning a sedation regimen. The duration of action of particular sedatives and sedative combinations are discussed separately.

#### Patient Assessment

Only after careful patient assessment can the practitioner decide whether sedation in an emergency room setting, where the airway is uncontrolled and unprotected, is feasible and safe. Administering sedative medications without first evaluating a patient is an invitation for disaster. It is important first to be aware of the child's medical history, previous allergic or adverse drug reactions, current medications, and presence of coexisting diseases before proceeding with deliberate alteration of a patient's state of consciousness (63). In addition to these basic details, the patient with musculoskeletal injuries has to be evaluated for time of last oral intake, hemodynamic status, presence of other injuries, and status of the airway.

#### Oral Intake Precautions

In the management of elective patients, significant pulmonary aspiration is rare in pediatrics (143).

Note that for elective patients, multiple studies support and encourage the liberal intake of clear liquids up until 2 to 3 hours before scheduled surgery in otherwise healthy children (32,95,122,129). Acceptable clear liquids are apple juice, water, sugar, water, and gelatin. Milk (including breast milk), milk products, and juices with pulp are not clear liquids. For elective patients, most pediatric anesthesiologists now adhere to the so-called 2-4-6-8 rule regarding oral intake. This rule restricts clear fluids to 2 hours before elective surgery, breast milk to 4 hours before elective surgery, baby formula (cow's milk formula) to 6 hours before elective surgery, and solid food to 8 hours before elective surgery (50).

Emergency patients are definitely at risk for aspiration (143). Sedatives depress the protective reflexes (63). Caution is necessary to avoid the morbidity and possible mortality of aspiration pneumonia.

It is known that in trauma patients, the time interval between last oral intake and time of injury is a critical factor in the retention of gastric contents (99). It is clear that children injured within 1 to 2 hours after eating present with large gastric volumes (19). Although fasting can reduce an injured child's gastric volume (95), it is not clear how long of a fast is ideal. Also, in the presence of pain, anxiety, and with the administration of opioid pain relievers, all of which may occur in the patient with a fracture, gastric emptying may be radically slowed (57). If the procedure can wait, it is safe to say that a fasting period is in order. Starting intravenous fluids to prevent dehydration is important. Note that at present, there is no reliable method of assessing the volume of gastric contents, although different methods have been suggested (55). Patient hunger on presentation for surgical treatment is not a good indicator of an empty stomach (95).

In the author's opinion, the safest recommendations regarding sedation of patients with a potential full stomach are as follows:

1. If possible, postpone the procedure, even for as little as 4 hours.
2. Use the lightest effective level of sedation. Titrate sedation to effect, and avoid large bolus doses of medications.
3. Consider the administration of medications to reduce gastric volume (metoclopramide) or to increase gastric pH (histamine-2-receptor blockers). These medications, when indicated, should be administered intravenously 1 hour before sedative medications are given. The appropriate dose of metoclopramide (Reglan) is 0.15 mg/kg. The usual adult dose for metoclopramide is 10 mg, which should be more than sufficient for any child. Famotidine (Pepcid), a histamine-2-receptor blocker, may be given in a dose of 0.3 to 0.4 mg/kg intravenously, with a maximum dose of 20 mg.
4. Within 15 minutes of administering sedation, consider the use of oral nonparticulate antacids (Bicitra, sodium citrate) to raise gastric pH. Unfortunately, these

medications are not very palatable.

- Note that pregnancy, morbid obesity, gastroesophageal reflux, bowel obstruction, and increased intracranial pressure all magnify the risk of regurgitation and aspiration of gastric contents. Therefore, additional caution is necessary in managing patients with any of these conditions. Patients with coexisting bowel obstruction should not be sedated, and patients with increased intracranial pressure should not be sedated without the input and knowledge of the attending neurosurgical staff.
- If treatment cannot wait and the surgical procedure or the patient is not amenable to regional anesthesia, the safest approach is a general anesthetic with a rapid sequence induction and a protected airway (endotracheal tube). This approach is also safest for uncooperative children who urgently need a computed tomography (CT) scan, an magnetic resonance imaging (MRI) scan, or other detailed diagnostic studies.

### Hemodynamic Status

The magnitude of blood loss from a child's injuries is not always readily apparent. In children, long bone fractures and head injuries may easily have associated large concealed hemorrhages (140,153). It is important to assess the patient's volume status accurately before administering sedative medications. In a hypovolemic child, sedatives may interfere with catecholamine-mediated compensatory mechanisms and produce profound hemodynamic instability, leading to cardiovascular collapse.

Note that in an injured child, blood pressure monitoring alone does not provide a good indication of the patient's underlying volume status (105,155). Children maintain a normal blood pressure for their age in the face of large intravascular volume deficits (155). More reliable signs of ongoing hypovolemia in children include sinus tachycardia, mottling, cool extremities, poor urine output (less than 1 to 2 mL/kg/h), and altered state of consciousness. Each of these signs can imply poor perfusion of different organ systems skin, musculoskeletal system, kidneys, and central nervous system (CNS), respectively). Volume replacement, not sedation, should be the initial goal in the management of hypovolemic children.

### Coexisting Nonmusculoskeletal Injuries

Serious head injury accounts for 70% of pediatric trauma deaths (30,105). Respiratory depression from sedation, with resultant hypercapnia and hypoxia, may aggravate an underlying closed head injury and worsen its prognosis (153). In addition, any pharmacologic change in the patient's state of consciousness may confuse the neurologic evaluation. Other injuries to major body cavities or injuries associated with major blood loss should be assessed carefully before any sedative medications are administered.

### Status of the Airway

A tenuous airway can easily become a completely obstructed airway in a sedated child. There are common problems in children associated with airway obstruction. For example, children with large tonsils and adenoids may have obstructive sleep apnea (89). Obstructive sleep apnea, which is associated with a history of loud snoring and daytime sleepiness, may be acutely exacerbated with the administration of sedative medications (36). Other potentially dangerous problems include micrognathia (short jaw), limited ability to open the mouth, and limited movement of the neck, either congenital or acquired (12).

### Treatment Facility

What about the medical facility where the child is receiving treatment? On-site resuscitation equipment, including equipment for airway management (Table 3-5) and equipment for vascular access (Table 3-6) must be available for children of all ages and sizes (63). In addition, a positive pressure oxygen delivery system capable of delivering at least 90% oxygen for at least 60 minutes must also be readily available (63). A working suction apparatus (63) must be easily accessible to handle patient secretions, as well as for unexpected regurgitation and vomiting. These recommendations are essential for patient safety and for optimum patient care.

Ventilation Face Masks\*  
Infant, child, small adult, medium adult, large adult  
Swallowing bag and valve set  
Oral airways  
Infant, child, small adult, medium adult, large adult  
Nasal airways  
Small, medium, large  
Laryngoscope handles  
Laryngoscope blades  
Straight (Miller) No. 1, 2, 3  
Curved (Macintosh) No. 1, 2, 3  
Endotracheal tubes  
2.5-6.0 uncuffed  
6.0-8.0 cuffed  
Appropriate sized stylets for endotracheal tubes  
(Must be lubricated before insertion)  
Appropriate sized suction catheters for endotracheal tubes  
Yankauer-type suction  
Nasogastric tubes  
10-, 18-French  
Nebulizer set-up for treatment of bronchospasm

\* The correct sized ventilation face mask will fit over the child's face from the bridge of the nose to the cleft of the chin. This guideline is also correct when using patient-administered nitrous oxide analgesia.  
Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures. Pediatrics, 1992;89:1110-1115, with permission.

### TABLE 3-5. AIRWAY MANAGEMENT EQUIPMENT

Intravenous catheters  
(24-18 gauge)\*  
Intraosseous bone-marrow needle  
Intravenous tubing  
Pediatric drip (60 drops/mL)  
Pediatric burette-type  
Adult drip (10 drops/mL)  
Intravenous fluids  
Lactated Ringer's  
Normal saline  
Miscellaneous equipment  
Tourniquets  
Alcohol wipes  
Arm boards

\* In resuscitation situations, no more than 90 sec should be spent attempting to gain peripheral venous access. If attempts have been unsuccessful, then central venous cannulation, intraosseous cannulation, or peripheral venous cutdown should be done according to the expertise of available personnel.  
Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures. Pediatrics, 1992;89:1110-1115, with permission.

### TABLE 3-6. VASCULAR ACCESS EQUIPMENT

## SEDATIVE MEDICATIONS

Having now considered the preliminary step of patient assessment, the practitioner must now decide which sedative or sedatives to use. The ideal sedative should be easy to administer, quick in onset, devoid of side effects, and rapid in termination of effects. The abundance of references in the literature extolling the virtues of different sedative drugs and drug combinations is the best indicator that we do not yet have the ideal sedative. Each of the drugs that is discussed has only some of the properties of an ideal sedative medication. Also, patients demonstrate great variability in response to medications. It is important to treat each patient as an individual and to not expect to be able to fit every child with a fracture into any particular sedation regimen. For patients who cannot be adequately sedated, the orthopaedic surgeon should consult an anesthesiologist for provision of a brief, well-controlled general anesthetic.

### Nitrous Oxide

Self-administered 50% nitrous oxide (50% nitrous oxide and 50% oxygen) has been found to be moderately useful in providing sedation and analgesia for the reduction of children's fractures. Evans and co-workers (45) found it to be comparable in efficacy to intramuscular meperidine (2 mg/kg) and promethazine (1 mg/kg). However, in a different study, Hennrikus and co-workers noted that 46% of their patients experienced significant pain with nitrous oxide alone as a sedative and analgesic for fracture reduction (67). Patients with completely displaced radius and ulna fractures had a statistically higher incidence of failure to achieve analgesia (67). With the addition of a hematoma block (discussed in a subsequent section), Hennrikus and his coinvestigators were able to obtain a 97% incidence of adequate

sedation and analgesia (66). This study does illustrate the important point that where possible, the use of regional anesthesia, in combination with almost any sedation regimen is an excellent way to enhance pain relief and to minimize the need for systemic sedative and analgesics.

In general, nitrous oxide is a weak sedative and analgesic. It does have the advantages of rapid onset, relative ease of utilization, and rapid termination of effects (88). Because it diffuses rapidly into enclosed air-filled spaces, its use is contraindicated in patients with bowel obstruction or pneumothorax (88). Nitrous oxide is also contraindicated in patients with altered intracranial compliance (88).

Although nitrous oxide is perhaps a useful part of the sedation armamentarium, this author does not believe that the literature supports the use of nitrous oxide alone as a reliable sedative and analgesic for pediatric orthopedic procedures.

## **Ketamine**

Ketamine, which is structurally related to phencyclidine, was first synthesized in 1963. Developed to produce the "anesthetic state (analgesia, amnesia, loss of consciousness and immobility)" without total CNS depression, it was approved for general clinical use in 1970 (31,157).

The commercial preparation of ketamine is a racemic mixture of two optical isomers with differing activity (157). Ketamine is typically administered intravenously or intramuscularly (59,136). Rectal (118), oral (64,145), and intranasal administration (154) have been described in the literature.

Ketamine is metabolized in the liver, primarily by *N*-methylation to norketamine. Norketamine has about one third the sedative and analgesic potency of ketamine. As such, ketamine should be administered cautiously or in reduced doses to patients with impaired hepatic function.

Intravenous ketamine, 1 to 2 mg/kg, produces unconsciousness within 30 to 60 seconds (136). Peak plasma concentrations occur within 1 minute. Return of consciousness occurs within 10 to 15 minutes, although complete recovery may be delayed (136). Dose requirements and recovery times from ketamine are age related (24,87).

Ketamine has been found to have interactions at multiple binding sites, including *N*-methyl-D-aspartate (NMDA) and non-NMDA receptors, nicotinic and muscarinic cholinergic receptors and opioid receptors (83). Agonist actions of ketamine on opioid receptors play only a minor role in its analgesic effects (83). Note that naloxone, a narcotic antagonist that is further discussed in the section on opioids, does not reverse the analgesic effect of ketamine (83). The psychotomimetic effects of ketamine, however, may involve interaction with a specific subclass of opioid receptors known as kappa receptors (83). For analgesia, the main site of action is the NMDA receptor. The reader is referred to other sources for further information on this topic (83).

### **Central Nervous System Effects of Ketamine**

Ketamine produces a state known as dissociative anesthesia. Dissociative anesthesia refers to a cataleptic state characterized by functional and electrophysiologic dissociation between the thalamocortical and limbic systems (157). Patients keep their eyes open and exhibit a slow nystagmic gaze. Corneal and pupillary reflexes remain intact. Generalized hypertonicity may be present. Even though ketamine has effects on nicotinic acetylcholine receptors in skeletal muscle, this effect is of minor significance, because ketamine increases muscle tone by central mechanisms (83). Patients receiving ketamine may exhibit purposeful movements but not necessarily in response to surgical stimulation (157).

Ketamine's analgesic effect is intense and may outlast its sedative effect (59). In one study of minor surgical procedures with ketamine anesthesia, no additional analgesics were required for 24 hours postoperatively (69). Amnesia persists for about one hour after apparent recovery from ketamine (136).

Emergence phenomena are relatively rare in children, although young adults are especially susceptible to this problem (69). Changes in mood and body image, out-of-body experiences, floating sensations and frank delirium are all possible (157). Emergence phenomena result from misinterpretation of auditory and visual stimuli at the neurologic level (157). Although usually terminating within 24 hours (136), prolonged emergence phenomena lasting as long as 10 to 12 months have been reported in children (102). An increased incidence of emergence reactions is seen in patients older than 16 years, female patients, patients who have received doses of intravenous ketamine above 2 mg/kg, and patients with a history of abnormal personalities (157). There is no evidence that emergence in a quiet environment decreases the incidence of this problem (157). Benzodiazepines (e.g., diazepam and midazolam) are the most effective treatment for ketamine-induced delirium and hallucinations (157). In fact, the administration of a benzodiazepine 3 to 5 minutes before ketamine is effective in almost entirely eliminating the possibility of emergence delirium (88).

Transient diplopia (31), ataxia (60), and disequilibrium (60) may occur after ketamine use. Early attempts at ambulation should be discouraged (60). Ketamine does not induce seizures and is not necessarily contraindicated in patients with an underlying seizure disorder (157).

Ketamine is contraindicated in patients with increased intracranial pressure or with abnormal intracerebral compliance. Thus, patients who have sustained a head injury as part of their ongoing trauma should not receive this drug (144). It is interesting to note that there are some reports actually suggesting that there is a neuroprotective effect for ketamine (83). However, the recommendation that ketamine be avoided in head-injured patients still stands firm for now.

### **Respiratory Effects of Ketamine**

Ketamine can have some potentially troublesome effects on the airway. It causes the production of increased salivary and tracheobronchial secretions, which can lead to coughing, laryngospasm, and airway obstruction. This problem may be especially treacherous in patients with an ongoing respiratory infection. Glycopyrrolate (Robinul), an anticholinergic, should be administered 3 to 5 minutes before ketamine (at the same time that the benzodiazepine is given) to ameliorate this problem (88). The dose for glycopyrrolate is 5 to 10  $\mu$ g/kg, given intravenously. For large children, a dose of 0.2 mg (200  $\mu$ g) of glycopyrrolate given intravenously is sufficient. Unless there is some other strong indication for its use, ketamine should be avoided in patients with ongoing infections of the respiratory tract.

Although ketamine does not usually produce significant depression of ventilation (136), apnea has been reported with its administration (37). Apnea is more likely to occur when the drug is given intravenously in rapid boluses (37) or in combination with other respiratory depressants (136). However, there are reports of apnea in otherwise healthy children sedated in the emergency department with intramuscular ketamine alone in the usual recommended dosage (96,126).

In addition, ketamine does not protect against aspiration of gastric contents (26,141). In this regard, ketamine is no different from any other sedative and analgesic except maybe for self-administered 50% nitrous oxide in oxygen. Ketamine should never be given in an unmonitored setting, such as a patient's room on a regular hospital ward, or a clinic that does not have appropriate monitoring and resuscitation equipment (see the first part of this chapter).

### **Cardiovascular Effects of Ketamine**

Ketamine stimulates the sympathetic nervous system and leads to the release of endogenous catecholamines. Through such an effect, ketamine produces a dose-dependent increase in heart rate and blood pressure (144), and therefore, it is useful in the operating room in patients with mild hypovolemia. As a byproduct of its sympathetic stimulation, ketamine produces bronchodilation, and as such, it has been useful in the anesthetic management of patients with asthma (88). However, because ketamine is a direct myocardial depressant, its administration to patients who are profoundly hypovolemic, and whose sympathetic nervous system is already maximally stimulated, will lead to cardiovascular collapse. The reader is reminded that any sedation given to a hypovolemic patient must be administered very judiciously and preferably after the volume status is corrected.

### **Review of Relevant Literature (Ketamine)**

In 1990, Green and co-workers reviewed a collective experience of nearly 12,000 children sedated with ketamine for various procedures (60,61). In 1998, Green and coworkers (62) published their experience with 1,022 pediatric patients aged 15 years and younger sedated with ketamine 4 mg/kg intramuscularly for a variety of emergency room procedures, consisting mainly of laceration repairs and fracture reductions. From this group of patients, the authors reported two cases of apnea, four cases of laryngospasm, one case of respiratory depression, and seven cases of partial airway obstruction (e.g., airway malalignment) responding to repositioning of the head. They also reported a 6.7% incidence of vomiting but no cases of aspiration. In an interesting study by Kennedy and colleagues (81), intravenous

ketamine combined with midazolam (Versed) was compared with fentanyl combined with midazolam in the management of pediatric fractures. This particular study is further discussed later.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

Salient points regarding the safe use of ketamine are summarized in [Table 3-7](#). Monitoring and procedural guidelines for deeply sedated patients ([63](#)) should be followed whenever ketamine is used. If used intramuscularly, the dose should be limited to 4 mg/kg. If the drug is used intravenously, the total dose should be limited to 2 mg/kg. The reader is reminded to use glycopyrrolate, and to consider strongly the administration of midazolam (Versed) 0.05 to 0.1 mg/kg for the prevention of agitation and delirium. Note that there is an increased risk of respiratory depression whenever more than one sedative medication is administered. Note also that no reversal drug exists for ketamine.

- I. Methods of Administration and Dosage
  - A. IM, 4 mg/kg
  - B. IV, 1–2 mg/kg
  - C. PO, 6–10 mg/kg
  - D. Rectal, 5–10 mg/kg
- II. Contraindications
  - A. Altered state of consciousness
  - B. Increased intracranial pressure
  - C. Active upper respiratory infections (increased quantity of secretions and possible increased risk of laryngospasm)
  - D. Full stomach
  - E. Prior unfavorable experience with ketamine
  - F. Patients < 16 yr old (increased incidence of emergence delirium)
- III. Advantages
  - A. Provides sedation, amnesia, intense analgesia
  - B. Sympathetic-mediated activity may be beneficial for children with asthma
- IV. Disadvantages
  - A. Increases production of saliva and tracheobronchial secretions. Coadministration of glycopyrrolate 0.01 mg/kg recommended
  - B. Potential for loss of the airway from:
    - 1. Laryngospasm secondary to increased secretions
    - 2. Aspiration from laryngeal incompetence
    - 3. Apnea
  - C. Emergence delirium: Rare in young children. No advantage to quiet environment. Midazolam may help, but may contribute to oversedation.

**TABLE 3-7. KETAMINE IN PEDIATRIC SEDATION**

### **Benzodiazepines**

Initial interest in the use of benzodiazepines developed when these drugs were noted to exert taming effects in animals ([133](#)). Benzodiazepines provide anxiolysis, hypnosis, centrally mediated ([40](#)) relaxation of muscle tone, antegrade and retrograde amnesia, and anticonvulsant activity ([113](#)). Benzodiazepines have no analgesic activity and require supplementation for painful procedures ([133](#)).

#### **Pharmacology**

Midazolam (Versed) is the primary benzodiazepine used for pediatric sedation. It offers several advantages over other benzodiazepines ([158](#)). It is water soluble and, therefore, usually relatively painless on injection ([113,158](#)). It does occasionally cause discomfort on injection, although the pain is not as severe as that with diazepam (Valium). At physiologic pH, midazolam becomes highly lipid soluble, facilitating transport into the CNS and onset of sedative effects ([158](#)). Initial recovery, which is due to redistribution of the drug away from the CNS, occurs in about 30 minutes. The elimination half-life of midazolam is significantly shorter than that of diazepam ([158](#)). On a milligram-per-milligram basis, midazolam is at least two to three times as potent as diazepam ([56](#)). Electroencephalographic (EEG) studies indicate that the blood–brain equilibration time is 4.8 minutes for midazolam versus 1.6 minutes for diazepam ([25](#)). Therefore, when titrating midazolam for sedation, it is important to wait 5 minutes between doses.

#### **Central Nervous System Effects**

Anxiolysis and centrally mediated relaxation of skeletal muscle tone is presumed to occur from a benzodiazepine-induced increase in the availability of glycine inhibitory neurotransmitter ([133](#)). Facilitating the action of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA) is responsible for the sedative effects of benzodiazepines ([133](#)). It remains unclear what the site of action is for the production of amnesia ([133](#)).

Note that midazolam and diazepam produce direct depression of the central respiratory drive ([54](#)) and that apnea may occur ([18,72,113](#)), especially after parenteral administration of these drugs. Although it is generally considered very safe, orally administered midazolam has been reported to produce airway obstruction in a child with congenital airway anomalies ([85](#)). In general, the incidence of respiratory complications increases with the presence of major vital organ disease ([72](#)). However, even in healthy adult volunteers, intravenous sedation with midazolam (0.1 mg/kg) can depress the ventilatory response to hypoxia ([3](#)). Concomitant administration of opioids ([72,133,161](#)) greatly increases the risk of respiratory complications. Therefore, extra vigilance and careful titration of medications to effect are even more important when using more than one sedative or analgesic medication.

#### **Other Systemic Effects**

With careful titration, significant hemodynamic changes are unusual with midazolam ([113](#)). Loss of protective airway reflexes is also unlikely under these circumstances ([113](#)) as long as the physician pays careful attention to the effects of each incremental dose on the patient's state of consciousness. Caution is always urged if the patient's stomach is full. Slurring of speech is a typical sign of sedation with benzodiazepines ([113](#)). Children may also exhibit loss of anxiety, unsolicited smiling, and even laughter.

In reporting their experience with 2,617 children sedated for endoscopic procedures, Massanari and co-workers ([91](#)) noted that 36 patients exhibited paradoxical reactions to midazolam, including inconsolable crying, combativeness, and agitation. The authors of this study were able to treat these reactions with flumazenil, a benzodiazepine antagonist, which is further discussed below.

#### **Review of Relevant Literature (Midazolam)**

In children, midazolam can be administered by oral, nasal, sublingual, intravenous, intramuscular, and rectal routes ([88](#)). A liquid oral formulation, whose concentration is 2 mg midazolam/mL, now exists in the United States. It is marketed as Versed Syrup 2 mg/mL and is manufactured by Hoffman-LaRoche pharmaceuticals. If this formulation is not available at a particular location, then the practitioner can order the parenteral form (usually the 5 mg/mL concentration) to be mixed in 5 to 10 mL of a sweet-tasting syrup ([106](#)). Acetaminophen syrup or ibuprofen syrup are useful vehicles for mixing parenteral midazolam, keeping in mind the appropriate pediatric doses of acetaminophen and ibuprofen ([Table 3-8](#)). The author's personal preference is to mix the midazolam in 3 to 5 mL of Tylenol syrup. For nasal administration, which the author does not recommend, the parenteral preparation is used with no additives. The reason for discouraging the use of nasal midazolam is because most children find its administration to be very unpleasant. In one study, 84% of children given intranasal midazolam cried in response to administration of the medication ([80](#)). Although sublingual administration is a good idea from a pharmacologic point of view (see discussion under [morphine](#)), it requires a degree of patient cooperation that may be difficult to reliably obtain in children. In other words, an uncooperative child may be unwilling or unable to hold a medication under his or her tongue.

- I. Method of Administration
- Diarepam: 0.1 to 0.3 mg/kg IV or PO. IM administration should be avoided because it is painful.
  - Midazolam
    - PO, 0.5–0.75 mg/kg
    - Rectal\*, 0.3–0.4 mg/kg
    - IM, 0.03–0.1 mg/kg
    - IV, 0.05–0.1 mg/kg
- II. Contraindications
- Previous unfavorable experience with benzodiazepines
  - (?) Early pregnancy (possible teratogenicity)
  - Altered state of consciousness
- III. Advantages
- Generally provide excellent sedation and amnesia
  - Reversible if necessary (flumazenil, 10 µg/kg) up to a total dose of 1.0 mg
- IV. Disadvantages
- No analgesic effect
  - Respiratory depression, especially with parenteral administration
  - Combination with narcotics may lead to oversedation or respiratory arrest
- \* Many children find the intranasal administration of midazolam to be very unpleasant. This method of administering midazolam is not recommended.

**TABLE 3-8. BENZODIAZEPINES IN PEDIATRIC SEDATION**

Hennes and colleagues (65) have used oral midazolam to allay the anxiety of children requiring laceration repair in the emergency department. Fatovich and Jacobs (47) also noted decreased anxiety in children requiring laceration repair after oral midazolam versus in a control group that received only a placebo. Note that if the practitioner decides to use orally administered midazolam, the appropriate dose is 0.5 to 0.75 mg/kg (48) and a waiting period of 10 to 30 minutes is required for onset of effects. The maximum amount of midazolam that should be administered orally has not been determined, but in practice, this amount is usually limited to 20 to 25 mg. Note that analgesic supplementation in the form of either local anesthetics, opioids, or both is required for painful procedures. Patients who receive parenteral benzodiazepines must be monitored with pulse oximetry, because oxygen desaturation may occur (125).

### Drug reversal

Flumazenil (Romazicon) reverses the sedative effects of benzodiazepines (76,82,107). The flumazenil dose for children is 10 µg/kg intravenously. Flumazenil administration may then be continued at 5 µg/kg/minute until the child awakens, or until a total dose of 1 mg has been given (77). The elimination half-life of flumazenil is 30 minutes, compared with 1 to 2 hours for midazolam. Patients who receive flumazenil should be observed for at least 2 hours before discharge to ensure that re-sedation from the original benzodiazepine does not occur. In the author's opinion, the use of flumazenil should be limited to situations of relative or absolute benzodiazepine overdose leading to respiratory or hemodynamic compromise. Routinely reversing benzodiazepines is both unnecessary and, in the absence of persistent monitoring, potentially dangerous.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

Salient points regarding the use of midazolam and other benzodiazepines are summarized in Table 3-9. Although supplemental analgesia is required for painful procedures, such as the reduction of fractures, the anxiolysis and amnesia that midazolam produces make it an excellent medication for children with orthopaedic injuries. Careful intravenous titration of midazolam in increments of 0.05 mg/kg may be undertaken, combined with a regional anesthetic block (Bier block, hematoma block, for example) for pain relief. The author believes that oral midazolam, with its mandatory 10- to 30-minute waiting period, and with its lack of titratability to effect, is probably best reserved for use as a preoperative medication before elective surgical procedures. Also, for emergency patients, intravenous titration is the best and most efficient way to achieve desirable levels of patient sedation and cooperation. The combination of midazolam and opioids is discussed in the next section.

- I. Method of Administration
- Morphine: 0.05–0.1 mg/kg IM or IV
  - Meperidine: 0.5–1.0 mg/kg IM or IV
  - Fentanyl: in increments of 0.001 mg/kg IV (maximum total dose, 0.004–0.005 mg/kg)
  - Nalbuphine: 0.1 mg/kg IM or IV
- Patients younger than 3 mo old should be given no more than half of these doses initially. IV titration to desired effect is the ideal way to administer all sedative medications.
- II. Contraindications
- Altered state of consciousness
  - Previous unfavorable experience (excludes that medication only)
  - Sedation for nonpainful procedure
- III. Advantages
- Provide excellent analgesia
  - Reversible if necessary (naloxone 0.001–0.005 mg/kg IV [titrated to effect])
- IV. Disadvantages
- Risk of respiratory depression and apnea
  - Increased risk of respiratory depression and apnea when combined with other sedatives
  - No amnesic effects
- V. Additional Side Effects (more likely when used in recurrent doses for treatment of pain)
- Nausea, vomiting, pruritus, constipation, decreased gastric motility

**TABLE 3-9. OPIOIDS IN PEDIATRIC SEDATION**

## Opioids

Opioids include all exogenous substances, natural or synthetic, that bind to specific receptors and produce morphine-like effects (138). There are several types and subtypes of opioid receptors (8,138). Opioids vary in their respective affinity for receptor types, accounting for the difference in side effects. Opioids are classified as pure receptor agonists (e.g., morphine, meperidine, fentanyl), agonist-antagonists (e.g., nalbuphine), or pure antagonists (e.g., naloxone) (138).

### Opioid Agonists

All opioid agonists produce dose-dependent respiratory depression and apnea (138). Nausea and vomiting occur because of direct stimulation of the chemoreceptor trigger zone in the floor of the fourth ventricle of the medulla oblongata (138).

#### Morphine

Morphine is a well-known analgesic. It is usually administered intravenously or intramuscularly, although sublingual and rectal routes have been described (33). Oral morphine is usually used for long-term pain control in patients with severe, chronic pain. Rectal administration of morphine is not recommended because it has been associated with delayed absorption, delayed respiratory depression, and death (33,58). In general, rectally administered medications are absorbed unpredictably (135) and access of the medication to the rectal mucosa may be variably impeded by rectal stool content.

The usual starting dose for intravenous or intramuscular morphine is 0.05 to 0.1 mg/kg. In infants younger than 3 months old, the dose should be reduced by at least one half because of increased susceptibility to respiratory depression (114). Morphine should be reserved for painful procedures lasting at least 30 minutes (33). Morphine is not very lipid soluble, and its delay in leaving the CNS accounts for a potential duration of action of 3 to 4 hours (8,33). Hypotension secondary to vasodilation, histamine release, or vagally mediated bradycardia can occur even with the administration of small doses of morphine (8). Histamine release along the course of the vein into which the morphine is administered is not by itself an allergic reaction (138). The overall incidence of true allergic reactions to opioids is very small (138).

#### Meperidine

The use of meperidine (Demerol) parallels that of morphine. The initial intravenous or intramuscular dose is 0.5 to 1.0 mg/kg. Again, the dose should be reduced by at least one half in infants younger than 3 months of age (114). Normeperidine, a metabolic breakdown product of meperidine, has been associated with seizures, agitation, tremors, and myoclonus (68,78). Meperidine is not recommended for patients with an underlying seizure disorder. Accumulation of normeperidine is more likely in situations of prolonged meperidine administration. Therefore, meperidine should be used cautiously, if at all, in the treatment of chronic pain (33). As with

morphine, meperidine may produce hypotension due to various mechanisms (8). Histamine release has also been reported with meperidine (8).

### Fentanyl

Fentanyl is a synthetic narcotic 100 times more potent than morphine and 1,000 times more potent than meperidine on a milligram-per-milligram basis. Fentanyl is highly lipid soluble and rapidly penetrates the CNS (8). When administered in low doses, its duration of action is from 30 to 45 minutes. For sedation, fentanyl is given intravenously in increments of 0.5 to 1 µg/kg. The maximum total dose is 4 to 5 µg/kg (33). As a preoperative medication, fentanyl is available in an oral raspberry flavored lollipop known as the Fentanyl Oralet (88). Currently available sizes for the Oralet are 200 µg, 300 µg, and 400 µg. As a preoperative medication, the recommended dose ranges from 10 to 20 µg/kg. Troublesome side effects of this preparation include nausea and vomiting, pruritus, and oxygen desaturation (121).

Reonset of respiratory depression up to 4 hours after fentanyl administration has been reported (131). Glottic closure (5), and muscular rigidity (6,120,128) can occur, especially, although not exclusively, with administration of higher doses. Respiratory arrest may occur, especially with the coadministration of other sedatives (161). For these reasons, fentanyl should be titrated slowly to effect.

### Opioid Agonist-Antagonists

A so-called ceiling effect or limit on the degree of respiratory depression has been demonstrated for various opioid agonist-antagonists, including nalbuphine (116) and butorphanol (149). Nalbuphine and morphine have the same analgesic potency on a milligram-per-milligram basis (114). Nalbuphine has a shorter elimination half-life (73). Opioid agonist-antagonists have no particular advantage over properly dosed opioids (38). The major problem with opioid agonist-antagonists is that their ceiling effect on respiratory depression is often accompanied by a ceiling effect for analgesia (138). Also, agonist-antagonists reduce the analgesic effectiveness of pure agonists (e.g., morphine, meperidine, fentanyl, codeine) if additional analgesia is required (38). In patients who are receiving opioids on a long-term basis, administration of opioid agonist-antagonists can precipitate acute withdrawal symptoms (38).

### Drug Reversal With Opioid Antagonists

Naloxone (Narcan), which has no agonist activity of its own, displaces opioids from opioid receptors (138). Note that rapid reversal of narcotic effects may precipitate severe hypertension, pulmonary edema, ventricular or supraventricular irritability, seizures, and cardiac arrest (7,44). Dysphoria, nausea, and vomiting may also occur. Acute narcotic withdrawal in this situation reflects sympathetic nervous system stimulation from abrupt reversal of analgesia and sudden perception of pain (8). Therefore, naloxone should be titrated to effect (relief of respiratory depression) in increments of 1 to 5 µg/kg intravenously. Naloxone has a duration of action of 30 to 45 minutes, and re sedation is possible. Close patient observation is required, and supplemental naloxone doses may be necessary. Current dosage recommendations in cases of frank opioid intoxication are significantly higher (43). Caution is always advised against precipitating acute narcotic withdrawal. The first priority in the treatment of narcotic overdose is a patent, well-maintained airway.

Similar to the situation with flumazenil for reversal of benzodiazepines, the author believes that the routine use of naloxone to reverse narcotic sedative medications is unwarranted and, for reasons noted earlier, potentially dangerous. Naloxone use should be reserved for situations of airway compromise brought on by relative opioid overdose, and it should never be utilized as a way of expediting patient discharge after a procedure requiring sedation.

### Review of Relevant Literature (Opioids)

With careful monitoring and judicious administration, combinations of opioids for analgesia and benzodiazepines for amnesia and anxiolysis are probably the most useful sedatives in existence today for the management of children's fractures. Varela and co-workers (150) reported excellent patient and physician satisfaction using intravenous meperidine and midazolam for intravenous sedation for the closed reduction of fractures in children. In this particular investigation, the average meperidine dose was 1.47 mg/kg and the average midazolam dose was 0.11 mg/kg. There were no episodes of apnea or cardiorespiratory complications. These investigators stress that careful patient monitoring is important, not only during but after the procedure as well. Intravenous morphine 0.1 mg/kg plus midazolam 0.1 mg/kg is another useful sedation mixture for children with fractures, especially when combined with a hematoma block (personal communication, J. Edeen, M.D.). The hematoma block is discussed later in the chapter. With this particular approach, the midazolam is administered first, followed by the morphine about 5 minutes later. The hematoma block is performed, and the fracture is then reduced. Again, careful patient monitoring as previously outlined is essential to good care.

In the absence of specific contraindications, including tenuous airway status, unstable hemodynamic status, or history of specific allergic reactions, for the performance of painful procedures in children, a combination of a benzodiazepine and a narcotic is probably ideal (110,127,161), as long as the principles of careful titration and close patient monitoring are observed.

Salient points regarding the use of opioids as well as opioid and benzodiazepine combinations for pediatric sedation are summarized in Table 3-10 and Table 3-11. Opioid and benzodiazepine combinations provide amnesia, analgesia, and sedation. The tradeoff is that of additive respiratory depression and additive depression of protective airway reflexes. In both elective and emergent situations, the practitioner must

I. Method of Administration  
IV titration to effect  
A. Midazolam: In increments of 0.05 mg/kg to a maximum of 0.1 mg/kg. Wait 5 min between doses.  
B. Fentanyl: Begin 5 min after last midazolam dose. Give in increments of 0.001 mg/kg to a maximum of 0.003 mg/kg. Wait 2 to 3 min between doses.

II. Contraindications  
A. Altered state of consciousness  
B. Previous unfavorable experience with either medication  
C. Specific contraindications to benzodiazepines or opioids (see Tables 3-9 and 3-10).

III. Advantages  
A. Provides sedation, amnesia (midazolam), and analgesia (fentanyl)  
B. Reversible if necessary (see Tables 3-9 and 3-10).

IV. Disadvantages  
A. Additive respiratory depressant effects  
B. Additive depressant effects on protective airway reflexes with increased risk for regurgitation and aspiration of gastric contents

\* An excellent review of the advantages and problems associated with this drug regimen is provided by Vaster and colleagues<sup>11</sup>.

TABLE 3-10. FENTANYL AND MIDAZOLAM IN PEDIATRIC SEDATION\*

I. Method of Administration  
20 to 75 mg/kg orally or rectally (maximum single dose, 1.0 g; if a second dose is given, the maximum total dose is either 100 mg/kg or 2.0 g, whichever is lower).

II. Contraindications  
A. Compromised hepatic function  
B. History of obstructive sleep apnea\*  
C. Previous unfavorable experience with chloral hydrate

III. Advantages  
No specific advantages for sedation and treatment of children with fractures

IV. Disadvantages  
A. Prolonged time to peak effect (as long as 60 min)  
B. Difficult to titrate  
C. Prolonged observation period required

\* Caution is required when using any sedative medication in patients with obstructive sleep apnea.

TABLE 3-11. CHLORAL HYDRATE IN PEDIATRIC SEDATION

1. Thoroughly evaluate the patient, as has been discussed earlier in the chapter.
2. Follow standard practice guidelines for deep sedation (63).

3. Pay careful attention to dosing limits ([Table 3-11](#)).
4. Be certain that both flumazenil and naloxone are available. These medications are to be used strictly for the treatment of absolute or relative overdose of benzodiazepines and opioids, respectively. Do not use these medications to expedite discharge from the emergency room.

### **Midazolam and Ketamine Compared With Midazolam and Fentanyl**

While studying a group of 260 children between the ages of 5 and 15 presenting for the emergency room reduction of fractures, Kennedy and coworkers ([81](#)) compared the effectiveness of two regimens for sedation and analgesia. For the first reduction attempt in each patient, all of the children initially received midazolam 0.1 mg/kg (maximum dose of 2.5 mg) at 3-minute intervals until either objective signs of onset of drug effect developed (slurred speech, glassy eyes) or until three doses of midazolam were given. One minute after the administration of the last dose of midazolam, half the patients began receiving fentanyl, titrated in increments of 0.5  $\mu$ g/kg until either a decreased response to verbal or painful stimulation was noted, or a maximum dose of 2  $\mu$ g/kg had been administered. The maximum amount of fentanyl used was 100  $\mu$ g. The other half of the patients, instead of receiving fentanyl, were given ketamine, titrated in increments of 0.5  $\mu$ g/kg, using the same criteria as for fentanyl to determine onset of effect. Ketamine titration continued, if necessary, until a maximum amount of 2  $\mu$ g/kg had been given. All of the patients who were given ketamine also received glycopyrrolate, 5  $\mu$ g/kg as an antisialagogue. Additional amounts of midazolam and the study drug were given if analgesia and sedation were judged to be insufficient or if additional reduction attempts were necessary. Treatment failures occurred in those patients who experienced significant adverse drug effects or who obtained insufficient sedation and analgesia.

Patient distress was quantified using various behavioral scales. The reader is referred to the article itself for more detailed information about the measurement process ([81](#)). Whereas pre-sedation scores were basically the same for both groups, during fracture reduction, the investigators reported a significantly lower score of patient distress for the children receiving midazolam and ketamine than for those receiving midazolam and fentanyl ([81](#)). In general, the ketamine and midazolam combination was favored by parents and by the involved orthopaedic surgeons ([81](#)). On the other hand, however, two patients receiving midazolam and ketamine required brief assisted ventilation with a bag, valve, and mask apparatus, and one patient developed laryngospasm. Vomiting occurred more often in the group taking ketamine, and group taking the ketamine had a longer mean time to recovery and discharge.

This is a very detailed study that looks very closely at two viable regimens for sedation of children with fractures. Again, with careful monitoring and careful attention to drug titration, the practitioner may wish to try both to determine which is more useful in their individual practice. The authors of this study favor the ketamine and midazolam regimen and provide good evidence for their conclusions ([81](#)).

### **Pediatric Cocktail**

The so-called pediatric cocktail (DPT) or "lytic" cocktail is a mixture of meperidine (Demerol) and two phenothiazines: promethazine (Phenergan), and chlorpromazine (Thorazine). For multiple reasons, this sedative regimen should be avoided. Prolonged and profound sedation occur, often far outlasting the procedure for which the sedation was intended. One study has reported a mean total recovery time of 19 hours, plus or minus 15 hours, in children receiving DPT in the emergency department ([142](#)). Orthostatic hypotension is possible, because promethazine and chlorpromazine are alpha-adrenergic blockers ([33](#)). Severe respiratory depression and death, both during and after the procedure, have occurred in patients sedated with DPT. All three medications in this mixture lower the seizure threshold, and phenothiazines can produce dystonic reactions ([33](#)). Note that there is no reversal agent for phenothiazine overdose.

## **AUTHOR'S PREFERRED METHOD OF TREATMENT**

The author strongly believes that the use of the pediatric cocktail should be abandoned ([127](#)).

### **Chloral Hydrate**

Of greatest use in sedating children for nonpainful diagnostic procedures such as radiographic studies, chloral hydrate is administered in a dose of 20 to 75 mg/kg orally or rectally. The maximum single dose is 1 g. If more than one dose has to be given, the upper limit for the total dose is either 100 mg/kg or 2 g, whichever is lower.

Although, theoretically, rectal dosing should be more efficient, because of the lack of a first pass hepatic effect ([135](#)), 30 to 60 minutes may elapse before chloral hydrate takes effect no matter how it is administered. Children receiving chloral hydrate must be observed for at least several hours. Respiratory depression is unusual, but children with sleep apnea and adenotonsillar hypertrophy may be particularly vulnerable to airway obstruction after sedation with chloral hydrate ([16](#)). At least one death has been reported following its use ([74](#)). These problems emphasize that even sedatives thought to have little risk of producing respiratory depression must be administered under properly supervised conditions ([63](#)) and with strict adherence to dosage guidelines.

## **AUTHOR'S PREFERRED METHOD OF TREATMENT**

Chloral hydrate is of minimal use in the sedation and treatment of patients with fractures. It provides no analgesia, and it lacks the rapidity of onset and titratability of intravenous opioids and benzodiazepines. The practitioner should be familiar with this medication, however, because it remains in common use for nonpainful pediatric procedures. Salient features regarding its administration are summarized in [Table 3-12](#).

1. Numbness of the lips and tongue, metallic taste in the mouth.
2. Lightheadedness
3. Visual and auditory disturbances (double vision and tinnitus)
4. Shivering, muscle twitching, tremors (initial tremors may involve the muscles of the face and distal parts of the extremities)
5. Unconsciousness
6. Convulsions
7. Coma
8. Respiratory arrest
9. Cardiovascular depression and collapse

\* With gradual increases in plasma concentration, these signs and symptoms may occur in order as listed. With the sudden development of high plasma concentrations of a local anesthetic agent, the first manifestation of toxicity may be a convulsion, respiratory arrest, or cardiovascular collapse. In young children, or in children who are heavily sedated, subjective evidence of impending local anesthetic toxicity (manifestations 1, 2, 3) may be difficult to elicit.

**TABLE 3-12. MANIFESTATIONS OF LOCAL ANESTHETIC TOXICITY\***

### **Barbiturates**

In general, barbiturates have a lower margin of safety than benzodiazepines ([132](#)). In addition, barbiturates seem to lower the pain threshold, and are therefore a poor choice for producing sedation in the presence of a painful condition, such as a fracture ([132](#)). With these points in mind, barbiturates should not be used for sedating children with fractures.

### **Propofol**

Propofol is a substituted isopropylphenol that is a rapid-acting intravenous anesthetic ([136](#)). Because it is virtually insoluble in aqueous solutions, it has to be dissolved in lecithin-containing formulations. The orthopaedist may have seen this whitish medication administered by the anesthesiologist in the operating room, where it has gained the popular name of "milk of amnesia."

Propofol has a fast onset of action, owing to its high lipid solubility, and an extremely short duration of action. Awakening is rapid, with little to no "hangover" effect as

seen with other drugs (136). It also has antiemetic effects (88).

However, there are several reasons for which the drug should not be used in the management of children with fractures in an emergency room setting. First and foremost, it is easy to suddenly lose the airway in a patient given propofol. Therefore, this drug has really should be administered by an anesthesiologist (79). Second, the drug provides no analgesic effect and, therefore, has to be combined with an opioid, which, in turn, will intensify the respiratory depressant effects of propofol. Third, in children, the administration of propofol is associated with opisthotonic posturing and myoclonus (88), which is certainly not helpful in the reduction of a fracture. Propofol has vasodilatory and negative inotropic effects, which can lead to hypotension (88). Finally, there is some concern that propofol may be associated with seizures (88), although Momota and co-workers (97) have used propofol to stop seizure activity from local anesthetic overdose.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

Regarding children with fractures, propofol should be reserved for administration in the operating room as part of a regimen of general anesthesia by an anesthesiologist.

## REGIONAL ANESTHESIA IN THE CHILD WITH A MUSCULOSKELETAL INJURY

Within the limitations and guidelines that are discussed later, the use of regional anesthesia to relieve pain in children with musculoskeletal injuries is reasonable and worthwhile.

### Regional Anesthetic Agents

Regional or local anesthetic medications prevent nerve impulse propagation by interfering with the function of the sodium channel on the axonal membrane (139). Commonly used local anesthetics have either an amino amide or amino ester linkage in their molecular structure (146). Amino amide local anesthetics include lidocaine (Xylocaine), bupivacaine (Marcaine, Sensorcaine), mepivacaine, prilocaine, etidocaine, and the relatively new agent ropivacaine. Amino ester local anesthetics include procaine (Novocain), chlorprocaine, tetracaine, benzocaine, and cocaine.

Medications within each group have important intrinsic differences in potency, duration of action, and potential for toxicity (36,146). For example, lidocaine is significantly less toxic a drug than bupivacaine but it also has a shorter duration of action. An important feature of ropivacaine is that even though its duration of action is similar to bupivacaine, it produces less CNS toxicity and less cardiac toxicity (123). Duration of action for the various local anesthetic medications is also determined in part by the type of regional block performed. For example, single dose brachial plexus blocks tend to have a far longer duration than do single dose epidural or subarachnoid blocks (36).

### Local Anesthetic Toxicity

At least three types of adverse reactions can occur from local anesthetic agents. Clinically, the most important is systemic toxicity of the CNS and cardiovascular system from relative overdose into the circulation. This type of reaction is not a medication allergy but simply a function of placing too much medication into the bloodstream. Note that in the presence of a major artery, even a few drops of local anesthetic can lead to seizure activity. An example of this problem is seen in the performance of stellate ganglion blocks in the neck for the management of reflex sympathetic dystrophy of the upper extremity, where the vertebral artery can be accidentally entered during administration of the medication.

In most cases, however, the severity of systemic toxicity is directly related to the concentration of local anesthetic in the bloodstream (Table 3-13) (36). Seizures and cardiac arrest may be the initial manifestations of systemic toxicity in patients who rapidly attain a high serum level of medication (42,98,109). Agents with greater intrinsic potency, such as bupivacaine and etidocaine, require lower levels for production of symptoms (36). Dysrhythmias and cardiovascular toxicity may be especially severe with bupivacaine, and resuscitation of these patients may be prolonged and difficult (2,36).

**Preventive Measures**

1. Ensure availability of oxygen administration equipment, airway equipment, suction equipment, and medications for treatment of seizures (diazepam or midazolam, thiopental, succinylcholine).
2. Ensure constant verbal contact with patient (for symptoms of toxicity) and monitor cardiovascular signs and oxygen saturation.
3. Personally prepare the dose of local anesthetic and ensure it is within the accepted dosage range.
4. Give the anesthetic slowly, and fractionate the dose.

**Treatment**

1. Establish a clear airway; suction if required.
2. Give oxygen by face mask. Begin artificial ventilation if necessary.
3. Give diazepam 0.1-0.3 mg/kg IV in incremental doses until convulsions cease. Midazolam (0.05-0.1 mg/kg) may be used instead, also in increments until convulsions cease.
4. Thiopental in increments of 1-2 mg/kg IV may be used to control the seizures.
5. Succinylcholine (1 mg/kg IV) may be used if there is inadequate control of ventilation with the other medications. Artificial ventilation and possibly endotracheal intubation are required after using succinylcholine.
6. Use advanced cardiac life-support measures as necessary to support the cardiovascular system (treat liberally with local anesthetics of increased potency, such as bupivacaine).

TABLE 3-13. PREVENTION AND TREATMENT OF ACUTE LOCAL ANESTHETIC SYSTEMIC TOXICITY

The prevention and treatment of acute local anesthetic systemic toxicity are outlined in Table 3-14.

Agent	Injection Dose (mg/kg)	
	Plain	With Epinephrine*
Lidocaine† (Xylocaine)	5	7
Bupivacaine‡ (Marcaine, Sensorcaine)	2.5	3
Mepivacaine (Carbocaine)	4	7
Prilocaine§	5.5	8.5

\* The addition of epinephrine (vasoconstrictor) reduces the rate of local anesthetic absorption into the bloodstream, permitting use of a higher dose.  
 † For IV regional anesthesia ( Bier blocks), the maximum lidocaine dose is 3 mg/kg. Preservative-free lidocaine without epinephrine should be used for either Bier blocks or hematoma blocks.  
 ‡ Owing to its cardiotoxicity, bupivacaine should never be used for IV regional anesthesia or for hematoma blocks.  
 § Of the amide local anesthetics, prilocaine is the least likely to produce CNS and cardiovascular toxicity. However, a byproduct of prilocaine metabolism may lead to severe methemoglobinemia in young children. Prilocaine is, therefore, contraindicated in children younger than 6 mo old.

TABLE 3-14. MAXIMUM RECOMMENDED DOSES OF COMMONLY USED LOCAL ANESTHETICS IN CHILDREN

Although the potential for CNS toxicity may be diminished with barbiturates or benzodiazepines, given either as premedications or during treatment of convulsions, these measures do not alter the cardiotoxic threshold of local anesthetic agents. With rapid and appropriate treatment, the fatality rate from local anesthetic convulsions should be greatly decreased (36).

It is absolutely essential to stay within accepted dose limits when using any local anesthetic (Table 3-15). To aid in dose calculations, a simple formula for converting percent concentration to milligrams to milliliter is provided in Table 3-16.

Percentage concentration  $\times 10 =$  Number of mg/mL

Examples: 0.25% bupivacaine has 2.5 mg bupivacaine/mL;

2% lidocaine has 20 mg lidocaine/mL.

**TABLE 3-15. CONVERSION FORMULA FROM % CONCENTRATION TO MILLIGRAMS/MILLILITER**

IM†: Morphine, 0.1–0.15 mg/kg q 3–4 h  
Meperidine, 1.0–1.5 mg/kg q 3–4 h  
IV: Morphine, 0.05–0.1 mg/kg q 2 h  
Meperidine, 0.5–1.0 mg/kg q 2 h

\* Infants < 3 mo old should be dosed in increments of one third to one half because of increased risk of respiratory depression. † Intramuscular dosing should rarely be used.

Adapted from Roger, L. and Moro, M. Acute postoperative and chronic pain in children. In Rasch, D.K. and Webster D.E. eds.: *Clinical manual of pediatric anesthesia*. New York: McGraw-Hill, 1994:297, with permission.

**TABLE 3-16. PARENTAL OPIOID DOSING SCHEDULE FOR ANALGESIA IN CHILDREN\***

Although rare, true immune-mediated allergic reactions to local anesthetics are possible, more commonly with amino esters than with amino amides ( [22,52](#)). The reader is referred to other sources for the detailed management of allergic or anaphylactic reactions ( [28,100](#)).

Local nerve damage and reversible skeletal muscle changes have been reported from the use of local anesthetics ( [36](#)).

### Intravenous Regional Anesthesia

Intravenous regional anesthesia was first described by August Bier in 1908 ( [15](#)). Although it declined in popularity as brachial plexus blocks were developed, it was revived in 1963, when its safe and successful use for the reduction of forearm fractures in adults was reported ( [70](#)). Since then, multiple reports have attested to its usefulness in the treatment of upper limb injuries in adults and children ( [10,17,29,46,53,104,147](#)).

The mechanism of action is uncertain. It may involve both direct transport of local anesthetic to major nerve trunks or the blockade of small nerve endings ( [53](#)).

Intravenous regional anesthesia is reliable, with a higher success rate than other approaches to upper extremity blocks ( [53](#)). Other advantages include its simplicity, rapidity of onset, and rapidity of recovery. Rapidity of recovery may be considered both an advantage as well as a disadvantage, because, with local anesthetic alone, the analgesic effect is lost once the tourniquet is deflated. A recent report in adults examined the addition of the nonsteroidal antiinflammatory medication ketorolac (Toradol) to the local anesthetic solution for intravenous regional anesthesia and found that patients did obtain prolonged analgesia after the tourniquet was released ( [124](#)). However, no pediatric studies have been performed on this drug combination.

Intravenous regional anesthesia is unsuitable for lesions above the elbow ( [71](#)). In addition, if the fracture involves the supracondylar area of the humerus, the cuff may limit the degree of hyperflexion needed to produce an adequate reduction. Intravenous regional anesthesia is contraindicated in patients with underlying heart block, known hypersensitivity to local anesthetic agents, and seizure disorders ( [71](#)). Although not totally contraindicated, caution is urged when using this technique in patients with underlying hemoglobinopathies such as sickle cell disease.

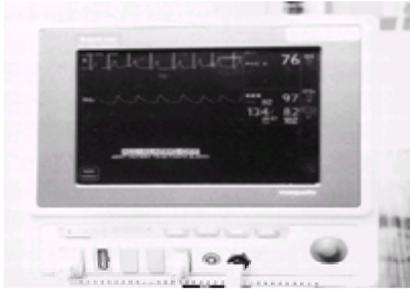
## AUTHOR'S PREFERRED METHOD OF TREATMENT

The basic steps involved in performing an intravenous regional block are as follows:

1. Confirm the immediate availability of a functioning positive-pressure oxygen delivery system, as well as appropriate airway management equipment ( [Table 3-5](#)) ( [63](#)). Also, confirm the immediate availability of medications for the treatment of anesthetic-induced convulsions ( [Table 3-13](#)).
2. Start an intravenous infusion in the contralateral arm ( [Fig. 3-3](#)). A patent intravenous line is of paramount importance in treating the complications of this block. Obtain a baseline set of vital signs, including systolic and diastolic blood pressure. Monitor pulse oximetry ( [Fig. 3-3](#)) as well as the ECG continuously ( [Fig. 3-4](#)).

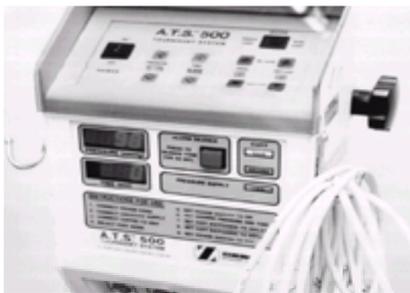


**FIGURE 3-3.** Intravenous infusion for fluids, sedative medications, and if necessary, resuscitative medications has been started in the contralateral hand. A pulse oximeter probe is present on the patient's index finger.



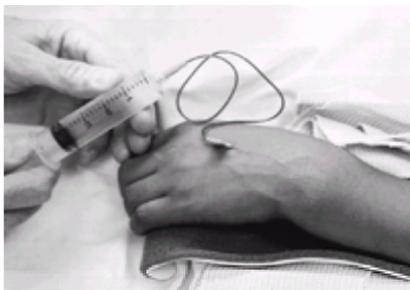
**FIGURE 3-4.** Continuous display of the electrocardiogram (top waveform) and continuous display of the plethysmographic tracing from the pulse oximeter (second line). Intermittent blood pressure reading is displayed.

3. Select an appropriate tourniquet. An orthopaedic tourniquet that can be fastened securely should be used. Because Velcro may become less adhesive with time, check the tenacity of the tourniquet before use. As an added safety measure, the tourniquet may be covered with strong adhesive tape or an Ace bandage after application. The tourniquet should fully encircle the arm and overlap back on itself by at least 6 cm (personal communication, K.E. Wilkins, M.D.). The arm may be minimally padded with cast padding underneath the tourniquet (17). If a pneumatic tourniquet is used, the physician must be familiar with the location of the tourniquet pressure gauge (Fig. 3-5) (29) and valves, because these features may vary in location from model to model. Narrow-cuffed double tourniquets may not effectively occlude arterial flow, and their use has been discouraged (71). Tourniquet discomfort should not be a problem during short procedures. If this problem develops, a second tourniquet can be applied distally over the anesthetized area of the arm.



**FIGURE 3-5.** Tourniquet controls may vary in position from model to model. It is imperative for the practitioner to be aware of the function of each of the controls to avoid accidental premature deflation of the tourniquet.

4. Palpate the radial pulse of the injured limb.
5. Place and secure a short 22-gauge cannula in a vein on the dorsum of the hand of the fractured limb. A 23-gauge butterfly needle may also be used (Fig. 3-6). Note that although it may be possible to “thread” a butterfly needle securely into a large vein, butterfly needles may still slip out of the vein more easily than a plastic intravenous catheter. The author recommends the use of intravenous catheters whenever possible. If a distal vein is unavailable, a proximal vein or even an antecubital vein can be used. However, the use of proximal veins in the arm may result in a less effective block (71).



**FIGURE 3-6.** Venipuncture for administration of local anesthetic has been performed with a butterfly needle. An intravenous catheter may be more secure than a needle, which can become dislodged from within the vein.

6. With the tourniquet deflated, exsanguinate the limb by vertically elevating it above the level of the heart for 60 seconds.
7. Rapidly inflate the tourniquet to a pressure of 225 to 250 mm Hg or 150 mm Hg above the patient's systolic blood pressure (51). Check for disappearance of the radial pulse. Cross-clamping the tubing of the cuff after inflation is discouraged (71), because it might prevent detection of a small leak. Constant observation of the cuff pressure gauge is recommended.
8. Lower the extremity and slowly inject the local anesthetic. It is recommended that this injection be done over a period of 60 seconds. A concentration of 0.5% plain lidocaine (5 mg/mL) is used (Fig. 3-7). Bupivacaine is contraindicated for this block because of its cardiotoxicity. To prevent thrombophlebitis, the local anesthetic solution must be free of any additives or preservatives (71). In different studies, the recommended dose of lidocaine has varied from 1.5 to 3.0 mg/kg (10,17,29,46,53,104,147). A dose of 1.5 mg/kg appears to be safe and effective, and may produce a decreased incidence of complications (17). One study has recommended a maximum lidocaine dose of 100 mg for this block (46). The skin of the extremity becomes mottled as the drug is injected. The patient, unless he or she is very sedated, and the parents, if they are watching, should be warned that the extremity will look and feel strangely (personal communication, K.E. Wilkins, M.D.). Analgesia and muscle relaxation develop within 5 minutes of injection (71). Note that for fractures at the wrist, placement of a regular penrose



**FIGURE 3-7.** Preservative-free 0.5% lidocaine without epinephrine for intravenous regional anesthesia.

drain tourniquet around the distal forearm may improve distribution of the local anesthetic solution at the fracture site (Fig. 3-8) (personal communication, K.E. Wilkins, M.D.).



**FIGURE 3-8.** Penrose drain tourniquet on the forearm to improve distribution of local anesthetic at the fracture site.

9. To improve analgesia for fracture reduction, the last 2 mL of local anesthetic solution may be injected directly into the fracture hematoma ( [Fig. 3-9](#)) (personal communication, K.E. Wilkins, M.D.). The technique of local infiltration anesthesia, or hematoma block, is discussed further in a subsequent section of this chapter.



**FIGURE 3-9.** Hematoma block performed with the last 2 mL of the intravenous lidocaine solution to enhance analgesia at the fracture site.

10. Perform the surgical procedure ( [Fig. 3-10](#)).



**FIGURE 3-10.** Fracture reduction under appropriately monitored sedation and intravenous regional anesthesia.

11. Leave the cuff inflated for at least 15 minutes ( [71](#)), even if the surgical procedure takes less time.
12. Release the cuff at the end of the operation, or at the end of a 15-minute interval, whichever is longer. Cuff deflation, followed by immediate reinflation, as a method of delaying systemic absorption of local anesthetic is impractical because it cannot be done rapidly enough to prevent significant entry of local anesthetic into the general circulation ( [71](#)).
13. Monitor the patient closely for at least 15 minutes for any complications related to the block. The treatment of local anesthetic-induced systemic toxicity has been discussed ( [Table 3-13](#)).
14. Of course, depending on whatever sedation has been administered, the patient should be monitored until discharge criteria are met ( [Table 3-1](#)).

An assistant must be present to watch the patient, the tourniquet, and the monitors at all times.

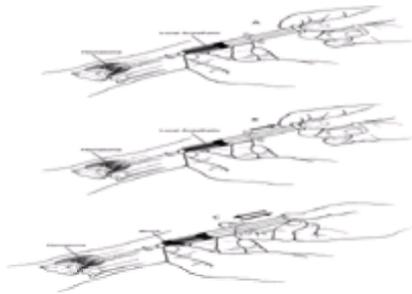
#### **Local Infiltration Anesthesia: Hematoma Block**

Hematoma block has been successfully used in the treatment of distal forearm fractures ( [27,94](#)). This simple technique involves injecting local anesthetic into the fracture hematoma, the location of which is confirmed by aspirating blood into the syringe ( [Fig. 3-9](#)). When done alone, 1% lidocaine in a dose of 1 to 2 mg/kg is used ( [94](#)). As indicated earlier, when combined with a Bier block, the last 1 to 2 mL of local anesthetic solution are used. Although the medication is rapidly absorbed into the circulation, the resulting systemic blood levels of local anesthetic are well below those required for CNS toxicity ( [94](#)). Hematoma blocks may be used in patients who have received a regional block yet still have residual pain at the fracture site.

Alioto and co-workers ( [4](#)) have described the use of an intraarticular hematoma block for the manipulative reduction of ankle fractures in a population group that included both children and adults. The youngest patient in their study group who received a hematoma block was 12 years old. The technique they describe consisted of a direct injection of 2% lidocaine 1 mg/kg (the authors used 2 mg lidocaine per pound) into the tibiotalar joint space. Absolute sterile technique was used, with the skin overlying the fracture site being prepared with a povidone-iodine preparation. A 22- or 25-gauge needle was used for the injection of the local anesthetic solution. The authors of this investigation recommend directing the needle in a slightly cephalad direction to avoid injury to the talar and tibial articular surfaces. Aspiration of blood was used to confirm entry into the intraarticular space. The injection was performed only if no resistance was encountered. A minimum of 15 minutes was allowed to pass after the injection, during which time the patient was closely observed and monitored for any evidence of local anesthetic systemic toxicity (see earlier). The injection was considered adequate if the patient was subsequently able to elevate the injured limb off the bed without assistance and perform active ankle dorsiflexion and plantarflexion. The authors of this investigation found the intraarticular ankle hematoma block to be safe, effective, and well tolerated by patients ( [4](#)). Although some of their patients tolerated the injection and the subsequent fracture reduction without the use of any other supplemental analgesics or sedatives, most children would probably require the same type of sedation that would be used before performing a Bier block or a hematoma block of the upper extremity.

#### **AUTHOR'S PREFERRED METHOD OF TREATMENT**

Full aseptic technique, including adequate skin preparation and the use of sterile gloves, is recommended. The incidence of infection from introduction of a needle into the hematoma is greatly decreased if antiseptic precautions are taken ( [75](#)). The hematoma is localized by aspirating blood into the syringe. The local anesthetic solution is given gradually by alternate injection (barbotage) of a small amount of medication and withdrawal of a small amount of hematoma until all of the medication has been given ( [Fig. 3-11](#)). In the case of the intraarticular hematoma block at the ankle ( [4](#)), a slow direct injection is used, with careful ECG monitoring for any evidence of dysrhythmias.

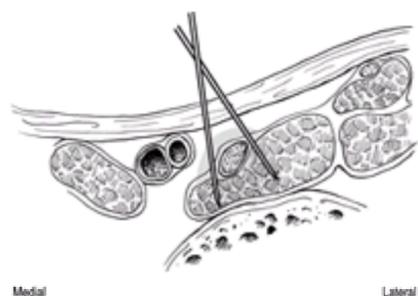


**FIGURE 3-11.** Barbotage. When performing a hematoma block, the local anesthetic is given by barbotage. A: Half the anesthetic is injected in the hematoma. B: The blood from the hematoma is withdrawn until the original volume is regained. C: This mixed material is repeatedly injected and reaspirated until the anesthetic is dispersed in the hematoma. The final aspirate should contain a volume equal to the original anesthetic. Thus, the final volume of fluid in the hematoma has not been increased.

Reported complications with hematoma blocks in the upper extremity include compartment syndrome (162), temporary paralysis of the anterior interosseous nerve (162), and increased incidence of carpal tunnel syndrome (84).

### Femoral Nerve Block

Berry (14) has described a technique of rapid analgesia for patients with a fracture of the femoral shaft using 1% lidocaine with 200,000 epinephrine or 1.5% lidocaine with 1:200,000 epinephrine deposited at the femoral nerve, just below the inguinal ligament ( Fig. 3-12). As always, the anesthetic dose should remain within accepted limits. For lidocaine with epinephrine, 7 mg/kg is the permissible dose limit.



**FIGURE 3-12.** Section of right thigh immediately below the inguinal ligament, showing femoral nerve under cover of fascia iliaca and its block by a barrage technique. (Reproduced with permission from Anesthesia, 1977, Volume 32, page 577. Analgesia in Patients with Fractured Shaft of Femur. F.R. Berry, M.D.)

Cooperation and tolerance among young children for regional blocks varies, so careful patient selection is advised.

## POSTOPERATIVE ANALGESIA IN THE CHILD WITH A MUSCULOSKELETAL INJURY

Safe and effective postoperative analgesia in children with musculoskeletal injuries can be accomplished with opioids, nonsteroidal antiinflammatory agents (NSAIDs), or local anesthetic agents.

Simultaneous use of more than one modality may be beneficial in order to minimize side effects from any one particular approach (e.g., the use of NSAIDs to decrease the incidence of nausea, vomiting, or even respiratory depression from opioids). The end point is to make patients comfortable while minimizing adverse reactions.

### Postoperative Analgesia With Opioids

Opioids have long been the mainstay of postoperative analgesia. It is important for the practitioner to understand the rationale behind different dosage regimens to maximize pain relief for the patient.

#### Intermittent Dosing

Although commonly used, traditional intermittent as-needed dosing of intramuscular and intravenous opioids ( Table 3-17) makes little pharmacologic sense for control of severe pain (49,148). Wide variations in plasma opioid levels occur, leading to periods of sedation alternating with prolonged periods of no pain relief at all ( 49). In addition, for pediatric care, intramuscular dosing is a particularly poor choice ( 148), because children often chose to hide their pain rather than risk having to undergo an injection. The end result with intermittent dosing, especially with intramuscular narcotics, is unnecessary undertreatment of pain.

<p>Loading dose: Morphine, 0.025–0.05 mg/kg          Maintenance dose: Morphine, 0.01–0.02 mg/kg          Lockout interval: 6–10 min          4-hr maximum: Morphine, 0.8 mg/kg/4 h</p> <p><b>Treatment of Side Effects</b></p> <p><b>Pruritus</b>          Diphenhydramine (0.5 mg/kg IV) Q8          Low-dose naloxone (0.5–1.0 µg/kg)</p> <p><b>Nausea/vomiting</b>          Metoclopramide (0.1 mg/kg IV) Q8          Droperidol (10–20 µg/kg IV or IM) Q8          Ondansetron (0.15 mg/kg IV over 15 min)          Low-dose naloxone as for pruritus</p> <p><b>Urinary retention (1 mg/kg in the face of adequate fluid intake)</b>          Low-dose naloxone infusion as above</p> <p><b>Respiratory depression</b>          Vital signs, specify parameters that require treatment and method for contacting responsible physician.          Stop PCA pump.          Give 100% oxygen and maintain the airway.          Give naloxone (1–5 µg/kg IV bolus). Repeat as needed.          Consider naloxone infusion (2–5 µg/kg/h).</p> <p><small>Rogers J, Adron M. Acute postoperative and chronic pain in children. In: Routh CR, and Webster CR, eds. Clinical manual of pediatric anesthesia. New York: Elsevier, 1994:268, with permission.</small></p>
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**TABLE 3-17. PATIENT-CONTROLLED ANALGESIA IN CHILDREN**

### Patient-Controlled Analgesia

Patient-controlled analgesia (PCA) is a sensible approach to the problems inherent with intermittent as-needed dosing of opioids ( 49). With PCA, intravenous

self-titration of small doses of opioids at frequent intervals eliminates the wide variations in plasma drug levels seen with intermittent dosing ( 49). It also allows patients to gain control over their pain management ( 23), which may be of psychological importance to the patient's well-being.

PCA was first evaluated in adolescents in 1987, after several years of successful use in adults ( 23). Since then, this modality has been used for children as young as 6 years of age (11). Depending on the intelligence and cooperative ability of the child, it is conceivable that PCA could be used for younger individuals, although careful assessment of each individual situation is required.

When compared with traditional intermittent dosing, improved pain control and greater patient satisfaction have been demonstrated ( 11). Note that further improvement in pain relief may be achieved with the addition of a continuous background infusion of opioids to maintain the plasma concentrations of the analgesic during sleep. However, adding a background infusion may increase the risk of opioid-associated nausea, sedation, and hypoxemia ( 39,159).

Conceivably, for younger children or for children otherwise unreliably capable of pushing the button on the PCA cord, "parent-controlled analgesia" may be useful. The author has used this approach in a patient as young as 1 1/2 years of age. In this particular situation, however, the parents were very motivated and intelligent, and had done this before for their child after another surgical procedure. In general, PCA is safest when only the patient is operating the device.

Parameters that must be considered are the loading dose, the maintenance dose, and the lockout interval (the period during which no further administration of medication will occur despite attempts to do so by the patient), and the 4-hour maximum dose ( Table 3-18). For PCA, morphine is more effective than meperidine (151). Opioids other than morphine should be used only for patients allergic to morphine ( 20), or for whom morphine produces intolerable side effects. Whenever possible, the persistent use of one medication helps avoid dosing errors ( 20).

Agent	Dose	Formulations*
Codeine	0.5-1 mg/kg q 4-6 h (oral) single dose 0.5 mg	50 mg/mL suspension
Codeine with acetaminophen (Tylenol with Codeine)	0.5-1 mg/kg q 4-6 h (oral) single dose 0.5 mg/kg acetaminophen q 4-6 h	10 mg/mL suspension + 100 mg/mL acetaminophen†
Codeine with salicylate (Tylenol with Codeine)	0.5-1 mg/kg q 4-6 h (oral) single dose 0.5 mg/kg acetaminophen q 4-6 h	10 mg/mL suspension + 100 mg/mL salicylate†
Hydrocodone with acetaminophen (Vicodin, Norco, Zexin, Co-Done, Duricef, Norflex, Vicodin)	0.5-1 mg/kg q 4-6 h (oral) single dose 0.5 mg/kg acetaminophen q 4-6 h	5 mg/mL suspension + 100 mg/mL acetaminophen†
Hydrocodone (Hycodan)	0.5-1 mg/kg q 4-6 h (oral) single dose 0.5 mg/kg acetaminophen q 4-6 h	5 mg/mL suspension
Meperidine (Demolor, Peri, Demolor-ER)	0.5-1 mg/kg q 4-6 h (oral) single dose 0.5 mg/kg acetaminophen q 4-6 h	50 mg/mL suspension
Morphine (Morphine sulfate, Duramorph)	0.5-1 mg/kg q 4-6 h (oral) single dose 0.5 mg/kg acetaminophen q 4-6 h	10 mg/mL suspension
Hydroxyzine (Hydroxyzine hydrochloride)	0.5-1 mg/kg q 4-6 h (oral) single dose 0.5 mg/kg acetaminophen q 4-6 h	5 mg/mL suspension
Propofol and Diprivan (Propofol)	0.5-1 mg/kg q 4-6 h (oral) single dose 0.5 mg/kg acetaminophen q 4-6 h	Propofol - 100 mg/mL

TABLE 3-18. DOSING SCHEDULES AND FORMULATIONS FOR ORAL OPIOIDS IN CHILDREN

The use of the PCA pump should be explained to patients preoperatively. Effective use of a loading dose will avoid the problem of having to play catch-up with out-of-control levels of pain.

Mishaps have occurred with PCA pumps due to programming errors (156), so ward personnel must be totally familiar with the equipment. Treatment of opioid-related side effects is outlined in Table 3-18.

### Oral Administration of Opioids

Oral dosing of opioids is extremely useful for the continued management of diminishing postoperative pain, once oral analgesics are tolerated. Several oral analgesics are available, and their appropriate use is summarized in Table 3-19. None of these medications is devoid of side effects, including mood changes, nausea, vomiting, constipation, dizziness, and pruritus. The occurrence and degree of side effects vary from patient to patient, so the physician should be prepared to change dosing regimens based on patient response. The use of NSAIDs (see the following section) as part of the analgesic regimen may be helpful in reducing or eliminating troublesome opioid-related side effects.

Agent	Dose	Formulations*
Meperidine (oral)	0.5-1 mg/kg q 4-6 h (oral) single dose for treatment of pain, not specifically for analgesia	50 mg/mL suspension Tablets (25, 50, 100, 200 mg)
Meperidine (oral)	0.5-1 mg/kg q 4-6 h	50 mg/mL suspension
Meperidine (IM, IV)	0.5-1 mg/kg q 4-6 h	Tablets (25, 50, 100 mg) suspensions (50 mg/mL)
Choline Magnesium Trisilicate (oral)	0.5-1 mg/kg q 4-6 h	50 mg/mL suspension
Trisilicate (oral)	0.5-1 mg/kg q 4-6 h	Tablets (50, 100 mg)
Acetaminophen (oral, rectal)	0.5-1 mg/kg q 4-6 h	100 mg/mL suspension 100 mg/mL suspension 100 mg/mL suspension 100 mg/mL suspension 100 mg/mL suspension

TABLE 3-19. DOSING SCHEDULES AND FORMULATIONS FOR NSAIDS IN CHILDREN

### Other Modes of Opioid Administration

Epidural opioids are being used in children after major surgery with excellent results ( 112). The author encourages close cooperation between surgeons and anesthesiologists to avail children of this modality of analgesia whenever feasible.

### Postoperative Analgesia With Nonsteroidal Antiinflammatory Drugs

NSAIDs have moderately good analgesic properties (148). Unlike opioids, which produce analgesia by effects on CNS receptors, NSAIDs act peripherally by inhibiting prostaglandin synthesis and decreasing inflammation (137,152). Inflammatory mechanisms play an important part in the pathogenesis of postoperative pain (148), and therefore, the use of NSAIDs makes good sense in the postoperative setting. Also, although NSAIDs have some troubling side effects of their own, they do not produce respiratory depression, nausea, and vomiting, which are some of the bothersome features of opioids. Thus, using NSAIDs either as an adjunct or as a substitute for opioids where feasible should decrease or eliminate the possibility of drug-induced nausea, vomiting, or respiratory depression in the surgical patient (148).

Potential side effects of this class of drugs include platelet dysfunction, gastritis, and acute renal dysfunction ( 93,152). A history of sensitivity to aspirin or a history of nasal polyps may be associated with potentially fatal cross-sensitivity to other NSAIDs ( 1,137). In children with asthma, the prevalence of aspirin sensitivity may be as high as 28% (111). Therefore, asthmatic children should probably receive only those NSAIDs that do not cross-react with aspirin. These medications include acetaminophen, salsalate (Disalcid), and choline magnesium trisilicylate (Trilisate) ( 130) (Table 3-19).

In a child with a chronic underlying bleeding disorder, NSAIDs are not necessarily contraindicated. Consultation with the child's hematologist is advised regarding the use of specific medications in this class.

Ketorolac, unlike other NSAIDs, can be administered not only orally but also intravenously and intramuscularly. A loading dose of 1.0 mg/kg may provide similar analgesia as 0.1 mg/kg of morphine (90). The pharmacology of ketorolac has been extensively reviewed (86), and both its mode of action and adverse reactions are generally typical of NSAIDs. The major controversy with this drug remains its effect on hemostasis and bleeding. Rusy and co-workers (117) found that ketorolac contributed to increased blood loss and more difficulty in achieving surgical hemostasis in pediatric tonsillectomy patients. Caution is advised in administering ketorolac or any other NSAID in a perioperative situation in which bleeding has been or can be significant. Ketorolac has been associated with an increased incidence of nonunion in patients undergoing spine fusion.

Suggested dosing schedules for some of the more common NSAIDs are listed in Table 3-8. Acetaminophen is considered a member of this class of medications, although its mechanism of action is central and its effects on prostaglandin synthesis and the inflammatory response are comparatively very weak (152).

### Postoperative Analgesia With Local Anesthetic Agents

Regional anesthesia is an excellent means of providing postoperative analgesia without respiratory depression and with minimal physiologic alterations (160). Both central (epidural) and peripheral (e.g., brachial plexus blocks) nerve blocks may be used for this purpose. The physician must ensure that the pain relief achieved does not mask the signs and symptoms of developing vascular or neurologic compromise (41,101).

### TREATMENT OF POSTOPERATIVE NAUSEA

Postoperative nausea is common in children, although not particularly after peripheral orthopaedic procedures (90). The pharmacologic treatment is outlined in Table 3-20. Additional helpful measures include not forcing intake of oral fluids until the child is hungry, and minimizing early postoperative ambulation, especially when opioids have been given (12).

Agent	Dosage	Route
Promethazine (Phenergan)	0.25–0.5 mg/kg	IV or per rectum
Metoclopramide (Regan)	0.1 mg/kg (maximum dose, 5 mg)	IV
Ondansetron (Zofran)	0.15 mg/kg (maximum dose, 4 mg)	IV

\* Additional helpful measures include ensuring adequate IV hydration, avoiding forced oral fluids until the patient is hungry, and avoiding forced early ambulation, especially when opioids are given for pain relief.

TABLE 3-20. PHARMACOLOGIC APPROACH TO POSTOPERATIVE NAUSEA\*

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### CHAPTER REFERENCES

1. Abrishami MA, Thomas J. Aspirin intolerance—a review. *Annals Allergy Asthma Immuno*. 1977;39:28–37.
2. Albright GA. Cardiac arrest following regional anesthesia with etidocaine or bupivacaine. [Editorial]. *Anesthesiology* 1979;51:285–287.
3. Alexander, CM, Gross JB. Sedative doses of midazolam depress hypoxic ventilatory responses in humans. *Anesth Analg* 1988;67:377–382.
4. Alioto RJ, Furia JP, Marquardt JD. Hematoma block for ankle fractures: a safe and efficacious technique for manipulations. *J Orthop Trauma* 1995;9:113–116.
5. Arandia HY, Patil VU. Glottic closure following large doses of fentanyl [Letter]. *Anesthesiology* 1987;66:574–575.
6. Askgaard B, Nilsson T, Ibler M, et al. Muscle tone under fentanyl–nitrous oxide anaesthesia measured with a transducer apparatus in cholecystectomy incisions. *Acta Anaesthesiol Scand* 1977;21:104.
7. Azar I, Turndorf H. Severe hypertension and multiple atrial premature contractions following naloxone administration. *Anesth Ana* 1979;58:524–525.
8. Bailey PL, Stanley TH. Pharmacology of intravenous narcotic anesthetics. In: Miller RD (ed). *Anesthesia*, 2nd ed. New York: Churchill-Livingstone, 1986:745–797.
9. Barker SJ, Hyatt J, Shah NK, Kao YJ. The effect of sensor malpositioning of pulse oximeter accuracy during hypoxemia. *Anesthesiology* 1993;79:248–254.
10. Barnes CL, Blasler RD, Dodge BM. Intravenous regional anesthesia: a Safe and cost-effective outpatient anesthetic for upper extremity fracture treatment in children. *J Pediatr Orthop* 1991;11:717–720.
11. Berde CB, Lehn BM, Yee JD, et al. Patient-controlled analgesia in children and adolescents: A prospective comparison with intramuscular administration of morphine for postoperative analgesia. *J Pediatr* 1991;118:461–466.
12. Berry FA. Anesthesia for the child with a difficult airway. In: Berry FA, ed. *Anesthetic management of difficult and routine pediatric patients*, 2nd ed. New York: Churchill-Livingstone, 1990:15–52.
13. Berry FA. General philosophy of patient preparation, premedication, and induction of anesthesia. In: Berry FA, ed. *Anesthetic management of difficult and routine pediatric patients*, 2nd ed. New York: Churchill-Livingstone, 1990:15–52.
14. Berry FR. Analgesia in patients with fractures shaft of femur. *Anaesthesia* 1977;32:576–577.
15. Bier A. Über einen neuen weg lokalanästhesie an den gliedmaßen zu erzeugen. *Verhandlungen der Deutschen Gesellschaft für Chirurgie (Berlin)* 1908;37:204–213.
16. Bilban P, Baraldi E, Pettenazzo A, et al. Adverse effect of chloral hydrate in two young children with obstructive sleep apnea. *Pediatrics* 1993;92:461–463.
17. Bolte RG, Stevens PM, Scott SM, Schunk JE. Mini-dose Bier block intravenous regional anesthesia in the emergency department treatment of pediatric upper-extremity injuries. *J Pediatr Orthop* 1994;14:534–537.
18. Braunstein MC. Apnea with maintenance of consciousness following intravenous diazepam. *Anesth Analg* 1979;58:52–53.
19. Bricker SRW, McCluckie A, Nightingale DA. Gastric aspirates after trauma in children. *Anaesthesia* 1989;44:721–724.
20. Broadman LM. Patient-controlled analgesia in children and adolescents. In: Ferrante FM, Ostheimer GW, Covino BG, eds. *Patient-controlled analgesia*. Boston: Blackwell Scientific Publications, 1990:129–138.
21. Brooks TD, Paulus DA, Winkle WE. Infrared heat lamps interfere with pulse oximeters [letter]. *Anesthesiology* 1984;61:630.
22. Brown DT, Beamish D, Wildsmith JAW. Allergic reaction to an amide local anaesthetic. *Br J Anaesth* 1981;53:435–437.
23. Brown RE Jr, Broadman LM. Patient-controlled analgesia for postoperative pain control in adolescents [abstract]. *Anesth Analg* 1987;66:S22.
24. Brown TCK, Fisk GC. *Anaesthesia for children*. Oxford: Blackwell Scientific Publications, 1979:29–31.
25. Bührer M, Maitre PO, Crevoisier C, Stanski DR. EEG effects of benzodiazepines. II. Pharmacodynamic modeling of the EEG effects of midazolam and diazepam. *Clin Pharmacol Ther* 1990;48:555–567.
26. Carson IW, Moore J, Balmer JP, et al. Laryngeal competence with ketamine and other drugs. *Anesthesiology* 1973;38:128–133.
27. Case RD. Haematoma block—a safe method of reducing Colles' fractures. *Injury* 1985;16:469–470.
28. Chan CYJ. Pediatric pharmacology. In: Rasch DK, Webster DE, eds. *Clinical manual of pediatric anesthesia*. New York: McGraw-Hill, 1994:27–46.
29. Colizza WA, Said E. Intravenous regional anesthesia in the treatment of forearm and wrist fractures and dislocations in children. *Can J Surg* 1993;36:225–228.
30. Coln D. Trauma in children. In: Levin DL, Morriss FC, eds. *Essentials of pediatric intensive care*. St. Louis: Quality Medical Publishing, 1990:671–676.
31. Corssen G, Miyasaka M, Domino EF. Changing concepts in pain control during surgery: dissociative anesthesia with CI-581, a Progress Report. *Anesth Analg* 1968;47:746–759.
32. Cote' CJ. NPO after midnight for children—a reappraisal. *Anesthesiology* 1990;72:589–592.
33. Cote' CJ. Sedation for the pediatric patient—a review. *Pediatr Clin North Am* 1994;41:31–51.
34. Cote' CJ, Goldstein EA, Cote' MA, et al. A single-blind study of pulse oximetry in children. *Anesthesiology* 1988;68:184–188.
35. Cote' CJ, Todres ID. The pediatric airway. In: Ryan JF, Todres ID, Cote' CJ, Goudsouzian NG, eds. *A practice of anesthesia for infants and children*. New York: Grune and Stratton, 1986:35–57.
36. Covino BG. Clinical pharmacology of local anesthetic agents. In: Cousins MJ, Bridenbaugh PO, ed. *Neural blockade in clinical anesthesia and management of pain*, 2nd ed. Philadelphia, J.B. Lippincott, 1988:111–144.
37. Dachs RJ, Ines GM. Intravenous ketamine sedation of pediatric patients in the emergency department. *Ann Emerg Med* 1997;29:146–150.
38. Deshpande JK, Anand KJS. Basic aspects of acute pediatric pain and sedation. In: Deshpande JK, Tobias JD, ed. *The pediatric pain handbook*. St. Louis: Mosby, 1996:1–48.
39. Doyle E, Robinson D, Morton NS. Comparison of patient-controlled analgesia with and without a background infusion after lower abdominal surgery in children. *Br J Anaesth* 1993;71:670–673.
40. Dretchen K, Ghoneim MM, Long JP. The interaction of diazepam with myoneural blocking agents. *Anesthesiology* 1971;34:463–468.
41. Dunwoody JM, Reichert CC, Brown KLB. Compartment syndrome associated with bupivacaine and fentanyl analgesia in pediatric orthopaedics. *J Pediatr Orthop* 1997;17:285–288.

42. Eddie R, Deutsch S. Cardiac arrest after interscalene brachial-plexus block. *Anesth Analg* 1977;56:446–447.
43. Emergency drug doses for infants and children and naloxone use in newborns: clarification. *Pediatrics* 1989;83:803.
44. Estilo AE, Cottrell JE. Hemodynamic and catecholamine changes after administration of naloxone. *Anesth Analg* 1965;61:349–353.
45. Evans JK, Buckley SL, Alexander AH, Gilpin AT. Analgesia for the reduction of fractures in children: a comparison of nitrous oxide with intramuscular sedation. *J Pediatr Orthop* 1995;15:73–77.
46. Farrell RG, Swanson SL, Walter JR. Safe and effective IV regional anesthesia for use in the emergency department. *Ann Emerg Med* 1985;14:288–292.
47. Fatovich DM, Jacobs IG. A randomized, controlled trial of oral midazolam and buffered lidocaine for suturing lacerations in children (the SLIC trial). *Ann Emerg Med* 1995;25:209–214.
48. Feld LH, Negus JB, White PF. Oral midazolam preanesthetic medication in pediatric outpatients. *Anesthesiology* 1990;73:831–834.
49. Ferrante FM. Patient characteristics influencing effective use of patient-controlled analgesia. In: Ferrante FM, Ostheimer, GW, Covino BG, ed. *Patient-controlled analgesia*. Boston: Blackwell Scientific Publications, 1990:51–60.
50. Ferrari LR, Rooney FM, Rockoff MA. Preoperative fasting practices in pediatrics. *Anesthesiology* 1999;90:978–980.
51. Finegan BA, Bukht MD. Venous pressure in the isolated upper limb during saline injection. *Can Anaesth Soc J* 1984;31:364–367.
52. Fisher M McD, Graham R. Adverse responses to local anaesthetics. *Anaesthesia and Intensive Care* 1984;12:325–327.
53. FitzGerald B. Intravenous regional anesthesia in children. *Br J Anaesth* 1976;48:485–486.
54. Forster A, Gardaz JP, Suter PM, Gemperle M. Respiratory depression by midazolam and diazepam. *Anesthesiology* 1980;53:494–497.
55. Fujigaki T, Fukusaki M, Nakamura H, et al. Quantitative evaluation of gastric contents using ultrasound. *J Clin Anesth* 1993;5:451–455.
56. Galletly D, Forrest P, Purdie G. Comparison of the recovery characteristics of diazepam and midazolam. *Br J Anaesth* 1988;60:520–524.
57. Gibbs PC, Modell JH. Aspiration pneumonitis. In: Miller RD, ed. *Anesthesia*, 2nd ed. New York: Churchill-Livingstone, 1986:2023–2050.
58. Gourlay GK, Boas RA. Fatal outcome with use of rectal morphine for postoperative pain control in an infant. *Br Med J* 1992;304:766–767.
59. Grant IS, Nimmo WS, McNicol LR, Clements JA. Ketamine disposition in children and adults. *Br J Anaesth* 1983;55:1107–1111.
60. Green SM, Johnson NE. Ketamine sedation for pediatric procedures: Part 2, review and implications. *Ann Emerg Med* 1990;19:1033–1046.
61. Green SM, Nakamura R, Johnson NE. Ketamine sedation for pediatric procedures: Part 1, a prospective series. *Ann Emerg Med* 1990;19:1024–1032.
62. Green SM, Rothrock SG, Lynch EL, et al. Intramuscular ketamine for pediatric sedation in the emergency department: safety profile in 1,022 cases. *Ann Emerg Med* 1998;31:688–697.
63. Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures. *Pediatrics* 1992;89:1110–1115.
64. Gutstein HB, Johnson KL, Heard MB, Gregory GA. Oral ketamine preanesthetic medication in children. *Anesthesiology* 1992;76:28–33.
65. Hennes HM, Wagner V, Bonadio WA, et al. The effect of oral midazolam on anxiety of preschool children during laceration repair. *Ann Emerg Med* 1990;19:1006–1009.
66. Hennrikus WL, Shin AY, Klingelberger CE. Self-administered nitrous oxide and a hematoma block for analgesia in the outpatient reduction of fractures in children. *J Bone Joint Surg Am* 1995;77:335–339.
67. Hennrikus WL, Simpson RB, Klingelberger CE, Reis MT. Self-administered nitrous oxide analgesia for pediatric fracture reductions. *J Pediatr Orthop* 1994;14:538–542.
68. Hershey LA. Meperidine and central nervous system toxicity. [Editorial.] *Ann Intern Med* 1983;98:548–549.
69. Hollister GR, Burn JMB. Side effects of ketamine in pediatric anesthesia. *Anesth Analg* 1974;53:264–267.
70. Holmes MC. Intravenous regional analgesia, a useful method of producing analgesia of the limbs. *Lancet* 1963;1:245–247.
71. Holmes MC. Intravenous regional neural blockade. In: Cousins MJ, Bridenbaugh PO, eds. *Neural blockade in clinical anesthesia and management of pain*, 2nd ed. 1988:443–459.
72. Iber FL, Livak A, Kruss DM. Apnea and cardiopulmonary arrest during and after endoscopy. *J Clin Gastroenterol* 1992;14:109–113.
73. Jailon P, Gardin ME, Lecoq B, et al. Pharmacokinetics of nalbuphine in infants, young healthy volunteers, and elderly patients. *Clin Pharmacol Ther* 1989;46:226–233.
74. Jastak JT, Pallasch T. Death after chloral hydrate sedation: report of a case. *J Am Dent Assoc* 1988;116:345–347.
75. Johnson PQ, Noffsinger MA. Hematoma block of distal forearm fractures. Is it safe? *Orthop Rev* 1991;20:977–979.
76. Jones RDM, Chan K, Roulson CJ, et al. Pharmacokinetics of flumazenil and midazolam. *Br J Anaesth* 1993;70:286–292.
77. Jones RDM, Lawson AD, Andrew LJ, et al. Antagonism of the hypnotic effect of midazolam in children: a randomized double-blind study of placebo and flumazenil administered after midazolam-induced anaesthesia. *Br J Anaesth* 1991;66:660–666.
78. Kaiko RF, Foley KM, Gabrinski PY, et al. Central nervous system excitatory effects of meperidine in cancer patients. *Ann Neuro* 1983;13:180–185.
79. Kaplan RF. *Sedation and analgesia in pediatric patients for procedures outside the operating room*. #221, American Society of Anesthesiologists Annual Refresher Course Lectures, October, 1997.
80. Karl HW, Keifer AT, Rosenberger JL, et al. Comparison of the safety and efficacy of intranasal midazolam or sufentanil for preinduction of anesthesia in pediatric patients. *Anesthesiology* 1989;76:209–215.
81. Kennedy RM, Porter FL, Miller JP, Jaffe DM. Comparison of fentanyl/midazolam with ketamine/midazolam for pediatric orthopedic emergencies. *Pediatrics* 1998;102:956–963.
82. Klotz U, Kanto J. Pharmacokinetics and clinical use of flumazenil (Ro 15-1788). *Clin Pharmacokinet* 1988;14:1–12.
83. Kohrs R, Durieux ME. Ketamine: teaching an old drug new tricks. *Anesth Analg* 1998;87:1186–1193.
84. Kongsholm MJ, Olerud C. Neurological complications of dynamic reduction of Colles' fractures without anesthesia compared with traditional manipulation after local infiltration anesthesia. *J Orthop Trauma* 1987;1:43–47.
85. Litman RS. Airway obstruction after oral midazolam [Letter]. *Anesthesiology* 1996;85:1217–1218.
86. Litvak KM, McEvoy GK. Ketorolac, an injectable non-narcotic analgesic. *Clin Pharm* 1990;9:921–935.
87. Lockhart CH, Nelson WL. The relationship of ketamine requirement to age in pediatric patients. *Anesthesiology* 1974;40:507–508.
88. Lowe S, Hershey S. Sedation for imaging and invasive procedures. In: Deshpande JK, Tobias JD, ed. *The pediatric pain handbook*. St. Louis: Mosby, 1996:263–317.
89. Magnat D, Orr WC, Smith RO. Sleep apnea, hypersomnolence, and upper airway obstruction secondary to adenotonsillar enlargement. *Arch Otolaryngol Head Neck Surg* 1977;103:383–386.
90. Mason LJ. *Challenges in pediatric anesthesia*. International Anesthesia Research Society Review Course Lectures, 1999:64–70.
91. Massanari M, Novitsky J, Reinstein LJ. Paradoxical reaction in children associated with midazolam use during endoscopy. *Clin Pediatr* 1997;36:681–684.
92. Maxwell LG, Yaster M. The myth of conscious sedation. *Arch Pediatr Adolesc Med* 1996;150:665–667.
93. McIntire SC, Rubenstein RC, Gartner JC Jr, et al. Acute flank pain and reversible renal dysfunction associated with nonsteroidal anti-inflammatory drug use. *Pediatrics* 1993;92:459–460.
94. Meinig RP, Quick A, Lobmeyer L. Plasma lidocaine levels following hematoma block for distal radius fractures. *J Orthop Trauma* 1989;3:187–189.
95. Miller M, Wishar HY, Nummo WS. Gastric contents at induction of anaesthesia—is a 4-hour fast necessary? *Br J Anaesth* 1983;55:1185–1187.
96. Mitchell RK, Koury SI, Stone CK. Respiratory arrest after intramuscular ketamine in a 2-year-old child. *Am J Emerg Med* 1996;14:580–581.
97. Momota Y, Artu AA, Powers KM, et al. Posttreatment with propofol terminates lidocaine-induced epileptiform electroencephalogram activity in rabbits: Effects on cerebrospinal fluid dynamics. *Anesth Analg* 1988;87:900–906.
98. Moore DC, Crawford RD, Scurlock JE. Severe hypoxia and acidosis following local anesthetic-induced convulsions. *Anesthesiology* 1983;53:1185–1187.
99. Morris RE, Miller GW. Preoperative management of the patient with a full stomach. *Clin Anesth* 1976;11:25–29.
100. Morriss FC. Anaphylaxis. In: Levin DL, Morriss FC, ed. *Essentials of pediatric intensive care*. St. Louis: Quality Medical Publishing, 1990:98–105.
101. Mubarak SJ, Wilton CTN. Compartment syndromes and epidural anesthesia. (Editorial.) *J Pediatr Orthop* 1997;17:282–284.
102. Myers EF, Charles P. Prolonged adverse reactions to ketamine in children. *Anesthesiology* 1978;49:39–40.
103. Noorily SH, Norrily AD. Anesthesia for pediatric ear, nose, and throat procedures. In: Rasch DK, Webster DE, eds. *Clinical manual of pediatric anesthesia*. New York: McGraw-Hill, 1994:380–402.
104. Olney BW, Lugg PC, Turner PL, et al. Outpatient treatment of upper extremity injuries in childhood using intravenous regional anaesthesia. *J Pediatr Orthop* 1988;8:576–579.
105. Perkin RM, Levin DL. Shock. In: Levin DL, Morriss FC, eds. *Essentials of pediatric intensive care*. St. Louis: Quality Medical Publishing, 1990:78–79.
106. Peterson MD. Making oral midazolam palatable for children [Letter]. *Anesthesiology* 1990;73:1053.
107. Philip BK, Simpson TH, Hauch MA, Malampati SR. Flumazenil reverses sedation after midazolam-induced general anesthesia in ambulatory surgery patients. *Anesth Analg* 1990;71:371–376.
108. Practice guidelines for sedation and analgesia by non-anesthesiologists. *Anesthesiology* 1996;84:459–471.
109. Prentiss JE. Cardiac arrest following caudal anesthesia. *Anesthesiology* 1979;50:51–53.
110. Proudfoot J, Roberts M. Providing safe and effective sedation and analgesia for pediatric patients. *Emergency Medicine Reports* 1993;14:207–218.
111. Rachelefsky GS, Coulson A, Siegel SC, Stiehm ER. Aspirin intolerance in chronic childhood asthma: detected by oral challenge. *Pediatrics* 1975;56:443–448.
112. Rasmussen GE. Epidural and spinal anesthesia and analgesia. In: Deshpande JK, Tobias JD, eds. *The pediatric pain handbook*. St. Louis: Mosby, 1996:81–112.
113. Reeves JG, Fragen RJ, Vinik HR, Greenblatt DJ. Midazolam: pharmacology and uses. *Anesthesiology* 62:310–324.
114. Rita L, Seleny F, Goodarzi M. Comparison of the calming and sedative effects of nalbuphine and pentazocine for paediatric premedication. *Can Anaesth Soc J* 1980;27:546–549.
115. Rogers J, Moro M. Acute postoperative and chronic pain in children. In: Rasch DK, Webster DE, eds. *Clinical manual of pediatric anesthesia*. New York: McGraw-Hill, 1994:291–306.
116. Romagnoli A, Keats AS. Ceiling effect for respiratory depression by nalbuphine. *Clin Pharmacol Therapeut* 1980;27:478–485.
117. Rusy LM, Houck CS, Sullivan, LJ, et al. A double blind evaluation of ketorolac tromethamine versus acetaminophen in pediatric tonsillectomy patients, effects on analgesia and bleeding. *Anesth Analg* 1995;80:226–229.
118. Saint-Maurice C, Laguenie G, Couturier C, Goutail-Flaud F. Rectal ketamine in pediatric anesthesia [Letter]. *Br J Anaesth* 1979;51:573–574.
119. Sacchetti A, Schafermeyer R, Gerardi M, et al. Pediatric analgesia and sedation. *Ann Emerg Med* 1994;23:237–250.
120. Scamman FL. Fentanyl-oxygen-nitrous oxide rigidity and pulmonary compliance. *Anesth Analg* 1983;62:332–334.
121. Schechter NL, Weisman SJ, Rosenblum M, et al. The use of oral transmucosal fentanyl citrate for painful procedures in children. *Pediatrics* 1995;95:335–339.
122. Schreiner MS, Triebwasser A, Keon TP. Ingestion of liquids compared preoperative fasting in pediatric outpatients. *Anesthesiology* 1990;72:593–597.
123. Scott DB, Lee A, Fagan D, et al. Acute toxicity of ropivacaine compared with that of bupivacaine. *Anesth Analg* 1989;69:563–569.
124. Scott RB, Steinberg RB, Kreitzer JM, Duprat KM. Intravenous regional anesthesia using lidocaine and ketorolac. *Anesth Analg* 1995;81:110–113.
125. Sievers TD, Yee JD, Foley ME, Berde CB. Midazolam for conscious sedation during pediatric oncology procedures: safety and recovery parameters. *Pediatrics* 1991;88:1172–1179.
126. Smith JA, Santer LS. Respiratory arrest following intramuscular ketamine injection in a 4-year-old child. *Ann Emerg Med* 1993;22:613–615.
127. Snodgrass WR, Dodge WF. Lytic/DPT cocktail: time for rational and safe alternatives. *Pediatr Clin North Am* 1989;36:1285–1291.

128. Sokoll MD, Hoyt JL, Gergis SD. Studies in muscle rigidity, nitrous oxide, and narcotic analgesic agents. *Anesth Analg* 1972;51:16–20.
129. Splinter WM, Stewart JA, Muir JG. The effect of preoperative apple juice on gastric contents, thirst and hunger in children. *Can J Anaesth* 1989;36:55–58.
130. Stevenson DD, Simon RA. Aspirin sensitivity: respiratory and cutaneous manifestations. In: Middleton E Jr, Reed CE, Ellis EF, et al., eds. *Allergy principles and practice*, 3rd ed. St. Louis: C. V. Mosby, 1988:1537–1554.
131. Stoeckel H, Hengstmann JH, Shuttler J. Pharmacokinetics of fentanyl as a possible explanation for recurrent respiratory depression. *Br J Anaesth* 1979;51:741–745.
132. Stoelting RK. Barbiturates. In: *Pharmacology and physiology in anesthetic practice*. Philadelphia: JB Lippincott, 1987:102–116.
133. Stoelting RK. Benzodiazepines. In: *Pharmacology and physiology in anesthetic practice*. Philadelphia: JB Lippincott, 1987:117–133.
134. Stoelting RK. Endotracheal intubation. In: Miller RD, ed. *Anesthesiology*, 2nd ed. New York; Churchill-Livingstone, 1986:523–552.
135. Stoelting RK. Inhaled anesthetics. In: *Pharmacology and physiology in anesthetic practice*. Philadelphia: JB Lippincott, 1987: 2–34.
136. Stoelting RK. Nonbarbiturate induction drugs. In: *Pharmacology and physiology in anesthetic practice*. Philadelphia: JB Lippincott, 1987:134–147.
137. Stoelting RK. Nonopioid and nonsteroidal analgesic, antipyretic, and anti-inflammatory drugs. In: *Pharmacology and physiology in anesthetic practice*. Philadelphia: JB Lippincott, 1987:240–250.
138. Stoelting RK. Opioid agonists and antagonists. In: *Pharmacology and physiology in anesthetic practice*. Philadelphia: JB Lippincott: 1987:69–101.
139. Strichartz GR. Neural physiology and local anesthetic action. In: Cousins MJ, Bridenbaugh PO, eds. *Neural blockade in clinical anesthesia and management of pain*, 2nd ed. Philadelphia, J.B. Lippincott, 1987:24–45.
140. Striker TW. Anesthesia for trauma in the pediatric patient. In: Gregory GA, ed. *Pediatric anesthesia*, 2nd ed. New York: Churchill-Livingstone, 1989:1273–1288..
141. Taylor PA, Towey RM. Depression of laryngeal reflexes during ketamine anaesthesia. *Br Med J* 1971;2:688–689.
142. Terndrup TE, Dire DJ, Madden CM, et al: A prospective analysis of intramuscular meperidine, promethazine, and chlorpromazine in pediatric emergency department patients. *Ann Emerg Med* 1991;20:31–35.
143. Tired L, Nivoche Y, Hatton F, et al. Complications related to anaesthesia in infants and children. A prospective survey of 40,240 anaesthetics. *Br J Anaesth* 1988;61:263–269.
144. Tobias JD. Sedation in the pediatric intensive care unit. In: Deshpande JK, Tobias JD, eds. *The pediatric pain handbook*. St. Louis: Mosby, 1996:235–261.
145. Tobias JD, Phipps S, Smith B, Mulhern RK. Oral ketamine premedication to alleviate the distress of invasive procedures in pediatric oncology patients. *Pediatrics* 1992;90:537–541.
146. Tucker GT, Mather LE. Properties, absorption, and disposition of local anesthetic agents. In: Cousins MJ, Bridenbaugh PO, ed. *Neural blockade in clinical anesthesia and management of pain*, 2nd ed. Philadelphia: JB Lippincott, 1988:47–110..
147. Turner PL, Batten JB, Hjorth D, et al. Intravenous regional anaesthesia for the treatment of upper limb injuries in childhood. *Aust N Z J Surg* 1986;56:153–155.
148. Tyler DC. Pharmacology of pain management. *Pediatr Clin North Am* 1994;41:59–69.
149. Vandam LD. Butorphanol. *N Engl J Med* 1980;302:381–384.
150. Varela CD, Lorfing KC, Schmidt TL. Intravenous sedation for the closed reduction of fractures in children. *J Bone Joint Surg Am* 1995;77A:340–345.
151. Vetter TR. Pediatric patient-controlled analgesia with morphine versus meperidine. *J Pain Symptom Manage* 1992;7:204–208.
152. Walson PD, Mortensen ME. Pharmacokinetics of common analgesics, anti-inflammatories and antipyretics in children. *Clin Pharmacokinet* 1989;17(Suppl 1):116–137.
153. Webster DE. The pediatric trauma patient. In: Rasch DK, Webster DE, eds. *Clinical manual of pediatric anesthesia*. New York: McGraw-Hill, 1994:189–216.
154. Weksler N, Ovadia L, Mutai G, Stav A. Nasal ketamine for paediatric premedication. *Can J Anaesth* 1993;40:119–121.
155. Wertz RC. Anesthesia for pediatric trauma. In: Steen JK, Grande CM, eds. *Trauma anesthesia*. Baltimore: Williams & Wilkins, 1991:312–329.
156. White PF. Mishaps with patient-controlled analgesia. *Anesthesiology* 1987;66:81–82.
157. White PF, Way WL, Trever AJ. Ketamine—its pharmacology and therapeutic uses. *Anesthesiology* 1982;56:119–136.
158. Wright SW, Chudnofsky CR, Dronen SC, et al. Comparison of midazolam and diazepam for conscious sedation in the emergency department. *Ann Emerg Med* 1993;22:201–205.
159. Wu MYC, Purcell GJ. Patient-controlled analgesia—the value of a background infusion [Letter.] *Anaesthesia and Intensive Care* 1990;18:575–576.
160. Yaster M, Maxwell LG. Pediatric regional anesthesia. *Anesthesiology* 1989;70:324–338.
161. Yaster M, Nichols DG, Deshpande JK, Wetz RC. Midazolam-fentanyl intravenous sedation in children: case report of respiratory arrest. *Pediatrics* 1990;86:463–467.
162. Younge D. Hematoma block for fractures of the wrist: a cause of compartment syndrome. *J Hand Surg* 1989;14B:194–195.

## MANAGEMENT OF THE MULTIPLY INJURED CHILD

VERNON T. TOLO

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### INCIDENCE OF INJURIES

#### Trauma

The most common cause of death in children over the age of 1 year is trauma, not only in the United States but worldwide. Estimates of cost to the American public for the care of pediatric trauma range from over \$1 billion (53) to \$13.8 billion (56) annually. Although isolated long bone fractures still comprise the bulk of orthopaedic injuries in children, a surprising number of these young patients have multiple system injuries.

The cause of death from trauma in children generally is severe head injury. Boys are injured twice as often as girls and may account for even a greater proportion of hospital admissions related to pediatric trauma. Blunt trauma is the mechanism of injury in most children and preadolescents, whereas penetrating trauma more often is the source of multiple injuries in adults. Although blunt trauma in the youngest children often is due to child abuse, vehicular accidents and falls from a height account for the more severe multiple injuries in the rest of childhood (12).

Multiple injuries in teenagers more closely mirror the causes in adults. In the adolescent age group, alcohol abuse now is considered a major factor in over a third of injuries resulting from accidents (55). Orthopaedists treating teenagers involved in vehicular accidents need to be aware of the potential alcohol use in this age group and be prepared to refer adolescents for appropriate counseling to avoid future accidents and injuries.

#### Fractures

Although they are rarely the cause of mortality in a child with multiple injuries, fractures and other injuries to the musculoskeletal system are commonly a major part of the injuries (12,18,20,63). In one series from a pediatric trauma center treating children with polytrauma, femoral shaft fractures accounted for 22% of the fractures and 9% of the fractures were open (12). Although they are less common, fractures of the spine, pelvis, and scapula and clavicle were associated with longer stays in the hospital and in the intensive care unit, in addition to having the highest associated mortality rates.

Knowledge of fracture associations leads to improved diagnostic skill and fracture care. Femoral and adjacent pelvic fractures often occur together. If a pedestrian child has been struck by an automobile, there often are fractures in the ipsilateral upper extremity and lower extremity (11). The coexistence of a femoral fracture and a head injury indicates substantial high-energy trauma and has a more guarded prognosis than does either of these injuries alone.

#### Child Abuse

Child abuse continues to be a societal problem that crosses all socioeconomic and ethnic groups. This diagnosis must be suspected in all cases of multiple injuries in children younger than 2 years of age, if there is no obvious and witnessed plausible explanation of the injuries. Abuse continues to be the most common cause of traumatic death in infants and toddlers. Abuse should be considered a possible cause of injury in all young children with multiple long bone fractures in association with head injury. Even a single long bone fracture associated with a head injury or abdominal injury should raise suspicion of child abuse. Although the *corner fracture* usually is thought of as being most characteristic of child abuse, the most common fracture caused by abuse is a single transverse fracture of the femur or humerus, not multiple fractures (42). Although rib fractures occur in only about 5% of children with multiple injuries from trauma of other causes, they are more common in child abuse (25,63). Whereas blunt compressive trauma to the thorax from other causes may result in lateral rib fractures, the rib fractures seen in child abuse are posterolateral and adjacent to the transverse processes of the thoracic spine.

### COMMON MECHANISMS OF INJURY

#### Falls

Falls are one of the two primary mechanisms of multiple injuries in children (12,28,72). Occurring more often in younger children, these injuries are either due to the direct impact or to deceleration forces present at the time of landing. Direct impact usually causes fractures, whereas internal injury more often results from the post-impact forces. Falls through a second floor window that may be next to a bed often occur in toddlers and younger children. Although a variety of injuries can result from these falls, the position of the body at impact and the surface on which the child lands are important factors that affect the injury severity (28).

#### Motor Vehicles

Accidents involving motor vehicles account for most multiple-system injuries in school-age children and preadolescents. These injuries occur when a vehicle strikes a

child on foot or riding a bicycle, or when the child is a passenger in a car involved in an accident.

Whereas most states require that infants and toddlers be restrained in car seats when riding in a car, standard adult shoulder and lap belts do not adequately restrain children who are too big for car seats and too small for the standard restraints. Adjustable restraints to accommodate the size of the car occupant better have been proposed to solve this problem. In addition, there is increasing public sentiment to require seat belt use on school buses, a policy that has been in place for handicapped student transport for some time.

The most important point is that some type of restraining system should be used by people of all ages when in a vehicle. Although teaching children better safety while on foot or on bicycles may or may not be effective in changing the incidence of injury, the safety of automobile travel can certainly be improved by enforcement of the use of restraints by all—both young and old.

## ROLE OF THE PEDIATRIC TRAUMA CENTER

After the rapid transport of wounded soldiers to a specialized treatment center proved very effective in improving survival in the military setting, trauma centers, using the same principles of rapid transport and immediate care, have been established throughout the United States. These trauma centers are supported by the states on the premise that the first hour after injury is the most critical in influencing the rates of survival from the injuries. Rapid helicopter or ambulance transport to an on-site team of trauma surgeons in the trauma center has led to an improvement in the rates of acute survival after multiple injuries have occurred.

The first trauma centers focused on adult patients, because more adults than children are severely injured. However, pediatric trauma centers have been established at several medical centers across the United States with the idea that the care of pediatric polytrauma patients differ from the care given to adults and that special treatment centers are important for optimal results (33,34). The American College of Surgeons has established specific criteria for pediatric trauma centers, which include the same principles of rapid transport and rapid treatment by an in-house surgical team as in adult trauma centers. A pediatric general surgeon is in the hospital at all times and heads the pediatric trauma team. This surgeon evaluates the child first while the other surgical specialists are immediately available as needed. General radiographic services and computed tomography capability must be available at all times for patient evaluation and an operating room must be immediately available.

Although there is some evidence that survival rates for severely injured children are improved if the children are brought to a pediatric trauma center rather than a community hospital (77), the costs associated with such a center (particularly the on-call costs of personnel) have limited the number of pediatric trauma centers. Knudson et al. (44) studied the results of pediatric multiple injury care in an adult level I trauma center and concluded that the results were comparable to national standards for pediatric trauma care. The use of a general trauma center for pediatric trauma care may be an acceptable alternative if it is not feasible to fund a separate pediatric trauma center.

## INITIAL RESUSCITATION AND EVALUATION

Regardless of the mechanism causing the multiple injuries, the initial medical management focuses on the life-threatening, nonorthopaedic injuries to stabilize the child's condition (56). The responsibility for the initial life-saving resuscitation rarely is the responsibility of the orthopaedist. The initial role of the orthopaedist generally is to treat the extremity injuries, even if this just consists of temporary splint application to allow patient transport to the trauma center or even around the hospital for imaging studies. It is recommended, however, that the orthopaedist stay up to date in principles of resuscitation of children to be prepared if the occasion arises when these are needed.

### The Child Is Different

The initial steps in resuscitation of a child are essentially the same as those used for an adult (2,20,56). In severe injuries, the establishment of an adequate airway immediately at the accident site often means the difference between death and survival. The cervical spine needs to be stabilized for transport if the child is unconscious or if neck pain is present. A special transport board with a cut-out for the occipital area is recommended for children younger than 6 years of age, because the size of the head at this age is larger in relation to the rest of the body. If a young child is placed on a normal transport board, the cervical spine is flexed because of this larger head size, a position that is best avoided if a neck injury is suspected (37).

### Fluid Replacement

Once an adequate airway is established, the amount of hemorrhage from the injury, either internally or externally, should be assessed. This blood loss should be replaced initially with intravenous crystalloid solution. In younger children, rapid intravenous access may be difficult. In this situation, the use of intraosseous fluid infusion should be considered for administration of both fluid and medications. Guy et al. (32) reported successful intraosseous infusion into the tibias of 15 children between the ages of 3 months and 10 years. The intraosseous needles were placed by prehospital and hospital personnel, and colloid, crystalloid solution, and blood were all given by this route. No complications occurred in the surviving children. Bielski et al. (7), in a rabbit tibia model, likewise demonstrated no adverse effects on the histology of bone or the adjacent physis with intraosseous injection of various resuscitation drugs and fluids.

Because death is common if hypovolemic shock is not rapidly reversed, the child's blood pressure must be maintained at an adequate level for organ perfusion. Most multiply injured children have sustained blunt trauma rather than penetrating injuries, and most of the blood loss is internal from visceral injury or from pelvic and femoral fractures. Because of this problem, the blood loss may be easily underestimated at first. Despite the need to stabilize the child's blood pressure, caution needs to be exercised in children with head injuries so that over hydration is avoided, because cerebral edema is better treated with relative fluid restriction. Excessive fluid replacement also may lead to further internal fluid shifts, which often produce a drop in the arterial oxygenation from interstitial pulmonary edema, especially when there has been direct trauma to the thorax and lungs. The appropriate amount of fluid replacement can best be guided by early placement of a central venous catheter during initial resuscitation. Similarly, a urinary catheter is essential during the resuscitation to monitor urine output as a means of gauging adequate organ perfusion.

## EVALUATION AND ASSESSMENT

### Trauma Rating Systems

After the initial resuscitation has stabilized the injured child's condition, it is essential to perform a quick but thorough check for other injuries. A number of injury rating systems have been proposed, but the Injury Severity Score (ISS) is a valid, reproducible rating system that can be widely applied in the pediatric polytrauma setting (Table 4-1) (91). Another injury rating system for children that has been shown to be valid and reproducible is the Pediatric Trauma Score (PTS) (Table 4-2) (81). The injury rating system chosen varies among trauma centers, but whether the ISS or PTS is used, these systems allow an objective means to assess mortality risk at the time of initial treatment, as well as allowing some degree of prediction of future disability.

TABLE 4-1. INJURY SEVERITY SCORE

Component	Category		
	+2	+1	-1
Size	<20 kg	10-20 kg	<10 kg
Airway	Normal	Maintainable	Unmaintainable
Systolic BP	<50 mm Hg	50-50 mm Hg	<50 mm Hg
OG	Awake	Obtunded/LOC	Comatose/brat
Open wound	None	Minor	Major/penetrating
Skeletal	None	Closed fracture	Open/multiple fractures
Total			

\* This scoring system includes six common determinants of the clinical condition in the injured child. Each of the six determinants is assigned a grade: +2, minimal or no injury; +1, minor or potentially major injury; -1, major, or immediate life-threatening injury. The scoring system is arranged in a manner standard with advanced trauma life-support protocol, and thereby provides a quick assessment scheme. The ranges are from -6 for a severely traumatized child to +12 for a least traumatized child. This system has been confirmed in its reliability as a predictor of injury severity. From Tepas, J. (1981). Tabern, B., and Bryant, M. The Pediatric Trauma Score as a predictor of injury severity in the injured child. *J Pediatr Surg* 12:14-18, 1987, with permission.

**TABLE 4-2. PEDIATRIC TRAUMA SCORE\***

Head injury is most often evaluated and rated by the Glasgow Coma Scale (GCS), which evaluates eye opening (1 to 4 points), motor function (1 to 6 points), and verbal function (1 to 6 points) on a total scale of 3 to 16 points ( [Table 4-3](#)) (80). There are some limitations in the use of the GCS in children who are preverbal or who are in the early verbal stages of development, but in other children, this rating system has been a useful guide to predicting early mortality and later disability. As a rough guide in verbal children, a GCS score of fewer than 8 points means a significantly worse chance of survival for these children than for those with a GCS above 8 points. The GCS should be noted on arrival in the trauma center and should be repeated 1 hour after the child arrives at the hospital. Serial changes in the GCS, either better or worse, correlate with improvement or worsening of the neurologic injury. Repeated GCS assessments over the initial 72 hours after injury may be of prognostic significance. In addition to the level of oxygenation present at the initial presentation to the hospital, the 72-hour GCS motor response score has been noted to be very predictive of later permanent disability as a sequel to the head injury ( [58](#)).

Response	Action	Score
Best motor response	Obeys	6
	Localizes	5
	Withdraws	4
	Abnormal flexion	3
	Extensor response	2
Verbal response	Nil	1
	Oriented	5
	Confused conversation	4
	Inappropriate words	3
	Incomprehensible sounds	2
Eye opening	Nil	1
	Spontaneous	4
	To speech	3
	To pain	2

\* This scale is used to measure the level of consciousness using the eye opening, best verbal, and best motor responses. The range of scores are from 3 for the most severe to 15 for the least involved. This is a measure of level and progression of changes in consciousness. From Jeanneret B, Trandate JB, Galbraith S, et al. Severe head injuries in three countries. *J Neurosurg* 1977;60:291-298, with permission.

**TABLE 4-3. GLASGOW COMA SCALE\***

### Physical Assessment

In a child with multiple injuries, a careful abdominal examination is essential to allow early detection of injuries to the liver, spleen, pancreas, or kidneys. Ecchymoses on the abdominal wall must be noted, because this is often a sign of significant visceral injury ( [75](#)). Swelling, deformity, or crepitus in any extremity is noted, and appropriate imaging studies are arranged to evaluate potential extremity injuries more fully. If extremity deformity is present, it is important to determine whether or not the fracture is open or closed. Sites of external bleeding are examined, and pressure dressings are applied to prevent further blood loss. It has been reported that the presence of a pelvic fracture and one or more other skeletal injuries should serve as a marker for the presence of head and abdominal injuries ( [89](#)). Major arterial injuries associated with fractures of the extremity usually are diagnosed early by the lack of a peripheral pulse. However, abdominal venous injuries following blunt trauma are less common and are less commonly diagnosed before exploratory laparotomy. About half of abdominal venous injuries have been reported to be fatal, so the trauma surgeon needs to consider this diagnosis in children who continue to require substantial blood volume support after the initial resuscitation has been completed ( [62](#)).

The initial splinting of suspected extremity fractures is routinely done by the transporting team members at the scene of the accident. However, once the injured child is in the hospital, the orthopaedist should personally inspect the extremities to determine the urgency with which the extremity injuries need to be definitively treated. Most important are whether or not a vascular injury has occurred and whether the fracture is open or closed. Any neurologic deficit also is noted to document the extremity function before any treatment. The back and spine should be carefully examined. If there is not an open fracture and if the distal peripheral vascular function is normal, there is less urgency in treating the fracture and splinting will suffice until the other organ system injuries are stabilized.

### Imaging Studies

#### Radiographs

Imaging studies need to be performed as quickly as possible after the initial resuscitation and physical examination are complete. Any extremity suspected of having a significant injury should be examined radiographically to assess the degree of injury. If the child has a head injury or if neck pain is noted on the examination, a lateral cervical spine radiograph is obtained. If the lateral cervical spine radiograph is normal, no further views of the cervical spine are needed at this stage. If a cervical spine injury is present, the lateral radiograph of this area almost always will detect it. If there is suspicion of a cervical spine injury on the neutral lateral view, a lateral cervical spine radiograph in the flexed position, with the orthopaedist supervising the amount of flexion, will help detect any cervical instability. The cervical spine of a young child is much more flexible than the cervical spine in an adult. Under the age of 12 years, the movement of C1 on C2 during flexion of the neck can normally be up to 5 mm, whereas in adults, this movement should be less than 3 mm. Likewise in this young age group, C2 can move up to 3 mm forward on C3 during neck flexion. No forward movement of C2 on C3 should be present in a skeletally mature individual when the neck is flexed. This so-called pseudosubluxation of C2 on C3 in a child should not be diagnosed as instability that requires treatment because this is a normal finding in most young children ( [16](#)). Because it is difficult to detect a fracture of the thoracic or lumbar spine clinically, radiographs of this area, primarily a lateral view, should be assessed, particularly in a comatose child.

#### Computed Tomography

Computed tomography (CT) is essential in evaluating a child with multiple injuries. If a head injury is present, the CT of the head will detect skull fractures and intracranial bleeding. With abdominal swelling, pain, or bruising, a CT of the abdomen provides excellent visualization of the liver and spleen and allows quantification of the amount of hemorrhage present. Because most hepatic and splenic lacerations are treated nonoperatively at present ( [13,38,71](#)), the CT scan and serial hematocrit levels are used to determine whether surgical treatment of these visceral lacerations is needed. If a pelvic fracture is seen or suspected on the initial radiographs, a CT of the pelvis will help to determine the need for operative treatment of this fracture and the length of time protection from walking is needed. If an abdominal CT is being done to evaluate visceral injury and there is suspicion of a pelvic fracture, it is simple to request that the abdominal CT be extended distally to include the pelvis. A CT of a fractured vertebra will provide the information needed to classify the fracture as stable or unstable and determine whether operative treatment is needed.

#### Intravenous Pyelography

There is a strong correlation of urologic injury with anterior pelvic fractures, as well as with liver and spleen injury. Although CT and ultrasonography are used to evaluate renal injuries, the intravenous pyelogram still has a role in helping to diagnose bladder and urethral injuries ( [61](#)).

### **Radionuclide Scans**

Bone scans have a limited role in the acute evaluation of a child with multiple injuries. In conjunction with a skeletal survey, a technetium-99m bone scan is routinely used in children with suspected child abuse to detect previously undetected new or old fractures. Heinrich et al. ( [36](#)) reported that bone scans in 48 children with multiple injuries often demonstrated an unsuspected injury. Nineteen previously unrecognized fractures were identified by obtaining radiographs of the areas with increased isotope uptake. Of their 48 patients, six had a change in their orthopaedic care as a result of this bone scan, although this treatment was usually simple cast application of a nondisplaced fracture. Nonetheless, the bone scan can be a valuable screening tool in a child with multiple injuries from any cause. In some instances, the bone scan can be useful to differentiate a normal variation in skeletal ossification (normal uptake) from a fracture (increased uptake), particularly in an extremity or a spinal area where pain is present.

### **Magnetic Resonance Imaging**

Magnetic resonance imaging (MRI) is used primarily for the detection of injury to the brain or the spine and the spinal cord. In young children, the bony spine is more elastic than the spinal cord. As a result, a spinal cord injury can occur without an obvious spinal fracture in children with multiple injuries, particularly in automobile accidents ([4,24](#)). In the SCIWORA (spinal cord injury without radiographic abnormality) syndrome, MRI is valuable in demonstrating the site and extent of spinal cord injury and in defining the level of injury to the disks or vertebral apophysis. A fracture through the vertebral apophysis is similar to a fracture through the physis of a long bone and may not be obvious on planar radiographs.

MRI also is very useful in evaluating knee injuries, particularly when a bloody knee effusion is present. If blood is present on knee arthrocentesis, MRI can assist in diagnosing an injury to the cruciate ligaments or menisci. In addition, a chondral fracture that cannot be seen on routine radiographs may be demonstrated by MRI.

### **Ultrasonography**

Ultrasound evaluation has been shown to be an accurate means of detecting hemoperitoneum following injury. Some trauma centers have replaced peritoneal lavage and laparoscopy with serial ultrasound evaluations to monitor liver, spleen, pancreas, and kidney injury in children with multiple injuries ( [13,38,71](#)). One of the problems with ultrasonography is the operator-dependent nature of this imaging study. As a result, CT is more often used for assessment and monitoring of visceral injury in children sustaining multiple injuries, and a recent comparison of CT and ultrasonography demonstrated the superiority of CT for diagnosing visceral injury in children with polytrauma ([70](#)).

## **NONORTHOPAEDIC CONDITIONS IN THE MULTIPLY INJURED CHILD**

### **Head Injury**

#### **Prognosis for Recovery**

Head injuries occur in children with multiple injuries even more often than orthopaedic injuries. It has been clearly demonstrated that a child recovers more quickly and more fully from a significant head injury than does an adult ( [18,50,93](#)). Even children who are in a coma for several hours to several days often recover full motor function. Mild cognitive or learning deficits may persist however, so educational testing needs to be considered for children who have had head injury and coma. The two factors that have been identified to produce poorer functional recovery and more severe permanent neurologic deficits are a low oxygen saturation level at the time of presentation to the emergency department or trauma center and a low GCS score 72 hours after the head injury.

Despite the fact that excellent motor recovery is expected in most children after a head injury, Greenspan and MacKenzie reported that 55% of children in their series had one or more health problems at 1-year follow-up, many of which were relatively minor ( [29](#)). Headaches were present in 32% and extremity complaints in 13% of patients. The presence of a lower extremity injury with a head injury led to a higher risk of residual problems.

Because of the more optimistic outlook for children with head injuries than for adults with similar injuries, orthopaedic care must be provided in a timely way, and the orthopaedist must base the orthopaedic care on the assumption that full neurologic recovery will ensue. Waiting for a child to recover from a coma is not appropriate, and comatose children tolerate general anesthesia well. The orthopaedic injury must be treated in the same way that it would be in an alert injured child to obtain the best outcome. Unless the musculoskeletal injuries are treated with the assumption that full neurologic recovery will take place, long bone fractures may heal in angled or shortened positions. Once neurologic recovery occurs, the primary functional deficit will then be from ill-managed orthopaedic injuries rather than from the neurologic injury.

### **Intracranial Pressure**

After a head injury, intracranial pressure measurements are commonly monitored to prevent excessive intracranial pressure, which may lead to further permanent disability or death. Normally, intracranial pressure does not exceed 15 mm Hg, and all attempts should be made to keep this pressure under 30 mm Hg after a head injury.

This is accomplished by elevating the head of the bed to 30 degrees, lowering the pCO<sub>2</sub>, and restricting intravenous fluid administration. Ventilator assistance is used to lower the pCO<sub>2</sub>, which, in turn, helps lessen cerebral edema. If the other injuries allow and adequate blood pressure is sustained, fluid restriction is also preferred to lessen the cerebral edema. Elevation of serum norepinephrine has been shown to correlate well with the severity of head injury in patients with injury of multiple organ systems ([94](#)).

Motion at the site of a long bone fracture will cause an elevation of the intracranial pressure in children with multiple injuries. Because of this problem, long bone fractures must be immobilized to limit fracture motion until definitive fracture care can be provided. Initial immobilization usually is accomplished by splinting or casting of the fractures, or by use of traction for femoral shaft fractures. The use of external or internal fixation of fractures should be strongly considered to help control elevation of intracranial pressure. Fracture stabilization also aids in dressing changes for the treatment of adjacent soft tissue injury as well as allowing in-hospital transport for imaging studies and other necessary treatments ([84,85](#)).

### **Secondary Orthopaedic Effects of Head Injuries**

A head injury can have later impact on the management of musculoskeletal injuries, even after the acute phase has passed. Persistent spasticity, the development of contractures, heterotopic bone formation in soft tissue, and changes in fracture healing rates are all seen in children with sequelae of a head injury.

**Spasticity.** Spasticity may develop within a few days of head injury. The early effect of this spasticity is to cause shortening at the sites of long bone fractures if traction or splint or cast immobilization is being used. If fracture displacement or shortening occurs in a circumferential cast, the bone ends may cause pressure points between the bone and the cast, leading to skin breakdown at the fracture site, with a higher risk for deep infection. Even with skeletal traction for femoral fractures, fracture shortening and displacement will occur as the spasticity overcomes the traction forces. Once spasticity develops and long bone fractures displace, internal or external fixation is needed to maintain satisfactory reduction. This operative stabilization should be performed as soon as the spasticity becomes a problem for fracture reduction because fracture healing is accelerated by a head injury ( [83,84](#) and [85](#)).

**Contractures.** The persistence of spasticity in the extremities often leads to subsequent contractures of the joints spanned by the spastic muscles. Contractures can arise quite quickly, and early preventative stretching or splinting should begin while the child is in the intensive care unit. Nonselective mass action muscle activity associated with brain injury can be used to help prevent these early contractures. If the child lies in bed with the hips and knees extended, there will usually be a strong plantarflexion of the feet at the ankles from the spasticity. If the hip and knee are placed in a flexed position, it will be much easier to dorsiflex the foot at the ankle, so positioning in this way will prevent early equinus contractures from developing so quickly. Stretching and splinting can often be effective in preventing

contractures, but if these measures are not successful, there should be no hesitation to treat these contractures surgically if they are interfering with subsequent rehabilitation.

**Heterotopic Bone Formation.** Heterotopic bone may form in the soft tissues of the extremity as early as a few weeks after a head injury with persistent coma. Usually, this is in the vicinity of the hip or elbow but may occur elsewhere as well. There is some evidence that heterotopic bone formation can be stimulated by surgical incisions. In head-injured teenagers who undergo antegrade reamed femoral intramedullary nailing of femoral fractures, heterotopic bone often forms at the nail insertion site that later restricts hip motion ( 41). Obtaining weekly serum alkaline phosphatase levels on a child with persistent coma may allow early detection of the heterotopic bone, although alkaline phosphatase is also elevated during healing of fractures. A sudden increase of alkaline phosphatase a few weeks after the onset of coma, even with fractures co-existing, may mean that heterotopic bone is starting to form and a more careful examination of the extremities is in order ( 59). Technetium-99 bone scans show increased isotope uptake in the soft tissue where heterotopic bone forms, and this imaging study should be considered if new swelling is noted in the extremity of a comatose child. Other diagnoses that must be considered in a comatose child with new swelling of the extremity are a new long bone fracture or a deep venous thrombosis (79).

Observation and excision are the two primary approaches taken in managing heterotopic bone formation in an injured child. If the child remains comatose, usually little treatment is administered. There is no conclusive data to support medical treatment if an early diagnosis of heterotopic bone formation is made. However, it may be useful to try to block some of the heterotopic bone formation by use of salicylates or nonsteroidal antiinflammatory medication once an early diagnosis is established. If the child has recovered from the head injury and has heterotopic bone that does not interfere with rehabilitation, surgical excision is not warranted. If there is significant restriction of joint motion from the heterotopic bone, this bone should be excised to facilitate rehabilitation. The timing of the heterotopic bone excision is somewhat controversial, but current thinking leans toward resection whenever heterotopic bone significantly interferes with rehabilitation, rather than waiting for 12 to 18 months until the bone is more mature. After surgical excision, it is essential to use salicylates or nonsteroidal antiinflammatory drugs immediately after the excision and for several weeks thereafter to block new heterotopic bone formation at the operative site. Mital et al. ( 59) reported success in preventing recurrence of heterotopic bone after excision by use of salicylates at a dosage of 40mg/kg/day in divided doses for 6 weeks postoperatively.

**Fracture Healing Rates.** For reasons that are not entirely clear, long bone fractures heal more quickly in children and adults who have associated head injuries. It has been demonstrated that polytrauma patients in a coma have a much higher serum calitonin level than do conscious patients with similar long bone fractures, but how or whether this finding influences fracture healing is still unclear ( 22).

### Peripheral Nerve Injuries

Although persistent neurologic deficits in a child who has multiple injuries usually are sequelae of a head injury, peripheral nerve injury should be carefully assessed as the rehabilitation process proceeds. In one clinical review of brain-injured children, 7% had evidence of an associated peripheral nerve injury documented by electrodiagnostic testing ( 66). The peripheral nerve injury most often is associated with an adjacent fracture or with a stretching injury of the extremity. It is important to recognize these injuries, because surgical peripheral nerve repair with nerve grafts offers excellent chances of nerve function recovery in young patients, if the nerve injury does not recover on its own.

### Abdominal Trauma

Abdominal viscera, both solid and hollow, are at high risk of significant injury in children with multiple skeletal injuries. Abdominal swelling, tenderness, or bruising are all signs of injury. CT or ultrasonography evaluation has largely displaced peritoneal lavage or laparoscopy as the initial method of evaluation of abdominal injury. Abdominal injury is not unusual if a child in an accident has been wearing a lap seat belt ( 87). Bond et al. (10) noted that the presence of multiple pelvic fractures strongly correlated (80%) with the presence of abdominal or genitourinary injury, whereas the child's age or mechanism of injury had no such correlation with abdominal injury rates. The usual practice is to treat hepatic and splenic lacerations nonoperatively, by monitoring the hematocrit, by repeating the abdominal examination frequently, and by serial CT scans or ultrasound examinations ( 15,17). Once the child's overall condition has stabilized, the presence of abdominal injuries that are being observed should not delay the fracture care as long as the child is stable enough to undergo general anesthesia.

### Genitourinary Injuries

Injuries to the genitourinary system generally occur in conjunction with pelvic fractures. Most injuries to the bladder and urethra are associated with fractures of the anterior pelvic ring ( 5). The injury is usually at the bulbourethra, but the bladder, prostate, and other portions of the urethra can also be injured. If the injury is severe, kidney injury may also occur, but most urologic injuries that occur with pelvic fractures are distal to the ureters ( 1).

Tears of the vagina and resultant vesicovaginal fistulae may be associated with displaced fractures of the anterior pelvic ring. If the iliac wings are displaced or the pelvic ring shape is changed, it is important to reduce these fractures to reconstitute the birth canal in female patients. There are increased rates of caesarean section in young women who have had a pelvic fracture compared with those without this injury ( 19).

It is important to inform adolescent females with displaced pelvic fractures of this later potential problem with vaginal delivery.

### Fat Embolism

Although fat embolism and acute respiratory distress syndrome are relatively common in adults with multiple long bone fractures, they are rare in young children and uncommon in teenagers (51). When they are present, the signs and symptoms are the same as in adults: axillary petechiae, hypoxemia, and radiographic changes of pulmonary infiltrates appearing within several hours of the fractures ( 30). It is likely that some degree of hypoxemia develops in some children after multiple fractures, but the full clinical picture of fat embolism seldom develops. If a child with multiple fractures but without a head injury develops a change in sensorium and orientation, hypoxemia is most likely the cause, and arterial blood gases are essential to determine the next step in management. The other primary cause of mental status change after fractures is overmedication with narcotics for pain control.

If fat embolism is diagnosed by low levels of arterial oxygenation, the treatment is the same as in adults. Usually, this management consists of endotracheal intubation, positive pressure ventilation, and hydration with intravenous fluid. The effect of early fracture stabilization, intravenous alcohol, or high-dose corticosteroids on the syndrome of fat embolism has not been studied well in children with multiple injuries.

### Nutritional Requirements

Multiple injuries place large caloric demands on the body. If an injured child requires ventilator support for several days, caloric intake via a feeding tube or a central intravenous catheter is necessary to improve healing and help prevent complications from developing. The baseline caloric needs of a child can be determined based on the weight and age of the child. Children on mechanical ventilation in a pediatric intensive care unit have been shown to require 150% of the basal energy or caloric requirements for age and weight ( 82). The daily nitrogen requirement for a child in the acute injury phase is 250 mg/kg.

## ORTHOPAEDIC MANAGEMENT OF THE MULTIPLY INJURED CHILD

### Timing

Because fractures are rarely life-threatening in children with multiple system injuries, splinting of the fractures will generally suffice as the initial orthopaedic care needed, while the child's overall condition is stabilized. When is the optimal time for the definitive treatment of the fracture or fractures and what is that optimal treatment? Should the child have all fractures treated operatively to allow mobilization out of bed, as is commonly recommended in adults with multiple fractures, or is a combination of operative and nonoperative management more appropriate? Are there times when implants used in adults can also be used in children, or should separate pediatric implants be used when operative treatment is chosen? How rigid does fracture fixation need to be in children?

### Pelvic Fractures

Fractures of the pelvis are common in children and adolescents with multiple injuries. The central injuries to the spine and pelvis have been reported to be associated

with the most intense hospital care and higher mortality rates than other injury combinations ( 12). The immediate problem often is control of bleeding, either from the retroperitoneum near the fracture or from the peritoneum from injured viscera ( 40). However, death of children with pelvic fractures appears to be caused more often by an associated head injury rather than an injury to the adjacent viscera or vessels ( 60).

The fractures of the anterior pelvic ring are the primary cause of urethral injury ( 1,5). Bilateral anterior and posterior pelvic fractures are most likely to cause severe bleeding (57), although death from blood loss in children is uncommon ( 60). Injury to the sciatic nerve or the lumbosacral nerve roots may result from hemipelvis displacement through a vertical shear fracture. In fact, nonorthopaedic injuries associated with pelvic fractures led to long-term morbidity or mortality in 30% of patients (11 of 36) in one published review of pediatric pelvic fractures ( 26).

Most pelvic fractures in children are treated nonoperatively. In a child or preadolescent, an external fixator can be used to close a marked pubic diastasis or to control bleeding by stabilizing the pelvis for transport and other injury care. The external fixator will not reduce a displaced vertical shear fracture, but the stability provided is helpful to control the hemorrhage while the child's condition is stabilized ( 69,86).

## Open Fractures

**Background.** Most serious open fractures in children result from high-velocity blunt injury involving vehicles. However, many low-energy blunt injuries can cause puncture wounds in the skin adjacent to fractures, especially displaced radial, ulnar, and tibial fractures. It has been estimated that in children with multiple injuries, about 10% of the fractures are open. When open fractures are present, 25% to 50% of patients have additional injuries involving the head, chest, abdomen, and other extremities.

**Wound Classification.** The classification used to describe the soft tissues adjacent to an open fracture is based primarily on the system described by Gustilo and Anderson (30,31). Primary factors that are considered and ranked in this classification system are the size of the wound, the degree of wound contamination, and the presence or absence of an associated vascular injury ( Table 4-4).

Type I	An open fracture with a wound <1 cm long and clean
Type II	An open fracture with a laceration >1 cm long without extensive soft-tissue damage, flaps, or avulsions
Type III	Massive soft tissue damage, compromised vascularity, severe wound contamination, marked fracture instability
Type IIIA	Adequate soft tissue coverage of a fractured bone despite extensive soft tissue laceration or flaps, or high-energy trauma irrespective of the size of the wound
Type IIIB	Extensive soft-tissue injury loss with periosteal stripping and bone exposure; usually associated with massive contamination
Type IIIC	Open fracture associated with arterial injury requiring repair

Adapted from Gustilo RB, Mendoza RM, Williams DN. Problems in the management of type III (severe) open fractures: a new classification of type III open fractures. *J Trauma* 1984;24:742-746; Gustilo RB, Anderson JJ. Prevention of infection in the treatment of 1025 open fractures of long bones: retrospective and prospective analyses. *J Bone Joint Surg Am* 1976;58:453-456, with permission.

**TABLE 4-4. CLASSIFICATION OF OPEN FRACTURES**

*Type I.* Type I fractures usually result from a spike of bone puncturing the skin (inside-out). The wound is less than 1 cm in size, and there is minimal local soft tissue damage or contamination.

*Type II.* A type II wound is generally larger than 1 cm and is associated with a transverse or oblique fracture with minimal comminution. There is adjacent soft tissue injury, including skin flaps or skin avulsion and a moderate crushing component of adjacent soft tissue is usually present.

*Type III and Subgroups.* The most severe open fractures are classified as type III, with associated subgroups A, B, or C, with the letters indicating increasing severity of injury. These fractures typically result from high-velocity trauma and are associated with extensive soft tissue injury, a large open wound, and significant wound contamination. In a type IIIA fracture, there is soft tissue coverage over the bone, which often is a segmental fracture. In a type IIIB fracture, bone is exposed at the fracture site, with treatment typically requiring skin or muscle flap coverage of the bone. Type IIIC fractures are the most severe and, in addition to extensive soft tissue loss and contamination, have an injury to a major artery in that segment of the extremity.

This classification is widely used and has been shown to correlate in adults with sequelae of the injury, including the potential for infection, delayed union, nonunion, amputation, and residual impairment. Probably due to the better vascular supply to the extremities of children, the final functional results of type III fractures in children appear to be superior to results after similar fractures in adults.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

**Three Stages.** I consider the treatment of open fractures in children to be similar to that for open fractures in adults. The primary goals are to prevent infection of the wound and fracture site, while allowing soft tissue healing, fracture union, and eventual return of optimal function. My initial emergency care includes the so-called ABCs of resuscitation, application of a sterile povidone-iodine (Betadine) dressing, and preliminary alignment and splinting of the fracture for patient transport. If profuse bleeding is present, a compression dressing is applied to limit blood loss. In the emergency room, masks and gloves should be worn as each wound is thoroughly inspected. Tetanus prophylaxis is provided, and the initial dose of intravenous antibiotics is given. The dose of tetanus toxoid is 0.5 mL intramuscularly to be given if the patient's immunization status is unknown, or if it is more than 5 years since the last dose. The second stage of management is the primary surgical treatment, including initial and (if necessary) repeat debridement of the tissues in the area of the open fracture until the entire wound appears viable. The fracture is reduced and stabilized. If the bone ends are not covered with viable soft tissue, muscle or skin flap coverage is considered. My third and final stage of this management is bony reconstruction as needed if bone loss has occurred and, ultimately, rehabilitation of the child.

**Cultures.** The role of cultures obtained in the emergency room is controversial. Cultures in this setting are probably of little use in future management of the open fracture. The cultures for characterizing the bacterial contamination present are better obtained in the operating room at the time of debridement ( 45).

**Antibiotic Therapy.** Antibiotic therapy decreases the risk of infection in children with open fractures. Wilkins and Patzakis ( 92) reported a 13.9% infection rate in 79 patients who received no antibiotics after open fractures, whereas there was a 5.5% infection rate in 815 patients with similar injuries who had antibiotic prophylaxis. Bacterial contamination has been noted in 70% of open fractures in children, with both gram-positive and gram-negative infections noted, depending on the degree of wound contamination and adjacent soft tissue injury. I limit antibiotic administration generally to 72 hours after surgical treatment of the open fracture.

For all type I and some type II fractures, I use a first-generation cephalosporin. For type III and some more severe type II fractures, I use a combination of a cephalosporin and aminoglycoside (cephalothin 150 mg/kg/day q8h, and gentamicin). For farm injuries or grossly contaminated fractures, penicillin is added to the cephalosporin and aminoglycoside. All antibiotics are given intravenously and for 72 hours. Oral antibiotics are occasionally used if significant soft tissue erythema at the open fracture site remains after the intravenous antibiotics have been completed.

I continue the antibiotic regimen beyond 48 to 72 hours if there is (a) delayed wound closure, (b) open reduction and internal fixation of fractures, and (c) secondary bone reconstruction procedures.

**Debridement and Irrigation.** I consider debridement and irrigation of the open fracture in the operating room to be the most important step in the primary management of open fractures in children. Some authors have reported that significantly higher infection rates occurred if debridement and irrigation were done more than 6 hours after open fractures in children ( 46). A recent report, however, demonstrated an overall 1% to 2% infection rate after open long bone fractures, with no increased rate in infection if the debridement was delayed even as long as 24 hours ( 76). I believe that it is important to perform the debridement and irrigation as quickly as is feasible, but a modest delay may not lead to severe consequences or chronic infection. The debridement needs to be performed carefully and

systematically to remove all foreign and nonviable material from the wound. My order of debridement typically is

1. Excision of the necrotic tissue from the wound edges
2. Extension of the wound to adequately explore the fracture ends
3. Debridement of the wound edges to bleeding tissue
4. Resection of necrotic skin, fat, muscle, and contaminated fascia
5. Fasciotomies as needed
6. Thorough pulsatile irrigation of the fracture ends and wound

Because secondary infection in ischemic muscle can be a major problem in wound management and healing, I ensure that all ischemic muscle is widely debrided back to muscle, which bleeds at the cut edge and contracts when pinched with the forceps.

When I am debriding and irrigating an open diaphyseal fracture, I always bring the proximal and distal bone ends into the wound for visual inspection. This often means that the open wound needs to be extended somewhat, but that is preferable to leaving the fracture site contaminated. I carefully remove devitalized bone fragments and contaminated cortical bone with curettes or a small rongeur. If there is a possibly nonviable bone fragment, judgment is needed as to whether this bone fragment should be removed or left in place. My experience is that reconstruction of a large segmental bone loss has a better outcome in children than in adults, because children have a better potential for bone regeneration and have better vascular supply to their extremities. As with all diaphyseal debridements, I identify and protect major neurovascular structures in the area of the fracture. I consider the debridement complete when all contaminated, dead, and ischemic tissues have been excised; the bone ends are clean with bleeding edges; and only viable tissue lines the wound bed.

I usually use a pulsed lavage system to irrigate the open fracture with 10 L of sterile normal saline. In the past, I have used bacitracin and polymyxin antibiotics in this irrigation solution but irrigate only with the normal saline. The antibiotic addition is more costly, and I believe that the key is local irrigation and debridement to allow the intravenous antibiotics to reach the wound and control the infection. I obtain cultures from the depths of the wound near the fracture ends just before the irrigation. If repeat debridement is needed, cultures are again obtained and are often useful in guiding the final antibiotic coverage used.

After the debridement and irrigation are complete, I try to use the local soft tissue to cover the neurovascular structures, the tendons, and the bone ends. If local soft tissue coverage is inadequate, consideration should be given to local muscle flaps or other coverage methods. The area of the wound that has been incised to extend the wound for fracture inspection can be primarily closed with interrupted nylon sutures. The remaining wound that is open is dressed with a moistened Betadine dressing, which is changed the following day. If the wound appears clean, sequential wet-to-dry saline dressings are used until wound healing occurs. If the wound does not appear clean at 48 hours, repeat debridement in the operating room is carried out. This cycle is repeated until the wound can be sutured closed or has a split thickness skin graft or local flap to cover it.

**Fracture Stabilization.** Fracture stabilization in children with open fractures decreases pain, protects the soft tissue envelope from further injury, decreases the spread of bacteria, allows stability important for early soft tissue coverage, and improves the fracture union rate.

In general terms, my principles for stabilization of open fractures in children include allowing access to the soft tissue wound and the extremity to allow for debridement and dressing changes, allowing weight bearing when appropriate and preserving full motion of the adjacent joints to allow full functional recovery.

I often use casts or splints to stabilize type I fractures and occasionally type II fractures with relatively small wounds and minimal soft tissue involvement. Most of these injuries involve the radius or ulna in the upper extremity or the tibia in the lower extremity. In the forearm, a flexible intramedullary implant in either the radius or ulna or both, provides enough stability of the fracture to allow dressing changes through the cast or splint. Splint or cast immobilization generally is not satisfactory for the more unstable type II and most type III injuries.

For intramedullary fixation, I prefer flexible titanium implants of 2 mm to 4 mm diameter for stabilizing open fractures in the forearm when reduction of either the radius or ulna fracture is unstable. The ulnar implant is inserted proximally, whereas the radial implant is inserted just proximal to the distal radial physis. One or both bones can be stabilized, and the implants removed easily after fracture healing. I also use these flexible intramedullary nails more often for type I and some type II fractures of the femoral shaft. For type III fractures, especially if there is a large or contaminated soft tissue wound present, I still prefer to use external fixation.

External fixation is my treatment of choice for most type II and type III fractures of the tibia and femur in children. The benefits of external fixation include easy access to the wound for debridement and dressing changes plus any soft tissue or bone reconstruction needed. External fixation allows patient transport around the hospital for other reasons associated with the multiple injuries. External fixation preserves the length of the long bone at the appropriate level and allows weight bearing relatively soon after the injury. I find that a uniplanar frame is best for most fractures and is relatively easy to apply. For some segmental fractures in the metaphysis and diaphysis, as well as soft tissue injuries, the Ilizarov device may be a better choice.

I use open reduction and internal fixation for open intraarticular fractures. If the fracture involves the physis, I avoid threaded pins or screws across the physis and use smooth Steinmann pins for stabilization, if needed. For fractures that involve the metaphysis and diaphysis, I may combine open reduction and internal fixation with external fixation. For diaphyseal fractures in preteens, I prefer flexible intramedullary nails to compression plates for internal fixation of type I and type II fractures. For treatment of a *floating joint*, usually the knee or elbow, I carry out operative stabilization of at least one and usually both fractures ([9,49](#)).

**Wound Management.** I prefer to provide soft tissue coverage of the open fracture and adjacent soft tissue defect by 5 to 7 days after the injury. The wound is debrided every 48 hours until it is clean, if the initial dressing change indicates residual necrotic tissue. In the lower extremity, the fractures are externally fixed when appropriate, and I attempt to obtain early soft tissue coverage to limit the risk of later infection. Most type I wounds heal with local dressing changes. For some type II and type IIIA fractures, I use delayed wound closure or a split-thickness skin graft over underlying muscle cover.

Large soft tissue loss is most often a problem with types IIIB and IIIC fractures. In the proximal tibia area, I often work with the plastic surgeons to provide a gastrocnemius rotational flap, followed by secondary coverage of the muscle with a skin graft. In the middle third of the leg, a soleus flap is used with skin graft coverage, whereas a vascularized free muscle transfer is necessary if local coverage is inadequate.

The flaps and grafts I use for reconstructing severe injuries are either muscle flaps or composite grafts. For a massive loss of soft tissue and bone, composite grafts of muscle and bone often are necessary. The younger the child the better the likelihood that autogenous graft will fill in a bone defect if there is a well-vascularized bed from the muscle flap. Free flaps, especially from the latissimus dorsi, are useful in the mid-tibial and distal tibial regions to decrease infection rates and improve the union rates. Vascularized fibular grafts rarely are used acutely to reconstruct bone defects but may be useful in later reconstruction, after soft tissue wounds are healed.

For the rare bone loss defect in a child, I rely on the healing capacity of young periosteum and bone and the vascular supply of a child's extremity. An external fixator is used to hold the bone shortened about 1 to 2 cm to decrease the size of the bone loss. In a growing child, 1 to 2 cm of overgrowth can be expected in the subsequent 2 years after these severe injuries so the final leg length will be satisfactory. Autogenous bone graft can be used early, but if there is surviving periosteum at this site, I have been surprised by how much new bone the child has formed, sometimes to the extent that bone grafting is unnecessary. In teenagers with bone loss, once the soft tissue has healed, bone transport using either a uniplanar lengthening device or an Ilizarov device is my preferred method of reconstruction, although use of an allograft or vascularized fibular graft may be considered.

**Amputation.** In the most severe of open fractures, attempts should generally be made to preserve all extremities, even with those type IIIC open fractures that are usually treated with primary amputation in adults. Wounds and fractures that do not heal in adults often heal satisfactorily in children and preservation of limb length and physis are important in young children. If amputation is absolutely necessary, as much length as possible should be preserved. For example, if the proximal tibial physis is preserved in a child with a short proximal tibial stump at age 7 years, 3 to 4 inches more growth of the tibial stump can be expected by the time skeletal maturity is reached. As a result, this below-knee amputation would likely be superior in final function to a knee disarticulation done at the time of injury.

Although amputations performed to treat congenital limb deficits are usually done through the joint to limit bone spike formation at the end of the stump, I prefer to maintain maximal possible length if amputation becomes necessary as a result of a severe injury.

**Management of Other Fractures.** When a child with an open fracture is brought to the operating room for irrigation and debridement of the open fracture, the

orthopaedist should take advantage of the anesthetic and treat the other fractures as well, whether operative treatment or closed reduction and casting is needed.

## STABILIZATION OF FRACTURES

### Beneficial Effects

In addition to promoting fracture healing, fracture stabilization also provides a number of nonorthopaedic benefits to a child with multiple injuries. Pulmonary contusions at the time of injury often lead to increasing respiratory problems in the first few days after injury ( 65). If the lungs have been severely contused, protein leaks into the alveolar spaces, making ventilation more difficult. Surfactant dysfunction follows and is most abnormal in patients with the most severe respiratory failure (67). As the time from the injury increases, pulmonary function deteriorates and general anesthesia becomes more risky. In patients with severe pulmonary contusions and multiple fractures, the use of extracorporeal life support may be the only treatment available to allow patient survival ( 74).

In adults with multiple injuries, early operative stabilization of fractures decreases pulmonary and other medical complications associated with prolonged bed rest that is a part of nonoperative fracture treatment ( 6). Most adult trauma centers follow the treatment protocol of early fracture stabilization, even though Poole et al. ( 68) reported that, despite early fracture stabilization simplifying patient care, pulmonary complications in patients with marked chest trauma were not prevented and the course of the head injury was not affected. In children, medical complications are less common so the recommendations to mandate early fracture stabilization are somewhat more difficult to support in the young patients. Nonetheless, bruises on the chest or rib fractures should alert the orthopaedist to potential pulmonary contusions as a part of the injury complex (64). Initial chest radiographs may not clearly demonstrate the degree of pulmonary parenchymal injury, and arterial blood gas determinations are more useful in estimating the anesthetic risk of these patients during operative care of the fractures.

**Timing.** In a child with multiple closed fractures, splinting is needed at the time of the initial resuscitation. Definitive treatment should proceed expeditiously once the child's condition has been stabilized. Loder ( 52) reported that in 78 children with multiple injuries, early operative stabilization of fractures within the first 2 or 3 days after injury led to a shorter hospital stay, a shorter stay in the intensive care unit, and a shorter time on ventilator assistance was needed. In addition, there were fewer complications in those who underwent surgical treatment of the fractures less than 72 hours after injury. Although there may have been other factors besides the timing of surgery that affected the eventual outcomes in this study, it would seem prudent to try to complete the fracture stabilization within 2 to 3 days from the time of injury.

**Operative Fixation.** The type of operative stabilization of closed fractures of long bones in multiply injured children commonly depends, as in other orthopaedic arenas, on the training, experience, and personal preference of the orthopaedist. The most common methods used are intramedullary rod fixation, external fixation, and AO compression plating, though Kirschner wires or Steinmann pins may be used in conjunction with casts.

**Intramedullary Rod Fixation.** There has been a recent increase in the use of flexible intramedullary rods of 2- to 4-mm diameter for stabilization of unstable closed fractures of the radius and ulna in patients up to the early teenage years and for stabilization of closed femoral shaft fractures in patients between the ages of 5 and 11 years (85,90).

Forearm fractures generally can be reduced closed, with the intramedullary implant passed across the fracture site under fluoroscopy to stabilize the fracture ( 48). The ulnar implant is placed from proximal to distal and is inserted in the lateral proximal metaphyseal area. The radial implant is contoured before insertion in the radial aspect of the distal radius, just proximal to the distal radial physis. Stability of both fractures may be achieved by instrumenting only the radius or the ulna, but both bones may require implant fixation. A cast is used for further immobilization. The implants are easily removed from the wrist area and the elbow region 6 to 12 months after insertion and after fracture healing has taken place.

Despite the utility of flexible intramedullary implants for stabilizing forearm fractures in children, the radius and ulna in young patients have significant remodeling capacity and not all fractures require anatomic reduction. A closed reduction and cast immobilization often is sufficient treatment. Complications, including loss of reduction, infection, hardware migration, nerve injury, and delayed union, have been reported with the use of pediatric intramedullary implants in the forearm, though 95% of patients (19 of 20) had excellent or good results on follow-up ( 21).

If flexible intramedullary nails are used in the femur, the most common insertion site is the medial and lateral metaphyseal region of the distal femur, just proximal to the physis. Two rods are used to cross the fracture site and obtain purchase in the proximal femur, usually with one at the base of the femoral neck and the other at the base of the greater trochanter. A cast is not necessary postoperatively, although a fracture brace can be used to help control rotation at the fracture site and provide some patient comfort during early walking. The implants are removed before 1 year from the time of fracture fixation ( 35,39).

The use of reamed antegrade intramedullary rods to treat femoral shaft fractures in the pediatric population should be reserved, in my view, for those at least older than the age of 11 years who probably have closure of the proximal femoral physis. In younger children, rod insertion at the piriformis fossa may interfere with the vascular supply to the femoral epiphysis, may cause growth arrest of the greater trochanter apophysis with resultant coxa valga, or may interfere with the appositional bone growth at the base of the femoral neck, thereby thinning this region and potentially predisposing the child to a femoral neck fracture ( 14). The specific indications for intramedullary fixation of the femur are discussed in more detail in [Chapter 22](#).

**Compression Plates.** Some have advocated the use of compression plates to stabilize long bone fractures, especially in the femoral shaft, in children with multiple injuries. Kregor et al. (47) reported an average overgrowth of the femur of 9 mm, and all fractures healed in a near-anatomic position. The disadvantages of compression plating is the need for more extensive operative exposure at the site of the fracture and the usual need to remove the plate once healing is complete. In addition, refracture may occur through the screw holes left after plate removal if physical activity is resumed too quickly. Stiffness of adjacent joints is rarely a problem in children unless there has been an associated severe soft tissue injury. As a result, the number of cortices the screws cross on each side of the fracture may be fewer in children than in adults, because a cast or splint is routinely used in young patients.

Although some authors have recommended open reduction and compression plate fixation of displaced radial and ulnar fractures ( 95), I prefer flexible intramedullary nails, as noted earlier. The use of compression plates in the forearm requires a larger operative incision with the resultant scar and a second extensive procedure for plate removal. I do not believe that the healing capability of the young child requires the rigid fixation of compression plating to obtain fracture union.

**External Fixation.** The most common indications for use of external fixation in a child with multiple injuries include open fractures with significant soft tissue injury, fractures in association with a head injury and coma, and so-called floating knee fractures of the femur and tibia ( 3,8,9,43,49,69,73,84). The use of an external fixator in these circumstances allows the child to be transported for imaging studies or to the operating room for management of nonorthopaedic injuries. A unilateral fixator generally is sufficient to hold the fracture reduced in this age group.

When applying the external fixator, the pin sites should be predrilled and the pins placed in the operating room under fluoroscopic control. The caliber of the pin should be less than 30% of the diameter of the bone into which it is to be inserted. The distal and proximal pin sites must be chosen at a level to avoid the physis, and I recommend leaving at least 1 to 2 cm between the pin and physis, partly to avoid any adverse effect on the physis should a pin track infection occur. The proximal tibial physis is more distal anteriorly below the tibial tubercle, and this area must be avoided or a recurvatum deformity of the proximal tibia and knee will result.

The external fixator usually is left in place until fracture healing is complete, but it can be removed once the reason for placement has resolved (such as waking from coma or healing of skin wound) (23,84). If the fixator is removed early, a walking cast is applied. Transverse open fractures reduced out to length take longer to heal than do oblique fractures reduced with slight overlap. Once the fixator is removed, there is a risk of refracture that varies widely among the published reports. When a rigid transfixion type of fixator was used, a 21% refracture rate was noted ( 83). When a more flexible unilateral frame was used, the refracture rate was lower ( 3,8). A recent report indicated that if three of the four cortices at the fracture site appear to be healing on anteroposterior and lateral radiographs of the fracture, the refracture rate after frame removal should be small (77).

### Outcomes of Treatment of the Multiply Injured Child

In one review of 74 children with multiple injuries, 59 (80%) survived, but after 1 year, 22% were disabled, mainly from a brain injury. At 9 years after the injuries, 12% had significant physical disability, whereas 42% had cognitive impairment. In this group, however, the SF-36 or functional outcome survey did not differ from the control population. The best predictor of long-term disability was the Glasgow Outcome Scale from 6 weeks after injury on ( 88). In a shorter term review, it has been

demonstrated that 6 months after injury 8% to 19% of injured children and adolescents had some significant limitation ( 27).

Whichever method of fracture treatment—operative or nonoperative—is chosen for a child with multiple injuries, it is important that the orthopaedist be involved in the care of the child from the start. While recognizing the need to care for the other organ system injuries the child has sustained, it is important to advocate for the expeditious and appropriate treatment of the fractures that are present. Failure to do so will leave the multiply injured child with musculoskeletal disability once healing of the other injuries occurs. After multiple injuries, the most common long-term problems relate to either sequelae of the head injury or of the orthopaedic injuries.

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## CHAPTER REFERENCE

1. Abou-Jaoude WA, Sugarman JM, Fallat ME, Casale AJ. Indicators of genitourinary tract injury or anomaly in cases of pediatric blunt trauma. *J Pediatr Surg* 1996;31:86–89.
2. Armstrong PF. Initial management of the multiply injured child: the ABCs. *Instruct Course Lect* 1992;41:347–350.
3. Aronson J, Tursky EA. External fixation of femur fractures in children. *J Pediatr Orthop* 1992; 12:157–163.
4. Aufdemaur M. Spinal injuries in juveniles: necropsy findings in 12 cases. *J Bone Joint Surg Br* 1974;56:513–519.
5. Batislam E, Ates Y, Germiyanboglu C, et al. Role of tile classification in predicting urethral injuries in pediatric pelvic fractures. *J Trauma* 1997;42:285–287.
6. Beckman SB, Scholten DJ, Bonnell BW, Bukrey CD. Long bone fractures in the polytrauma patient: the role of early operative fixation. *Am Surg* 1989;55:356–358.
7. Bielski RJ, Bassett GS, Fideler B, Tolo VT. Intraosseous infusions: effects on the immature physis—an experimental model in rabbits. *J Pediatr Orthop* 1993;13:511–515.
8. Blasler RD, Aronson J, Tursky EA. External fixation of pediatric femur fractures. *J Pediatr Orthop* 1997;17:342–346.
9. Bohn WW, Durbin RA. Ipsilateral fractures of the femur and tibia in children and adolescents. *J Bone Joint Surg Am* 1991;73:429–439.
10. Bond SJ, Gotschall CS, Eichelberger MR. Predictors of abdominal injury in children with pelvic fracture. *J Trauma* 1991;31:1169–1173.
11. Brainard BJ, Slaughterbeck J, Benjamin JB. Fracture pattern and mechanisms in pedestrian-motor vehicle trauma: the ipsilateral dyad. *J Orthop Trauma* 1992;6:279–282.
12. Buckley SL, Gotschall C, Robertson WW Jr, et al. The relationships of skeletal injuries with trauma score, injury severity score, length of hospital stay, hospital charges, and mortality in children admitted to a regional pediatric trauma center. *J Pediatr Orthop* 1994;14:449–453.
13. Buess E, Illi OE, Soder C, Hanimann B. Ruptured spleen in children—15-year evolution in therapeutic concepts. *Eur J Pediatr Trauma* 1992;14:449–453.
14. Canale ST, Tolo VT: Fractures of the femur in children. *J Bone Joint Surg Am* 1995;77:294–315.
15. Canarelli JP, Boboyono JM, Ricard J, et al. Management of abdominal contusion in polytraumatized children. *Int Surg* 1991;76:119–121.
16. Cattell HS, Filtzer DL. Pseudosubluxation and other normal variations in the cervical spine in children. *J Bone Joint Surg Am* 1965;47:1295–1309.
17. Coburn MD, Pfeifer J, DeLuca FG. Nonoperative management of splenic and hepatic trauma in the multiply injured pediatric and adolescent patient. *Arch Surg* 1995;130:332–338.
18. Colombani PM, Buck JR, Dudgeon DL, et al. One-year experience in a regional pediatric trauma center. *J Pediatr Surg* 1985;20:8–13.
19. Copeland CE, Bosse MJ, McCarthy ML, et al. Effect of trauma and pelvic fractures on female genitourinary, sexual, and reproductive function. *J Orthop Trauma* 1997;11:73–81.
20. Cramer KE. The pediatric polytrauma patient. *Clin Orthop* 1995;318:125–135.
21. Cullen MC, Roy DR, Giza E, Crawford AH. Complications of intramedullary fixation of pediatric forearm fractures. *J Pediatr Orthop* 1998;18:14–21.
22. DeBastiani G, Mosconi F, Spagnol G, et al. High calcitonin levels in unconscious polytrauma patients. *J Bone Joint Surg Br* 1992;74:101–104.
23. Evanoff M, Strong ML, MacIntosh R. External fixation maintained until fracture consolidation in the skeletally immature. *J Pediatr Orthop* 1993;13:98–101.
24. Evans DL, Bethem D. Cervical spine injuries in children. *J Pediatr Orthop* 1989;9:563–568.
25. Garcia VF, Gotshall CS, Eichelberger MR, Bowman, LM. Rib fractures in children: a marker of severe trauma. *J Trauma* 1990;30:695–700.
26. Garvin KL, McCarthy RE, Barnes CL, Dodge BM. Pediatric pelvic ring fractures. *J Pediatr Orthop* 1990;10:577–582.
27. Gofin R, Adler B, Hass T. Incidence and impact of childhood and adolescent injuries: a population-based study. *J Trauma* 1999;47:15–21.
28. Greenberg MI. Falls from heights. *J Am Coll Emerg Phys* 1978;7:300–301.
29. Greenspan AI, MacKenzie EJ. Functional outcome after pediatric head injury. *Pediatrics* 1994;94:425–432.
30. Gustilo RB, Anderson JT. Prevention of infection in the treatment of 1,025 open fractures of long bones: retrospective and prospective analysis. *J Bone Joint Surg Am* 1976;58:453–458.
31. Gustilo RB, Mendoza RM, Williams DN. Problems in the management of Type III (severe) open fractures: a new classification of Type III open fractures. *J Trauma* 1984;24:742–746.
32. Guy J, Haley K, Zuspan SJ. Use of intraosseous infusion in the pediatric trauma patient. *J Pediatr Surg* 1993;28:158–161.
33. Haller JA Jr, Shorter N, Miller D, et al. Organization and function of a regional pediatric trauma center: does a system of management improve outcome? *J Trauma* 1983;23:691–696.
34. Harris BH. Creating pediatric trauma systems. *J Pediatr Surg* 1989;24:149–152.
35. Heinrich SD, Dvaric DM, Darr K, MacEwen GD. The operative stabilization of pediatric diaphyseal femur fractures with flexible intramedullary nails: a prospective analysis. *J Pediatr Orthop* 1994;14:501–507.
36. Heinrich SD, Gallagher D, Harris M, Nadell JM. Undiagnosed fractures in severely injured children and young adults: identification with technetium imaging. *J Bone Joint Surg Am* 1994;76:561–572.
37. Herzenberg JE, Hensinger RN, Dedrick DK, Phillips WA: Emergency transport and positioning of young children who have an injury of the cervical spine. *J Bone Joint Surg Am* 1989;71:15–22.
38. Hoffmann R, Nerlich M, Muggia-Sullam M, et al. Blunt abdominal trauma in cases of multiple trauma evaluated by ultrasonography: a prospective analysis of 291 patients. *J Trauma* 1992;32:452–458.
39. Huber RI, Keller HW, Huber PM, Rehm KE. Flexible intramedullary nailing as fracture treatment in children. *J Pediatr Orthop* 1996;16:602–605.
40. Ismail N, Bellemare JF, Mollitt DL, et al. Death from pelvic fracture: children are different. *J Pediatr Surg* 1996;31:82–85.
41. Keret D, Harcke HT, Mendez AA, Bowen JR. Heterotopic ossification in central nervous system-injured patients following closed nailing of femoral fractures. *Clin Orthop* 1990;256:254–259.
42. King J, Diefendorf D, Athorp J, et al. Analysis of 429 fractures in 189 battered children. *J Pediatr Orthop* 1988;8:585–589.
43. Kirschenbaum D, Albert MC, Robertson WW Jr, Davidson RS. Complex femur fractures in children: treatment with external fixation. *J Pediatr Orthop* 1990;10:588–591.
44. Knudson MM, Shagoury C, Lewis FR. Can adult trauma surgeons care for injured children? *J Trauma* 1992;32:729–737.
45. Kreder HJ, Armstrong PF. The significance of perioperative cultures in open pediatric lower-extremity fractures. *Clin Orthop* 1994;302:206–212.
46. Kreder HJ, Armstrong PF. A review of open tibia fractures in children. *J Pediatr Orthop* 1995;15:482–488.
47. Kregor PJ, Song KM, Routt ML, et al. Plate fixation of femoral shaft fractures in multiply injured children. *J Bone Joint Surg Am* 1993;75:1774–1780.
48. Lascombes P, Prevot J, Ligier JN, et al. Elastic stable intramedullary nailing in forearm fractures in children: 85 cases. *J Pediatr Orthop* 1990;10:167.
49. Letts M, Vincent N, Gouw G. The “floating knee” in children. *J Bone Joint Surg Br* 1986;68B:442–446.
50. Levin HS, High WM, Ewing-Cobb L, et al. Memory functioning during the first year after closed head injury in children and adolescents. *Neurosurgery* 1988;22:1043–1052.
51. Limbird TJ, Ruderman RJ. Fat embolism in children. *Clin Orthop* 1978;136:267–269.
52. Loder RT. Pediatric polytrauma: orthopaedic care and hospital course. *J Orthop Trauma* 1987;1:48–54.
53. MacKenzie EJ, Morris JA Jr, deLissovoy GV, et al. Acute hospital costs of pediatric trauma in the United States: how much and who pays? *J Pediatr Surg* 1990;25:970–976.
54. Magid D, Fishman EK, Ney DR, et al. Acetabular and pelvis fractures in the pediatric patient: value of two- and three-dimensional imaging. *J Pediatr Orthop* 1992;12:621–625.
55. Maio RF, Portnoy J, Blow FC, Hill EM. Injury type, injury severity, and repeat occurrence of alcohol-related trauma in adolescents. *Alcohol Clin Exp Res* 1994;18:261.
56. Maksoud JG, Moront ML, Eichelberger MR: Resuscitation of the injured child. *Semin Pediatr Surg* 1995;4:93–99.
57. McIntyre RC Jr, Bensard DD, Moore EE, et al. Pelvic fracture geometry predicts risk of life-threatening hemorrhage in children. *J Trauma* 1993;33:423–429.
58. Michaud LJ, Rivara FP, Grady MS, Reay DT. Predictors of survival and severity of disability after severe brain injury in children. *Neurosurgery* 1992;31:254–264.
59. Mital MA, Garber JE, Stinson JT. Ectopic bone formation in children and adolescents with head injuries: its management. *J Pediatr Orthop* 1987;7:83–90.
60. Musemeche C, Fischer RP, Cotler HB, Andrassy RJ. Selective management of pediatric pelvic fractures: a conservative approach. *J Pediatr Surg* 1987;22:538–540.
61. Onuora VL, Patil MG, al-Jasser AN. Missed urological injuries in children with polytrauma. *Injury* 1993;24:619–621.
62. Payiga VJ, Valentine RJ, Myeers SI, et al. Blunt pediatric vascular trauma: analysis of 41 consecutive patients undergoing operative intervention. *J Vasc Surg* 1994;20:419–424.
63. Pecllet MH, Newman KD, Eichelberger MR, et al. Patterns of injury in children. *J Pediatr Surg* 1990;25:85–90.
64. Pecllet MH, Newman KD, Eichelberger MR, et al. Thoracic trauma in children: an indicator of increased mortality. *J Pediatr Surg* 1990;25:961–965.
65. Pfenninger J. Pulmonary problems following multiple trauma in children. *Intensive Care Med* 1989;15(Suppl 1):950–952.
66. Philip PA, Philip M. Peripheral nerve injuries in children with traumatic brain injury. *Brain Injury* 1992;6:53–58.
67. Pison U, Seeger W, Buchhorn R, et al. Surfactant abnormalities in patients with respiratory failure after multiple trauma. *Am Rev Resp Dis* 1989;140:1033–1039.
68. Poole GV, Miller JD, Agnew SG, Griswold JA. Lower extremity fracture fixation in head-injured patients. *J Trauma* 1992;32:654–659.
69. Reff RB. The use of external fixation devices in the management of severe lower extremity trauma and pelvic injuries in children. *Clin Orthop* 1984;188:21–33.
70. Richardson MC, Hollman AS, Davis CF. Comparison of computed tomography and ultrasonographic imaging in the assessment of blunt abdominal trauma in children. *Br J Surg* 1997;84:1144–1146.
71. Roche BG, Bugmann P, LeCoultré C. Blunt injuries to liver, spleen, kidney, and pancreas in pediatric patients. *Eur J Pediatr Surg* 1992;2:154–156.
72. Rozycki GS, Maull KI. Injuries sustained by falls. *Arch Emerg Med* 1991;8:245–252.
73. Schranz PJ, Gultekin C, Colton CL. External fixation of fractures in children. *Injury* 1992;23:80–82.
74. Senunas LE, Goulet JA, Greenfield MVH, Bartlett RH. Extracorporeal life support for patients with significant orthopaedic trauma. *Clin Orthop* 1997;339:32–40.
75. Sivit CJ, Taylor GA, Newman KD, et al. Safety-belt injuries in children with lap-belt ecchymosis: CT findings in 61 patients. *AJR Am J Roentgenol* 1991;157:111–114.
76. Skaggs DL, Kautz S, Kay RM, Tolo VT. Effect of delay of surgical treatment on rate of infection in open fractures in children. *J Pediatr Orthop* 2000;20:19–22.
77. Skaggs DL, Leet AI, Money MD, et al. Secondary fractures associated with external fixation in pediatric femur fractures. *J Pediatr Orthop* 1999;19:582–586.
78. Smith JS Jr, Martin LF, Young WW, Macioce DP. Do trauma centers improve outcome over nontrauma centers: the evaluation of regional trauma care using discharge abstract data and

- patient management categories. *J Trauma* 1990;30:1533–1538.
79. Sobus KM, Sherman N, Alexander MA. Coexistence of deep venous thrombosis and heterotopic ossification in the pediatric patient. *Arch Phys Med Rehabil* 1993;74:547–551.
  80. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. *Lancet* 1974;2:81–84.
  81. Tepas JJ 3rd, Mollitt DL, Talbert JL, Bryant M. The Pediatric Trauma Score as a predictor of injury severity in the injured child. *J Pediatr Surg* 1987;22:14–18.
  82. Tilden SJ, Watkins S, Tong TK, Jeevanandam M. Measured energy expenditure in pediatric intensive care patients. *Am J Dis Child* 1989;143:490–492.
  83. Tolo VT. External skeletal fixation in children's fractures. *J Pediatr Orthop* 1983;3:435–442.
  84. Tolo VT. External fixation in multiply-injured children. *Orthop Clin North Am* 1990;21:393–400.
  85. Tolo VT. Orthopaedic treatment of fractures of the long bones and pelvis in children who have multiple injuries. *Instr Course Lect* 2000;49:415–423.
  86. Torode I, Zieg D. Pelvic fractures in children. *J Pediatr Orthop* 1985;5:76–84.
  87. Tso EL, Beaver BL, Haller JA Jr. Abdominal injuries in restrained pediatric passengers. *J Pediatr Surg* 1993;28:915–919.
  88. van der Sluis CK, Kingma J, Eisma WH, ten Duis HJ. Pediatric polytrauma; short-term and long-term outcomes. *J Trauma* 1997;43:501–506.
  89. Vasquez WD, Garcia VF. Pediatric pelvic fractures combined with an additional skeletal injury as an indicator of significant injury. *Surg Gynecol Obstet* 1993;177:468–472.
  90. Verstreken L, Delonge G, Lamoureaux J. Orthopaedic treatment of paediatric multiple trauma patients: a new technique. *Int Surg* 1988;73:177–179.
  91. Wesson DE, Spence LJ, Williams JI, Armstrong PF. Injury scoring system in children. *Can J Surg* 1987;30:398–400.
  92. Wilkins J, Patzakis M. Choice and duration of antibiotics in open fractures. *Clin Orthop* 1991;22:433–437.
  93. Winogron HW, Knights RM, Bawden HN. Neuropsychological deficits following head injury in children. *J Clin Neuropsychol* 1984;6:269–286.
  94. Woolf PD, McDonald JV, Feliciano DV, Kelly MM, Nichols D, Cox C. The catecholamine response to multisystem trauma. *Arch Surg* 1992;127:899–903.
  95. Wyrsh B, Mencio GA, Green NE. Open reduction and internal fixation of pediatric forearm fractures. *J Pediatr Orthop* 1996;16:644–650.

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The growth plate (physis) of an epiphysis or an apophysis may be injured in various ways ([1,2,3,4](#)). The most common injury is fracture, but other insults such as disuse, radiation, infection, tumor, vascular impairment, neural involvement, metabolic abnormality, frostbite, burns, electrical injuries, laser injuries, chronic stress, and iatrogenic injury can also damage the physis sufficiently to interrupt growth.

When the entire growth plate is arrested, bone length is retarded. If the physis at each end of a bone is arrested, longitudinal bone growth ceases completely. When the bone is compared with the contralateral mate, there is a discrepancy in bone length. In the forearm or lower leg, the length inequality can also be relative to the ipsilateral companion bone (the radius and ulna or tibia and fibula, respectively).

When only part of the physis is damaged, length retardation can be accompanied by angular deformity as the undamaged portion of the physis continues to grow. The deformity and length inequality depend on the site (specific bone), the location within the site, the extent (quantity), and the duration of the physeal damage.

This chapter is an overview of physeal injuries and is divided into three parts: physeal fractures, other physeal injuries, and premature physeal arrest. Injuries at specific anatomic sites are discussed in their respective chapters.

## PHYSEAL FRACTURES

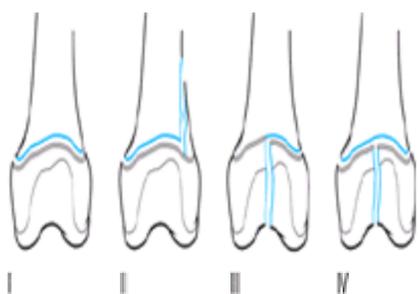
### Historical Review

Fractures of the physis have been of interest since antiquity. Historians note the fable of the Amazons, whose custom of separating the epiphyses of newborn males ensured female supremacy and beauty. Hippocrates receives credit for the first written medical account of this injury ([5,6](#)). A Neapolitan surgeon, Marcus Aurelius Severinus, noted the problem of separation of the proximal and distal tibial epiphyses in 1632. Written observations, case reports, articles, theses, and treatises followed and are best summarized in Poland's 1898 book *Traumatic Separation of the Epiphysis* ([6](#)).

### Classification

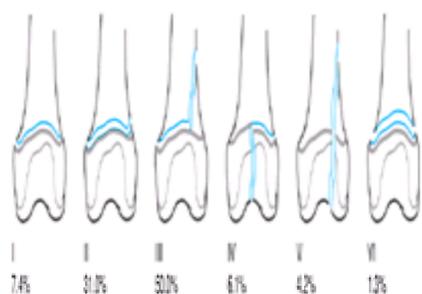
#### Early Classifications

Poland's book ([6](#)) established the fracture as a significant and not rare entity. He documented four specific injuries, provided drawings of each, and thereby produced the first true classification ([Fig. 5-1](#)). Following Roentgen's discovery of the x-ray (1895), the subject was studied more scientifically; before this, all observations were made from compound fractures, dissections of patients with fatal injuries, or of limbs with traumatic amputations ([7,8,9,10,11,12,13,14,15,16,17,18,19,20,21,22,23,24,25,26,27,28,29,30,31,32](#)).



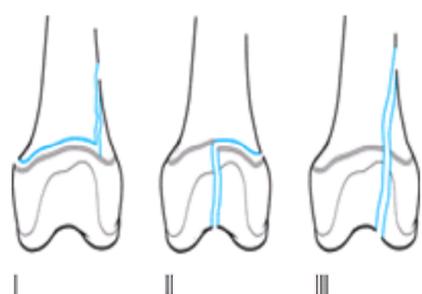
**FIGURE 5-1.** Classification of Poland (1898). (Redrawn from Peterson HA. Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439–448; with permission.)

In 1933, Bergenfeldt documented radiographically 310 physeal injuries in 295 patients and defined six types ( Fig. 5-2). These six types included the first three of Poland and added a fracture through the epiphysis, metaphysis, and physis. These four fractures subsequently were used by Salter and Harris ( 28) as their first four types.



**FIGURE 5-2.** Classification of Bergenfeldt (1933), with percentage of each type. (Redrawn from Peterson HA. Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439–448; with permission.)

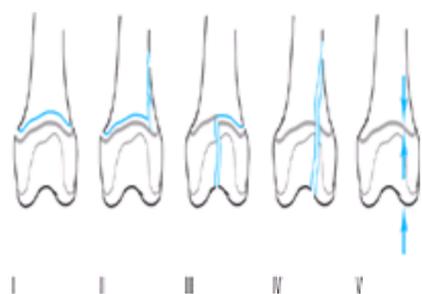
In 1936, Aitken (7) described three types of physeal fractures of the distal tibia ( Fig. 5-3), two of which (I and II) were described by Poland, and the third, by Bergenfeldt. Aitken documented the same three physeal fractures in the distal femur ( 10) and the proximal tibia (9). After his 1965 article (8) discussing these three types of fractures in a general context, Aitken's three types of fractures became the standard by which most physicians reported physeal fractures.



**FIGURE 5-3.** Classification of Aitken (1936). (Redrawn from Peterson HA. Physeal fractures: Part 3, classification. *J Pediatr Orthop* 1994;14:439–448; with permission.)

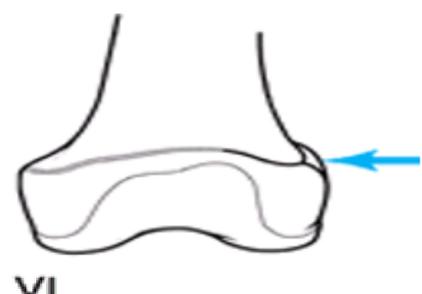
### Salter-Harris Classification

In 1963 Salter and Harris (28) published their classic article, “Injuries Involving the Epiphyseal Plate,” in which five types of injuries were described ( Fig. 5-4). The first four types are a combination of those described by Poland (types I to III), Bergenfeldt (types I to V), and Aitken (types I to III). Salter and Harris added the concept of compression injury, which they designated type V. They proposed that the mechanism of this injury is by longitudinal compression, which damages the germinal layer of physeal cells. Because there was no osseous injury, radiographs at the time of injury were by definition normal. This differs from the crushing of physeal cells that can occur with any physeal fracture, as described by Letts (18). Because no structure is broken, this is not a fracture; the term “injury” seems more appropriate.



**FIGURE 5-4.** Classification of Salter and Harris (1963). (Redrawn from Peterson HA. Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439–448; with permission.)

Salter's associate, Mercer Rang (26) added an injury in 1969 that has become known as a Salter-Harris, or Rang, type VI. This was described as a rare injury produced by a direct blow to the periosteum or perichondrial ring ( Fig. 5-5). It was never specified whether the damage was produced by peripheral transverse compression of physeal cells or ischemia due to vascular changes. Because no mention was made of open trauma, it is assumed that these were closed injuries. Like a Salter-Harris type V, this should also be called an injury rather than a fracture, because nothing is broken and the original radiograph is normal. Other authors (2,3,19,20 and 21) have interpreted this injury as an avulsion of the perichondrial ring with portions of attached metaphyseal and epiphyseal bone, while still considering it a Salter-Harris or Rang type VI. Although drawings have been provided, the only case depicted radiographically ( 3) is an open lawn mower excision of the metaphysis, physis, and epiphysis; this is more appropriately classified as a part missing (see Classification later). None of these Rang type VI injuries were found in a recent population-based study of 951 cases. Neither Salter (29) nor Rang (27) included this type in subsequent publications.



VI

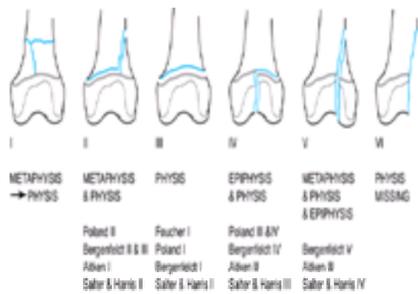
**FIGURE 5-5.** Physeal injury of Rang (1969). (Redrawn from Peterson HA. Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439–448; with permission.)

The Salter-Harris classification gained widespread acceptance throughout the world (1,2,3,4). In recent years, however, several authors have deviated from this classification. In 1980, Weber (32), who was unable to find any type V injuries, returned to the Aitken classification. In 1983, Rang (27) noted that the Aitken classification is “widely used,” and in 1993, Kling (17) stated that the Aitken classification is “now used in Europe.” Other authors (12,13,14,19,20,21,30,32), finding the classification incomplete or lacking in substantiation of prognosis, have developed new classifications, notably Ogden (19,20,21) in 1981 and Shapiro (30) in 1982.

The classification of physeal fractures is a work in progress. This search for a classification that will allow the collection of meaningful statistical data and a better means of communication is a progressive quest for knowledge. This knowledge, in turn, should improve criteria for prognosis, management, and recommendations for follow-up of patients with physeal fractures.

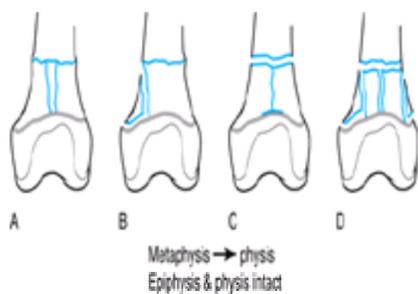
### AUTHOR'S CLASSIFICATION

In 1994, a new classification (24) based on the first population-based epidemiologic study (Fig. 5-6) arranged fracture types from the least involvement or damage to the physis (type I) to the greatest involvement (type VI).



**FIGURE 5-6.** Classification of Peterson. (Redrawn from Peterson HA. Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439–448; with permission.)

Type I is a transverse fracture of the metaphysis with fracture line or lines extending to the physis (Fig. 5-7) (23). There is no fracture along the physis and no displacement of the epiphysis on the metaphysis (Fig. 5-8). There may be a small eccentric cortical fragment not attached to either the epiphysis or the metaphysis. Comminution is common (Fig. 5-9), and compounding is rare. The mechanism of injury is most likely longitudinal compression as evidenced by the cortical torus or buckling, widening of the metaphysis, or comminution. Radiographs 2 to 4 weeks after injury typically show transmetaphyseal sclerosis indicative of a healing compression fracture (Fig. 5-8B). This fracture made up 15.5% of fractures in the Olmsted County population study, but it is probably much more prevalent, because metaphyseal fractures were not reviewed and neither the hand surgeons or the pediatric orthopaedists were aware of this fracture before the study. The most common sites are the distal radius, finger phalanges, and metacarpals. Nonoperative treatment by closed reduction and immobilization usually results in a good outcome. Only one patient with this fracture type (0.7%) was treated surgically in the Olmsted County study. Premature physeal closure occurred in five (3.4%) adolescents, none of whom required treatment.



**FIGURE 5-7.** Peterson type I fracture of the metaphysis with extension to the physis. **A:** Torus or buckle complete transmetaphyseal fracture with one or more fracture lines extending to the physis. The fracture does not extend along the physis, and the epiphysis is not displaced on the metaphysis. The metaphysis is frequently wider than normal. The transmetaphyseal fracture is a compression fracture, often best visualized 2 to 4 weeks postfracture as an increased sclerotic osseous density. **B:** Transverse metaphyseal compression fracture with peripheral cortical fragment. This fragment may be displaced eccentrically, indicating disruption of the physis in this area. **C:** Complete transverse metaphyseal fracture with fracture line extension to the physis. **D:** Comminuted fracture of the metaphysis with multiple fracture extensions to and along the physis. None of these fractures meets the requirements of a type II fracture, which is a fracture of only part of the metaphysis extending to and along the physis. The designations **A** to **D** are not an attempt to subdivide or classify this fracture type but are used only to show the multiple possibilities. (Redrawn from Peterson, HA. Physeal fractures: part 2, two previously unclassified types. *J Pediatr Orthop* 1994;14:431–438; with permission.)

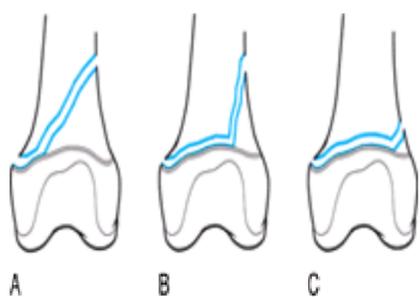


**FIGURE 5-8.** Peterson type I fracture in the distal radius of a 6+5-year-old boy. **A:** AP view shows complete transverse metaphyseal fracture with longitudinal extension to the physis. The metaphysis is wider than normal, and the epiphysis is not displaced on the metaphysis. **B:** AP view 34 days postfracture shows healing sclerosis transversely and longitudinally at sites of fracture. (From Peterson HA. Physeal fractures: part 2, two previously unclassified types. *J Pediatr Orthop* 1994;14:431–438; with permission.)



**FIGURE 5-9.** Peterson type I fracture in the distal radius of a 14+1-year-old boy. **A:** AP view shows comminuted displaced fracture of metaphysis. Longitudinal cortical buckling fracture on radial side extends to the physis. **B:** AP view during reduction in fingertrap traction. Transmetaphyseal fracture with extension to physis and no displacement of epiphysis is now evident. The metaphysis is wider than normal. Fractured ulnar styloid. **C:** AP view, 14 months postfracture. There is normal function, and the distal radial physis is open and growing. Nonunion of ulnar styloid is seen. (From Peterson HA. Physeal fractures: part 2, two previously unclassified types. *J Pediatr Orthop* 1994;14:431–438, with permission.)

Type II is a separation of part of the physis, with a portion of the metaphysis attached to the epiphysis (Thurstan Holland sign). Involvement and potential damage of the physis may be minimal (Fig. 5-10) or nearly all the physis may be disrupted, leaving only a small metaphyseal fragment (Fig. 5-10C). Most commonly, the metaphyseal portion attached to the physis is a quarter to a third the width of the physis (Fig. 5-10B). Although attention is usually focused on the size of the metaphyseal fragment, the more important factor is the amount of physeal tissue disrupted. Indeed, the Thurstan Holland metaphyseal fragment may be so tiny that it is not seen on routine anteroposterior or lateral radiographs (Fig. 5-10C). Tangential (oblique) views may be necessary to reveal the fragment. In this context, it differs little from a type III injury (Fig. 5-6), which involves complete physeal disruption with no osseous fracture. Type II (Fig. 5-10C) and III injuries can be managed similarly, usually by closed reduction and immobilization. There are no recorded cases of premature physeal closure between the metaphyseal fragment and the epiphysis. Brashear, applying longitudinal compression by bending the knee joints of rats, produced a type II fracture each time. None of these fractures developed physeal closure at the compression site (metaphyseal fragment/epiphyseal interface). If premature physeal closure occurs, it is at the site of the sharp edge of the fractured metaphysis, which excoriates or compresses the physis (18).



**FIGURE 5-10.** Peterson type II physeal fracture. See text for discussion. (Redrawn from Peterson HA. Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439–448; with permission.)

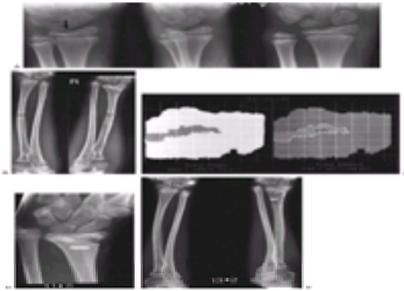
Regardless of the amount of physis disrupted, the essential features of a type II injury are disruption of part of the physis between the epiphysis and the metaphysis, however small this may be; fracture of only part of the metaphysis, with a metaphyseal fragment attached to the physis; and no continuity from the epiphysis to the intact major metaphyseal/diaphyseal complex. Comminution and open fracture are uncommon. This fracture is the most common type in all previous series and is made up 53.6% of fractures in the Olmsted County study. The most common site is in finger phalanges, where it occurred 47.6% of the time. Initial management was surgical for 23 (4.5%) patients. Thirty-three (6.5%) developed premature physeal closure. Twelve (2.4%) underwent late surgical correction.

A type III injury is a separation of the epiphysis from the diaphysis through any of the layers of the physis, disrupting the complete physis (Fig. 5-6). This injury is rarely open and cannot be comminuted. The only anatomic variations are the different layers of the physis through which the fracture traverses. At present, this can be determined only histologically. In two studies, the transphyseal fracture was histologically noted to involve all zones of cartilage cells (germinal, proliferating, hypertrophying, and provisionally calcified) (16,31). This helps explain why premature growth arrest may occur following fractures along the physis (Fig. 5-11). This injury made up 13.2% of physeal injuries in the Olmsted County study. It occurs most commonly in the distal fibula. Thirteen acute fractures (10.3%) were treated surgically. Nine patients (7.1%) had late corrective surgery.



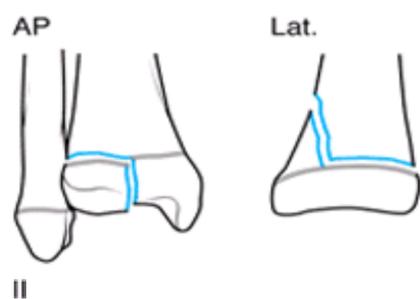
**FIGURE 5-11.** Salter–Harris I fracture of the distal radial physis in a 7+6-year-old girl with subsequent physeal arrest and bar excision. **A:** AP and lateral show dorsally displaced distal radial epiphysis. **B:** Seven and a half weeks postinjury. Physis and metaphysis are irregular and sclerotic. **C:** One year postfracture there is relative overgrowth of ulna. **D:** Age 14+4 years after physeal bar resection. The patient is normally active, participating competitive volleyball, and is asymptomatic. The distal radius is growing (greater than 100%) faster than the ulna, as evidenced by the reduction of the ulnar plus deformity and the increasing distance between the metal markers. The Cranioplast plug stayed with the epiphysis.

Type IV is a fracture of the epiphysis extending to and along the physis (Fig. 5-6). It may be comminuted or “double.” Open fractures are uncommon. Because this fracture most often occurs when part of the physis, usually central, has begun to close, it is more common in older children. The disrupted articular surface requires anatomic reduction and maintenance of reduction, often by open reduction and internal fixation. Premature growth arrest is common, but it usually is complete rather than partial, and rarely causes angular deformity. Most children with this injury are relatively mature, and bone-length discrepancy is uncommon. Significant length discrepancy occurs only in young patients (Fig. 5-12). This fracture made up 10.9% of fractures in the Olmsted County study. The most common sites are the finger phalanges and the distal tibia (medial malleolus and lateral plafond). Eighteen fractures (17.3%) were treated initially by surgery, and 15 (14.4%) underwent late surgery.



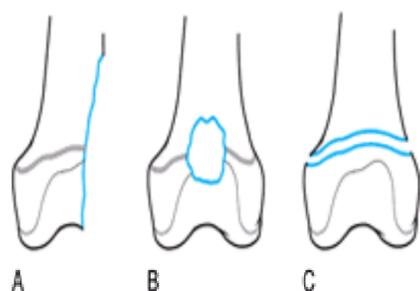
**FIGURE 5-12.** Salter–Harris III fracture, distal radius in a 9+1-year-old girl with subsequent physeal arrest and bar excision. **A:** Patient fell 8 feet, landing on her outstretched right hand. Fracture of the epiphysis extending to the physis ( *arrow*) might be better visualized with additional oblique radiographs or tomograms. **B:** Two years 6 months postfracture (age 11+17). Physeal bar medial distal radius. The right radius is 23 mm shorter than the left. The ulnae are of equal length. **C:** MRI transverse depiction of distal radial physis using 3-D rendering (ANALYZE) shows linear bar comprising 10.5% of the physis. **D:** Physeal bar excised through peripheral approach and cavity filled with Cranioplast. Metal markers 11 mm apart. Note normal growth (relative overgrowth) of ulna. **E:** Eighteen months after bar excision (age 13+2). The distal right radius has not only resumed normal growth, but is growing faster than normal (greater than 100%), as evidenced by improvement of radial–ulnar length discrepancy. The metal markers are 29 mm apart. The radial articular angle is improved, and remodeling has resulted in a more ulnarward position of the proximal metal marker.

Type V is a fracture that traverses the metaphysis, physis, epiphysis, and usually the articular cartilage ( [Fig. 5-6](#)). The triplane fracture ( [22](#)) meets all of these criteria ( [Fig. 5-13](#)) and is therefore a complex type V fracture, which is otherwise depicted in only one plane, usually the sagittal. Comminution and open injuries are common. Type V fractures are best managed by anatomic reduction and maintenance of reduction to align both the articular cartilage and the growth cartilage. This is particularly true in the young patient with significant growth remaining. This usually requires open reduction and internal fixation. Premature growth arrest is common and occurs even with anatomic reduction. This fracture made up 6.5% of fractures in the Olmsted County study. The most common sites are the distal humerus (lateral condyle), finger phalanges, and distal tibia, where the fracture pattern is variable ( [11](#)). Twelve fractures (19.4%) were treated initially by surgery, and 12 (19.4%) were treated by subsequent surgery.



**FIGURE 5-13.** Triplane fracture (1957). (Redrawn from Peterson HA. Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439–448; with permission.)

Type VI is a fracture in which part of the physis has been removed or is missing ( [Fig. 5-14](#)) ( [23](#)). Usually an accompanying part of the epiphysis or metaphysis, or both, is also missing. This occurs only with open fractures such as those caused by lawn mowers, farm machinery (e.g., auger, corn picker, power take-off, corn sheller), snowmobiles, gunshots, and motorboat propellers ( [Fig. 5-15](#)). Premature partial closure of the remaining exposed surface of the physis nearly always occurs but sometimes not until years later. This fracture made up only 0.2% of fractures in the Olmsted County study, but it is more common among referral patients. All type VI injuries require initial surgery, at least wound care; most if not all require late reconstructive or corrective surgery, especially those in young children.



**FIGURE 5-14.** Peterson type VI fracture of the physis with a portion of the physis missing. **A:** Longitudinal fracture with piece comprising epiphysis, physis, and metaphysis missing. In rare cases a portion of the diaphysis may also be missing. The absent portion may vary in location and in size from small to large. Common mechanisms of injury are from lawn mowers, snowmobiles, automobiles, farm implements, and motorboat propellers. **B:** Penetrating injury may remove physeal cartilage, along with adjacent epiphyseal and metaphyseal bone in any plane. A transverse orientation of penetration causes the most severe physeal damage. The most common penetrating object is a bullet. **C:** Lacerating injury directly in the plane of the physis, removing some or all of the physis with relatively little damage to the epiphysis or metaphysis. This injury is rare. The designations A to C are not an attempt to subdivide or classify the fracture type but are used only to show the multiple possibilities. These fractures have only one criterion: part of the physis is missing. A physeal bar invariably develops, sometimes years postfracture. (Redrawn from Peterson HA. Physeal fractures: part 2, two previously unclassified types. *J Pediatr Orthop* 1994;14:431–438; with permission.)



**FIGURE 5-15.** Peterson type VI fracture of the proximal tibia in a 10+4-year-old girl. **A:** Standing teleoroentgenogram. Right genu varum following removal of right proximal tibial medial condyle in lawn mower accident that occurred at age 4+11 years. Patient had previously undergone proximal tibial valgus osteotomy and

physeal bar excision. Note lateral subluxation of tibia on femur. **B:** Tomogram confirms recurrent physeal bar medially. Note this relative overgrowth of the unopposed medial femoral condyle. This patient was treated by bracing and, at age 13, by medial tibial cadaver allograft and physeal closure of the lateral side of the proximal tibia, with concomitant physeal arrest of the contralateral normal proximal tibial physis.

### Salter-Harris Type V, Present Status

The Salter-Harris type V injury is not included in this new classification of fractures because with this injury, there is no fracture. Because the radiograph taken at the time of injury is normal and growth arrest is discovered only in retrospect, this entity, if it exists at all, cannot be suspected during the evaluation or treatment of an acute injury. Therefore, it is included in the later section, "Other Physeal Injuries."

Comparison of classifications may have more than historical value. New mechanisms of injury and new or improved imaging techniques may lead to the inclusion of new fracture types, or of a previously described but discarded fracture type.

### Epidemiology (The Olmsted County Study)

Mann and Rajmaira (35) reported that of 2650 long-bone fractures in children, about 30% involved the physes ( 33,34,35,36,37,38,39). Worlock and Stower (39) reported that of 826 fractures of all bones in children, 18.5% involved the physes. An epidemiologic study of physeal fractures was performed over a 10-year period in Olmsted County, Minnesota, from 1979 to 1988 (Table 5-1) (38). This was the first population-based study in which both the numerator (fractures) and denominator (population at risk) were known. No referral cases were included, thereby omitting preselected difficult or high-risk cases.

Types	Number (%)	Immediate Surgery (%)	Late Surgery (%)
I	147 (15.5)	1 (0.7)	0 (0)
II	510 (53.6)	23 (4.5)	12 (2.4)
III	126 (13.2)	13 (10.3)	9 (7.1)
IV	104 (10.9)	18 (17.3)	15 (14.4)
V	62 (6.5)	12 (19.4)	12 (19.4)
VI	2 (0.2)	2 (100)	1 (50)
Total	951 (100)	69 (7.3)	49 (5.2)

\* Data pertain to physeal fractures among children in Olmsted County, Minnesota, 1979-1988. From Peterson HA, Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439-448.

TABLE 5-1. NUMBER AND SURGERY OF PHYSEAL FRACTURES ACCORDING TO PETERSON CLASSIFICATION

Of the 951 physeal fractures in the study, the male-to-female ratio was 2:1. The incidence rates were highest among boys age 14 and girls ages 11 and 12 ( Fig. 5-16). The overall age- and sex-adjusted incidence of physeal fractures was 279.2 per 100,000 person-years (95% confidence interval, 261.4 to 296.9). Fractures occurred approximately equally on right and left limbs (52.6% versus 47.4%,  $p > .05$ ). The most common site was the phalanges of the fingers, which accounted for 37% of all physeal fractures, followed by the distal radius (18%) ( Table 5-2). The distal end of each long bone was fractured more frequently than the proximal end. Seventy-one percent of fractures occurred in the upper extremity, 28% in the lower extremity, and 1% in the axial skeleton.

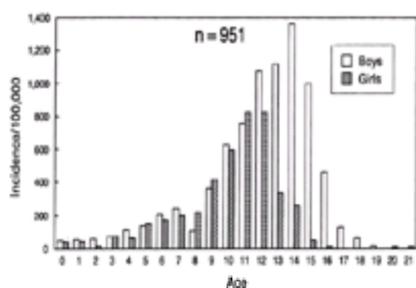


FIGURE 5-16. Incidence of physeal fractures by gender for 1-year age groups with all types of fractures. Boys peak at age 14 years, and girls peak at age 11 to 12 years. (From Peterson HA, Madhok R, Benson JT, et al. Physeal fractures: part 1, epidemiology in Olmsted County, Minnesota, 1979-1988. *J Pediatr Orthop* 1994;14:423-430; with permission.)

Skeletal Site	Type I		Type II		Type III		Type IV		Type V		Type A		Type B		All Types	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Phalanges (Proximal)	21	2.2	200	21.0	42	4.4	12	1.3	36	3.8	2	0.2	206	21.6	314	33.0
Distal radius	21	2.2	366	38.5	3	0.3	10	1.1	36	3.8	0	0	375	39.5	511	53.7
Distal tibia	0	0	47	5.0	27	2.8	0	0	0	0	0	0	74	7.8	121	12.7
Distal tibia	0	0	30	3.2	1	0.1	0	0	0	0	0	0	31	3.3	31	3.3
Metatarsals	2	0.2	27	2.8	1	0.1	0	0	0	0	0	0	28	3.0	30	3.2
Phalanges (Distal)	6	0.6	30	3.2	8	0.8	8	0.8	1	0.1	0	0	43	4.5	60	6.3
Distal radius	8	0.8	5	0.5	1	0.1	28	2.9	2	0.2	0	0	37	3.9	43	4.5
Distal tibia	0	0	7	0.7	0	0	0	0	0	0	0	0	7	0.7	7	0.7
Proximal humerus	0	0	18	1.9	0	0	0	0	0	0	0	0	18	1.9	18	1.9
Distal femur	4	0.4	6	0.6	1	0.1	0	0	0	0	0	0	11	1.2	11	1.2
Metatarsals	2	0.2	6	0.6	1	0.1	0	0	0	0	0	0	9	0.9	9	0.9
Proximal radius	0	0	0	0	1	0.1	0	0	0	0	0	0	1	0.1	1	0.1
Proximal tibia	1	0.1	0	0	1	0.1	0	0	0	0	0	0	2	0.2	2	0.2
Proximal tibia	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Clavicle, medial	0	0	0	0	1	0.1	0	0	0	0	0	0	1	0.1	1	0.1
Acromioclavicular	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Proximal femur	1	0.1	0	0	0	0	0	0	0	0	0	0	1	0.1	1	0.1
Proximal femur	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total	104	10.9	510	53.6	126	13.2	104	10.9	62	6.5	2	0.2	951	100	951	100

TABLE 5-2. SITES AND TYPES OF PHYSEAL FRACTURES AMONG CHILDREN IN OLMSTED COUNTY, MINNESOTA, 1979-88 AND SALTER-HARRIS CLASSIFICATION WITH TWO NEW TYPES (A AND B)

### Evaluation

The history of injury and the clinical evaluation (symptoms and physical findings) usually define appropriate body parts for radiographic evaluation (40,41,42,43,44,45,46,47,48,49,50,51,52,53,54,55,56,57,58,59,60,61,62,63,64,65,66,67,68,69,70,71). Radiographs in two planes, usually anteroposterior and lateral, are sufficient to determine the fracture type and the management plan for most fractures. If the radiographs are indeterminate, oblique or three-quarter views should be taken (51,63,64). Occasionally stress views, tomograms, arthrograms, computed tomography (CT) scans, magnetic resonance imaging (MRI), or ultrasound is needed to adequately display the fracture. Stress views are most useful when the injury is near a uniplanar joint (e.g., the elbow, knee, or ankle) ( 51). Tomograms help delineate fragmentation and orientation of fragments. CT scans may best reveal the complexities of triplane fractures ( 22,64). Fractures involving a large volume of cartilage, such as the proximal or distal humerus in infants, in the past were best appreciated by arthrography ( 40,41,49,56,57,71). Recently, this method has been

replaced by MRI ([54,55,64,65,70](#)) and ultrasound ([43,52,57](#)). MRI has also been used in older children with acute injuries to identify possible physeal damage ([44,53,59,60,67,70](#)) and is recommended primarily to identify complex fractures ([60](#)). The role of scintigraphy ([47,68](#)) in assessing acute physeal fractures is yet to be determined.

## Treatment

The goal of treatment of all physeal fractures is to maintain function and normal growth ([72,73,74,75,76,77,78,79](#)). Maintenance of growth is obviously more important in a young child than in an older adolescent, who has little growth remaining. Consistent attainment of these goals is most likely when all structures are anatomically reduced. Thus, the goal becomes to obtain and maintain anatomic reduction. This may be done by closed or open means. All reductions, whether closed or open, should be gentle to prevent damage to the delicate physeal cartilage. Forceful, repeated manipulations should be avoided. During open reduction, placing direct pressure on the physis with instruments should be avoided.

Physeal fractures should be reduced immediately, because delay makes reduction more difficult. The younger the child, the more rapidly the healing callus blends with articular and physeal cartilage, making reduction difficult.

### **Type I (Metaphyseal Compression With Extension Into the Physis)**

The type I fracture has the least potential damage to the physis and therefore needs the least aggressive treatment. Closed reduction and casting usually achieve satisfactory position and alignment of the fragments ([Fig. 5-8](#) and [Fig. 5-9](#)). Fractures of the metaphysis and physis heal rapidly, and cast immobilization for 3 to 4 weeks is usually sufficient. Because the physis is involved in the injury, growth arrest is possible (3.4% in the Olmsted study), and these fractures need follow-up long enough to ensure that normal growth has resumed. This period varies depending on the patient's age and the site and severity of injury; at least 3 months is appropriate for most patients, longer for patients with comminuted or markedly displaced fractures.

### **Salter-Harris II**

Most type II fractures can be reduced easily with closed manual reduction. Scraping of the metaphyseal fragment across the intact physis can be decreased by good patient relaxation to reduce muscle tension. This is probably best achieved by general anesthesia or a nerve block such as an axillary or lumbar epidural block. The metaphyseal fragment usually prevents overreduction ([Fig. 5-10A](#) and [Fig. 5-10B](#)). The intact periosteum on the side of the metaphyseal fragment imparts further stability to the reduced fracture, and internal fixation is often unnecessary.

In a young patient, incomplete reduction may be more acceptable than repeated overzealous manipulation, which may cause gouging of the physis. In an older patient, a more accurate reduction is necessary because spontaneous correction with growth is less likely. Occasionally, a type II fracture cannot be satisfactorily reduced. A common example is the distal tibia, where periosteum becomes impinged at the fracture site. Tendon, nerve, and vascular-structure impingement also has been reported at various sites; these types also need surgical extrication. If the reduced fragments are unstable, internal fixation is appropriate. This is best accomplished by pins or screws from metaphysis to metaphysis, avoiding the physis. Occasionally, the metaphyseal fragment attached to the epiphysis (Thurstan Holland sign) is so small that it cannot be internally fixed to the main metaphysis ([Fig. 5-10C](#)). In this case, small-diameter, smooth pins may be placed from the epiphysis, across the physis, and into the metaphysis. Growth arrest is less likely if the pins avoid the perichondrial ring, are as longitudinally as feasible, and remain in place a short time, preferably 3 weeks or less. The insertion of biodegradable pins across the physis is a new concept that is being evaluated, most intensely in Finland ([72,74,75,76,78,79](#)). Whether these pins impart adequate stability without predisposing to physeal arrest remains to be seen. In one of the more recent studies, growth arrest was noted and the authors suggested that the procedure be limited to adolescents with little growth remaining ([78](#)).

Type II fractures may involve a small ([Fig 5-10A](#)) or large ([Fig 5-10C](#)) portion of the physis; the more of the physis involved, the greater the chance of growth arrest. The prognosis also depends greatly on the site of injury. When the physis is irregular and undulating, as in the distal femur or proximal tibia, displacement of the metaphyseal surface against the physeal surface produces scraping of these irregular surfaces and an increased likelihood of physeal arrest. A smooth, flat physis, such as the distal radius, is much less prone to arrest. The degree of displacement and the patient's age are also obviously important. Repeated reduction attempts increase the chance of arrest. Type II fractures may involve only a small amount of physis ([Fig. 5-10A](#)) or a large amount of physis ([Fig. 5-10C](#)); the latter increases the chance of physeal damage.

### **Salter-Harris I**

Type III fractures ([Fig. 5-6](#)) are similar to those type II fractures which have a very small metaphyseal fragment ([Fig. 5-10C](#)). Because all layers of the physis may be involved ([Fig. 5-11](#)), the prognosis for growth arrest is slightly greater than with type II fractures ([Table 5-1](#)). Type III fractures should be managed by closed reduction because internal fixation would likely require crossing the physis. In a young child, it is better to accept an imperfect reduction than risk the hazards of internal fixation across the physis.

### **Salter-Harris III**

In type IV fractures, the cartilage of the physis and the articular surface are both disrupted ([Fig. 5-6](#)). The best result is achieved by anatomic reduction of the articular surface to reduce the likelihood of degenerative arthrosis and anatomic reduction of the physeal cartilage to reduce the chance of growth arrest. Usually, these fractures occur in older children, when the physis is beginning to close and growth arrest is not a problem. Anatomic reduction often requires open reduction to expose the articular surfaces, especially in young children. The most desirable internal fixation is epiphysis to epiphysis, if possible, especially in young children.

### **Salter-Harris IV**

For the type V fracture, anatomic reduction and maintenance of reduction are essential to align both the physis and the articular surface. If there is any displacement, open reduction and internal fixation are usually required. Closed reduction with percutaneous internal fixation may be acceptable in some situations ([77](#)). Internal fixation is best accomplished from epiphysis to epiphysis or metaphysis to metaphysis, particularly in young children. Growth arrest is likely, and these fractures need observation for at least a year, even if earlier evaluation is good.

### **Type VI (Epiphyseal and Physeal Loss)**

Because type VI fractures are open injuries, all require initial debridement, often with wound packing and secondary closure and sometimes with skin graft or flap closure. Children with these injuries must be followed until maturity, because most if not all develop angular deformities and relative shortening of the involved bone ([Fig. 5-15A](#)). There is a high likelihood of physeal bar formation on the exposed bone surface ([Fig. 5-15B](#)).

## Prognosis

The prognosis of a physeal fracture depends on the following factors, in descending degree of importance: the severity of injury, including displacement, comminution, and whether it is open or closed; the patient's age; the physis injured; and the radiographic type of fracture ([1,2,3,4,24,28,29,38,80,81](#) and [82](#)). Treatment depends on these factors and in itself has an important bearing on the prognosis.

### **Severity of Injury**

The mechanism of injury may be thought of as the activity the patient was involved in at the time of injury (e.g., automobile accident, football, diving) or as an anatomic description of the injury (e.g., varus, extension, rotation). The term *high-velocity injury* is often used to describe fractures with significant displacement, comminution, and compounding. The term *severity of injury* is used here to include all of these features and is the most important factor in prognosis. Any damage to or loss of the germinal or proliferative layer of physeal cells has a negative implication concerning growth.

### **Age of Patient**

The patient's age is also important. A physeal injury in a 13-year-old girl or a 15-year-old boy rarely results in significant length discrepancy or angular deformity, because little growth remains. Any physeal injury in a young child, however, has the potential for premature arrest, and appropriate follow-up is required.

### Site of the Injury

The site of injury influences the outcome. A type I fracture of the distal femur or proximal tibia, both of which have large, undulating, multiplanar physes, are prone to arrest, even with mild separation or displacement. Because these sites contribute more longitudinal growth than any other site in the body, deformity and length discrepancy are common. Conversely, the proximal radius and ulna and distal humerus contribute so little growth to the forearm and humerus, respectively, that injury and premature arrest from any fracture type at these sites rarely causes significant angular deformity or length inequality. A type I fracture of the distal fibula, rarely develops arrest, and ankle valgus deformity would occur only with arrest at a young age. The proximal humerus has such good remodeling potential that significant displacement and angulation can be accepted, even in older children.

The site of the injury also determines the blood supply of the physis. The growth-producing germinal and proliferating cells receive their nourishment through blood vessels from the epiphysis. If this blood supply is destroyed, these cells die and growth ceases. Fortunately, most epiphyses receive their blood supply directly from multiple sources. When the arterial supply is limited to a few arteries that must reach the epiphysis by crossing the periphery of the physis (e.g., the proximal femur), any displacement of the epiphysis may occlude the blood supply, resulting in avascular necrosis of the epiphysis and physeal cell death.

### Amount of Physis Injured

The type of fracture is determined by the mechanism of injury, the patient's age, and the site of injury. For example, a type II fracture of the distal radius is common at any age; a type II fracture of the distal humerus is rare at all ages. The type of fracture also relates to the amount of physis damaged. Thus, although this new classification is based primarily on anatomic considerations from the least amount of physis involved (type I) to the greatest amount (type VI), it also was found to be correlated to the prognosis in a general way. In the Olmsted study, the amount of surgery performed, both initially at the time of injury and later to correct a complication, increased as the type number increased ([Table 5-1](#)).

## COMPLICATIONS

Sepsis can occur in any open physeal fracture, just as in any open diaphyseal fracture ([83,84,85,86,87,88,89,90,91,92,93,94,95,96,97,98](#)). Practically speaking, this is seen exclusively in open injuries, primarily type IV and VI fractures. Precautions and treatment are similar to those for any other open bone or joint injury. Overgrowth after physeal fracture is essentially unknown, except of the capitellum in type V fractures of the lateral humeral condyle, where overgrowth is rarely sufficient to cause significant angular deformity or length discrepancy. Hypoplasia of an epiphysis after trauma also is rare, and it is usually associated with damage of the germinal layer of the physis or with vascular impairment that produces avascular necrosis. The accompanying angular deformity usually is minor, but in the trochlea, it can be sufficient to cause ulnar nerve palsy ([92](#)). Likewise, malunion of properly recognized and treated physeal fractures is uncommon ([86](#)). Delayed union or nonunion of type V fractures occurs occasionally, and has been noted in the lateral humeral condyle ([85,89](#)), the distal femur ([87](#)), and the medial malleolus ([Fig. 5-17](#)). Nonunion may also occur after open reduction and internal fixation ([Fig. 5-18](#)). This is usually treated by attempts at osteosynthesis or reconstructive surgery.

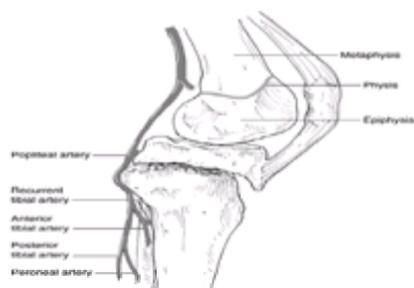


**FIGURE 5-17.** Nonunion of type V fracture. **A:** Type V fracture of distal tibial medial malleolus in a 4-year-old boy. The fracture was treated by cast immobilization. **B:** Age 6+10. Union of the small metaphyseal fragment to the metaphysis, but nonunion of the epiphyseal fragment to the epiphysis. There is no physeal bar. The physis is growing more rapidly medially, as evidenced by the growth arrest line in the metaphysis. The upward displacement of the fragment before union of the metaphyseal fragment allows some ankle varus.



**FIGURE 5-18.** Nonunion of Salter–Harris IV fracture. **A:** Type V fracture of distal tibial medial malleolus in a 3+4-year-old girl. Treatment was open reduction, excision of metaphyseal fragment, and internal fixation. **B:** Five weeks postfracture, medial malleolar fragment is not united. **C:** Five months postfracture there is an established nonunion.

Compartment syndrome and arterial occlusion are not commonly associated with physeal fractures but have been reported with fractures of the proximal tibia ([83,93,94,97](#)) ([Fig. 5-19](#)) or the distal radius ([88,91](#)). Avascular necrosis of the proximal femoral capital epiphysis is a dreaded complication of any fracture of the proximal femoral physis, particularly if the hip is also dislocated ([95](#)). Avascular necrosis can occur at the proximal ([90](#)) and the distal humerus ([98](#)) and at the proximal radius, but it is rare at all other anatomic sites.



**FIGURE 5-19.** Occlusion of the popliteal artery by direct pressure from posterior displacement of the proximal tibial metaphysis. (Redrawn from Burkhart SS, Peterson HA. Fractures of the proximal tibial epiphysis. *J Bone Joint Surg Am* 1979;61:996–1002; with permission.)

All of these complications are uncommon. By far, the most frequent complication of a physeal fracture is premature growth arrest, resulting in diminished bone length, angular deformity, or both. This complication is so prevalent that it deserves special attention and is discussed later in this chapter.

## OTHER PHYSEAL INJURIES

Physeal injuries, other than fracture, that are sufficient to cause premature partial or complete arrest share two characteristics: normal radiographs at the time of insult, and premature physeal arrest noted weeks, months, or years later. These injuries are uncommon. The injuries that meet these criteria can be classified as follows:

1. Disuse
2. Radiation
3. Infection
4. Tumor
5. Vascular impairment
6. Neural involvement
7. Metabolic abnormality
8. Cold injury (frostbite)
9. Heat injury (burn)
10. Electric injury
11. Laser injury
12. Stress injury
13. Longitudinal compression
14. Developmental
15. Iatrogenic

### Disuse

Disuse of an extremity, from any cause, results in atrophy of muscle and other soft tissue ([99,100,101,102,103,104,105,106,107,108,109,110](#) and [111](#)). If disuse is prolonged, growth of bone length also is retarded. If disuse is marked, some or all of the physis may cease growing completely. Bed rest, crutches, cast immobilization, and traction, even in the absence of fracture, are all associated with growth arrest of the distal femoral physis and proximal or distal tibial physis. These modalities have been used in the management of patients with congenital hip dislocation ([100,104](#)), rheumatoid arthritis ([104](#)), polio ([101,108](#)), tuberculosis ([107,111](#)), and Perthes' disease, but are now used more sparingly for various reasons, one of which is the possibility of growth arrest.

Complete closure of one or all of the physes in an extremity after a single diaphyseal fracture has been reported and was attributed to disuse of the extremity ([3,61,99,102,103,104,105,106,109,110](#)). Some of those multiple physeal closures have been attributed to Salter-Harris type V compression injuries, but it is difficult to envision multiple-site compression injuries with a single femoral or tibial transverse or spiral shaft fracture. Conversely, some Salter-Harris type V injuries reported in the literature may have been caused by posttraumatic disuse rather than sudden traumatic physeal compression.

The precise etiology of physeal arrest associated with disuse is unknown. Gill speculated that the accompanying osteoporosis predisposed the patient to microfractures that traversed the physis. Ross ([110](#)) suggested that the partially degenerated cartilage cannot withstand the abnormal stresses of a faulty gait. Diminished arterial supply is a more likely possibility.

### Radiation

The growth-inhibiting and destructive effects of x-rays on physeal cartilage have been demonstrated in both animals and humans ([112,113,114,115,116,117,118,119,120,121,122,123,124,125,126,127,128,129,130](#) and [131](#)). Growth-inhibiting effects are common when a physis is in the treatment field during therapeutic irradiation ([115,116,119,127,129,130](#) and [131](#)) but have not been shown to occur with diagnostic x-ray studies or the use of repeated tomograms. The main variables determining the extent of demonstrable bone change caused by radiation therapy are the patient's age at the time of therapy, the amount of the radiation delivered (including both dose per treatment and total dose delivered), the field size, the site to which the radiation was delivered, and the growth potential of this site. The younger the patient and the greater amount of radiation delivered, the greater is the extent of subsequent bone changes ([Fig. 5-20](#)). A radiation dose as low as 400 R can produce growth retardation, and as the dose increases, so does the extent of cellular damage and eventual development of dysplastic bone. All physes, including those in the spine, are at risk ([120,124,125](#) and [126](#)).



**FIGURE 5-20.** Physeal arrest associated with radiation. **A:** A 5+0-year-old with Ewing's sarcoma proximal left fibula; diagnosis was made by open biopsy. Metastasis to lung confirmed by transthoracotomy biopsy. Patient received radiation therapy (5000 cGy to the left leg in 24 fractions over 8 weeks and 1500 cGy to the lungs in 12 fractions over 2 weeks). **B:** The left tibia did not grow, and at age 15+0, it is still 12.2 cm shorter than the right despite numerous corrective surgical procedures. Ankle disarticulation facilitated below-knee prosthetic fitting. The patient is tumor-free at age 22 years.

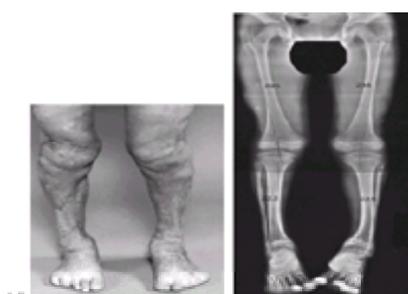
### Infection

Septic arthritis and metaphyseal osteomyelitis often are followed by physeal arrest ([132,133,134,135,136,137,138,139,140,141,142](#) and [143](#)). Because the interval between infection and physeal arrest may be years ([Fig. 5-21](#)), intraarticular and osseous infections need long-term follow-up. The arrest is often partial, resulting in both angular deformity and length discrepancy. Typically, there is no residual infection when the arrest is noted. Physeal bar excision in patients with previous infection may, however, have a higher incidence of postexcision infection. Of 178 physeal bar excisions reviewed at the Mayo Clinic, the only three postoperative infections were in patients in whom the bar was due to metaphyseal osteomyelitis.



**FIGURE 5-21.** Physeal arrest associated with metaphyseal osteomyelitis. **A:** An infant girl had a cutdown inserted into the right ankle saphenous vein on her second day of life. Staphylococcal osteomyelitis in the distal right femur was treated by incision and drainage on day 17 of life. Care was taken to avoid contact of the curet with the physis. A scanogram 4 years 11 months postoperatively demonstrates right distal femoral valgus and relative shortening of femur (1.2 cm). Coronal tomogram of right knee **B:** shows central bar with tenting or cupping. A 4-year follow-up of this case following bar excision is documented in Peterson HA. Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258; with permission.

Physeal arrest also has been reported after some systemic infections, particularly meningococemia (61). In this instance, the arrest may be associated with vascular impairment (Fig. 5-22).



**FIGURE 5-22.** Physeal arrest associated with systemic infection. **A:** A 7+10-year-old boy had meningococemia at age 2 years complicated by ischemia of the lower extremities. He underwent fasciotomies, debridement of necrotic tissue, and extensive skin grafting. The legs, however, survived with only the loss of toes. Subsequently he had heel cord lengthenings and soft tissue flap grafts to cover both patellae. **B:** Scanogram show bilateral ankle varus and physeal bars of the distal tibiae. He later developed a bar of the proximal left tibia.

## Tumor

Several benign tumors often abut the physis, and the physis is susceptible to injury during curettage and bone-grafting procedures. The physis may close even when the tumor is untreated. In addition to unicameral cyst (145,147,148,149 and 150) and enchondroma (144,146), other childhood tumors that occur near growth plates and might cause growth arrest are aneurysmal cyst, chondroblastoma, chondromyxoid fibroma, and fibrous dysplasia.

## Vascular Impairment

The germinal and proliferative layers of physeal cartilage cells are primarily responsible for longitudinal bone growth (151,152,153,154,155,156,157,158,159 and 160). These cells are nourished by vessels in the epiphysis, and any vascular disturbance of the epiphysis can result in physeal damage (157,158,159,160). Some investigators have suggested that the epiphyseal blood supply is probably the most important determinant in the development of bone bridges (3,81). If the blood supply to the metaphysis is also disrupted, the likelihood of a bone bridge increases.

Arterial spasm associated with traction (154) and even the application of a cast reduces blood flow to the extremity (153), which can alter physeal growth (3,158,159). Premature physeal closure has been documented after temporary arterial insufficiency (Fig. 5-23) (155) and after intravenous fluid extravasation (156). Some Salter-Harris type V and Rang type VI injuries reported in the literature may have been caused by vascular dysfunction rather than by physeal germinal cell compression.



**FIGURE 5-23.** Physeal arrest associated with temporary arterial impairment. At age 13 months, this girl with congenital dislocation of the right hip had an open reduction, complicated by severing of the common femoral artery. Multiple arterial anastomotic grafting procedures were successful in saving the extremity. A progressive asymptomatic right ankle varus developed. This coronal tomogram at age 2+6 shows premature partial closure of the distal tibial physis. Further documentation of this case is provided in Peterson HA. Premature physeal arrest of the distal tibia associated with temporary arterial insufficiency. *J Pediatr Orthop* 1993;13:672–675; with permission.

The exact mechanism of premature physeal closure with thalassemia major is unknown (152). In sickle cell disease (61), there is increased viscosity predisposing to sludging, erythrocyte entrapment in small vessels, and blockade of small vessels producing ischemia.

## Neural Involvement

The etiology of impairment of physeal growth linked with nerve deficits associated with poliomyelitis, cerebral palsy (spastic hemiparesis), sacral tumors, and major nerve transection is unknown, but it is probably related to nutrition—more precisely, to diminished vascular supply (161,162). Length inequality in nerve-related disorders tends to progress slowly, and premature physeal closure is unlikely (162). Two exceptions are congenital insensitivity to pain (Fig. 5-24) and meningomyelocele (161). The growth retardation in these conditions is partial or complete arrest and may be caused by increased trauma and repeated transphyseal

fractures in the insensate limb.



**FIGURE 5-24.** Physeal arrest associated with neural involvement. **A:** A 4+3-year-old boy with congenital insensitivity to pain. The left tibia is 22 mm shorter than the right and has premature physeal closure at both ends. The poorly visualized proximal tibial physis and exuberant metaphyseal bone, as well as the deformity and compression of both tali and os calcis, are typical of this entity. **B:** Coronal tomogram of distal tibia shows central physeal bar without the usual indistinct physis and exuberant metaphyseal bone (see proximal left tibia). The patient was treated with bar excision.

#### Metabolic Abnormality ([163](#),[164](#),[165](#),[167](#),[168](#),[169](#),[170](#),[171](#),[172](#) and [173](#))

Vitamin A intoxication can cause premature physeal arrest ([167](#),[168](#),[169](#),[170](#),[171](#),[172](#) and [173](#)). The marked metaphyseal and epiphyseal cupping and growth retardation that sometimes occurs with vitamin C deficiency may also produce physeal closure ([170](#),[172](#)), the condition may correct spontaneously with an appropriate diet. Careful serial radiographic studies are necessary before considering bar excision or contralateral epiphyseodesis ([171](#)).

Tissue resistance to growth hormone in patients with uremia has been implicated as the cause of growth retardation in patients with chronic renal failure ([169](#)). Also, high doses of calcitriol may directly inhibit chondrocyte activity within physeal cartilage and adversely affect linear growth in children with end-stage renal disease ([166](#)). Insulin-like growth factor-binding proteins may also contribute to growth inhibitors in children with chronic renal failure ([168](#)).

With increasing use and success of bone marrow transplantation, more children survive cancer. Chemotherapy and irradiation both have the potential to damage endocrine glands, which contributes to growth impairment through epiphyseal growth plate dysfunction ([164](#)). Immunosuppressive drugs may affect growth by differentially decreasing rates of cellular multiplication in the physis ([163](#)). The central portion of the physis is the most sensitive, and a cone-shaped epiphysis develops. This is similar, if not identical, to the changes seen with vascular and metabolic abnormalities.

#### Cold Injury (Frostbite)

Skeletal abnormalities after cold injury to the hands of growing children are complex and unique ([174](#),[175](#),[176](#),[177](#),[178](#),[179](#),[180](#),[181](#),[182](#),[183](#),[184](#),[185](#),[186](#),[187](#),[188](#),[189](#),[190](#) and [200](#)). Premature closure of the phalangeal physes is believed to result from direct injury to vulnerable chondrocytes in the cartilaginous physis ([195](#)) or from ischemic vascular changes ([178](#),[182](#),[191](#),[197](#)). Children of intermediate age (5 to 10 years) appear most susceptible, probably owing to a combination of carelessness and decreased adult supervision. Radiographic signs of physeal closure become evident 6 to 12 months after cold exposure, and follow a pattern of decreasing frequency from the distal to the proximal physes ([Fig. 5-25](#)). The index and little fingers are most often involved, followed by the ring finger and the middle finger ([176](#)). The thumb metacarpal, protected by the surrounding thenar muscles and the proximity of the radial artery, is rarely involved ([61](#),[187](#)). Presenting complaints are joint pain, stiffness, and weakness of fingers. Late sequelae include relative shortening of digits, skin redundancy, joint laxity, and degenerative joint changes ([184](#),[190](#),[200](#)).



**FIGURE 5-25.** Physeal arrest due to cold. An 11-year-old boy 1 year after frostbite complains of swelling and tenderness of finger joints accentuated by activity. Radiographs show absence or malformation of the epiphyses of 12 finger phalanges and irregularity of corresponding articular surfaces. The thumbs are normal clinically and radiographically. (From Wenzl JE, Burke EC, Bianco AJ. Epiphyseal destruction from frostbite of the hands. *Am J Dis Child* 1967;114:668–670; with permission.)

The radiographic changes are characteristic. Usually, the physis disappears completely, resulting eventually in a short finger. The physis and epiphysis often have a V shape. There may be no obvious destruction of the epiphysis, but eventual premature fusion of part of the epiphyseal line may lead to angular deformity. The affected phalanges are shorter and smaller than normal, and the juxtaarticular bone is expanded and irregular, with a coarse cancellous spongiosa. The same expanded and irregular appearance is seen on the contiguous articular surface of the more proximal phalanx, where there is no epiphysis ([3](#)).

In children, there is a striking lack of correlation between the extent of initial soft tissue injury and eventual skeletal changes. Many patients do not seek medical attention for the initial injury. The relative absence of soft-tissue ischemic changes, combined with late evidence of physeal damage, supports the hypothesis of direct cellular damage to physeal cartilage as the likely etiologic mechanism ([190](#)).

Very few children require surgical treatment ([184](#)). When deformities do require treatment, physeal arrest, arthrodesis, angular osteotomy, soft tissue arthroplasty, or tenorrhaphy can be performed as indicated ([175](#),[189](#)). Function of the hand, however, generally remains satisfactory without surgery.

#### Heat Injury (Burn)

The mechanism causing physeal damage in severely burned limbs is not well understood ([3](#),[201](#),[202](#),[203](#),[204](#),[205](#),[206](#) and [207](#)). Because physeal cartilage is more sensitive to irradiation and cold than articular cartilage, it may also be more sensitive to heat ([207](#)). The peripheral zone of Ranvier, being more superficial, is more readily subject to the effects of heat ([3](#)). Prolonged ischemia of soft tissues around the physis might impair physeal growth, or the restrictive or strangling effect of thick scar about the metaphysis and adjacent joint areas might inhibit physeal growth ([175](#),[202](#),[205](#)).

#### Electrical Injuries

Physeal arrest caused by electrical injury, possibly including lightning, is rare ([208](#),[209](#),[210](#),[211](#) and [212](#)). Multiple factors determine the effects of electric current on chondro-osseous tissue: the type of current (alternating current is three to four times as dangerous as direct current), voltage, amperage, duration of contact with the

electric current, the path taken through the body, the resistance at the points of contact and exit, and the patient's general state of health ( 209). During electrical accidents, tissue temperatures may momentarily reach several thousand degrees Celsius and may cause heat-induced liquefaction and necrosis of cartilage and bone. In general, the tissues offering the greatest resistance to tissue flow suffer the greatest damage. Electrical injury may result in cell death or may alter cellular activity temporarily or permanently (210). After electrical accidents, tissue repair, including callus formation, is poor.

Osseous changes in children are similar to those in adults but may also include additional abnormalities secondary to the effect of the current on the physal cartilage (211) The epiphyseal center and physal cartilage may be affected by the current, and the metaphyseal region remodels poorly. Partial premature arrest of the distal femur has been reported (208). There are only two reported cases of excision of physal bars caused by an electrical burn ( 212).

### Laser Injuries

A laser beam applied directly to physal cartilage damages the cartilage selectively without affecting the adjacent bone ( 213). The damaged physis is replaced by bone, which forms a bone bridge between the metaphysis and epiphysis identical to bone bridges after fracture. Care must be taken when using lasers near physes in growing children (Fig. 5-26).



**FIGURE 5-26.** Physeal arrests from laser injury. At age 5+6 years, this girl had warts removed from the opposing sides of the left dominant ring and long finger at the level of the distal interphalangeal joints. The parents report that the procedure was aggressive and that the wounds took 3 to 4 months to heal. Physical therapy aided in regaining joint motion. Progressive angulatory deformity occurred. At age 6 years 2 months, corrective closing wedge osteotomies on the middle phalanges were performed and internally fixed with internal wire. Angulatory deformity recurred. **A:** By age 10 years 9 months, there is deformity and relative shortening of both distal phalanges. The distal phalanx of the long finger has a physeal bar on the ulnar side, and increasing angulatory deformity is anticipated. The distal phalanx of the ring physis finger developed a bar on the radial side that caused deformity of the distal phalanx. This physis is now completely closed, and no further deformity or growth is anticipated. **B:** Normal right ring and long fingers. **C:** Clinical appearance both hands (age 11 years 4 months).

### Stress Injuries

#### Widening and Irregularity

Widening and irregularity of the physis without accompanying displacement of the epiphysis have been recognized as “stress-induced” changes in adolescent athletes (214,215,216,217,218,219,220,221,222,223,224,225,226,227,228,229,230,231 and 232). These characteristics are common in the distal radial and ulnar physes of elite gymnasts and often are bilateral (214,217,218,221,224,229,230 and 231). Stress injury of the distal radial physis also occurs in a significant percentage (up to 25%) of nonelite gymnasts (223,224). These lesions initially involve the volar aspect of the radial epiphysis and subsequently the entire physis. In about 20% of patients, similar changes are present in the distal ulnar physis. Similar changes involve the physes of the distal femur ( 226), proximal tibia (219), and distal fibula (226) in adolescent runners, and in the proximal ulna ( 222) and the proximal humerus in baseball pitchers (220,221) and racket sports (216). The changes are typically unilateral when they are associated with throwing or racket sports.

#### Signs and Symptoms

Patients describe pain localized to the site of the involved physis. Symptoms usually develop during training and become more intense as the workout progresses. Initially, the pain is relieved by rest. Clinical examination reveals painful limitation of extremes of motion at the affected site. Tenderness is localized to the line of the physis.

#### Radiographic Changes

Radiographs show evidence of widening of the physis and irregularity and sclerosis of the metaphysis without accompanying displacement of the epiphysis ( Fig. 5-27). In patients with long-standing symptoms—implying continued activity—premature physal arrest may occur ( 215,218,225). Growth arrest has not been described at sites other than the distal radius.



**FIGURE 5-27.** Stress injury of the physis. Persistent widening and irregularity of the distal radial physis in a 13-year-old female elite gymnast. A year earlier, right wrist pain resulted in radiographs that showed similar but less severe changes. Treatment consisting of cast and rest resulted in improvement of both pain and radiographic appearance. Return to competition resulted in present picture. Earlier radiographs of this patient were published in Ruggles DL, Peterson HA, Scott SG. Radial growth plate injury in a female gymnast. *Med Sci Sports Exerc* 1991;23:393–396; with permission.

MRI has suggested metaphyseal and epiphyseal ischemia of the physis (223) or metaphyseal injury (232).

#### Etiology

Most authors assume that these changes are due to repetitive shearing or compressive stresses, as might occur with vaulting or floor exercises. However, traction stress, as could occur with the uneven and parallel bars, is also possible, especially in gymnasts who use dowel grips ( 231). Most gymnasts compete in all events. In skeletally immature athletes, the growth plate is weaker than the ligaments, joint capsules, and bone about the joint, so stresses are focused in the physis.

Compressive forces, both intermittent and sustained, have been shown to injure chondrocytes ( [227](#)).

A single episode of stress or injury could result in occult microscopic fissures within the physis, which could, in time, proceed to widening of the physis and irregularity of the opposing bone margins of the metaphysis and epiphysis. Bright and colleagues ( [80](#)) reported that histologic examination of the tibiae of rats loaded to 50% failure energy revealed internal cracks within the physis. These cracks appeared in all layers of the cartilage but were most common in the hypertrophic zone. Subsequent growth of the cartilage resulted in widening of the physis. Thus, the initial radiograph would be normal, but a subsequent radiograph 2 to 3 weeks later may demonstrate radiographic changes within the physis.

### **Treatment**

The treatment of stress injuries is symptomatic and consists of reduction or temporary cessation of the activity that created the injury. It may take up to 6 months for the patient to become asymptomatic. An increased prevalence of ulnar positive variance in gymnasts can be attributed to premature closure of the distal radial physis. Surgical arrest of the distal radius and ulna has been used for bilateral irregular closure of the distal radial physis ( [215](#)). There are no reports of bar excision for this problem.

### **Longitudinal Compression (Salter-Harris Type V)**

The Salter-Harris type V classification ( [28,233,234,235,236,237,238,239,240,241,242,243,244](#) and [245](#)) proposes that a single, sudden, longitudinal force applied to an immature bone can compress the physis sufficiently to kill the cartilage growth cells without causing fracture of adjacent trabecular or cortical bone. Radiographs at the time of injury do not show osseous abnormality. The diagnosis is suspected only months to years later, when growth arrest becomes manifest. Salter and Harris's original drawing shows partial arrest with subsequent angular deformity. Their illustrative case ( [28,29](#)) does not include the initial normal radiographs but does show a healed proximal tibial fracture with many features of their type IV fracture (displacement and angulation of the medial condyle only).

Examples of this compression injury are rare in the literature. Most cases are recorded as part of a series with no individual details; there are a few case reports ( [233,234,235,236,238,245](#)). When details of individual cases are given, invariably it is an associated nonphyseal fracture in the extremity that has been treated by immobilization, cast, or traction; thus, disuse and possible arterial impairment are also present. In addition, in all reports, the arrest was complete, not partial, as depicted by Salter and Harris, further implicating the possibility of disuse or arterial insufficiency.

### **Developmental Physeal Abnormalities**

Deformities of the proximal tibia (Blount) ( [246,247](#)) and distal radius (Madelung) ( [250,251](#)) appear to result from gradually progressing physeal and epiphyseal abnormalities that may eventually result in partial physeal arrest ( [248](#)). These conditions are never noted at birth, and the etiology is unknown.

A bracket epiphysis or delta phalanx may be noticeable at birth, is therefore most likely congenital, and responds well to excision of the physeal bar ( [252](#)).

Preexisting bone deformity may predispose the patient to physeal fracture ( [249](#)). In addition, premature physeal arrest may occur at any physis at any age with no apparent etiology ( [104](#)), although this is rare.

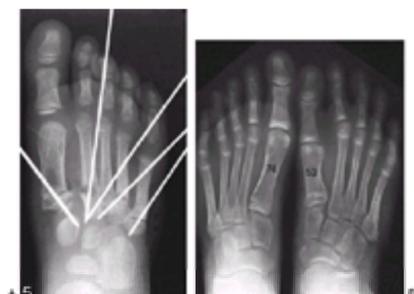
### **Iatrogenic Injuries**

#### **Surgical Insults**

In a sense, several of the above-mentioned "other physeal injuries," such as those caused by cast application, traction, radiation, the prescribing of drugs (metabolic), and the use of laser can be considered iatrogenic, or physician induced. The ones discussed here ( [253,254,255,256,257,258,259,260,261,262,263,264,265,266,267,268,269,270,271,272,273](#) and [274](#)) are primarily problems related to surgery. During the treatment of deformity, fracture, infection, or tumor, the physis may be damaged. Subperiosteal dissection extending to the perichondrial ring of Ranvier may result in premature closure of that area of the physis. It also may be impossible to avoid the physis during curettage of infected tissue or tumors, corrective osteotomy for malunion or deformity ( [261,265,270,274](#)), or internal fixation of physeal fractures. Each of these procedures can damage a physis sufficiently to affect growth and should be avoided when possible. Injury of the periphery of a physis is more likely to result in physeal arrest than is an insult to its center ( [3](#)).

#### **Transphyseal Pins**

Multiple factors determine whether the presence of a pin or pins across a physis will affect growth ( [256,271](#)): the presence or absence of threads, the pin's obliquity to the physis, the pin's location in the physis (central or peripheral), the number and size of pins, and the amount of time the pin is left in place. Usually, a single, smooth, small pin perpendicular to the center of the physis, left in place a short time (e.g., 3 weeks), does not result in physeal closure. There are exceptions ( [Fig. 5-28](#)). Of all of these factors, the presence of threads is most likely to result in physeal closure. The presence of a traction pin across, or even close to a physis, is sometimes associated with subsequent physeal closure, particularly in the proximal tibia ( [Fig. 5-29](#)) ( [106,254](#)). Whether the growth arrest is secondary to the pin or is an occult physeal injury associated with diaphyseal fracture is unknown.



**FIGURE 5-28.** Physeal arrest from transphyseal pin. This boy with clubfoot was treated in infancy by serial casting. **A:** At age 4+5, he underwent osteotomy of all five metatarsals. The osteotomy of the first metatarsal was 1 cm distal to the proximal physis. The single, smooth 0.062 Kirschner wire was perpendicular to and in the center of the proximal physis of the first metatarsal. **B:** Eight years after surgery, the right proximal first metatarsal physis is closed; the length of the right first metatarsal is 53 mm, and the left metatarsal is 74 mm.

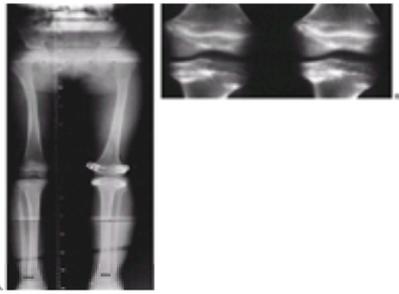


**FIGURE 5-29.** Physeal arrest from traction pin. This 11+6-year-old girl has developed left genu recurvatum following proximal tibial skeletal traction for a distal femur

fracture 3 years 6 months previously (age 8+0). Lateral x-ray studies of both knees showed that there is 20 degrees recurvatum of the left proximal tibia. There is a physal bar of the left anterior tibial tubercle.

### Staples

Staples are commonly used to retard longitudinal growth or to correct angular bone growth (255,263,273). If they are left in place too long, they can cause permanent arrest (257). Of equal concern is the occasional peripheral bar that forms after staple removal (Fig. 5-30) (264).



**FIGURE 5-30.** Physeal arrest associated with staple removal. **A:** Orthoroentgenogram. A 10+11-year-old girl with right spastic hemiparesis. Twenty-one months previously, staples were placed in the normal left distal femur and proximal tibia, resulting in overcorrection of 11 mm (the normal left lower extremity is 11 mm shorter than the right). The staples were removed at this time. **B:** Thirteen months later at age 12+0, there was progressive left genu varum relative shortening (the left lower extremity is now 23 mm shorter than the right). Two coronal tomograms of the left knee confirm physeal bars on the medial side of both the distal femur and the proximal tibia. Note increasing distance between the tracts from the tines of the staples on the lateral side of the distal femur and proximal tibia and no increase in this distance on the medial side. Note that the angles of the tine tracks laterally are parallel, and medially are converging. Also note growth arrest line of the distal femur, with growth present laterally and not medially. Therefore, these medial arrests had to occur at time of staple removal.

### Other Iatrogenic Injuncts

Drilling across a physis is rarely done. The larger and more numerous the drill holes, the greater the likelihood of physeal arrest (259,260,262,269). However, drilling across the distal femoral and proximal tibial physes for anterior cruciate repair has not resulted in premature arrest (253,267,268), probably because the drill hole is filled with a substance (tendon or foreign material) that acts much like an interposition material used for bar excision. The mean growth of the proximal tibial physis of 24 children treated with an uncemented sliding tibial component that crossed the physis perpendicularly was 69% of that of the contralateral normal side (258).

## PHYSEAL ARREST

### Etiology

Premature complete physeal arrest produces bone-length retardation with no angular deformity (275,276,277,278,279,280 and 281). If the physis at each end of the bone is arrested, there is no growth at all.

Premature partial arrest of growth of a physis retards bone lengthening and may cause progressive angular deformity of the involved bone. The arrest is produced when bone forms from metaphysis to epiphysis, crossing the physis. This continuity of bone is known as a bone bar or a bone bridge. As the remaining physis grows, angular deformity occurs (Fig. 5-31). The site, size, location, and duration of the bar determine the clinical deformity. If the bar is located laterally in a physis—for example, the distal femur—the normal physis medially continues to grow, producing genu valgum deformity (278,279,280 and 281). If the bone bar is anterior, the normal physis grows posteriorly, producing genu recurvatum (Fig. 5-29) (254). If the bar is central, the periphery may grow, causing cupping, tenting, or dip deformity of the metaphysis, combined with relative shortening of the bone but little, if any, angular deformity (Fig. 5-21B and Fig. 5-23) (167,260,275,276).



**FIGURE 5-31.** Physeal arrest following transphyseal fracture. **Left:** A 3+7-year-old boy sustained a Salter–Harris IV fracture of the distal tibia with proximal displacement of the medial malleolar fragment. Treatment was cast immobilization without reduction. **Right:** Same child 22 months later (age 5+5). Note physeal bar medially, growth arrest line perpendicular to longitudinal axis of the tibia, asymmetric growth of remaining normal physis, producing a 45-degree varus angulation of the physis, and adaptive contouring of the lateral edge of the tibial epiphysis, allowing ankle varus to be less than the physeal varus. There is normal growth of the fibula (not overgrowth). The cartilaginous medial malleolus is unossified. Long-term follow-up of this case has been published in Peterson HA. Operative correction of post-fracture arrest of the epiphyseal plate: case report with 10-year follow-up. *J Bone Joint Surg Am* 1980;62:1018–1020; with permission.

Bone bars may result after any injury to physeal cells. The most common cause is fracture, although bars may occur after other types of physeal damage [e.g., disuse, radiation, infection, tumors, vascular abnormalities; iatrogenic injuries (see previous section)]. For some bone bars, no etiology is apparent; these have been called congenital or developmental (e.g., from Blount-Barber syndrome). However, bone bars have never been reported at birth.

Any fracture that involves the physis may result in a bone bar. Posttraumatic bars result from damage to the germinal or palisading layer of physeal cells. The physeal cell damage probably occurs at the time of injury, but it may occur during fracture reduction (closed or open), or it may be associated with internal fixation. Bone bars can be anticipated after comminuted type IV and VI injuries and, if they are followed closely, they can be detected as early as a few weeks after the injury. Some bone bars do not become clinically evident until years after the injury, underscoring the need to follow any significant physeal injury for years, if not until maturity. This is particularly true after metaphyseal osteomyelitis (Fig. 5-21).

### Anatomic Factors

Anatomic differences in the various physes also are important in the production of a bone bar. Factors include the size of the physis, its rate of growth, and the

contours of the physis (that is, whether the physis lies on one plane or is irregular). Although physes of the phalanges and distal radius are by far the most frequently injured (33,36,38), they are small and uniplanar, and are an uncommon site of a bone bar. In contrast, the physes of the proximal tibia and distal femur are large and irregular in contour (multiplanar), and account for 60% to 70% of the growth of their respective bones. Together, they account for only 2.2% of all physeal injuries (Table 5-2) (36,38,281), but they are responsible for 50% of the bone bars requiring treatment (Table 5-3).

	No.	%
Distal femur	61	34
Distal tibia	51	29
Proximal tibia	29	16
First metatarsal	5	3
Proximal femur	2	1
Distal fibula	2	1
Proximal phalanx, great toe	2	1
Distal radius	15	8
Distal ulna	5	3
Proximal humerus	3	2
Phalanges	2	1
Metacarpal	1	0.5
Pelvis (triradiate)	1	0.5
	178	100

**TABLE 5-3. PHYSEAL BRIDGE RESECTION**

### **Influence of Age**

The patient's age at the time of physeal injury is perhaps the paramount factor. Injury of the physis of a 14- or 15-year-old girl or a 16- or 17-year-old boy is of little consequence because they have so little growth remaining that deformity is unlikely to become clinically manifest. Any bone bar in an infant or young child, however, is a significant problem with wide-ranging clinical effects. Long clinical follow-up is mandatory in these children.

### **Assessment**

#### **Clinical Examination**

Bone bars are usually first noted clinically because of the angular deformity or relative shortening of the involved extremity (282,283,284,285,286,287,288,289,290,291,292,293,294,295,296,297,298,299,300,301,302,303,304,305,306,307,308,309,310 and 311). The history, physical examination, and routine radiographs localize the involved physis. Clinical evaluation of limb-length discrepancy, angular deformity, joint motion, and functional impairment should be recorded. Radiographs of the appropriate body area must be taken in at least two planes, usually coronal and sagittal. The relation of the growth-arrest line, the physis, and the joint surface needs close scrutiny (Fig. 5-31) (50,295,302,303). Much can be learned from good-quality plain radiographs. Depending on these findings, additional studies become appropriate.

#### **Imaging Studies**

##### **Skeletal Age**

Skeletal age must be determined in older children to assess the potential for remaining growth. There must be enough growth remaining (2 years or 2 cm) to make the option of bar excision worthwhile. Comparison of a radiograph of the hand with an atlas is the most commonly used (291,309).

##### **Leg-Length Measurements**

Both the involved and the uninvolved extremity are measured clinically, and the lengths are documented by radiographs. Three radiographic methods are in common use: teleoroentgenography, orthoroentgenography, and scanography.

A teleoroentgenogram (293) is a single radiograph taken from a distance great enough (usually 6 feet) to reduce magnification and to include all long bones of each extremity. It has the disadvantage of magnification, which increases as the child grows, making serial evaluation less precise.

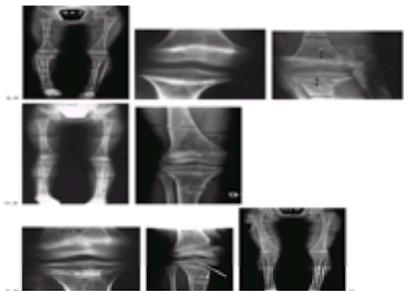
An orthoroentgenogram (290) is a multiple-exposure radiograph designed to obtain a straight projection through each joint of the extremity to obviate magnification. It has the disadvantage of recording an incorrect length if the child moves between exposures. Additional films are necessary to assess alignment.

A scanogram (308) uses an x-ray tube in linear motion with a slit diaphragm. Any movement by the patient is detected by motion. It includes all osseous structures so that any angular deformity or bone abnormality can be detected (Fig. 5-22B). There is no magnification. The film can be measured directly with a tape measure, and thus can be remeasured if there is any discrepancy with the clinical findings. These true measurements may be compared with previous and future scanograms in longitudinal studies. The quality of film detail is sufficient so that no additional coronal view is necessary to assess alignment; this reduces expense. Scanograms are superior to other methods, with the only disadvantage being the modification of radiographic equipment (306,308) (Fig. 5-15A and Fig. 5-30A).

Scanograms, having no magnification, become even more valuable when analyzing growth between two metal markers after treatment (Fig. 5-22B, Fig. 5-32A, Fig. 5-32B and Fig. 5-33A, Fig. 5-33D, Fig. 5-33H) and make more elaborate techniques such as stereophotogrammetry (286) unnecessary.



**FIGURE 5-32.** Metal markers using Kirschner wires. **A:** Close-up of a scanogram 5 months after bar excision and insertion of Cranioplast in a girl, now age 5+4, whose preoperative radiographs are shown in Figure 5-21. The physis is open, and the two metal markers inserted at the time of bar excision are now 28 mm apart. The Cranioplast plug is close to the proximal marker and to the physis. **B:** Close-up of scanogram 4 years postoperatively. Both femora had grown 9.8 cm; thus, growth of this femur is 100% of the contralateral femur. The metal markers are 83 mm apart, indicating 55 mm of growth from the distal femur since **A**. The use of the scanogram obviates any magnification of distances between markers. Angulation between the Kirschner wires is unchanged. The femoral shaft–femoral condyle angle has improved from 63 degrees to 64 degrees. Note that the plug initially stayed with the epiphysis, as evidenced by the increased distance of the plug from the proximal metal marker (compare with **A**). Later, the epiphysis grew away from the plug, as evidenced by the increased distance of the plug from the distal marker (compare with **A**). The ultimate distance between the markers was 110 mm. This case was illustrated more completely in Peterson HA. Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258, with permission.



**FIGURE 5-33.** Bar recurrence successfully treated by bar reexcision. **A:** Scanogram of a girl age 4+7 who had a febrile illness with swelling of the right knee in infancy. The right leg is 35 mm shorter than the left (femur -19 mm, tibia -16 mm). **B:** Coronal tomogram depicts bars of both distal femur and proximal tibia. **C:** At the time of excision of bars and insertion of Cranioplast at both sites, metal markers (half of a silver vascular clip) were inserted in the center, longitudinal to each other. The distance between the femoral markers is 17 mm and between the tibial markers 18 mm. A third marker in the distal femur was inserted too far peripheral, laterally, thereby decreasing its value for measuring future longitudinal growth. With growth and bone remodeling, it became extraosseous, negating its value as a marker. At the time of surgery, a marker was also placed in the mid left contralateral normal tibia, which provided useful information in assessing growth and recurrence later. Scanograms at 6 months showed the right leg to grow faster than the left leg (2 years 3 months postoperatively, the leg-length discrepancy had decreased from 35 to 23 mm, and the growth of the right leg was 121% of the normal left). Subsequently, there was progressively less growth. **D:** Scanogram 4 years 8 months postoperatively (age 9+3) shows continued growth of both operative areas (metal markers farther apart) but with a progressive reduction in the rate. During the previous 6 months, the right femur grew 15 mm, the left 17 mm, 88%; the right tibia 12 mm, the left 14 mm, 86% for this interval. Overall growth of the right leg since surgery is now 102% of the uninjured left leg. **E:** Close-up of **D** shows femoral markers 78 mm and tibial markers 52 mm apart (total growth 95 mm). The Cranioplast plugs stayed in the metaphyses. They did not migrate; rather, the epiphyses grew away from them. Although the metal markers in the proximal tibia were continuing to become farther apart, much of the growth of the tibia was distally, as determined by comparison with the metal marker in the normal mid-left tibia. This caused suspicion of a developing recurrent bar. The lateral peripheral marker in the femur ( *arrow*) is now nearly extracortical because of diaphyseal remodeling. **F:** Coronal tomogram confirms recurrent bar formation in both the tibia and femur. Note diminished cupping compared with [Figure 5-33B](#). **G:** Repeat bar excision of both the proximal tibia and distal femur was performed at 9+5 years. Note clear visualization of remaining physes of both bones. **H:** Scanogram at age 11+8 years, 7 years after first surgery. The femoral markers are 109 mm apart, and the tibial markers are 72 mm apart. All physes are closed. There has been no surgery on the left leg and no osteotomies or lengthening on the right leg. The right leg is 26 mm shorter than the left (femur -15 mm, tibia -11 mm). Growth of the right femur from the time of original surgery was 14.5 cm on the right, 14.1 cm on the left (the operated right femur grew 103% compared with the normal left). The operated right tibia grew 12.3 cm, and the left tibia grew 11.8 cm (the right tibia grew 104% compared with the left). An orthoroentgenogram sent from home at age 12+10 showed that the total leg-length discrepancy was 21 mm (compared with 35 mm preoperatively). All physes were closed, and no further treatment was recommended. There was no surgery on the normal left leg. This case illustrates the need to follow patients continuously (6-month intervals) until maturity because bar formation can recur at any time. Scanograms are indispensable for accurate measurement of both total bone length and distance between markers. Placement of a metal marker in the contralateral unoperated bone is the only way to determine accurately the relative growth of the operated physis and its ipsilateral physis (same bone, other end) compared with the two physes of the contralateral bone. The bar recurrence was successfully treated by bar reexcision. At completion of growth, growth of the operated right leg exceeded growth of the normal left leg by 14 mm. This case was illustrated in Peterson HA. Partial growth plate arrest and its treatment. In Morrissy RT, ed. *Lovell and Winter's pediatric orthopaedics*, 3rd ed. JB Lippincott, Philadelphia, 1990:1071–1089.

Additional methods of documenting bone length, including CT scan, MRI, and microdose digital radiography ([282,306](#)), are not commonly used because of their cost, poor osseous detail, or alteration of length (too long or too short) on the film, requiring the technician to determine loci for measuring the length at the time of film procurement ([306](#)). Usually, films by these techniques cannot be measured directly by ruler.

#### Localization of the Bar

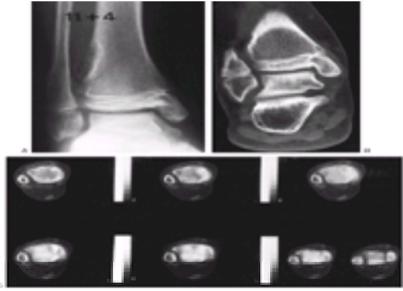
A documenting image (map) of the location, area, and contours of the bar is essential in determining the feasibility of bar excision and the surgical approach. Several methods are available, including tomography, CT, scintigraphy, and MRI.

**Tomography.** Until recently, tomography was the most commonly used method of evaluating the location, extent, and contours of the bar. Tomographic techniques include linear, circular, ellipsoidal, spiral, and hypocycloidal ([306](#)). The latter two are termed multiplanar ([Fig. 5-34](#)). False-positive and false-negative findings have been reported with standard uniplanar tomography. Thin (1-mm) multiplanar tomograms taken every third millimeter in two planes, usually the coronal and sagittal, are used to construct a map of the physis as if it were laid out on a flat surface ([287](#)). The contours and area of both the bar and the entire physis can then be determined. Because of the multiple views required, the amount of radiation exposure is high ([289](#)) and the mapping is time-consuming and subject to interpretation errors.



**FIGURE 5-34.** Physeal arrest visualized by tomography. **A:** An 11+4-year-old boy sustained a type V fracture of the distal radius 3 years earlier. This coronal 1-mm-thick trispiral tomogram shows significant deformity and growth alteration, but no physeal bar. **B:** A cut 3 mm away reveals a large physeal bar. It is difficult to envision a transverse CT scan in any plane that would add information about the location, size, or contours of this bar. (From Peterson HA. Partial growth plate arrest. In: Morrissy RT, ed. *Lovell and Winter's pediatric orthopaedics*, 3rd ed. JB Lippincott, Philadelphia, 1990:1071–1089; with permission.)

**CT Imaging.** CT images are difficult to make in coronal and sagittal projections because of difficulty placing the body part in the gantry. When possible, CT scans in an axial projection show the bridge, although less clearly than multiplanar tomograms ([Fig. 5-35](#)). The cuts are thicker and fewer in number, making map determination less precise than with multiplanar tomography. CT scans made in the transverse plane are difficult to interpret because of normal undulations of the physis, with increased contour irregularity produced by the bar and its accompanying physeal contour abnormality. The entire physis can rarely, if ever, be captured on one cut. The adjacent normal juxtaphyseal bone sclerosis can easily mimic a bony bridge. In the 1980s, CT scanning had no advantage over multiplanar tomography or MRI and were sometimes misleading ([288,301,307,310,311](#)). The recent advent of helical computed tomography has significantly improved the images so that they are now superior to tomography and rival MR imaging ([299](#)).



**FIGURE 5-35.** Physeal arrest visualized by CT scans. **A:** Plain radiograph of an 11+4-year-old boy 4 months after an undisplaced type V fracture illustrates a medial bar. The growth arrest line is wider laterally. **B:** Tangential coronal views of the ankle obtained by flexing the knee maximally in the gantry. This CT scan gives no additional information compared with the plain radiograph or tomograms. **C:** Transverse CT scan through the distal tibia. The physis is not visualized on any cut. The physis and bar are poorly defined despite the fact that this should be an ideal situation (a relatively even-contoured physis with minimal deformity caused by the bar). (From Peterson HA. Partial growth plate arrest. In: Morrissy RT, ed. *Lovell and Winter's pediatric orthopaedics*, 3rd ed. JB Lippincott, Philadelphia, 1990:1071–1089.)

**Scintigraphy.** Scintigraphy, using technetium, a pinhole collimator, and a gamma camera aimed axially toward the epiphysis, has been used to assess physeal bars (286,294). It can be used only when there is no bone intervening between the epiphysis and the camera—for example, the distal femur with the knee flexed. All other physes would have intervening bone, making the technique inapplicable. The computer-generated maps are imprecise (306).

**Magnetic Resonance Imaging.** Adaptation of MRI for evaluation of physeal bars (301,307,310,314,315,322) has several advantages. The images are of excellent quality, and there is no radiation (as for tomograms and CT scans). Acquisition time averages 8 minutes, and sedation is usually not necessary. Thin cuts can be assembled contiguously in any plane from the original computerized data. These produce images clearer than reconstructed CT images. The data can be processed to depict the entire physis (and its bar) on one plane, despite contour irregularities caused by the bar. This precludes the laborious and less accurate mapping (287) (which can also be done on the multiple-cut MRI images). Two computerized techniques are available.

**Three-Dimensional Rendering.** A computer-generated illustration is formed by loading sagittal or coronal images from an orthogonal series into a computer work-station that contains rendering software (the program ANALYZE is an example) (283). Each image is processed (rendered) manually by the technician, defining the physis (this may take several hours of technician time). The rest of the image (bone and all soft tissue) is then discarded. A series of sectional images of the physis is produced. The computer combines successive images into a three-dimensional model. When this three-dimensional model is viewed in an axial plane, the result is a physeal map with a defect corresponding to the physeal bar (Fig. 5-12C). The computer can determine the number of pixels in the entire physis and in the bar, so that the bar can be calculated as a percentage of the entire physis (Fig. 5-11C).

**Three-Dimensional Projection.** A computer-generated image can also be formed from a fat-suppressed three-dimensional volume acquisition in the axial plane (283,304) (Fig. 5-36). Slice thickness may be as thin as 0.7 mm (skip 0, up to 60 slices total). This three-dimensional information is processed on the MRI console, using maximum-intensity projection image-analysis software used in MR angiography (standard software for all institutions performing MR angiography). No manual segmentation of the physis is required. The clarity of the result is improved by excluding surrounding soft tissues from this projection volume. The result is a true anatomic image based on volume data (voxels), depicting only the physis and the physeal bar, and does not depend on a technician rendering each image. Technician time is needed only to outline the periphery of the physis and of the bar with a computer mouse.



**FIGURE 5-36.** Physeal arrest visualized by MRI. An 8-year-8-month-old-boy injured his right knee jumping on a trampoline. **A:** He sustained a displaced type III fracture. **B:** Treatment was closed reduction and percutaneous pinning. **C:** Two years 4 months later (age 11+0), the right femur was 32 mm shorter than the left and there was absence of the central portion of the distal physis. Note growth arrest lines on the proximal and distal tibia and the distal left femur but not the distal right femur. **D:** Coronal MR image shows what appears to be multiple central physeal bars. **E:** Projection MRI shows the distal femur and patella cartilage (gray) and central bone (black). This volume acquisition obviates contour abnormalities of the physis and depicts structures as a flat single plane surface. This is not a transverse cut. Thus, it is not necessary to determine the precise longitudinal level (as for a CT scan). The dense white images are vascular structures. **F:** The technician has outlined the entire physis with a computer mouse (*white line*). This technique is much more sensitive than other techniques, and the bars have been found to be much more irregular than had been previously thought. The irregularity of these two bars accounts for the appearance of multiple bars on the coronal MR images (**D**). **G:** The technician has outlined the medial bar with the computer mouse. The number of voxels in the bar compared with the entire physis is 6.3%. The lateral bar measured 3.6% for a total bar of 9.9%. Note position of the bars compared with position of the crossed pins on Figure 5-35B.

In comparing three-dimensional renderings and three-dimensional projections, the results were similar but were more precise using three-dimensional projection. Three-dimensional projections are less expensive because technician interpretation and special software are unnecessary. In all patients who underwent surgery, the bar found surgically, matched the illustration on the image.

We have found the projection method to be the most reliable, safe, cost-effective, and least time-consuming method, and we now use it exclusively for physeal bar evaluation (304).

## Management

### Complete Arrest

Premature complete closure of a physis causes cessation of growth at that physis (312,313,314,315,316,317,318,319,320,321,322,323,324,325,326,327,328,329,330,331,332,333, 334,335,336,337,338,339,340,341,342,343,344,345 and 346). Because the physis is completely closed, there is no progressive angular deformity. Continuing growth of the contralateral physis produces length inequality between the two bones involved. The amount of inequality is determined by the physis injured (specifically, its contribution to the growth of that bone) and the patient's age at the time of growth cessation. In older children with little growth remaining, no treatment is required. In younger children, consideration for treatment depends on the specific physis injured and the amount of length discrepancy calculated to be present at maturity. Treatment options include a shoe-lift for lower limb discrepancy, physeal arrest of the contralateral or companion (radius/ulna or tibia/fibula) bone, ipsilateral bone lengthening, contralateral bone shortening, or a combination. Because no physis remains, bar excision and physeal distraction are not options.

## Upper Extremity Physes

Complete arrest of the proximal humerus physis rarely results in sufficient discrepancy to produce functional impairment. If the discrepancy exceeds 6 cm, bone lengthening may be considered ([142](#)). The contralateral humerus should never be arrested or shortened.

Complete arrest of the physes of the proximal radius or ulna likewise never causes sufficient forearm length discrepancy to consider physeal arrest of the contralateral forearm. Arrest of the distal radial physis at a very early age could cause sufficient distal radial-ulnar variance to consider arrest of the distal ulnar physis, ulnar shortening, or even radial lengthening. Because much greater length occurs from the distal ends of these bones than from the proximal ends, postinjury arrest of the distal end of either is often treated by surgical arrest of the other or by lengthening of the involved bone.

## Lower Extremity Physes

In the lower extremities, limb-length inequality causes pelvic tilt and spine curvature, which predispose the patient to low back pain. Arrest of the capital femoral physis at a young age (e.g., from hip dysplasia, Perthes' disease, trauma, avascular necrosis, or even slipped capital epiphysis) can result in significant femoral length discrepancy. Surgical arrest of the contralateral capital femoral physis is not warranted because of its surgical inaccessibility and potential for avascular necrosis. It would be more appropriate to have the patient wear a shoe-lift, arrest the contralateral distal femoral physis at a later date, or lengthen the ipsilateral femur at times determined by growth charts or at maturity.

Complete premature arrest of a distal femoral physis can be treated by permanent use of a shoe-lift, physeal arrest of the contralateral femoral physis, femoral lengthening, or contralateral femoral shortening at maturity. The choice depends on the degree of calculated discrepancy at maturity and the body height and desires of the patient.

Complete arrest of the proximal or distal tibial physis can be treated similarly to the distal femur, with the addition of physeal arrest of the ipsilateral fibula if significant relative overgrowth of the fibula is likely. Contralateral tibial shortening should never be undertaken as an elective procedure, because any significant surgical tibial shortening will result in weakness of the anterior tibialis muscle and footdrop.

## Partial Arrest

### Treatment Alternatives

Premature partial closure of the physis can be treated in many ways ([280,331,332](#) and [333](#)). If the patient is a teenager approaching maturity and little growth remains in the involved physis, no treatment may be necessary. If the patient is young with significant growth remaining, both length discrepancy and angular deformity may occur. Management may require a combination of modalities.

1. Shoe-lift. This is applicable when a lower extremity bar is central and causes no angular deformity and the leg-length discrepancy is expected to be minor at maturity (2.5 cm or less). This is the only effective nonoperative treatment and was used extensively for centuries before the advent of modern surgery.
2. Arrest of the remaining growth of the injured physis. This should be considered in an older child with a beginning or progressive angular deformity when limb-length inequality will be minor (lower extremity) or of relatively little functional consequence (upper extremity).
3. Arrest of the remaining growth of the injured physis and the physis of the adjacent bone (forearm and lower leg).
4. Arrest of the remaining growth of the injured physis, the physis of the adjacent bone if one is present, and the corresponding physis or physes of the contralateral bone or bones.
5. Open or closed wedge osteotomy to correct angular deformity without operative arrest of the remaining normal physis ([336,337,346](#)). In a young patient, the untreated bone bar would reproduce angular deformity after osteotomy. Osteotomy can be repeated several times before attainment of full growth if this method is used alone. Some final relative shortening of the involved bone should be expected.
6. Lengthening of the involved bone.
7. Shortening of the contralateral or companion (ulna or fibula) bone.
8. Excision of the physeal bar and insertion of an interposition material (see later).
9. Fracture of the bone bar by physeal distraction using an external distractor, with or without excising the bar ([316,317,318,325,330,334](#)). This has been done successfully on small bars in optimal locations. The procedure can be combined with correction of both angulation and length discrepancy, but it should be done only in older children nearing maturity because the procedure is likely to result in complete closure of the physis ([312,321](#)).
10. Transplantation of an epiphysis and physis from another bone to fill the defect following resection of a bar. Many experiments using various strategies have been performed, with varying success ([313,314,315,319,320,322,323,326,327,328,329,335,338,339,340,341,342,343,344](#) and [345](#)). In humans, in addition to problems of vascularity, growth, and tissue rejection, a major obstacle is the availability of a suitable, dispensable donor physis. With ongoing advances in microvascular techniques and tissue rejection problems, this method might be considered in the future, but is now not clinically applicable.
11. Combinations of the above-mentioned procedures. It is unusual for a patient to be treated optimally with only one of the above-mentioned modalities. Even when bar excision allows several inches of longitudinal growth, some other modality, such as a shoe-lift, closure of the contralateral physis, or osteotomy to correct angular deformity, is often beneficial.

## Functional Considerations

Leg-length discrepancy of 2.5 cm or less usually causes little, if any, functional impairment or low back pain and can be left untreated, or a shoe-lift can be used on the short side. Leg-length discrepancy anticipated to be 2.5 to 5 cm at maturity can be managed by arrest of growth of the contralateral bone if the child has sufficient growth remaining to correct the discrepancy. Bone shortening on the contralateral side may be considered if all physes are closed or the child is nearing maturity. Bone shortening should be considered only for the femur, because shortening of the tibia is usually accompanied by uncorrectable muscle weakness, especially of foot dorsiflexion. Lengthening of the femur or tibia, or repeated lengthenings, may be considered for discrepancies of 4 to 5 cm or more. The patient's anticipated height at maturity is a factor in all of these instances.

Arm-length discrepancy results in functional impairment only when the discrepancy is extreme. Discrepancies of 10 cm or less are best left untreated. Surgical shortening of the contralateral upper extremity has never been reported and, to the author's knowledge, has no application. Lengthening of the humerus ([142](#)) and forearm has been reported but carries potential morbidity and should be done only by surgeons with experience in the procedure.

All of the above-mentioned treatment options have been used in the management of physeal bars and should always be considered. However, excision of the bar, when successful, may negate the need for the other modalities and their potential morbidity. If excision is unsuccessful, the other options can still be used.

## Treatment by Bar Excision

### Experimental Studies

In animal studies, several investigators created a physeal bar, allowed the bar to develop, excised the bar and inserted an interposition material, and then sacrificed the animal to observe the result ([347,348,349,350,351,352,353,354,355](#) and [356](#)). Although the results have varied, there has been enough success to confirm that a bar can be successfully excised and growth reestablished. Interposition materials used include bone wax, fat, cartilage, silicone rubber, and polymethylmethacrylate. When no interposition material was inserted, a bone bar promptly reformed ([269,356](#)). Because of variables in the experiments, it is difficult to determine superiority of one interposition material over another.

Because cartilage is the damaged tissue, cartilage would seem to be the ideal interposition material. Possible sources of cartilage are another physis, an apophysis such as the iliac crest, and a laboratory-procured chondrocyte allograft transplant ([347,352,353](#) and [354](#)). There are technical difficulties procuring and inserting another physis. Apophyseal cartilage may not have the same growth potential as epiphyseal cartilage. Chondrocyte allograft transplants require initial cartilage procurement, followed by laboratory time for the cartilage matrix to develop. Immune response problems might occur if the material is transferred from one human to another. Nevertheless, as cartilage is the damaged tissue, cartilage would be the ideal interposition material. It is hoped that more investigations will solve this

problem in the near future.

A load-sharing interposition material, such as polymethylmethacrylate, may be superior in resection of large bars in weight-bearing areas ( [351](#)).

A study in rabbits ([356](#)) suggested that re-formation of a bar after excision can be inhibited by the use of oral indomethacin without the use of an interposition material. Indomethacin produces a nonspecific inhibition of osteoblastic activity that is triggered by fracture or postoperative inflammation. No clinical trial using oral indomethacin in conjunction with bar excision in humans has been reported. Whether indomethacin can be given in sufficient doses in humans to prevent the bone bar from re-forming without inhibiting normal bone growth remains to be seen.

#### Clinical Experience ([357,358,359,360,361,362,363,364,365,366,367,368,369,370,371,372,373,374,375,376,377,378, 379,380,381,382,383,384 and 385](#))

The first bone bar in a human was reported in 1967 by Langenskiöld ([374](#)), who described a 15-year-old boy with genu recurvatum secondary to a bone bar in the anterior proximal tibia; the etiology was unknown. The bone bar was excised, and the space was filled with autogenous fat. During the 1.5-year follow-up, the angle of genu recurvatum improved by 10 degrees, but there was no documentation of longitudinal growth.

Longitudinal growth was first documented in the distal tibia of a 5-year-old boy in 1968 ( [381](#) ). During a 10-year follow-up, the involved tibia grew 16.7 cm. Sheet Silastic and Gelfoam were used as interposition materials.

#### Interposition Materials

Fat ([361,368,369,371,372,373,374,384,385](#)), bone wax and fat ([212,252](#)), Silastic ([349,350,360,365,384](#)), methylmethacrylate ([375,376](#)), and Cranioplast ([331,332,333,367,377,378,379,380 and 381](#)) are the most popular interposition materials, but too few patients have been followed to maturity to determine superiority of one interposition material over the others.

Fat has the distinct advantage of being autogenous. Langenskiöld used buttock fat because of its more firm and globular consistency. Fat has the disadvantage of a lack of hemostasis in the resected cavity. When the tourniquet is released, fat tends to float out of the cavity. Closing periosteum over the cavity to contain the fat predisposes the patient to new bone formation peripherally. This is undesirable because it tends to tether growth again. The operative defect weakens the structure of the bone, and a cast is used to protect a weight-bearing bone from fracture ( [385](#) ). When fat grafts work well, the intraoperative cavity enlarges as the bone (and fat graft) grow ([368,369](#)). Postoperative fracture has been reported ( [368](#) ).

Silastic ([1,349,350,360,365,384](#)) has many properties similar to Cranioplast but has not been used since its withdrawal from commercial markets by Dow Corning in 1987.

### AUTHOR'S PREFERRED TECHNIQUE OF TREATMENT

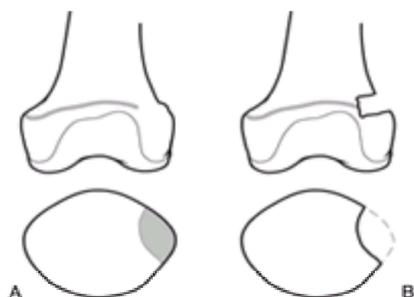
The objectives of surgical excision of a bone bar are to remove the bar completely and to preserve as much of the normal remaining physis as possible. This requires knowledge of surgical anatomy ([358](#)) and careful preoperative evaluation and planning. The excision may be difficult if the bar is irregular or the epiphyseal/metaphyseal complex is deformed ([Fig. 5-34](#)).

### THE AUTHOR'S CLASSIFICATION

In 1984, I grouped physeal bars into one of three types based on location and contour: (a) peripheral, (b) elongated, and (c) central ( [333](#) ). Subsequently, Bright ([1](#)) and Ogden ([3](#)) classified these as types I, II, and III, although they used the terms linear or longitudinal instead of elongated.

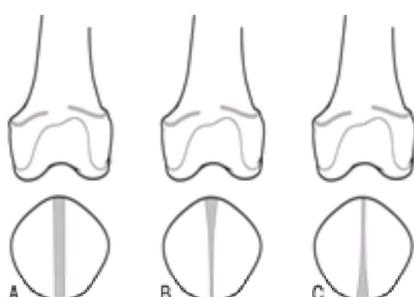
#### Preferred Surgical Approaches

I approach peripherally located bars directly from the periphery ( [Fig. 5-37](#) ). I am careful to excise the periosteum overlying the bar to prevent subsequent bar re-formation. Under direct vision, the bar can then be removed until normal physis is visible on all sides of the cavity ( [Fig. 5-37B](#) ). I find that optical loupes are helpful and ordinary surgical light is satisfactory. The bone bar is then removed initially using an osteotome, curet, and rongeur. The final exposure of the normal physis is done more precisely with a motorized bur. This allows excellent visualization of the physis and removal of as little metaphyseal bone as necessary, and facilitates contouring of the cavity in the physis. The heat generated by the bur has no apparent deleterious effect on the viability of the remaining physis.



**FIGURE 5-37.** Peripheral bar shown in AP view (above), transverse section through physis (below). **A:** With map of bar composed from MRI. **B:** Bar exposed by direct approach. (Redrawn from Peterson HA. Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258; with permission.)

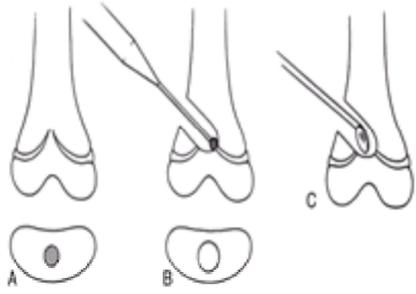
An elongated, linear, or longitudinal, bar extends completely across the physis and is common after Salter–Harris IV fracture ( [Fig. 5-38A, Fig. 5-38B, Fig. 5-38C](#) ). I first carefully evaluate these bars with MRI maps to determine the surgical approach and to ensure complete removal of the bar. Headlamp light to illuminate the depth of the excision is beneficial and is preferable to optic loupes.



**FIGURE 5-38.** Elongated bar extending from anterior to posterior surfaces. Although these three bars have the same appearance as the anteroposterior view (above), they have different contours and areas on transverse orientations (below). To achieve complete bar removal with retention of as much normal physis as possible, the

bar in **B** would be optimally approached posteriorly, and that in panel **C** anteriorly. (Redrawn from Peterson HA. Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258; with permission.)

Centrally located bars have normal physis peripherally and an intact perichondrial ring of Ranvier ( [Fig. 5-39A](#)). I almost always approach these bars through the metaphysis by removing a window of cortical bone and cancellous metaphyseal bone to expose the bar from inside out ( [Fig. 5-40](#); [Fig. 5-39B](#)). This preserves the perichondrial ring of Ranvier. Again, headlamp light here is extremely helpful. After removal of the entire bar, I am careful to inspect normal physis circumferentially in the cavity. I have found that using a small (5 mm in diameter) dental mirror ( [Fig. 5-39C](#)) makes this easier.



**FIGURE 5-39.** Central bar as seen anteroposteriorly (above) and transversely (below). **A:** Bar in center with growth peripherally results in cupping or tenting of the physis. **B:** Excision of central bar through window in metaphysis. **C:** Visualization of the entire physis using a dental mirror. (From Peterson HA. Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258; with permission.)

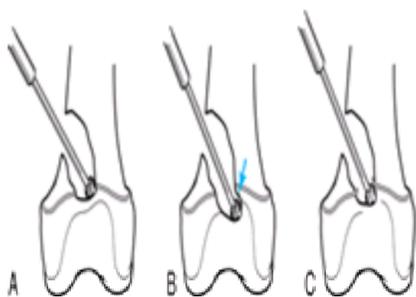


**FIGURE 5-40.** AP radiograph of distal femur of an 11+0-year-old boy taken at time of bar excision to document desired depth of bur, removal of bar, and presence of remaining normal physis. Previous radiographs of this patient are shown in [Figure 5-36](#).

Other authors have reported modifications of this basic technique. Some use an operating microscope to aid visualization of the bar ( [212,252,370,371,373](#)). Ultraviolet visualization of tetracycline-labeled bone in rabbits has been reported to allow complete excision of experimentally created physal arrests while minimizing excision of normal physis ( [382](#)). I have not found these modifications to be necessary and have no personal experience with them.

### The Final Cavity

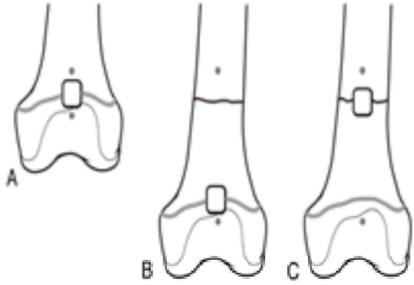
I always try to make the sides of the cavity flat and smooth ( [Fig. 5-41A](#)). Enlarging the cavity in the epiphysis may encourage the interposition material to remain across the physis, but I have not found this feature to enhance results ( [Fig. 5-41B](#)). Care must be taken not to weaken the epiphysis, predisposing it to fracture. I do not undermine the metaphyseal and epiphyseal bone away from the physis, as advocated by Ogden ( [Fig. 5-41C](#)) (3). I am concerned that the protruding physis would be deprived of its blood supply and more of the physis would have been removed than necessary.



**FIGURE 5-41.** Contour of cavity. **A:** As normal physis is exposed, a smooth surface helps to identify and visualize the physis. **B:** Bone of the epiphysis may be undermined in an attempt to allow the plug to stay with the epiphysis as the bone grows in length. If this is done, a small rim of epiphyseal bone ( *arrow*) should be preserved to maintain viability of the physis. **C:** Undermining bone away from the physis may improve visualization of the physis but will deprive it of its blood supply. (Redrawn from Peterson HA. Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258; with permission.)

### Metal Markers

I place metal markers in the metaphysis and the epiphysis to allow accurate radiographic measurement of subsequent growth ( [Fig. 5-42](#)). These metal markers in the metaphysis and the epiphysis also help differentiate overgrowth of the physis at the other end of the bone, which may falsely enhance the result. I try to place these markers carefully in cancellous bone, not in contact with the cavity, because they might become attached to the interposition material, or if fat is used, they might migrate into the cavity. I am also sure to place them in the same longitudinal plane proximally and distally to the defect. A position in the center of the bone is preferable because eccentric markers may become extraosseous due to growth and metaphyseal remodeling. Any metal marker will do; half of a vascular clip, stainless steel, or silver was commonly used in early cases. Transversely oriented Kirschner wires (K-wires) parallel with each other and with the physis, one in the metaphysis and one in the epiphysis, allow accurate assessment of angular growth ( [Fig. 5-32](#)). Titanium Kirschner wires avoid artifact on subsequent MRI evaluation. At present, 10-mm lengths of Titanium 0.062 K-wires are used.



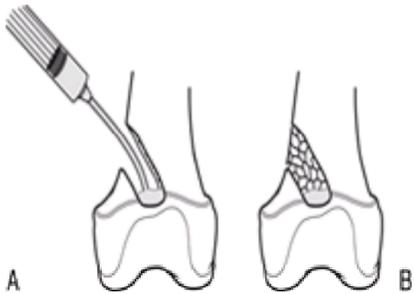
**FIGURE 5-42.** Metal markers. **A:** Metal markers are placed in cancellous bone of the epiphysis and metaphysis, away from the interposition plug, longitudinally oriented to each other and as close to the center of the bone as feasible. **B:** A plug that stayed with the epiphysis as the physis grew away from the proximal marker and the growth arrest line. **C:** A plug that stayed with the metaphysis as the physis grew. The plug has not migrated. (Redrawn from Peterson HA. Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258; with permission.)

### Preferred Interposition Material

Next, I place the interposition material into the cavity. The object is to fill the cavity to prevent blood from occupying the cavity, organizing, and re-forming a bone bar.

I prefer Cranioplast as an interposition material because of its several advantages. It is easily available and inexpensive, there is no Food and Drug Administration (FDA) control (as for Silastic), and no second incision is needed (as for fat). It is light, easy to handle and mold, thermally nonconductive, and radiolucent. Both the liquid (monomer) and the powder (polymer) are sterile as packaged and can be mixed in the operating room. It is unnecessary to take cultures. It provides hemostasis because it occupies the entire desired portion of the cavity; it is strong, so that no postoperative immobilization is necessary; and there are no apparent side effects. I find it especially useful to support the epiphysis after excavation of a large bar ( [351,385](#)).

In a cavity that is gravity dependent, I pour the Cranioplast in a liquid state. If the cavity is not in a dependent position, I place the Cranioplast in a syringe and push it into the defect through a short polyethylene tube ( [Fig. 5-43A](#)). Alternatively, I may allow the Cranioplast to set partially and then push it into the defect like putty. As little Cranioplast as possible should be allowed to remain in the metaphysis. After the Cranioplast has set, the rest of the metaphyseal cavity is filled with previously removed cancellous bone ( [Fig. 5-43B](#)).



**FIGURE 5-43.** Insertion of Cranioplast. **A:** Only enough Cranioplast is inserted, here shown by use of a syringe and catheter, to bridge all physal surfaces. **B:** The remainder of the defect is filled with bone chips harvested at the time of exposure. (Redrawn from Peterson HA. Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258; with permission.)

Misunderstandings concerning methylmethacrylate are related to terminology. Pure methylmethacrylate, or Cranioplast, was first produced in 1927. It has been used for over five decades by neurosurgeons to repair skull defects and has been found to be an inert and safe material. When used as an isolated substance, it has caused no rejection, infection, or neoplastic change ( [362](#)). Its thermogenic properties are minor, as evidenced by the neurosurgical practice of pouring it in a semiliquid form directly on dura and brain tissue before setting. It is also radiolucent.

When initial results from total hip arthroplasty revealed prosthesis loosening, a search for a bone cement was undertaken. Cranioplast was found to work well, but the radiolucent property made subsequent loosening difficult to detect. Barium was added to the methylmethacrylate. This achieved radiopacity, but it decreased the setting time and increased the exothermic property significantly. Thus, the material that is now generally referred to as methylmethacrylate is, in fact, Cranioplast with barium added. This type of methylmethacrylate is undesirable as an interposition material for bar excisions because of its radiopacity and possibly because of its exothermic property. The radiopacity obviates detection of recurrent bar formation.

### Osteotomy

Mild angular deformity secondary to peripheral bars may correct spontaneously with growth after excision of the bar. Angular deformities of more than 20 degrees will probably not correct spontaneously and usually require osteotomy ( [212,336,337](#)). This can be done at the same time as bar excision, or later.

**Postoperative Care.** Postoperatively, if Cranioplast is inserted and no osteotomy is performed, no cast or other immobilization is necessary. Joint motion and weight-bearing are encouraged on the day of operation, or as soon as operative discomfort subsides.

## COMPLICATIONS

### Bar Re-formation

The major complication of bar excision is bar re-formation. Although this may occur after excision of a bar of any size, in any location, and at any site, it is more likely to occur with large bars (those that occupy 50% or more of the entire physis). A recurrent bar may form at any time, early or late, after initial excision. If it occurs soon after excision and significant growth remains, the recurrent bar can be reexcised with some hope of success ( [Fig. 5-33F, Fig. 5-33G, Fig. 5-33H](#)). More commonly, when bar formation occurs, it occurs near the completion of growth, which underscores the need for careful follow-up (see the next [section](#)). Bar re-formation signals a less-than-desirable outcome but does not preclude application of all other types of bar management (see earlier).

### Unhealthy Physis

In addition to the size of the bar and the adequacy of bar removal, other factors that may be associated with bar re-formation are the patient's health and the rate of growth of the physis at the time of excision. A protocol for using growth hormone after bar excision to enhance the short-term growth activity was used in one patient with equivocal results.

### Technical Errors

Other complications of bar excision are primarily technical. Fracture of the medial portion of the distal tibial epiphysis has been noted after too-generous removal of epiphyseal bone of a medial distal tibial bar. A referral case, in which a large bar had been completely replaced with bone wax, resulted in recurrent transcutaneous extrusion of small bits of wax until the wax was entirely surgically removed.

### Postoperative Infection

Postoperative infection can occur, as in any orthopaedic surgery. However, in the author's experience, it has occurred only in three patients in whom the original cause of the bar was infection. This infection is chronic and requires debridement and antibiotics like other bone infections. Once the infection is cleared, bar management proceeds using any of the previously discussed methods.

### Follow-Up

**Reexcision May Be Necessary.** Follow-up until maturity is essential. Reestablished physal growth may cease at any time. Recurrent bar formation has been successfully treated by reexcising the bar (Fig. 5-33) (331,364,367). If a bar re-forms near maturity or if the entire physis ceases growing on the injured side earlier than its contralateral counterpart (a fairly common finding), physal arrest on the contralateral side may be considered.

### Scanograms

Scanograms (305,306,308) are the most precise way to measure the increasing distance between the two metal markers (Fig. 5-33A, Fig. 5-33D, Fig. 5-33H). As the child grows, length and circumference of the extremity increase, allowing magnification of the distance between the markers on regular roentgenograms, teleroentgenograms, and orthoroentgenograms. This magnification falsely enhances the result. Scanograms have no magnification. They can be measured and remeasured directly on the film by tape measure. They also show any deformity or other abnormality of the entire bone.

It is suspected that the physis on the opposite end of an injured or operated bone sometimes overgrows to compensate for any damage at the other end (364). Although the amount of growth of bone attributable to each physis has been fairly well established (for example, distal femur, 70%; proximal tibia, 60%; distal tibia, 40%), the only way to determine precisely the amount and percentage of growth contributed by each physis of a bone after bar excision, compared with its contralateral member, is to place a single metal marker in the diaphysis of the normal contralateral bone. This has been found valuable in determining comparative growth, which aids in finding bar recurrence (Fig. 6-33D and Fig. 5-33H).

## RESULTS

Assessing results is difficult because so many factors are involved. When the procedure works well, it is most gratifying and may be the only procedure needed. This renewed growth may diminish the angular deformity and the rate of progression of limb-length inequality; occasionally, there may even be reduction of the length inequality (e.g., the treated limb grows faster than the normal limb; Fig. 5-33A and Fig. 5-33H).

Only patients followed to maturity should be included in any reported series. Some operated physes, although growing well after the procedure, close earlier than their contralateral physes. Thus, in some patients, surgical arrest of the contralateral physis is performed toward the end of growth to negate additional discrepancy. This approach favorably influences the result expressed as a percentage.

### Fifty Percent Rule

Excision of bars constituting 50% or more of the entire physis usually fail to restore satisfactory longitudinal growth. Bars more than 50% of the physis may be excised in very young children because the alternatives are undesirable, because the procedure occasionally works, and because if the procedure is unsuccessful, all other methods of management can still be used.

### The Mayo Clinic Experience

From 1968 through 1996, 178 patients were treated by bar excision at the Mayo Clinic (Table 5-3). Bar formation was at the knee (distal femur and proximal tibia) in 50% of the cases, whereas these sites account for only 2.2% of all physal injuries (Table 5-2). This disparity is explained by the anatomy of these physes. Their undulations and irregularities in multiple planes insulate them from injury, but when injury occurs, the same undulations predispose the patient to damage of the growth layer of the physis. All operations were performed by four staff pediatric orthopaedists, with little variation in technique. Cranioplast was used for the interposition material in 153 patients, fat in 23, methyl methacrylate in 1, and sheet Silastic and Gelfoam in 1 (381).

In 98 patients followed to maturity, the average growth of the operated side was 84% of that of the unoperated side. The average was 78% for the distal femur, 88% for the proximal tibia, and 93% for the distal tibia (Table 5-4).

	Growth <sup>1</sup> No.	%	Average Age (Yr)	Time Interval (Yr)	Bar Area >45%	Bar Area <45%	Control Bars
Distal femur	43	78	10.5	3	11 (26)	32 (74)	10 (23)
Prox tibia	18	88	11.6	2.1	2 (11)	16 (89)	6 (33)
Dist tibia	37	93	11.1	1.9	3 (8)	34 (92)	5 (14)

<sup>1</sup>Growth = % of growth of operated vs. contralateral non-operated physis

<sup>2</sup>Time age = interval between injury and bar excision

<sup>3</sup>Bar area = area of bar expressed as a percentage of entire area of physis. Number is number of cases; () = percentage of cases

TABLE 5-4. RESULTS OF 98 MATURE CASES

Thirteen of these 98 patients (13%) had no accompanying surgery. Adjunctive surgery in the remaining 85 patients (87%) was usually performed for length discrepancy and angular deformity existing before the bar excision, which did not correct even with a successful bar excision. Resumption of normal growth that stops progression of angular deformity and length discrepancy is a successful outcome of bar excision. Improvement of angular deformity and length discrepancy (the operated side grows more than the normal side) sometimes occurs, but parents should not be led to believe that it will. There were 37 osteotomies to correct angular deformity, performed either concomitantly with the bar excision or later. Forty patients had epiphysiodeses of the contralateral bone or of the adjacent bone in the case of the lower leg. Thirteen patients had lengthenings of the involved bone. Eighteen recurrent bars were reexcised.

Of the 43 distal femoral lesions, 30 were in boys and 13 in girls. The interval between injury and bar excision was 2.8 years. The average age at time of bar excision was 10.5 years. The area of the bar was more than 45% in 11, less than 30% in 12, and less than 30% in 20. The site of the lesion was medial (13), lateral, central (10), posterior (9), and anterior (6) (some lesions occupied more than one locus). The operated side grew 78% as much as the normal side. Additional procedures included epiphysiodesis (23), osteotomy (11), and lengthening (9). There were nine recurrent bars and two infections (the cause of the bar in these two was osteomyelitis).

Of the 18 proximal tibial lesions, nine were found in boys and nine were found in girls. The interval between injury and surgery was 2.1 years. The average age at time of surgery was 11.7 years. The area of the bar was more than 45% in two patients, more than 30% in one patient, and less than 30% in 15 patients. The site of arrest was central (6), lateral (5), medial (4), anterior (4), and posterior (1) (some lesions occupied more than one locus). The average growth of the operated side was 88%

of the uninjured side. Additional procedures include osteotomy, epiphysiodesis (9), and lengthening (3). There were two recurrences and one infection (the cause of the bar in this case was osteomyelitis).

Of the 37 distal tibial lesions, 26 were in boys and 11 were in girls. The interval between injury and surgery was 1.9 years. The average age at time of surgery was 11.1 years. The area of the bar was more than 45% in three patients, more than 30% in four patients, and less than 30% in 30 patients. The site of the bar was medial (14), anterior (10), central (5), posterior (5), and lateral (3). The average growth was 93% of that of the contralateral nonoperated side. Additional surgery included osteotomy (15), physeal arrest (8), and lengthening (1). There were 7 recurrent bars, 2 fractures, and no infections.

From this data, it can be seen that the more distal the lesion, the better the result (Table 5-4). The overall result, expressed as a percentage of growth of the contralateral normal physis, improves as the site progresses distally and correlates directly with the percentage of lesions smaller than 45% of the physis. Factors favoring the distal tibia were the short time interval between injury and bar excision, the few larger lesions (8%), and the many smaller lesions (92%). Factors contributing to the less satisfactory results in the distal femur include the long time interval, the greater number of large lesions (26%), the fewer small lesions (74%).

There was no correlation of result with gender or location of the bar within the physis. However, several spectacular results were obtained in central bars, and the presence of an intact cartilage ring of Ranvier is believed to be beneficial.

### Interposition Material Removal

The criteria for the subsequent removal of interposition material or for bone grafting of defects left by fat have not been established. If the physis grows away from the interposition material, the material becomes located in the metaphysis or diaphysis. As the metaphysis remodels, the interposition material, although remaining the same size, occupies a greater proportion of the transverse plane of the shaft of the bone (compare Fig. 5-33C and Fig. 5-33E). This may predispose the bone to pathologic fracture and has done so in one patient after the Cranioplast was removed. In one patient, remodeling of the cortex allowed the Cranioplast to extrude and become extraosseous. Patients should be advised that the interposition material may have to be removed in the future. All Cranioplast plugs have been embedded firmly in the bone. There has been no loosening, such as occurs when methylmethacrylate is used with a prosthesis. In the few that have been removed, histologic evaluation revealed only a thin surrounding layer of fibrous tissue and no untoward reaction. The material is not easily removed, and usually a thin area of surrounding bone must be excised. A motorized bur or an osteotome has been used to remove Cranioplast plugs. The author's practice is to not remove the Cranioplast.

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## CHAPTER REFERENCES

### Physeal Fractures

#### Introduction

1. Bright RW. Physeal injuries. In Rockwood CA, Wilkens KE, King RE, eds. *Fractures in children*. Philadelphia: JB Lippincott, 1991;87-170.
2. Canale ST. Physeal injuries. In Green NE, Swionkowski MF, eds. *Skeletal trauma in children*. Philadelphia: WB Saunders, 1994;15-55.
3. Ogden JA. Injury to the growth mechanisms. In *Skeletal injury in the child*, 3rd ed. New York: Springer-Verlag, 2000;147-242.
4. Tachdjian MO. Fractures and dislocations. In: *Pediatric Orthopaedics*, 2nd ed. Philadelphia: WB Saunders, 1990;3013-3373.

#### Historical Review

5. Foucher JT. De la divulsion des epiphyses. Cong of Med de France 1963 (Reprinted in English in *Clin Orthop* 1984;188:3-9.);1:63-72.
6. Poland J. *Traumatic separation of the epiphyses*. London: Smith, Elder & Company, 1898. (Portions reprinted in *Clin Orthop* 1965);:7-18.

#### Classification

7. Aitken AP. The end result of the fractured distal tibial epiphysis. *J Bone Joint Surg* 1936;18:685-691.
8. Aitken AP. Fractures of the epiphysis. *Clin Orthop* 1965;41:19-23.
9. Aitken AP, Ingersoll RE. Fractures of the proximal tibial epiphyseal cartilage. *J Bone Joint Surg Am* 1956;38:787-796.
10. Aitken AP, McGill HK. Fractures involving the distal femoral epiphyseal cartilage. *J Bone Joint Surg Am* 1952;34:96-108.
11. Cass JR, Peterson HA. Salter and Harris Type IV injuries of the distal tibial epiphyseal growth plate with emphasis on those involving the medial malleolus. *J Bone Joint Surg Am* 1983;65:1059-1070.
12. Chadwick CJ, Bentley G. The classification and prognosis of epiphyseal injuries. *Injury* 1987;18:157.
13. Chadwick CJ, Bentley G. Chadwick and Bentley classification of distal tibial growth plate injuries. In: Uthoff NK, Wiley JJ, eds. *Behavior of the growth plate*. New York: Raven Press, 1988;105-110.
14. Dias LS, Tachdjian MO. Physeal injuries of the ankle in children: classification. *Clin Orthop* 1978;136:230-233.
15. Johnson EW, Fahl JC. Fractures of the distal epiphysis of the tibia and fibula in children. *Am J Surg* 1957;93:778-781.
16. Johnston RM, Jones WW. Fractures through human growth plates. *Orthop Trans* 1980;4:295.
17. Kling TF. Management of physeal injuries. In: Chapman MW, ed. *Operative orthopaedics*, 2nd ed. Philadelphia: JB Lippincott, 1993;3035-3049.
18. Letts RM. Compression injuries of the growth plate. In: Uthoff HK, Wiley JJ, eds. *Behavior of the growth plate*. New York: Raven Press, 1988;111-118.
19. Ogden JA. Injury to the growth mechanism of the immature skeleton. *Skeletal Radio*. 1981;6:237-253.
20. Ogden JA. Skeletal growth mechanism injury patterns. *J Pediatr Orthop* 1982;2:371-377.
21. Ogden JA. Skeletal growth mechanism injury patterns. In: Uthoff HK, Wiley JJ, eds. *Behavior of the growth plate*. New York: Raven Press, 1988;85-96.
22. Peterson HA. Distal tibial triplane fractures. In: Heckman JD, ed. *Perspectives in orthopaedic surgery*. St. Louis: Quality Medical Publishers, 1991;21-34.
23. Peterson HA. Physeal fractures: part 2, Two previously unclassified types. *J Pediatr Orthop* 1994;14:431-438.
24. Peterson HA. Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439-448.
25. Peterson HA. Classification of physeal fractures. In: dePablos J, ed. *Surgery of the growth plate*. Madrid: Ediciones Ergon, S.A., 1998;181-195.
26. Rang M. *The growth plate and its disorders*. Baltimore: Williams & Wilkins, 1969.
27. Rang M. Injuries of the epiphyses, the growth plate, and the periochondral ring. In: *Children's fractures*, 2nd ed. Philadelphia: JB Lippincott; 1983;10-25.
28. Salter RB, Harris WR. Injuries involving the epiphyseal plate. *J Bone Joint Surg Am* 1963;45:587-622.
29. Salter RB. Salter-Harris classification of epiphyseal injuries. In: Uthoff NK, Wiley JJ, eds. *Behavior of the growth plate*. New York: Raven Press, 1988;97-103.
30. Shapiro F. Epiphyseal growth plate fracture-separation. A pathophysiologic approach. *Orthopaedics* 1982;5:720-736.
31. Smith DG, Geist RW, Couperman DR. Microscopic examination of a naturally occurring epiphyseal growth plate fracture. *J Pediatr Orthop* 1985;8:306-308.
32. Weber BG, Brunner CH, Freuler F. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980.

#### Epidemiology

33. Fischer MD, McElfresh EC. Physeal and epiphyseal injuries of the hand: Patterns of injury and results of treatment. *Hand Clin*. 1994;10:287-301.
34. Landin LA. Fracture patterns in children. *Acta Orthop Scand* 1983;54(Suppl 1):1-109.
35. Mann DC, Rajmaira S. Distribution of physeal and nonphyseal fractures in 2650 long-bone fractures in children ages 0 to 16 years. *J Pediatr Orthop* 1990;10:713-716.
36. Mizuta T, Benson WM, Foster BK, et al. Statistical analysis of the incidence of physeal injuries. *J Pediatr Orthop* 1987;7:518-523.
37. Oh WH, Craig C, Banks HH. Epiphyseal injuries. *Pediatr Clin North Am* 1974;21:47-52.
38. Peterson HA, Madhok R, Benson JT, et al. Physeal fractures: part I, epidemiology in Olmsted County, Minnesota, 1979-1988. *J Pediatr Orthop* 1994;41:423-430.
39. Worlock P, Stower M. Fracture patterns in Nottingham children. *J Pediatr Orthop* 1986;6:656-660.

## Evaluation

40. Arkbania BA, Silberstein MJ, Rende RJ, et al. Arthrography in the diagnosis of fractures of the distal end of the humerus in infants. *J Bone Joint Surg Am* 1986;68A:599–602.
41. Barrett WP, Almquist EA, Staheli LT. Fracture separation of the distal humeral physis in the newborn. *J Pediatr Orthop* 1984;4:617–619.
42. Broker FH, Burbach T. Ultrasonic diagnosis of separation of the proximal humeral epiphysis in the newborn. *J Bone Joint Surg Am* 1990;72:187–191.
43. Brown J, Eustace S. Neonatal transphyseal supracondylar fracture detected by ultrasound. *Pediatr Emerg Care* 1997;13:410–412.
44. Carey J, Spence L, Blickman H, Eustace S. MRI of pediatric growth plate injury: correlation with plain film radiographs and clinical outcome. *Skeletal Radio*. 1998;27:250–255.
45. Dias JJ, Lamont AC, Jones JM. Ultrasonic diagnosis of neonatal separation of the distal humeral epiphysis. *J Bone Joint Surg Br* 1988;70:825–828.
46. Diaz MJ, Hedlund GL. Sonographic diagnosis of traumatic separation of the proximal femoral epiphysis in the neonate. *Pediatr Radio*. 1991;21:238–240.
47. Etchebehere EC, Etchebehere M, Gamba R, et al. Orthopedic pathology of the lower extremities: scintigraphic evaluation in the thigh, knee, and leg. *Semin Nucl Med* 1998;28:41–61.
48. Eustace S. MR Imaging of acute orthopedic trauma to the extremities [Review]. *Radiol Clin North Am* 1997;35:615–629.
49. Hansen PE, Barnes DA, Tullos HS. Arthrographic diagnosis of an injury pattern in the distal humerus of an infant. *J Pediatr Orthop* 1982;2:569–572.
50. Harris HA. Lines of arrested growth in the long bones in childhood: the correlation of histological and radiographic appearance in clinical and experimental conditions. *Br J Radiol* 1931;4:561–588.
51. Heim M, Blankstein A, Israeli A, Horoszowski H. Which x-ray views are required in juvenile ankle trauma? *Arch Orthop Trauma Surg* 1990;109:175–176.
52. Howard CB, Shinwell E, Nyska M, Meller I. Ultrasound diagnosis of neonatal fracture separation of the upper humeral epiphysis. *J Bone Joint Surg Br* 1992;74:471–472.
53. Jaramillo D, Shapiro F. Musculoskeletal trauma in children. *Magn Reson Imaging Clin North Am* 1998;6:521–536.
54. Jaramillo D, Hoffer FA. Cartilaginous epiphysis and growth plate: Normal and abnormal and MR imaging findings. *Am J Radiol* 1992;158:1105–1110.
55. Jaramillo D, Hoffer FA, Shapiro F, Rand F. MR imaging of fractures of the growth plate. *Am J Radiol* 1990;155:1261–1265.
56. Marzo JM, d'Amato C, Strong M, Gillespie R. Usefulness and accuracy of arthrography in the management of lateral humeral condyle fractures in children. *J Pediatr Orthop* 1990;10:317–321.
57. Nimkin K, Kleinman PK, Teeger S, Spevak MR. Distal humeral physeal injuries in child abuse: MR imaging and ultrasonography findings. *Pediatr Radio*. 1995;25:562–565.
58. Oestreich AE, Ahmad BS. The periphysis and its effect on the metaphysis: I. Definition and normal radiographic pattern. *Skeletal Radio*. 1992;21:283–286.
59. Ohashi K, Brandser EA, el-Khoury GY. Role of MR imaging in acute injuries of the appendicular skeleton. *Radiol Clin North Am* 1997;35:591–613.
60. Petit P, Panuel M, Faure F, et al. Acute fracture of the distal tibial physis: role of gradient-echo MR imaging versus plain film examination. *Am J Roentgenol* 1996;166:1203–1206.
61. Poznanski AK. Annual oration: diagnostic clues in the growing ends of long bones. *J Can Assoc Radiol* 1978;29:7–21.
62. Rogers LF. The radiology of epiphyseal injuries. *Radiology* 1970;96:289–299.
63. Rogers LF. Radiology of skeletal trauma. In: Rogers LF. *Special Considerations in Children*. New York: Churchill-Livingstone; 1992;109–148.
64. Rogers LF, Poznanski AK. Imaging of epiphyseal injuries. *Radiology* 1994;191:297–308.
65. Smith BG, Rand F, Jaramillo D, Shapiro F. Early MR imaging of lower extremity physeal fracture-separations: a preliminary report. *J Pediatr Orthop* 1994;14:526–533.
66. So YC, Fang D, Leong JC, Bong SC. Varus deformity following lateral humeral condylar fractures in children. *J Pediatr Orthop* 1985;5:569–572.
67. Stuart J, Boyd R, Wilson-Derbyshire B, Phillips B. Magnetic resonance assessment of inversion ankle injuries in children. *Injury* 1998;29:29–30.
68. Wegener WA, Heyman S. Growth plate fracture. An acute fracture of the proximal tibia with a photopenic defect on bone scintigraphy. *Clin Nucl Med* 1990;15:447.
69. Werenskiold B. A contribution to the roentgen diagnosis of epiphyseal separations. *Acta Radiol* 1927;8:419–426.
70. White PG, Mah JY, Friedman L. Magnetic resonance imaging in acute physeal injuries. *Skeletal Radio*. 1994;23:627–631.
71. Yates C, Sullivan JA. Arthrographic diagnosis of elbow injuries in children. *J Pediatr Orthop* 1987;7:54–60.

## Treatment

72. Böstman O, Mäkelä EA, Törmälä P, Rokkanen P. Transphyseal fracture fixation using biodegradable pins. *J Bone Joint Surg Br* 1989;71:706–707.
73. Hope PG, Williamson DM, Coates CJ, Cole WG. Biodegradable pin fixation of elbow fractures in children. A randomized trial. *J Bone Joint Surg Br* 1991;73:965–968.
74. Mäkelä EA, Vainionpää S, Vihtonen K, et al. The effect of a penetrating biodegradable implant on the epiphyseal plate: an experimental study on growing rabbits with special regard to polyglactin 910. *J Pediatr Orthop* 1987;7:415–420.
75. Mäkelä EA, Vainionpää S, Vihtonen K, et al. The effect of a penetrating biodegradable implant on the growth plate: An experimental study on growing rabbits with special reference to polydioxanone. *Clin Orthop* 1989;241:300–308.
76. Mäkelä EA, Böstman O, Kekomäki M, et al. Biodegradable fixation of distal humeral physeal fractures. *Clin Orthop* 1992;283:237–243.
77. Mintzer CM, Waters PM, Brown DJ, Kasser JR. Percutaneous pinning in the treatment of displaced lateral condyle fractures. *J Pediatr Orthop* 1994;14:462–465.
78. Partio EK, Ruompo P, Hirvensalo E, et al. Totally absorbable fixation in the treatment of fractures of the distal femoral epiphyses. A prospective clinical study. *Arch Orthop Trauma Surg* 1997;116:213–216.
79. Svensson P, Janarv P, Hirsch G. Internal fixation with biodegradable rods in pediatric fractures: one-year follow-up of 50 patients. *J Pediatr Orthop* 1994;14:220–224.

## Prognosis

80. Bright RW, Richmond V, Burstein AH, Elmore SM. Epiphyseal-plate cartilage. A biomechanical and histological analysis of failure modes. *J Bone Joint Surg Am* 1974;56:688–703.
81. Dale GG, Harris WR. Prognosis of epiphyseal separations. An experimental study. *J Bone Joint Surg Br* 1958;40:116–122.
82. Lombardo SJ, Harvey JP. Fractures of the distal femoral epiphysis. Factors influencing prognosis: a review of 34 cases. *J Bone Joint Surg Am* 1977;59:742–751.

## Associated Injuries and Complications

83. Burkhart SS, Peterson HA. Fractures of the proximal tibial epiphysis. *J Bone Joint Surg Am* 1979;61A:996–1002.
84. Compere EL. Growth arrest in long bones as a result of fractures that include the epiphyses. *JAMA* 1953;105:2140.
85. Flynn JC. Nonunion of slightly displaced fractures of the lateral humeral condyle in children—an update. *J Pediatr Orthop* 1989;9:691–696.
86. Fontanetta P, MacKenzie DA, Rossman M. Missed, maluniting, and malunited fractures of the lateral humeral condyle in children. *J Trauma* 1978;18:329–335.
87. Goldberg BA, Mansfield DS, Davino NA. Nonunion of a distal femoral epiphyseal-separation. *Am J Orthop* 1996;25:773–777.
88. Hernandez J, Peterson HA. Fracture of the distal radial physis complicated by compartment syndrome and premature physeal closure. *J Pediatr Orthop* 1986;6:627–630.
89. Jeffrey CC. Nonunion of the epiphysis of the lateral condyle of the humerus. *J Bone Joint Surg Br* 1958;40B:396–405.
90. Martin RP, Parsons DL. Avascular necrosis of the proximal humeral epiphysis after physeal fracture. A case report. *J Bone Joint Surg Am* 1997;79:760–762.
91. Matthews LS. Acute volar compartment syndrome secondary to a distal radius fracture in an athlete: a case report. *Am J Sports Med* 1983;11:6–7.
92. Minami A, Sugawara M. Humeral trochlear hypoplasia secondary to epiphyseal injury as a cause of ulnar nerve palsy. *Clin Orthop* 1988;228:227–231.
93. Pape JM, Goulet JA, Hensinger RN. Compartment syndrome complicating tibial tubercle avulsion. *Clin Orthop* 1993;295:201–204.
94. Polakoff DR, Bucholz RW, Ogden JA. Tension band wiring of displaced tibial tuberosity fractures in adolescents. *Clin Orthop* 1986;209:161–165.
95. Robertson RC, Peterson HA. Traumatic dislocation of the hip in children: review of Mayo Clinic series. In: Harris WH, ed. *The hip*. St. Louis: CV Mosby, 1974;154–168.
96. Salter RB. Specific problems of epiphyseal plate injuries. In: Gossling HR, Pillsbury SL, eds. *Complications of fracture management*. Philadelphia: JB Lippincott, 1984;215–229.
97. Shelton WR, Canale ST. Fractures of the tibia through the proximal tibial epiphyseal cartilage. *J Bone Joint Surg Am* 1979;61:167–173.
98. Yang Z, Wang Y, Gilula LA, Yamaguchi K. Microcirculation of the distal humeral epiphyseal cartilage: implications for post-traumatic growth deformities. *J Hand Surg* 1998;23:165–172.

## OTHER PHYSEAL INJURIES

### Disuse

99. Beals RK. Premature closure of the physis following diaphyseal fractures. *J Pediatr Orthop* 1990;10:717–720.
100. Botting TD, Scrase WH. Premature epiphyseal fusion at the knee complicating prolonged immobilization for congenital dislocation of the hip. *J Bone Joint Surg Br* 1965;47:280–282.
101. Currarino G. Premature closure of epiphyses in the metatarsals and knees: a sequel of poliomyelitis. *Radiology* 1966;87:424–428.
102. Hresko T, Kasser JR. Physeal arrest about the knee associated with nonphyseal fractures in the lower extremity. *J Bone Joint Surg Am* 1989;71:698–703.
103. Hunter LY, Hensinger RN. Premature monomelic growth arrest following fracture of the femoral shaft: A case report. *J Bone Joint Surg Am* 1978;60:850–852.
104. Kestler OC. Unclassified premature cessation of epiphyseal growth about the knee joint. *J Bone Joint Surg* 1947;29:788–797.
105. Morton KS, Starr DE. Closure of the anterior portion of the upper tibial epiphysis as a complication of tibial shaft fractures. *J Bone Joint Surg Am* 1964;46:570–574.
106. Pappas AM, Anas P, Toczylowski HM. Asymmetrical arrest of the proximal tibial physis and genu recurvatum deformity. *J Bone Joint Surg Am* 1984;66:575–581.
107. Parke W, Colvin OS, Almond AH. Premature epiphyseal fusion at the knee in tuberculous disease of the hip. *J Bone Joint Surg Br* 1949;31:63–73.
108. Ratliff AH. The short leg in poliomyelitis. *J Bone Joint Surg Br* 1959;41:56–69.
109. Ratliff AH. Complications after fractures of the femoral neck in children and their treatment. *J Bone Joint Surg Br* 1970;52B:175.
110. Ross D. Disturbance of longitudinal growth associated with prolonged disability of the lower extremity. *J Bone Joint Surg Am* 1948;30:103–115.

111. Sissons HA. Osteoporosis and epiphyseal arrest in joint tuberculosis. An account of the histological changes in involved tissues. *J Bone Joint Surg Br* 1952;34:275–290.

### **Radiation**

112. Arguelles F, Gomar F, Garcia A, Esquerdo J. Irradiation lesions of the growth plate in rabbits. *J Bone Joint Surg Br* 1977;59:85–88.
113. Barnard HJ, Geyer RW. Effects of x-radiation on growing bones. *Radiology* 1962;78:207–214.
114. Baserga R, Lisco H, Carter DB. The delayed effects of external gamma irradiation on the bones of rats. *Am J Pathol* 1961;39:455–472.
115. Butler MS, Robertson WW, Rate W, et al. Skeletal sequelae of radiation therapy for malignant childhood tumors. *Clin Orthop* 1990;251:235–240.
116. DeSmet AA, Kuhns LR, Fayos JV, Hoyt JF. Effects of radiation therapy on growing long bones. *Am J Roentgenol* 1976;127:935–939.
117. Engel D. An experimental study on the action of radium on developing bones. *Br J Radiol* 1938;NS11:779–803.
118. Frantz CH. Extreme retardation of epiphyseal growth from roentgen irradiation. *Radiology* 1950;55:720–724.
119. Katz LD, Lewson JP. Radiation-induced growth abnormalities. *Skeletal Radio* 1990;19:50–53.
120. Katzman H, Waugh T, Berdon W. Skeletal changes following irradiation of childhood tumors. *J Bone Joint Surg Am* 1969;51:825–842.
121. Kember NF. Cell survival and radiation damage in growth cartilage. *Br J Radiol* 1967;40:496–505.
122. Langenskiöld A. Growth disturbance appearing 10 years after roentgen ray injury. *Acta Chir Scand* 1953;105:350.
123. Langenskiöld A, Edgren W. Imitation of chondrodysplasia by localized roentgen ray injury. An experimental study of bone growth. *Acta Chir Scand* 1950;99:353.
124. Neuhauser EB, Wittenborg MH, Berman CZ, Cohen J. Irradiation effects of roentgen therapy on the growing spine. *Radiology* 1952;59:637–650.
125. Peterson HA. Spinal deformity secondary to tumor, irradiation, and laminectomy. In: Bradford DS, Hensinger RM, eds. *The pediatric spine*. New York: Thieme, 1985;273–285.
126. Peterson HA. Iatrogenic spine deformities. In: Weinstein SL, ed. *The pediatric spine: principles and practice*. New York: Raven Press, 1994:651–664.
127. Probert JC, Parker BR. The effect of radiation therapy on bone growth. *Radiology* 1975;114:155.
128. Regen EM, Wilkins WE. The effect of large doses of x-rays on growth of young bone. *J Bone Joint Surg* 1936;18:61–68.
129. Reidy JA, Lingley JR, Gall EA, Barr JS. The effects of roentgen irradiation on epiphyseal growth. II. Experimental studies upon the dog. *J Bone Joint Surg* 1947;29:853–878.
130. Robertson WW, Butler MS, D'Angio GJ, Rate WR. Leg-length discrepancy following irradiation for childhood tumors. *J Pediatr Orthop* 1991;11:284–287.
131. Spangler D. The effect of x-ray therapy for closure of epiphyses. *Radiology* 1941;37:310.

### **Infection**

132. Bergdahl S, Ekengren K, Eriksson M. Neonatal hematogenous osteomyelitis: risk factors for long-term sequelae. *J Pediatr Orthop* 1985;5:564–568.
133. Blanche DW. Osteomyelitis in infants. *J Bone Joint Surg Am* 1952;34:71–85.
134. Bos CF, Mol LJ, Obermann WR, Tjin-Ton ER. Late sequelae of neonatal septic arthritis of the shoulder. *J Bone Joint Surg Br* 1998;80:645–650.
135. Chen SC, Huang SC, Wu CT. Nonspinal tuberculous osteomyelitis in children. *J Formos Med Assoc* 1998;97:26–31.
136. Dick HM, Tietjen R. Humeral lengthening for septic neonatal growth arrest: case report. *J Bone Joint Surg Am* 1978;60:1138–1139.
137. Ellefson BK, Frierson MA, Rancy EM, Ogden JA. Humeral varus: a complication of neonatal, infantile and childhood injury and infection. *J Pediatr Orthop* 1994;14:479–486.
138. Langenskiöld A. Growth disturbance after osteomyelitis of femoral condyles in infants. *Acta Orthop Scand* 1984;55:1–13.
139. Lindblood B, Ekengren K, Aurelius G. The prognosis of acute hematogenous osteomyelitis and its complications during early infancy after the advent of antibiotics. *Acta Pediatr Scand* 1965;54:24.
140. Morrey BF, Peterson HA. Hematogenous pyogenic osteomyelitis in children. *Orthop Clin North Am* 1975;6:935–951.
141. Peters W, Irving J, Letts M. Long-term effects of neonatal bone and joint infection on adjacent growth plates. *J Pediatr Orthop* 1992;12:806–810.
142. Peterson HA. Surgical lengthening of the humerus. Case report and review. *J Pediatr Orthop* 1989;9:596–601.
143. Price CT, Mills WL. Radial lengthening for septic growth arrest. *J Pediatr Orthop* 1983;3:88–91.

### **Tumor**

144. Chew DK, Menelaus MB, Richardson MD. Ollier's disease: varus angulation at the lower femur and its management. *J Pediatr Orthop* 1998;18:202–208.
145. Clayer M, Boatright C, Conrad E. Growth disturbances associated with untreated benign bone cysts [Review]. *Aust N Z J Surg* 1997;67:872–873.
146. Gabos PG, Bowen JR. Epiphyseal-metaphyseal enchondromatosis. A new clinical entity. *J Bone Joint Surg Am* 1998;86:782–792.
147. Herring JA. Instructional case: simple bone cyst with growth arrest. Guest Discussant: Hamlet A. Peterson, M.D. *J Pediatr Orthop* 1987;7:231–235.
148. Madhavan P, Ogilvie C. Premature closure of the upper humeral physis after fracture through simple bone cyst. *J Pediatr Orthop* 1998;7:83–85.
149. Neer CS, Francis KC, Marcove RC, et al. Treatment of unicameral bone cyst. A follow-up of 175 cases. *J Bone Joint Surg Am* 1966;48:731–745.
150. Stanton RP, Abdel-Mota'al MM. Growth arrest resulting from unicameral bone cyst. *J Pediatr Orthop* 1998;18:198–201.

### **Vascular Impairment**

151. Brashear HR. Epiphyseal avascular necrosis and its relation to longitudinal bone growth. *J Bone Joint Surg Am* 1963;45:1423–1438.
152. Currarino G, Erlandson ME. Premature fusion of epiphyses in Cooley's anemia. *Radiology* 1964;83:656–664.
153. Hultén O. The influence of a fixation bandage on the peripheral blood vessels and the circulation. *Acta Chir Scand* 1951;101:151–159.
154. Mustard WT, Simmons EH. Experimental arterial spasm in the lower extremities produced by traction. *J Bone Joint Surg Br* 1953;35:437.
155. Peterson HA. Premature physeal arrest of the distal tibia associated with temporary arterial insufficiency. *J Pediatr Orthop* 1993;13:672–675.
156. Sanpera I, Fixsen JA, Hill RA. Injuries to the physis by extravasation. A rare cause of growth plate arrest. *J Bone Joint Surg Br* 1994;76:278–280.
157. Tomita Y, Tsai T, Steyers C, et al. The role of the epiphyseal and metaphyseal circulations on the longitudinal growth in the dog: an experimental study. *J Hand Surg* 1985;11A:375.
158. Trueta J. The role of the vessels in osteogenesis. *J Bone Joint Surg Br* 1963;45:402–418.
159. Trueta J, Amato VP. The vascular contribution to osteogenesis. III. Changes in the growth cartilage caused by experimentally induced ischaemia. *J Bone Joint Surg Br* 1960;42:571–587.
160. Trueta J, Morgan JD. The vascular contribution to osteogenesis. I. Studies by the injection method. *J Bone Joint Surg Br* 1960;42:97–109.

### **Neural Involvement**

161. Rodgers WB, Schwend RM, Jaramillo D, et al. Chronic physeal fractures in myelodysplasia: magnetic resonance analysis, histologic description, treatment, and outcome. *J Pediatr Orthop* 1997;17:615–621.
162. Troupp H. Nervous and vascular influence on longitudinal growth of bone: an experimental study on rabbits. *Acta Orthop Scand* 1961;51(Suppl):7–78.

### **Metabolic Abnormality**

163. Bright RW, Elmore SM. Some effects of immunosuppressive drugs on the epiphyseal plates of rats. *Surg Forum* 1967;18:485.
164. Cohen A, Rovelli R, Zecca S, et al. Endocrine late effects in children who underwent bone marrow transplantation: review [Abstract]. *Bone Marrow Transplant* 1999;21(Suppl 2):564–567.
165. Griffin JB. Hypervitaminosis A. *Bull Hosp Spec Surg* 1959;1:10–19.
166. Kuizon BD, Goodman WG, Juppner H, et al. Diminished linear growth during intermittent calcitriol therapy in children undergoing CPPD. *Kidney Int* 1998;53:205–211.
167. Pease CN. Focal retardation and arrestment of growth of bones due to vitamin A intoxication. *JAMA* 1962;182:980–985.
168. Powell DR, Liu F, Baker BK, et al. Insulin-like growth factor binding proteins vs growth inhibitors in children with chronic renal failure. *Pediatr Nephrol* 1996;10:343–347.
169. Saborio P, Krieg RJ, Hahn S, Chan JC. Pathophysiology of growth retardation in chronic renal failure. *Chung-Han Min Kuo Hsiao Erh Koi Hsueh Hui Tsa Chih* 1998;39:21–27.
170. Silverman FN. An unusual osseous sequel to infantile scurvy. *J Bone Joint Surg Am* 1953;35:215–220.
171. Silverman FN. Recovery from epiphyseal invagination: sequel to an unusual complication of scurvy. *J Bone Joint Surg Am* 1970;52A:384–390.
172. Sprague PL. Epiphyseometaphyseal cupping following infantile scurvy. *Pediatr Radio* 1976;4:122–123.
173. Wolbach SB. Vitamin A deficiency and excess in relation to skeletal growth. *J Bone Joint Surg* 1947;29:171–192.

### **Cold Injury (Frostbite)**

174. Bennett RB, Blount WP. Destruction of epiphyses by freezing. *JAMA* 1935;105:661–662.
175. Benoit RR. Thermal injuries of the growth plate. In: Uthoff HK, Wiley JJ, eds. *Behavior of the growth plate*. New York: Raven Press, 1988;119–122.
176. Bigelow DR, Ritchie GW. The effects of frostbite in childhood. *J Bone Joint Surg Br* 1963;45:122–131.
177. Blair JR, Shatski R, Orr KD. Sequelae injury to cold in 100 patients—follow-up study four years after occurrence of cold injury. *JAMA* 1957;163:1203.
178. Blaustein A, Siegler R. Pathology of experimental frostbite. *NY J Med* 1954;54:2968.
179. Brown FE, Spiegel SK, Boyle EW. Digital deformity: an effect of frostbite in children. *Pediatrics* 1983;71:955–959.

180. Carrea CF, Dozin F, McCarty DJ. Arthritis after frostbite injury in children. *Arthritis Rheum* 1979;22:1082–1087.
181. Carrea CF, Dozin F, Flaherty L, McCarty OJ. Radiographic changes in the hands following childhood frostbite injury. *Skeletal Radiol* 1981;6:33–37.
182. Crimson JM, Fuhrman FA. Studies on gangrene following cold injury. *J Clin Invest* 1947;26:486.
183. Crouch C, Smith WL. Long-term sequelae of frostbite. *Pediatr Radio* 1990;20:365–366.
184. Dowdle JA, Kleven LH, House JH, Thompson WW. Frostbite—Effect on the juvenile hand. *Orthop Trans* 1978;2:13.
185. Dreyfuss JR, Glimcher MJ. Epiphyseal injury following frostbite. *N Engl J Med* 1955;253:1065–1068.
186. Florkiewicz L, Kozlowski K. Symmetrical epiphyseal destruction by frostbite. *Arch Dis Child* 1962;37:51–52.
187. Galloway H, Suh J, Parker S, Griffiths H. Frostbite. *Orthopedics* 1991;14:198–200.
188. Graline BJ, Postner JM, Rosch J. Angiography in the diagnosis and therapy of frostbite. *Radiology* 1976;119:305.
189. Hakstian RW. Cold-induced digital epiphyseal necrosis in childhood (symmetric focal ischemic necrosis). *Can J Surg* 1972;15:168–178.
190. House JH, Fidler MD. Frostbite of the hand. In: Green DP, editor. *Operative Hand Surgery*, 2nd ed. New York: Churchill-Livingstone, 1988;2165–2174.
191. Hurlay LA. Angioarchitectural changes associated with rapid rewarming subsequent to freezing injury. *Angiology* 1957;8:19.
192. Lee KE, Pelker RR. Effect of freezing on histologic and biomechanical failure patterns in the rabbit capital femoral growth plate. *J Orthop Res* 1985;3:154.
193. Lindholm A, Nilsson O, Svartholm F. Epiphyseal destruction following frostbite. *Arch Environ Health* 1968;17:681–684.
194. Nakazato T, Ogino T. Epiphyseal destruction of children's hands after frostbite. *J Hand Surg* 1986;11A:289–292.
195. Scow RO. Destruction of cartilage cells in the newborn rat by brief refrigeration with consequent skeletal deformities. *Am J Pathol* 1949;25:143–153.
196. Selke SC. Destruction of phalangeal epiphyses by frostbite. *Radiology* 1969;93:859–860.
197. Shumacker HB, Lempke RE. Recent advances in frostbite with particular reference to experienced studies concerning functional pathology and treatment. *Surgery* 1951;30:873.
198. Thelander HE. Epiphyseal destruction by frostbite. *J Pediatr* 1950;36:105–106.
199. Tischler JM. The soft-tissue and bone changes in frostbite injuries. *Radiology* 1972;102:511–513.
200. Wenzel JE, Burke EC, Bianco AJ. Epiphyseal destruction from frostbite of the hands. *Am J Dis Child* 1970;114:668–670.

### Heat Injury (Burn)

201. Bantz E, Auerbach J. Leg burns from mopeds. *Pediatrics* 1982;70:304.
202. Evans EB, Smith JR. Bone and joint changes following burns—a roentgenographic study, preliminary report. *J Bone Joint Surg Am* 1959;41:785–799.
203. Frantz CH, Delgado S. Limb-length discrepancy after third-degree burns about the foot and ankle. *J Bone Joint Surg* 1966;48:443–450.
204. Gelfand DW, Law EF, MacMillan BG, Barclay TL. The radiographic assessment of skeletal pathology in severely burned children: a review of 250 cases. In: Matter P, Barclay TO, Lonikova Z, eds. *International Congress of Research in Burns*. Vienna: Hans Huber Publishers, 1971.
205. Olney DB. A review of the long-term results of electric-bar fire burns of the hand in children. *Hana* 1983;15:179–184.
206. Stark RH, Matloub HS, Sangen JR, et al. Warm ischemic damage to the epiphyseal growth plate—a rabbit model. *J Hand Surg* 1987;12A:54.
207. VanDemark RE. Burned hands in infants. *South Dakota J Med Pharmacol* 1957;10:1–3.

### Electrical Injuries

208. Ahstrom JP. Epiphyseal injuries of the lower extremity. *Surg Clin North Am* 1965;1:119.
209. Brinn LB, Mosely JE. Bone changes following electrical injury. *Am J Radiol* 1966;97:682.
210. Duffner DW. Management of electroshock injury. *Orthop Rev* 1982;11:57.
211. Ogden JA, Southwick WO. Electrical injury involving the immature skeleton. *Skeletal Radiol* 1981;6:187.
212. Vickers DW. Premature incomplete fusion of the growth plate: causes and treatment by resection (physeolysis) in 15 cases. *Aust NZ J Surg* 1980;50:393–401.

### Laser Injuries

213. Morein G, Gassner S, Kaplan I. Bone growth alteration resulting from application of CO<sup>2</sup> laser beam to the epiphyseal growth plates. *Acta Orthop Scand* 1978;49:244.

### Stress Injuries

214. Albanese SA, Palmer AK, Kerr DR, et al. Wrist pain and distal growth plate closure of the radius in gymnasts. *J Pediatr Orthop* 1989;9:23–29.
215. Bak K, Boeckstyns M. Epiphysodesis for Bilateral Closure of the Distal Radial Physis in a Gymnast. *Scand J Med Sci Sports* 1997;7:363–366.
216. Boyd KT, Batt ME. Stress fracture of the proximal humeral epiphysis in an elite junior badminton player. *Br J Sports Med* 1997;31:252–253.
217. Caine D, Howe W, Ross W, Bergman G. Does repetitive physeal loading inhibit radial growth in female gymnasts: review. *Clin J Sports Med* 1997;7:302–308.
218. Caine D, Roy S, Singer KM, Broekhoff J. Stress changes of the distal radial growth plate: a radiographic survey and review of the literature. *Am J Sports Med* 1992;20:290–298.
219. Cahill BR. Stress fracture of the proximal tibial epiphysis: a case report. *Am J Sports Med* 1977;5:186–187.
220. Carson WG, Gasser SI. Little leaguer's shoulder. A report of 23 Cases. *Am J Sports Med* 1998;26:575–580.
221. Carter SR, Aldridge MJ. Stress injury of the distal radial growth plate. *J Bone Joint Surg Br* 1988;70:834–836.
222. Chan D, Aldridge MJ, Maffulli N, Davies AM. Chronic stress injuries of the elbow in young gymnasts. *Br J Radiol* 1991;64:1117–1118.
223. DiFiori JP, Mandelbaum BR. Wrist pain in a young gymnast: unusual radiographic findings and MRI evidence of growth plate injury. *Med Sci Sports Exerc* 1996;28:1453–1458.
224. DiFiori JP, Puffer JC, Mandelbaum BR, Dorey F. Distal radial growth plate injury and positive ulnar variance in nonelite gymnasts. *Am J Sports Med* 1997;25:763–768.
225. Fliegel CP. Stress-related widening of the radial growth plate in adolescents. *Ann Radiol (Paris)* 1986;29:374–376.
226. Godshall RW, Hansen CA, Rising DC. Stress fractures through the distal femoral epiphysis in athletes. *Am J Sports Med* 1981;9:14.
227. Greco F, dePalma L, Specchia N, Mannarini M. Growth-plate cartilage metabolic response to mechanical stress. *J Pediatr Orthop* 1989;9:520–524.
228. Liebling MS, Bordon WE, Ruzal-Shapiro C, et al. Gymnast's wrist (pseudorickets growth plate abnormality) in adolescent athletes: findings on plain films and MR imaging. *Am J Roentgenol* 1995;164:157–159.
229. Mandelbaum BR, Bartolozzi AR, Davis CA, et al. Wrist pain syndrome in the gymnast: pathogenetic, diagnostic, and therapeutic considerations. *Am J Sports Med* 1989;17:305–317.
230. Roy S, Caine D, Singer K. Stress changes of the distal radial epiphysis in a young gymnast. A report of 21 cases and review of the literature. *Am J Sports Med* 1985;13:301–308.
231. Ruggles DL, Peterson HA, Scott SG. Radial growth plate injury in a female gymnast. *Med Sci Sports Exerc* 1991;23:393–396.
232. Shih C, Chang CY, Penn IW, et al. Chronically stressed wrists in adolescent gymnasts: MR imaging appearance. *Radiology* 1995 (published erratum appears in *Radiology* 197:319, 1997) 5:855–859.

### Longitudinal Compression (Salter-Harris Type V)

233. Abram LJ, Thompson GH. Deformity after premature closure of the distal radial physis following a torus fracture with a physeal compression injury. Report of a case. *J Bone Joint Surg Am* 1987;69:1450–1453.
234. Aminian A, Schoenecker PL. Premature closure of the distal radial physis after fracture of the distal radial metaphysis. *J Pediatr Orthop* 1995;15:495–498.
235. Bowler JR, Mubarak SJ, Wenger DR. Tibial physeal closure and genu recurvatum after femoral fracture: occurrence without a tibial traction pin. Case report. *J Pediatr Orthop* 1990;10:653–657.
236. Connolly JF, Eastman T, Huurman WW. Torus fracture of the distal radius producing growth arrest. *Nebr Med J* 1985;June:204–207.
237. Gomes LS, Volpan JB, Goncalves RP. Traumatic separation of epiphyses. An experimental study in rats. *Clin Orthop* 1988;236:286–294.
238. Keret D, Mendez AA, Harcke HT, MacEwen GD. Type V physeal injury: a case report. *J Pediatr Orthop* 1990;10:545–548.
239. Mendez AA, Barta E, Grillot MB, Lin JJ. Compression (Salter-Harris type V) physeal fracture: an experimental model in the rat. *J Pediatr Orthop* 1992;12:29–37.
240. Moen CT, Pelker RP. Biomechanical and histological correlations in growth plate failure. *J Pediatr Orthop* 1984;4:180–184.
241. Ogden JA, Ganey T, Light TR, Southwick WO. The pathology of acute chondro-osseous injury in the child. *Yale J Biol Med* 1993;66:219–233.
242. Peterson HA, Burkhardt SS. Compression injury of the epiphyseal growth plate: fact or fiction? *J Pediatr Orthop* 1981;1:377–384.
243. Rang M, Armstrong P, Crawford AH, et al. Symposium: management of fractures in children and adolescents. Part I. *Contemp Orthop* 1991;25:517–544.
244. Trueta J, Trias A. The vascular contribution to osteogenesis. IV. The effect of pressure upon the epiphyseal cartilage of the rabbit. *J Bone Joint Surg Br* 1961;43:800.
245. Valverde JA, Albinana J, Certucha A. Early posttraumatic physeal arrest in distal radius after a compression injury. *J Pediatr Orthop* 1996;5:57–60.

### Developmental Physeal Abnormalities

246. Beck CL, Burke SW, Roberts JM, Johnston CE. Physeal bridge resection in infantile Blount disease. *J Pediatr Orthop* 1987;7:161–163.
247. Bradway JK, Klassen RA, Peterson HA. Blount disease: a review of the English literature. *J Pediatr Orthop* 1987;7:472–480.
248. Cook PA, Yu JS, Wiand W, et al. Madelung deformity in skeletally immature patients: Morphologic assessment using radiography, CT, and MRI. *J Comput Assist Tomogr* 1996;20:505–511.
249. Takahara M, Sasaki I, Kimura T, et al. Second fracture of the distal humerus after varus malunion of a supracondylar fracture in children. *J Bone Joint Surg Br* 1998;80:791–797.
250. Vender MI, Watson HK. Acquired Madelung-like deformity in a gymnast. *J Hand Surg* 1988;13A:19–21.

251. Vickers DW. Madelung deformity: surgical prophylaxis (physiolysis) during later growth period by resection of the dyschondroostosis lesion. *J Hand Surg* 1992;17B:401–407.
252. Vickers D. Epiphysiolysis. *Curr Orthop* 1989;3:41–47.

### Iatrogenic Injuries

253. Bisson LJ, Wickiewicz T, Levinson M, Warren R. ACL reconstruction in children with open physes. *Orthopedics* 1998;21:259–263.
254. Bjerkreim I, Benum P. Genu recurvatum. A late complication of tibial wire traction in fractures of the femur in children. *Acta Orthop Scand* 1975;46:1012–1019.
255. Blount WP, Clarke RG. Control of one growth by epiphyseal stapling. *J Bone Joint Surg Am* 1949;31:464.
256. Boyden EM, Peterson HA. Partial premature closure of the distal radial physis associated with Kirschner wire fixation. *Orthopedics* 1991;14:585–588.
257. Christensen NO. Growth arrest by stapling. *Acta Orthop Scand* 1973;151(Suppl):1.
258. Cool WP, Carter SR, Grimer RJ, et al. Growth after extendible endoprosthetic replacement of the distal femur. *J Bone Joint Surg Br* 1997;79B:938–942.
259. Ford LT, Canales GM. A study of experimental trauma and attempts to stimulate growth of the lower femoral epiphyses in rabbits. III. *J Bone Joint Surg Am* 1960;42:439–446.
260. Ford LT, Key JA. A study of experimental trauma to the distal femoral epiphysis in rabbits. *J Bone Joint Surg Am* 1956;38:84–92.
261. Gamble JG, Decker S, Abrams RC. Short first ray as a complication of multiple metatarsal osteotomies. *Clin Orthop* 1982;164:241–244.
262. Garcés GL, Mugica-Garay I, Coviella NL, Guerado E. Growth plate modifications after drilling. *J Pediatr Orthop* 1994;14:225–228.
263. Harris WR. Epiphyseal injuries. *Instruct Course Lect* 1958;15:206–214.
264. Hass SL. Mechanical retardation of bone growth. *J Bone Joint Surg Am* 1948;30:506–512.
265. Holden D, Siff S, Butler J, Cain T. Shortening of the first metatarsal as a complication of metatarsal osteotomies. *J Bone Joint Surg Am* 1984;66:582–587.
266. Johnson JT, Southwick WO. Growth following transepiphyseal bone grafts. *J Bone Joint Surg Am* 1960;42:1381–1395.
267. Lo IK, Bell DM, Fowler PJ. Anterior cruciate ligament injuries in the skeletally immature patient. *Instruct Course Lect* 1998;47:351–359.
268. Matara MJ, Siegel MG. Arthroscopic reconstruction of the ACL with semitendinosus-gracilis autograft in skeletally immature adolescent patients. *Am J Knee Surg* 1997;10:60–69.
269. Nordentoft EL. Experimental epiphyseal injuries: grading of traumas and attempts at treating traumatic epiphyseal arrest in animals. *Acta Orthop Scand* 1969;40:176.
270. Peterson HA. Brachymetatarsia of the first metatarsal treated by surgical lengthening. In: Simons GW, ed. *The clubfoot, the present and a view of the future*. New York: Springer-Verlag, 1994;360–369.
271. Pritchett JW. Does pinning cause distal radial growth plate arrest? *Orthopedics* 1994;17:550–552.
272. Sarafin J. Effect of longitudinal transection of the epiphysis and metaphysis on cartilaginous growth. *Am Diag Orthop Lit* 1970;1:17.
273. Siffert RS. The effect of staples and longitudinal wires on epiphyseal growth. An experimental study. *J Bone Joint Surg Am* 1956;38:1077–1088.
274. Steedman JT, Peterson HA. Brachymetatarsia of the first metatarsal treated by surgical lengthening. *J Pediatr Orthop* 1992;12:780–785.

### Physeal Arrest

#### Etiology

275. Barash ES, Siffert RS. The potential for growth of experimentally produced hemiepiphysis. *J Bone Joint Surg Am* 1966;48:1548–1553.
276. Caffey J. Traumatic cupping of the metaphysis of growing bones. *Am J Roentgenol* 1970;108:451–460.
277. Friedenbergs ZB. Reaction of the epiphysis to partial surgical resection. *J Bone Joint Surg Am* 1957;39:332–340.
278. Friedenbergs ZB, Brashear R. Bone growth following partial resection of epiphyseal cartilage. *Am J Surg* 1956;91:362.
279. Key JA, Ford LT. A study of experimental trauma to the distal femoral epiphysis in rabbits—II. *J Bone Joint Surg Am* 1958;40:887–896.
280. Langenskiöld A. Traumatic premature closure of the distal tibial epiphyseal plate. *Acta Orthop Scand* 1967;38:520.
281. Peterson CA, Peterson HA. Analysis of the incidence of injuries to the epiphyseal growth plate. *J Trauma* 1972;12:275–281.

#### Assessment

282. Altongy JF, Harcke TH, Bowen JR. Measurement of leg-length inequality by microdose digital radiographs. *J Pediatr Orthop* 1987;7:311–316.
283. Borsa JJ, Peterson HA, Ehman RL. MR imaging of physeal bars. *Radiology* 1996;199:683–687.
284. Bright RW. Partial growth arrest: Identification, classification, and results of treatment. *Orthop Trans* 1982;6:655.
285. Bylander B, Aronson S, Egund N, et al. Growth disturbance after physeal injury of the distal femur and proximal tibia studied by roentgen stereophotogrammetry. *Arch Orthop Trauma Surg* 1981;98:225–285.
286. Bylander B, Hansson LI, Kärrholm J, Naversten Y. Scintimetric evaluation of posttraumatic and postoperative growth disturbance using 99m-Tc MDP. *Acta Radiol* 1983;24:85–96.
287. Carlson WO, Wenger DR. A mapping method to prepare for surgical excision of a partial physeal arrest. *J Pediatr Orthop* 1984;4:232.
288. DeCampo JF, Boldt DW. Computed tomography of partial growth plate arrest: initial experience. *Skeletal Radio* 1986;15:526–529.
289. Gabel GT, Peterson HA, Berquist TH. Premature partial physeal arrest. Diagnosis by magnetic resonance imaging in two cases. *Clin Orthop* 1991;272:242–247.
290. Green WT, Wyatt GM, Anderson M. Orthoroentgenography as a method of measuring the bones of the lower extremities. *Clin Orthop* 1968;61:10–15.
291. Greulich WW, Pyle SI. *Radiographic atlas of skeletal development of the hand and wrist*. Stanford, CA: Stanford University Press, 1959.
292. Havranek R, Lizler J. Magnetic resonance imaging in the evaluation of partial growth arrest after physeal injury in children. *J Bone Joint Surg Am* 1991;73:1234–1241.
293. Hickey PM. Teleroentgenography as an aid in orthopedic measurements. *Am J Radiol* 1924;11:232–233.
294. Howman-Giles R, Trochei M, Yeates K, et al. Partial growth plate closure: apex view on bone scan: technique. *J Pediatr Orthop* 1985;5:109–111.
295. Hynes D, O'Brien T. Growth disturbance lines after injury of the distal tibial physis: Their significance in prognosis. *J Bone Joint Surg Br* 1988;70:231–233.
296. Jaramillo D, Laor T, Zaleske DS. Indirect trauma to the growth plate: Results of MR imaging after epiphyseal and metaphyseal injury in rabbits. *Radiology* 1993;187:171–178.
297. Jaramillo D, Shapiro F, Hoffer FA, et al. Posttraumatic growth plate abnormalities: MR imaging of bony-bridge formation in rabbits. *Radiology* 1990;175:767–773.
298. Kumar R, Madewell JE, Swischuk LE. The normal and abnormal growth plate. *Radiol Clin North Am* 1987;25:1133.
299. Loder RT, Swinford AE, Kuhns LR. The use of helical computed tomographic scan to assess bony physeal bridges. *J Pediatr Orthop* 1997;17:356–359.
300. Mayer V, Marchisello PJ. Traumatic partial arrest of tibial physis. *Clin Orthop* 1984;183:99.
301. Murray K, Nixon GW. Epiphyseal growth plate: evaluation with modified coronal CT. *Radiology* 1988;166:263–265.
302. Ogden JA. Growth slowdown and arrest lines. *J Pediatr Orthop* 1984;4:409–415.
303. Park EA. The imprinting of nutritional disturbances on the growing bone. *Pediatrics* 1964;33:815–862.
304. Peterson HA. Magnetic resonance imaging of growth plates. In: dePablos J, ed. *Surgery of the growth plate*. Madrid: Ediciones Ergon, SA, 1998;22–28.
305. Peterson HA. Letter to the editors. *J Pediatr Orthop* 1994;14:823.
306. Peterson HA. Scanning the bridge. In: Uthoff HK, Wiley JJ, eds. *Behavior of the growth plate*. New York: Raven Press, 1988;247–258.
307. Porat S, Nyska M, Nyska A, Fields S. Assessment of bony bridge by computed tomography: experimental model in the rabbit and clinical application. *J Pediatr Orthop* 1987;7:155–160.
308. Pugh DG, Winkler NT. Scanography for leg-length measurement: an easy, satisfactory method. *Radiology* 1966;87:130–133.
309. Tanner JM, Whitehouse RH, Marshall WA. *Assessment of skeletal maturity and prediction of adult height (TW2 methods)*. London: Academic Press; 1975.
310. Young JW, Bright RW, Whitley NO. Computed tomography in the evaluation of partial growth plate arrest in children. *Skeletal Radio* 1986;15:530.
311. Young JW, Bright RW, Whitley NO. The value of CT in the assessment of partial growth arrests in children. *Radiology* 1984;153:95.

#### Management

312. Apte SS, Kenwright J. Physeal distraction and cell proliferation in the growth plate. *J Bone Joint Surg Br* 1994;76:837–843.
313. Bowen CV, Ethridge CP, O'Brien BM, et al. Experimental microvascular growth plate transfers. Part I: Investigation of vascularity. *J Bone Joint Surg Br* 1988;70:305–310.
314. Bowen CV, O'Brien BM, Gumley GJ. Experimental microvascular growth plate transfers. Part II: investigation of feasibility. *J Bone Joint Surg Br* 1988;70B:311–314.
315. Brown K, Marie P, Lyszakowski T, et al. Epiphyseal growth after free fibular transfer with and without microvascular anastomosis. Experimental study in the dog. *J Bone Joint Surg Br* 1983;65B:493–501.
316. Canadell J, dePablos J. Breaking bony bridges by physeal distraction: A new approach. *Int Orthop* 1985;9:223–229.
317. Connolly JF, Huurman WW, Lippiello L, Pankaj R. Epiphyseal traction to correct acquired growth deformities: an animal and clinical investigation. *Clin Orthop* 1986;202:258–267.
318. Connolly JF, Huurman WW, Ray S. Physeal distraction treatment of fracture deformities. *Orthop Trans* 1979;3:231.
319. Cundy PJ, Jofe M, Zuleske DJ, et al. Physeal reconstruction using tissue donated from early postnatal limbs in a murine model. *J Orthop Res* 1991;9:360–366.
320. Eades JW, Peacock EE. Autogenous transplantation of an interphalangeal joint and proximal phalangeal epiphysis: case report and 10-year follow-up. *J Bone Joint Surg Am* 1966;48A:775–778.
321. Fjeld T, Steen H. Growth retardation after experimental limb lengthening by epiphyseal distraction. *J Pediatr Orthop* 1990;10:463–466.
322. Harris WR, Martin R, Tile M. Transplantation of epiphyseal plates. *J Bone Joint Surg Am* 1965;47:897–914.
323. Heikel HV. Experimental epiphyseal transplantation. Part II: histological observations. *Acta Orthop Scand* 1960–1961;30:1.
324. Hoffman S, Siffert RS, Simon BE. Experimental and clinical experiences in epiphyseal transplantation. *Plast Reconstr Surg* 1972;50:58.
325. Jones CB, Dewar ME, Aichroth PM, et al. Epiphyseal distraction monitored by strain gauges. *J Bone Joint Surg Br* 1989;71:651.
326. Lalanandham T, Ehrlich MG, Zaleske DJ, et al. Viability and metabolism of cartilage transplanted to physeal regions. *J Pediatr Orthop* 1990;10:450–458.

327. Nettelblad H, Randolph MA, Weiland AJ. Heterotopic microvascular growth plate transplantation of the proximal fibula: An experimental canine model. *Plast Reconstr Surg* 1986;77:814–820.
328. Nettelblad H, Randolph MA, Weiland AJ. Free microvascular epiphyseal-plate transplantation. *J Bone Joint Surg Am* 1984;66:1421–1430.
329. Olin AO, Creaseman C, Shapiro F. Free physeal transplantation in the rabbit: an experimental approach to focal lesions. *J Bone Joint Surg Am* 1984;66:7.
330. Peltonen JI, Karaharju EO, Alitalo I. Experimental epiphyseal distraction producing and correcting angular deformities. *J Bone Joint Surg Br* 1984;66:598.
331. Peterson HA. Management of partial physeal arrest. In: Chapman MW, ed. *Operative orthopaedics*, 2nd ed. Philadelphia: JB Lippincott, 1993;3065–3075.
332. Peterson HA. Partial growth plate arrest and its treatment. In: Morrissy RT, ed. *Lovell and Winter's pediatric orthopaedics*, 3rd ed. Philadelphia: JB Lippincott, 1990;1071–1089.
333. Peterson HA. Review: partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258.
334. Ray SK, Connolly JF, Huurman WW. Distraction treatment of deformities due to physeal fractures. *Surg Forum* 1978;29:543.
335. Ring PA. Transplantation of epiphyseal cartilage: an experimental study. *J Bone Joint Surg Br* 1955;37:642–657.
336. Scheffer MM, Peterson HA. Opening-wedge osteotomy for angular deformities of long bones in children. *Adv Orthop Surg* 1995;19:16–18.
337. Scheffer MM, Peterson HA. Opening-wedge osteotomy for angular deformities of long bones in children. *J Bone Joint Surg Am* 1994;76:325–334.
338. Spira E, Farin I. Epiphyseal transplantation: a case report. *J Bone Joint Surg Am* 1964;46:1278–1282.
339. Teot L, Bosse JP, Gilbert A, Tremblay GR. Pedicle graft epiphysis transplantation. *Clin Orthop* 1983;180:206–218.
340. Tsai TM, Ludwig L, Tonkin M. Vascularized fibular epiphyseal transfer. A clinical study. *Clin Orthop* 1986;210:228–234.
341. Whitesides ES. Normal growth in transplanted epiphysis. *J Bone Joint Surg Am* 1977;59:546–547.
342. Wilson JN. Epiphyseal transplantation. A clinical study. *J Bone Joint Surg Am* 1966;48:245.
343. Wolohan MJ, Zaleske DJ. Hemiepiphyseal reconstruction using tissue donated from fetal limbs in a murine model. *J Orthop Res* 1990;9:180–185.
344. Zaleske DJ, Ehrlich MG, Piliero C, et al. Growth-plate behavior in whole joint replantation in the rabbit. *J Bone Joint Surg Am* 1982;64:249–258.
345. Zaleske DJ, Floyd WE, Hallet J, et al. Epiphyseal replacement using developing tissue donors in a murine model: A combined histologic and radiographic study. *J Orthop Res* 1988;6:155–165.
346. Zehntner MK, Jakob RP, McGarity PL. Growth disturbance of the distal radius epiphysis after trauma: Operative treatment by corrective radial osteotomy. *J Pediatr Orthop* 1990;10:411–415.

### Experimental Studies

347. Barr SJ, Zaleske DJ. Physeal reconstruction with blocks of cartilage of varying development time. *J Pediatr Orthop* 1992;12:766–773.
348. Bright RW. Further canine studies with medical elastomer X7-2320 after osseous bridge resection for partial physeal plate closure. *Orthop Trans* 1981;5:264.
349. Bright RW. Surgical correction of partial growth plate closure. Laboratory and clinical experience. *Orthop Rev* 1978;8:149.
350. Bright RW. Operative correction of partial epiphyseal plate closure by osseous bridge resection and silicone rubber implant. *J Bone Joint Surg Am* 1974;56:655–664.
351. Bueche MJ, Phillips WA, Gordon J, et al. Effect of interposition material on mechanical behavior in partial physeal resection: a canine model. *J Pediatr Orthop* 1990;10:459–462.
352. Foster BK, Hansen AL, Gibson GJ, et al. Reimplantation of growth plate chondrocytes into growth plate defects in sheep. *J Orthop Res* 1990;8:555–564.
353. Hanson AL, Foster BK, Gibson GJ, et al. Growth plate chondrocyte cultures for reimplantation into growth plate defects in sheep. Characterization in cultures. *Clin Orthop* 1990;256:286–298.
354. Kawabe N, Ehrlich MG, Mankin GH. Growth plate reconstruction using chondrocyte allograft transplants. *J Pediatr Orthop* 1987;7:381–388.
355. Österman K. Operative elimination of partial premature closure: an experimental study. *Acta Orthop Scand* 1972;147(Suppl):7–79.
356. Sudmann E, Husby OS, Bang G. Inhibition of partial closure of epiphyseal plate in rabbits by indomethacin. *Acta Orthop Scand* 1982;53:507–511.

### Clinical Experience

357. Alford BA, Oshman DG, Sussman MD. Radiographic appearances following surgical correction of the partially fused epiphyseal plate. *Skeletal Radio*. 1986;15:146.
358. Birch JG, Herring JA, Wenger DR. Surgical anatomy of selected physes. *J Pediatr Orthop* 1984;4:224–231.
359. Bollini G, Tallet JM, Jacquemier M, Bouyala JM. Case report: new procedure to remove a centrally located bar. *J Pediatr Orthop* 1990;10:662–666.
360. Botte MJ, Sutherland DH, Mubarak SJ. Treatment of partial epiphyseal arrest by resection of the osseous bridge and interposition with silicone rubber. *Orthop Trans* 1986;10:457.
361. Broughton NS, Dickens DR, Cole WG, Menelaus BB. Epiphyseolysis for partial growth plate arrest. Result after four years or at maturity. *J Bone Joint Surg Br* 1989;71:13–16.
362. Cabanela ME, Coventry MB, MacCarty CS, Miller WE. The fate of patients with methylmethacrylate cranioplasty. *J Bone Joint Surg Am* 1972;54A:278–281.
363. Coleman SS. Physiolytic resection of premature incomplete fusion of the physis. *Orthop Trans* 1986;10:554–555.
364. Heikel HV. Has epiphyseodesis in one end of a long bone a growth-stimulating effect on the other end? An experimental study. *Acta Orthop Scand* 1961;31:18.
365. Hume MC, Burstein SM. Silastic implantation after epiphyseal bar resection. *Orthop Trans* 1988;12:151.
366. Kasser JR. Physeal bar resections after growth arrest about the knee. *Clin Orthop* 1990;255:68–74.
367. Klassen RA, Peterson HA. Excision of physeal bars: the Mayo Clinic experience 1968–1978. *Orthop Trans* 1982;6:65.
368. Langenskiöld A, Österman K, Valle M. Growth of fat grafts after operation for partial bone growth arrest: demonstration by CT scanning. *J Pediatr Orthop* 1987;7:389–394.
369. Langenskiöld A, Videman T, Nevalainen T. The fate of fat transplants in operations for partial closure of the growth plate. Clinical examples and an experimental study. *J Bone Joint Surg Br* 1986;68:234–238.
370. Langenskiöld A. Surgical treatment of partial closure of the growth plate. *J Pediatr Orthop* 1981;1:3–11.
371. Langenskiöld A, Österman K. Surgical treatment of partial closure of the epiphyseal plate. *Reconstr Surg Traumatol* 1979;17:48.
372. Langenskiöld A. Partial closure of epiphyseal plate: principles of treatment. *Int Orthop* 1978;2:95.
373. Langenskiöld A. An operation for partial closure of an epiphyseal plate in children, and its experimental basis. *J Bone Joint Surg Br* 1975;57:325–330.
374. Langenskiöld A. The possibilities of eliminating premature partial closure of an epiphyseal plate caused by trauma or disease. *Acta Orthop Scand* 1967;38:267–279.
375. Mallet J, Rey JC. Treatment of traumatic partial epiphysiodesis in a child by epiphysiolysis. *Int Orthop* 1978;1:309.
376. Mallet J. Les épiphysiodèses partielles traumatiques de L'extrémité inférieure du tibia chez l'enfant: leur traitement avec désépiphysiodèse. *Rev Chir Orthop* 1975;61:5.
377. Peterson HA. Treatment of physeal bony bridges by means of bridge resection and interposition of cranioplast. In: dePablos J, ed. *Surgery of the growth plate*. Madrid: Ediciones Ergon, S.A., 1998;299–307.
378. Peterson HA. Treatment of physeal bony bridges of the distal femur and proximal tibia. In: dePablos J, ed. *The immature knee*. Barcelona: Masson, S.A., 1998;333–342.
379. Peterson HA. Growth plate injuries and physeal bridge resection. In: Buckwalter JA, Ehrlich MG, Sandell LJ, Trippel SB, eds. *Skeletal growth and development: clinical issues and basic science advances*. American Academy of Orthopaedic Surgeons, Rosemont, Illinois, 1998;561–575.
380. Peterson HA. Treatment of physeal bony bridges by means of bridge resection and interposition of cranioplast. *Mapfre Medicina* 1993;4(Suppl II):226–230.
381. Peterson HA. Operative correction of postfracture arrest of the epiphyseal plate: case report with 10-year follow-up. *J Bone Joint Surg Am* 1980;62:1018–1020.
382. Post WR, Jones ET. Tetracycline labeling as an aid to complete excision of partial physeal arrest: a rabbit model. *J Pediatr Orthop* 1992;12:736–760.
383. Talbert RE, Wilkins KE. Physeal bar resection: factors contributing to success. *Orthop Trans* 1987;11:549.
384. Versveld GA. Surgical management of partial closure of the growth plate (Abstract). *J Bone Joint Surg Br* 1984;66:460.
385. Visser JD, Nielsen HK. Operative correction of abnormal central epiphyseal plate closure by transmetaphyseal bone bridge resection and implantation of fat. *Neth J Surg* 1981;33:140.

## PATHOLOGIC FRACTURES ASSOCIATED WITH TUMORS AND UNIQUE CONDITIONS OF THE MUSCULOSKELETAL SYSTEM

JOHN P. DORMANS  
JOHN M. FLYNN

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As a child grows, the pattern of injury and the response to injury and treatment change. One of the more difficult situations encountered in caring for children occurs when the diagnosis and treatment of a complex injury are complicated by a preexisting underlying disease process or condition of the skeleton. The diagnosis of both the injury and the underlying condition depends on an accurate understanding of how these conditions alter the presentation of the child, given that evaluation of the traumatic injury may reveal the underlying condition for the first time. This chapter describes the pertinent clinical and radiographic features of the conditions of the pediatric musculoskeletal system that can predispose a child to pathologic fracture, including specific patterns of injury and special concerns of treatment. For most orthopaedic surgeons, experience with these conditions is limited, and diligence is needed to avoid pitfalls of treatment. The physician must always be aware of the possibility of a pathologic fracture, especially when a patient presents with a fracture following minimal trauma.

A pathologic fracture is defined as a fracture that occurs through abnormal bone. These fractures occur in bone that lacks normal biomechanical and viscoelastic properties. Pathologic fractures may result from intrinsic or extrinsic processes. Examples of intrinsic processes include the osteopenia of osteogenesis imperfecta or replacement of bone with tumor. With extrinsic processes, the weakness is caused by something that lessens the inherent structural integrity of bone, such as radiation or a hole in bone from biopsy or internal fixation. Additionally, pathologic fractures may result from localized (a bone cyst, for example) or generalized processes (such as osteopetrosis), and the fracture may be correctable (rickets) or noncorrectable (metastatic cancer). A fracture similar to a pathologic fracture can occur through anatomically normal bone that is weakened by normal structures such as a vascular foramina ([Fig. 6-1](#)).



**FIGURE 6-1.** A fracture through the vascular foramina of this 9-year-old boy's left clavicle. The fracture healed uneventfully.

Pathologic fractures can occur in children with generalized bone conditions and in those with tumors or tumor-like processes of bone. Although the presenting episode of a child with generalized bone disease (osteogenesis imperfecta, osteopetrosis, and rickets) may be a fracture, more commonly, the diagnosis has been made based on clinical findings such as history, physical examination, radiographs, or laboratory findings. Often, the history is most helpful. For example, pathologic fracture secondary to generalized osteopenia associated with chronic drug therapy (steroids or anticonvulsants) may become evident through the patient's history. Some of the key points in analysis of a pediatric patient with a musculoskeletal tumor or tumor-like lesion are shown in [Table 6-1](#).

1. Age of patient (see Table 6-2)
2. Location of lesion (see Table 6-3)  
Epiphysis, metaphysis, diaphysis?  
Central or eccentric?
3. What is the lesion doing to the bone? (Pattern of involvement)
  - a. Zone of transition (narrow or wide; can measure)
  - b. Geographic versus "moth eaten" versus permeative
4. What is the bone doing to the lesion?  
Periosteal response or "walling off"  
No response?  
Early, immature?  
Late, mature?
5. Is there a characteristic appearance of the lesion, ie matrix?  
Lytic, calcified, ossified, "Ground-glass"

**TABLE 6-1. EVALUATION OF A PEDIATRIC PATIENT WITH A MUSCULOSKELETAL TUMOR OR TUMOR-LIKE LESION**

Hipp and colleagues proposed a way of quantifying the risk of pathologic fracture in 1995 ( 3). They defined the *factor of risk* as the load applied to the involved bone divided by the load required for bone failure. Other attempts have been made to measure the risk of pathologic fracture in patients with underlying conditions of bone (5,27,34). Unfortunately, retrospective studies have failed to find any predictive methods based on radiographic findings that can accurately forecast fracture in most situations. New methods that apply engineering principles to information from computed tomography (CT) scans may provide better noninvasive estimates for the risk of pathologic fracture ( 3).

With pathologic fractures through a tumor or tumor-like lesion, the age of the patient can also be helpful in making the diagnosis ( Table 6-2). Most tumors and tumor-like processes are recognizable by radiographic appearance; the location of the lesion can also be helpful ( Table 6-3) (2).

Age (years)	Benign	Malignant
0-5	Langerhans cell histiocytosis Osteomyelitis	Ewing's sarcoma Lukemia Neuroblastoma (metastatic) Wilms' tumor (metastatic) Neuroblastoma (metastatic)
5-10	Unicameral bone cyst Aneurysmal bone cyst Nonossifying fibroma Fibrous dysplasia Osteoid osteoma Langerhans' cell histiocytosis Osteomyelitis	Ewing's sarcoma Osteosarcoma Rhabdomyosarcoma
10-20	Unicameral bone cyst Aneurysmal bone cyst Osteoid osteoma Fibrous dysplasia Chondroblastoma Osteofibrous dysplasia	Osteosarcoma Ewing's sarcoma (Chondrosarcoma) Rhabdomyosarcoma Synovial cell sarcoma

**TABLE 6-2. PEAK AGE OF COMMON PEDIATRIC MUSCULOSKELETAL CONDITIONS, (TUMORS AND TUMOR-LIKE LESIONS OF BONE)**

Metaphyseal—Any lesion	Epiphyseal	Diaphyseal	Epiphyseal	Diaphyseal
Chondroblastoma Brooke's abscess of the epiphysis osteomyelitis Giant cell tumor Fibrous dysplasia	Chondroblastoma Brooke's abscess of the epiphysis osteomyelitis Langerhans' cell histiocytosis Ewing's sarcoma Lukemia Subacute osteomyelitis Osteosarcoma (secondary)	Chondroblastoma Ewing's sarcoma Lukemia Subacute osteomyelitis Osteosarcoma (secondary)	Chondroblastoma Ewing's sarcoma Lukemia Subacute osteomyelitis Osteosarcoma (secondary)	Chondroblastoma Ewing's sarcoma Lukemia Subacute osteomyelitis Osteosarcoma (secondary)

**TABLE 6-3. LOCATION OF TUMOR AND TUMOR-LIKE LESIONS OF BONE**

Biopsy is sometimes needed to determine the cause of a pathologic fracture, especially with pathologic fractures through tumors or tumor-like processes. In this situation, the surgeon must ensure that biopsy is performed on representative areas of the bony lesion.

Pathologic fractures differ from fractures in normal bone in that the etiology, natural history, and treatment of the underlying abnormality of the bone must be taken into account. Accurate and careful determination of these underlying diagnoses is critical for the appropriate care of these fractures. Fracture management principles often are altered for pathologic fractures because of the abnormal condition responsible for the fracture. The treatment plan must consider both the treatment of the fracture and treatment of the underlying cause of the fracture.

Once a fracture occurs secondary to a previously unrecognized condition, subsequent fractures often can be prevented, usually through patient education. Iatrogenic pathologic fracture often can be prevented by the appropriate use of internal fixation (avoidance of unnecessary cortical penetration with drills and guide pins for example), protection of an extremity with internal fixation by cast or brace when appropriate, and the use of rounded edges of bone biopsy sites ( 1).

### FRACTURES ASSOCIATED WITH CYSTS, TUMORS, OR TUMOR-LIKE PROCESSES

Benign tumors can be classified according to their aggressiveness ( Table 6-4). Stage 1, or latent benign lesions, are usually asymptomatic, discovered incidentally, and seldom associated with pathologic fracture. Stage 2 lesions are intermediate in behavior, and stage 3, or aggressive benign lesions, are usually symptomatic, grow rapidly, and may be associated with pathologic fracture.

- Stage 1, Latent Benign**  
Asymptomatic  
Often discovered incidentally  
Seldom associated with pathologic fracture
- Stage 2, Active Benign**  
Majority  
Tend to grow steadily  
May be symptomatic
- Stage 3, Aggressive Benign**  
Generally symptomatic  
Discomfort, usually tender  
May be associated with pathologic fracture  
Growth rapid

**TABLE 6-4. CLASSIFICATION OF BENIGN LESIONS ACCORDING TO THEIR AGGRESSIVENESS**

## Unicameral Bone Cysts

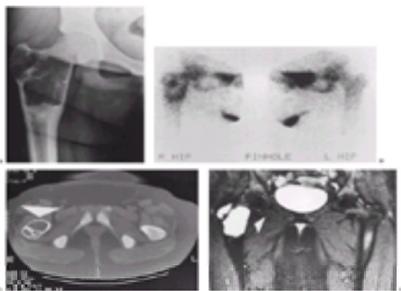
Unicameral bone cysts (UBCs) are radiolucent expansile fluid-filled cystic lesions found most commonly in the metaphyses of long bones. The term *unicameral* suggests a single-chambered cyst, but often, especially after treatment or fracture, the lesions are multiloculated with radiolucent fibrous septa segregating the primary lesion into multiple small chambers (11). The cysts usually contain yellow serous fluid. In order of decreasing frequency, the cysts most commonly occur in the proximal humerus, proximal femur, proximal tibia, distal tibia, distal femur, calcaneus, distal humerus, radius, fibula, ilium, ulna, and rib (34). Approximately 70% of these cysts are found in either the proximal humerus or femur. Some authors have suggested that UBCs evolve from an accumulation of interstitial fluid in the bone because of a defect in venous or lymphatic drainage (14,15,24).

Approximately 75% of patients who have UBCs present with pathologic fractures (7,12,18,19,36,44). These cysts usually are diagnosed within the first two decades of life (13,15,21), and the ratio of males to females is about 2:1 (26). Fractures are often incomplete or minimally displaced. Patients with pathologic fractures through UBCs present with mild to moderate pain in the extremity after either mild trauma or no history of injury. In one series (8), 40% of pathologic femoral neck fractures in children were due to UBCs. Pathologic fracture with collapse of the articular surface of the femoral head and joint incongruity also has been reported (17).

Although the fracture commonly heals within 6 weeks of injury, the UBC usually persists, often with further fracture. Only about 10% of cysts heal after fracture. In a 1993 report of 52 pathologic fractures due to UBC, Ahn and Park (4) found that only 8% of cysts healed at an average of 5.5 years after injury.

### Radiographic Findings

The classic appearance of a UBC is a centrally located, radiolucent, slightly expansile lesion of the metaphysis (Fig. 6-2) (38). The width of the lesion seldom exceeds that of the adjacent physis [a feature characteristic of the more expansile aneurysmal bone cyst (ABC)]. Occasionally, UBCs are located in the diaphysis when the physis has migrated away from the lesion with growth (Fig. 6-3) (23). The so-called fallen fragment sign, described by Reynolds (38) in 1969, is a fracture fragment seen on plain radiographs at the bottom of a cyst suggesting a hollow cavity in the bone rather than a solid tumor (Fig. 6-4). A cortical fragment also may be tilted into the interior of the lesion. The differential diagnosis of UBC includes ABC, fibrous dysplasia, enchondroma, giant cell tumor, and eosinophilic granuloma.



**FIGURE 6-2.** A: A 14-year-old girl presented with right hip pain. X-ray study reveals a large lytic expansile lesion of the proximal femur. B: A technetium scan was performed, and the anterior cortex was thin with fluid within the lesion. C: MRI was performed, and signal intensity was equal to that within the bladder, and a diagnosis of unicameral bone cyst was made. D: It was treated with curettage and bone graft.



**FIGURE 6-3.** A 12-year-old boy sustained a pathologic fracture while throwing a tennis ball. A: Radiographs at presentation revealed a lytic lesion of the proximal humerus with pathologic fracture seen distal to the lesion. There is also the suggestion of a pathologic fracture through the cyst. The lesion was believed to represent a unicameral bone cyst, and the fracture was treated in a hanging arm cast. B: At 8-weeks follow-up, fracture lines were beginning to obliterate but the cyst persisted. A nondisplaced healing fracture through the cyst is also well visualized. C: At 8-week follow-up, the patient was believed likely to be prone to have a recurrent fracture and was taken to surgery. With the patient under general anesthesia, the lesion was injected with radiopaque dye and was found to be a cystic lesion. Steroid injection was done. D: At 12-week follow-up, the patient is asymptomatic, with radiographic improvement of the lesion.



**FIGURE 6-4.** A 13-year-old boy sustained a pathologic fracture. A: X-ray studies at presentation revealed a lytic lesion of the proximal humerus with pathologic fracture. A so-called fallen fragment sign is seen at the bottom of a cyst suggesting a hollow cavity in the bone rather than a solid tumor. The lesion was believed to represent a UBC, and the fracture was treated in a hanging arm cast. B: At 8-week follow-up, fracture lines were beginning to obliterate. C: At 6 month follow-up, the cyst has partially healed.

### Natural History—Predicting Fracture

The natural history of UBCs is variable, but typically they gradually improve with growth. Most UBCs persist into adulthood, but some disappear spontaneously at puberty (24). The cysts are traditionally described as active if they are adjacent to the physis (24) or latent if more than 0.5 cm from the physis (35) (Table 6-5). Ahn and Park (4) noted that pathologic fracture occurred when the transverse diameter of the cyst was 85% or more, whereas Nakamura et al. (33) reported that the

chance of fracture was high if the cyst wall was less than 5 mm in width.

	Active	Inactive or "Latent"
Age of the patient	≤10–12 years	>12 years
Location	Abutting the physis	Separated from physis by a zone of normal cancellous bone
X-ray appearance	Single cavity	Multiloculated cavity
Intralesional pressure	>30 cm H <sub>2</sub> O	6–10 cm H <sub>2</sub> O
Pathology	Thin shiny membrane, few osteocytes, little or no hemosiderin, osteoclasts	Thick membrane, frequent giant cells, cholesterol slits, hemosiderin, osteoblasts

TABLE 6-5. STAGING OF UNICAMERAL BONE CYSTS

Complications after fracture of the proximal femur include malunion, growth arrest, and avascular necrosis of the femoral head. Kay and Nason (30) noted growth disturbance in five of their 21 patients (24%) with pathologic proximal humeral fracture before treatment of the UBC. Moed and LaMont (31) reported three patients in whom premature closure of the medial proximal humeral physis resulted in humeral shortening of 2 to 5 cm. Malunion may be a problem in fractures through cysts of the proximal femur (15), and avascular necrosis of the femoral head has been reported after displaced femoral neck fractures (26) and after an undisplaced femoral neck fracture in an 8-year-old boy (48). Infrequently, the cyst may extend from the metaphysis into the epiphysis (5,7,20) and collapse of the surface of the femoral head has been reported.

### Treatment

Pathologic fractures through UBCs invariably heal with simple immobilization; the cyst, however, persists in nearly 85% of patients, and additional fractures are common. Neer et al. (34) observed an additional 2.5 fractures per patient during observation periods after the initial injury, and Oppenheim and Galleno (36) emphasized that prolonged observation with inevitable refracture may be stressful to patients and may adversely restrict children from normal childhood activities.

In the upper extremity, minimally displaced, stable pathologic fractures through UBCs are treated with simple immobilization for 4 to 6 weeks to obtain healing. Usually, a sling is adequate treatment for stable fractures of the proximal humerus, and once healed, the options for further treatment are discussed with the patient and family (see later).

With pathologic fracture through UBC in the proximal femur, preliminary traction before surgical intervention may be appropriate (49). Most authors recommend internal fixation if the fracture is unstable or displaced (7,26,29,34). Malunion is common in these fractures (15). Intertrochanteric osteotomies may be necessary later to correct alignment (25).



## OPERATIVE TREATMENT

**Curettage and Bone Grafting.** Many surgical techniques have been developed for the treatment of UBCs, with varying rates of success. Earlier interventions consisted primarily of curettage and bone grafting. Lesions treated with this technique have a recurrence rate varying from 18% to 40% (6,7,19,34,35,47). Delaying the treatment until the cyst converts from an active to a latent stage has been advocated by some physicians, but a 2-year period of observation may be necessary (7). Some data suggest that the response to treatment may be the same for latent as for active cysts (23). After fracture, most authors advocate at least a 6-week delay in treatment to allow the fracture to heal before curettage and bone grafting (15,39). There seems to be no substantial difference in the rate of healing with the use of either autogenous bone graft or allograft.

Open curettage and bone grafting has fallen out of favor for the treatment of upper extremity and smaller lower extremity UBCs due to the invasive nature of the operation and also the still significant persistence and recurrence rates associated with this option. Curettage and bone graft remains a popular method of treatment for larger UBCs of the proximal femur, however; the reported recurrence rates range from 10% to 40% (6,7,19,36). Although partial persistence or recurrence of the UBC may occur, interim healing usually occurs such that the risk of pathologic fracture and displacement is lessened and subsequent persistence or recurrence can be treated with corticosteroid injections (Fig. 6-5). Follow-up of the treated lesions with periodic x-ray studies is suggested.



FIGURE 6-5. A 7-year-old boy was playing basketball and felt a severe pain in his left hip and was unable to bear weight on that side. **A:** X-ray studies revealed a comminuted pathologic fracture through a large lytic lesion extending from the base of the femoral neck to the subtrochanteric region (comminuted type I—B hip). The appearance of the lesion is believed to be most compatible with a UBC. **B:** The fracture and cyst were approached laterally. Open reduction and internal fixation were performed using a pediatric hip screw and side plate. The cyst contents were consistent with UBC; it was curetted and packed with cancellous bone graft. The patient was placed in a hip spica cast for 6 weeks with healing of the fracture. **C:** At 4-month follow-up, the fracture has healed and the cyst appears to be consolidating. **D:** At 2-year follow-up, x-ray studies show a partial recurrence or persistence of the cyst. The cyst was subsequently grafted again. **E:** At 4-year follow-up, x-ray studies show no evidence of recurrence or persistence of the cyst. The patient has no discomfort and has returned to full activities.

The patient with a calcaneal UBC usually presents with pain due to microfracture (22,32,37). UBCs in this location may not respond as well to injections with methylprednisolone (22,32), and several authors have recommended primary curettage and bone grafting (22,32).

Because of the high rates of recurrence after curettage and bone grafting, a number of approaches were developed, including intentional fracture of the cyst (46), crushing of the lesion with onlay bone grafting (5), and packing of the defect with plaster of Paris pellets (37). Radical excision of the lesion with or without grafting produced recurrence rates of 9% or less (19,21,30), but this procedure is a larger operation with greater risk, can be difficult technically, and requires substantial bone graft.

**Aspiration and Injection.** In 1974, Scaglietti et al. began empiric injection of UBCs with methylprednisolone and in 1979 (43), reported healing rates (mostly radiographic improvement of the cyst) of 96% in 72 patients. Two needles were inserted into each cyst, and the initial corticosteroid dose varied from 40 to 200 mg for large cysts. Complete healing was initially reported in 55% of cysts undergoing this treatment. Local recurrence in 45% of cases was treated with up to five

additional injections of 40 to 80 mg of methylprednisolone at 2- to 3-month intervals until healing occurred.

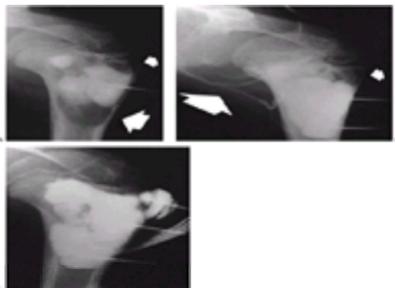
The mechanism for cyst healing after methylprednisolone injection remains unknown. Scaglietti et al. ( 43) suggested that an antiinflammatory response initiated by the corticosteroids promoted cyst healing. Shindell et al. ( 45) observed elevated levels of prostaglandins in cysts; after serial injections of corticosteroid, levels decreased. Because prostaglandins stimulate osteoclastic activity, these authors suggested that a corticosteroid-induced repression of prostaglandins may indirectly aid in cyst healing. In animal experiments, however, corticosteroids are rapidly cleared from the medullary canal of long bones after injection ( 16). Others have suggested that cyst healing occurs through decompression of cyst fluid pressure by multiple trephination ( 13,16,42).

Methylprednisolone injection for UBCs became popular because of its relatively low morbidity and an apparent long-term effectiveness approaching that of more invasive techniques. However, incomplete healing and recurrence are common after the initial injection of corticosteroid, and multiple injections of corticosteroid may be necessary in 50% to 92% of patients (intervals ranging from 2 to 6 months) ( 11,36).

In those cysts that respond to methylprednisolone treatment, the cortical margins of the lesion usually thicken. By 6 months, the central portion of the cyst assumes a frosted-glass appearance, and by 12 months, the cyst may heal with dense sclerotic bone ( 12,40).

*Aspiration and Injection—Operative Technique.* A two-needle injection technique is most commonly used. Some authors (36) recommend the use of two Craig-type needles or Jamshidi biopsy needles so that a biopsy specimen can be obtained at the time of injection. The initial dose of methylprednisolone varies from 40 to 200 mgs (11,18,43), and the volume can be adjusted to match radiographic volume of the cyst. The injection can be given under local anesthetic ( 10), but most prefer general anesthesia. Several authors (11,36,37) have emphasized the importance of outlining the cyst with radiopaque dye injection before placement of the methylprednisolone. With this technique, intracystic fibrous septa were found in 92% of lesions in one series ( 11) and these may prevent complete filling of the cyst by corticosteroids with later incomplete healing. This cystogram also allows the surgeon to verify that the cyst is indeed fluid filled. If it does not fill with contrast material, other diagnoses, such as fibrous dysplasia or enchondroma, should be considered. Capanna et al. ( 11) recommend vigorous saline irrigation of the cyst through two needles to lyse the fibrous septa, whereas Oppenheim and Galleno ( 36) simply reinject the areas of cyst that are not filled by contrast material in the initial injection.

*Aspiration and Injection—The Humerus.* With fluoroscopic guidance, the humerus is rotated until an area of thin cortex is identified, and two 20-gauge spinal needles are passed through this area into the cyst so that the needle tips are at opposite ends of the cyst cavity. A more stout, disposable biopsy needle can be used for areas of thicker bone. The presence of serous fluid with removal of the needle stylet is indicative of UBC. A cystogram is then performed by injection of several milliliters of Renografin dye both to confirm the fluid-filled nature of the cyst and to ascertain whether the cyst is indeed unicameral or multiloculated. If the dye does not fill a cyst cavity, the diagnosis of UBC should be questioned. If the cyst is multiloculated, the needles can be used to break up any septations that exist. Methylprednisolone is then injected through one of the needles using a dose ranging from 40 to 200 mgs ( Fig. 6-6). To guard against recurrent fracture, the arm is protected in a sling for 2 to 6 weeks and x-ray studies are performed every 6 weeks initially to monitor healing. Incomplete healing or persistence of the cyst can be treated with additional injections or with other techniques. Aspiration, cystogram, and steroid injection can also be done for small, stable UBCs of the lower extremity, but if the cyst is potentially unstable and there is a risk of malunion (e.g., varus of the femoral neck), one should consider surgical fixation and bone grafting.



**FIGURE 6-6. A:** A radiopaque dye injection of a unicameral cyst of the proximal humerus. Note the poor filling of the inferior portion of the cyst ( *large arrow*) and the proximal portion ( *small arrow*). This suggests that fibrous septa are present in the cyst. **B:** More radiopaque dye was placed in the distal portion of the cyst through a second needle, and a venous drainage of the lesion is now visualized ( *large arrow*). The proximal portion of the cyst is still poorly filled ( *swag arrow*). **C:** With a third needle superiorly, the proximal portion of the cyst is now filled. The total dose of corticosteroid is divided into equal portions and injected separately through the three needles to ensure complete dispersal throughout the lesion. A single dose through the first needle might not have saturated the entire cyst.

## Complications

In addition to recurrence of the cyst, complications that occur with corticosteroid treatment include recurrent pathologic fractures ( 12,20,36) and avascular necrosis of the femoral head (8,12). Nakamura et al. (33) measured the bone mineral content of the corticosteroid injected cyst by densitometer and found that if there was no increase in density 2 months after injection, then the chance of refracture was high. In one series, growth disturbance was a problem in 20% of 141 patients with bone cysts treated by corticosteroid injection (8). Systemic reactions to the corticosteroid injection, such as corticosteroid flush or increased appetite and temporary weight gain, are rare (16,36).

## Newer Methods

Some authors believe that relieving the pressure of the interstitial fluid in the lesions can heal the cyst. Chigira et al. ( 13) treated six patients by puncturing the cysts with multiple Kirschner wires, which were then left in place; cysts subsequently recurred in 6 to 8 months in two patients. Santori et al. ( 42) decompressed UBCs with Enders nails or Rush pins without curettage or grafting, and during short-term follow-up, they noted healing in all 11 patients. The effect of the fixation on the adjacent physes awaits long-term follow-up.

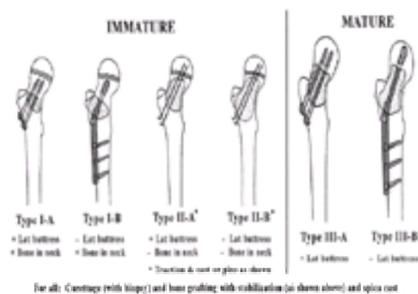
New grafting materials also are becoming available. Demineralized bone matrix (DBM) or commercial product paste is composed of demineralized bone particles ranging from 100 to 500  $\mu\text{m}$  in a glycerol base. It has been used in the treatment of delayed unions and nonunion, in some primary bone-healing situations, and more recently, for the treatment of UBCs (27,28,41). Killian et al. (27) used DBM in 11 patients with UBCs, and nine cysts healed (within 4 to 5 months) after a single injection. At 2 years' follow-up, no cysts were deemed active or recurrent (26).

Other materials also have been used. Packing of the defect with plaster of Paris pellets (calcium sulfate) was described by Peltier in 1978 ( 37). Osteoset pellets bone void filler is a new material made of medical grade calcium sulfate. This radiopaque product can be used in an open grafting situation or can be injected percutaneously. The biodegradable pellets are resorbed in 30 to 60 days when used according to labeling. This product is not intended to provide structural support during the healing process and therefore is contraindicated when structural support is required.

Adjuvants such as liquid nitrogen have been used for the treatment of UBCs ( 46), but their efficacy or safety has not been established. Current research includes the use of aspirated bone marrow injected into UBCs.

## Internal Fixation of Proximal Femoral Pathologic Fractures

If there is a significant loss of proximal femoral bone because of the UBC, there is a high risk of a coxa vara deformity after treatment without internal fixation. Both the location of the UBC and the amount of bone loss dictate whether fixation can stabilize the fracture after grafting and what type of fixation is appropriate. We have classified pathologic fractures of the femoral neck in children into six types ( Fig. 6-7) (9).



**FIGURE 6-7.** Our classification system for the treatment of pathologic fractures of the proximal femur associated with bone cysts in children. **A:** In Type IA, a moderately-sized cyst is present in the middle of the femoral neck. There is enough bone in the femoral neck and lateral proximal femur (lateral buttress) to allow fixation with cannulated screws after curettage and bone grafting. **B:** In Type IB, a large cyst is present at the base of the femoral neck. Although there is enough bone in the femoral neck, there is loss of lateral buttress, so a pediatric hip screw and a side plate should be considered rather than cannulated screws, after curettage and bone grafting. **C,D:** In Type IIA-B, a large lesion is present in the femoral neck, so there is not enough bone beneath the physis to accept screws. There are 2 options for treatment of these bone cysts: (1) After curettage and bone grafting, parallel pins across the physis can be used in combination with a spica cast. (2) The patient can be treated in traction until the fracture heals (with subsequent spica cast) followed by curettage and bone grafting. **E,F:** In Type IIIA-B, the physis is closing or closed. The lateral buttress is present in Type IIIA hips, so cannulated screws can be used to stabilize the fracture after curettage and bone grafting. In Type IIIB hips, the loss of lateral buttress makes it necessary to use a pediatric hip screw and a side plate following curettage and bone grafting. In all types, we recommend spica cast immobilization following surgery.

## AUTHORS' PREFERRED METHOD OF TREATMENT

Overall, methylprednisolone injection has a favorable rate of success compared with open surgical treatment; the ease of treatment, relatively low operative morbidity, and the information obtained with aspiration and cystogram make it a favorable initial choice for treatment of UBCs in the upper extremity and smaller cysts in the lower extremity. Because incomplete healing and recurrence are common after the initial injection of corticosteroid and because multiple injections of corticosteroid are necessary in most patients, new grafting materials are being used more commonly at our institution, especially if the first steroid injection fails. Grafton demineralized bone matrix (Osteotech, Eatontown, NJ) and Osteoset pellets (Wright Medical Technology, Arlington, TN) are the two most often used at present.

We recommend simple immobilization for most fractures occurring through UBCs. Spontaneous healing of the cyst can occur, although infrequently. Once the fracture has healed, generally by 6 to 8 weeks, further treatment of the cyst can be rendered, if necessary.

Displaced pathologic fractures of the proximal femur are based on the location of the cyst. The amount of bone loss dictates whether fixation can stabilize the fracture after grafting and what type of fixation would be best to use. We use the classification of pathologic fractures of the proximal femur in children shown in [figure 6-7](#).

### Aneurysmal Bone Cysts

ABCs are eccentric or central, expansile osteolytic lesions usually occurring in the metaphyseal ends of long bones or in the posterior elements of the spine during adolescence. Nearly 75% of ABCs are found in patients younger than 20 years old, and 50% are seen in individuals between 10 and 20 years of age ([50,54](#)). Girls are affected slightly more often than boys. ABCs are relatively rare, accounting for approximately 1.5% of all primary bone tumors ([50](#)).

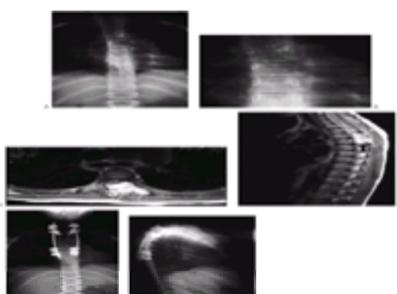
The long bones are involved in 65% of patients. In order of decreasing frequency, the most commonly involved bones are the distal femur, proximal tibia, proximal humerus, and distal radius. The vertebrae are involved in 12% to 27% of patients ([54,59](#)), some of whom have symptoms of radicular pain. The lumbar vertebrae are most commonly affected. The primary site of involvement is the posterior elements of the spine with frequent extension into the vertebral body ([58](#)).

The lesions are not true cysts but rather sponge-like collections of interconnected fibrous tissue and blood-filled spaces ([59](#)). They tend to be destructive lesions, which replace bone and thin the cortices of the host bone. The elevated viable periosteum usually maintains a thin osseous shell.

The etiology of ABCs is unknown. Some have considered them primary lesions of bone ([79](#)), whereas others have noted a secondary association with other lesions such as UBCs, nonossifying fibromas, fibrous dysplasia ([52](#)), and osteogenic sarcoma. They also can occur in association with fractures of the long bones ([58,63](#)). The most common presenting symptom is localized pain of less than 6 months' duration ([54,79](#)). Patients with ABCs are three times more likely to have pain during exercise rather than pain at rest ([71](#)).

### Radiographic Findings

ABCs are eccentric or central lytic lesions of bone, sometimes with extension beyond the cortex ([50,52,79](#)). Septation is common, giving rise to the so-called soap bubble or honeycomb appearance. Lesions in the short tubular bones, such as the metacarpals and metatarsals, are commonly more central. Spinal ABCs usually are located in the posterior elements of the spine but may also occur in the vertebral bodies and can be associated with pathologic fracture and vertebral collapse ([Fig. 6-8](#)) ([66,79](#)).

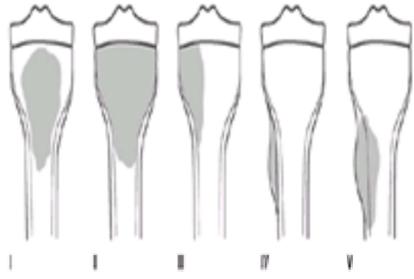


**FIGURE 6-8.** An 8-year-old girl presented with a 4-week history of back pain. **A:** Radiographs show absence of the pedicle of T5 on the left. **B:** Close-up view of the same areas. **C:** The axial T2-weighted image through the body of T5 shows multiple blood-fluid levels within the left sided expansile destructive mass. **D:** Sagittal proton density MRI of the thoracic spine shows a blood fluid level (arrow) within an expansile mass. **E:** Postoperative x-ray studies showing instrumentation and fusion after extended curettage and removal of tumor. The patient is pain free without recurrence or deformity at 4 years after surgery. (From Cohen RB, Dormans JP, Guttenberg ME, Hunter JV. Back pain in an 8-year-old girl. *Clin Orthop* 1997;343:249–252; with permission.)

The x-ray picture often evolves with time. Initially, there is frank osteolysis of the margins of the bone, and periosteal elevation; with growth of the lesion, there is progressive destruction of bone with poorly demarcated margins. A stabilization phase follows, with formation of a bone shell with septa. Later, with further ossification, a bony mass begins to form ([59](#)).

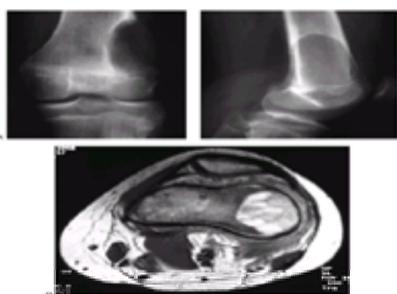
Campanacci et al. ([54](#)) have classified the ABCs into three groups ([Fig. 6-9](#)). An aggressive cyst has signs of reparative osteogenesis with ill-defined margins and no

periosteal shell. An active cyst has an incomplete periosteal shell and a defined margin between the lesion and the host bone. An inactive cyst has a complete periosteal shell and a sclerotic margin between the cyst and the long bone.



**FIGURE 6-9.** Classification of morphologic types of ABC. (From Capanna R, Bettelli G, Biagini R, et al. Aneurysmal cysts of long bones. *Ital J Orthop Traumatol*, 1985;XI:421–429; with permission.)

Angiography may aid diagnosis and treatment; abnormal peripheral vascularization is often present (71). Percutaneous dye injection has been used as an additional diagnostic technique and in evaluation of vertebral lesions (72). Technetium bone scan usually shows an increased uptake of the isotope in the periphery of the lesion, but homogeneous uptake is also seen (68). Magnetic resonance imaging (MRI) often is helpful in demonstrating the characteristic septations and fluid levels, but these findings are not pathognomonic for ABC (Fig. 6-8 and Fig. 6-10) (78).



**FIGURE 6-10.** A 14-year-old-girl presented with distal thigh pain from microfractures through the thinned wall of an aneurysmal bone cyst of the distal femur. **A:** X-ray studies show an eccentric, expansile, lytic lesion of the lateral aspect of the distal femoral metaphysis. There is a narrow zone of transition with a sclerotic border. **B:** MRI shows septation of the lesion with the fluid-fluid levels, which are characteristic of ABC. The patient was treated with extended curettage and bone grafting.

### Natural History

ABCs are benign but usually behave in a locally aggressive manner. Pathologic fractures occur in 11% to 35% of patients with ABCs of the long bones (67,71). The humerus and femur are the most commonly fractured long bones (57,71). Other sites of fracture occur, but these are rare. In one series, vertebral body ABC was associated with fracture in 27% of patients (79).

Although rare, epiphyseal involvement by the lesion through metaphyseal extension has been reported (51,60,69). Capanna et al. (56) reported nine patients with invasion of the physis by large metaphyseal ABCs. In five of these patients, growth disturbance of the involved physis subsequently developed.

Conservative treatment with immobilization is inappropriate as a definitive treatment for pathologic fractures of ABCs. Although the pathologic fracture will heal, the ABC will persist and enlarge and a recurrent pathologic fracture will occur.

### Treatment

Although ABC healing after simple biopsy has been reported (54), this does not occur often and observation is not recommended because these lesions usually are locally aggressive. Simple curettage and bone grafting have been associated with high recurrence rates (51,54,59,66,79), ranging from 20% to 30% (54,59). There appears to be a higher rate of recurrence in patients younger than 15 years of age (79). Freiberg et al. (62) treated ABCs with curettage and bone grafting in seven patients younger than 10 years of age and noted recurrence in five of the seven patients at an average of 8 months after the first procedure.

Selective arterial embolization is used most commonly in locations where a tourniquet cannot be used and control of bleeding can be difficult (e.g., spine, pelvis, and the proximal portions of the extremities). Green et al. (64) reported on eight patients treated with selective arterial embolization. In seven patients, embolization was performed in conjunction with open bone grafting and, in one patient, as definitive treatment. At a follow-up of 3 years, there were no recurrences and no complications related to embolization.

Treatment of ABCs by cryotherapy in conjunction with curettage has a recurrence rate of between 8% and 14% (51,73,77). Dabezies et al. (58) obtained healing of ABCs associated with fractures by collapsing the cyst manually after curettage. Polymethylmethacrylate cementation also has been described as an adjuvant to curettage for the treatment of ABCs. Ozaki et al. (74) compared curettage and bone grafting in 30 patients with curettage and cementation in 35 patients. At follow-up ranging from 24 to 161 months, the recurrence rate was 37% for curettage and bone grafting compared with 17% for curettage and cementation.

Injection has been used by some physicians (61). Guibaud et al. (65) reported on the use of percutaneous embolization with an alcoholic solution of Zein (Ethibloc; Ethnor Laboratories/Ethicon, Norderstedt, Germany) in 18 patients. In two patients, the cystogram showed marked venous drainage and embolization was not attempted. Six patients underwent repeat embolization. At follow-up ranging from 18 months to 4 years, there were no recurrences.

Complete *en bloc* resection is reserved for active or recurrent ABCs (50,56,66,71) and is most feasible in the proximal fibula, distal ulna, ribs, pubic rami, metatarsals (54), and metacarpals (53). Resection of metacarpal lesions with replacement by a fibular autograft often results in soft tissue scarring with reduced joint motion (53). Campanacci et al. (54) recommended saucerization of peripheral active and aggressive cysts.

Irradiation should be avoided. Its use has been associated with the development of sarcoma (79). It has been used for lesions that are surgically inaccessible (57), but it is contraindicated in the pelvis, where the reproductive organs may be affected, and in areas of active growth of the long bones (54).

ABCs of the spine can be difficult to treat because of the relative inaccessibility of the lesion, proximity of the lesion to the spinal cord and nerve roots, and the potential for spinal instability (75,81). Papegelopoulos et al. (75) reported on 52 consecutive patients with spinal ABCs treated over an 83-year period and recommended preoperative selective arterial embolization, intralesional excisional curettage, bone grafting, and fusion of the affected area if instability is present (Fig. 6-8). Turker et al. (81) described three patients with ABCs of the spine and emphasized the need for spinal stabilization and fusion in conjunction with removal of the

lesion.

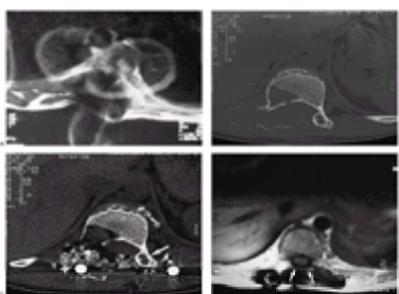
## AUTHORS' PREFERRED METHOD OF TREATMENT

The first step in effective treatment of a patient with an ABC is to confirm the diagnosis with open biopsy and frozen section; this biopsy usually is done at the same surgical setting as the definitive surgical procedure. It is important to remember that ABC can be secondary to other tumors such as nonossifying fibroma, giant cell tumor, and chondroblastoma; telangiectatic osteosarcoma may be difficult to distinguish from ABC with an inadequate biopsy specimen because the aplastic tumor cells are seen only at the periphery of the lesion ([70,76](#)).

Once the diagnosis is made, treatment should be initiated as soon as possible because most ABCs are aggressive and often grow and invade rapidly. Preoperative planning is important to ensure adequate exposure, preparation for blood loss, internal fixation, grafting material, and in selected cases, preoperative embolization.

Achieving adequate exposure with a large cortical window for thorough extended curettage is a key component for successful treatment. The use of a high-speed burr allows systematic intralesional excisional curettage. Adjuvants phenol and alcohol, liquid nitrogen, and polymethyl methacrylate (PMMA) usually are reserved for large or recurrent ABCs but may be considered in the initial management. Bone grafting is done for all lesions and can consist of autograft, allograft, bone substitutes, or a combination of these methods, depending on the circumstances. Most patients with fractures through ABCs have microfractures that do not alter treatment. For those with more significant or unstable fractures, internal stabilization is used when appropriate, particularly in the hip, femur, or tibia. The classification and recommendations in [figure 6-7](#) are applicable for those with proximal femoral fractures associated with ABCs. A walking hip spica cast (i.e., a unilateral hip spica cast with the hip and knee in 20 to 30 degrees of flexion) is sometimes appropriate for young children with stable fractures. Close follow-up is recommended initially because recurrence can be rapid and aggressive.

For ABC of the spine, we recommend preoperative selective arterial embolization, intralesional extended excisional curettage and bone grafting. Instrumentation and fusion of the affected area should be performed if instability or the potential for instability exists. If instrumentation is used, titanium instrumentation allows follow-up MRI with less metal artifact compared with stainless steel implants ([Fig. 6-11](#)) ([80](#)).



**FIGURE 6-11.** When dealing with pathologic fractures secondary to tumors or tumor-like processes of the spine, if instrumentation is needed, titanium instrumentation allows much better postoperative visualization with both CT and MRI for the detection of tumor recurrence as compared with standard stainless steel instrumentation. **A:** Postoperative MRI of the spine with standard stainless steel instrumentation showing a large degree of artifact that makes interpretation difficult. **B:** Preoperative CT scan of a patient with an ABC of the spine. **C:** Postoperative CT scan of the same patient showing an adequate view of the surgical area. **D:** Postoperative MRI of a patient with a previous spinal tumor again adequately showing the surgical site to monitor for recurrence or persistent tumor.

## Tumors of Bone

### *Fibrous Cortical Defects and Nonossifying Fibromas*

Fibrous cortical defects (FCDs) and a larger variant known as nonossifying fibroma (NOFs) may be associated with pathologic fractures in children. Both lesions contain fibrous tissue, foam cells, and multinucleated giant cells ([84](#)). Most pathologic fractures occur in boys ([83](#)), and age at presentation varies from 6 to 14 years ([91](#)).

FCDs are small metaphyseal lesions ranging from 1 to 2 cm in diameter and most commonly occur in the distal femur, proximal tibia, and fibula. They are eccentric and usually are surrounded by the thinned cortex, with the medullary wall of the lesion tending to be sclerotic. FCDs are common and can be seen on x-ray studies of the lower extremity in approximately 25% of pediatric patients ([93](#)). In view of their usually asymptomatic nature, it is difficult to estimate the true incidence. They usually require no treatment other than observation.

### *Radiographic Findings*

NOFs also are eccentric lesions of the metaphysis, but they may achieve a length of 5 cm ([90](#)) or more and can extend across a substantial portion of the width of the long bone. They present at a similar age as FCDs, and follow a similar distribution of bone involvement, and multiple lesions are present in approximately one third of patients ([83,87](#)). On x-ray study, the lesions are usually eccentric, radiolucent cystlike areas that can be either uniloculated or multiloculated; in small bones such as the fibula, they may occupy the entire width of the shaft ([84](#)). Sclerotic scalloping is often present along the endosteal margin ([89](#)). Usually, NOFs are asymptomatic unless a pathologic fracture is present ([84](#)). They become clinically significant when they present with or predispose to pathologic fracture.

### *Natural History*

Arata et al. ([82](#)) found that 43% of pathologic fractures through NOFs were in the distal tibia. Several previous reports suggested that these lesions regress spontaneously ([83,84,85,87,90,92](#)) Ritschl et al. ([92](#)) described the radiomorph course of NOFs, demonstrating that the defects become sclerotic and resolve. Typically, this tumor remains asymptomatic and is commonly an incidental radiographic finding. However, lesions with extensive cortical involvement can cause pathologic fractures.

Previous reports suggest that the absolute size of the lesion correlates directly with the risk of pathologic fracture ([82](#)). Based on this factor, prophylactic curettage and bone grafting of larger NOFs have been recommended. Arata et al. ([82](#)) noted that all pathologic fractures associated with NOFs in the lower extremity occurred through lesions involving *more than 50% of the transverse cortical diameter*. These large lesions were defined as exhibiting more than 50% cortical involvement on anteroposterior (AP) and lateral x-ray studies and a height measurement of more than 33 mm ([82](#)). Although the authors recommended careful observation of these large NOFs, they suggested that “prophylactic curettage and bone grafting be considered if there is a reasonable chance of fracture.” Their series does not include any large lesions meeting their size criteria that did not fracture, and their hypothesis has never been tested in any published series. Drennan et al. ([87](#)) suggested that large NOFs causing pain may predispose to fracture and recommended prophylactic curettage and bone grafting for selected larger lesions.

Fractures through NOFs exhibit excellent healing potential ([82,84,87](#)), but the lesion usually persists after healing of the fracture ([Fig. 6-12](#) and [Fig. 6-13](#)). Recurrent fractures have been reported, but the incidence of documented refracture is low ([82,87](#)). Fracture union takes place normally, but often multiple radiolucencies remain ([84](#)). Overall, fracture healing does not usually obliterate the lesions so recurrent fracture can occur ([82,89](#)).



**FIGURE 6-12. A:** An 8-year-old boy was referred after being casted at another institution for a pathologic fracture of the right femur. The x-ray studies in the cast show a pathologic fracture of the right distal femur through an NOF. **B:** At 14 weeks after the injury, the fracture has united with some posterior displacement of the distal fragment. **C:** At 7 months after fracture, there has been good remodeling at the fracture site with persistence of the NOF. The patient returned to full activities and has had no further problems at 7-year follow-up.



**FIGURE 6-13. A:** A 13-year-old athlete presented with a pathologic fracture through the distal tibia after a fall on the ice. **A:** The patient was treated with a long-leg cast immobilization. After the fracture had healed, the patient underwent open curettage and bone grafting of the lesion. **B:** Follow-up x-ray studies show healing of both the fracture and the lesion, and the patient has returned to full activities.

Large lesions, defined as having a diameter more than 50% of the width of a long bone on both AP and lateral x-rays, are believed to be prone to pathologic fracture; most authors have recommended curettage and bone grafting for these large lesions ([83,84,86,87,89](#) and [91](#)).

Easley and Kneisel et al. ([88](#)) reported that although absolute size parameters were helpful in predicting pathologic fracture, they did not imply a requirement for prophylactic curettage and bone grafting. In their series, 13 (59%) large NOFs had not had pathologic fracture despite exceeding the previously established size threshold. In the nine (41%) patients in whom pathologic fracture occurred, healing was uneventful after closed reduction and cast immobilization, and no refractures occurred. They suggested that most patients with large NOFs can be monitored without intervention, because previous studies support spontaneous resolution of most of these lesions ([82,84,87](#)). All fractured NOFs in their series healed with closed reduction and immobilization. It may be reasonable to restrict the activity of patients with large NOFs based on the nine patients in their study with pathologic fractures caused by trauma.

Fractures usually are treated with immobilization until healing is obtained ([83,86](#)). Surgery is necessary only if the residual lesion is of significant size to predispose the patient to further pathologic fractures or there is doubt about the identity of the lesion ([84](#)). Displaced pathologic supracondylar fractures of the distal femur may require open reduction, bone grafting, and intramedullary fixation ([87](#)). Subperiosteal resection of a pathologic humeral shaft fracture followed by bone grafting has resulted in pseudoarthrosis ([89](#)).

## AUTHORS' PREFERRED METHOD OF TREATMENT

Treatment is based on the size and location of the lesion and the type of pathologic fracture. Small lesions without fracture can be observed and may require 1 to 3 years to spontaneously resolve ([83](#)). Substantial lesions of the lower extremity in active children, even if they are asymptomatic, should either be followed carefully with serial x-ray studies or should undergo curettage and bone grafting to avoid pathologic fracture. Although absolute size parameters may be useful in predicting pathologic fracture, they do not imply a requirement for prophylactic curettage and bone grafting. Most patients with large NOFs can be monitored without surgical intervention, and fractures can be successfully managed with nonoperative treatment. Our experience is that a considerable number of incidentally discovered large NOFs do not fracture. Although we cannot readily identify an accurate denominator, we infer that many large NOFs remain unidentified and nonproblematic. Patient and family wishes and the individual's activity demands also obviously influence the decision. Given the historic evidence for spontaneous resolution and favorable healing characteristics of NOFs, patients with lesions larger than 50% of the width of the bone should be approached individually.

### **Giant Cell Tumors of Bone**

Giant cell tumors of bone are rare in the pediatric population; most occur in skeletally mature individuals. In a series of 221 patients with giant cell tumors of bone ([96](#)), only 20% of patients were younger than 20 years of age. In order of decreasing frequency, these tumors most commonly occur in the distal femur, proximal tibia, proximal humerus, and distal radius. The incidence of pathologic fractures with these lesions is approximately 16% ([94,95,96,97](#) and [98](#)).

Radiographically, they are osteolytic, metaphyseal lesions that can extend into the epiphysis, usually after physeal closure ([Fig. 6-14](#)). They can be eccentric, but larger lesions can involve the full width of the bone. Little or no sclerosis usually is present around the margin of the tumor. Although heavy trabeculation may be present, new periosteal bone formation is uncommon.



**FIGURE 6-14. A:** X-ray studies showed an eccentric, lytic, destructive lesion involving the distal portion of the medial metaphysis with extension into the epiphysis. **B:** CT scan showed the destructive lesion with thinning of the cortex and no external soft tissue mass. Open biopsy and frozen sections were consistent with giant cell tumor of the distal femur. An extended curettage was then performed with phenol as an adjuvant and subsequent cementation using methylmethacrylate bone cement. **C:** Follow-up x-ray studies at 2 years after surgery showing no evidence of recurrence. The patient

is asymptomatic and has full range of motion and function of the knee.

Giant cell tumors usually are treated by extended curettage in combination with adjuvants, such as phenol or liquid nitrogen, and filling with material such as PMMA (Fig. 6-14) (94,95 and 96). *En bloc* or wide resection is a more aggressive option. Simple curettage and bone grafting are associated with a high recurrence rate. In one series (96), simple curettage and bone grafting had a 34% recurrence rate, whereas wide resection of the lesion resulted in only a 7% recurrence rate. Wide resection is most appropriate for giant cell tumors involving expendable bones and for aggressive lesions with significant involvement of the articular surface. The location and extent of the lesion and the proximity of the tumor to articular cartilage and physis influence the treatment of giant cell tumors in children.

**Pathologic Fractures Associated With Giant Cell Tumors of Bone**

Pathologic fractures are associated with giant cell tumors in 6% to 30% of patients. The complexity of treatment is markedly increased if a pathologic fracture is present. Management depends on the type of fracture and fracture displacement ( Table 6-6).

Scenario	Treatment
1. Fracture nondisplaced, structurally insignificant, nonarticular	No change in treatment plan (usually extended curettage and cementation with adjuvant such as phenol)
2. Fracture simple, but displaced	Joint preserving options:
a. Fracture can be reduced closed	a) closed reduction and delayed extended curettage*
b. Fracture cannot be reduced closed	b) Open reduction, extended curettage, simultaneous internal fixation and cementation*
3. Fracture displaced, articular, open reduction, delayed curettage and cementation cannot be achieved satisfactorily	Resection or partial resection of the joint

\* If a fracture can be reduced and held by closed methods, it may be preferable to delay the definitive surgery until a fracture healing has occurred. Fracture healing is biologically faster than the growth of the tumor, a delay of 2 to 6 weeks will have a minimal effect on tumor progression.

\* A temporary antibiotic and phenol solution (see Fig. 6-14) can be applied to the site of fracture after reduction and fixation of the fracture. The use of antibiotic and phenol solution after fixation of the fracture of bone and cartilage around the internal fixation system.

**TABLE 6-6. TREATMENT OF GIANT CELL TUMORS**

A biopsy may be needed before fracture treatment if the diagnosis is not certain. Most pathologic fractures are undisplaced, structurally insignificant, or nonarticular, and require no change in the treatment plan. For more significant fractures, an attempt at preserving the joint should be made. Overall, the presence of a pathologic fracture itself does not seem to directly influence the recurrence rate of giant cell tumor; it may influence the reconstruction options and the overall functional result.

**Enchondroma**

Solitary enchondroma is a rare lesion in children. In one series of enchondromas ( 101), 57% of patients were between 11 and 30 years of age. The common presenting symptom is pain, usually associated with a pathologic fracture. Most common sites of involvement in decreasing order of frequency are the phalanges, metacarpals, metatarsals, humerus, and femur. Pathologic fracture is commonly the presenting symptom for enchondromas located in the phalanges of the hands or feet, but is rare for enchondromas in other locations.

On x-ray studies, the lesions can be central or eccentric and a stippled calcification of the cartilage tumor matrix may be seen. In the long bones, lesions tend to be central with only slight bulging of the cortex. The short tubular bones with enchondromas show a cloudy radiolucency with bulging and thinning of the cortex ( Fig. 6-15) (101).



**FIGURE 6-15.** An 8-year-old boy presents with pain and swelling of the ulnar border of his right hand. **A:** X-ray studies showed an expansile, lucent lesion of the diaphysis of the patient's right fifth metacarpal with microfractures. The patient had an open incisional biopsy with frozen section, which was consistent with enchondroma with subsequent curettage and bone grafting. **B:** Gross appearance of material removed at the time of surgery, which is consistent with enchondroma. **C:** At 6-month follow-up, the fracture is well healed, and there is no sign of recurrent tumor.

Children can also have multiple enchondromas or enchondromatosis (Ollier's disease), which is commonly seen between 2 and 10 years of age ( Fig. 6-16). X-ray studies show lesions similar to solitary enchondroma, but usually with deformity and shortening of the extremity due to growth disturbance ( Fig. 6-16E). A unique x-ray finding that is believed to be pathognomonic for enchondromatosis is the presence of linear radiolucencies extending from the metaphysis down the shaft of the long bone.



**FIGURE 6-16.** Multiple enchondromatosis. **A:** A 10-year-old girl with multiple enchondromas sustained a spontaneous pathologic fracture of the femur while running. Nine months before the injury, she had sustained a fracture of the same femur, which had been treated with a one-and-a-half hip spica cast for 3 months. **B:** The lateral x-ray study shows overriding of the fracture. **C:** The fracture was treated with 3 weeks of skeletal traction, and then the extremity was placed in a cast brace for 9 weeks. On x-ray, the excessive anterior bow of the femur has been somewhat corrected by deliberate posterior angulation of the fracture. **D:** At 3-year follow-up, the fracture is well healed. **E:** The anteroposterior x-ray study of the hand in this patient demonstrated multiple expansile enchondromas of the small bones. **F:** An x-ray

study of the humerus shows the streaked-mud appearance of the lateral humerus (*arrow*).

When these lesions are associated with multiple hemangiomas, the general condition is known as Maffucci's syndrome ([100,102](#)). In this syndrome, 30% of patients have one or more pathologic fractures ([102](#)). Approximately half of these fractures go on to delayed union or nonunion. Unilateral skeletal involvement occurred in 48% of patients, with involvement in the order of frequency of the hand, foot, tibia, femur radius and ulna, humerus, and ribs. Skeletal deformities tend to stabilize at maturity. Sarcomatous degeneration has been reported in approximately 15% of patients.

Asymptomatic lesions can be observed. Biopsy may be necessary when the identity of the lesion is uncertain. Symptomatic lesions respond well to curettage and bone grafting ([99,100](#)). Treatment is individualized for displaced fractures ([Fig. 6-15](#)).

## AUTHORS' PREFERRED METHOD OF TREATMENT

Solitary enchondromas often require biopsy to establish the diagnosis. For asymptomatic patients with small lesions with classic x-ray findings, biopsy usually is not necessary. Curettage and bone grafting is necessary for those lesions with acute or impending pathologic fracture. Fixation is not necessary for those lesions of the short tubular bones but may be necessary for lesions of the proximal femur or long bone of the lower extremity. Standard fracture care is adequate to treat most injuries.

### **Osteochondromas**

Osteochondromas are one of the most common tumors of bone in children, and clinical symptoms usually are related to irritation of the surrounding soft tissue structures. Peroneal nerve palsy may occur in association with osteochondroma of the proximal fibula ([103](#)). Radiation induced osteochondromas also can occur ([104](#)).

Although they are rare, fractures may occur through the base or stalk of a pedunculated osteochondroma ([105](#)); conservative treatment is adequate ([Fig. 6-17](#)). Fractures through solitary osteochondromas should be observed, and excision should be reserved for those patients with persistent symptoms after healing.



**FIGURE 6-17.** A 10-year-old-boy with pain over the right medial tibia after a direct blow. An exostosis is present, and the transverse radiolucency at its base may represent a fracture. The patient continued to have symptoms after healing of the fracture and the osteochondroma was excised.

### **Eosinophilic Granuloma (Langerhans' Cell Histiocytosis)**

Eosinophilic granuloma of bone is a benign condition with either solitary or multiple lytic bone lesions. It is considered part of a general family of diseases collectively known as Langerhans' cell histiocytosis (LCH) or histiocytosis X, which also includes Hand-Schüller-Christian and Letterer-Siwe diseases ([111,115](#)). Bone lesions contain large coffee bean-shaped histiocytes, eosinophils, and multinucleated giant cells ([117](#)).

Nearly 75% of patients with eosinophilic granuloma of bone are younger than 20 years old, and 34% are younger than 4 years old. The ratio of male to female is approximately 3:2 ([117](#)). Nearly 50% of patients present with localized pain and tenderness ([108,117](#)); the duration of symptoms ranges from 2 weeks to 2 months, with occasional associated trauma. Swelling may be present over superficial bones such as the skull, clavicle, and tibia ([108](#)). In order of decreasing frequency, the sites of involvement are the skull, femur, pelvis, ribs, humerus, spine, clavicle, mandible, tibia, radius, scapula, and fibula. In one series ([113](#)), 14% of patients with solitary eosinophilic granuloma of bone had additional lesions within 2 years of diagnosis, whereas 85% of patients with multiple lesions at the time of initial diagnosis had additional bone involvement. The incidence of pathologic fracture in patients with LCH is approximately 14% ([108,117](#)). Laboratory tests usually are not very helpful; eosinophilia is infrequent ([117](#)).

Patients with vertebral body involvement present with back pain and may have neurologic signs or symptoms. Although the thoracic spine is the most commonly involved part of the spine, both cervical and lumbar lesions have been reported ([113](#)).

### **Radiographic Findings**

In children, long bone lesions may occur in both the diaphysis and metaphysis, with destructive osteolysis that erodes the cortex and overlying expansion by periosteal layering ([108,113,114](#)). Epiphyseal involvement is rare. Defects in bone may be lobulated, and aggressive subperiosteal bone formation may suggest malignancy. The size of the lytic area may vary from 1 to 4 cm ([117](#)).

Classic vertebral plana is uncommon with eosinophilic granuloma of the spine in a child. When present, however, usually only one vertebra is involved, and it assumes a coin-on-end appearance with intact adjacent disk spaces. X-ray studies of patients with skull involvement show characteristic punched-out round lytic lesions that are beveled on tangential views.

The lesion may mimic osteomyelitis, Ewing's sarcoma, Brodie's abscess, metastatic disease, and osteogenic sarcoma ([108](#)). Bone scans tend to be unpredictable, with the incidence of false-negative scans ranging from 28% to 35% ([106](#)). In one series ([106](#)), the increased uptake of isotope was seen in 60% of patients, and there was an 11% incidence of cold lesions found only in the vertebral column and the ribs. Although bone scans may be useful in identifying recurrent lesions ([106](#)), the skeletal survey is most valuable in identifying the lesions. A bone scan should be used only when the x-ray studies are normal or equivocal.

### **Treatment**

Biopsy usually is needed for diagnosis because of the lesion's tendency to mimic more serious conditions. Once the diagnosis is established, treatment options may include curettage, curettage and bone grafting, irradiation, chemotherapy ([108,117](#)), and corticosteroid injection ([115](#)). All of these forms of treatment result in healing of the lesions ([108](#)). For small lesions and an established diagnosis, observation may be the best option; marginal sclerosis about the lytic area suggests healing.

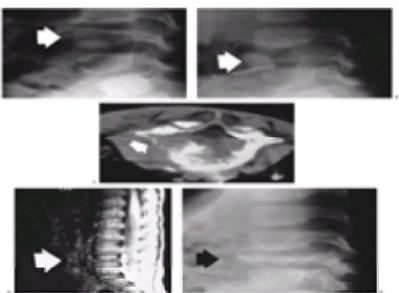
Sbarbaro and Francis ([117](#)) reported an average healing time of 16 months after curettage without grafting. McCollough ([113](#)) noted that if a lesion increased in size after surgery, then additional bone or soft tissue lesions usually appeared. Yasko et al. ([119](#)) described a percutaneous needle biopsy technique for diagnosis with subsequent methylprednisolone injection for patients with localized LCH; 34 of 35 lesions injected with methylprednisolone healed, and there were no complications. Other investigators ([107](#)) have had similar results. Radiation therapy has been recommended for some inaccessible lesions ([108](#)), but one series suggested an

association of this treatment with subsequent lymphosarcoma (108). Chemotherapy, usually consisting of oral methotrexate and prednisone, is used in patients with severe, painful, or progressive lesions or visceral involvement (115).

Fracture bracing is useful for both acute fractures and prophylactic use for impending fracture or after biopsy of lower extremity lesions (109). Surgery may be necessary for unstable fractures (Fig. 6-18). The diagnosis of vertebral lesions usually is best established by biopsy, especially if there are any atypical radiographic feature of a lesion such as a soft tissue mass. After diagnosis, they usually are treated with activity modification and a spinal orthosis (Fig. 6-19) (110,112). Seimon (118) recommended biopsy "only if there is any uncertainty in diagnosis," and irradiation only for progressive disease. Surgery may be necessary when there are associated neurologic deficits (113). Remodeling is seen with some spinal lesions but does not seem to correlate with patient age (110,112,116).



**FIGURE 6-18.** Eosinophilic granuloma. **A:** A 4-year-old boy with a pathologic fracture of the right femoral neck secondary to eosinophilic granuloma (arrow). **B:** The pins were removed, and at 3-month follow-up, the fracture was healed with acceptable alignment.



**FIGURE 6-19.** Eosinophilic granuloma. **A:** A 5-year-old boy presented with mild back pain and normal results of physical examination. A lateral x-ray study showed equivocal posterior wedging of L4 (arrow). Bone scan was read as normal. **B:** Nineteen days later on follow-up, he had marked spasm of the lower back. The lateral x-ray study now shows vertebral plana of L4 (arrow). **C:** CT scan shows marked expansion and erosion of the pedicle (arrow) and vertebral body. **D:** MRI shows marked collapse of L4 with thickening of the adjacent intervertebral disks. Soft tissue mass is also seen adjacent to L4 (arrow). Eosinophilic granuloma was diagnosed by Craig needle biopsy guided by CT scan, and the patient was treated with a spinal orthosis. Radiation treatment was also recommended, and a total of 600 rads was given in three divided doses. **E:** At 2-month follow-up, the compressed vertebra was beginning to regain density (arrow). The patient was asymptomatic. Bracing was continued for a total of 6 months.

## AUTHORS' PREFERRED METHOD OF TREATMENT

Pathologic fracture is uncommon in patients with LCH. The correct diagnosis should be established with biopsy for most lesions, and the use of newer diagnostic methods such as immunohistochemistries (such as CD1A) can be helpful in confirming the diagnosis of these lesions. The natural history is one of gradual regression and healing. Standard fracture care is usually sufficient for pathologic fractures.

### **Malignant Bone Tumors and Metastasis**

The two most common primary sarcomas of the long bones in children are Ewing's sarcoma and osteosarcoma. Destructive lesions also can be caused by metastatic cancer to bone.

Careful staging (124,130) and subsequent biopsy (121,131,132,135) are critical in the evaluation of children with malignant bone tumors. To avoid pathologic fracture from biopsy of bone, an oval hole with smooth edges should be used and should be filled with PMMA (130). Many malignant tumors have a large soft tissue mass that can be biopsied, obviating the need to make a hole in the bone. There has been a great deal of progress in the understanding of the molecular biology and genetics of cancer (125,126,127 and 128). These advances have already led to better diagnostic analysis of these tumors. Immunohistochemical, molecular genetic, and cytogenetic tests often are critical in establishing the correct diagnosis, especially small round blue cell tumors. The evaluation and biopsy of these children should preferably be done at a center where these techniques are known and available (121,129,131,132).

One of the major advances in the care of children with isolated extremity sarcoma has been the development of limb-sparing surgical techniques for local control of the tumors. Pathologic fracture has been cited as a contraindication to this procedure because of concerns about tumor dissemination by fracture hematoma (Fig. 6-20). Until recently, there has been little clinical data in the literature on which to base the treatment of these patients. A number of recent studies, however, have shown that pathologic fractures heal with neoadjuvant chemotherapy and do not affect survival rates (123,133,137). Abudu et al. (120) reviewed the surgical treatment and outcome of pathologic fractures in 40 patients with localized osteosarcomas and found that limb-sparing surgery with adequate margins of excision could be achieved in many patients without compromising survival, but that 19% of those treated with limb-sparing surgery had local recurrences. Scully et al. (134) reviewed the surgical treatment of 18 patients with osteosarcomas pathologic fractures. Of the 10 patients who had limb-sparing surgery, three had local recurrences and six had distant recurrences. Although the distant recurrence rate for patients undergoing amputation was no different from the rate for those undergoing limb salvage, the difference in local tumor control approached statistical significance. All patients who developed local recurrence died. The authors stated that surgical treatment should be individualized (134). Limb-sparing surgery is possible and appropriate in carefully selected patients as long as wide margins can be safely achieved and the function of the child will be better than that achieved with an amputation and a well-fitting prosthesis.



**FIGURE 6-20.** A 15-year-old girl was referred with a pathologic fracture of the femoral shaft after a fall while going down stairs. On close questioning, she stated that she had had pain in her thigh for several weeks before the fall and that the fall occurred after her leg gave way while going down the stairs. As an infant, she had already been treated with a cast for 2 months. **A:** The patient had been casted at another hospital, and x-ray studies in the cast show a transverse fracture of the mid-femoral shaft with destructive changes and worrisome periosteal elevation. **B:** MRI showed destructive changes of the mid-shaft of the femur with a soft tissue mass and bleeding from the fracture. An open biopsy established the diagnosis of Ewing's sarcoma of the femur, which was treated with neoadjuvant chemotherapy.

Pathologic fracture after limb-sparing surgery is a major complication, occurring most commonly after allograft reconstruction ([122,136](#)). Berrey et al. ([122](#)) reviewed 43 patients in whom allografts used in reconstruction after resection of tumors had subsequently fractured. Four fractures healed with immobilization alone, and the remainder of patients attained satisfactory results with open reduction and grafting, replacement of the internal fixation device, or total joint replacement ([122](#)). San-Julian and Canadell ([136](#)) reported on 12 patients with 14 fractures (10.2% of 137 patients with allografts for limb-sparing surgery in their series). They recommended intramedullary fixation whenever possible to reduce the incidence of allograft fracture.

Pathologic fractures also can occur in children with metastatic disease but are less common in children than in adults. Most are microfractures and can be managed with conservative fracture management techniques.



## AUTHORS' PREFERRED METHOD OF TREATMENT

In all suspicious lesions, careful staging and biopsy are the appropriate treatments by individuals who have experience in the management of children with musculoskeletal sarcomas. Furthermore, access to special diagnostic modalities, such as immunohistochemistry and cytogenetics, will lessen the chances of misdiagnosis. The decision for or against limb-sparing surgery in patients with pathologic fractures should be individualized based on factors such as the fracture displacement, fracture stability, histologic and radiologic response to chemotherapy, and most important, the ability to achieve wide margins for local tumor control. Pathologic fractures that occur after reconstruction through allograft or endoprosthetic reconstruction often can be successfully treated with bone grafting or exchange of allograft or endoprosthesis.

## BONE AND FIBROUS TISSUE DISEASES

### Fibrous Dysplasia

Fibrous dysplasia, a developmental abnormality of bone presenting as expansile fibrous lesions, can result in pathologic fracture and deformity. Three forms exist: monostotic fibrous dysplasia, polyostotic fibrous dysplasia, and McCune-Albright syndrome (MAS). In 1937, McCune and Bruch ([154](#)) and Albright et al. ([138](#)) described patients with these osseous lesions in association with cutaneous pigmentation and endocrine dysfunction. Later, Lichtenstein and Jaffe ([152,153](#)) coined the term fibrous dysplasia and further divided the entity into monostotic and polyostotic forms without endocrine disorder. The common factor is expansile fibrous tissue lesions of the bone, which contain woven bone formed by metaplasia with poorly oriented bone trabeculae.

Studies have demonstrated that MAS is caused by activating mutations in the gene for the alpha subunit of the heterotrimeric stimulatory G protein of adenylate cyclase (*GNAS1* gene), located on the long arm of chromosome 20 at locus 20q13.2-q13.3 ([139,155](#)). The mutation in this gene likely occurs in embryonic development and is expressed in a mosaic pattern, resulting in the often lateralized pattern of skin and bone involvement in patients with MAS. This mutation is not present in tissue from patients with aggressive fibromatosis involving bone or osteofibrous dysplasia ([139](#)).

### Monostotic Fibrous Dysplasia

#### Clinical Presentation

The diagnosis usually is made between the ages of 10 to 15 years of age, although neonatal fibrous dysplasia of the fibula has been reported. The lesions usually are asymptomatic until a fracture occurs, then patients may have pain and swelling ([146](#)). Incomplete fractures are most common. The sites of fracture in order of decreasing frequency are the proximal femur, tibia, ribs, and bones of the face ([146](#)). Cutaneous lesions usually are not present in monostotic fibrous dysplasia. Although pregnancy may stimulate the lesions ([176](#)), overall progression is rare after initial presentation. Sarcomatous degeneration has an incidence of approximately 0.5% and generally occurs approximately 15 years after initial diagnosis.

The central dilemma in monostotic fibrous dysplasia is distinguishing the lesion from other benign disorders. The differential diagnosis usually includes eosinophilic granuloma, UBC, giant cell tumor, enchondroma, solitary fibroma, and osteomyelitis. MRI can be helpful in evaluating these lesions, but biopsy is sometimes necessary to establish the correct diagnosis.

#### Radiographic Findings

Radiographically, lesions of monostotic fibrous dysplasia usually are elliptical, central lesions in the mid-diaphysis. The borders of the lesion are commonly sclerotic; trabeculation is more common than a ground-glass appearance. There often is a slight bowing of the tibia, but bowing of the femur is uncommon.

With evaluation by CT scan, the extent of cortical thinning can be studied and relative central densities measured. Eosinophilic granuloma, neoplasm, and osteomyelitis have a density on CT scan ranging from 20 to 40 Hounsfield units, whereas fibrous dysplasia has a higher density, ranging from 70 to 130 Hounsfield units. MRI may be useful to differentiate fibrous dysplasia from other lesions, especially UBC.

### Injury

Pathologic fractures occur in nearly 45% of patients. Fractures of the long bones are generally not displaced; many are microfractures. Although the fractures heal rapidly, endosteal callus is poorly formed and periosteal callus is normal ([148](#)). With mild deformity, the cortex thickens on the concave side of the long bone. Nonunion is rare.

### Treatment

Conservative treatment with immobilization is indicated for most fractures that occur in conjunction with monostotic fibrous dysplasia. Traction with subsequent casting can be used for femoral shaft fractures in young children; casts or cast-bracing for upper and other lower extremity fractures is often appropriate ([146](#)).

Operative intervention is indicated for fractures of severely deformed long bones and those through large cystic areas. Bone graft can be resorbed, but total obliteration of the lesion often can be accomplished with grafting. Deformity can occur, and internal fixation may be required for stabilization. Complete *en bloc* extraperiosteal excision with grafting has been shown to be successful for severe lesions but is seldom needed. Both painful lesions without fracture and impending pathologic fractures can be treated with bone grafting.

Proximal femoral lesions with pathologic fracture are especially troublesome because of the propensity for malunion with coxa vara. For fractures through small lesions, either cast immobilization or curettage with grafting can be used; osteotomy can be done for residual deformity ([145](#)). For larger lesions, internal fixation is necessary. Proximal femoral pathologic fractures have been stabilized with lag screws, blade plates ([146](#)), intramedullary nails, and Enders nails. Cast immobilization and protected weight bearing are necessary after these procedures to protect the reduction. Spine fractures are rare but can be treated with bed rest followed by immobilization with an orthosis ([146](#)).

## Polyostotic Fibrous Dysplasia

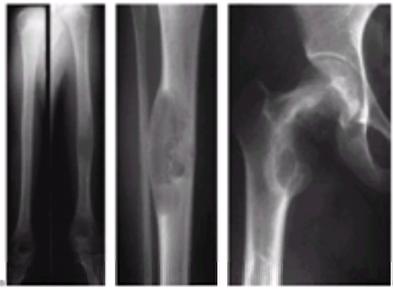
### Clinical Presentation

Most patients with polyostotic fibrous dysplasia present before age 10 years with pain, limp, deformity, or pathologic fracture ( 146,152). The bones most commonly affected are the femur, tibia, humerus, radius, facial bones, pelvis, ribs, and phalanges ( 152). Involvement is often unilateral, usually affecting a single extremity. In one series, 50% of patients had facial involvement ( 146). Spine involvement occurs with polyostotic fibrous dysplasia, and limb-length discrepancy is common (146,148). Although most laboratory studies are normal, serum alkaline phosphatase levels may be elevated ( 148). Some authors believe that polyostotic fibrous dysplasia does not usually progress significantly after adulthood ( 146,158), but others (148) believe that puberty does not affect the bone lesions.

Both intramuscular myxoma and myositis ossificans progressiva have been associated with polyostotic fibrous dysplasia. When it occurs, sarcomatous degeneration of the lesions occurs 10 to 12 years after the initial diagnosis of fibrous dysplasia; osteogenic sarcomas, chondrosarcomas, and giant cell sarcomas also have been reported. The warning signs for sarcoma in existing lesions of fibrous dysplasia are pain and rapid enlargement of the lesion.

### Radiographic Findings

Polyostotic fibrous dysplasia appears as multiple expansile lesions with cortical erosion. Most have a multilocular appearance associated with a scalloped pattern of endosteal erosion (175). A ground-glass appearance on x-ray study is caused by the metaplastic woven bone comprising the lesion. A radiolucent cystic appearance also is common. Cartilage may be present in approximately 10% of lesions, and radiographic stippling may be seen ( 153). In contrast to those in monostotic fibrous dysplasia, the lesions in the polyostotic form have little increased peripheral density, and they usually form a characteristic fusiform expansion of the bone ( Fig. 6-21). Bowing of the long bones is common, and normal tubulation may not occur with growth (148).



**FIGURE 6-21.** Polyostotic fibrous dysplasia in a 10-year-old girl. **A:** A fusiform expansile lesion is present in the mid-shaft of the humerus. **B:** A more eccentric expansile lesion is seen in the tibial shaft. There is scalloping due to endosteal erosion with a central ground-glass appearance. The lesion was painful and was treated with curettage and bone graft. (Courtesy of Jack Henry, M.D., San Antonio, Texas.) **C:** Polyostotic fibrous dysplasia in a 26-year-old woman. A large erosive lesion is present in the inferior neck of the femur. This lesion was treated by curettage with both fibula and iliac crest bone graft. (Courtesy of Gregorio Canales, M.D., San Antonio, Texas.)

Distinguishing polyostotic from monostotic fibrous dysplasia may be difficult. Plain x-ray skeletal surveys usually are done; technetium bone scans are helpful in identifying multiple lesions that may not be present on plain x-ray studies ( 143,149). Harris et al. (148) suggested that the hallmarks of polyostotic fibrous dysplasia are the characteristic long bone lesions with extension from epiphysis to epiphysis and increased density at the base of the skull. They suggest that a survey of the pelvis and femurs is most helpful in noting multiple lesions. Epiphyseal lesions can also occur ( 148).

### Injury

In one series of 37 patients with polyostotic fibrous dysplasia, nearly 85% had at least one fracture and 40% had an average of three fractures ( 148). Fractures are most common in the femur, humerus, radius, and wrist (152,153). Like fractures in monostotic fibrous dysplasia, fractures in the polyostotic form generally are not displaced and healing is not delayed; nonunion can occur, however ( 148). Rib fractures are rare, and generally rib lesions are asymptomatic. A shepherd's crook deformity of the humerus associated with polyostotic fibrous dysplasia has been reported ( 143,157). Compression of the spinal cord by fibrous tissue also has been reported.

### Treatment

The fractures of polyostotic fibrous dysplasia usually occur through very diseased bone and are associated with marked deformity. They often require more aggressive treatment than fractures seen in the monostotic form. Conservative immobilization techniques usually are appropriate for most shaft fractures in children before puberty. Fractures of the femur can be treated with traction and subsequent casting in young patients. After adolescence, however, the recurrence of deformity after surgery is less, and curettage and grafting should be considered for fractures, especially for large lesions with associated deformity ( 148,153). Stephenson et al. (156) found that in patients younger than 18 years of age, closed treatment or curettage and bone grafting of lower extremity fractures gave unsatisfactory results, but internal fixation produced more satisfactory outcomes. A Sofield procedure (osteotomy and intramedullary nailing) can be used for tibial deformity.

The greatest challenge in polyostotic fibrous dysplasia is treatment of fractures of the proximal femur. With recurrent fracture and deformity, a severe coxa vara resembling a shepherd's crook develops. Curettage of the lesion with abundant bone grafting has been recommended for mild deformities ( 149,153), and fixation usually is needed for large lesions. Femoral neck fracture or osteotomy for deformity can be stabilized with internal fixation. For severe shepherd's crook deformity, medial displacement valgus osteotomies with plate fixations are needed to restore the biomechanical stability of the hip ( 147). For severe lesions, Funk and Wells (145) recommended complete excision of the intertrochanteric area and advancement of the psoas and gluteus medius tendons. Breck ( 140) recommended securing the side plate of the femoral nail with bolts and washers rather than with screws to obtain better stability. Massive autogenous bone grafting is believed to be superior to donor bone graft (148), but it may be impractical because the iliac crests of a young child may be small and the pelvis may have coexisting disease.

The use of bisphosphonates may offer hope for a medical treatment for patients with severe fibrous dysplasia. Radiation therapy is not recommended ( 152).

## McCune-Albright Syndrome

### Clinical Presentation

McCune-Albright syndrome is a rare variant of fibrous dysplasia associated with endocrine dysfunction (such as precocious puberty in females), characteristic bone lesions (osteitis fibrosa disseminata), and areas of cutaneous pigmentation ( 138,154). It affects both women and men and has been associated with pituitary adenoma, gigantism, hyperthyroidism, and Cushing's syndrome (150). The skin lesions generally are flat, multiple melanotic areas that stop at the midline and parallel the distribution of bone lesions ( 146). The irregular margins have been compared to the coast of Maine in contrast to the smoother lesions found in neurofibromatosis, which are likened to the coast of California. Skin lesions may also be absent.

On x-ray studies, a polyostotic presentation is seen. With precocious sexual development, there is rapid bone maturation with early physal closure and short stature (146). Pathologic fractures in childhood occur with a pattern and appearance similar to those in polyostotic fibrous dysplasia.

## Treatment

The literature is limited regarding specific treatment of McCune-Albright syndrome, but both conservative treatment and aggressive surgical intervention have been used with apparent success. Internal fixation may be necessary in severe cases ([Fig. 6-22](#)). With the recent progress in understanding the genetic basis of this disorder, newer treatment alternatives may become available.



**FIGURE 6-22.** **A:** A 7-year-old girl presented with McCune-Albright syndrome. Skull radiographs show multiple lesions consistent with this condition. **B:** Expansile rarefied lesions are present throughout the humerus and the radius. **C** and **D:** Both proximal femurs have areas of rarefaction with expansile lesions (*arrows*). **E:** Both tibiae and fibulae show areas of expansile fibrous dysplasia (*arrows*).

## AUTHORS' PREFERRED METHOD OF TREATMENT

Conservative treatment with immobilization is indicated for most fractures in children with monostotic fibrous dysplasia. In younger children, immediate casting, or traction and subsequent casting are used for most femoral shaft fractures. Because fractures in patients with polyostotic fibrous dysplasia usually occur through very abnormal bone and can result in marked deformity, they often require more aggressive treatment (e.g. internal fixation).

After adolescence, the occurrence of deformity after surgery is less frequent. Nonoperative treatment of fractures and curettage and cancellous bone grafting do not generally produce satisfactory results in children with fibrous dysplasia of the lower extremity. Curettage and grafting are indicated for fractures of severely deformed long bones and those through large cystic areas, with internal fixation appropriate for the location and age. Bone graft can be resorbed after placement in extensive lesions, and proximal deformity can occur after corrective osteotomy.

### Proximal Femoral Lesions

One of the most common sites of fracture and deformity is the proximal femur. Proximal femoral lesions with pathologic fracture are especially difficult because of the tendency for varus deformity and repeated fracture. Stable fractures through small lesions can be treated with cast immobilization, but one must be vigilant and ready to intervene at any sign of varus displacement.

Femoral neck fractures can be stabilized *in situ* with a cannulated screw, or compression screw and side plate, depending on the extent of involvement and the nature and location of the fracture. Fixation can be combined with valgus osteotomy if there is preexisting deformity or with curettage and grafting if there is a large area of bone loss. Postoperative cast immobilization and protected weight bearing usually are necessary. Varus deformity is best treated with valgus osteotomy of the subtrochanteric region and internal fixation early in the course of the disease to restore the normal neck shaft angle and mechanical axis. Intramedullary load-sharing fixation (such as flexible intramedullary nails) can be used for juvenile patients with femoral shaft fractures.

For larger lesions with more severe deformity, and in older patients, rigid fixation often is necessary. Depending on the situation, intramedullary load-sharing fixation devices that support not only the femoral neck but also the shaft of the femur (such as *custom intramedullary reconstruction nails*) are better and should be used when possible. For severe shepherd's crook deformity, medial displacement osteotomies are needed to restore the biomechanical stability of the hip.

Patients with McCune-Albright syndrome should have careful preoperative evaluation in preparation for anesthesia and surgery. The reader is referred to an excellent review by Langer et al. ([151](#)) for further information.

### Osteofibrous Dysplasia of the Tibia and Fibula

#### Clinical Presentation

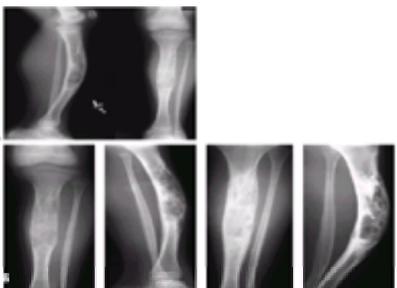
The term osteofibrous dysplasia of the tibia and fibula is advocated to describe specific, uncommon fibrous lesions of the tibia and fibula in young children ([160](#)). These lesions superficially resemble monostotic fibrous dysplasia but exhibit unique natural history, response to treatment, and specific histology.

Most patients present before the age of 5 years, but the range varies from 5 weeks to 15 years of age ([160,163](#)). There usually is painless enlargement of the tibia with slight to moderate anterior or anterolateral bowing. The disease process is almost always confined to one tibia, but the ipsilateral fibula can also be involved. Solitary involvement of the fibula is infrequent, and bilateral involvement of both tibias is rare. Both distal and proximal lesions can occur, and with fibular involvement, the lesion is located distally.

Biopsy specimens distinguish this entity from fibrous dysplasia or adamantinoma. The fibrous tissue present is less cellular than in fibrous dysplasia. Woven bone is at the center of the lesion, with newly formed bone trabeculae bordered by active osteoblasts and lamellar structure near the periphery of the lesion. This pattern is unusual in fibrous dysplasia. A particular zonal architecture is present in large biopsy specimens and shows increasing size and maturity of the bone trabeculae toward the edge of the lesion. Some authors believe that osteofibrous dysplasia may be related to adamantinoma ([164,165](#)).

#### Radiographic Findings

An eccentric intracortical lesion of osteolysis usually is present in the middle third of the tibia, with extension proximally or distally ([Fig. 6-23](#)) ([163](#)). The cortex overlying the lesion is expanded and thinned, and in the medullary canal, a dense band of sclerosis borders the lesion with narrowing of the medullary canal. A single area of radiolucency may be present and has a ground-glass appearance, but often there are several areas of involvement with a bubble-like appearance.



**FIGURE 6-23.** Osteofibrous dysplasia of the tibia. **A:** An 8-year-old girl presented with slight bowing of the left leg. X-ray study showed a circumscribed sclerotic and lytic lesion of the diaphysis of the tibia. Lateral radiographs show bowing of the tibia with a diaphyseal intracortical lesion. The patient was thought to have osteofibrous dysplasia, and was braced and followed yearly. **B:** Eight years later, the lesion has become slightly more extensive and is associated with slight progression of anterior bowing of the tibia. The patient underwent open biopsy, and pathologic diagnosis was consistent with adamantinoma. Chest CT showed no sign of lung involvement. The patient was treated with wide excision of the entire lesion and free vascular fibula bone graft reconstruction. **C:** The patient has had no evidence of further recurrence at 16-month follow-up.

The differential diagnosis usually includes both monostotic fibrous dysplasia and adamantinoma, but some authors ( [160](#)) believe that the characteristic x-ray appearance and the early onset of the disease effectively exclude these two disorders and that biopsy is not necessary. Others support the need for biopsy to establish diagnosis. Bracing with orthotics is advisable after open biopsy for diagnostic purposes.

### **Injury**

Pathologic fractures are present in nearly one third of patients ( [160](#)). These fractures are either incomplete or minimally displaced and heal well with both closed and open methods ( [160](#)), although delayed union may be a problem. Pseudarthrosis is rare.

### **Treatment**

The natural history of this disorder in untreated patients is variable. Most lesions show slight or moderate progression in late childhood, but others may show aggressive expansion with bowing of the tibia in patients up to 15 years of age. Rarely, the lesions may even regress or remain stationary over 3 to 4 years. Most lesions stop expanding after completion of skeletal growth. What appears to be osteofibrous dysplasia may actually be early adamantinoma ( [164](#)). MRI showing a soft tissue extension is indicative of adamantinoma. Open biopsy also may be needed to establish the biphasic histology of adamantinoma.

Curettage and grafting were associated with a local recurrence rate of up to 64% in children 14 years old or younger in one series ( [160](#)). Others reported a lower local recurrence rate. Ozaki et al. ( [162](#)) showed the course of six tibial lesions in five patients with osteofibrous dysplasia who were followed longer than 10 years (average: 16.8 years). Curettage and autogenic bone grafting were performed on two lesions, which then healed. Of four lesions on which curettage and autogenic bone grafts were performed, three healed and one recurred. The recurrent lesion healed after curettage and xenogeneic bone grafting. Three lesions healed without surgical treatment. During long-term follow-up, this disease showed a clear tendency for healing ( [162](#)). Wide extraperiosteal resection can be performed, but graft resorption can still occur ( [160](#)). Some authors ( [159,161](#)) believe that bracing until skeletal maturity is preferable to surgery.

Pathologic fractures in this disorder should heal with cast immobilization in plaster casts. If fractures recur, or if the lesion is rapidly progressive, wide extraperiosteal resection with grafting is necessary ( [160](#)). Open reduction with bone grafting and internal fixation is recommended for fractures with angular deformity. Early osteotomy is recommended for severe bowing deformity.

### **Neurofibromatosis**

Neurofibromatosis, also known as von Recklinghausen's disease, is an autosomal dominant condition with variable penetrance that occurs in 1 in 2,500 to 3,000 live births ( [177](#)). It affects neural tissue, vascular structures, skin, and the skeleton. The diagnosis of neurofibromatosis can be based on the presence of two of the four following criteria, according to Crawford and Bagamery ( [177](#)):

1. Multiple café-au-lait spots
2. Positive family history for neurofibromatosis
3. Diagnostic biopsy of a neurofibroma
4. Presence of pseudarthrosis of the tibia, hemihypertrophy, or a short, angular scoliosis

Crowe and Schull ( [178](#)) pointed out that adult patients with neurofibromatosis usually had more than five café-au-lait spots with a diameter of more than 1.5 cm. The presence of café-au-lait spots, however, is not pathognomonic for neurofibromatosis. Whitehouse ( [214](#)) noted that 23% of normal children have one or two café-au-lait spots with a diameter of more than 0.5 cm, and the presence of five or more café-au-lait spots is needed to suggest the diagnosis of neurofibromatosis. Although café-au-lait spots may be present at birth, usually they are not seen until the patient is 5 or 6 years old ( [177](#)). Generalized soft tissue hypertrophy of the limbs is present in 37% of adults with neurofibromatosis ( [196](#)), whereas children have an 11% incidence of limb-length discrepancy and only a 3% incidence of soft tissue enlargement of the extremities ( [177](#)).

A diagnostic biopsy of a dermal neurofibroma is considered a valuable criterion for the diagnosis of neurofibromatosis. These tumors, however, tend not to be clinically apparent until the child is older than 12 years of age ( [177](#)). Plain x-ray studies are not helpful in identifying these soft tissue tumors. MRI can be helpful in identifying the soft tissue masses. Technetium 99m-labeled diethylenetriaminepentaacetic acid (DTPA) accumulates in the soft tissue tumors of neurofibromatosis ( [191,192](#)). Routine isotopic imaging with this technique can identify lesions as small as 1.5 cm. Lesions as small as 0.8 cm were seen through a more advanced technique known as single proton emission computed tomography. Such techniques may be useful in identifying occult neurofibromatosis and pseudarthrosis of the long bones.

### **Injury**

Pseudarthrosis of the long bones in patients with neurofibromatosis can be a therapeutic dilemma. The appearance of pseudarthroses and their resistance to treatment has been postulated to be due to a deficiency of bone formation secondary to mesodermal dysplasia. More recently, the abnormal soft tissue associated with these pseudarthroses has been postulated to be the major associated factor in causing pseudarthrosis ( [216](#)). Wright et al. ( [216](#)) developed a rabbit tibia congenital pseudarthrosis model and suggested that a possible underlying abnormality in congenital pseudarthrosis might actually be the abnormal soft tissue surrounding the bone rather than the bone itself ( [216](#)).

Approximately 5% of patients with neurofibromatosis are thought eventually to have pseudarthrosis of the long bones. The tibia is the bone most often affected, but only 55% of the cases of congenital pseudarthroses of the tibia are thought to be associated with neurofibromatosis ( [176](#)).

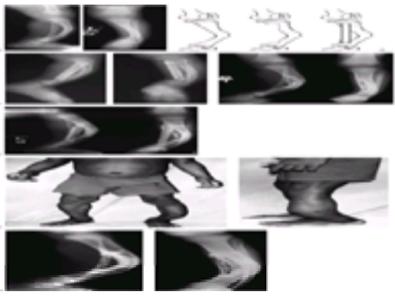
The term congenital pseudarthrosis in neurofibromatosis is misleading because a substantial number of patients do not have a pseudarthrosis at birth but rather develop it later after a pathologic fracture ( [199](#)). Brown et al. ( [175](#)) found that in six children with neurofibromatosis, anterior bowing of the leg developed at an average age of 8 months and then went on to fracture and pseudarthrosis an average of 4.5 months after the initial clinical observation of deformity.

The focus of the recent orthopaedic literature has been treatment of congenital pseudarthrosis of the tibia ( [199](#)), but pseudarthroses in other locations in children with neurofibromatosis also occur and can be a challenge. Pseudarthroses have been reported in the radius ( [171,175,184,185,186,187,188,189,190,191,192,193,194](#) and [195](#)), ulna ( [166,167,170,171,189,200,201](#)), both the radius and the ulna ( [167,170,193,195,205,207](#)), femur, clavicle, and humerus. Most patients had associated neurofibromatosis, but some with pseudarthroses in each of the above-mentioned locations did not.

### **Radiographic Findings**

Anterolateral bowing of the tibia with loss of the medullary canal usually is present before fracture ( [Fig. 6-24](#)) ( [175](#)). Another x-ray characteristic is a prefracture cystic lesion of the tibia with anterolateral bowing ( [176](#)). Biopsy specimens of these pseudarthroses invariably reveal fibrous tissue, but there are reports of ( [167,194](#)) found evidence of neural tissue in biopsy specimens. None of these findings, however, has been confirmed by electron microscopy to document the presence of Schwann cells. X-ray findings in patients with established pseudarthroses of the tibia include narrowing or obliteration of the medullary canal at the pseudarthrosis site, with

sclerosis and anterolateral angulation ( [Fig. 6-25](#)). Pseudarthrosis of the fibula is associated with valgus deformity of the ankle ( [Fig. 6-26](#)).



**FIGURE 6-24.** **A:** A 2-year-old boy with neurofibromatosis presented with anterolateral bowing, sclerosis, and partial obliteration of the medullary canal of the tibia without fracture. **B:** A modified McFarland technique for prophylactic bypass grafting was performed as shown. **C:** Immediate postsurgical x-ray studies of the tibia after prophylactic bypass grafting. **D:** Three years later, x-ray studies show continued growth of the tibia without fracture but some absorption of the allograft and relative loss of structural support by the allograft related to continued growth. **E:** X-ray studies after a second prophylactic bypass grafting. **F:** Clinical photographs of patient's lower extremities. **G:** Current x-ray study 5 years after the initial presentation. The patient has had no fractures and is functioning well without pain. (From Dormans JP. Modified Sequential McFarland Bypass Procedure for prepseudarthrosis of the tibia. *J Orthop Tech* 1995;3:176–180.)



**FIGURE 6-25.** **A and B:** Two examples of untreated congenital pseudarthrosis of the tibia in children with neurofibromatosis. **C:** After resection and reconstruction with a vascularized free fibula interposition.



**FIGURE 6-26.** Pseudarthrosis of the fibula is associated with valgus deformity of the ankle.

In upper extremity pseudarthroses of neurofibromatosis, the x-ray signs of the bone at risk are narrowing of the diaphysis ( [201](#)), sclerosis and hypoplasia with associated absent medullary canal ( [193](#)), and the presence of a cystic lesion in the bone. Once a fracture has occurred, a pseudarthrosis is likely when the fracture line persists for more than 7 weeks after injury ( [194](#)). The ends of the fracture gradually become tapered, there is little callus, and the cortex of the healing bone thickens with a decreased diameter of the medullary canal. The cause of these radiographic changes is unclear. Biopsy specimens from upper extremity pseudarthroses have consistently shown the presence of dense fibrous tissue without evidence of neurofibroma. Pseudarthroses have developed in children with neurofibromatosis after fracture through normal-appearing forearm bones ( [185,186](#)).

### Treatment

A tibia with an anterolateral bow in an infant or child will eventually fracture; corrective osteotomy to correct angular deformity will only accelerate the progression to pseudarthrosis and should not be done. Once the fracture occurs, there is no indication for closed treatment. In conjunction with excision of the hypotrophic bone ends, methods of treatment of this congenital pseudarthrosis include intramedullary fixation with iliac bone graft, fixation with vascularized fibular graft, and Ilizarov's compression of the pseudarthrosis with callotastic lengthening of the proximal tibia. All of these methods may be complicated by further pathologic fracture.

In contrast to pseudarthrosis of the upper extremity, there is substantial experience with treatment of pseudarthrosis of the tibia, but results are also disappointing. Bracing has proved ineffectual in the treatment of an established pseudarthrosis. Surgical procedures have included bypass grafts ( [197](#)), onlay grafts ( [173](#)), grafting with small bone chips, intramedullary nailing procedures ( [168,169](#)), and intramedullary rod fixation after segmental osteotomies ( [209](#)). The rate of union with these procedures ranges from 7% to 90%, and eventual amputation has been common. Electrical stimulation has been used with some success, but most series reporting its use have short patient follow-up and the electrical stimulation was used in combination with other surgical techniques. In one series, a 20% success rate was achieved using direct-current stimulation ( [174](#)). In another series, union was achieved in 10 of 12 patients with pseudarthrosis of the tibia through rigid intramedullary rod fixation and electrical stimulation through implanted electrodes ( [202](#)). The Farmer procedure, a skin and bone pedicle from the contralateral leg, has a reported union rate approaching 53% ( [198](#)). Free vascularized fibular grafts also have been used for reconstruction after excision of the involved tibia ( [198](#)). In one series, 11 of 12 patients with neurofibromatosis and pseudarthrosis of the tibia were successfully treated with free vascularized fibular grafts. Union of the pseudarthrosis occurred between 3 to 8 months after surgery ( [172,179,213](#)). A free vascularized iliac graft has been used in one patient with neurofibromatosis, resulting in union within 10 weeks ( [188](#)). Fabry et al. ( [181](#)) obtained union of pseudarthrosis of the tibia in two patients with compression through an Ilizarov fixator. In another series ( [204](#)), the fractures in three of five patients healed in 4.5 months. The other two patients needed supplementary iliac grafts, and eventually, the bone united. It is important to stress that treatment cannot be considered successful until skeletal maturity has been reached; many of these series included patients with short follow-up, and few included follow-up to skeletal maturity.

A prophylactic bypass grafting of the prepseudarthrotic tibia in neurofibromatosis has been performed with some success. This modification of the original McFarland bypass procedure, which was originally done for established pseudarthrosis, was successful in a series of patients from several centers reviewed by Strong and Wong-Chung ( [211](#)). A modified sequential McFarland bypass procedure for pre-pseudarthrosis of the tibia also has been described ( [Fig. 6-24](#)) ( [180](#)).

Amputation should be considered and discussed with the family early when previous operative interventions have been unsuccessful. Amputation usually is at the

Syme level, with prosthetic fitting around the pseudarthrosis. In a gait analysis study, Karol et al. ( 187) compared 12 patients with previously operated and healed congenital pseudarthroses of the tibia with four children with amputations for final treatment of congenital pseudarthroses of the tibia. They found marked disturbance of gait and muscle strength in patients with healed congenital pseudarthroses of the tibia. They concluded that patients with early onset of disease, early surgery, and transankle fixation had more inefficient gaits than amputees. Patients with forearm pseudarthroses can be pain free and function may be satisfactory with observation or splinting. However, persistence of an ulnar pseudarthrosis in a growing child often leads to bowing of the radius and posterior lateral subluxation or dislocation of the radial head ( 166,167,189,201). Healing after 6 months of casting has been reported in a 2-month-old infant with a congenital pseudarthrosis of the radius. There was no clinical evidence of neurofibromatosis at the time of treatment of this patient ( 183). Union after conventional bone grafting and fixation has been reported in only a small number of patients with congenital pseudarthrosis of the forearm ( 170,171,185,186,193,208). Many of these patients require multiple conventional bone grafting procedures and often years of immobilization. There are more reports of patients (and probably many more patients) with pseudarthroses of the forearm bones who did not respond to multiple grafting procedures ( 166,171,175,194,201). The results of treatment of congenital pseudarthrosis of the forearm in neurofibromatosis by free vascularized fibular grafts are encouraging. Allieu et al. ( 167) treated one patient with radial and ulnar pseudarthroses and another with ulnar pseudarthrosis with free vascularized fibular grafts. They obtained union in the patient with radial and ulnar pseudarthroses in 6 weeks and in the patient with ulnar pseudarthroses in 3 months. Earlier conventional grafting techniques had failed in both. Two additional patients with pseudarthroses of the radius without evidence of neurofibromatosis were treated with free vascularized fibular grafts, resulting union within 6 weeks ( 207,216). Mathlin et al. (195) reported six pseudarthroses of the forearm bones treated with vascularized fibular grafting with union in five ranging from 6 to 18 months after surgery. Other surgical options include excision of the ulnar pseudarthrosis to avoid a later tethering effect on the growing radius ( 166) and fusion of the distal radius and ulnar joint ( 201). Creation of a one-bone forearm is often technically successful, but both length and rotation of the forearm are sacrificed with this procedure ( 189,201).

Extreme care should be taken in surgical treatment of children with neurofibromatosis, in whom the periosteum of the long bones is believed to be less adherent to the bone than normal periosteum.

### Complications

Extensive subperiosteal hemorrhage with subsequent ossification was reported in a 9-year-old patient who underwent surgical epiphysiodesis of the proximal tibia and fibula ( 217). Massive subperiosteal hemorrhage due to minor trauma in children with neurofibromatosis occurred in the tibia and femur ( 217). The amount of blood loss in subperiosteal hemorrhages can be life-threatening. Yaghmai and Tafazoli ( 217) evacuated a subperiosteal hemorrhage of a femur in an 11-year-old boy with neurofibromatosis who presented with a rapidly growing mass of the thigh after a minor fall. An eggshell calcification was visible around the femur within weeks of the trauma. The cyst held 2,100 mL of serous fluid and went on to complete ossification within 12 weeks of surgery with marked thickening and distortion of the involved femur.

It is important preoperatively to rule out hypertension in children with neurofibromatosis because 16% of children with neurofibromatosis had hypertension in one series ( 212).

### Injuries of the Spine in Neurofibromatosis

Spinal deformity is the most common musculoskeletal abnormality seen in individuals with neurofibromatosis. Although scoliosis was present in 64% of patients with neurofibromatosis in one series ( 177), kyphoscoliosis may be the primary contributor to the development of paraplegia ( 215). Patients younger than 19 years of age may have paraplegia secondary to vertebral deformity, whereas those patients older than 19 are more likely to have neurologic deficits secondary to a neurofibroma. Complete dislocation of the spine with neurologic defect has been reported in two patients with neurofibromatosis ( 206). Rib penetration of the enlarged neural foramen with spinal cord compression in neurofibromatosis has also been reported in four patients ( 182,190). CT scan and MRI are useful for evaluating these patients. Resection through either an anterior or a posterior approach seems satisfactory ( 190).

## AUTHORS' PREFERRED METHOD OF TREATMENT

The treatment of congenital pseudarthrosis of the tibia remains controversial. When a child presents with prepseudarthrosis, (angulation without fracture), either bypass grafting with fibular allograft or bracing are reasonable options. Once pseudarthrosis has developed, our preference is inserting an intramedullary rod and bone grafting of both the tibia and fibula when possible. If these procedures fail, free vascularized fibula transfer or resection and bone transport with circular frame techniques can be considered. Amputation and prosthetic fitting should be considered early in patients with failure of the above-mentioned techniques and severe shortening and a stiff ankle and foot. Conservative options such as bracing or observation for upper extremity pseudarthroses may be justified in a patient with a nonprogressive deformity and a satisfactory functional use of the extremity. Conventional bone grafting and fixation procedures for treatment of pseudarthrosis of the upper extremity have very limited success, and other approaches should be considered. Free vascularized fibular grafts seem the treatment of choice for upper extremity pseudarthrosis associated with neurofibromatosis.

### CONGENITAL INSENSITIVITY TO PAIN

Congenital insensitivity to pain is a rare disorder characterized by the absence of normal subjective and objective responses to noxious stimuli in patients with intact central and peripheral nervous systems. The cause is unknown, but sporadic reports have appeared in the orthopaedic literature ( 219,220,221 and 222).

Orthopaedic manifestations of congenital insensitivity to pain include recurrent fractures, osteomyelitis, and neuropathic joints ( Fig. 6-27). Limb-length discrepancy may occur from physeal damage. Lack of pain perception is associated with the development of Charcot's joints, which may lead to later neuropathic arthropathy. The weight-bearing joints usually are affected, especially the knees and ankles.



**FIGURE 6-27. A:** This 6-year-old child with anhidrosis, congenital insensitivity to pain and attention deficit disorder presented with a history of swollen ankles and knees. This AP radiograph and **(B)** this lateral radiograph shows Charcot changes in the subtalar joint with calcaneous and distal fibula fracture. **C:** This AP radiograph of the right knee and **(D)** this lateral radiograph of the right knee show large, loose osteochondral fragments, medial subluxation of the femur on the tibia and extensive periosteal new bone formation in the distal femur. Soft tissue shadows are consistent with her huge knee hemiarthrosis. More than 100 mL of sterile serosanguineous fluid was aspirated from the knee at her initial visit. The effusion quickly returned in the days following the aspiration. Because management with casts at another hospital resulted in significant skin breakdown, we stabilized the knees with removable hinged braces. The effusions improved but did not resolve.

The differential diagnosis includes a spectrum of closely related sensory disorders including congenital sensory neuropathy, hereditary sensory radicular neuropathy, familial sensory neuropathy with anhidrosis, and familial dysautonomia (Riley-Day syndrome). Acquired conditions with pain insensitivity include syringomyelia, diabetes mellitus, tabes dorsalis, alcoholism, and leprosy. Loss of protective sensation promotes self-mutilation, burns, bruises, and fractures. The disease comes to light when the child develops teeth and then bites his or her tongue, lips, and fingers.

Management should aim at education and prevention of injury. Prevention of joint disease is the best early option ( 219,222). Joint injury should be recognized and

treated early to prevent progression to gross arthropathy. Early diagnosis of injury is important, with signs of instability, swelling, and local warmth prompting early investigation and treatment. Most fractures are treated nonoperatively, when appropriate. Immobilization, bed rest, or appropriate bracing usually is indicated (219,221).

In a severely unstable, degenerated joint, arthrodesis may eventually be appropriate; however, poor healing, nonunion, and pseudarthrosis are common in neuropathic joints. The condition appears to improve with time with the gradual recovery of pain sensation.

## MARROW DISEASE OF BONE

### Gaucher's Disease

Gaucher's disease is a hereditary disorder of lipid metabolism caused by a deficiency of the lysosomal enzyme glucocerebrosidase resulting in an abnormal accumulation of glucocerebroside (glucosylceramide) in macrophages of the reticuloendothelial system. It is the most common sphingolipidosis and is inherited as an autosomal recessive trait (232). It is very rare, with most cases noted in Ashkenazic Jews of eastern European origin (228). It has three forms of presentation. Type I is a chronic non-neuropathic form (i.e., without neurologic problems) with visceral (spleen and liver) and osseous involvement. This is the most common form (more than 90% of cases) and the type most commonly seen by orthopaedic surgeons. This type is also known as the adult form, but commonly, patients present during childhood (238). Type II is an acute, neuropathic type with central nervous system involvement and early infantile death. Type III is a subacute non-neuropathic type with chronic central nervous system involvement. These later two types, characterized as either infantile or juvenile, are notable for severe progressive neurologic disease and usually are fatal.

Lipid-laden histiocytes, known as Gaucher's cells, provoke clinical symptoms by their accumulation in the liver, spleen, and bone marrow. Osseous lesions result from marrow accumulation and include Erlenmeyer's flask appearance, osteonecrosis (particularly of the femoral head), and pathologic fractures, especially of the spine and femoral neck.

### Clinical Presentation

Most patients with Gaucher's disease are diagnosed before age 10 years (236). Common clinical findings include hepatosplenomegaly, yellowish pigmentation of the skin, pingueculae of the eyes (235,237), and bone lesions in 50% to 75% of patients (235). Bone pain, presenting as dull extremity ache, is present in most patients, and joint pain is equally common (228). Hemolytic anemia, leukopenia, and thrombocytopenia result from both hypersplenism and marrow replacement. These factors, along with abnormal liver function, tend to make these patients susceptible to infection and abnormal bleeding (224,225,226,227 and 228). Patients often have an increased serum acid phosphatase level and may have a decreased level of activity of glucocerebrosidase enzyme in white cells.

Bone lesions are most common in the femur, but they also occur in the pelvis, vertebra, humerus, and other locations (235). Infiltration of bone by Gaucher's cells leads to vessel thrombosis; compromise of the medullary vascular supply leads to localized osteonecrosis of the long bones (237), and avascular necrosis of the femoral head occurs in most patients in whom the disease is diagnosed in childhood (238).

Bone crisis and osteomyelitis in patients with Gaucher's disease demonstrate similar symptoms. Nearly half of patients with Gaucher's disease have episodes of bone crisis, also known as pseudo-osteomyelitis, in which they present with acute sharp pains of the extremity with associated local warmth, redness, and tenderness (239). Distinguishing this problem from osteomyelitis can be difficult. With bone crisis, a patient may have severe pain in the back or extremities, rubor, fever, and an elevated white blood cell count (239). Radiographs may show periosteal reaction or lytic lesions that are difficult to differentiate from osteomyelitis. Blood cultures are sterile, and aspiration of the affected bone is often necessary to provide correct diagnosis (224).

Osteomyelitis is present in a significant number of patients with Gaucher's disease. Hematogenous osteomyelitis was found in 10% of 49 patients in one series (224). Acute osteomyelitis is best managed conservatively when possible. Open irrigation and debridement of the bone may result in chronic osteomyelitis. Noyes and Smith (237) reviewed 18 patients with bone crisis who underwent biopsy to rule out osteomyelitis, and 61% of them went on to have postoperative osteomyelitis.

Plain x-ray studies usually are not helpful in differentiating crisis from infection (224). Technetium 99 bone scanning often demonstrates no increased uptake with a crisis (224) and shows decreased uptake in the area of osteonecrosis secondary to bone crisis, usually within 1 to 3 days after onset (225). Gallium 67 scintigraphy may be useful in differentiating crisis from infection by showing a lack of uptake in osteonecrosis and bone crisis, which may prove useful in excluding the presence of osteomyelitis (236). Bell et al. (224) recommended the use of CT scans to document the presence of purulent exudate in osteomyelitis in patients with Gaucher's disease. Recently, some authors have found MRI useful for excluding osteomyelitis in patients with Gaucher disease (237), but others (225) believe that MRI cannot distinguish between osteomyelitis and pseudo-osteomyelitis of bone crisis.

### Radiographic Findings

Radiographically, three patterns of bone involvement have been described. (a) Generalized infiltration of the bone marrow of long bones by Gaucher cells causes a decreased density and cortical thinning. There is a general tendency for failure of tubulation (229), and this problem is most pronounced in the distal femur, where it produces an Erlenmeyer's flask appearance (Fig. 6-28). (b) The bone is moth-eaten with occasional osteosclerosis, and a centrally located curvilinear or vertical radiolucent streak is seen in the distal femur. (c) Dense collections of Gaucher cells form localized bubbly expansile lytic lesions in the bone, and associated areas of aseptic necrosis with medullary infarction form diaphyseal lytic lesions with reparative periosteal reaction surrounding them (229).



**FIGURE 6-28.** A 2-year-old child with Gaucher's disease. Early flaring of the distal femur is already present and will likely develop into a classic Erlenmeyer's flask deformity. Note the moth-eaten appearance of the metaphysis (arrow).

Avascular necrosis of the femoral head is similar to that seen in Legg-Calvé-Perthes disease, and vertebral plana may be present with lesions of the spine (229). MRI is more sensitive than radiographs or CT in demonstrating marrow involvement.

### Injury

Pathologic fractures, especially of the femoral neck or shaft after biopsy and of the spine, usually are best managed conservatively. Katz et al. (232) reported 23 pathologic fractures in 9 children with Gaucher disease; 7 had multiple fractures. In decreasing order of frequency, the site of involvement included the distal femur, basilar neck of the femur, spine, and proximal tibia. Fractures also occurred infrequently in the distal tibia, proximal humerus, rib, and acetabulum. Fractures of the long bones were transverse and usually in the metaphysis. Fractures of the spine were either wedge shaped or centrally depressed at the end plate. The factors predisposing these children to fracture included significant medullary space infiltration, cortical bone erosion, osteonecrosis, and associated disuse osteoporosis.

(232).

In another report of 53 patients with Gaucher's disease aged 9 to 18 years (234), 11 had vertebral fractures, usually at two or three sites in each patient, with either anterior wedging, central vertebral collapse, or total rectangular collapse. Most patients had relief of their pain after 1 to 4 months of conservative treatment; two required decompression laminectomies, and one had a posterior lateral fusion to stabilize the spine.

Katz et al. (233) found that fractures of the upper extremities in Gaucher's disease were prone to occur in areas of prior crisis. Although external callus formed in 6 to 8 weeks in most patients, complete healing with internal callus took almost 2 years in some. Other authors have found fracture union to be rapid (229). Both delayed union and nonunion (237) have been reported in older patients with Gaucher's disease.

Pathologic femoral neck fractures with minimal associated trauma in children with Gaucher's disease often heal with a varus malunion and minimal subsequent remodeling; avascular necrosis of the femoral head also can be associated with femoral neck fractures (229,235). Goldman and Jacobs (229) stated that the presence of a mixed density of bone of the femoral neck on x-ray with narrowing of the medial cortex was a risk factor for fracture.

### **Treatment**

In the past, there was no specific treatment for Gaucher's disease. Splenectomy was often performed to help correct thrombocytopenia (228), but some authors believed that splenectomy worsened the orthopaedic complications of Gaucher's disease and recommended that it should be delayed as long as possible (332). Enzyme replacement therapy for the deficit found in Gaucher's disease has been developed (230). It is known as alglucerase (Ceredase) and is given in intravenous infusions every 2 weeks, with most patients having a decrease in the size of their livers and spleens, and improvement in their anemia. With the recommended dosage of 60 units of alglucerase per kilogram, a 30-kg child requires a yearly amount of enzyme costing \$163,800 (227). Low-dose imiglucerase (Cerezyme, Genzyme), a placental recombinant human-derived beta-glucocerebrosidase enzyme replacement, is also being used to treat patients with type I Gaucher's disease (244). Studies have shown that either a reduced dose or a low-dose-high-frequency regimen can achieve similar clinical effects with less cost (227,243). Less costly home intravenous enzyme replacement treatment is possible (245). After 1 year of treatment with replacement enzyme, patients with Gaucher's disease seem to have a decreased tendency for infection (243). The effects of the enzyme on the bone disease are unclear. Zevin et al. (242) found no change in bone appearance after 1 year of replacement therapy, whereas Hill et al. (231) noted improved appearance of the long bones and involved spine after 16 months of enzyme replacement therapy. Bone marrow transplantation also has been shown to be helpful in reversing the medical effects of Gaucher disease (241), but the mortality rate of patients undergoing allogeneic marrow transplantation is greater than 15% (227).

### **Fracture Management**

Pathologic fractures of the upper extremities and the spine respond well to conservative immobilization techniques. For fractures of the lower extremity, prolonged bed rest is to be avoided because of the additional complication of disuse osteoporosis. Early mobilization with non-weight bearing casts is necessary to avoid angulation of the fractures. Complete healing as defined by the appearance of internal callus may require from 10 to 32 weeks of immobilization. Corrective osteotomy may be necessary for residual angulation. Some authors have recommended conservative treatment with non-weight bearing for minimally angulated femoral neck fractures (228,232), with internal fixation reserved for unstable femoral fractures with progressive or marked displacement (232,235).

All patients with Gaucher's disease considered for a surgical procedure should undergo extensive preoperative evaluation of their abnormal clotting function. Excessive bleeding may even occur when clotting tests are normal (224). It is important for the anesthesiologist to recognize that patients with Gaucher's disease may be prone to upper airway obstruction because of infiltration of the upper airway with glycolipids and commonly may have an airway up to 50% smaller than predicted for age (240). These patients are prone to infection, likely due to abnormal neutrophil chemotaxis (223), and needle biopsy under operating room conditions is preferable to open biopsy (237).

## **AUTHORS' PREFERRED METHOD OF TREATMENT**

Conservative immobilization with non-weight bearing is suggested for long bone fractures when appropriate. Stable fractures of the femoral neck should be treated by guarded immobilization with frequent follow-up x-ray studies. Internal fixation should be used in femoral fractures that show signs of or potential for displacement. Preoperative planning is important, with careful evaluation of clotting function and preoperative consultation by the anesthesiologist. Femoral head osteonecrosis is managed symptomatically, at first with osteotomy or joint replacement later when necessary.

### **Sickle Cell Disease**

The term sickle cell disease (SCD) characterizes conditions caused by the presence of sickle cell hemoglobin (HbS). Variants of these conditions include sickle cell anemia (HbS-S), the less severe sickle cell disease (HbS-C), and the often asymptomatic sickle cell trait (HbA-S). The most common type of SCD, HbS-S, is a homozygous recessive condition in which individuals inherit the beta S globin gene from each parent. SCD has systemic effects particularly on splenic function and on the central nervous, renal, hepatic, and musculoskeletal systems. SCD affects approximately 1 in 400 African-Americans. Sickle cell trait affects 8% to 10% of the African-American population and other groups less frequently (267). With sickle cell trait, each individual has inherited a beta-S globin gene and a beta-A globin gene. Clinical manifestations of sickle cell trait usually are not apparent. The presence of these abnormal hemoglobins in red blood cells causes them to be mechanically fragile, and when they are deoxygenated, the cells assume a sickle shape, which makes them prone to clumping with blockage of the small vessels of the spleen, kidneys, and bones (262,263). Chronic hemolytic anemia is present in most severely affected patients, and marrow hyperplasia is found in both the long bones and the short tubular bones. These disorders are diagnosed by hemoglobin electrophoresis (252).

### **Clinical Presentation**

Musculoskeletal involvement results from small vessel occlusion by clumped sickle cells with bone infarction, avascular necrosis, and increased susceptibility to infection. These problems are most commonly seen in sickle cell anemia and sickle cell disease. Bone infarction is caused by blockage of marrow vascular channels by sickled erythrocytes. It is seen in patients as young as 6 to 12 months of age. Ultimate infarction occurs in as many as 74% of patients. Patients with acute long bone infarctions present with pain and swelling of the affected extremity and a low-grade fever. The long bone usually is tender, and infrequently, both erythema and warmth also are present (257). Acute symptoms usually resolve within a week. One presenting sign of sickle cell disease in an adolescent is an indolent ulcer over the lateral malleolus of the ankle with surrounding areas of patchy hyperpigmentation (260). Sickle cell dactylitis, or infarction of the small bones of the hands and feet, may resemble infection and is common in infancy and childhood (259). Young children getting their feet wet or walking in the snow can initiate episodes (253). Pain and swelling may last for 1 to 2 weeks. X-ray studies may show osteolysis and periosteal new bone formation.

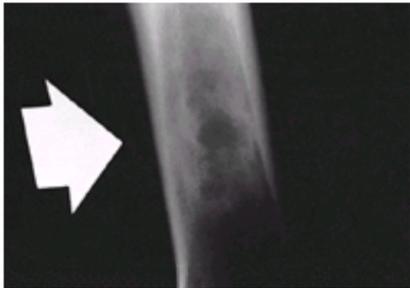
Acute bone infarct in patients with SCD may be difficult to distinguish from osteomyelitis. Osteomyelitis occurs in fewer than 1% of affected patients. Acute long bone infarctions are 50 times more common than bacterial osteomyelitis in patients with sickle cell disease (257), but in Africa, osteomyelitis is 200 times more common in patients with SCD than in normal individuals (256). Osteoarticular bacterial infection was diagnosed in 1.6% of 247 admissions in one series of children with sickle cell disease admitted to the hospital for musculoskeletal complaints (261). These authors recommended aspiration as the diagnostic procedure of choice for infection. Clinical and x-ray presentations in both of these conditions are quite similar, except that high fevers are more common in osteomyelitis. Patients present with a warm, swollen, painful extremity. Multifocal bone involvement was reported in 73% of patients with osteomyelitis in one series (265), and the most common sites in decreasing order of frequency were the humerus, metatarsals, tibia, femur, radius, metacarpals, and ulna. Routine evaluation should include blood cultures and needle aspiration of the affected area. Typical causative organisms in osteomyelitis include *Staphylococcus aureus*, *Salmonella*, and *Streptococcus pneumoniae*. Bennett et al. (247) reviewed bone and joint manifestations in 57 patients with sickle cell anemia and found that osteomyelitis occurred in 61% and that *Salmonella* was the causative organism in 71% of these patients. Others have also found that *Salmonella* is a common organism seen in osteomyelitis in SCD (256,265), but Epps and co-workers (255) found that *Staphylococcus* was the most common organism in their patients with SCD osteomyelitis. Al-Salem (246) also found that *Staphylococcus* was the most common organism in osteomyelitis in those patients with sickle cell trait. Both the metaphysis and diaphysis are common locations of infection (250,265). Septic arthritis is rare in patients with SCD. Surgical drainage of septic joints, osteomyelitis, and subperiosteal abscesses is indicated.

Other common problems in SCD include avascular necrosis of both the femoral head and proximal humerus. Wedging or flattening of the vertebral bodies is also seen in older patients (253). Fat emboli, secondary to marrow infarcts, can occur in patients with less severe SCD (HbS-C) and less frequently in patients with sickle cell

anemia. Pneumococcal sepsis is the leading cause of death in young children with SCD.

### **Radiographic Findings**

In young patients, marrow hyperplasia results in a generalized osteoporosis with widening of the medullary canal and thinning of the cortex. As the patients become older, endosteal bone apposition causes sclerosis ( [Fig. 6-29](#)) and the medullary canal may actually become narrowed with thickening of the cortex. Large bone infarctions may not be visible on plain x-ray studies because of limited circulation, but commonly within 2 weeks of onset of symptoms, areas of involvement appear moth-eaten, with irregularly distributed translucent areas commonly bound by elevated periosteum with new bone formation ( [252](#)). Widespread involvement may form a “bone within a bone” appearance ( [253](#)). Nearly 85% of long bone infarcts are found at the junction between the metaphysis and diaphysis, and 10% are centrally located ( [248](#)). Plain x-ray studies cannot distinguish between osteomyelitis and bone infarction in these patients. Although Keeley and Buchanan ( [257](#)) believe that bone scans are not helpful in the diagnosis of bone infarction, Koren et al. ( [258](#)) noted that in the first 48 hours of symptoms, there was decreased uptake in the affected area of bone infarct by technetium bone scan. Normal isotope uptake developed approximately 1 week later. They found that increased isotope uptake was common 2 to 4 weeks after the onset of symptoms. Gallium bone scan was thought to be a helpful adjunct to this technique when osteomyelitis was suspected. MRI has not been found useful in distinguishing osteomyelitis from acute infarcts ( [251](#)), but contrast material-enhanced CT scan can aid in diagnosis of osteomyelitis in these patients by its ability to visualize subperiosteal abscess ( [266](#)). Differentiation from infarction can be aided by aspiration and by comparing the results of technetium 99 scans with those of a bone marrow scan. Both partial and full femoral head involvement can occur in avascular necrosis of the capital femoral epiphysis in SCD ( [253](#)), and coxa vara also has been reported ( [259](#)).



**FIGURE 6-29.** A young man with sickle cell disease. The lateral x-ray of the femur shows evidence of past avascular necrosis, and scalloping and radiolucencies of the shaft with sclerosis are seen posteriorly ( *arrow*). This x-ray appearance can mimic osteomyelitis.

### **Injury**

Pathologic fractures of the long bones in SCD frequently may be the first symptom of the disorder ( [262](#)), and many authors report pathologic fractures in their series ( [247,250,252,254,259,264](#)). In a series of 81 patients with 198 long bone infarcts with occasional concurrent osteomyelitis, Bohrer ( [248](#)) found evidence of fracture in 25% of femoral lesions, 20% of humeral lesions, and a significant percentage also in tibial bone infarcts. Ebong ( [254](#)) reported pathologic fractures in 20% of patients with SCD and osteomyelitis. The most common site of fracture was the femur. The fractures are transverse and commonly located in the shaft of the long bone ( [253](#)), and although minimal trauma is needed to cause them ( [259](#)), they often have significant displacement ( [248,249](#)). The exact mechanism for pathologic fracture in these patients is unclear; although it is often associated with bone infarct, the fracture itself is seldom through the area of infarction ( [259](#)). Marrow hyperplasia may be a major contributing factor; not only does the hypercellular bone marrow expand the medullary canal with thinning of both trabecular and cortical bone, but it also extends into widened haversian and Volkmann canals ( [253](#)). This process probably weakens the bone sufficiently so that fractures occur. The healing process seems unaffected, and union usually occurs normally ( [262](#)).

### **Treatment**

Vaso-occlusive episodes are managed with nonsteroidal antiinflammatory medications, oxygen, and hydration. Symptomatic bone infarction should be treated with bed rest, analgesics, and intravenous or oral administration of fluids ( [257](#)). If osteomyelitis is suspected, identification of the organism should be attempted by both blood cultures and aspiration of subperiosteal fluid ( [265](#)). The choice of antibiotics is based on Gram's stain. For Gram-positive organisms, either cephalothin or nafcillin is used initially, and for Gram-negative organisms, either ampicillin or chloramphenicol is used. Changes in antibiotics are based on later culture results and sensitivities. The patient is monitored by C-reactive protein or sedimentation rate, and intravenous antibiotics usually are continued for at least 6 weeks. Operative treatment of patients with SCD is potentially hazardous. Extreme care must be taken to oxygenate the patient's tissues adequately during the procedure, and ideally, elective procedures should be preceded by multiple transfusions to reduce hemoglobin (S) to less than 30% of total hemoglobin levels. A randomized multicenter study found that a simple conservative transfusion regimen to raise Hb levels to 10 g/dL was as effective as an aggressive exchange transfusion regimen (to reduce HgS to less than 30%) in preventing perioperative complications. The conservative approach resulted in only half as many transfusion-associated complications ( [268](#)). Intravenous hydration also is important, with one and a half to two times the daily fluid requirements needed in addition to routine replacement of fluid losses. The use of a tourniquet in surgery for patients with SCD is somewhat controversial. Some authors believe that it is not indicated because it is potentially dangerous ( [253,255](#)); others found no increased risks associated with its use in these patients ( [259,267](#)).



### **AUTHORS' PREFERRED METHOD OF TREATMENT**

Pathologic fractures in patients with SCD usually heal well with conservative immobilization techniques ( [253](#)). Customary precautions should be followed in those patients who require open surgical procedures for displaced or unstable fractures. Osteonecrosis of the femoral head is an especially difficult problem in patients with SCD. Treatment options include conservative measures and core decompression (a multicenter trial is under way). Total joint replacement is occasionally indicated in young adults. Before general anesthesia, the patient's hematocrit should be raised to <sup>3</sup>30 Hb to <sup>3</sup>10 g/dL.

### **Leukemias**

Leukemia accounts for over 30% of cases of childhood cancer. Acute lymphocytic leukemia (ALL) is one of the most common malignant diseases in childhood and accounts for 80% of pediatric leukemias. There is an increased occurrence of lymphoid leukemias in patients with Down syndrome, immunodeficiencies, and ataxic telangiectasia. The peak incidence is at 4 years of age.

Leukemic involvement of bones and joints occurs frequently in patients with leukemia. Skeletal lesions occur more frequently in leukemic children than in adults because a child's small marrow reserve can be replaced quickly by leukemic cells. Approximately 50% to 75% of children with acute leukemia develop radiographic skeletal manifestations during the course of their disease ( [277,280](#)). Rogalsky et al. ( [282](#)) reported a 12% incidence of fracture associated with acute leukemic lesions.

### **Clinical Presentation**

Patients present with fatigue, pallor, purpura, fever, hepatosplenomegaly, or bone and joint pain; 20% to 60% of patients present with musculoskeletal signs or symptoms ( [275](#)). In one series, bone pain was a presenting symptom in 59% of patients ( [284,285](#)). Rogalsky et al. ( [282](#)) reported a 20.6% incidence of reports of problems with the skeletal system at initial presentation. Migratory arthritis may be present in some patients; point pain is believed to be secondary to leukemic cell infiltrate of the metaphyseal periosteum ( [285](#)). Leukemia may mimic osteomyelitis, rheumatic fever, septic arthritis, and tuberculosis ( [283](#)). Either leukocytosis or leukopenia is present, and the presence of immature leukocytes in the peripheral blood smear should suggest a diagnosis of leukemia. In the early phase of the disease, anemia, neutropenia, and thrombocytopenia occur in 80% of patients; 10% of children have normal peripheral blood counts. Bone marrow aspirate usually is

diagnostic.

### Radiographic Findings

No pathognomonic osseous manifestations occur in acute leukemia. Skeletal involvement occurs in approximately 50% of patients, and diffuse osteopenia is the most frequent manifestation. Lucencies and periostitis may mimic osteomyelitis.

Nonspecific juxtaepiphyseal lucent lines are a result of generalized metabolic dysfunction. Sclerotic bands of bone trabeculae are more typical in older children. A characteristic lesion seen within a month of onset of symptoms is a radiolucent metaphyseal band adjacent to the physis. These are usually bilateral and vary from 2 to 15 mm in width (285). Similar radiolucent bands are seen both in infants with scurvy and in older children with neuroblastoma.

Osteolytic lesions with punctate areas of radiolucency are found in the metaphyses and can either appear moth-eaten or as a confluent radiolucency. Similar lesions can be present in the diaphysis (273), the skull, pelvis, ribs, and bones of the hands and feet. Large geographic lesions also may be seen. Periosteal reaction often is present with osteolytic lesions and is most common in the posterior cortex of the distal femoral metaphysis, the medial neck of the femur, and the diaphyses of the tibia and fibula (286).

Medical management protocols usually include vincristine, prednisone, and L-asparaginase (VPL) or methotrexate, vincristine, L-asparaginase, and dexamethasone (MOAD) (272). Remission rates are now up to 98%, with cure rates approaching 80% (272).

The complication seen most commonly by orthopaedic surgeons is avascular necrosis (AVN) of the femoral head (278,287). This may occur after chemotherapy, after chemotherapy and allogenic bone marrow transplantation (BMT), or after graft-versus-host disease (GVH) related to BMT. (270) Although most AVN is attributed to glucocorticoid therapy, L-asparaginase can contribute to thrombophilia and has been implicated in the production of AVN. The risk of AVN is especially high after BMT in boys older than 16 years who are treated for GVH with steroids or irradiation. MRI is best for the early detection of AVN. Treatment modalities include weight relief, symptomatic treatment, core decompression, and total hip replacement. Bizot et al. (271) described the results in 27 patients treated with total hip arthroplasty for AVN after allogenic BMT.

Most bone lesions in leukemia improve during remission after treatment and tend to progress with worsening of the disease. The radiolucent metaphyseal bands usually are not affected by treatment, however, and further demineralization of the skeleton may occur with both corticosteroid use and methotrexate therapy. Diffuse demineralization of the skeleton occurs in almost all patients with widening of the medullary canal and thinning of the cortex (285). Although increased uptake of isotope is seen on technetium bone scan in 80% of patients, positive areas of isotope uptake correlate poorly with both sites of clinical bone pain and the presence of lesions on x-ray study (274).

Several authors (269,276,277,279) have attempted to evaluate the prognostic significance of the extent of bone involvement in childhood leukemia. Hughes et al. (278) reported that major skeletal involvement, in fact, may correlate with a better overall outcome in childhood leukemia. Heinrich et al. (277) concluded that children without radiographic skeletal abnormalities have an aggressive form of acute leukemia that results in a worse prognosis.

### Injury

Pathologic lesions predisposing children to a fracture usually resolve during treatment. Fracture is most commonly associated with osteoporosis of the spine, resulting in vertebrae plana. Fractures occasionally occur at other locations and usually after minor trauma.

Vertebral compression fractures are the most commonly reported fractures associated with leukemia. The thoracic vertebrae are the most commonly involved; uniform spinal osteoporosis often is present (278,281). A bone scan may aid in identifying clinically silent areas but may not correlate with areas of obvious destruction on radiographs. Spastic paraparesis has been reported in one patient with vertebral fracture due to leukemia (278).

### Treatment

Most fractures are treated using standard methods (Fig. 6-30). Newman and Melhorn (281) noted a prompt decrease in pain in four patients with vertebral fractures due to leukemia once chemotherapy was initiated. No bracing was used in these patients, and full activity was encouraged. In one of their patients observed for more than 5 years, there was no evidence of healing of the fractured vertebra on x-ray study. There are no specific treatment recommendations for any other associated pathologic fractures of leukemia in the literature.



**FIGURE 6-30. A:** A 13-year-old girl with acute lymphocytic leukemia presented with a pathologic fracture of the right hip. Note the destructive changes associated with the displaced femoral neck fracture. **B:** The patient subsequently underwent *in situ* screw fixation of the hip fracture with postoperative hip spica cast immobilization and subsequent continuation of chemotherapy. **C:** Follow-up x-ray studies 1 year after surgery shows healing with slight varus and without evidence of avascular necrosis. At present, the patient is alive and well, has no hip pain and walks with a very slight Trendelenburg gait.

## AUTHORS' PREFERRED METHOD OF TREATMENT

Prompt diagnosis and initiation of chemotherapy is the first step in the treatment of pathologic fractures associated with leukemia. Most fractures are stable microfractures and can be treated with conservative immobilization techniques with emphasis on early ambulation to avoid further problems with disuse osteoporosis. Most vertebral fractures can be treated nonoperatively with close observation.

### Hemophilia

Hemophilia is a sex-linked recessive disorder of clotting mechanism that presents most commonly as a functional deficiency of either factor VIII (hemophilia A) or factor IX (hemophilia B). Classic hemophilia, or hemophilia A, (factor VIII deficiency) is an inherited sex-linked recessive disorder. The incidence is 1 per 10,000 live male births in the United States (291). Christmas disease, or hemophilia B, is a sex-linked recessive factor IX deficiency and occurs in 1 per 40,000 live births.

When hemophilia is suspected, screening tests should be performed, including platelet count, bleeding time, prothrombin time, and partial thromboplastin time. Deficiency of factor VII, the most common form of hemophilia, causes a marked prolongation in the partial thromboplastin time (337). Once the disease is suspected, specific factor assays can document the type of hemophilia.

Musculoskeletal complications in a child with hemophilia include acute hemarthroses (knee, elbow, and ankle, in decreasing order of frequency), soft tissue and

muscle bleeds, acute compartment syndrome, carpal tunnel syndrome, and femoral nerve neuropraxia. The severity of the deficiency often is correlated with circulating levels of factor VIII or IX ( [Table 6-7](#)). The disease is classified as severe when clotting activity is less than 1%, moderate when clotting activity is 1% to 5%, and mild when clotting activity is more than 5%. By definition, each milliliter of normal human plasma contains one unit of factor activity, and the clinical severity of hemophilia correlates with the patient's percentage of normal levels of plasma factor activity ( [Table 6-7](#)). Early diagnosis and aggressive management are the keys to lessening complications.

Degree of Hemophilia	Percentage of Factor	Clinical Characteristics
Mild	20%–60%	Usually clinically occult, excessive bleeding after major trauma or surgery
Moderate	5%–20%	Excessive bleeding during surgery and after minor trauma
Moderately severe	1%–5%	Excessive bleeding with mild injury and infrequent spontaneous hemarthrosis
Severe	Less than 1%	Frequent excessive bleeding with trauma and spontaneous bleeding into the soft tissue and joints

**TABLE 6-7. SEVERITY OF HEMOPHILIA CORRELATED WITH PLASMA FACTOR ACTIVITY LEVELS**

### Treatment—Bleeding Episodes

During bleeding episodes, the primary therapy is intravenous replacement of the deficient factor. Several different treatment plans exist: On-demand therapy is the traditional method of hemophilia management; factor replacement is given at the first sign of a bleeding episode. Primary prophylaxis involves initiation of regular factor replacement therapy soon after the diagnosis of severe hemophilia (usually when the child is 1 to 2 years of age) with the intention of preventing joint bleeds. Secondary prophylaxis is used after a child has established a pattern of frequent bleeding but before frequent joint bleeds occur.

Empirically, one unit of factor VIII per kilogram of body weight will raise plasma activity by 2%, and a similar dose of factor IX will elevate the plasma level of that factor by 1.5% ([291](#)). The usual half-life of factor VIII varies from 6 to 12 hours and that of factor IX varies from 8 to 18 hours. Routinely in the nonbleeding patient, factor VIII must be given every 8 hours and factor IX must be given every 12 hours to maintain a stable factor level. Higher than usual dosages given more frequently are necessary in patients with active bleeding. In the past, factor replacement was accomplished through plasma transfusion, but the quantities necessary for adequate factor levels may result in circulatory overload and pulmonary edema ([331](#)). This problem was eventually solved through the use of cryoprecipitate, protein prepared from plasma that is rich in factor VIII and fibrinogen. More concentrated forms of both factor VIII and factor IX (Konyne 80) have become available. The reader is referred to an excellent review by Connelly and Kaleko ([298](#)) for a current state of the art review of gene therapy for patients with hemophilia A.

### Complications

Significant complications are caused by the use of replacement therapy in hemophiliacs. Antibodies to replacement factor, known as factor inhibitors, develop in 15% to 25% of patients with severe factor VIII deficiency (up to 50% if transient or insignificant inhibitors are included) ([291,331](#)). When inhibitors are present in quantities higher than 5 Bethesda units, very high doses of factor may be necessary to achieve significant activity levels. Many children with high titer factor VIII inhibitors are treated with daily high doses of factor VIII (immune tolerance) to reduce or eliminate the inhibitor. The level of inhibitors rises sharply 6 to 10 days after initial treatment ([301](#)), and the effectiveness of replacement therapy gradually deteriorates. In patients with hemophilia A with inhibitor levels greater than 20 Bethesda units, factor VIII replacement is ineffective. There has been some success in treating these patients with alternative therapy such as nonactivated factor IX concentrates, activated factor IX, porcine factor VIII ([340](#)), and activated prothrombin complex concentrate (Autoplex T) ([288](#)). Desmopressin (L-deamino-8D-arginine vasopressin) is becoming a treatment of choice in patients with mild hemophilia A and also has an effect on patients with von Willebrand's disease as well as platelet disorders ([401,404](#)). Patients can have an approximate threefold increase in factor VIII. Studies have shown effectiveness of this medication through administration by intravenous route or subcutaneous injections, as well as intranasal administration by spray.

A large percentage of the adult hemophiliac population treated with concentrated plasma-derived factor before 1985 became human immunodeficiency virus (HIV) positive. Because replacement therapy for 1 year (or one surgery episode) may expose hemophiliacs to the equivalent of 3,200 units of donor blood ([291](#)), these patients have been particularly susceptible to both hepatitis and HIV infection. The annual rate of hepatitis in hemophiliacs is estimated to be 5% ([294](#)). In a study of 181 patients with either hemophilia A or B, Ragni et al. ([332](#)) found that 45% were HIV seropositive, and 82% of patients treated with factor VIII concentrate were HIV positive. In this study, the peak of seroconversion occurred in 1982, with declining rates in the ensuing years, presumably owing to HIV antibody donor screening and heat inactivation of blood products. The overall incidence of acquired immunodeficiency syndrome (AIDS) for all these patients was 5.5%, but in patients who were HIV seropositive for more than 5 years, the incidence of AIDS approached 32%. All hemophilia patients are monitored for development of treatment-related viral infection; the incidence of infection today is extremely low. Serial examinations of T4 counts are performed for patients at risk. Medical therapies are still evolving for AIDS patients with hemophilia. Current gene therapy efforts are focused on developing a vector that is safe and gives long-term expression of the missing factor at levels that will significantly change the phenotype ([289](#)).

### Surgery in Hemophilia

In preparing a patient with hemophilia for surgery, the orthopaedist and the hematologist should work closely together. Preoperatively, the patient should be tested for the presence of inhibitor and a test dose of factor replacement should be given to determine the biologic half-life of that factor for that particular patient ([291](#)). Elective surgery usually is contraindicated in the presence of inhibitor. Most authors recommend a level of factor activity during surgery ranging from 70% to 100% ([291,317,331](#)), although others believe that approximately 50% is adequate ([328,330](#)). The initial dose is usually 40 units/kg ([331](#)), and it should be given 1 to 2 hours before surgery ([308,317](#)). In prolonged procedures with active bleeding, the factor level should be checked as often as every 3 hours and appropriate factor supplementation given as necessary ([331](#)). Tourniquets are recommended for extremity surgery. Although some authors believe electrocautery is adequate for obtaining hemostasis ([308,317](#)), others consider ligation of vessels preferable ([291,331](#)). The use of routine drains is not advised, but 24 hours of suction drainage is favored by some ([291,317,331](#)). Factor levels are checked immediately after surgery and then at least daily. Factor VIII is given every 6 hours, and factor IX is given every 8 hours. It is useful to check a trough level factor activity immediately before the next dose of factor supplementation. In the immediate postoperative period, factor levels are maintained at 30% to 40% ([291,317](#)), and these levels should be maintained until sutures are removed ([317](#)). During the rehabilitative period, maintenance levels of factor ranging from 20% to 50% immediately before sessions of physical therapy should be maintained ([291,317,328,331](#)). Intramuscular injections of analgesics should obviously be avoided, as should aspirin compounds and nonsteroidal antiinflammatory medications that affect platelet function. Both acetaminophen (Tylenol) and codeine medications are safe oral analgesics ([312](#)). In the past, hemophiliac patients had an increased risk of operative infections and delayed wound healing, but aggressive replacement therapy has minimized those problems ([331](#)).

### Injury

#### Fractures and Dislocations

**Clinical Presentation.** Most, but not all authors, believe that hemophiliac patients have an increased incidence of fracture ([290,301,308](#)). These patients have been predisposed to fracture because of poor muscle function, limitation of joint movement, and osteopenia secondary to recurrent joint hemarthrosis. Most authors have noted that healing of fractures proceeds primarily with endosteal callus and very little periosteal callus ([301,313,339](#)), but Lancourt et al. ([317](#)) observed significant periosteal calcification in these fractures with a normal rate of healing. Fractures occur in both the upper and lower extremities ([290,295,301,313,333](#)). Joint dislocations are rare in hemophiliac patients. Floman and Niska ([302](#)) reported on a 6-year-old boy who sustained a posterior dislocation of the hip with mild trauma that required a closed reduction under general anesthesia and immobilization in a hip spica cast. The joint was found to be ankylosed at 6-year follow-up. Ackroyd and Dinley ([289](#)) reported on two patients who had their patellas locked into the intercondylar notch of the distal femur after sustaining hyperflexion injuries of the knees, which had limited range of motion owing to arthropathy. These injuries were treated by flexion of the knee under general anesthesia, depression of the inferior

pole of the patella to unlock it, and then extension of the knee followed by splinting.

**Treatment.** Most fractures in hemophiliac patients are treated conservatively with immobilization. Factor replacement is important for about the first week after the fracture, and levels of factor activity recommended vary from 20% to 50% (290,291,295,301,308,317). Circumferential plaster casts are extremely hazardous in the treatment of these fractures because of the risk of swelling from bleeding as well as subsequent compartment syndrome and skin necrosis (338). A Robert Jones dressing may be preferable for fracture immobilization immediately after injury, and a cast should be applied once active swelling has stopped (308). All casts applied should be well padded and split, and the patient should be monitored carefully for swelling. Fractures of the femur can be treated with traction and subsequent spica casting (295). Some authors consider skeletal traction to be hazardous because of the risk of infection or bleeding (291,308), but Boardman and English (295) believe that with proper replacement therapy, skeletal pins can be used in the hemophiliac. In the presence of inhibitor, a large blood loss owing to fracture should be treated with transfusion with saline-washed packed red blood cells (313). Replacement therapy is advisable while fractures are manipulated and casts are changed. Most authors think that open reduction and internal fixation should be performed in hemophiliac patients for fractures that would customarily be treated with such methods (291,295,317).

### Muscle Hematoma

**Clinical Presentation.** Hematomas of the soft tissues in hemophiliacs occur in superficial tissues but are more of a clinical problem when they develop in muscle (317). Although most are spontaneous, a history of trauma was noted in 24% of 178 episodes of muscle hematoma in one series (292). In order of frequency, the most common sites of involvement in that series were the quadriceps, calf, anterior compartment of the leg, thigh adductors, hamstrings, and sartorius muscle. The first clinical symptoms are tenderness, stiffness, and swelling of the involved muscle group with pain on motion (292). Early volar compartment syndrome of the forearm, which responded to factor replacement, has been reported in a young child (322), and before the advent of replacement therapy, Volkmann ischemic contracture of the forearm could evolve from this injury (291). A hematoma in the iliopsoas muscle can present as severe groin pain, flexion deformity of the hip, and a tender mass palpable along the iliac crest. Passive extension of the hip increases pain, and significant swelling of the muscle can cause compression of the femoral nerve (with subsequent femoral nerve palsy and quadriceps muscle paralysis) by the inguinal ligament superiorly and the iliopsoas ligament medially. X-ray studies usually are not helpful in the diagnosis of intramuscular hemorrhage in the hemophiliac. Ultrasound (316), CT scan (336), and MRI are useful in documenting the presence of muscle hematoma. Wilson et al. (341) noted that in early hemorrhage of the muscle, the ultrasound shows increased echogenicity, but in established muscle hematoma, the echogenicity is decreased. This may be helpful in recurrence, because fresh hemorrhage into an organizing hematoma can be distinguished by this technique. One should not assume that all groin pain in hemophilia is due to an iliopsoas muscle hematoma. Although rare, hip hemarthrosis can occur, and septic arthritis of the hip has also been reported in hemophiliac patients. In this clinical situation, a hip ultrasound and aspiration can help make the correct diagnosis (341).

**Treatment.** The most important consideration in the treatment of a patient with muscle hematoma is early initiation of replacement therapy. Aronstam et al. (293) found that if replacement therapy was initiated within 2 hours of onset of symptoms, then excellent recovery ensued. The only exception to this finding was hematomas of the calf muscles, which responded equally well to replacement therapy if it was treated within 3 hours of the onset of symptoms. Most authors recommend raising factor levels to between 20% and 50% (291,292,308,320,325) and continuing treatment for 24 hours (292) to 5 days (317). The patient should not bear weight on the extremity (292), and the affected joints should be placed in a position of comfort, with a compression dressing and ice packs applied to the swollen muscle (292,308). Serial casting may be useful in regaining joint motion after active bleeding has ceased (308), and light traction may be useful for regaining hip motion in patients with iliopsoas hematoma. Quadriceps muscle function usually returns with recovery from quadriceps hematoma. Infrequently, ectopic bone may form in the soft tissues after hematoma (309). In severe cases of muscle hemorrhage that do not respond to conservative treatment, fasciotomy and neurolysis (with proper replacement therapy) may be necessary (317).

### Neurapraxia

In addition to the compression of the femoral nerve seen in iliopsoas hematoma, neurapraxia in hemophiliacs can occur in the peroneal, sciatic, median, and ulnar nerves (291). Carpal tunnel syndrome due to hemorrhagic compression of the median nerve has been reported (317,326). Factor levels for these patients should be raised to 80% to 100% of normal for 2 days and then maintained at 40% of factor levels for another 7 days with splinting of the extremity (291). Another neurologic complication in patients with hemophilia is significant intracranial bleeding from minor head trauma. This has been reported in 2% to 13% of children who have hemophilia and von Willebrand's disease (299).

### Pseudotumor of Hemophilia

**Clinical Presentation.** The pseudotumor of hemophilia is a cystic swelling of the muscle due to hematoma. Adjacent bone erosion is often evident on plain x-ray study and is seen most frequently in the ilium and femur (335). In children, a pathologic fracture can occur after a destructive pseudotumor of the femoral shaft. Pseudotumors can also evolve after fracture of the femur (296). These lesions may develop through subperiosteal hemorrhage, which causes pressure necrosis of the overlying muscle and underlying bone (339). Progressive enlargement can compress surrounding vital soft tissue structures, and extreme enlargement may eventually result in skin perforation, with infection and possible death (296). In children, the peripheral skeleton is most commonly affected by pseudotumor (308), and the small bones in the hands and feet are the most common sites; the prognosis is better with these sites than with more centrally located pseudotumors (290). Large pseudotumors may develop calcific deposits that are visible on plain x-ray studies. An established cyst may be associated with semilunar struts of bone projecting from the adjacent bone at the proximal and distal ends of the pseudocyst (296). With involvement of the small bones of hands and feet, interosseous expansile lesions with surrounding periosteal elevation are seen (317). The CT scan is useful in delineating the extent of pseudotumor (329).

**Treatment.** Aspiration is contraindicated in treatment of pseudocysts because not only does the needle track fail to heal but hematoma soon recurs, with the possibility of infection and bleeding from the needle wound (291,296,325,339). Very early treatment of small pseudotumors with replacement therapy, compression dressings, and prolonged immobilization may arrest their development (290,308,315,317). Factor levels of 50% were used in one series (290). Radiation therapy has been used to treat pseudotumors of hands and feet successfully but should be discouraged in children (368,375,376,400). Surgical excision of the pseudotumor may be necessary if the diagnosis is in question or if the lesion is enlarging with danger of skin perforation (291,297). Malignant degeneration is unlikely, and only one case of fibrous sarcoma has been associated with a pseudotumor of the chest wall (319). Surgical removal of a pseudotumor is quite demanding, and often residual cyst must be left behind when it is connected with vital structures. Amputation may be necessary if the lesion is complicated by infection (317).

### Hemarthrosis

**Clinical Presentation.** Patients with severe hemophilia have a high rate of hemarthrosis. The joints most commonly involved in decreasing order of frequency include the knees, elbows, ankles, hips, and shoulders (291). There usually is a prodrome of stiffness and pain before clinical swelling, and trauma usually is absent. The joint is held in a position of flexion that is most comfortable, and eventually, the joint becomes tense and swollen with decreased range of motion. A subacute hemarthrosis of the knee is said to be present when two or more episodes of acute hemarthrosis have preceded it. On clinical examination, the synovium is very thick and boggy with decreased range of motion of the joint. Pain is uncommon. In chronic hemarthrosis, generally a subacute arthropathy has been present for at least 6 months, and destructive changes are present on the x-ray studies of the knee with osteoporosis, overgrowth of the epiphysis with subchondral cysts, and eventual narrowing of the joint space (Fig. 6-31) (291). MRI was found to be useful in examining both the hypertrophied synovium in hemophilia arthropathy as well as subchondral cysts (311,327). Various systems of classification of joint arthropathy of the knee in hemophilia have been proposed (291,306). The chronic phase of articular involvement for patients with hemophilia can lead to articular cartilage degeneration from recurrent bleeds and effusions. Initially, synovial hypertrophy and chronic hyperemia occur, followed by epiphyseal overgrowth. Articular involvement may be graded as shown in Table 6-8. A diagnostic dilemma that can occur is septic arthritis in HIV-positive hemophiliacs with existing joint effusion. Merchan et al. (324) reported four patients with this complication, of which one was a 15-year-old boy with a septic elbow due to *Streptococcus pneumoniae* that was treated with 3 weeks of penicillin G therapy.



**FIGURE 6-31.** A 22-year-old man with hemophilia had frequent hemarthrosis of the knee. Moderately severe arthropathy is present, with joint space narrowing, spurting, and subchondral cyst (*arrow*).

Grade 1	Transitory synovitis; no bleeding sequelae, and with no more than three episodes in 3 months
Grade 2	Permanent synovitis with increased joint size, synovial thickening, and limitation of movement
Grade 3	Chronic arthropathy with axial deformity and muscular atrophy
Grade 4	Ankylosis

**TABLE 6-8. GRADES OF ARTICULAR INVOLVEMENT**

**Treatment.** The first step in the treatment of joint hemarthrosis is prompt factor replacement with levels ranging from 30% to 50% ([291,307,334,337](#)), and these levels may need to be maintained for up to 4 days for severe cases ([307](#)). Home care factor replacement provides a means of rapid initiation of therapy ([291](#)). With effective treatment, usually prompt pain relief is achieved in minutes after initiation of replacement therapy, and the extremity should be splinted and ice should be applied to the joint ([291](#)). In acute hemarthrosis of the knee with a very tense, swollen joint that is not responding well to replacement therapy, early aspiration may be of some value. For aspiration to be successful, a large needle such as a 16-gauge needle must be used ([317](#)), and aspiration must be accomplished within 24 hours of the onset of swelling because the clot begins to loculate after that time ([308](#)). Factor replacement must not be given before the aspiration because this will promote clotting of the hemarthrosis fluid and prevent removal; ideally, therapy is initiated at the same time as aspiration ([320](#)). The value of aspiration is uncertain because there was no difference in the range of motion between aspirated and unaspirated knees at 5-day follow-up in one controlled trial ([310](#)). In addition to factor replacement, splinting, ice packs, and compression bandages are helpful ([317](#)).

Intraarticular dexamethasone and nonsteroidal antiinflammatory drugs (NSAIDs) are used by some. A short course of corticosteroids seems to reduce the need for long-term replacement therapy ([314](#)). In subacute hemarthrosis, aspiration is not necessary, and factor replacement with levels from 20% to 30% is used three times weekly to protect the patient from further bleeding during physical therapy sessions ([300](#)). Mobilization should continue for 3 to 4 weeks, and if there is no response to therapy, synovectomy may be indicated ([317](#)). A target joint is defined as one with four bleeding episodes in a 6-month period. Treatment involves regular replacement therapy for at least 6 to 12 weeks to interrupt bleeding cycles. Replacement therapy can include recombinant or plasma-derived factor; most patients use recombinant factor. In general, 1 unit per kg of body weight of factor VIII concentrate provides a 2% increase in the factor VIII plasma activity. Today, acute joint bleeds are treated with a more intensive therapy than was used previously ([334,337](#)).

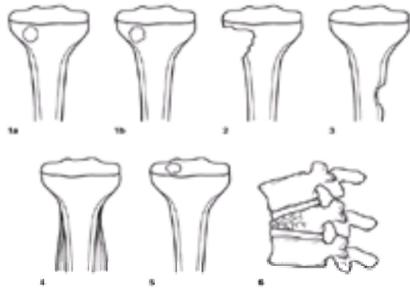
Management of the patient with chronic articular involvement is aimed at stopping this phase with a prophylactic factor replacement regimen ([334,387](#)). Greene et al. ([304](#)) reviewed their experience with prophylactic transfusions for hypertrophic synovitis in 19 children with severe hemophilia and found mixed results. Persistent, painful, or progressive synovitis can be managed with open or arthroscopic synovectomy, especially for the knee and elbow of those with breakthrough bleeds. Greene ([305](#)) reported good results in five children who underwent synovectomy of the ankle with a decreased rate of hemarthrosis episodes. Chemical (Rifampicin) or radioactive (Au-198) synovectomy has also been used. Erken ([300](#)) reported 35 patients who underwent medical synovectomy by intraarticular injection with the radiocolloid yttrium-90 silicate. Of these patients, 21 were younger than 10 years, and at a mean follow-up of 7 years, 47 joints were pain free and 13 joints had not experienced another hemorrhagic episode. There was no change in the radiographic appearance of the joints at follow-up. Erken suggested that this form of treatment is for patients who have failed to respond to intensive physical therapy as well as those patients who have inhibitors to factor replacement. Fixed joint contractures can be treated with reversed dynamic slings ([308,338](#)) or a turnbuckle-type cast ([291](#)). Slow continuous passive motion may be useful after surgery of the knee in hemophiliacs ([303](#)). Joint contractures resistant to these conservative therapies can be treated with either soft tissue procedures, osteotomies, arthrodesis, or joint replacement ([291](#)). Total joint arthroplasty may be helpful in selected candidates.

## AUTHORS PREFERRED METHOD OF TREATMENT

Collaboration between the orthopaedist and the hematologist is important in providing care for children with hemophilia. Most fractures in children with hemophilia can be treated with either traction or cast techniques. Care must be taken to avoid complications related to compression in these patients, and a monovalved, well-padded plaster cast provides a safe means of treatment. A fiberglass cast is not as desirable because a simple monovalve will fail to expand the cast completely. Operative treatment should be reserved for fractures that normally require surgery, and the usual precautions for hemophiliac patients for surgery are observed. Muscle hematomas are treated with a combination of replacement therapy, ice packs, bed rest, and a particular emphasis on rehabilitation. CT scans are extremely helpful in following the course of an iliopsoas muscle hematoma. Pseudotumors of the small bones of the hands and feet can be treated with excision and bone grafting. Hemarthrosis of the knee responds well to prompt factor replacement therapy and immobilization; aspiration is seldom needed. The orthopaedic surgeon caring for patients with hemophilia must be ever mindful of the possible presence of both hepatitis and HIV, and the utmost care should be used in any sort of invasive procedure with these patients.

## OSTEOMYELITIS

The character of pediatric acute hematogenous osteomyelitis in North America has changed somewhat during the past several decades. Although the typical clinical picture of acute osteomyelitis in children is still seen, more subtle presentations are more frequent. This may be due to a variety of reasons, including modification of the clinical course by antibiotics given before admission ([351](#)) and, possibly, increased awareness and an earlier presentation to a medical facility resulting in earlier diagnosis. Children often present with subacute osteomyelitis ([Fig. 6-32, Table 6-9](#)). Less common variants include Brodie's abscess, subacute epiphyseal osteomyelitis, viral osteomyelitis ([365](#)), and chronic recurrent multifocal osteomyelitis ([354](#)). Some patients present with a bone lesion that may be confused with other disease entities, including neoplasm ([345](#)). All can masquerade as osteomyelitis with fever and tenderness over areas of focal leukemic bone destruction ([366](#)). Biopsy often is needed to clarify the diagnosis. Even with appropriate antibiotic therapy, some patients have recurrent infection, growth disturbance, or pathologic fractures.



**FIGURE 6-32.** Classification of subacute osteomyelitis. (From Dormans JP, Drummond DS. Pediatric hematogenous osteomyelitis: new trends in presentation, diagnosis, and treatment. *J Am Acad Orthop Surg* 1994;2:333–341.)

Presentation	Subacute	Acute
Pain	Mild	Severe
Fever	Few patients	Majority
Loss of function	Minimal	Marked
Prior antibiotics	Often (30%–40%)	Occasional
Elevated WBC	Few	Majority
ESR	Majority	Majority
Blood cultures	Few positive	50% positive
Bone cultures	60% positive	85% positive
Initial x-ray study	Frequently abnormal	Often normal
Site	Any location (may cross physis)	Usually metaphysis

From Dormans JP, Drummond DS: Pediatric hematogenous osteomyelitis: New trends in presentation, diagnosis, and treatment. *J Am Acad Ortho Surg*. 2:333–341, 1994.

**TABLE 6-9. COMPARISON OF ACUTE TO SUBACUTE OSTEOMYELITIS**

### Definitions

Acute hematogenous osteomyelitis can be classified by age (neonatal, childhood, and adult osteomyelitis), organism (pyogenic and granulomatous infections), onset (acute, subacute, and chronic osteomyelitis), and routes of infection (hematogenous and direct inoculation). Chronic osteomyelitis is defined by most authors as osteomyelitis with symptoms that have been present for longer than 1 month.

### Clinical Presentation

Most patients with acute hematogenous osteomyelitis present with fever, pain, and localized tenderness at the site of infection. In order of decreasing frequency, the most commonly involved bones include the femur, tibia, humerus, fibula, radius, phalanges, calcaneus, ulna, ischium, metatarsals, and vertebral bodies. Although the erythrocyte sedimentation rate is commonly elevated in osteomyelitis, the white blood count is normal in 40% to 75% of patients (353). Blood cultures are positive for the infecting organism in 40% to 50% of patients (364), and direct cultures of the site of infection are positive in no more than 70% (353). The most common organism in hematogenous osteomyelitis is *Staphylococcus aureus*. In very young children, both *Haemophilus influenzae* type b and *Streptococcus pneumoniae* have a significant prevalence (353), but *Haemophilus influenzae* has virtually been eliminated as a musculoskeletal pathogen by immunization. Table 6-10 shows the most common organisms affective individuals by age.

Patient Type	Probable Organism	Initial Antibiotic
Neonate	Grp. B Strep, <i>S. aureus</i> , Gram-negative rods ( <i>H. influenzae</i> )	Cefotaxime (100–120 mg/kg/24 h) or oxacillin and piperacillin (5–7.5 mg/kg/24 h)
Infants & children	<i>S. aureus</i> (90%) if allergic to penicillin* if allergic to penicillin and cephalosporins*	Oxacillin (150 mg/kg/24 h) Cefazolin (100 mg/kg/24 h) Clindamycin (25–40 mg/kg/24 h) or Vancomycin (40 mg/kg/24 h)
Sickle cell disease	<i>S. aureus</i> or <i>Salmonella</i>	Oxacillin and ampicillin or chloramphenicol or cefotaxime (100–120 mg/kg/24 h)

\*Overall 80% due to *S. aureus*

**TABLE 6-10. INITIAL ANTIBIOTIC THERAPY FOR OSTEOMYELITIS**

### Radiographic Evaluation

Only 20% of patients have plain x-ray findings of osteomyelitis within 10 to 14 days after onset of symptoms; the earliest finding is loss of defined deep soft tissue planes (353). Because of this early insensitivity of plain x-ray studies, isotope scanning techniques have been used to aid in diagnosis with varying rates of success. In proven osteomyelitis, abnormal technetium scans are seen in 63% to 90% of patients (352,358). In one study (358), gallium scans were abnormal in all patients with osteomyelitis, but they were also abnormal in 30% of patients without a bone infection. In neonates with osteomyelitis, only 31.5% had abnormal technetium bone scans in one series (342). Although somewhat technically difficult, indium-labeled leukocyte scans are usually abnormal at sites of osteomyelitis. CT scans have proved to be helpful in the evaluation of primary epiphyseal bone abscess (343). MRI is becoming the study of choice for defining the stage and extent of osteomyelitis, after plain x-ray studies (350). Bone infection can be identified by soft tissue changes through MRI techniques in 92% of patients, but the presence of prior surgery or coexisting fractures affects the accuracy of this study (344).

### Injury

In 1932, Capener and Pierce (347) reviewed 1,068 patients with osteomyelitis and found only 18 pathologic fractures, 13 of which occurred in the femur. They thought these fractures were due to delayed recognition of the infection or inadequate treatment. Other factors were disuse osteopenia, presence of a weak involucrum, and excessive surgical removal of involved bone. In this preantibiotic era, most of these fractures were sustained after surgical treatment of the osteomyelitis, and the authors believed that conservation of the involucrum and proper immobilization could have prevented these injuries. White and Dennison (371) noted that before antibiotics, pathologic fractures of osteomyelitis were common, but union occurred with certainty in the presence of dense involucrum. Daoud and Saighi-Bouaouina (348) reported on 34 patients with hematogenous osteomyelitis complicated by pathologic fracture, pseudarthrosis, or significant segmental bone loss. The tibia was affected in 24 cases, the femur was affected in 8 cases, and the humerus was affected in 2 cases. A pathologic proximal femoral fracture has been reported in neonatal osteomyelitis (342). Although the condition is rare, hematogenous osteomyelitis can also evolve at the site of a closed fracture from 1 to 6 weeks after injury (346,355,363). Canale et al. (346) reported three children with osteomyelitis after closed fracture. They pointed out that progressive pain and swelling at a fracture site during healing are suggestive of possible osteomyelitis. Daoud et al. (349) reported 35 children with upper femoral osteomyelitis with associated septic arthritis. The incidence of AVN of the femoral head was approximately 50% both in the group that was treated with arthrotomy and in the group in which no surgery had been done. They postulated that AVN of the femoral head may be due to compression by abscess of the vessels lying on the posterior superior femoral neck. The complications

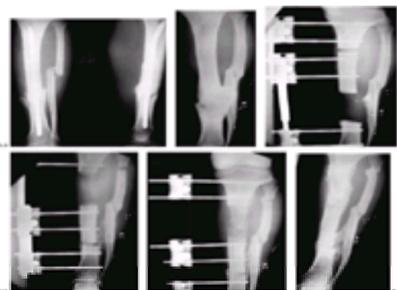
of fracture, dislocation, and displacement of the capital femoral epiphysis occurred in two thirds of their patients, and these usually were patients who presented long after an acute phase of the disease. They recommended surgical drainage of septic hips, and reduction and stabilization of hips with AVN using skin traction and plaster immobilization for 40 to 60 days. Lewallen and Peterson (357) documented osteomyelitis in 40% of 20 nonunions of diaphyseal open fractures in children. They believed that the presence of infection was a significant factor in their failure to heal. Long bone lesions also may be present in congenital rubella (361), and pathologic fractures have been reported in congenital cytomegalic inclusion disease (367).

### Treatment

Once osteomyelitis is clinically suspected, needle aspiration of the area of maximal tenderness of the long bone is recommended to obtain the causative organism either from subperiosteal fluid or from the metaphysis directly. If gross purulent material is obtained, surgical drainage should follow (353). Ultrasound or MRI may be helpful in evaluating equivocal cases. With the increased concern about medical economics, recent trends to decrease the duration of intravenous antibiotic treatment of these infections appear to be appropriate as long as certain criteria are met. If oral antibiotics are inappropriate or the organism is not isolated by culture, then longer term parenteral antibiotics must be considered. Total length of therapy is based on the type of organism isolated and the patient's response to treatment. Unkila-Kallio et al. (370) found that in acute hematogenous osteomyelitis in children, the serum C-reactive protein correlated more closely than erythrocyte sedimentation rate to the clinical course of osteomyelitis, with rapid elevation at the onset of disease and a significantly faster decline with effective treatment than the erythrocyte sedimentation rate.

### Fracture Management

Fractures associated with osteomyelitis may be difficult to treat and may be associated with complications such as malunion and growth disturbance (359). Pathologic fractures associated with osteomyelitis are rare in North America and usually are associated with neglected or chronic osteomyelitis, neonatal osteomyelitis, or septic arthritis. In children with chronic osteomyelitis, the purulent material elevates the periosteum and a supportive involucrum develops. Sequestrectomy of a portion of the necrotic diaphysis while leaving the supportive involucrum is often needed to bring the infection under control, but the timing of this procedure is controversial. Langenskiold (356) delayed sequestrectomy of a necrotic femoral shaft for 10 months in a 6-year-old patient to allow the involucrum to develop, but Daoud and Saighi-Bouaouina (348) recommended much earlier debridement. In patients with active infection, they performed sequestrectomy with debridement, followed by antibiotic therapy for up to 6 months. Prolonged cast immobilization was necessary. They obtained healing in 33 of 34 patients with pathologic fractures or pseudarthroses due to osteomyelitis. The mean healing time of fractures was 5 months in patients with involucrum. Patients with active infection without involucrum required debridement, antibiotics, and subsequent treatment with corticocancellous iliac graft. The mean healing time was 8.7 months. Their patients without active infection and no involucrum were treated with prolonged immobilization and cancellous bone graft supplemented by fixation. Angular deformities were treated with cast manipulation. Tudisco et al. (369) reported on 26 patients with chronic osteomyelitis with average follow-up of 23 years. Approximately 15% had shortening and angular deformity of the affected limb. Newer techniques for difficult cases have also been developed (Fig. 6-33).



**FIGURE 6-33.** A 20-year-old patient presented with recalcitrant chronic draining osteomyelitis of the left tibia 4 years after a grade 3A open tibia fracture treated with an intramedullary nail. **A, B:** X-ray studies at presentation show healing of the fracture but changes consistent with chronic osteomyelitis. **C:** X-ray studies after resection of the involved segment of bone and sequestrum, with early bone transport from above and below a corticotomy. **D:** Early regenerate is seen at the time of docking of the transported segment and the distal tibia. **E:** Healing at the docking site is seen after posterolateral bone grafting. **F:** X-ray studies 6 years after the resection and bone transport showing healing and remodeling of the tibia. The patient has returned to work, is free of pain, and has no signs of active infection.

Pathologic fractures also can occur in association with advanced or neglected septic arthritis in older children and in association with neonatal osteomyelitis and septic arthritis. Infants with septic arthritis, especially of the shoulder or hip, present with pseudoparalysis of the extremity. In the upper extremity, this can be confused with Erb's palsy. Plain x-ray studies may show only soft tissue swelling and equivocal widening of the joint (362); technetium bone scans often are unreliable in neonates (362,368). Ultrasound and MRI can both be helpful; aspiration should be done if clinical suspicion exists. Schmidt et al. (362) found that more than half of their patients with septic arthritis of the shoulder also had osteomyelitis, and they recommended arthrotomy of the shoulder with drilling of the proximal humeral metaphysis if aspiration of the joint was positive.

### AUTHORS' PREFERRED METHOD OF TREATMENT

With early recognition and appropriate treatment, osteomyelitis leading to pathologic fracture is uncommon. When osteomyelitis is associated with pathologic fracture, it usually is neglected chronic osteomyelitis, or rarely, neonatal osteomyelitis or septic arthritis. The most important step in the treatment of fracture associated with osteomyelitis is to control the underlying infection. At a minimum, this requires drainage and debridement of the infection with immobilization in association with antibiotic therapy. In advanced infections, sequestrectomy may be necessary. MRI is useful in identifying the sequestrum; an attempt should be made to leave as much supporting involucrum as possible at the time of sequestrectomy. Bone transport and lengthening also are occasionally useful. Prolonged immobilization with either plaster casts or external fixation devices may be needed, and segmental bone loss can be treated with bone transport or grafting.

### PATHOLOGIC FRACTURES AFTER LIMB LENGTHENING

Limb lengthening has evolved dramatically over the past several decades. Surgeons experienced with lengthening techniques can now correct problems that previously had no satisfactory solution. The very high complication rate that has come with these advances has decreased with newer techniques and more extensive surgical experience. Complications with the Wagner method, popular 20 to 30 years ago, were as high as 92% (375,377). Newer techniques, using gradual lengthening with either monolateral fixators or fine wire fixators such as the Ilizarov fixator, have decreased the complication rate.

Fractures that occur in association with limb lengthening fall into three general categories: (a) fractures through pin tracks, (b) fractures through regenerate bone, or (c) fractures through bone weakened by disuse osteoporosis. Fractures that occur through holes left after removal of screws or fine wires generally occur a few weeks after device removal. The incidence of these fractures can be minimized by protective weight bearing after removal of the device and using the smallest possible screw diameter that is appropriate for the fixation device needed.

Fractures through regenerate bone are true pathologic fractures. The bone that is formed by distraction callotaxis must be subjected to normal weight-bearing forces over a period of time before normal bony architecture is established. Fractures that occur through the lengthening gap can occur soon after removal of the fixator or years later. Various reports describe fractures through regenerative bone occurring as late as 2 to 8 years after lengthening (376,377 and 378). The incidence of fractures has been reported to be as high as 18% for Wagner lengthenings but only 3% for newer techniques (373,374,375,378,379 and 380). At present, most lengthenings are performed through the metaphysis, which has a larger bone diameter and better blood supply than the diaphysis, where Wagner lengthenings were done. When fractures occur in regenerate bone, they can be treated with simple cast immobilization. However, because this method further promotes osteopenia, many surgeons reapply a fixator, correct any malalignment caused by the fracture, and compress at the fracture site until healing. To ensure that the regenerate bone can bear the forces of normal activity, a variety of imaging methods have been used (372,376). When the regenerate bone attains the density and ultrastructural

appearance (development of the cortex and the medullary canal) of the adjacent bone, fixator removal is generally safe.

Pathologic fracture also can be caused by the osteopenia and joint contractures that can occur after months in an external fixation device. Some children, because of pain or anxiety, are reluctant to bear sufficient weight on their fixator devices, putting them at risk for disuse osteoporosis. Joint contractures can be related to either the lengthening itself or insufficient rehabilitation during and after lengthening. Many of the fractures due to these causes are avoidable; when they do occur, appropriate immobilization or internal fixation is used.

## FRACTURES IN CONDITIONS THAT WEAKEN BONE

### Osteogenesis Imperfecta

Osteogenesis imperfecta (OI) is a heterogeneous group of inherited disorders in which the structure and function of type I collagen is altered. The fragile bone is susceptible to frequent fractures and progressive deformity (390,405). OI is identifiable in 1 in 20,000 total births, with an overall prevalence of approximately 16 cases per million index patients (405,423). The wide spectrum of clinical severity—from perinatal lethal forms to clinically silent forms—reflects the tremendous genotypic heterogeneity (more than 150 different mutations of the type 1 procollagen genes *COL1A1* and *COL1A2* have been described). As the molecular basis of this continuum of severity is further elucidated, the phenotypic groupings of the various classifications and subclassifications may seem arbitrary. However, these classifications facilitate communication, predict natural history, and help the clinician plan management strategies (405). From a practical viewpoint of orthopaedic care, patients with OI can be divided into two groups. One group of patients with severe disease develops long-bone deformity through repetitive fractures, eventually requiring open treatment with intramedullary fixation. Another group of patients has mild disease with frequent fractures, but their injuries usually respond well to closed methods of treatment and there is less residual deformity.

### Clinical Presentation

Children with severe OI may present with a short trunk, marked deformity of the weight-bearing lower extremities, prominence of the sternum, triangular facies, thin skin, muscle atrophy, and ligamentous laxity; some develop kyphoscoliosis (386,397,410), basilar impression (408,417), and deafness (due to otosclerosis) (398). Despite this multitude of physical problems, children with OI usually have normal intelligence. Blue sclera, a classic finding in certain forms of OI, can also be present in normal infants, as well as in children with hypophosphatasia, osteopetrosis, Marfan's syndrome, and Ehlers-Danlos syndrome (402). Histologic findings in severe cases reveal a predominance of woven bone, an absence of lamellar bone, and thinning of the cortical bone with osteopenia.

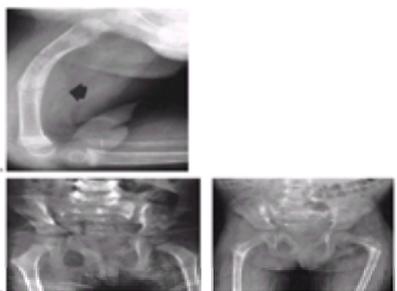
Patients with OI may present with swelling of the extremity, pain, and low-grade fever and a radiograph showing exuberant, hyperplastic, callus formation (Fig. 6-34). The callus may occur without fracture and can have a distinct butterfly shape (402), as opposed to the usual fusiform callus of most healing fractures. The femur is most commonly involved, but cases noting involvement of the tibia and humerus have been reported (416). The sedimentation rate and serum alkaline phosphatase may be elevated. Because osteosarcoma has been associated with OI (401,403), aggressive-appearing lesions may occasionally require biopsy to confirm their benign nature.



**FIGURE 6-34. A:** This 10-month-old boy with a history of osteogenesis imperfecta presented with a right thigh pain and swelling and refusal to bear weight. This AP radiograph and **(B)** this lateral radiograph of the right femur show the extraordinarily abundant, hyperplastic callus—with the characteristic butterfly shape—that can occur in osteogenesis imperfecta. This appearance may be mistaken for an infection or a neoplastic process.

### Radiographic Findings

Radiographic findings vary. In severe involvement, there is marked osteoporosis, thin cortical bone and evidence of past fracture with angular malunion (Fig. 6-35). Both anterior and lateral bowing of the femur and anterior bowing of the tibia are common. The long bones may be gracile with multiple cystic areas. Spinal radiographs may show compression of the vertebrae between the cartilaginous disk spaces (so-called codfish vertebra). The presence of wormian bones on a skull radiograph is relatively specific for OI. Subsequent development of multiple pathologic fractures with callus and deformity firmly establishes the diagnosis (Fig. 6-36).



**FIGURE 6-35. A:** X-ray study of a nondisplaced transverse femoral shaft fracture (arrow) in a 6-year-old boy with osteogenesis imperfecta. Mild anterior bowing of the femoral shaft is already present, and with further fractures, this deformity will worsen. **B:** X-ray study of a 5-year-old ambulatory girl with osteogenesis imperfecta. Note the marked osteopenia of the proximal femurs. **C:** Thirteen months later, the patient sustained bilateral proximal femoral stress fractures, which eventually required treatment with Bailey-Dubow rods.



**FIGURE 6-36. A:** This 6-week old girl presented with multiple fractures in different stages of healing. She was evaluated for osteogenesis imperfecta and nonaccidental injury. She had a strong family history of osteogenesis imperfecta. An AP radiograph of the left upper extremity shows marked osteopenia and a nondisplaced spiral fracture of the left proximal humerus. **B:** An AP radiograph of the right lower extremity showing osteopenia and healing fractures of both the femur and the tibia. There is already some varus angulation and bowing to the mid-shaft of the right femur.

The diagnosis of OI is based on clinical and radiographic findings. There is no specific laboratory diagnostic test, although fibroblast cell culture can detect the collagen abnormality in 85% of OI patients (389). In the absence of multiple fractures, the initial radiographic diagnosis can be difficult. It is crucial, but often difficult, to distinguish OI from nonaccidental injury (404,409). Unexplained fractures in mild, undiagnosed OI can drag a family through unnecessary legal proceedings; conversely, a child with OI may be abused but not exhibit classic fracture patterns (e.g., corner fractures) owing to the fragility of their bones. Although no test or finding is specific, skin biopsy plays an important role (414,421).

### Treatment

The multiple fractures in OI usually are transverse, diaphyseal, and seldom displaced, and they usually heal at a relatively normal rate in most patients (402,417). Most fractures in patients with OI occur before skeletal maturity. In a series of 31 patients, Moorefield and Miller (410) noted 951 fractures, 91% of which occurred before skeletal maturity. Fractures of the femur and tibia predominate. The humerus is the most commonly fractured bone in the upper extremity. Multiple long bone fractures may result in coxa vara, genu valgum, and leg-length discrepancy. Lateral dislocation of the radial head has been noted in some patients (402). Olecranon fractures, which are rare in unaffected children, are more common in patients with OI, especially the tarda form (391,422). Di Cesare et al. (391) reported an infant whose presentation with bilateral isolated olecranon fractures led to the diagnosis of OI.

Nonunion is more common in OI than in similar fractures in unaffected children. Although nonunion was mentioned in several series (410), Gamble et al. (393) emphasized the problem with a report of 12 nonunions in 10 patients. Almost all had type III OI (419) and presented with nonpainful clinical deformity and decreased functional ability. A history of inadequate treatment of the initial fracture was seen in 50% of these nonunions. One patient eventually required an amputation for a painful nonunion of a distal femoral supracondylar fracture.

### Prevention

The role of medical therapy to limit the fracture frequency in OI should be considered investigational but promising (396,413). The tremendous genotypic and phenotypic variations in OI must be considered as the results of these trials are analyzed: A drug that works well for children with certain forms of OI may be ineffective for others. Sodium fluoride, growth hormones, and anabolic steroids have all been shown to be ineffective. Calcitonin, which limits osteoclasts, has had variable success. Nishi et al. (413) reported that the fracture rate decreased in 10 patients with OI treated with either calcitonin injection or nasal spray.

At present the most promising agents are the bisphosphonates (385,396). Pamidronate, like other bisphosphonates, is a potent inhibitor of bone resorption. In a trial of 30 children with severe OI, Glorieux et al. (396) showed that cyclic intravenous administration of pamidronate every 4 to 6 months resulted in a 41.9% increase per year in bone mineral density, an increase in metacarpal cortical width, and a decrease in fracture incidence of 1.7 fractures per year. Mobility improved in 16 of the 30 children, and all reported substantial relief of chronic pain.

### Closed Methods

The orthopaedist caring for children with OI must balance good, standard fracture care (satisfactory reduction and casting) with the goal of minimizing immobilization to avoid a vicious circle: immobilization, weakness and osteopenia, then refracture (381,405,410). Plaster splints and casts, braces, and air splints have all been used (387,388,392,410,417).

Protected weight bearing is thought to reduce the incidence of lower extremity fractures (395). Customized splints and braces can add support to limbs weakened by fragile and deformed bone. Letts et al. (406) encouraged weight bearing in patients by protecting them with vacuum pants. The splinting system is a two-layer set of pants with Styrofoam beads between the layers. By evacuating the interval between the layers, a form-fitting orthosis results, much like the bean bag seating systems. Both decreased frequency of fracture and increased bone density were reported after use of this support system.



## OPERATIVE TREATMENT

Load-sharing devices (such as intramedullary rods) are used for internal fixation of long bone fractures or osteotomies in children with OI. Plates and screws should be avoided. In patients with OI, most internal fixation is used for stabilization after corrective osteotomies. The goals of these osteotomies are to improve function and reduce fractures in weight-bearing bones by correcting angulation. Porat et al. (415) found that the percentage of ambulatory patients in their series went from 45% to 75% after intramedullary rodding. The amount of bowing that requires osteotomy has not been defined. In one series (411), the average preoperative bowing was 71 degrees for the femur and 40 degrees for the tibia, but many patients had much less angulation.

Traditionally, multiple osteotomy and rodding procedures (Sofield technique) involved extensive incisions with significant soft tissue stripping and blood loss. Sijbrandij (418) reported a percutaneous technique in which the deformity is straightened by closed osteoclasis and Rush pins are inserted along the proximal axis of the long bones, partially transfixing them to stabilize them in a new alignment. Most centers now use limited incisions, thus minimizing blood loss and periosteal stripping, while ensuring optimally placed osteotomies and efficient, controlled instrumentation. The choice of fixation device should be based on the age of the patient and the width of the medullary canal of the bone. Both fixed-length rods (402,415,420) and extensible Bailey-Dubow rods (382,383,384,393,394,411,412,415) are used. Skeletally mature patients and patients with very small medullary canals are best treated with nonelongating rods, whereas skeletally immature patients with adequate width of the medullary canal are best treated with extensible rods (394). Luhmann et al. (407) reported a 20-year experience with extensible nails: both overlapping Rush rods and Bailey-Dubow rods. They first implanted the rods at an average age of 7 years old and averaged more than 5 years before the first revision. They recommended a posterior position in the canal and using the stronger overlapping Rush rods technique in the femur whenever the canal diameter permitted; they advised against using overlapping Rush rods in the tibia.

Displaced fractures of the apophysis of the olecranon in patients with OI can be treated with open reduction and stabilization by resorbable suture in infants and with open reduction and internal fixation with two Kirschner wires and tension band technique by figure-of-eight absorbable suture in older children (422) (Fig. 6-37). Tibial tubercle avulsion injuries should be treated by surgical stabilization if displaced.

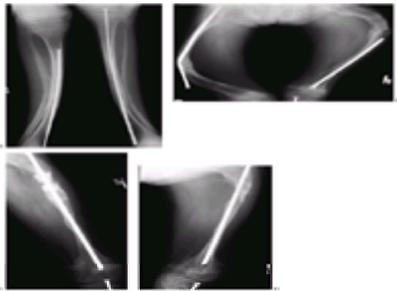


**FIGURE 6-37.** This 10-year-old girl with osteogenesis imperfecta presented with a displaced olecranon sleeve fracture of the left elbow. This was her first fracture. **A:** This lateral radiograph shows a widely displaced fracture involving the posterior portion of the articular surface. **B:** She was treated with open reduction and internal

fixation with two K-wires and tension band construct using #2 Vicryl suture. She was maintained in a cast for 3 weeks and then started on range-of-motion exercises.

## Complications

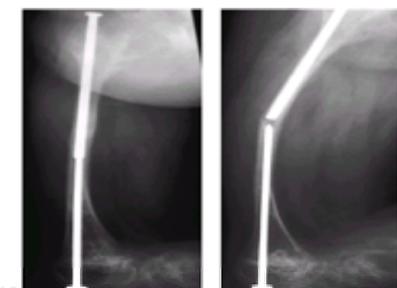
Complication rates are high (Fig. 6-38). Problems include fracture at the rod tip (Fig. 6-39), migration of the fixation device, joint penetration, loosening of components of extensible rods, and fractures through the area of uncoupled rods (Fig. 6-40). Harrison and Rankin (399) compared the complications of 23 extensible rods with those of 27 fixed length rods. They found that the refracture rate was higher in the fixed length rods and fewer surgical interventions were necessary when extensible rods were used. Gamble et al. (394) reported that, although the complication rate approached 69% for Bailey-Dubow rods (mostly due to loosening of the T-piece), the fixed length rods had a complication rate of 55%; however, the replacement rate for nonelongating rods was 24% but only 12% for the Bailey-Dubow rods. They recommended crimping the T-piece to the sleeve and burying it slightly under the bone of the greater trochanter to prevent displacement. Jerosch (400) found a similar 63.5% complication rate for Bailey-Dubow rods but also thought that they were the best device available. Porat et al. (415) found that the complication rate was 75% for Bailey-Dubow rods and 50% for nonelongating rods, with a similar percentage requiring reoperation for both types of nails. Zionts et al. (424) reported 40 complications in 40 extensible nailings of 15 children, finding a much higher complication rate when insertion of the rods was initiated before 5 years of age. Complications were also higher for tibial nailings.



**FIGURE 6-38. A:** The lower extremity deformities of this 14-year-old girl with severe osteogenesis imperfecta were managed throughout childhood with Bailey-Dubow rods. This AP radiograph of both legs shows successful lengthening and maintenance of alignment of the left tibia but failure of rod lengthening with valgus angulation of the right tibia. **B:** The AP radiograph of both femurs, also taken at age 14, shows signs of several of the problems in the management of the femoral deformities throughout childhood. The right Bailey-Dubow rod protrudes into the soft tissues distally. There is femoral bowing with a fracture and bending of the Bailey-Dubow rod at the junction of the male and female ends. In the left femur, part of the Bailey-Dubow rod has been removed because it was protruding into the soft tissues. There is a pathologic fracture through the bowing of the proximal femur. **C:** Deformities of both femurs were treated by osteotomy and exchange of the Bailey-Dubow rods for a Rush nail. **D:** One year after the exchange, the osteotomy has healed. The femoral alignment is good, but the proximal portion of the Rush rod is prominent in the soft tissues.



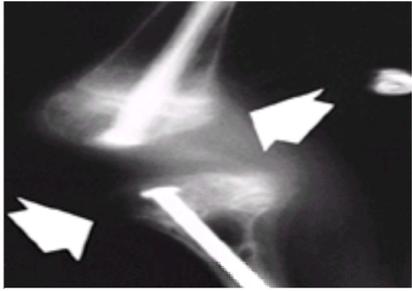
**FIGURE 6-39. A:** An 11-year-old boy with osteogenesis imperfecta fell and sustained a right subcapital femoral neck fracture (arrow). **B:** The fracture was reduced with gentle traction by a fracture table under general anesthesia and was stabilized with a cannulated screw, which was placed with difficulty through an open approach, just anterior to the Bailey-Dubow intramedullary rod.



**FIGURE 6-40. A:** This 10-year-old with severe osteogenesis imperfecta presented several years after Bailey-Dubow rod treatment for fractures and deformity of the right femur. This lateral femoral radiograph shows osteopenia, good position of the Bailey-Dubow rod, and signs that the rod has allowed significant lengthening of the femur without deformity. **B:** This lateral radiograph of the right femur, taken 2 years later at age 12, shows that the rod has separated. There is now a fracture in the gap between the male and female ends of the Bailey-Dubow rod. (Courtesy of Brian Grottkau M.D. and Michael Goldberg, M.D., Boston, Mass.)

Postoperative bracing is suggested for lower extremity fractures (411). Upper extremity fractures should also undergo prolonged splinting after removal of fracture fixation. Immobilization also may be adequate to treat stable, minimally displaced fractures just distal or proximal to the intramedullary rods (394).

Nonunions, after fracture or surgical intervention, may cause difficulty in both ambulation and transfer. In one series of nonunions (393), the average age at diagnosis was close to 10 years and most patients responded to treatment with intramedullary rods and bone grafting, with healing in approximately 9 weeks. Nonunion can occur after insertion of rods for upper extremity fractures. Growth arrest also can follow the use of intramedullary rods in the lower extremities (Fig. 6-41).



**FIGURE 6-41.** An 8-year-old girl with osteogenesis imperfecta with limb-length discrepancy. Lateral tomograms of the knee show a central physeal arrest of the proximal tibia and early physeal arrest of the distal femur (arrows). Such complications are not infrequent with the use of Bailey-Dubow rods.

## AUTHORS' PREFERRED METHOD OF TREATMENT

Protected weight bearing is the goal for patients with severe OI. Close follow-up is necessary in the first few years of life, with protective posterior plaster splinting for fractures. Orthoses are constructed for bracing of the lower extremities to aid in both standing and ambulation. Standing frames also are used. Once he or she is ambulatory, the patient is advanced to the use of a walker or independent ambulation.

Severe bowing of the extremities after recurrent fractures is an indication for osteotomy and intramedullary rodding. Whenever possible, surgery is delayed until the child is 6 or 7 years old. We recommend extensible rods in skeletally immature patients and nonelongating rods both in older patients and in younger patients whose canal is not wide enough for insertion of Bailey-Dubow rods. When possible, we use limited incisions for insertion of Bailey-Dubow rods to minimize blood loss and to avoid devascularization of the long bones, which occurs commonly in the so-called open shish-kabob technique.

### Operative Technique—Bailey-Dubow Rod

Multiple small skin incisions are made at the point of maximal deformity of the long bones, and a small periosteal incision is made to permit introduction of a drill bit. After the cortex is drilled repeatedly, manual osteoclasis completes the osteotomy and the long bone is straightened. A guide pin is drilled in to the medial edge of the tip of the greater trochanter, and down into the intertrochanteric femur, and then is tapped distally through the intramedullary canal through the knee. If callus from old fracture halts progress, the guide pin can be drilled through the obstruction and then tapped farther distally into the middle of the distal flexed knee joint. Next, the reamer is advanced over the guide pin and the female portion of the Bailey-Dubow rod is threaded over the guide pin and driven down the canal. The guide pin is pulled out of the knee joint through a small arthrotomy incision, and the T-piece is attached to the Bailey-Dubow sleeve. The T-piece is tapped down into the greater trochanter. The male portion is then slid into the sleeve to complete assembly of the expandable rod.

The patient is immobilized for approximately 4 weeks in a hip spica cast and then in a brace for 3 to 6 months. When there are fractures associated with intramedullary rods, these are treated with either rod revision or immobilization. Every effort should be made to keep patients ambulatory early after fractures occur to minimize disuse osteoporosis.

### Osteopetrosis

Osteopetrosis, also known as Albers-Schönberg disease, is a condition in which excessive density of bone occurs as a result of abnormal function of osteoclasts (425,442). The resultant bone of these children is dense, brittle, and highly susceptible to pathologic fracture. The incidence of osteopetrosis is approximately 1 per 200,000 births. The inherent problem is a failure of bone resorption with continuing bone formation and persistent primary spongiosa. The disorder classically has been divided into a severe infantile malignant type and a milder form that presents later in life. Intermediate forms have been identified in which osteopetrosis presents as renal tubular acidosis (448). Although the number of osteoclasts present in the affected bone is variable (408,435), in the severe form of this disease, the osteoclasts may be increased but function poorly (446).

Radiographically, the bones have a dense, chalklike appearance (Fig. 6-42). The spinal column may have a sandwich or rugger jersey appearance because of dense, sclerotic bone at each end plate of the vertebrae and less involvement of the central portion. The long bones tend to have a dense, marble-like appearance and may have an Erlenmeyer's flask shape at their ends owing to deficient cutback remodeling. Radiolucent transverse bands may be present in the metaphysis of the long bones, and these may represent a variable improvement in the resorption defect during growth of the child (441). There may be bowing of the bones due to multiple fractures (432), spondylolysis (440), or coxa vara (442). The small bones of the hands and feet may show a bone-within-bone appearance with increased density around the periphery. The unusual radiographic appearance may initially obscure occult, nondisplaced fractures.



**FIGURE 6-42.** This 2-year-old with osteopetrosis presented with forearm pain. An AP radiograph shows the characteristic increased bone density and absence of a medullary canal, especially in the distal radius and ulna. There is a typical transverse, nondisplaced fracture (arrow) in the distal ulnar diaphysis.

### Treatment

Pathologic fractures are quite common in patients with osteopetrosis (408,427,432,436,438,445,448). Patients with a severe form of the disease have more fractures than those with presentation later in childhood. Concurrent blindness can make patients more susceptible to accidental trauma. Patients with autosomal dominant osteopetrosis with rugger jersey spine and endobones of the pelvis (type II) are six times more likely to have fractures than patients with only sclerosis of the cranial vault (type I) (427).

Patients with the severe, congenital disease have transverse or short oblique fractures of the diaphysis, particularly the femur (Fig. 6-43). Distal physeal fractures with exuberant callus may be confused with osteomyelitis (441). Common locations for fractures include the inferior neck of the femur, the proximal third of the femoral shaft, and the proximal tibia (425,441). Although most fractures involve the long bones of the lower extremities, upper extremity fractures also occur frequently (425,432). The onset of callus formation after fracture in osteopetrosis is variable (425,432,435). Although many studies state that fractures in osteopetrosis heal at a normal rate (435,442), others report delayed union and nonunion (425). In a rat model of osteopetrosis, Marks and Schmidt (439) found delayed fracture healing and remodeling. Hasenhuttl (432) observed that in one patient with recurrent fractures of the forearm, each succeeding fracture took longer to heal, with the last fracture

taking nearly 5 months to unite.



**FIGURE 6-43.** **A:** This 9-year-old with osteopetrosis sustained similar bilateral subtrochanteric fractures of the femur over a 2-year period. This AP femoral radiograph and **(B)** this lateral femoral radiograph shows a healing transverse subtrochanteric fracture of the left femur. **C:** One year later, at age 10, she sustained a similar right transverse minimally displaced subtrochanteric femur fracture, which was treated with reduction and a spica cast. **D:** This AP radiograph taken at age 14 years shows that both proximal femur fractures have healed and there is mild residual coxa vara, especially on the right side.

The orthopaedist treating fractures in children with osteopetrosis should follow the principles of standard pediatric fracture care, with additional vigilance for possible delayed union and associated rickets ([432,445](#)). Immobilization is prolonged when delayed union is recognized. Armstrong et al. ([425](#)) surveyed the membership of the Pediatric Orthopaedic Society of North America and compiled the combined experience of 58 pediatric orthopaedic surgeons with experience treating pathologic fractures in osteopetrosis. In this comprehensive review, they concluded that nonoperative treatment should be strongly considered for most diaphyseal fractures of the upper and lower limbs in children, but surgical management is recommended for femoral neck fractures and coxa vara.

Open treatment of these fractures with fixation is technically difficult. One author ([431](#)) described insertion of fixation into this bone like “drilling into a rock.” In intramedullary fixation of femoral fractures, extensive reaming may be required because the intramedullary canal can be completely obliterated by sclerotic bone ([428](#)). Other authors have found fixation of hip fractures with fixation to be a formidable task ([425,441](#)), with damage occurring to the fixation devices on insertion. The bone is hard enough to break the edges off both chisels and drill bits. Armstrong et al. ([425](#)) cautioned that “the surgeon should expect to use several drill bits and possibly more than one power driver.”

In addition to these technical difficulties, patients with osteopetrosis are at risk for excessive bleeding and infection, probably related to the hematopoietic dysfunction caused by obliteration of the marrow cavity ([445](#)). Procedures should not be performed unless the platelet count is greater than  $50,000 \text{ mm}^3$ , and preoperative platelet transfusions may be necessary ([445](#)). Prophylactic antibiotic coverage is advised. Minor procedures should be performed percutaneously whenever possible ([445](#)).

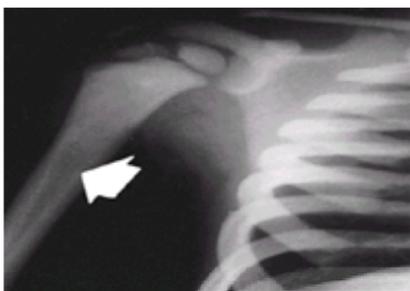
In the past, primary medical treatment for osteopetrosis included transfusions, splenectomy, calcitriol, and adrenal corticosteroids, but these techniques have proved ineffectual ([444,447](#)). Bone marrow transplantation for infant malignant osteopetrosis has proved to be an effective means of treatment for some patients, but it is not possible in all patients, it does not guarantee survival, and it may be complicated by hypercalcemia ([425,429,430,443](#)).

### Pyknodysostosis

Pyknodysostosis is a rare syndrome of short stature and generalized sclerosis of the entire skeleton. The dense brittle bones of affected children are highly susceptible to pathologic fractures. Henri Toulouse-Lautrec was thought to be afflicted by this disorder ([452](#)). Pyknodysostosis is inherited as an autosomal recessive trait, with an incidence estimated as 1.7 per 1 million births. The long bones are sclerotic, with poorly formed medullary canals; histologic section show attenuated haversian canal systems.

Patients with pyknodysostosis have short stature, a hypoplastic face, a nose with a parrot-like appearance, and both frontal and occipital bossing. Bulbous distal phalanges of the fingers and toes with spooning of the nails are common. Coxa vara, coxa valgum, genu valgum, kyphosis, and scoliosis may be present. Failure of segmentation of the lower lumbar spine has been reported. Results of laboratory studies usually are normal.

Radiographs show a sclerotic pattern very similar to that of osteopetrosis. In pyknodysostosis, however, the medullary canals, although poorly formed, are present and a faint trabecular pattern is seen ([Fig. 6-44](#)). Such sclerotic bone is also seen in Engelmann's disease, but clinically those patients are tall and eventually develop muscle weakness. The distal femur in a patient with pyknodysostosis usually has an Erlenmeyer's flask deformity similar to that found in patients with Gaucher's disease ([449](#)).



**FIGURE 6-44.** A young boy with pyknodysostosis. Although bone density is increased overall, a medullary canal is present in the proximal humerus with faint trabecular lines (*arrow*) in contrast to osteopetrosis, in which the medullary canal is usually completely obliterated. (Courtesy of John D. McKeever, M.D., Corpus Christi, Texas)

### Treatment

Although pathologic fractures are thought to be less common in pyknodysostosis than in OI, almost all patients with pyknodysostosis reported in the literature have had pathologic fractures. By age 22 years, one patient had sustained more than 100 fractures ([450](#)). The fractures are usually transverse and diaphyseal, and heal with scanty callus ([453](#)). The fracture line can persist for nearly 3 years after clinical union, with an appearance similar to a Looser line. Lower extremity fractures are the most common ([450](#)), and clinical deformity of both the femur and tibia is frequent.

Fracture healing has been described as both normal ([455](#)) and delayed ([450](#)). Nonunion is reported in the ulna, clavicle, and tibia ([453](#)). One series ([453](#)) with long-term follow-up suggests that fractures tend to heal readily in childhood, but nonunion can be a problem in adulthood. Edelson et al. ([451](#)) reported 14 new cases of pyknodysostosis from a small Arab village. They described a hangman's fracture of C2 in a 2-year-old child that went on to asymptomatic nonunion. There was 100% incidence of spondylolysis in their patients aged 9 years or older, with most located at L4–L5. None of the spondylolytic lesions showed uptake on technetium  $99\text{m}$  bone scan. Treatment was conservative, with one patient with symptomatic spondylolysis responding to bed rest.

Cast immobilization is successful in the treatment of most of these fractures. Taylor et al. ([456](#)) treated a femoral fracture in an 11-year-old boy with skin traction and a

one-and-a-half hip spica cast. At 6-month follow-up, clinical union with persistent fracture line was seen. In adults, both plates and screws and hip screws have been used for proximal femoral fractures (454). Delayed union of tibial fractures has been treated with both compression plating and bone grafting (453) and intramedullary nailing with cast immobilization. Roth (454) noted that treatment of a hip fracture with fixation was technically difficult. Cervical immobilization through a Minerva cast and soft cervical collar has been used for a C2 fracture in a child, and the patient did well, although immobilization was prematurely discontinued (450).

## Rickets

Rickets is a disease of growing children caused by either a deficiency of vitamin D or an abnormality of its metabolism. The osteoid of the bone is not mineralized, and broad unossified osteoid seams form on the trabeculae. With failure of physal mineralization, the zone of provisional calcification widens and the ingrowth of blood vessels into the zone is disrupted. In the rickets of renal failure, the effects of secondary hyperparathyroidism (bone erosion and cyst formation) also are present. Before widespread fortification of common foods, vitamin D deficiency was a common cause of rickets, but other diseases affecting the metabolism of vitamin D have become a more common cause. Regardless of the underlying cause, the various types of rickets share similar clinical and radiographic features. Although many of the metabolic findings are the same, there are some differences (Table 6-11).

Disorder	Cause	1,25(OH) <sub>2</sub> Vitamin D	PTH	Ca <sup>2+</sup>	P	Alk Phos
Vitamin D deficiency rickets	Lack of vitamin D in the diet	↓	↑	↓ or →	↓	↑
Gastrointestinal rickets	Decreased gastrointestinal absorption of vitamin D or Ca <sup>2+</sup>	↓ or →	↑	↓	↓	↑
Vitamin D-dependent rickets	Reduced 1,25(OH) <sub>2</sub> vitamin D production	↓	↑	↓	↓	↑
Vitamin D-resistant rickets—end organ insensitivity	Intestinal cell insensitivity to vitamin D causing decreased Ca <sup>2+</sup> absorption	↓ or →	↑	↓	↓	↑
Renal osteodystrophy	Renal failure causing decreased vitamin D synthesis, phosphate retention, hypocalcemia and secondary hyperparathyroidism	↓	↑	↓	↑	↑

TABLE 6-11. RICKETS: METABOLIC ABNORMALITIES

Both pathologic fractures (435,457,464,466,468) and epiphyseal displacement (458,465) can occur in rickets with associated renal disease. The treatment of rickets with associated pathologic fractures depends on identification of the underlying cause. In addition to nutritional rickets, many diseases of the various organ systems can affect vitamin D metabolism, and their treatment is necessary before the clinical rickets can be resolved.

### Nutritional Rickets

Inadequate dietary vitamin D and lack of exposure to sunlight can lead to a vitamin D deficiency (Fig. 6-45). Pathologic fractures from vitamin D deficiency rickets also occur in children on certain diets: unsupplemented breast milk (463), diets restricted by religious beliefs (457), and fad diets (460).



FIGURE 6-45. **A:** This 3-year-old girl presented with extremely severe nutritional rickets. An AP radiograph of the pelvis and both femora shows severe coxa vara, widening of all physes, and osteopenia of the distal femora, with flaring of the distal femoral metaphysis. **B:** Standing lower extremity radiograph shows further deformity of the distal tibia with Looser lines visible on the concavity of the deformity of the right distal fibula.

Fractures are treated with both cast immobilization and correction of the vitamin deficiency by oral vitamin D supplementation. Oral calcium supplements also may be necessary, and patients should consume a vitamin D–fortified milk source (457).

### Rickets in Malabsorption Diseases

Celiac disease caused by gluten-sensitive enteropathy affects intestinal absorption of fat-soluble vitamins (such as vitamin D), resulting in rickets. Biopsy of the small intestine shows characteristic atrophy of the villi. Treatment is oral vitamin D and a gluten-free diet. Infants with short gut syndrome may have vitamin D–deficiency rickets. This syndrome may develop after intestinal resection in infancy for volvulus or necrotizing enterocolitis, in intestinal atresia, or after resection of the terminal ileum and the ileocecal valve (471). Pathologic fractures have been reported, and treatment is immobilization and administration of vitamin D<sup>2</sup> with supplemental calcium gluconate.

Hepatobiliary disease also is associated with rickets (459,462) (Fig. 6-46). With congenital biliary atresia, the bile acids, essential for the intestinal absorption of vitamin D, are inadequate. By age 3 months, nearly 60% of patients with biliary atresia may have rickets (461). Intravenous vitamin D often is needed for effective treatment of these patients. After appropriate surgical correction of the hepatic syndrome, the bone disease gradually improves. The pathologic fractures that develop in these disorders (459) can be treated with immobilization.

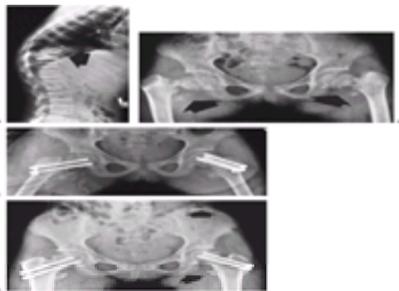


FIGURE 6-46. **A:** This 18-year-old boy with sclerosing cholangitis and a history of steroid use presented with several months of worsening low back pain. This lateral radiograph of his lumbar spine shows marked osteopenia, collapsed codfish vertebrae with sclerotic end plates and widened disc spaces and Schmorl's nodes. **B:** This MRI shows flattened concave vertebrae that are smaller in most locations than the adjacent intervertebral disc. He was successfully treated with 3 months in a

thoraco-lumbar-sacral-orthosis (TLSO) brace, followed by weaning from the brace and conditioning exercises.

Anticonvulsant therapy can interfere with the hepatic metabolism of vitamin D and result in rickets and pathologic fractures. Fewer fractures occur in institutionalized patients receiving vitamin D prophylaxis (467). In a study of 20 children with epilepsy, there was no difference in the bone mineral density of the femoral neck in patients on either phenobarbital or phenytoin compared with control subjects (470).

Ifosamide, a chemotherapeutic agent used for treatment of Ewing's sarcoma, can cause hypophosphatemic rickets in children. The onset of rickets may occur anywhere from 2 to 14 months after chemotherapy and can be corrected with the administration of oral phosphates (469). Other mineral deficiencies such as magnesium (a cofactor for parathyroid hormone) can cause rare forms of rickets (Fig. 6-47).



**FIGURE 6-47.** **A:** A 13-year-old girl presented with rickets caused by magnesium deficiency. She had back and hip pain after a seizure; the x-ray study shows compression fractures of the thoracic vertebrae (arrow). **B:** Displaced femoral neck fractures are also present with periarticular calcification (arrows). **C:** The fractures were openly reduced with fixation by multiple cannulated screws. She was placed in a hip spica cast to protect the fixation. **D:** At 12-month follow-up, the femoral neck fractures have united, with no evidence of avascular necrosis of the femoral heads. With medical treatment of the rickets, new bone has been added to the pelvis (arrows.)

### Rickets and Very-Low-Birth-Weight Infants

Very-low-birth-weight infants (1,500 g or less) can have pathologic fractures. In one study of 12 very-low-birth-weight infants, the incidence of pathologic fracture was 2.1%, nearly twice the rate of other premature infants with a birth weight of more than 1,500 g (472). The fractures are likely caused by a nutritional osteomalacia that may evolve into frank rickets in nearly 30% of very-low-birth-weight infants (472,474,476,478) (Fig. 6-48). During the last trimester of pregnancy, intrauterine growth rate is exponential and almost two thirds of the birth weight is gained at that time (480). Eighty percent of both calcium and phosphorus is acquired then (475). Bone loss can be graded by either loss of cortical bone of the humerus (479) or loss of bone of the distal radius (476). Other than craniotabes (thinning and softening of the skull bone, with widening of the sutures and fontanelles), the clinical signs of rickets are generally lacking in these patients (574). The risk factors predisposing these patients to both rickets and fractures include hepatobiliary disease (461,462,472), prolonged total parenteral nutrition (472), chronic lung disease (472), necrotizing enterocolitis (471), patent ductus arteriosus (473), and physical therapy with passive range-of-motion exercises (474,477). In a prospective study of 78 low-birth-weight infants, Koo et al. (477) observed a 73% incidence of rickets with associated pathologic fractures in patients with a birth weight of 800 g or less and only a 15% incidence of rickets with fractures in patients with a birth weight ranging from 1,000 to 1,500 g.



**FIGURE 6-48.** A 4-month-old premature infant of low birth weight with healing rickets. New periosteal bone is cloaking the humerus (arrow).

### Treatment

In most cases, pathologic fractures in very-low-birth-weight infants are found incidentally on chest x-ray or gastrointestinal studies. The fractures may be suspected when physical examination reveals swelling and decreased movement of an extremity. The differential diagnosis of these fractures is limited but important: OI, copper deficiency syndrome, child abuse, and pathologic fracture from overzealous physical therapy (474). Recurrent fractures, physical findings, and a positive family history are the hallmarks of OI; serum copper levels are useful in establishing copper deficiency syndrome. Neonatal osteomyelitis may also present a similar x-ray appearance. If risk factors for infection are present, the bone lesion should be aspirated and cultured (472).

In the series reported by Amir et al. (472), 12 (1.2%) of 973 preterm infants had fractures; 11 of 12 had more than 1 fracture. Radiographically, osteopenia is first seen at the fourth week of life. Typically, rib fractures are next seen at 6 to 8 weeks of life, then fractures of the long bones at 11 to 12 weeks (480). In one study, 54% of fractures were in the upper extremities, 18% in the lower extremities, 22% in the ribs, and approximately 6% in either the scapula or the clavicle (477). Most long bone fractures are metaphyseal and may be transverse or greenstick with either angulation or complete displacement (472). Callus is seen at the fracture site in less than a week, and complete remodeling occurs in 6 to 12 months (472,477). Passive range-of-motion exercises for these infants, by both physical therapists and parents, should be avoided unless it is absolutely necessary (474). Rib fractures have been associated with vigorous chest physiotherapy (477,478). Care also should be taken even with routine manipulation of the extremities during nursing care, and special care should be taken in restraining the extremities during surgical procedures (477).

Splinting is the treatment of choice for pathologic fractures of the long bones in very-low-birth-weight infants (Fig. 6-49). Hip spica casts are contraindicated because they may compromise cardiopulmonary support and hamper nursing care (477). Regardless of the means of immobilization, the prognosis is excellent for most of these fractures because they go on to complete remodeling within 12 months; prolonged follow-up is advised. Preventive measures are important to minimize the risk of fracture in low-birth-weight infants. Their nutritional need for high levels of calcium, phosphorus, and vitamin D should be recognized. Alternating high levels of calcium with low levels of phosphorus in hyperalimentation solutions can help meet these needs. Because growth arrest is possible after fractures, follow-up over the first 2 to 3 years of life is advised.



**FIGURE 6-49.** A tibia splint has been constructed for this premature infant by wrapping a tongue blade in a heavy layer of Webril and then fracturing the tongue depressor so that it assumes a sugar tong appearance. Tape is gently wound around the splint to hold it in place.

### **Rickets and Renal Osteodystrophy**

Renal osteodystrophy is common in patients with end-stage renal failure; it was diagnosed in 79% of patients in a series of 50 children ( 490). Typically developing about 1.4 years after diagnosis of the kidney disease ( 490), the clinical syndrome is a combination of rickets and secondary hyperparathyroidism with marked osteoporosis. These children present with short stature, bone pain, muscle weakness, delayed sexual development, and bowing of the long bones ( 483). The underlying renal disease may be chronic nephritis, pyelonephritis, congenitally small kidneys, or cystinosis ( 468). Identification of the renal disorder is important because patients presenting with rickets due to obstructive uropathy may respond to surgical treatment of the renal disease.

Specific clinical deformities include genu valgum (most common), genu varum, coxa vara, and varus deformities of the ankle ( 482,483,485). These deformities are most common in patients diagnosed before 3 years of age. Davids et al. ( 485) showed that periods of metabolic instability, characterized as an alkaline phosphatase of 500 U for at least 10 months, were associated with progression of deformity. With the adolescent growth spurt, osseous deformities can accelerate rapidly over a matter of weeks (485).

Radiographs show rickets and osteopenia with osteitis fibrosa cystica ( 482). Osteoclastic cysts (brown tumors) may form. Metaphyseal cortical erosions occur in the lateral clavicle, distal ulna and radius, neck of the humerus, medial femoral neck, medial proximal tibia, and middle phalanges of the second and third fingers ( 490). The proximal femur may become so eroded with tapering and thinning that it has been likened to a rotting fence post ( 502). In renal osteodystrophy, the Looser zone may represent a true stress fracture and, with minor trauma, may extend across the full thickness of the bone with development of a true fracture. Callus may be scanty in patients with fractures who have untreated renal disease, but in patients on hemodialysis, abundant callus may form at the fracture site ( 466).

### **Treatment**

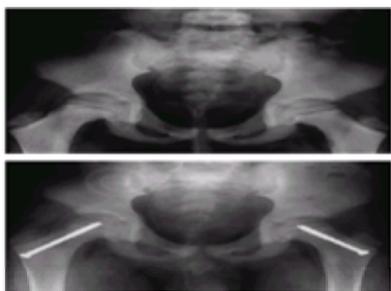
In renal osteodystrophy, pathologic fractures of the long bones, as well as rib fractures, vertebral compression fractures, and displacement of the epiphyses, occur frequently. Fractures occur in areas of metaphyseal erosion or through cysts. Immobilization is used to treat pathologic fractures through both generalized weakened bone and brown tumors.

Once the underlying bone disease is under control, open procedures such as curettage of cysts with bone grafting and open reduction of fractures may be considered when appropriate (466). Internal fixation is preferable to external fixation ( 484). Preoperative tests needed for these patients before surgery include electrolytes, calcium, phosphorus, and alkaline phosphatase. Before surgery, these patients may need dialysis, phosphate adjustment, either medical or surgical correction of hyperparathyroidism, or chelation therapy for aluminum toxication. Supplemental vitamin D should be discontinued or the dose halved 2 to 4 weeks before any procedure that may require immobilization (483). It is important to rule out dental abscess, which may be present in as many as 25% of patients with vitamin D-resistant rickets (494). Postoperative infection may be more common in patients who are on corticosteroid therapy after renal transplantation ( 496). Prophylactic antibiotics are highly recommended for surgery of all patients with renal osteodystrophy ( 487).

Osteonecrosis may occur in weight-bearing bones, with the femoral head most often symptomatic (501). Other sites of involvement include the distal femoral condyles and the talus (482,499). Osteonecrosis may result from the use of immunosuppressive corticosteroids, because there is less osteonecrosis when the corticosteroid dose is decreased (486). Ferris et al. (488) described osteochondritis dissecans lesions of the medial femoral condyles in young adults with hypophosphatemic rickets. Both loose bodies and so-called trap-door lesions were seen in these patients. Older patients developed calcification of the ligaments about the hip, knees, and spine. Lesions of bilateral osteochondritis dissecans may require repeated arthroscopic irrigations and removal of loose bodies ( 488). Joint stiffness in older patients due to ligamentous calcification about the knees, hips, and spine can be minimized if early corrective osteotomies of the lower extremities are performed to place them in the optimal position for function. Osteonecrosis in renal osteodystrophy can be treated with repeated joint aspiration, crutch ambulation, and physical therapy (485).

### **Rickets and Renal Osteodystrophy—Slipped Capital Femoral Epiphysis**

The incidence of epiphyseal displacement in children with renal osteodystrophy ranges from 20% to 30% ( 465,491). Sites of involvement include the distal femur, proximal femur, and proximal humerus, the heads of both the metatarsals and metacarpals, and the distal radial and ulnar epiphyses, which tend to displace in an ulnar direction (491). In the proximal femur, both femoral neck fractures (458) and slipped capital femoral epiphysis occur ( Fig. 6-50).



**FIGURE 6-50. A:** This 13-year-old boy with renal osteodystrophy presented with bilateral hip and thigh pain. This AP pelvis radiograph shows widening of the proximal femoral physes with sclerosis. Slipped capital femoral epiphyses were diagnosed. **B:** This AP pelvis radiograph taken 9 months after surgery shows narrowing of the physis and no evidence of further displacement of the capital femoral epiphyses.

Possible explanations for displacement of the proximal femoral epiphysis include metaphyseal erosion with subsequent fracture ( 458,491), and a layer of fibrous tissue that forms between the physis and the metaphysis because of the destructive effects of the renal osteodystrophy ( 492). The warning signs and risk factors for slipped capital femoral epiphysis in renal osteodystrophy include subperiosteal erosion of the medial femoral neck, increasing width of the physis, bilateral coxa vara, male gender, and an age between 10 and 20 years (458). With erosion of the cortex of the inferior medial femoral neck, the femoral head collapses, decreasing the neck shaft angle, and subjecting the physis to shear forces as it assumes a vertical orientation.

The aggressive medical treatment of renal osteodystrophy, including administration of vitamin D ( 493), calcitriol, hemo-dialysis, renal transplantation, and

parathyroidectomy, has improved the long-term survival and quality of life for these patients. Temporary limitation of weight bearing is recommended if there is little metaphyseal erosion, minimal coxa vara, and fusion of the physis is expected within 1 to 2 years (466). Goldman et al. (458) recommended prophylactic hip pinning or close follow-up if risk factors are present for slipped capital femoral epiphysis in patients with renal osteodystrophy. With continuing slippage after medical treatment, most authors recommend *in situ* fixation (458,483,497). Continuing displacement of the proximal femoral epiphysis may occur even after pinning. Fixation holds poorly, possibly because the wide radiolucent zone of the femoral neck in this disorder is not true physis but rather poorly mineralized woven bone and fibrous tissue (465). In a very young child, threaded pin fixation of the proximal femoral epiphysis may result in growth abnormality with trochanteric overgrowth.

Smooth pins can be used to stabilize the epiphysis temporarily until medical treatment resolves the underlying bone disease. For patients younger than 5 years, Hartjen and Koman (489) recommended treatment of slipped capital femoral epiphysis with reduction through Buck's traction and fixation with a single specially fabricated 4.5-mm cortical screw. The distal threads of the screw were machined off so that only the smooth shank of the screw extended across the physis. Subtrochanteric osteotomy with fixation or total hip arthroplasty may be necessary in older patients with severe coxa vara after slipped capital femoral epiphysis (458,483). Medical management of the underlying renal disease and of hyperparathyroidism is mandatory. Pinning of the proximal femur may not be necessary.

### Renal Osteodystrophy Complicated by Aluminum Toxicity

Oppenheim et al. (498) noted the contribution of aluminum toxicity to the development of fractures in renal osteodystrophy. Because phosphorus restriction is important in children with renal disease, aluminum hydroxide has been commonly used as a phosphate binder (481). Aluminum intoxication causes defective mineralization. Multiple pathologic fractures may occur with poor healing. Serum aluminum levels are not diagnostic, but the use of deferoxamine, a chelation agent, in an infusion test may provide the diagnosis (495). A bone biopsy often is necessary.

After treatment of the renal disease with correction of the aluminum toxicity by chelation agents, acute fractures will heal. Severe bowing of the long bones due to fractures can be treated with multiple osteotomies with intramedullary Rush rod or plate fixation (498). Recurrence of the syndrome is prevented by use of aluminum-free phosphate-binding agents such as calcium carbonate (500).

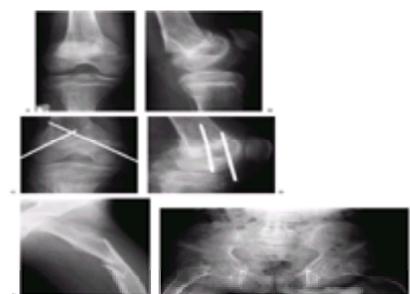
## AUTHORS' PREFERRED METHOD OF TREATMENT

Recognition of the underlying metabolic abnormalities is the most important aspect in the care of all of these injuries. Slipped capital femoral epiphysis may be the first presenting sign of renal failure (458). A slipped capital femoral epiphysis should be stabilized with *in situ* screw fixation in older children if progression is noted despite medical treatment. Multiple screws should be considered because the underlying metaphyseal bone is quite soft. For treatment of progressive slipped capital femoral epiphysis in very young children, some form of unthreaded fixation seems most logical.

Most fractures of the long bones respond readily to cast or splint immobilization with concurrent aggressive medical treatment of the underlying metabolic disease. Femoral neck fractures are treated with anatomic reduction and internal fixation. The underlying bone disease should be medically treated to ensure success of open procedures. Significant cysts should be treated with curettage and bone grafting. Angular deformities of the long bones should be corrected when the patient is close to maturity.

### Idiopathic Osteoporosis

Osteoporosis in a child generally is associated with either congenital disease such as osteogenesis imperfecta or metabolic disorders such as Cushing's syndrome. Rarely, children develop idiopathic osteoporosis with pathologic fractures (Fig. 6-51). Idiopathic osteoporosis is characteristically seen 2 years before puberty, but age at presentation may range from 4 to 16 years (515). Unique metaphyseal impaction fractures are a hallmark of this disorder (509). Biopsy specimens show a quantitative decrease in the amount of bone that has been linked to both increased resorption (510) and primary failure bone formation (515). Osteoblasts in this disorder seem to function normally when stimulated by oral 1,25-hydroxyvitamin D<sub>3</sub> (504). Symptoms can persist for 1 to 4 years after diagnosis, with spontaneous resolution in most patients after the onset of puberty. The only consistent metabolic abnormality is a negative calcium balance with high rates of fecal excretion of calcium (509). This finding supports the hypothesis that idiopathic juvenile osteoporosis results from intestinal malabsorption of calcium (507).



**FIGURE 6-51.** **A:** This case demonstrates multiple pathologic fractures in a previously healthy teenage boy who developed idiopathic osteoporosis. This AP radiograph of the right knee and **(B)** this lateral radiograph demonstrates a displaced distal femoral metaphyseal fracture with apex posterior angulation. **C:** This was treated with closed reduction and percutaneous pinning and application of a cast. **D:** This lateral radiograph shows satisfactory alignment with the pins in place. **E:** A few months later, he sustained a left proximal femur fracture, which was treated with a spica cast. **F:** This AP pelvis radiograph taken 3 years later shows healed proximal femoral fractures with varus angulation, and severe osteopenia of the pelvis and femora with profrusio of both acetabuli.

Although many children present with back pain as the only complaint, the most severely affected present with generalized skeletal pain (503,506,507,508,509 and 510,515). Patients have difficulty walking, and their symptoms may be initiated by mild trauma. In a review of 40 patients with idiopathic osteoporosis, Smith (515) observed that 87% had vertebral fractures and 42% had metaphyseal fractures. Symptoms of back or extremity pain can predate fractures by 6 months (503). Generally, 30% of bone mass must be absent before osteoporosis is detected on radiographs (511). Serum calcium, phosphorus, and alkaline phosphatase levels are usually normal (509). Low plasma calcitriol, a vitamin D metabolite that aids calcium absorption in the gut, has been observed in juvenile osteoporosis (512).

Radiographs of the spine show decreased density in the central areas of the vertebral bodies, and clarity of the dense vertebral end plates is increased. The long bones lose trabecular anatomy and show thinning of the cortex (509,514). Once symptoms begin, a mildly lucent area of newly formed bone, a so-called neo-osseous porosis, is observable in the metaphysis. This is considered weaker than the surrounding bone, which formed before onset of the disease (515).

### Treatment

Lower extremity and vertebral fractures (513) are common, although fractures of the proximal humerus, radius, ulna, and ribs are frequent (509). Nonunions of the tibia, radius, and ulna have been reported (507,509). Spinal cord compression has also been reported with vertebral fractures of osteoporosis in a child (505). Metaphyseal fractures can start as hairline cracks that gradually extend across the width of the shaft, and with further collapse in the femoral shaft, the cracks may telescope into the distal femur, with later distortion of the femoral condyles (509). Tibia and femoral shaft fractures may heal with bowing. Long bone shaft fractures are either transverse or oblique (509), and the callus formed seems to be normal (509,512). A technetium bone scan may be useful in showing healing fractures that are not obvious on plain x-ray studies (512). No clear-cut effective medical treatment has been found for idiopathic juvenile osteoporosis (508,509 and 510). Many patients have been treated by both vitamin D and calcium supplements with equivocal benefit, and usually mineralization of the skeleton does not improve until puberty, when the disease spontaneously resolves.

Dent and Friedman (507) summarized the treatment of fractures in juvenile osteoporosis when they stated that these fractures should undergo anatomic reduction with immobilization "as little as practical." They noted both severe deformity of the long bones and pseudarthrosis when the fractures could not be immobilized. The

bones usually are so soft that they are thought to be unsuitable for the usual forms of fixation, but femoral neck fractures in this disorder have been treated with internal fixation (509).

## Iatrogenic Osteoporosis

### Osteoporosis of Chemotherapy

Osteoporosis is common in association with chemotherapeutic agents. Methotrexate, for example, is believed to inhibit osteogenesis, causing both delayed union and nonunion of fractures (520). The incidence of pathologic fractures after methotrexate use ranges from 19% to 57% (518,520,522). Generalized demineralization of the skeleton is seen with marked radiolucency of the metaphyseal regions of the long bones. Radiographic changes in the metaphysis and epiphysis resemble those seen in scurvy (520). Minimally displaced transverse fractures occur in the long bones of both the upper and lower extremities, and the small bones of the feet (520,522). Schwartz and Leonidas (521) caution that stress fractures of the long bones that can occur after methotrexate therapy can be mistaken for recurrence of leukemia. If feasible from an oncologic viewpoint, methotrexate should be discontinued to allow these fractures to heal in a cast. The cast immobilization itself may result in additional osteopenia and fractures even though methotrexate is discontinued (521). Persistent nonunions require open reduction and internal fixation with bone graft (522). Patients with severe osteoporosis and bone pain without fracture also respond to a halt in methotrexate therapy (520).

### Immobilization Osteoporosis

Immobilization of an extremity for fracture treatment can result in loss of as much as a 44% of mineralization of trabecular bone. In some patients, osteoporosis may persist for 6 months after injury (516). Immobilization leads to bone resorption, especially in unstressed areas (517). In one study (516), bone density of the distal radius returned to normal in all patients at 1-year follow-up. Nilsson and Westlin (519) found a residual decrease in bone mineralization of the distal femur of 7% at nearly 11 years of follow-up in a study of 30 patients. It is likely that persistent osteoporosis after cast immobilization for fracture can contribute to refracture.

## Hyperparathyroidism

Primary hyperparathyroidism in childhood results from either hyperplasia of the parathyroid gland in very young children or parathyroid adenoma in older children (525). Increased parathyroid hormone increases osteoclastic activity, leading to general demineralization of the skeleton. In severely affected patients, osteitis fibrosa cystica may develop with fibrous tissue replacement of bone and formation of cysts (531). Hypercalcemia is common.

A particularly severe form of primary hyperparathyroidism seen in infants is congenital primary hyperparathyroidism, which results from an autosomal recessive trait (529) and is lethal without parathyroidectomy. These patients present with respiratory difficulty, hypotonia, poor feeding with constipation, and failure to thrive (536). Serum calcium is markedly increased in most patients, but a gradual rise above normal serum levels may occur in some infants with serial measurements (535). Radiographs reveal demineralization of the skeleton. Marked resorption is present in the femoral necks and distal tibiae, with decreased trabeculae and poorly defined cortices (529). Periosteal elevation is common, and when it is severe, the long bones may actually look cloaked with new bone (Fig. 6-52). Periosteal resorption of the bone of the middle phalanges is believed to be characteristic of this disease. Brown tumors are rare in infancy.



**FIGURE 6-52. A:** Newborn with hyperparathyroidism. There is marked demineralization of bone, and marked resorption is present in the proximal femora (arrows). **B:** Periosteal elevation is present along the ulna (arrows). (Courtesy of Bruce Mewborne, M.D., San Antonio, Texas.)

In older children and adolescents, the clinical presentation is more subtle. Weakness, anorexia, and irritability are present in 50% of patients, and constipation is present in 28% (525). Renal calculi also are present in 25% of patients, and polyuria, excessive thirst, bone pain, abdominal distention, pancreatitis, and swelling of the knees are occasionally present (524,525,528). Approximately 50% of older patients have osteopenia and other osseous signs of hyperparathyroidism (525). The serum calcium is only moderately elevated in many patients, but 24-hour urine calcium excretion is abnormally high (525,534). If the diagnosis is uncertain, selective venous catheterization for parathyroid hormone can be done, localizing the gland by either ultrasound, CT scan, or MRI (526).

### Treatment

Pathologic fractures of the long bones are common in patients with hyperparathyroidism (523,534), especially in infancy. Vertebral fractures, which occur in 4.4% of adult patients (527), are rare in infancy. Increased levels of parathyroid hormone results in decreased function and numbers of osteoblasts, and hence delayed union of pathologic fractures (533), but this problem has only been reported in adults; healing occurred after parathyroidectomy (532).

Most fractures are successfully treated with simple immobilization. Occasionally, a fracture through a cyst requires curettage and bone grafting after a period of initial healing (537). Partial or total parathyroidectomy is the primary treatment for hyperparathyroidism (526,530). Solitary bone cysts often heal after parathyroidectomy.

## Cushing's Syndrome

Cushing's syndrome results from excessive production of cortisol and its related compounds. If the hyperactivity of the adrenal cortex is due to pituitary gland stimulation, the syndrome is most precisely known as Cushing's disease (541). In children, hypercortisolism is most often caused by carcinoma, adenoma, hyperplasia of the adrenal cortex (540,543,547), Ewing's sarcoma (549), or exogenous corticosteroid therapy (538,550,551). The elevated adrenal corticosteroids inhibit the formation of osteoblasts (544), resulting in increased resorption of the bone matrix and decreased bone formation (546).

In infancy, carcinoma and adenoma are the primary causes of Cushing's syndrome; the remaining cases are secondary to hyperplasia of the adrenal cortex (543). Presenting symptoms include failure to thrive, short stature with excessive weight gain, moon facies, presence of a buffalo hump, hirsutism, weakness, and hypertension (543,547). Cutaneous striae are rare, and the genitalia are of normal size. Mortality is well over 50% (547). In older children, the clinical picture is somewhat different: truncal obesity, short stature, a lowered hairline, acne, weakness, emotional lability, hirsutism, cutaneous striae, hypertension, and ecchymosis (538). Patients with Cushing's syndrome due to adrenocortical neoplasm may have a palpable abdominal mass at an average age of approximately 5 years (540). Diagnosis usually is made by elevation of plasma 17 hydroxy steroids or increased 24-hour urinary excretion of 17-keto steroids (543). Cushing's syndrome also has been associated with hemihypertrophy (539). Corticosteroid therapy in children, both oral and inhaled (538,550,551), can also cause Cushing's syndrome, as well as the development of cataracts, ulcers, pancreatitis, myopathy, and hepatomegaly (542).

Radiographic findings may include severe osteopenia and a retarded bone age (543). Fractures of the ribs, vertebrae, and long bones have been reported in children with Cushing's syndrome (545,548).

### Treatment

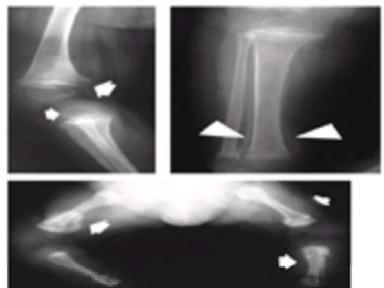
The primary treatment of Cushing's syndrome of childhood is total adrenalectomy (548). The associated fractures usually can be treated with standard immobilization techniques, but care should be taken not to increase the extent of osteopenia through excessive immobilization. In patients on corticosteroid therapy, the dose should be reduced, converted to an alternate-day schedule, or discontinued, if possible (538,549,550 and 551).

## Scurvy

The rarity of scurvy makes its diagnosis difficult. In children, scurvy may follow 6 to 12 months of a vitamin C–deficient diet (556). Because vitamin C is essential for normal collagen formation, deficiency of the vitamin results in defective osteogenesis, vascular breakdown, delayed healing, and wound dehiscence (553). With abnormal crosslinking, the defective collagen formed inhibits formation of osteoid matrix. Meanwhile, the chondroid tissue continues to be mineralized and can become completely calcified (559). Increased resorption and decreased formation of bone lead to generalized skeletal demineralization. The ends of the long bones resorb, leaving only a cuff of thickened periosteum to stabilize the epiphysis (552). Although scurvy is often due to a dietary deficiency of vitamin C (553,559,560), both aspirin and phenytoin are associated with decreased plasma levels of ascorbic acid. Vitamin C deficiency also is present in patients with myelomeningocele (558), although its contributions to fracture in that population is unclear.

Infants with scurvy present with irritability, tenderness of the lower extremities, weakness, pseudoparalysis, and possibly bleeding gums (if teeth have erupted). Subperiosteal hemorrhages may exist as well as hemorrhage into the subcutaneous tissues, muscles, urinary system, and gastrointestinal tract (555). Anemia also is a common finding. In older children, tenderness and swelling of the extremities are the most common findings. Fever was noted at the time of presentation in 70% of 52 patients in one series (557). Therefore, both osteomyelitis and septic arthritis must be initially considered in the differential diagnosis. In developing countries, older children with scurvy presenting with inability to walk may be misdiagnosed as having poliomyelitis (562).

Radiographs show profound demineralization. In advanced disease, the long bones become almost transparent with a ground-glass appearance and extreme thinning of the cortex. Calcium accumulates in the zone of provisional calcification adjacent to the physis and becomes densely white (Fränkel's line). Fractures generally occur in the scurvy line (Trummerfeld zone)—the radiolucent juxtaepiphyseal area above Frankel's line where the matrix is not converted to bone. Dense lateral spurs, known as Pelken's sign, may be seen (553). A characteristic finding of scurvy is the corner sign, in which a peripheral metaphyseal defect exists where fibrous tissue replaces absorbed cortex and cartilage (552). Cupping of the metaphysis is common in both scurvy and rickets; in rickets, the metaphysis is ragged, whereas in scurvy, the metaphysis is sharply outlined (553). The epiphysis becomes ringed with a thin, dense line (Wimberger's sign). The periosteal elevation caused by hemorrhage calcifies within 10 days of treatment with vitamin C (Fig. 6-53).



**FIGURE 6-53.** Scurvy. **A:** A 10-month-old boy presented with a 2-week history of refusal to walk with tenderness of the lower extremities. He had a history of milk and cereal intake only. There are signs of scurvy in the metaphysis (*large arrow*). The dense white line in the zone of the provisional calcification of the distal femur is known as Fränkel's line. The radiolucent juxtaepiphyseal line above the white line is known as the scurvy line. The peripheral metaphyseal defect, where fibrous tissue replaces absorbed cortex in cartilage, is known as the corner sign. Wimberger's sign is a thin, dense line surrounding the epiphysis (*small arrow*). **B:** This is a child with healing scurvy. There is marked periosteal calcification around the distal tibia (*arrows*). **C:** A newborn with scurvy. Periosteal hemorrhage has become calcified in the bones of the lower extremity (*arrows*). (Courtesy of Bruce Mewborne, M.D., San Antonio, Texas.)

## Treatment

Fractures and epiphyseal displacement occur in both infants and older children with scurvy (552,556,557,563,564). The most common sites of fracture, in order of frequency, are the distal femur, proximal humerus, costochondral junction of the ribs, and distal tibia (553). Fractures of the long bones generally are nondisplaced metaphyseal buckle fractures with mild angulation. In contrast, marked epiphyseal displacement occurs with a moderate amount of callus present even in untreated patients. Exuberant callus forms once vitamin C is administered. Standard immobilization, with administration of vitamin C, is adequate for most fractures. Remodeling potential is high in these patients (563). Even healed fractures that appear to have undergone growth arrest should just be observed, because the potential for continued growth with medical treatment of the vitamin C deficiency can be nearly normal (565). For infants who are older than 12 months of age and have begun weight bearing, spine films are recommended to rule out vertebral fractures (556).

The literature of fracture treatment in scurvy consists primarily of case reports. Hoeffel et al. (554) reported on a 14-month-old girl with scurvy with bilateral distal femoral epiphysis displacement. This condition resolved after treatment with vitamin C, but limb-length discrepancy developed on one side (561). In two patients with distal femoral fractures, healing went on to cupping of the metaphysis with an appearance similar to that in central growth arrest (557,564).

## Copper Deficiency and Scurvy-like Syndrome

Copper is a vital trace element needed in the production of collagen. Copper deficiency results in a decreased number of collagen crosslinks, with adverse effects on both bone and blood vessels (568). Copper deficiency can occur by 3 months in low-birth-weight infants (569) and after prolonged total parenteral nutrition. Copper deficiency can also develop as a result of excessive supplemental zinc ingestion (566).

Infants at risk for this syndrome are those who are primarily milk fed and are on semistarvation diets with concurrent vomiting and diarrhea (567). Both rib and wrist enlargement are frequent, (568) and neutropenia is common (569). The diagnosis is commonly based on clinical presentation and decreased levels of serum copper.

Radiographic findings in copper deficiency syndrome are very similar to those in rickets, including metaphyseal cupping, flaring, demineralization of the skeleton, and subperiosteal elevation with calcification (501). There are some radiographic differences between scurvy and copper deficiency syndrome (568). The corner sign is frequently absent in copper deficiency, the metaphyseal spurs are not strictly lateral but sickle shaped, and radiolucent bands of the metaphysis are absent. Bone age also is frequently retarded.

Pathologic fractures have been reported in copper deficiency syndrome. Cordano et al. (567) noted prompt healing of a distal femoral fracture in an infant, but the fracture recurred before treatment of the copper deficiency. Such injuries could be treated like those in scurvy, with simple immobilization and concurrent correction of the copper deficiency.

## FRACTURES IN NEUROMUSCULAR DISEASE

### Cerebral Palsy

Fractures of the extremities in patients with severe cerebral palsy are relatively rare, but their treatment can prove challenging. In a review of 1,232 institutionalized patients with cerebral palsy, McIvor and Samilson (575) documented 134 extremity fractures, primarily in quadriplegics. When the mechanism of injury was known, most of these fractures were the consequence of a fall, often associated with seizure activity. Approximately 46% of these fractures involved the femoral shaft, 6% were fractures of the head or neck of the femur, 15% involved the tibia and fibula, and 13% were humeral fractures. These authors believed that contracture or

paralytic dislocation of the hip joint predisposed these patients to femoral fractures ( [Fig. 6-54](#)).



**FIGURE 6-54. A:** This 11-year-old boy with quadriplegic cerebral palsy had a varus derotation osteotomy followed by spica casting. Two weeks after cast removal, he sustained this distal femoral fracture during physical therapy. An AP radiograph of the right knee and ( **B** ) a lateral radiograph of the right knee show a distal femoral metaphyseal fracture in a location typical for insufficiency fractures in children with neuromuscular disease. The fracture was minimally displaced and was treated with closed reduction and a carefully applied, well-padded spica cast. Note the osteopenia and the typical changes around the knee associated with long-standing knee flexion contracture.

Miller and Glazer ([578](#)) emphasized that spontaneous fractures can occur in patients with cerebral palsy without episodes of trauma, and factors such as disuse atrophy, nutritional deficiencies, and preexisting joint contractures contributed to these injuries. Nearly 50% of the full-time bedridden patients they studied developed spontaneous fractures. The diagnosis usually was delayed because the patients were noncommunicative. Anticonvulsant therapy may contribute to osteoporosis in patients with multiple fractures—low levels of serum vitamin D were seen in 42% of patients in one series ([573](#)). Fractures through osteoporotic bone can occur both above and below fixation devices.

Although long bone fractures in patients with cerebral palsy heal quickly with abundant callus ([578](#)), their treatment through either closed or open methods can be quite difficult. In a large series of patients, Mclvor and Samilson ([575](#)) recommended closed treatment through skeletal traction, hip spica cast, or long-leg cast. Approximately 65% of the femoral shaft fractures and 86% of distal femoral fractures went on to malunion. Despite malunion, most patients regained their prefracture function. Nearly 21% of their patients had refractures, and the authors believed that this was due to disuse osteopenia, inadequate reduction, or joint contractures. Closed treatment of these fractures can be complicated by the development of decubitus ulcers. Closed fractures, especially those of the femur, can become open injuries during treatment owing to spasticity or inadequate immobilization ([575,578](#)). Hip spica casts are difficult to use in patients with severe flexion contractures or dislocation of the hip. The healing time of femoral fractures treated through immobilization varies from 1 to 3.5 months ([575,578](#)). Fractures of the humerus have been treated with light hanging-arm casts or sling-and-swath bandages ([578](#)). Hip nails with side plates, compression plates, and intramedullary fixation also have been used for femoral shaft fractures in patients with cerebral palsy. The mean healing time has been 5.3 months ([575](#)).

Numerous reports note success in treating fractures of neurologically impaired children with internal or external fixation ([570,571](#)). Heinrich et al. ([571](#)) treated four femoral fractures in young patients with cerebral palsy with flexible intramedullary nails with good outcomes. Femoral neck fractures may require *in situ* pinning, but observation may be adequate in asymptomatic bedridden patients. Although he advocated open fixation of some lower extremity fractures in patients with mental retardation, Sherk ([580](#)) cautioned that some patients may have inadequate motivation to resume ambulation even with successful healing of their injuries. Medical management of these patients must also be emphasized. In patients with cerebral palsy and multiple fractures, Lee and Lyne ([573](#)) recommended metabolic supplementation, along with traditional fracture care.

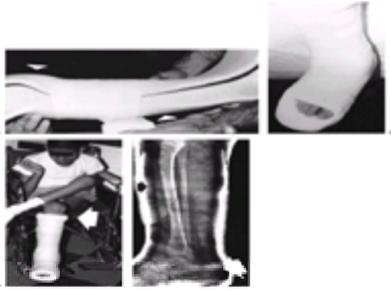
Fractures of the distal pole of the patella have been reported in children with cerebral palsy due to spasticity of the extensor mechanism of the knee in the presence of established knee flexion contracture ([572,574,579](#)). Lloyd-Roberts et al. ([574](#)) reported on eight patients with this injury who presented with deterioration in walking and decreased endurance. All had knee flexion contractures. Seven of the eight patients complained of pain and local tenderness at the distal pole of the patella. In a series of 88 patients, fragmentation was seen in only 8% ([579](#)). Patella alta and elongation of the patella are frequent in affected patients ([Fig. 6-55](#)) ([572,579](#)). Children predisposed to distal pole patellar fractures are spastic ambulators with flexion contractures of the knees, patella alta, and a history of falls ([572](#)). Extension casting may be helpful in symptomatic patients ([579](#)). If conservative treatment is unsatisfactory, then hamstring lengthening with correction of the knee flexion contracture can result in both healing of the fracture and relief of symptoms ([574,579](#)). Some authors ([572,574](#)) also have excised the avulsed distal pole of the patella to relieve chronic symptoms.



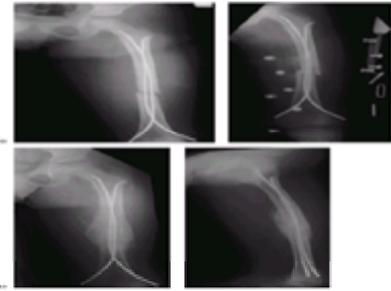
**FIGURE 6-55.** A 10-year-old girl with cerebral palsy had a healing stress fracture of an elongated patella ( *arrow* ). Patella alta is also present. The injury responded to conservative treatment.

## AUTHORS' PREFERRED METHOD OF TREATMENT

The goal of fracture treatment in cerebral palsy is to restore the child to his or her prefracture level of function. If the patient is ambulatory, conventional forms of fracture treatment should be used. In nonambulatory children with cerebral palsy, a goal of fracture care should be to preserve the ability to transfer. Special precautions should be used in closed treatment of fractures in these patients. The patients' spasticity and inability to communicate make them prone to skin problems, so casts should be properly applied and well padded, usually with felt and polyurethane foam. Extra padding should be placed over the patella, anterior ankle, and heel, and a snug cast mold should be placed above the calcaneus to prevent proximal migration of the heel ([Fig. 6-56](#)). When indicated, operative fracture fixation should be used in ambulatory patients. Elastic intramedullary nails can be a very effective way to treat femoral fractures in children with spasticity ([Fig. 6-57](#)).



**FIGURE 6-56.** Casting in neuromuscular fractures. **A:** A polyurethane foam short-leg cast is being placed on a patient. Two long rectangular sheets of foam ( *arrows*) are placed anteriorly and posteriorly over the stockinette, and Webril padding is wrapped around the foam. **B:** A long toe plate is needed to prevent injury to the foot of the patient. **C:** A thick, protective cuff of foam is formed by folding the polyurethane toward the center of the cast with the stockinette ( *arrow*). **D:** The Webril must be wrapped quite snugly to compress the foam against the underlying extremity evenly ( *black arrow*). Extra foam is placed over the anterior ankle and over the Achilles tendon to prevent proximal migration of the foot in the cast. A plaster cast is usually applied and covered with a layer of fiberglass for strength. A lateral x-ray verifies the position of the heel in the cast ( *white arrow*).



**FIGURE 6-57.** **A:** This 6-year-old boy sustained a severe closed head injury and left femur fracture when he was struck by a car. His femur was shortened 4 cm. His involuntary movements made management in traction very difficult. He was treated with retrograde titanium elastic nailing. **B:** At 13 days, early callus is seen. He was standing with a brace. **C:** At 3 months, AP and **(D)** lateral radiographs show a healed femur fracture in anatomic alignment. Nails were removed 6 months later. (From Flynn JM: Current treatment options for pediatric femur fractures. *U Penn Orthop J* 1998;11:27–35; with permission.)

Prevention should be an important part of managing fractures in children with cerebral palsy. Traditionally, long-leg casts or spica casts were used after multiple muscle lengthenings or hip osteotomies, then after several weeks, the cast was removed and therapy begun. After cast treatment, however, the osteopenia was worse, the joints were stiff, and fractures—especially in the distal femoral metaphysis—occurred during therapy or transfers. We and others ( [576,577](#)) use foam abduction pillows and knee immobilizers and an intensive therapy program in the immediate postoperative period to avoid the deconditioning, osteopenia, and joint stiffness that develop after prolonged cast immobilization. In ambulatory children who need hip osteotomies, use of rigid internal fixation allows standing and gait training within 2 weeks, preventing not only osteopenia but also the risk that the child may never regain the full level of preoperative function after a prolonged period of cast immobilization.

In nonambulatory children with severe cerebral palsy, some degree of both malunion and shortening may be accepted. Well-padded splints or casts are adequate treatment for many displaced fractures. Acute femoral shaft fractures can be treated with a heavily padded hip spica cast and distal fractures of the femur by a long-leg cast. Distal femoral buckle fractures in non-ambulatory children are safely treated with a knee immobilizer. If a long-leg cast is used for a fracture of the lower extremity and the joint of the involved side is dislocated, the rigid cast may function as a lever arm, with the posterior fracture of the proximal femur beyond the cast.

## Myelomeningocele

### Insufficiency Fractures

Children with myelomeningocele sustain fractures of the lower extremities through bone weakened by either disuse or immobilization after reconstructive surgical procedures. The incidence of fractures in several series of patients with myelomeningocele ranges from 12% to 31% ( [586,588,592,594,592,603](#)). The locations of these fractures, in order of decreasing frequency, are mid-shaft of the femur, distal femur, mid-shaft of the tibia, proximal femur, femoral neck, distal femoral physis, and proximal tibia ( [586](#)). Fractures of the distal tibia also have been reported in numerous series ( [590,594,595,602,603](#)). Both metaphyseal and diaphyseal fractures, usually resulting from minor trauma, are often either incomplete or impacted with intact periosteum ( [597](#)). They tend to heal rapidly—nonunion is rare ( [586](#)). Physeal fractures, however, may take 3 to 33 months to heal ( [603](#)).

Numerous factors predispose these patients to fracture. Children with flail limbs tend to pick up one leg and drop it out of the way when they roll over in bed or twist around while in a sitting position, and this may be enough force to cause a fracture ( [594](#)). Because protective sensation is absent, the child can neither anticipate impending injury nor be aware of injury once it has occurred. The level of neurologic involvement also affects the incidence of fractures. In a series of 76 fractures, Lock and Aronson ( [597](#)) found that 41% occurred with neurologic deficit at the thoracic level, 36% occurred with deficit at the upper lumbar level, and only 13% occurred in patients with lower lumbar or sacral deficits. Nearly 86% of these fractures occurred before 9 years of age, and 76% were associated with cast immobilization. Most fractures after immobilization occur within 4 weeks of cast removal ( [588](#)), and in one series ( [597](#)), 30% of patients with casts had multiple cluster fractures of either the casted extremity or the partially casted contralateral extremity. In addition to the inherent disuse osteoporosis from immobilization, casting causes stiffness of joints, with concentration of force on the osteoporotic bone adjacent to the joints ( [597](#)). Boytim et al. ( [583](#)) reported neonatal fractures in 6 infants with myelomeningocele and concluded that the risk of fracture was 17% for patients with thoracic or high lumbar level deficits with significant contracture of the lower extremities. The authors cautioned that particular care must be used to avoid fractures in these patients during physical therapy, x-ray positioning, or surgical procedures.

Stable fractures of the long bones may not require complete immobilization ( [588](#)). Femoral shaft fractures have been treated with padding and sandbags ( [591](#)). Skin traction of anesthetic limbs may cause massive skin necrosis ( [586,591](#)). Skeletal traction usually is inadvisable because of problems with decubitus ulcers and poor fixation in atrophic bone ( [586,591](#)). However, Drummond et al. ( [588](#)) treated 9 of 18 patients with skeletal traction without mention of failure or fixation.

Preventive measures include limiting cast immobilization after reconstructive surgery ( [587,588](#)). Solid side cushions may prevent fractures that occur when patients catch their lower extremities in bed rails ( [600](#)). The most important consideration was noted by Norton and Foley ( [599](#)) in 1959, when they stated “the quality of bone developed by activity appears to be the best protection against pathologic fractures,” and the orthopaedist should assist these patients in maintaining the highest activity level possible.

### Physeal Fractures

Fractures of the physes in patients with myelomeningocele are relatively uncommon, but for the unwary, the diagnosis can be difficult. The clinical presentation may mimic infection, with elevated temperature and swelling, redness, and local warmth at the fracture site ( [601,602](#)). Fractures of the proximal tibia may be confused with septic arthritis of the knee, with swelling up to the mid-thigh and limited knee flexion. Both the white blood cell count and erythrocyte sedimentation rate are often elevated. Immobilization of these injuries usually results in a dramatic decrease in swelling and redness of the extremity within 2 to 3 days of casting. With healing, the

x-ray picture can be alarming, with epiphyseal plate widening, metaphyseal fracture, and periosteal elevation ( 593). The radiographic differential diagnosis should include osteomyelitis, sarcoma, leukemia, and Charcot's joint ( 601,602 and 603).

Recurrent trauma to the physis, from either continued walking or passive joint motion after injury, results in an exuberant healing reaction ( Fig. 6-58) (590). Repetitive trauma delays resumption of normal endochondral ossification, resulting in abnormal thickening of the cartilage in the zone of hypertrophy and the physeal widening seen on radiographs (Fig. 6-59) (603). In a study of 19 chronic physeal fractures, Rodgers et al. (601) compared MRI with histology and found that adjacent to this thickened, disorganized zone of hypertrophy is juxtametaphyseal fibrovascular tissue that enhances gadolinium on MRI. Delayed union is common, and premature growth arrest occurs in 29% to 55% of patients (597,603). Anshuetz et al. (582) reported a unique syndrome in three patients with myelomeningocele and fracture. These children sustained fractures of the lower extremities during long-term immobilization and with cast removal went on to dramatic cardiopulmonary distress with increased pulse rate, hypotension, and increased respiratory rate. Fever also developed with decreased hematocrit levels. They suggested that the etiology of this problem was loss of intravascular volume into the fracture sites and recommended intravenous replacement of fluid losses, along with careful splinting of associated fractures.



**FIGURE 6-58. A:** This 7-year-old boy with spina bifida presented with an increasing swelling of the distal tibia. This lateral radiograph of the entire tibia shows slight widening of the distal tibial physis with subtle signs of posterior displacement of the distal tibial physis. There was a high suspicion of a fracture, so the leg was treated with a well-padded cast. **B:** This lateral radiograph, taken 2 months later, shows slight physeal widening with extraordinary periosteal new bone formation.



**FIGURE 6-59.** This 11-year-old boy with L4 myelomeningocele presented with a warm, swollen right ankle and no clear history of trauma. This AP radiograph of both ankles shows right distal tibial physeal widening with adjacent sclerosis, consistent with a chronic physeal injury. The child was treated in a well-padded, non-weight-bearing short-leg cast. He had complete resolution of symptoms when the cast was removed 6 weeks later.

Physeal injuries in patients with myelomeningocele are more difficult to treat than metaphyseal or diaphyseal long bone fractures and require lengthy immobilization with strict avoidance of weight bearing to avoid destructive repetitive trauma to the physis ( 590). Either a plaster cast or a snug-fitting total-contact orthosis is suggested for immobilization, and union can be determined by return of the physis to normal width on x-ray study ( 603). Kumar et al. (595) emphasized that application of a long-leg cast for 8 to 12 weeks is necessary to obtain satisfactory healing of physeal fractures of the tibia, and weight bearing is to be avoided until union occurs.

### Cast Technique

Immediate casting (588) and bivalved casting (591) have been used for long-bone fractures in children with myelomeningocele. A bilateral hip spica cast is suggested for supracondylar fractures of the femur, because use of a one-and-a-half hip spica cast may predispose the uninjured side to fracture ( 586). A bulky Webril dressing approximately 1.5 cm thick wrapped with an elastic bandage can be used instead of a plaster or fiberglass cast. Lock and Aronson ( 597) used Webril immobilization for an average of 1 to 3 weeks in their patients with fractures and discontinued immobilization when callus was visible. They found similar outcomes in patients treated with Webril dressings and those treated with casts; however, there was much less difficulty with pressure sores in the group treated with Webril dressings. Kumar et al. (595) used a polyurethane padded long-leg posterior plaster splint for metaphyseal and diaphyseal fractures for 3 weeks, followed by bracing.

Drennan and Freehafer (586) recommend a well-padded cast for 2 to 3 weeks for infants with fracture and braces or Webril immobilization for incomplete fractures that followed surgery. Injuries with deformity were placed in a cast. Mobilization was begun as soon as practical to prevent further osteopenia—patients with shaft fractures began ambulation 2 weeks after injury. Shortening was not a problem in their series. Lock and Aronson ( 597) cautioned that brace treatment of acute fractures may cause pressure sores. Drummond et al. (588) reported on 18 fractures treated by closed techniques that resulted in three malunions, two shortenings, and two episodes of pressure sores; one patient had four refractures. Drabu and Walker ( 585) noted a mean loss of knee movement of 58 degrees in 67% of fractures about the knee. The stiffness began 2 months after fracture and was well established by 6 months but resolved almost completely in all patients 3 years after injury. They suggested that aggressive physical therapy to restore knee motion is probably not necessary in these injuries.



## OPERATIVE TREATMENT

Operative fixation of fractures in children with myelomeningocele is associated with a high rate of infection ( 591). Wenger et al. (603) reported that most patients with proximal femoral epiphyseal displacement can be treated with hip spica casts. Reduction and pinning with subtrochanteric osteotomy may be necessary in certain patients. Bailey-Dubow rods may be valuable in multiple recurrent pathologic fractures of the femoral or tibial shaft ( 596). If operative treatment is necessary, it should be noted that the incidence of malignant hyperthermia seems to be higher in patients with myelomeningocele than in other children ( 581).

### Latex Allergy

Life-threatening anaphylactic reactions due to latex allergy in children with myelomeningocele have been reported with increasing frequency ( 584,597). Minor allergic reactions, such as rash, edema, hives, and respiratory symptoms are common when children with myelodysplasia are exposed to latex products such as gloves, catheters, and balloons. Between 18% and 40% of children with myelodysplasia are allergic to latex ( 589). Meeropol et al. (598) emphasized that every child with myelomeningocele should be screened for latex allergy, and those with a positive history should be evaluated individually by the anesthesiologist for preoperative prophylaxis. Current preoperative prophylaxis begins 24 hours before surgery and is continued for 24 hours after surgery. Medications used include diphenhydramine, 1 mg/kg every 6 hours (maximum 50 mg); methylprednisolone, 1 mg/kg every 6 hours (maximum 125 mg); and cimetidine, 5 mg/kg every 6 hours (maximum 300 mg). A latex-free environment must also be provided throughout the hospitalization.

## AUTHORS' PREFERRED METHOD OF TREATMENT

In nonambulatory patients, mild malunion and shortening can be tolerated, and stable or minimally angulated fractures can be treated with either polyurethane splints or Webril dressings. Fractures with significant deformity may require reduction and immobilization in a cast heavily padded with polyurethane foam. In children who walk, fractures should be carefully aligned with heavily padded casts that allow continued protective weight bearing, if possible. Hip spica casts may be necessary for femoral shaft fractures. Fractures of the proximal femur should be treated by immobilization and any later deformity corrected by osteotomy. Any patient considered for operative intervention should be screened for latex allergy, and if needed, given appropriate prophylaxis. Physeal fractures are treated with padded long-leg casts and non-weight bearing. Long-term follow-up is encouraged for physeal injuries because of the risk of growth arrest. Significant discrepancies can be addressed through contralateral epiphysiodesis or bridge resection.

### Muscular Dystrophy

Fractures of the lower extremity in children with Duchenne muscular dystrophy must be managed so as not to cause premature loss of the ability to walk (609) or transfer (606). In patients 9 to 10 years old, increasing muscle weakness and joint contractures contribute to falls, and a loss of normal muscle bulk and fat limit the cushioning on impact (608). Patients in lower extremity braces seem to sustain few fractures in falls (609), probably because the overlying orthoses provide some protection (608). Patients confined to a wheelchair can fall because they have poor sitting balance, and fractures are frequent because these patients are more osteoporotic than ambulatory individuals (608).

Concentric "osseous atrophy" occurs in the long bones of patients with Duchenne muscular dystrophy; osteoporosis is also common (604,607). Fractures are seldom displaced and are frequently minimally painful because there is minimal muscle spasm (608). Fractures tend to heal rapidly. The most commonly fractured bone is the femur (Fig. 6-60) (605,606,608), followed by the proximal humerus (608).



**FIGURE 6-60. A:** This 14-year-old nonambulatory boy with Duchenne muscular dystrophy crashed his wheelchair into the bumper of a stationary car, sustaining a displaced subcapital femoral fracture. This AP pelvis radiograph at the time of injury shows a displaced fracture and marked osteopenia of the pelvis and proximal femur. **B:** The fracture was treated by reduction and internal fixation with two cannulated screws.

There are two goals of fracture care in children with muscular dystrophy: limb stability and maintenance of maximal function during fracture healing. In ambulatory patients, treatment methods should allow children to maintain the ability to walk as the fracture heals. When ambulatory ability is tenuous, even minor bruises or ankle sprains (609) may end walking ability. As little as 1 week in a wheelchair can prematurely end ambulation (609); patients at bed rest for more than 2 weeks will likely lose the ability to ambulate (606). Hsu (605) reported that 25% of ambulatory patients with muscular dystrophy lost the ability to walk after sustaining fractures. In one of these patients, the ankle was casted in 20 degrees of plantar flexion, and the resulting contracture prevented ambulation at the end of treatment.

Treatment of specific fractures should be individualized. Upper extremity fractures can be treated with lightweight slings (608). Lower extremity fractures can be treated with either light walking casts or long-leg double upright braces (608,609). Splints also can be used until the patients are pain free. Routine activities are begun as soon as possible (605). Protected standing and ambulation with physical therapy are crucial in maintaining independent ambulation (609).

Hsu and Garcia-Ariz (606) reported on 20 femoral fractures in 16 patients with muscular dystrophy. Six of the seven ambulatory patients were able to walk after treatment. In the nonambulatory patients in this series, most had supracondylar femoral fractures (Fig. 6-61), which were splinted for 2 to 3 weeks, with emphasis on physical therapy to maintain functional abilities. Although union was achieved rapidly, hip and knee flexion contractures often increased in these patients, and up to 20 degrees of angulation of the fracture was routinely accepted. One patient with slipped capital femoral epiphysis was treated successfully with pinning *in situ*.



**FIGURE 6-61.** This 13-year-old nonambulatory boy with Duchenne muscular dystrophy sustained this typical impacted, minimally displaced distal femoral metaphyseal fracture when he fell from his wheelchair. Note the marked periarticular osteopenia.

## AUTHORS' PREFERRED METHOD OF TREATMENT

The first goal of fracture treatment in children with muscular dystrophy is to avoid making matters worse. The patient should be mobilized as soon as possible in a lightweight cast or orthosis. Aggressive physical therapy should be used to maintain functional status. In a very young child, mid-shaft femoral fractures can be treated by traction and hip spica techniques, but in an older patient, ambulatory cast bracing might be a better choice.

### Arthrogryposis and Poliomyelitis

Arthrogryposis is a rare disorder with an incidence of 3 in 10,000 live births (614). In this disease, muscle fiber is lost in utero, joint motion is limited during development, and taut ligaments and capsular tightness result in joint stiffness. Dislocations can occur with severe shortening of the involved muscles. Fractures may occur in 25% of infants with arthrogryposis multiplex congenita (610). Both difficult delivery and forceful manipulation of the extremities in these children can lead to fracture (610). Diamond and Alegado (610) reported on 16 fractures in 9 infants with arthrogryposis; an ipsilateral dislocated hip was present in 35% of patients. Most fractures involved the femur, with the remainder mostly tibial fractures, one humeral fracture, and one clavicular fracture. Epiphyseal separations occurred in the proximal tibia, distal femur, and proximal humerus. Clinical symptoms included poor feeding, irritability, and fussiness when handled. The involved extremity was

thickened, and there was often an increased white blood cell count. Plain radiographs after acute injury, especially with epiphyseal separations, were not helpful, and arthrography was used in one patient to evaluate a distal femoral epiphyseal separation. With healing, these fractures develop exuberant callus with rapid union, and ready remodeling of angulated mid-shaft fractures.

Short-term immobilization is adequate to treat undisplaced fractures in these patients. Heavy plaster splints are not necessary (612). Some authors (611) recommend closed reduction of acutely displaced epiphyseal fractures and fractures with rotatory malalignment. Postnatal fractures are most common in patients with either knee contracture or dislocation of the hip, and postnatal injury could possibly be reduced by avoidance of forceful manipulation of these extremities. Older patients with lower extremity contractures do not seem to have difficulty with pathologic fractures (613).

Acute poliomyelitis has become a relatively rare disease in most Western countries but occasionally occurs in children who live in less developed countries. There are few reports in the literature concerning fractures in patients with poliomyelitis. Robin (612) reported 62 fractures in patients with poliomyelitis. More than half were fractures of the femur, and 90% of those injuries were supracondylar fractures. More than half of the fractures occurred after cast immobilization, and joint stiffness also was associated with a significant number of fractures. There were no epiphyseal injuries in this series.

Treatment of these fractures is simple immobilization. Because most fractures have very little displacement, reduction is seldom necessary; if there is preexisting deformity, manual osteoclasia through the fracture site can be used to correct deformities. Robin (612) stressed that joint mobility must be obtained before general mobilization of the patient to reduce the incidence of fracture after plaster immobilization. He also emphasized that fractures in these patients heal rapidly, and immobilization times should be reduced accordingly, with walking or standing in casts to decrease osteoporosis.

### Spinal Cord Injury

Fortunately, childhood spinal cord injury is rare, and reports of pathologic fractures usually are included in larger series of patients with fractures and myelomeningocele because of the clinical similarities in presentation (621,622). Fractures of the femur, especially supracondylar fractures, are most common (619), but tibial fractures also are common (619). In most patients, these are pathologic fractures through osteoporotic bone. Children may be much more susceptible to these fractures than adults. Although fractures in paraplegics appear to heal rapidly with abundant callus (619), animal experiments suggest that in denervated limbs, the quality of the callus in fractures is compromised (620). Children with traumatic peripheral nerve lesions may have distal tibial physeal lesions similar to those in patients with myelomeningocele, neuropathic arthropathy of the small joints of the foot, and soft tissue ulcers (623).

Conservative treatment of fractures in patients with spinal cord injury is most commonly recommended. Skin traction is contraindicated because of the possibility of skin necrosis (618). Comarr et al. (616) also use pillow sandbag splints and, for certain fractures, treat patients with a turning frame. Open reduction and internal fixation of these fractures is controversial. The conservative techniques used for the treatment of similar fractures in children with myelomeningocele might best be applied to fractures in children with traumatic paraplegia (617,621,622).

Although crutch and brace ambulation in paraplegics reportedly restores bone integrity (615), fractures continue to occur through osteoporotic bone in children actively ambulating with brace support (621). Some authors advocate light protective braces (616), but Katz (621) suggested that continuous splinting will worsen the disease osteoporosis. He recommended careful manipulation of the lower extremities when they are out of ambulatory braces to reduce the incidence of fracture. Robin (622) emphasized that immobilization should be discontinued as soon as possible after fracture healing. Patient activity should be limited until knee motion is restored.



### AUTHORS' PREFERRED METHOD OF TREATMENT

Heavily padded casts or splints are recommended for most lower extremity fractures in children with traumatic paraplegia. Returning to ambulation with protection by walking braces is permitted once callus and joint motion are adequate. Every effort should be made to restore the child's prefracture function. Moderate malunion and shortening are acceptable in patients who are nonambulatory. Operative treatment of such fractures should be reserved for selected patients whose function would be significantly compromised by less than anatomic reduction.

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### CHAPTER REFERENCES

1. Clark CR, Morgan C, Sonstegard DA, Matthews LS. The effect of biopsy-hole shape and size on bone strength. *J Bone Joint Surg Am* 1977;59:213-217.
2. Copley L, Dormans JP. Benign pediatric bone tumors, evaluation and treatment. *Pediatr Clin North Am* 1996;43:949-966.
3. Hipp JA, Springfield DS, Hayes WC. Predicting pathologic fracture risk in the management of metastatic bone defects. *Clin Orthop* 1995;312:120-135.

### Unicameral Bone Cyst

4. Ahn JI, Park JS. Pathological fractures secondary to unicameral bone cysts. *Int Orthop* 1994;18:20-22.
5. Badgley CE. Unicameral cyst of the long bones: treatment by crushing cystic walls and onlay grafts. *J Bone Joint Surg Am* 1957;39A:1429-1430.
6. Baker DM. Benign unicameral bone cyst: a study of forty-five cases with long-term follow-up. *Clin Orthop* 1970;71:140-151.
7. Boseker EH, Bickel WH, Dahlin DC. A clinicopathologic study of simple unicameral bone cysts. *Surg Gynecol Obstet* 1968;127:550-560.
8. Campanacci M, Capanna R, Picci P. Unicameral and aneurysmal bone cysts. *Clin Orthop* 1986;204:25-36.
9. Campbell RM Jr. Problem injuries in unique conditions of the musculoskeletal system. In: Rockwood CA Jr, Wilkins KE, Beaty JH, eds. *Fractures in children*, Vol 3, 4th ed. Philadelphia: Lippincott-Raven, 1996;167-320.
10. Campos OP. Treatment of bone cyst by intracavity injection of methyl prednisone acetate: a message to orthopaedic surgeons. *Clin Orthop* 1982;165:43-48.
11. Capanna R, Albinini U, Caroli GC, Campanacci M. Contrast examination as a prognostic factor in the treatment of solitary bone cysts by cortisone injection. *Skeletal Radio*. 1984;12:97-102.
12. Capanna R, Monte AD, Gitelis S, Campanacci M. The natural history of unicameral bone cysts after steroid injection. *Clin Orthop* 1982;166:204-211.
13. Chigira M, Maehara S, Arita S, Udagawa E. The aetiology and treatment of simple bone cyst. *J Bone Joint Surg Br* 1983;65:633-637.
14. Cohen J. A simple bone cyst: studies of cyst fluid in six cases with a theory of pathogenesis. *J Bone Joint Surg Am* 1960;42:609-616.
15. Cohen J. Unicameral bone cyst: a current synthesis of reported cases. *Orthop Clin North Am* 1977;8:715-736.
16. Colville MR, Aronson DD, Precevski P, Crissman JD. The systemic and local effects of an intramedullary injection of methyl prednisolone acetate in growing rabbits. *J Pediatr Orthop* 1987;7:412-414.
17. Czitrom AA, Pritzker KPH. Simple bone cysts causing collapse of the articular surface of the femoral head and incongruity of the hip joint: a case report. *J Bone Joint Surg Am* 1980;62:842-845.
18. DePalma L, Santucci A. Treatment of bone cysts with methyl prednisolone acetate: a 9 to 11 year follow-up. *Int Orthop* 1987;11:23-28.
19. Fahey JJ, O'Brien ET. Subtotal resection and grafting in selected cases of solitary unicameral bone cyst. *J Bone Joint Surg Am* 1973;55:59-68.
20. Fernbach SK, Blumenthal DH, Poznanski AK. Radiographic changes in unicameral bone cyst following direct injection of steroids: a report on 14 cases. *Radiology* 1981;140:689-695.
21. Ganland JJ, Cole FL. Modern concepts in treatment of unicameral bone cyst of the proximal humerus. *Orthop Clin North Am* 1975;6:487-498.
22. Glaser DL, Dormans JP, Stanton RP, Davidson RS. Surgical management of calcaneal unicameral bone cysts. *Clin Orthop* 1999;360:231-237.
23. Harrer MF, Dormans JP, Stanton R, Davidson RS. Diaphyseal unicameral bone cysts in children. *Orthop Transactions* 1997-1998;21:1187-1188.
24. Jaffe HL. *Tumors and tumorous conditions of bone and joints*. Philadelphia: Lea & Febiger, 1958.
25. Kaelin AJ, MacEwen GD. Unicameral cysts: natural history and risk of fracture. *Int Orthop* 1989;13:275-282.
26. Khermouh O, Weissman SL. Coxa vara, avascular necrosis and osteochondritis dissecans complicating solitary bone cyst of the proximal femur. *Clin Orthop* 1977;126:143-146.
27. Killian JT, Wilkinson L, White S, Brassard M. Treatment of unicameral bone cyst with demineralized bone matrix. *J Pediatr Orthop* 1998;18:621-624.
28. Kresler TW, Kling TF, Rougraff BT. Unicameral bone cysts. *Curr Opin Orthop* 1994;5:75-81.
29. Malkawi H, Shannak A, Amr S. Surgical treatment of pathological subtrochanteric fractures due to benign lesions in children and adolescents. *J Pediatr Orthop* 1984;4:63-69.
30. McKay DW, Nason SS. Treatment of unicameral bone cyst by subtotal resection without grafts. *J Bone Joint Surg Am* 1977;59A:515-518.
31. Moed BR, LaMont RL. Unicameral bone cysts complicated by growth retardation: a report of 3 cases. *J Bone Joint Surg Am* 1982;64:1379-1381.
32. Moreau G, Letts M. Unicameral bone cysts in the calcaneus in children. *J Pediatr Orthop* 1994;14:101-104.
33. Nakamura T, Takagi K, Kitgawa T, Harada M. Microdensity of solitary bone cyst after steroid injection. *J Pediatr Orthop* 1988;8:566-568.
34. Neer CS II, Francis KC, Johnston AD, Kiernan HA Jr. Current concepts on treatment of solitary unicameral bone cyst. *Clin Orthop* 1973;97:40-51.
35. Neer CS II, Frands KC, Marcove RC, et al. Treatment of unicameral bone cyst: a follow-up study of one hundred seventy-five cases. *J Bone Joint Surg Am* 1966;48:731-745.

36. Oppenheim WL, Galleno H. Operative treatment versus steroid injection in the management of unicameral bone cyst. *J Pediatr Orthop* 1984;4:1-7.
37. Peltier LF, Jones RH. Treatment of unicameral bone cyst by curettage and packing with plaster-of-paris pellets. *J Bone Joint Surg Am* 1978;60:820-822.
38. Reynolds J. The "fallen fragment sign" in the diagnosis of unicameral bone cyst. *Radiology* 1969;92:949-953.
39. Robins PR, Peterson HA. Management of pathologic fractures through unicameral bone cyst. *JAMA* 1972;222:80-81.
40. Rosenborg M, Mortensson W, Hirsch G, et al. Considerations in corticosteroid treatment of bone cysts. *J Pediatr Orthop* 1989;9:240-243.
41. Rougraff BT, Kling TF. Treatment of inactive unicameral bone cysts with injectable autogenic bone graft and autogenous bone marrow. *Orthop Trans* 1995-1996;19:788.
42. Santori F, Ghera S, Castelli V. Treatment of solitary bone cyst With intramedullary nailing. *Orthopedics* 1988;11:873-878.
43. Scaglietti O, Marchetti PG, Bartolozzi P. The effects of methyl prednisolone acetate in the treatment of bone cysts: results of three years of follow-up. *J Bone Joint Surg Br* 1979;61:200-204.
44. Schreuder HWB, Conrad EU, Bruckner JD, et al. Treatment of simple bone cysts in children with curettage and cryosurgery. *J Pediatr Orthop* 1997;17:814-820.
45. Shindell R, Hurman WW, Lippiello L, Connolly JF. Prostaglandin levels in unicameral bone cyst treated by intralesional steroid injection. *J Pediatr Orthop* 1989;9:516-519.
46. Siegel IM. Brisement force with controlled collapse in treatment of solitary unicameral bone cyst. *Arch Surg* 1966;92:109-114.
47. Spence KF, Sell KW, Brown RH. Solitary bone cyst: treatment with freeze-dried cancellous bone allograft. *J Bone Joint Surg Am* 1969;51:87-96.
48. Taneda H, Zauma H. Avascular necrosis of the femoral epiphysis complicating a minimally displaced fracture of solitary bone cyst of the neck of the femur in a child. *Clin Orthop* 1994;304:172-175.
49. Weisel A, Hecht HL. Development of a unicameral bone cyst. *J Bone Joint Surg Am* 1980;62:664-666.

#### Aneurysmal Bone Cyst

50. Besse BE, Dahlin DC, Pugh DG, Ghormley RK. Aneurysmal bone cyst: additional considerations. *Clin Orthop* 1956;7:93-102.
51. Biesecker JL, Marcov RC, Huvos AG, Mike V. Aneurysmal bone cyst: a clinical pathologic study of 66 cases. *Clinicopathol Cancer* 1970;26:615-625.
52. Bonakdarpour A, Levy WM, Aegener E. Primary and secondary aneurysmal bone cyst: a radiological study of 75 cases. *Radiology* 1978;126:75-83.
53. Burkhalter WE, Schroeder FC, Eversmann WW Jr. Aneurysmal bone cyst occurring in the metacarpals: a report of three cases. *J Hand Surg* 1978;3:579-584.
54. Campanacci M, Capanna R, Picci P. Unicameral and aneurysmal bone cyst. *Clin Orthop* 1986;204:25-36
55. Capanna R, Albinini U, Picci P, et al. Aneurysmal bone cyst of the spine. *J Bone Joint Surg Am* 1985;67:527-531.
56. Capanna R, Springfield DS, Biagini R, et al. Juxtaepiphyseal aneurysmal bone cyst. *Skeletal Radio*. 1985;13:21-25.
57. Clough JR, Price CHG. Aneurysmal bone cyst: pathogenesis and long-term treatment of results. *Clin Orthop* 1973;97:52-63.
58. Dabiezies EJ, D'Ambrosia RD, Chuniard RG, Ferguson AB Jr. Aneurysmal bone cyst after fracture: a report of three cases. *J Bone Joint Surg Am* 1982;64:617-621.
59. Dabska M, Buraszewski J. Aneurysmal bone cyst pathology, clinical course and radiologic appearances. *Cancer* 1969;23:371-389.
60. Dyer R, Stelling CB, Fechner RE. Epiphyseal extension of an aneurysmal bone cyst. *AJR Am J Roentgenol* 1981;137:172-173.
61. Fraser RK, Coates CJ, Cole WG. An angiostatic agent in treatment of a recurrent aneurysmal bone cyst. *J Pediatr Orthop* 1993;13:668-671.
62. Freiberg AA, Loder RT, Heidelberger KL, Hensing RN. Aneurysmal bone cysts in young children. *J Pediatr Orthop* 1994;14:86-91.
63. Ginsburg LD. Congenital aneurysmal bone cyst: a report with comments on the role of trauma in the pathogenesis. *Radiology* 1974;110:175-176.
64. Green JA, Bellemore MC, Marsden FW. Embolization in the treatment of aneurysmal bone cysts. *J Pediatr Orthop* 1997;17:440-443.
65. Guibaud L, Herbreteau D, Dubois J, et al. Aneurysmal bone cysts: percutaneous embolization with an alcoholic solution of Zein—series of 18 cases. *Radiology* 1998;208:369-373.
66. Hay MC, Paterson D, Taylor TKF. Aneurysmal cyst of the spine. *J Bone Joint Surg Br* 1978;60:406-411.
67. Hooper JC. Aneurysmal bone cyst after penetrating the tibial epiphysis after curettage. *Med J Aust* 1971;1:200-201.
68. Hudson TM. Scintigraphy of aneurysmal bone cyst. *Am J Roentgenol* 1984;142:761-765.
69. Jaffe KA, Dunham WK. Treatment of benign lesions of the femoral head and neck. *Clin Orthop* 1990;257:134-137.
70. Kaufman RA, Towbin RB. Telangiectatic osteosarcoma simulating the appearance of an aneurysmal bone cyst. *Pediatr Radio* 1981;11:102-104.
71. Koskinen EVS, Visuri TI, Holmstrom T, Roukkula MA. Aneurysmal bone cyst. Evaluation of resection and of curettage in twenty cases. *Clin Orthop* 1976;118:136-146.
72. MacPherson RI. Aneurysmal bone cyst of spine diagnosed by percutaneous opacification. *J Can Assoc Radiol* 1980;31:210-212.
73. Marcove RC, Sheth DS, Takemoto S, Healey JH. The treatment of aneurysmal bone cyst. *Clin Orthop* 1995;311:157-163.
74. Ozaki T, Hillmann A, Lindner N, Winkelmann W. Cementation of primary aneurysmal bone cysts. *Clin Orthop* 1997;337:240-248.
75. Papagelopoulos PJ, Currier BL, Shaughnessy WJ, et al. Aneurysmal bone cysts of the spine. *Spine* 1998;23:621-628.
76. Reed RJ, Rothenberg M. Lesions of bone that may be confused with aneurysmal bone cysts. *Clin Orthop* 1964;35:150-162.
77. Schreuder HWB, Veth R, Pruszczynski M, et al. Aneurysmal bone cysts treated by curettage cryotherapy and bone grafting. *J Bone Joint Surg Br* 1997;79:20-25.
78. Sullivan RJ, Meyer JS, Dormans JP, Davidson RS: Diagnosing aneurysmal and unicameral bone cysts with magnetic resonance imaging. *Clin Orthop* 1999;366:186-190.
79. Tillman BP, Dahlin DC, Lipscomb PR, Stewart JR. Aneurysmal bone cyst: an analysis of ninety-five cases. *Mayo Clin Proc* 1968;43:478-495.
80. Torpey B, Dormans JP, Drummond DS. Titanium instrumentation and fusion for spinal cord tumor stabilization. *J Spinal Disor* 1995;8:76-81.
81. Turker RJ, Mardjetko S, Lubicky J. Aneurysmal bone cysts of the spine: excision and stabilization. *J Pediatr Orthop* 1998;18:209-213.

#### Fibrous Cortical Defect and Nonossifying Fibroma

82. Arata MA, Peterson HA, Dahlin DC. Pathologic fractures Through non-ossifying fibromas. *J Bone Joint Surg Am* 1981;63:980-988.
83. Campbell CJ, Harkess J. Fibrous metaphyseal defect of bone. *Surg Gynecol Obstet* 1957;104:329-336.
84. Cunningham JB, Ackerman LV. Metaphyseal fibrous defects. *J Bone Joint Surg Am* 1956;38:797-808.
85. Dahlin DC. *Bone tumors: general aspects and data on 6,221 cases*. Springfield, IL: Charles C Thomas, 1978.
86. Devlin JA, Bowman HE, Mitchell WL. Nonosteogenic fibroma of bone: a review of the literature with addition of six cases. *J Bone Joint Surg Am* 1955;37:472-486.
87. Drennan DB, Maylahn DJ, Fahey JJ. Fractures through non-ossifying fibromas. *Clin Orthop* 1974;103:82-88.
88. Easley ME, Kneisl JS. Pathologic fractures through nonossifying fibromas: Is prophylactic treatment warranted? *J Pediatr Orthop* 1997;17:808-813.
89. Evans GA, Park WM. Familial multiple nonosteogenic fibromata. *J Bone Joint Surg Br* 1978;60:416-419.
90. Hatcher CH. The pathogenesis of localized fibrous lesions in the metaphyses of long bones. *Ann Surg* 1945;122:1016-1030.
91. Phalen JT. Fibrous cortical defect in nonosseous fibroma of bone. *Surg Gynecol Obstet* 1964;119:807-810.
92. Ritschl P, Karmel F, Hajek P. Fibrous metaphyseal defects: determination of their origin and natural history using a radiomorphological study. *Skeletal Radio*. 1988;17:8-15.
93. Selby S. Metaphyseal cortical defects in the tubular bones of growing children. *J Bone Joint Surg Am* 1961;43:395-400.

#### Giant Cell Tumor

94. Campanacci M, Baldini N, Boriani S, Sudanes A. Giant cell tumor of bone. *J Bone Joint Surg Am* 1987;69:106.
95. Dahlin DC, Cupps RE, Johnson EW Jr. Giant-cell tumor: a study of 195 cases. *Cancer* 1970;25:1061.
96. McDonald DJ, Sim FH, McLeod RA, Dahlin DC. Giant cell tumor of bone. *J Bone Joint Surg Am* 1986;68:235.
97. Muscolo DL, Ayerza MA, Calabrese ME, Gruenberg M. The use of a bone allograft for reconstruction after resection of a giant cell tumor close to the knee. *J Bone Joint Surg Am* 1993;75:1657.
98. Sung HW, Kuo DP, Shu WP, et al. Giant cell tumor of bone: analysis of two hundred and eight cases: Chinese patients. *J Bone Joint Surg Am* 1982;64:755.

#### Enchondroma

99. Bauer RD, Lewis MM, Posner MA. Treatment of enchondromas of the hand with allograft bone. *J Hand Surg* 1988;13A:908-916.
100. Bean W. Dyschondroplasia and hemangiomas (Maffucci's syndrome): II. *Arch Intern Med* 1958;102:544-550.
101. Jaffe HL, Lichtenstein L. Solitary benign enchondroma of bone. *Arch Surg* 1943;46:480-493.
102. Lewis RJ, Ketcham AS. Maffucci's syndrome: functional and neoplastic significance. *J Bone Joint Surg Am* 1973;55:1465-1479.

#### Osteochondroma

103. Cardelia JM, Dormans JP, Drummond DS, et al. Proximal fibular osteochondroma with associated peroneal nerve palsy: a review of six cases. *J Pediatr Orthop* 1995;15:574-577.
104. Mahboubi S, Dormans JP, D'Angio G. Malignant degeneration of radiation-induced osteochondroma. *Skeletal Radio*. 1997;26:195-198.
105. Theodorou SD, Karamitsos S, Tsouparopoulos D, Hatzipavlou AG. Rare complications of exostosis: fractures and injuries to the common peroneal nerve. *Acta Orthop Belg* 1978;44:496-505.

#### Eosinophilic Granuloma

106. Crone-Munzebrock W, Brassow F. A comparison of radiographic and bone scan findings in histiocytosis X. *Skeletal Radio*. 1983;9:170-173.
107. Egler RM, Thompson RC, Boute PA, Nesbit ME. Intralesional infiltration of corticosteroids in localized Langerhans' cell histiocytosis. *J Pediatr Orthop* 1992;12:811-814.
108. Fowles JV, Bobeckko WP. Solitary eosinophilic granuloma in bone. *J Bone Joint Surg Br* 1970;52:238-243
109. Greis PE, Hankin FM. Eosinophilic granuloma: the management of solitary lesions of bone. *Clin Orthop* 1990;257:204-211.

110. Levine SE, Dormans JP, Meyer JS, Corcoran TA. Langerhan's cell histiocytosis of the spine in children. *Clin Orthop* 1996;323:288–293
111. Lieberman PH, Jones CR, Steinman RM, et al. Langerhan's cell (eosinophilic) granulomatosis: a clinicopathologic study encompassing 50 years. *Am J Surg Pathol* 1996;20:519–522.
112. Mammano S, Candiotti S, Balsano M. Cast and brace treatment of eosinophilic granuloma of the spine: long-term follow-up. *J Pediatr Orthop* 1997;17:821–827.
113. McCullough CJ. Eosinophilic granuloma of bone. *Acta Orthop Scand* 1980;51:389–398.
114. Meyer JS, Harty MP, Mahboubi S, et al. Langerhans' cell histiocytosis: presentation and evolution of radiologic findings with clinical correlation. *Radiographics* 1995;15:1135–1146.
115. Osenbach RK, Youngblood LA, Menezes AH. Atlanto-axial instability secondary to solitary eosinophilic granuloma of C2 in a 12-year-old girl. *J Spinal Disord* 1990;3:408–412.
116. Raab P, Hohmann F, Kühl J, Krauspe R. Vertebral remodeling in eosinophilic granuloma of the spine: a long-term follow-up. *Spine* 1998;23:1351–1354.
117. Sbarbaro JL Jr., Frands KC. Eosinophilic granuloma of bone. *JAMA* 1961;1:706–710.
118. Seimon LP. Eosinophilic granuloma of the spine. *J Pediatr Orthop* 1981;1:371–376.
119. Yasko AW, Fanning CV, Ayala AG, et al. Percutaneous techniques for the diagnosis and treatment of localized Langerhan's cell histiocytosis (eosinophilic granuloma of bone). *J Bone Joint Surg Am* 1998;80:219–228.

#### Malignant Bone Tumor

120. Abudu A, Sferopoulos NK, Tillman RM, et al. The surgical treatment and outcome of pathologic fractures in localised osteosarcoma. *J Bone Joint Surg Br* 1996;78:694–698.
121. Ayala A, Ro J, Fanning C, et al. Core needle biopsy and fine-needle aspiration in the diagnosis of bone and soft-tissue lesions. *Hematol Oncol Clin North Am* 1995;9:633.
122. Berrey BH, Lord CF, Gebhardt MC, Mankin HJ. Fractures of allografts: frequency, treatment, and end-results. *J Bone Joint Surg Am* 1990;72:825–833.
123. Ehara S, Kattapuram SV, Egglin TK. Ewing's sarcoma: radiographic pattern of healing and bony complications in patients with long-term survival. *Cancer* 1991;68:1531–1535.
124. Enneking WF. A system of staging musculoskeletal neoplasms. *Instr Course Lect* 1988;37:8.
125. Gebhardt MC. Molecular biology of sarcomas. *Orthop Clin North Am* 1996;27:421–429.
126. Hahn M, Dormans JP. Primary bone malignancies in children. *Curr Opin Pediatr* 1996;8:71–74.
127. Himmelstein BP, Dormans JP. Malignant bone tumors of childhood. *Pediatr Clin North Am* 1996;43:967–984.
128. Kruzelock R, Hansen M. Molecular genetics and cytogenetics of sarcomas. *Hematol Oncol Clin North Am* 1995;9:513.
129. Letson GD, Greenfield GB, Heinrich SD. Evaluation of the child with a bone or soft tissue neoplasm. *Orthop Clin North Am* 1996;27:431–451.
130. Massengill A, Seeger L, Eckardt J. The role of plain radiography, computed tomography, and magnetic resonance imaging in sarcoma evaluation. *Hematol Oncol Clin North Am* 1995;9:571.
131. Mankin HJ, Lange TA, Spanier SS. The hazards of biopsy in patients with malignant primary bone and soft-tissue tumors. *J Bone Joint Surg Am* 1982;64:1121–1127.
132. Peabody TD, Simon MA. Making the diagnosis: keys to successful biopsy in children with bone and soft-tissue tumors. *Orthop Clin North Am* 1996;27:453–459.
133. Pochanugool L, Subhadharaphandou T, Dhanachai M, et al. Prognostic factors among 130 patients with osteosarcoma. *Clin Orthop* 1997;345:206–214.
134. Scully SP, Temple HT, O'Keefe RJ, et al. The surgical treatment of patients with osteosarcoma who sustain a pathologic fracture. *Clin Orthop* 1996;324:227–232.
135. Simon M: Biopsy of musculoskeletal tumors. *J Bone Joint Surg Am* 1982;64:1253.
136. San-Julian M, Canadell J. Fractures of allografts used in limb preserving operations. *Int Orthop* 1998;22:32–36.
137. Wunder JS, Paulian G, Huvos AG, et al. The histologic response to chemotherapy as a predictor of the oncological outcome of operative treatment of Ewing's sarcoma. *J Bone Joint Surg Am* 1998;80:1020–1033.

#### Fibrous Dysplasia

138. Albright F, Butler AM, Hampton AO, Smith P. Syndrome characterized by osteitis fibrosa disseminata, areas of pigmentation and endocrine dysfunction, with precocious puberty in females: a report of five cases. *N Engl J Med* 216:727-746, 1937.
139. Alman BA, Greel DA, Wolfe HJ. Activating mutations of Gs protein in monostotic lesions of bone. *J Orthop Res* 1996;14:311–315.
140. Breck L. Treatment of fibrous dysplasia of bone by total femoral plating and hip nailing. *Clin Orthop* 1972;82:82–83.
141. Bryant DD, Grant RE, Tang D. Fibular strut grafting for fibrous dysplasia of the femoral neck. *J Natl Med Assoc* 1992;84:893–897.
142. Daffner RH, Kirks DR, Gehweiler JA Jr, Heaston DK. Computed tomography of fibrous dysplasia. *Am J Roentgenol* 1982;139:943–948.
143. DePalma AF, Ahmad I. Fibrous dysplasia associated with shepherd's crook deformity of the humerus. *Clin Orthop* 1973;97:38–39.
144. Enneking WF, Gearen PF. Fibrous dysplasia of the femoral neck. *J Bone Joint Surg Am* 1986;68:1415.
145. Funk FJ, Wells RE. Hip problems in fibrous dysplasia. *Clin Orthop* 1973;90:77–82.
146. Grabias SL, Campbell CJ. Fibrous dysplasia. *Orthop Clin North Am* 1977;8:771–783.
147. Guille JT, Kumar SJ, MacEwen GD. Fibrous dysplasia of the proximal part of the femur. *J Bone Joint Surg Am* 1998;80:648–658.
148. Harris WH, Dudley HR Jr, Barry RJ. The natural history of fibrous dysplasia: an orthopaedic, pathological, and roentgenographic study. *J Bone Joint Surg Am* 1962;44A:207–233.
149. Higashi T, Iguchi M, Shimura A, Kruglik GD. Computed tomography in bone scintigraphy in polyostotic fibrous dysplasia: a report of a case. *Oral Surg* 1980;50:580–583.
150. Kupcha PC, Guille JT, Tassanawipas A, Bowen JR. Polyostotic fibrous dysplasia and acromegaly. *J Pediatr Orthop* 1991;11:95–99.
151. Langer RA, Yook I, Capan LM. Anesthetic considerations in McCune-Albright syndrome: case report with literature review. *Anesth Analg* 1995;80:1236–1239.
152. Lichtenstein L. Polyostotic fibrous dysplasia. *Arch Surg* 1938;36:874–898.
153. Lichtenstein L, Jaffe HL. Fibrous dysplasia of bone: a condition affecting one, several or many bones, the graver cases of which may present abnormal pigmentation of skin, premature sexual development, hyperthyroidism or still other extra skeletal abnormalities. *Arch Pathol* 1942;33:777–815.
154. McCune DJ, Bruch H. Osteodystrophia fibrosa, report of a case in which condition was combined with precocious puberty, pathologic pigmentation of the skin and hyperthyroidism, with review of the literature. *Am J Dis Child* 1937;54:806–848.
155. Nixon GW, Condon VR. Epiphyseal involvement in polyostotic fibrous dysplasia: a report of two cases. *Radiology* 1973;106:167–170.
156. Stephenson RB, London MD, Hankin FM, Kaufer H. Fibrous dysplasia: an analysis of options for treatment. *J Bone Joint Surg Am* 1987;69:400–409.
157. Tsuchiya H, Tomita K, Matsumoto T, Watanabe S. Shepherd's crook deformity with an intracapsular femoral neck fracture in fibrous dysplasia. *Clin Orthop* 1995;310:160–164.
158. Warrick CK. Polyostotic fibrous dysplasia—Albright's syndrome. A review of the literature and report of four male cases, two of which were associated with precocious puberty. *J Bone Joint Surg Br* 1949;31:175–183.

#### Osteofibrous Dysplasia of the Tibia and Fibula

159. Akamatsu N, Hamada Y, Kohcio H, Siddiqi NA. Osteofibrous dysplasia of the tibia treated by bracing. *Int Orthop* 1992;16:180–194.
160. Campanacci M, Laus M. Osteofibrous dysplasia of the tibia and fibula. *J Bone Joint Surg Am* 1981;63:367–375.
161. Komiya S, Inoue A. Aggressive bone tumorous lesion in infancy: osteofibrous dysplasia of the tibia and fibula. *J Pediatr Orthop* 1993;13:577–581.
162. Ozaki T, Hamada M, Sugihara S, et al. Treatment outcome of osteofibrous dysplasia. *J Pediatr Orthop* 1988;7:199–202.
163. Park Y, Unni K, McLeod RA, Pritchard DJ. Osteofibrous Dysplasia: clinicopathologic study of 80 cases. *Human Pathol* 1993;1339–1347.
164. Springfield DS, Rosenberg AE, Mankin HJ, Mindell ER. Relationship between osteofibrous dysplasia and adamantinoma. *Clin Orthop* 1994;309:234–244.
165. Sweet DE, Vinh TN, Devaney K. Cortical osteofibrous dysplasia of long bone and its relationship to adamantinoma. *Am J Surg Pathol* 1992;16:282–290.

#### Neurofibromatosis

166. Ali MS, Hooper G. Congenital pseudarthrosis of the ulna due to neurofibromatosis. *J Bone Joint Surg Br* 1982;64:600–602.
167. Allieu Y, Gomis R, Yoshimura M, et al. Congenital pseudarthrosis of the forearm—two cases treated by free vascularized fibular graft. *J Hand Surg* 1981;6:475.
168. Anderson DJ, Schoenecker PL, Sheridan JJ, Rick MM. Use of an intramedullary rod for the treatment of congenital pseudarthrosis of the tibia. *J Bone Joint Surg Am* 1992;74:161–168.
169. Baker JK, Cain TE, Tullos HS. Intramedullary fixation for congenital pseudarthrosis of the tibia. *J Bone Joint Surg Am* 1992;74:169–178.
170. Bayne LG. Congenital pseudarthrosis of the forearm. *Hand Clin* 1985;1:457–465.
171. Bell DF. Congenital forearm pseudarthrosis: report of six cases and review of the literature. *J Pediatr Orthop* 1989;9:438–443.
172. de Boer HH, Verbout AJ, Nielsen HKL, van der Eijken JW. Free vascularized fibular graft for tibial pseudarthrosis in neurofibromatosis. *Acta Orthop Scand* 1988;59:425–429.
173. Boyd HB, Sage FP. Congenital pseudarthrosis of the tibia. *J Bone Joint Surg Am* 1958;40:1245–1270.
174. Brighton CT, Friedenberg ZB, Zemski LM, Pollis BR. Direct current stimulation non-Union and congenital pseudarthrosis: exploration of its clinical application. *J Bone Joint Surg Am* 1975;57:368–377.
175. Brown GA, Osebold WR, Ponseti IV. Congenital pseudarthrosis of long bones: a clinical, radiographic, histologic and ultrastructural study. *Clin Orthop* 1977;128:228–240.
176. Crawford AH. Neurofibromatosis in the pediatric patient. *Orthop Clin North Am* 1978;9:11–23.
177. Crawford AH, Bagamery N. Osseous manifestations of neurofibromatosis in childhood. *J Pediatr Orthop* 1986;6:72–88.
178. Crowe FW, Schull WJ. Diagnostic importance of café-au-lait spots in neurofibromatosis. *Arch Intern Med* 1953;91:758–766.
179. Dormans JP, Krajchich JI, Zuker R, Demuyck M. Congenital pseudarthrosis of the tibia: treatment with free vascularized fibular graft. *J Pediatr Orthop* 1990;10:623–628.
180. Dormans JP. Modified sequential McFarland bypass procedure for pre-pseudarthrosis of the tibia. *J Orthop Tech* 1995;3:176–180.
181. Fabry G, Lammens J, Van Melkebeek J, Stuyck J. Treatment of congenital pseudarthrosis with Ilizarov technique. *J Pediatr Orthop* 1988;8:67–70.
182. Flood BM, Butt WP, Dickson RA. Rib penetration of the intervertebral foraminae in neurofibromatosis. *Spine* 1986;11:172–174.
183. Greenberg LA, Schwartz A. Congenital pseudoarthrosis of the distal radius. *South Med J* 1975;68:1053–1054.
184. Gregg PJ, Price BA, Ellis HA, Stevens J. Pseudarthrosis of the radius associated with neurofibromatosis: a case report. *Clin Orthop* 1982;171:175–179.

185. Kaempffe FA, Gillespie R. Pseudarthrosis of the radius after fracture through normal bone in a child who had neurofibromatosis: a case report. *J Bone Joint Surg Am* 1989;71:1419-1421.
186. Kameyama O, Ogawa R. Pseudarthrosis of the radius associated with neurofibromatosis: report of a case and review of the literature. *J Pediatr Orthop* 1990;10:128-131.
187. Karol LA, Haideri NF, Halliday SE, et al. Gait analysis and muscle strength in children with congenital pseudarthrosis of the tibia: the effect of treatment. *J Pediatr Orthop* 1998;18:381-286.
188. Leung PC. Congenital pseudarthrosis of the tibia: three cases treated by free vascularized iliac crest graft. *Clin Orthop* 1983;175:45-50.
189. Lloyd-Roberts GC. Treatment of defects of the ulna in children by establishing cross-union with the radius. *J Bone Joint Surg Br* 1973;55B:320-327.
190. Major MR, Huizenga BA. Spinal compression by displaced ribs in neurofibromatosis. *J Bone Joint Surg Am* 1988;70:1100-1102.
191. Mandell GA, Harcke HT, Sharkey C, et al. SPECKT imaging of para-axial neurofibromatosis with technetium-99m DTPA. *J Nucl Med* 1987;28:1688-1694.
192. Mandell GA, Herrick WC, Harcke HT, et al. Neurofibromatosis: location by scanning with Tc-99m DTPA. *Radiology* 1985;157:803-806.
193. Manske PR. Forearm pseudarthrosis-neurofibromatosis. *Clin Orthop* 1979;139:125-127.
194. Masihuz-Zaman. Pseudarthrosis of the radius associated with neurofibromatosis: a case report. *J Bone Joint Surg Am* 59:977-978, 1977.
195. Mathoulin C, Gilbert A, Azze RG. Congenital pseudarthrosis of the forearm: Treatment of six cases With vascularized fibular graft and a review of the literature. *Microsurgery* 1993;14:252-259.
196. McCarroll HR. Clinical manifestations of congenital neurofibromatosis. *J Bone Joint Surg Am* 1950;32A:601-617.
197. McFarland B. Pseudarthrosis of the tibia in childhood. *J Bone Joint Surg Br* 1951;33:36-46.
198. Morrissy RT. Congenital pseudarthrosis of the tibia: factors that affect results. *Clin Orthop* 166:21-27, 1982.
199. Morrissy RT, Riseborough EJ, and Hall JE. Congenital pseudarthrosis of the tibia. *J Bone Joint Surg Br* 63:367-375, 1981.
200. Murray HH, Lovell WW. Congenital pseudarthrosis of the tibia: a long-term follow-up study. *Clin Orthop* 1982;166:14-20.
201. Ostrowski DM, Eilert RE, Waldstein G. Congenital pseudarthrosis of the ulna: a report of two cases and a review of the literature. *J Pediatr Orthop* 1986;5:463-467.
202. Paterson DC, Simonis RB. Electrical stimulation in the treatment of congenital pseudarthrosis of the tibia. *J Bone Joint Surg Br* 1985;67:454-462.
203. Pho RWH, Levack B, Satku H, Patradul H. Free vascularized fibular graft in the treatment of congenital pseudarthrosis of the tibia. *J Bone Joint Surg Br* 1985;67:64-70.
204. Rathgeb JM, Ramsey PL, Cowell HR. Congenital kyphoscoliosis of the tibia. *Clin Orthop* 1974;103:178-190.
205. Richin PF, Kranik A, Van Herpe L, Suffecol SL. Congenital pseudarthrosis of both bones of the forearm. *J Bone Joint Surg Am* 1976;58:1032-1033.
206. Rockower S, McKay D, Nason S. Dislocation of the spine in neurofibromatosis: a report of two cases. *J Bone Joint Surg Am* 1982;64:1240-1242.
207. Sellers DS, Sowa DT, Moore JR, Weiland AJ. Congenital pseudarthrosis of the forearm. *J Hand Surg* 1988;13A:89-93.
208. Shertzer JH, Bickel WH, Stubbins SG. Congenital pseudarthrosis of the ulna: a report of two cases. *Minn Med* 1969;52:1061-1066.
209. Sofield HA, Millar EA. Fragmentation, realignment, and intermedullary rod fixation of deformities of the long bones in children: a ten-year appraisal. *J Bone Joint Surg Am* 1969;41:1371-1391.
210. Sprague BL, Brown GA. Congenital pseudarthrosis of the radius. *J Bone Joint Surg Am* 1974;56:191-194.
211. Strong ML, Wong-Chung J. Prophylactic bypass grafting of the prepseudarthrotic tibia in neurofibromatosis. *J Pediatr Orthop* 1991;11:757.
212. Viridis R, Balestrazzi P, Zampoli M, et al. Hypertension in children with neurofibromatosis. *J Hum Hypertens* 1994;89:395-397.
213. Weiland AJ, Daniel RK. Congenital pseudarthrosis of the tibia: treatment with vascularized autogenous fibular grafts: a preliminary report. *Johns Hopkins Med J* 1980;147:89-95.
214. Whitehouse D. Diagnostic value of the café-au-lait spot in children. *Arch Dis Child* 1966;41:316-319.
215. Winter RB, Moe JH, Bradford DS, et al. Spine deformity in neurofibromatosis: a review of one hundred and two patients. *J Bone Joint Surg Am* 1979;61:677-694.
216. Wright J, Dormans JP, Rang M. Pseudarthrosis of the rabbit tibia: a model for congenital pseudarthrosis? *J Pediatr Orthop* 1991;11:277-283.
217. Yaghmai I, Tafazoli M. Massive subperiosteal hemorrhage in neurofibromatosis. *Radiology* 1977;122:439-441.
218. Yoshimura M, Shimamura K, Iwai Y, et al. Free vascularized fibular transplant: a new method for monitoring circulation of the grafted fibula. *J Bone Joint Surg Am* 1983;65:1295-1301.

#### Congenital Insensitivity to Pain

219. Guidera KJ, Multhopp H, Ganey T, Ogdne JA. Orthopaedics manifestations in congenitally insensate patients. *J Pediatr Orthop* 1990;10:514-521.
220. Guille JT, Forlin E, Bowen JR. Charcot disease of the shoulders in a patient who had familial sensory neuropathy with anhidrosis. *J Bone Joint Surg Am* 1992;74:1415-1417.
221. Kuo RS, Macnicol MF. Congenital insensitivity to pain: orthopaedic implications. *J Pediatr Orthop* 1996;5:292-295.
222. Okumo T, Inoue A, Izumo S. Congenital insensitivity to pain with anhidrosis. *J Bone Joint Surg Am* 1990;72:279-282.

#### Gaucher's Disease

223. Aker M, Zimran A, Abrahamov A, et al. Abnormal neutrophil chemotaxis in gaucher disease. *Br J Haematol* 1993;83:187-191.
224. Bell RS, Mankin HJ, Doppelt SH. Osteomyelitis in Gaucher disease. *J Bone Joint Surg Am* 1986;68:1380-1388.
225. Bilchik TA, Heyman S. Skeletal scintigraphy of pseudo-osteomyelitis in Gaucher's disease: two case reports and review of the literature. *Clin Nucl Med* 1992;17:279-282.
226. Cremin BJ, Davey H, Goldblatt J. The magnetic resonance features. *Clin Radiol* 1990;41:244-247.
227. Figueroa ML, Rosenbloom BE, Kay AC, et al. A less costly regimen of alglucerase to treat gaucher's disease. *N Engl J Med* 1992;327:1632-1636.
228. Golblatt J, Sacks S, Beighton P. The orthopaedic aspects of Gaucher disease. *Clin Orthop* 1978;137:208-214
229. Goldman AB, Jacobs B. Femoral neck fractures complicating Gaucher disease in children. *Skeletal Radio*. 1984;12:162-168.
230. Hermann G, Pastores GM, Abdelwahab IF, Lorberboym AM. Gaucher disease: Assessment of skeletal involvement and therapeutic responses to enzyme replacement. *Skeletal Radio*. 1997;26:687-696.
231. Hill SC, Parker CC, Brady RO, Barton NW. MRI of multiple platyspondyly in Gaucher disease: response enzyme replacement therapy. *J Comput Assist Tomogr* 1993;17:806-809.
232. Katz K, Cohen IJ, Ziv N, et al. Fractures in children who have Gaucher disease. *J Bone Joint Surg* 1987;69:1361-1370.
233. Katz K, Horev G, Rivlin E, et al. Upper limb involvement in patients with Gaucher's disease. *J Hand Surg* 1993;18A:871-875.
234. Katz K, Sabato S, Horev G, et al. Spinal involvement in children and adolescents with Gaucher disease. *Spine* 1993;18:332-335.
235. Lachiewicz PF. Gaucher's disease. *Orthop Clin North Am* 1984;15:765-774.
236. Miller JH, Ortega JA, Heisel MA. Juvenile Gaucher disease simulating osteomyelitis. *Am J Roentgenol* 1981;137:880-882.
237. Rosenthal DI, Scott JA, Barranger J, et al. Evaluation of Gaucher disease using magnetic resonance imaging. *J Bone Joint Surg Am* 1986;68:802-808.
238. Schein AJ, Arkin AM. Hip-joint involvement in Gaucher's disease. *Clin Orthop* 1973;90:4-10.
239. Schubiner H, Letourneau M, Murray DL. Pyogenic osteomyelitis versus pseudo-osteomyelitis in Gaucher's disease: a report of a case and review of the literature. *Clin Pediatr* 1981;20:667-669.
240. Tobias JD, Atwood R, Lowe S, Holcomb GW III. Anesthetic considerations in the child with Gaucher disease. *J Clin Anesth* 1993;5:150-153.
241. Tsai P, Lipton JM, Sahdev I, et al. Allogenic bone marrow transplantation in severe Gaucher disease. *Pediatr Res* 1992;31:503-507.
242. Zevin S, Abrahamov A, Hadas-Halpern I, et al. Adult-type Gaucher disease in children: genetics, clinical features, and enzyme replacement therapy. *Q J Med* 1993;86:565-573.
243. Zimran A, Elstein D, Kannai R, et al. Low-dose enzyme replacement therapy for Gaucher's disease: effects of age, sex, genotype, and clinical features on response to treatment. *Am J Med* 1994;97:3-13.
244. Zimran A, Elstein D, Levy-Lahad E, et al. Replacement therapy with miglucerase for type 1 Gaucher's disease. *Lancet* 1995;345:1479-1480.
245. Zimran A, Hollak CEM, Abrahamov A, et al. Home treatment with intravenous enzyme replacement therapy for Gaucher disease: an international collaborative study of 33 patients. *Blood* 1993;82:1107-1109.

#### Sickle Cell Disease

246. Al-Salef AH, Ahmed HA, Quaisaruddin S, et al. Osteomyelitis and septic arthritis in sickle cell disease in the eastern province of Saudi Arabia. *Int Orthop* 1992;16:398-402.
247. Bennett OM, Namnyak SS. Bone and joint manifestations of sickle cell anemia. *J Bone Joint Surg Br* 1990;72:494-499.
248. Bohrer SP. Acute long bone diaphyseal infarcts in sickle cell disease. *Br J Radiol* 1970;43:685-697.
249. Bohrer SP. Fracture complicating bone infarcts and/or osteomyelitis in sickle-cell disease. *Clin Radiol* 1971;22:83-88.
250. Bohrer SP. Growth disturbances of the distal femur following sickle-cell bone infarcts and/or osteomyelitis. *Clin Orthop* 1974;25:221-235.
251. Bonnerot V, Sebag G, deMontalembert M, et al. Gadolinium-DOTA enhanced MRI of painful osseous crises in children with sickle cell anemia. *Pediatr Radio*. 1994;24:92-95.
252. Chung SMJ, Alavi A, Russell MO. Management of osteonecrosis sickle-cell anemia and its genetic variance. *Clin Orthop* 1978;130:158-174.
253. Diggs LW. Bone and joint lesions in sickle cell disease. *Clin Orthop* 1967;52:119-143.
254. Ebong WW. Pathological fracture complicating long bone osteomyelitis in patients with sickle cell disease. *J Pediatr Orthop* 1986;6:177-181.
255. Epps CH, Bryant DD, Coles MJM, Castro O. Osteomyelitis in the patients who have sickle cell disease: diagnosis and management. *J Bone Joint Surg Am* 1991;73:1281-1294.
256. Golding JSR, MacIver JE, West LN. The bone changes in sickle-cell anaemia and its genetic variance. *J Bone Joint Surg Br* 1959;41:711-718.
257. Keeley K, Buchanan GR. Acute infarction of long bones in children with sickle-cell anemia. *J Pediatr* 1982;101:170-175.
258. Koren A, Garty I, Katzuni E. Bone infarction in children with sickle cell disease: early diagnosis and differentiation from osteomyelitis. *Eur J Pediatr* 1984;142:93-97.
259. Middlemiss JH, Raper AB. Skeletal changes in haemoglobinopathies. *J Bone Joint Surg Br* 1966;48:693-702.
260. Onuba O. Bone disorders in sickle cell disease. *Int Orthop* 1993;17:397-399.
261. Pavlock Dalton G, Drummond DS, Davidson RS, Robertson WW. Bone infarction versus infection in sickle cell disease in children. *J Pediatr Orthop* 1996;16:540-544.
262. Piehl FC, Davis RJ, Prugh SI. Osteomyelitis in sickle cell disease. *J Pediatr Orthop* 1993;13:225-227.
263. Sennara H, Gorry F. Orthopedic aspects anemia and allied hemoglobinopathies. *Clin Orthop* 1978;130:154-157.
264. Smith JA. Bone disorders in sickle cell disease. *Hematol Oncol Clin N Am* 1996;10:1345-1356.
265. Specht EE. Hemoglobinopathic *Salmonella* osteomyelitis: orthopedic aspects. *Clin Orthop* 1971;79:110-118.
266. Stark JE, Glasier CN, Blasler RD, et al. Osteomyelitis in children with sickle cell disease: early diagnosis with contrast-enhanced CT. *Radiology* 1991;179:731-733.

267. Stein RE, Urbaniak J. Use of tourniquet during surgery in patients with sickle cell hemoglobinopathies. *Clin Orthop* 1980;151:231–233.
268. Vichinsky EP, Haberkern CM, Neumayr L, et al. A comparison of conservative and aggressive transfusion regimens in the perioperative management of sickle cell disease. *N Engl J Med* 1995;333:206–213.

### Leukemia

269. Aur RJA, Westbrook HW, Riggs W Jr. Childhood acute lymphocytic leukemia. Initial radiological Bone involvement and prognosis. *Am J Dis Child* 1972;124:653.
270. Beredjikian PK, Drummond DS, Dormans JP, et al. Orthopaedic manifestations of graft-versus-host disease. *J Pediatr Orthop* 1998;18:572–575.
271. Bizot P, Witvoet J, Sedel L. Avascular necrosis of the femoral head after allogenic bone-marrow transplantation. *J Bone Joint Surg Br* 1996;78:878–883.
272. Bleyer WA. Acute lymphoblastic leukemia in children: advances and prospectus. *Cancer* 1990;65(Suppl 3):689–695.
273. Bos GD, Simon MA, Spiegel PG, Moohr JW. Childhood leukemia presenting as a diaphyseal radiolucency. *Orthop Clin North Am* 1978;135:66–68.
274. Claussen N, Gotze H, Pedersen A, et al. Skeletal scintigraphy and radiography at onset of acute lymphocytic leukemia in children. *Med Pediatr Oncol* 1983;11:291–296.
275. Gallagher DJ, Phillips DJ and Heinrich SD. Orthopaedic manifestations of acute pediatric leukemia. *Orthop Clin North Am* 1996;27:635–644.
276. Hann IM, Gupta S, Palmer MA, et al. The prognostic significance of radiological and symptomatic bone involvement in childhood acute lymphocytic leukemia. *Med Pediatr Oncol* 1979;6:51–55.
277. Heinrich SD, Gallagher D, Warrior R, et al. The prognostic significance of the skeletal manifestations of acute lymphoblastic leukemia of childhood. *J Pediatr Orthop* 1994;14:105–111.
278. Hughes RG, Kay HEM. Major Bone lesions in acute lymphoblastic leukaemia. *Med Pediatr Oncol* 1982;10:67–70.
279. Masera G, Carnelli V, Ferrari M, et al. Prognostic significance of radiological bone involvement in childhood acute lymphoblastic leukaemia. *Arch Dis Child* 1977;52:530–533.
280. Meehan PL, Viroslav S, Schmitt EW. Vertebral collapse in childhood leukemia. *J Pediatr Orthop* 1995;15:592–595.
281. Newman AJ, Melhorn DK. Vertebral compression in childhood leukemia. *Am J Dis Child* 1973;125:863–865.
282. Rogalsky RJ, Black GB, Reed MH. Orthopaedic manifestations of leukemia in children. *J Bone Joint Surg Am* 1986;68:494–501.
283. Samuda GM, Cheng MY, Yeung CY. Back pain and vertebral compression: an uncommon presentation of childhood acute lymphoblastic leukemia. *J Pediatr Orthop* 1987;7:175–178.
284. Silverstein MN, KeDy PJ. Leukemia with osteoarticular symptoms and signs. *Ann Intern Med* 1963;59:637–645.
285. Thomas LB, Forkner CE, Frei E III, et al. The skeletal lesions of acute leukemia. *Cancer* 1961;14:608–621.
286. Van Slyck EJ. The bony changes in malignant hematologic disease. *Orthop Clin North Am* 1972;3:733–744.
287. Wei S, Esmail AN, Bunin N, Dormans JP. Avascular necrosis in children with acute lymphoblastic leukemia. *J Pediatr Orthop* 2000;20:331–335.

### Hemophilia

288. Abildgaard CF, Penner JA, Watson-Williams EJ. Anti-inhibitor coagulant complex (autoplex) for treatment of factor VIII inhibitors in hemophilia. *Blood* 1980;56:978–984.
289. Ackroyd CE, Dinley RJ. The locked patella: an unusual complication of hemophilia. *J Bone Joint Surg Br* 1976;58:511–512.
290. Ahlberg AKM. On the natural history of hemophilic pseudotumor. *J Bone Joint Surg Am* 1975;57:1133–1135.
291. Ahlberg A, Nilsson IM. Fractures in haemophiliacs with special reference to complications and treatment. *Acta Chir Scand* 1967;133:293–302.
292. Arnold WD, Hilgartner MW. Hemophilic arthropathy: current concepts of pathogenesis and management. *J Bone Joint Surg Am* 1977;59:287–305.
293. Aronstam A, Browne RS, Wassef M, Hamad Z. The clinical features of early bleeding into the muscles of the lower limb in severe haemophiliacs. *J Bone Joint Surg Br* 1983;65:19–23.
294. Biggs R. Haemophilia treatment in the united kingdom: 1969 to 1974. *Br J Haematol* 1977;35:487–500.
295. Boardman KP, English P. Fractures and dislocations in hemophilia. *Clin Orthop* 1980;148:221–232.
296. Brant EE, Jordan HH. Radiologic aspects of hemophilic pseudotumors in bone. *Am J Roentgenol* 1972;115:525–539.
297. Castanda VL, Parmley RT, Bozzini M, Feldmeier JJ. Radiotherapy of pseudotumors of bone in hemophiliacs with circulating inhibitors of factor VIII. *Am J Hematol* 1991;36:55–59.
298. Connelly S, Kaleko M. Gene therapy for hemophilia A. *Thromb Haemost* 1997;78:31–36.
299. Dietrich AM, James CD, King DR, et al. Head trauma in children with congenital coagulation disorders. *J Pediatr Surg* 1994;29:28–32.
300. Erken EHW. Radiocolloids in the management of hemophilic arthropathy in children and adolescents. *Clin Orthop* 1991;264:129–135.
301. Feil E, Bentley G, Rizza CR. Fracture management in patients with haemophilia. *J Bone Joint Surg Br* 1974;56:643–649.
302. Floman Y, Niska M. Dislocation of the hip joint complicating repeated hemarthrosis in hemophilia. *J Pediatr Orthop* 1983;3:99–100.
303. Greene WB. Use of continuous passive slow motion in the postoperative rehabilitation of difficult pediatric knee and elbow problems. *J Pediatr Orthop* 1983;3:419–423.
304. Greene WB, McMillan CW, Warren MW. Prophylactic transfusions for hypertrophic synovitis in children with hemophilia. *Clin Orthop* 1997;343:19–24.
305. Greene WB. Synovectomy of the ankle for hemophilic arthropathy. *J Bone Joint Surg Am* 1994;76:812–819.
306. Greene WB, Yankaskas BC, Guilford WB. Roentgenographic classifications of hemophilic arthropathy. *J Bone Joint Surg Am* 1989;71:237–244.
307. Gregosiewicz A, Wosko I, Kandzierski G. Intraarticular bleeding in children with hemophilia: the prevention of arthropathy. *J Pediatr Orthop* 1989;9:182–185.
308. Houghton GR, Duthie RB. Orthopaedic problems in haemophilia. *Clin Orthop* 1979;138:197–216.
309. Hutcheson J. Peripelvic new bone formation in hemophilia. *Radiology* 1973;109:529–530.
310. Ingram GIC, Mathews JA, Bennett AE. A controlled trial of joint aspiration in acute haemophilic haemarthrosis. *Br J Haematol* 1972;23:649–654.
311. Idi-Peretti I, LeBalc'h T, Yvart J, Bittoun J. MR imaging of hemophilic arthropathy of the knee: classification and evolution of the subchondral cysts. *MRI* 1992;10:67–75.
312. Kasper CK, Rapaport SI. Bleeding times and platelet aggregation after analgesics in hemophilia. *Ann Intern Med* 1972;77:189–193.
313. Kemp HS, Matthews JM. The management of fractures in haemophilia and Christmas disease. *J Bone Joint Surg Br* 1968;50:351–358.
314. Kisker CT, Burke C. Double-blind studies on the use of steroids in the treatment of acute hemarthrosis in patients with hemophilia. *N Engl J Med* 1970;282:639–642.
315. Krill Jr CE, Mauer AM. Pseudotumor of calcaneus in Christmas disease. *J Pediatr* 1970;77:848–855.
316. Kumari S, Fulco JD, Karayaldn G, Lipton R. Gray scale ultrasound: evaluation of iliopsoas hematomas in hemophiliacs. *AJR Am J Roentgenol* 1979;133:103–106.
317. Lancourt JE, Gilbert MS, Poster MA. Management of bleeding and associated complications of hemophilia in the hand and forearm. *J Bone Joint Surg Am* 1977;59:451–460.
318. Lethagen S, Regnarson Tennvall G. Self-treatment with desmopressin intranasal spray in patients with bleeding disorders: effect on bleeding symptoms and socioeconomic factors. *Ann Hematol* 1993;66:257–260.
319. Lieberg OU, Penner JA, Bailey RW. Fibrosarcoma presenting as a pseudotumor of hemophilia: a report of an unusual case. *J Bone Joint Surg Am* 1975;57:422–424.
320. Lurie A, Bailey BP. The management of acute haemophilic haemarthroses and muscle haematomata. *S Afr Med J* 1972;46:656–659.
321. Lusher JM, Wartier I. Hemophilia A. *Hematol Oncol Clin North Am* 1992;6:1021–1033.
322. Madigan RR, Hanna WT, Wallace SL. Acute compartment syndrome in hemophilia: a case report. *J Bone Joint Surg Am* 1981;63:1327–1329.
323. Magallon M, Monteagudo J, Altisent C, et al. Hemophilic pseudotumor: multicenter experience over a 25-year period. *Am J Hematol* 1994;45:103–108.
324. Merchan ECR, Magallon M, Manso F, Martin-Villar J. Septic arthritis in HIV positive haemophiliacs. *Int Orthop* 1992;16:302–306.
325. Miller EH, Flessa HC, Glueck HI. The management of deep soft tissue bleeding in hemarthrosis in hemophilia. *Clin Orthop* 1972;82:92–107.
326. Moneim MS, Gribble TJ. Carpal tunnel syndrome in hemophilia. *J Hand Surg* 1984;9A:580–583.
327. Nuss R, Kilcoyne RF, Geraghty S, et al. Utility of magnetic resonance imaging for management of hemophilic arthropathy in children. *J Pediatr* 1993;123:388–392.
328. Patel MR, Pearlman HS, Lavine LS. Arthrodesis in hemophilia. *Clin Orthop* 1972;86:168–174.
329. Pettersson H, Ahlberg A. Computed tomography in hemophilic pseudotumor. *Acta Radiol* 1982;23:453–457.
330. Pietrogrande V, Dioguardi N, Mannucci PM. Short term evaluation of synovectomy in haemophilia. *Br Med J* 1972;2:378–381.
331. Post M, Telfer MC. Surgery in hemophilic patients. *J Bone Joint Surg Am* 1975;57:1136–1145.
332. Ragni MV, Winkelstein A, Kingsley L, et al. 1986 update of HIV seroprevalence, seroconversion, AIDS incidence, and immunologic correlates of HIV infection in patients with hemophilia A and B. *Blood* 1987;70:786–790.
333. Robins RHC, Murrell JS. Traumatic ischaemia in a haemophilic: a report of a case of prolonged haemostasis with cryoprecipitate during decompression and skin grafting. *J Bone Joint Surg Br* 1971;53:113–117.
334. Rodriguez-Merchan EC. Pathogenesis, early diagnosis, and prophylaxis for chronic hemophilic synovitis. *Clin Orthop* 1997;343:6–11.
335. Rosenthal RL, Graham JJ, Selirio E. Excision of pseudotumor with repair by bone graft of pathologic fracture of femur in hemophilia. *J Bone Joint Surg Am* 1973;55:827–832.
336. Shirkhoda A, Mauro MA, Staab EV, Blatt PM. Soft tissue hemorrhage in hemophilic patients: computed tomography and ultrasound study. *Radiology* 1983;147:811–814.
337. Shopnick RI, Brettler DB. Hemostasis; a practical review of conservative and operative care. *Clin Orthop* 1996;328:34–38.
338. Stein H, Dickson RA. Reversed dynamic slings for knee-flexion contractures in the hemophilic. *J Bone Joint Surg Am* 1975;57:282–283.
339. Valderrama JAF, Matthews JM. The haemophilic pseudotumor or haemophilic subperiosteal haematoma. *J Bone Joint Surg Br* 1965;47:256–265.
340. White GC II, McMillan CW, Blatt PM, Roberts HR. Factor VIII inhibitors: a clinical overview. *Am J Hematol* 1982;13:335–342.
341. Wilson DJ, Green DJ, MacLamon JC. Arthrosonography of the painful hip. *Clin Radiol* 1984;35:17–19.

### Osteomyelitis

342. Ash JM, Gilday DL. The futility of bone scanning in neonatal osteomyelitis: concise communication. *J Nucl Med* 1980;21:417–420.
343. Azouz EM, Greenspan A, Marton D. CT evaluation of primary epiphyseal bone abscesses. *Skeletal Radio* 1993;22:17–23.
344. Berquist TH, Brown ML, Fitzgerald RH Jr, May GR. Magnetic resonance imaging: application in musculo-skeletal infection. *Magn Resonance Imaging* 1985;3:219–230.
345. Cabanela ME, Sim FH, Beabout JW, Dahlin DC. Osteomyelitis appearing as neoplasms: a diagnostic problem. *Arch Surg* 1974;109:68–72.
346. Canale ST, Puhl J, Watson FM, Gillespie R. Osteomyelitis following closed fractures: a report of three cases. *J Bone Joint Surg Am* 1975;57:415–418.
347. Capener M, Pierce KC. Pathological fractures in osteomyelitis. *J Bone Joint Surg Am* 1932;14:501–510.

348. Daoud A, Saighi-Bouaouina A. Treatment of sequestra, pseudarthroses and defects in the long bones of children who have chronic hematogenous osteomyelitis. *J Bone Joint Surg Am* 1989;71:1448–1467.
349. Daoud A, Saighi-Bouaouina A, Descamps L, Maestro M. Hematogenous osteomyelitis of the femoral neck in children. *J Pediatr Orthop [B]* 1993;2:83–95.
350. Dormans JP, Drummond DS. Pediatric hematogenous osteomyelitis: new trends in presentation, diagnosis, and treatment. *J Am Acad Orthop Surg* 2:333-341, 1994.
351. Ferguson AB Jr. Osteomyelitis in children. *Clin Orthop* 1973;96:51–56.
352. Howie DW, Savage JP, Wilson TG, Paterson D. The technetium phosphate bone scan in the diagnosis of osteomyelitis in children. *J Bone Joint Surg Am* 1983;65:431–437.
353. Jackson MA, Nelson JD. Etiology and management of acute suppurative bone and joint infections in pediatric patients. *J Pediatr Orthop* 1982;2:313–323.
354. Jurik AG, Helmig O, Ternowitz T, Moller RN: Chronic multifocal osteomyelitis: a follow-up study. *J Pediatr Orthop* 1988;8:49–58.
355. Kavanaugh JH. Occult infected fracture of the femur: a report of two cases with long-term follow up. *J Trauma* 1978;18:813–815.
356. Langenskiold A. Femur remodeled during growth after osteomyelitis causing coxa vara and shaft necrosis. *J Pediatr Orthop* 1982;2:289–294.
357. Lewallen RP, Peterson HA. Nonunion of fractures in children: a review of 30 cases. *J Pediatr Orthop* 1985;5:135–142.
358. Lewin JS, Rosenfield NS, Hoffer PB, Downing D. Acute osteomyelitis in children: combined Tc-99m and Ga-67 imaging. *Radiology* 1986;158:795–804.
359. Morrey BF, Peterson HA. Hematogenous pyogenic osteomyelitis in children. *Orthop Clin North Am* 1975;6:935–951.
360. Nelson JD, Bucholz RW, Kusmiesz H, Shelton S. Benefits and risks of sequential parenteral-oral cephalosporin therapy for suppurative bone and joint infections. *J Pediatr Orthop* 1982;2:255–262.
361. Sacks R, Habermann ET. Pathological fracture in congenital rubella: a case report. *J Bone Joint Surg Am* 1977;59:557–559.
362. Schmidt D, Mubarak S, Gelberman R. Septic shoulders in children. *J Pediatr Orthop* 1981;1:67–72.
363. Seftion GK. Osteomyelitis after closed femoral fracture in a child. *J R Coll Surg Edinb* 1982;27:113.
364. Septimus EJ, Musher DM. Osteomyelitis: recent clinical and laboratory aspects. *Orthop Clin North Am* 1979;10:347–359.
365. Silverman FN. Virus diseases of bone: do they exist? *Am J Roentgenol* 1976;126:677–703.
366. Sitarz AL, Berdon WE, Wolff JA, Baker DH. Acute lymphocytic leukemia masquerading as acute osteoporosis: a report of two cases. *Pediatr Radiol* 1980;9:33–35.
367. Smith RK, Specht EE. Osseous lesions and pathologic fractures in congenital cytomegalic inclusion disease: a report of a case. *Clin Orthop* 1979;144:280–283.
368. Sundberg SB, Savage JP, Foster BK. Technetium phosphate bone scan and the diagnosis of septic arthritis in childhood. *J Pediatr* 1989;9:579–585.
369. Tudisco C, Farsetti P, Gatti S, Ippolito E. Influence of chronic osteomyelitis on skeletal growth: analysis at maturity of 26 cases affected during childhood. *J Pediatr Orthop* 1991;11:358–363.
370. Unkila-Kallio L, Kallio MJT, Eskola J, Peltola H. Serum C-reactive protein, erythrocyte sedimentation rate, and white blood cell count in acute hematogenous osteomyelitis of children. *Pediatrics* 1994;93:59–95.
371. White M, Dennison WM. Acute haematogenous osteitis in childhood: a review of two hundred and twelve cases. *J Bone Joint Surg Br* 1952;34:608–623.

#### Pathologic Fractures After Limb Lengthening

372. Blane C, Herzenberg J, DiPietro M. Radiographic imaging for limb lengthening in children. *J Pediatr Radiol* 1991;21:117–120.
373. Eldridge J, Bell D. Problems with substantial limb lengthening. *Orthop Clin North Am* 1991;22:625–631.
374. Epps C, Bowen RJ. *Complications in pediatric orthopaedic surgery*. Philadelphia: JB Lippincott, 1995:747.
375. Hood R, Riseborough E. Lengthening of the lower extremity by the Wagner method. *J Bone Joint Surg Am* 1981;63:1122–1131.
376. Maffulli N, Hughes T. Ultrasonographic monitoring of limb lengthening. *J Bone Joint Surg Br* 1992;74B:130–132.
377. Malhis T, Bowen J. Tibial and femoral lengthening. A report of 54 cases. *J Pediatr Orthop* 1982;2:487–491.
378. Mosca V, Moseley C. Complications of Wagner leg lengthening and their avoidance. *Orthop Trans* 1986;10:462.
379. Osterman K, Merikanto J. Diaphyseal bone lengthening in children using Wagner device: long-term results. *J Pediatr Orthop* 1991;11:449–451.
380. Price C, Cole J. Limb lengthening by callotasis for children and adolescents. *Clin Orthop* 1990;250:105–111.

#### Osteogenesis Imperfecta

381. Alman B, Frasca P. Fracture failure mechanisms in patients with osteogenesis imperfecta. *J Orthop Res* 1987;5:139–143.
382. Bailey RW. Further clinical experience with extensible nail. *Clin Orthop* 1981;159:171–176.
383. Bailey RW, Dubow HI. Studies of longitudinal bone growth resulting in an extensible nail. *Surg Forum* 1963;14:455–458.
384. Bailey RW, Dubow HI. Evolution of the concept of an extensible nail accommodating to normal longitudinal bone growth: clinical considerations and implications. *Clin Orthop* 1981;159:157–170.
385. Bembi B, Parma A, Bottega M, et al. Intravenous pamidronate treatment in osteogenesis imperfecta. *J Pediatr* 1997;131:622–625.
386. Benson DR, Newman DC. The spine and surgical treatment in osteogenesis imperfecta. *Clin Orthop* 1981;159:147–153.
387. Bleck EE. Nonoperative treatment of osteogenesis imperfecta: orthotic and mobility management. *Clin Orthop* 1981;159:111–122.
388. Byers PH, Bonadio JF, Steinmann B. Osteogenesis imperfecta: update in perspective. *Am J Med Genet* 1984;17:429–435.
389. Cohn DH, Byers PH. Clinical screening for collagen defects in connective tissue diseases. *Clin Perinato* 1990;17:793–809.
390. Cole WG. The molecular pathology of osteogenesis imperfecta. *Clin Orthop* 1997;343:235–248.
391. DiCesare PE, Sew-Hoy A, Krom W. Bilateral isolated olecranon fractures in an infant as presentation of osteogenesis imperfecta. *Orthopedics* 1992;15:741–743.
392. Furey JG, McNamee DC. Airsplints for long-term management of osteogenesis imperfecta. *J Bone Joint Surg Am* 1973;55:645–649.
393. Gamble JG, Rinsky LA, Strudwick WJ, Bleck EE. Non-union of fractures in children who have osteogenesis imperfecta. *J Bone Joint Surg Am* 1988;70:439–443.
394. Gamble JG, Strudwick WJ, Rinsky LA, Bleck EE. Complications of intramedullary rods in osteogenesis imperfecta: Bailey-Dubow rods versus non-elongating rods. *J Pediatr Orthop* 1988;8:645–649.
395. Gerber LH, Binder H, Weintrob J, et al. Rehabilitation of children and infants with osteogenesis imperfecta. *Clin Orthop* 1995;251:254–262.
396. Glorieux FH, Bishop NJ, Plotkin H, et al. Cyclic administration of pamidronate in children with severe osteogenesis imperfecta. *N Engl J Med* 1998;339:947–952.
397. Hanscomb DA, Winter RB, Lutter L, et al. Osteogenesis imperfecta: radiographic classification, natural history, and treatment of spinal deformities. *J Bone Joint Surg Am* 1992;74:598–616.
398. Harkey HL, Crockard HA, Stevens JM, et al. The operative management of basilar impression in osteogenesis imperfecta. *Neurosurgery* 1990;27:782–786.
399. Harrison WJ, Rankin KC. Osteogenesis imperfecta in Zimbabwe: comparison between treatment with intramedullary rods of fixed-length and self-expanding rods. *J R Coll Surg Edinb* 1998;43:328–332.
400. Jerosch J, Mazzotti I, Tomasevic M. Complications after treatment with osteogenesis imperfecta with a Bailey-Dubow rod. *Arch Orthop Trauma Surg* 1998;117:240–245.
401. Jewell FC, Lofstrom JE. Osteogenic sarcoma occurring in fragilitas ossium: a case report. *Radiology* 1940;34:741–743.
402. King JD, Bobechko WP. Osteogenesis imperfecta: an orthopaedic discussion and surgical review. *J Bone Joint Surg Br* 1971;53:72–89.
403. Klenerman L, Ockenden BG, Townsend AC. Osteosarcoma occurring in osteogenesis imperfecta: a report of two cases. *J Bone Joint Surg Br* 1967;49:314–323.
404. Knight DJ, Bennet GC. Nonaccidental injury in osteogenesis imperfecta: a case report. *J Pediatr Orthop* 1990;10:542–544.
405. Kocher MS, Shapiro F. Osteogenesis imperfecta. *J Am Acad Orthop Surg* 1998;6:225–236.
406. Letts M, Monson R, Weber K. The prevention of recurrent fractures of the lower extremities in severe osteogenesis imperfecta using vacuum pants: a preliminary report in four patients. *J Pediatr Orthop* 1988;8:454–457.
407. Luhmann SJ, Sheridan JJ, Capelli RN, Schoenecker PL. Management of lower-extremity deformities in osteogenesis imperfecta with extensible intramedullary rod technique: a 20-Year experience. *J Pediatr Orthop* 1998;18:88–94.
408. McKusick VA. Heritable disorders of connective tissue, 3rd ed. St. Louis: CV Mosby, 1972.
409. Minch CM, Kruse RW. Osteogenesis imperfecta: a review of basic science and diagnosis. *Orthopedics* 1998;21:558–567.
410. Moorefield WG, Miller GR. Aftermath of osteogenesis imperfecta disease in adulthood. *J Bone Joint Surg Am* 1980;62:113–119.
411. Nicholas RW, James P. Telescoping intramedullary stabilization of the lower extremities for severe osteogenesis imperfecta. *J Pediatr Orthop* 1990;10:219–223.
412. Niemann MW. Surgical treatment of the tibia in osteogenesis imperfecta. *Clin Orthop* 1981;159:134–140.
413. Nishi Y, Hamamoto K, Kajiyama M, et al. Effect of long-term calcitonin therapy by injection and nasal spray on the incidence of fractures in osteogenesis imperfecta. *J Pediatr* 1992;121:477–480.
414. Patterson CR, Burns J, McAllion SJ. Osteogenesis imperfecta: the distinction from child abuse and recognition of a variant form. *Am J Med Genet* 1993;45:187–192.
415. Porat S, Heller E, Seidman DS, Meyer S. Functional results of operation in osteogenesis imperfecta: elongating and nonelongating rods. *J Pediatr Orthop* 1991;11:200–203.
416. Robert JB. Bilateral hyperplastic callus formation in osteogenesis imperfecta. *J Bone Joint Surg Am* 1976;58:1164–1166.
417. Shoenfeld Y, Fried A, Ehrenfeld NE. Osteogenesis imperfecta: a review of the literature with presentation of 29 cases. *Am J Dis Child* 1975;129:679–687.
418. Sijbrandij S. Percutaneous nailing in the management of osteogenesis imperfecta. *Int Orthop* 1990;14:195–197.
419. Sillence D. Osteogenesis imperfecta: an expanding panorama of variants. *Clin Orthop* 1981;159:11–25.
420. Sofield HA, Millar EA. Fragmentation, realignment, and intramedullary rod fixation of deformities of the long bones in children. *J Bone Joint Surg Am* 1959;41:1371–1391.
421. Steiner RD, Pepin M, Byers PH. Studies of collagen synthesis and structure in the differentiation of abuse from osteogenesis imperfecta. *J Pediatr* 1996;128:542–547.
422. Stott NS, Zions LE. Displaced fractures of the apophysis of the olecranon in children who have osteogenesis imperfecta. *J Bone Joint Surg Am* 1993;75:1026–1033.
423. Wynne-Davies R, Hall CM, Apley AG, eds. *Atlas of skeletal dysplasias*. New York: Churchill Livingstone, Inc, 1985.
424. Zions LE, Ebramzadeh E, Stott NS. Complications in the use of the Bailey-Debow extensible nail. *Clin Orthop* 1998;348:186–195.

#### Osteopetrosis

425. Armsrtong DG, Newfield JT, Gillespie R. Orthopaedic management of osteopetrosis: results of a survey and review of the literature. *J Pediatr Orthop* 1999;19:122–132.
426. Beighton P, Hamersma H, Cremin BJ. Osteopetrosis in south africa: the benign, lethal, and intermediate forms. *S Afr Med J* 1979;55:659–665.
427. Bollerslev J, Andersen PE Jr. Fracture patterns in two types of autosomal dominant osteopetrosis. *Acta Orthop Scand* 1989;60:110–112.
428. Cameron HU, Dewar FP. Degenerative osteoarthritis associated with osteopetrosis. *Clin Orthop* 1977;127:148–149.
429. Coccia PF, Krivit W, Cervenka J, et al. Successful bone marrow transplantation for infantile malignant osteopetrosis. *N Engl J Med* 1980;302:701–708.
430. Gerritsen EJ, Vossen JM, van Loo IH, et al. Autosomal recessive osteopetrosis: variability of findings at diagnosis and during the natural course. *Pediatrics* 1994;93:247–253.
431. Greene WB, Torre BA. Femoral neck fracture in a child with autosomal dominant osteopetrosis. *J Pediatr Orthop* 1985;5:483–485.
432. Hasenhuttl K. Osteopetrosis: review of the literature and comparative studies on a case with twenty-four-year Follow-up. *J Bone Joint Surg Am* 1962;44:359–369.
433. Horton WA, Schimke RN, Iyama T. Osteopetrosis: a further heterogeneity. *J Pediatr* 1980;97:580–585.
434. Hoyt CS, Billson FA. Visual loss in osteopetrosis. *J Dis Chilc* 1979;133:955–958.
435. Jaffe HL. *Metabolic, degenerative, and inflammatory diseases of bone and joints*. Philadelphia: Lea & Febiger, 1972.
436. Kaplan FS, August CS, Fallon MD, et al. Osteopetrorickets: the paradox of plenty, pathophysiology and treatment. *Clin Orthop* 1993;294:64–78.
437. Loria-Cortes R, Quesada-Calvo E, Cordero-Chaverri C. Osteopetrosis in children: a report of 26 cases. *J Pediatr* 1977;91:43–47.
438. Manusov EG, Douville DR, Page LV, Trivedi DV. Osteopetrosis (marble bone disease). *Am Fam Physician* 1993;47:175–180.
439. Marks SC, Schmidt CJ. Bone remodelling as an expression of altered phenotype: studies of fracture healing in untreated and cured osteopetrotic rats. *Clin Orthop* 1978;137:259–264.
440. Martin RP, Deane RH, Collett V. Spondylolysis in children who have osteopetrosis. *J Bone Joint Surg Am* 1997;79:1685–1689.
441. Milgram JW, Jasty M. Osteopetrosis: a morphological study of twenty-one cases. *J Bone Joint Surg Am* 1982;64:912–929.
442. Popoff SN, Marks SC Jr. The heterogeneity of osteoporosis reflects the diversity of of cellular influences during skeletal development. *Bone* 1995;17:437–445.
443. Rawlingson PSM, Green RHA, Cognis AM, et al. Malignant osteoporosis: hypercalcaemia after bone marrow transplantation. *Arch Dis Child* 1995;66:638–639.
444. Reeves JE, Huffer WE, August CS, et al. The hematopoietic effects of prednisone therapy in four infants with osteopetrosis. *J Pediatr* 1979;94:210–214.
445. Shapiro F. Osteopetrosis: current clinical considerations. *Clin Orthop* 1993;294:34–44.
446. Shapiro F, Glimsher MJ, Holtrop ME, et al. Human osteopetrosis: a histological, ultrastructural, and biochemical study. *J Bone Joint Surg Am* 1980;62:384–399.
447. van Lie Peters EM, Aronson DC, Everts V, Dooren LJ. Failure of calcitriol treatment in a patient with malignant osteopetrosis. *Eur J Pediatr* 1993;152:818–821.
448. Whyte MP. Carbonic anhydrase II deficiency. *Clin Orthop* 1993;294:52–63.

#### **Pycnodysostosis**

449. Benz G, Schmid-Ruter E. Pycnodysostosis with heterozygous beta-thalassemia. *Pediatr Radiol* 1977;5:164–171.
450. Busenberry JF, Kane JJ. Pycnodysostosis: report of three new cases. *Am J Roentgenol* 1967;99:717–723.
451. Edelson JG, Obad S, Geiger R, et al. Pycnodysostosis. Orthopaedic aspects with a description of 14 new cases. *Clin Orthop* 1992;2804:263–276.
452. Maroteaux P, Lamay M. The malady of Toulouse-Lautrec. *JAMA* 1965;191:715–717.
453. Meredith SC, Simon MA, Laros GS, Jackson MA. Pycnodysostosis: a clinical, pathological, and ultramicroscopic study of a case. *J Bone Joint Surg Am* 1978;60:1122–1127.
454. Roth VG. Pycnodysostosis presenting with bilateral subtrochanteric fractures: a case report. *Clin Orthop* 1976;117:247–253.
455. Tachdjian MO. *Pediatric orthopaedics*, 2nd ed. Philadelphia WB Saunders, 1989.
456. Taylor MM, Moore TM, Harvey JP Jr. Pycnodysostosis: a case report. *J Bone Joint Surg* 1978;60A:1128–1130.

#### **Rickets**

457. Bachrach S, Fisher J, Parks JS. An outbreak of vitamin D deficiency rickets in a susceptible population. *Pediatrics* 1979;64:871–877.
458. Goldman AB, Lane JM, Salvati E. Slipped capital femoral epiphyses complicating renal osteodystrophy: a report of three cases. *Radiology* 1978;126:333–339.
459. Holda ME, Ryan JR. Hepatobiliary rickets. *J Pediatr Orthop* 1982;2:285–287.
460. Iddin DV, Levitsky LL, Schieg W, et al. Resurgence of nutritional rickets associated with breast feeding and special dietary practices. *Pediatrics* 1980;65:232–235.
461. Kobayashi A, Kawai S, Utsunomiya T, Ohbe Y. Bone disease in infants and children with hepatobiliary disease. *Arch Dis Child* 1974;49:641–646.
462. Kooh SW, Jones G, Reilly BJ, Fraser D. Pathogenesis of rickets and chronic hepatobiliary disease in children. *J Pediatr* 1979;94:870–874.
463. Lebrun JB, Moffatt MEK, Mundy RJT, et al. Vitamin D deficiency in a Manitoba community. *Can J Public Health* 1993;84:394–396.
464. Mankin HJ. Rickets, osteomalacia, and renal osteodystrophy: part I. *J Bone Joint Surg Am* 1974;46:101–128.
465. Mehls O, Ritz E, Krempien B, et al. Slipped epiphysis and renal osteodystrophy. *Arch Dis Child* 1975;50:545–554.
466. Parfitt AM. Renal osteodystrophy. *Orthop Clin North Am* 1972;33:681–698.
467. Sherk HH, Cruz M. Vitamin D prophylaxis and a lowered incidence of fractures in anticonvulsant rickets and osteomalacia. *Clin Orthop* 1977;129:251–257.
468. Smith R. The pathophysiology and management of rickets. *Orthop Clin North Am* 1972;3:601–621.
469. Sweeney LE. Hypophosphataemic rickets after ifosfamide treatment in children. *Clin Radio* 1993;47:345–347.
470. Timberlake RW, Cook SD, Thomas KA, et al. Effects of anticonvulsant drug therapy on bone mineral density in a pediatric population. *J Pediatr Orthop* 1988;8:467–470.
471. Touloukian RJ, Gertner JM. Vitamin D deficiency rickets as a late complication of short gut syndrome during infancy. *J Pediatr Surg* 1981;16:230–235.

#### **Rickets and Very-Low-Birth-Weight Infants**

472. Amier J, Tatz K, Yosipovich Z, Wielunski E, Reisner SH. Fractures in premature infants. *J Pediatr Orthop* 1988;8:41–44.
473. Bosley ARJ, Verrier-Jones ER, Campbell MJ. Aetiological factors in rickets of prematurity. *Arch Dis Child* 1980;55:683–686.
474. Helfer RE, Scheurer SL, Alexander R, et al. Trauma to the bones of small infants from passive exercise: a factor in the etiology of child abuse. *J Pediatr* 1984;104:47–50.
475. Kelly HJ, Sloan RE, Hoffman W, Sander C. Accumulation of nitrogen and six minerals in the human fetus during gestation. *Hum Bio* 1950;2:61–74.
476. Koo WWK, Gupta JM, Nayanar VV, et al. Skeletal changes in preterm infants. *Arch Dis Child* 1982;57:447–452.
477. Koo WWK, Sherman R, Succop P, et al. Fractures and rickets in very low birth weight infants: conservative management and outcome. *J Pediatr Orthop* 1989;9:326–330.
478. Koo WWK, Oestreich AE, Sherman R, et al. Osteopenia, rickets, and fractures in preterm infants. *Am J Dis Child* 1985;139:1045–1046.
479. Poznanski AK, Kuhns LR, Guire KE. New standards of cortical mass in the humerus of neonates: a means of evaluating bone loss in the premature infant. *Radiology* 1980;134:634–639.
480. Roberts WA, Badger VM. Osteomalacia of very-low-birth-weight infants. *J Pediatr Orthop* 1984;4:593–598.

#### **Rickets and Renal Osteodystrophy**

481. Andreoli SP, Bergstein JM, Sherrard DJ. Aluminum intoxication from aluminum-containing phosphate binders in children with azotemia not undergoing dialysis. *N Engl J Med* 1984;310:1079–1084.
482. Apel DM, Millar EA, Moel DI. Skeletal disorders in a pediatric renal transplant population. *J Pediatr Orthop* 1989;9:505–511.
483. Cattell HS, Levin S, Kopits S, Lyne ED. Reconstructive surgery in children with azotemic osteodystrophy. *J Bone Joint Surg Am* 1971;53:216–228.
484. Chalmers J. Subtrochanteric fractures in osteomalacia. *J Bone Joint Surg Br* 1970;52:509–513.
485. Davids JR, Fisher R, Lum G, Gliński SV. Angular deformity of the lower extremity in children with F osteodystrophy. *J Pediatr Orthop* 1992;12:291–299.
486. Davidson JK, Tsakiris D, Briggs JD, Junor BJR. Osteonecrosis and fractures following renal transplantation. *Clin Radio* 1985;36:27–35.
487. Eyres KS, Brown J, Douglas DL. Osteotomy and intramedullary nailing for the correction of progressive deformity in vitamin D-resistant hypophosphataemic rickets. *J R Coll Surg Edinb* 1993;38:50–54.
488. Ferris B, Walker C, Jackson A, Kirwan E. The orthopaedic management of hypophosphatemic rickets. *J Pediatr Orthop* 1991;11:367–373.
489. Hartjen CA, Koman LA. Treatment of slipped capital femoral epiphysis resulting from juvenile renal osteodystrophy. *J Pediatr Orthop* 1990;10:551–554.
490. Msu AC, Kooh SW, Fraser D, et al. Renal osteodystrophy in children with chronic renal failure: an unexpectedly common and incapacitating complication. *Pediatrics* 1982;70:742–750.
491. Kirkwood JR, Ozonoff MB, Steinbach HL. Epiphyseal displacement after metaphyseal fracture in renal osteodystrophy. *Am J Roentgenol* 1972;115:547–554.
492. Krempien B, Mehls O, Ritz E. Morphological studies on pathogenesis of epiphyseal slipping in uremic children. *Virchows Arch* 1974;362:129–143.
493. Llach F, Nikakhtar B. Current advances in the therapy of secondary hyperparathyroidism in osteitis fibrosa. *Miner Electrolyte Metab* 1991;17:250–255.
494. McWhorter AG, Seale NS. Prevalence of dental abscess in a population of children with vitamin D-resistant rickets. *Pediatr Dent* 1991;13:91–96.
495. Milliner DS, Nebeker HG, Ott SM, et al. Use of deferoxamine infusion test in the diagnosis of aluminum-related osteodystrophy. *Ann Intern Med* 1984;101:775–780.
496. Nelson CL, Evans CM, Popowniak K. Musculoskeletal complications of renal transplantation. *Surg Clin North Am* 1971;51:1205–1209.
497. Nixon JR, Douglas JF. Bilateral slipping of the upper femoral epiphysis in end-stage renal failure: a report of two cases. *J Bone Joint Surg Br* 1980;62:18–21.
498. Oppenheim WL, Namba R, Goodman WG, Salusky IB. Aluminum toxicity complicating renal osteodystrophy: a case report. *J Bone Joint Surg Am* 1989;71:446–452.
499. Ruderman RJ, Poehling GG, Gray R, et al. Orthopaedic complications of renal transplantation in children. *Transplant Proc* 1979;11:104–106.
500. Salusky IB, Coburn JW, Foley J, et al. Effects of oral calcium carbonate on control of serum phosphorus and changes in plasma aluminum levels after discontinuation of aluminum-containing gels in children receiving dialysis. *J Pediatr* 1986;108:767–770.
501. Stern PJ, Watts HG. Osteonecrosis after renal transplantation in children. *J Bone Joint Surg Am* 1979;61:851–856.

502. Teall CG. A radiological study of the bone changes in renal infantilism. *Br J Radiol* 1928;1:49–58.

#### Idiopathic Osteoporosis

503. Berglund G, Landquist B. Osteopenia in adolescents. *Clin Orthop* 1960;17:259–264.
504. Bertelloni S, Baroncelli GI, DeNero G, Saggese G. Idiopathic juvenile osteoporosis: evidence of normal osteoblast function by 1,25-dihydroxyvitamin D<sup>3</sup> stimulation test. *Calcif Tissue Int* 1992;51:20–30.
505. Cloutier MD, Hayles AB, Riggs BL, et al. Juvenile osteoporosis: report of a case including description of some metabolic and microradiographic studies. *Pediatrics* 1967;40:649–655.
506. Dent CE. Osteoporosis in childhood. *Postgrad Med J* 1977;53:450–456.
507. Dent CE, Friedman M. Idiopathic juvenile osteoporosis. *Q J Med* 1965;134:177–210.
508. Gooding CA, Ball JH. Idiopathic juvenile osteoporosis. *Radiology* 1969;93:1349–1350.
509. Houang MTW, Brenton DP, Renton P, Shaw DG. Idiopathic juvenile osteoporosis. *Skeletal Radio* 1978;3:17–23.
510. Jowsey J, Johnson KA. Juvenile osteoporosis: bone findings in seven patients. *J Pediatr* 1972;81:511–517.
511. Lane JM, Vigorita BJ. Osteoporosis: current concepts review. *J Bone Joint Surg Am* 1983;65:274–278.
512. Marder HK, Tsang RG, Hug G, Crawford AC. Calcitriol deficiency in idiopathic juvenile osteoporosis. *J Dis Child* 1982;136:914–917.
513. Marhaug G. Idiopathic juvenile osteoporosis. *Rheumatol Int* 1992;22:45–47.
514. Singh M, Nagrath AR, Maini PS. Changes in trabecular pattern of the upper end of the femur as an index of osteoporosis. *J Bone Joint Surg Am* 1970;52:457–467.
515. Smith R. Idiopathic osteoporosis in the young. *J Bone Joint Surg Br* 1980;62:417–427.

#### Iatrogenic Osteoporosis

516. Elsasser U, Ruegsegger P, Anliker M, et al. Loss and recovery of trabecular bone in the distal radius following fracture-immobilization of the upper limb in children. *Clin Wochenschr* 1979;57:763–767.
517. Lane M, Nesbit M Jr, Hall TC, et al. Chemotherapy: discussion. *Cancer* 1976;37(Suppl):1055–1057.
518. Nesbit M, Krivit W, Heyn R, Sharp H. Acute and chronic effects of methotrexate on hepatic, pulmonary, and skeletal systems. *Cancer* 1976;37(Suppl):1037–1054.
519. Nilsson BE, Westlin NE. Restoration of bone mass after fracture of the lower limb in children. *Acta Orthop Scand* 1971;42:78–81.
520. Ragab AH, Frech RS, Vietti TJ. Osteoporotic fractures secondary to methotrexate therapy of acute leukemia in remission. *Cancer* 1970;25:580–585.
521. Schwartz AM, Leonidas JC. Methotrexate osteopathy. *Skeletal Radio* 1984;11:13–16.
522. Stanisavljevic S, Babcock AL. Fractures in children treated with methotrexate for leukemia. *Clin Orthop* 1977;125:139–144.

#### Hyperparathyroidism

523. Adam A, Ritchie D. Hyperparathyroidism with increased bone density in the areas of growth. *J Bone Joint Surg Br* 1954;36:157–260.
524. Anspach WE, Clifton WN. Hyperparathyroidism in children: a report of two cases. *Am J Dis Child* 1939;58:540–557.
525. Bjernulf A, Hall K, Sjögren I, Werner I. Primary hyperparathyroidism in children: brief review of the literature and a case report. *Acta Paediatr Scand* 1970;59:249–258.
526. Bleck EE, Kleinman RG. Special injuries of the musculoskeletal system. In: Rockwood CA Jr, Wilkins KE, King RE (eds). *Fractures in children*. Philadelphia: JB Lippincott, 1984:173–228.
527. Clark OH, Duh QY. Primary hyperparathyroidism: a surgical perspective. *Endocrinol Metab Clin North Am* 1989;18:701–714.
528. Dauphine RT, Riggs BL, Scholz DA. Back pain and vertebral crush fractures: an unemphasized mode of presentation for primary hyperparathyroidism. *Ann Intern Med* 1975;83:365–367.
529. Eaton DGM, Hewitt CAH. Renal function in hyperparathyroidism with complicating nephrocalcinosis. *Acta Paediatr* 1993;82:111–112.
530. Grantmyre EB. Roentgenographic features of "primary" hyperparathyroidism in infancy. *J Can Assoc Radio* 1973;24:257–260.
531. Jackson CE, Frame B. Diagnosis and management of parathyroid disorders. *Orthop Clin North Am* 1972;3:699–712.
532. Jaffe HL. *Metabolic, degenerative, and inflammatory diseases of bone and joints*. Philadelphia: Lea & Febiger, 1972.
533. Lancourt JE, Hochberg F. Delayed fracture healing in primary hyperparathyroidism. *Clin Orthop* 1977;114:214–218.
534. Parfitt AM. The actions of parathyroid hormone on bone relation to bone remodeling and turnover, calcium homeostasis, and metabolic bone disease. *Metabolism* 1976;25:1033–1069.
535. Rajasuriya K, Peiris OA, Ratnaika VT, DeFonseca CP. Parathyroid adenomas in childhood: a case report and a review of the current literature. *Am J Dis Child* 1964;107:442–449.
536. Randal C, Lauchlan SC. Parathyroid hyperplasia in a infant. *Am J Dis Child* 1963;105:364–367.
537. Wilkerson H, James J. Self-limiting neonatal primary hyperparathyroidism associated with familial hypocalciuric hypercalcaemia. *Arch Dis Child* 1993;69:319–321.

#### Cushing's Syndrome

538. Ansell BM. Overview of the side effects of corticosteroid therapy. *Clin Exp Rheumatol* 1991;9(Suppl 6):19–20.
539. Benson PF, Vulliamy DG, Taubman JO. Congenital hemihypertrophy and malignancy. *Lancet* 1963;1:468–469.
540. Chudler RM, Kay R. Adrenocortical carcinoma in children. *Urol Clin North Am* 1989;16:469–479.
541. Cushing H. The basophil adenomas of the pituitary body and their clinical manifestations. *Bull Johns Hopkins Hosp* 1932;50:137–195.
542. Eberlein WR, Bongiovanni AM, Rodriguez CS. Diagnosis and treatment: the complications of steroid treatment. *Pediatrics* 1967;40:279–282.
543. Gilbert MG, Cleveland WW. Cushing's syndrome in infancy. *Pediatrics* 1970;46:217–229.
544. Harris WH, Heaney RP. Skeletal renewal and metabolic bone disease. *N Engl J Med* 1969;280:303–311.
545. Iannaccone A, Gabrilove JL, Brahm SA, Sofer LJ. Osteoporosis in Cushing's syndrome. *Ann Intern Med* 1960;52:570–586.
546. Jowsey J, Riggs BL. Bone formation in hypercortisolism. *Acta Endocrinol (Copenh)* 1970;63:21–28.
547. McArthur RG, Bahn RC, Hayles AB. Primary adrenocortical nodular dysplasia as a cause of Cushing's syndrome in infants and children. *Mayo Clin Proc* 1982;57:58–63.
548. McArthur RG, Colutier MD, Hayles AB, Sprague RG. Cushing's disease in children: findings in thirteen cases. *Mayo Clin Proc* 1972;47:318–326.
549. Preeyasombat C, Sirikulchayanonta V, Mahachokekeltwattana P, et al. Cushing's syndrome caused by Ewing's sarcoma secreting corticotropin releasing factor-like peptide. *Am J Dis Child* 1992;146:1103–1105.
550. Pritis K, Everard ML, Milner AD. Unexpected side-effects of inhaled steroids: a case report. *Eur J Pediatr* 1991;150:448–449.
551. Varonos S, Ansell BM, Reeve J. Vertebral collapse in juvenile chronic arthritis: its relationship with corticosteroid therapy. *Calcif Tissue Int* 1987;41:75–78.

#### Scurvy

552. Banks SW. Bone changes in acute and chronic scurvy: an experimental study. *J Bone Joint Surg Am* 1943;15:553–565.
553. Grewar D. Infantile scurvy. *Clin Pediatr* 1965;4:82–89.
554. Hoeffel JC, Lascombes P, Mainard L, Durup de Balcine D. Cone epiphysis of the knee and scurvy. *Eur J Pediatr Surg* 1993;3:186–180.
555. Lee RV. Scurvy: a contemporary historical perspective (third of three parts). *Conn Med* 1984;48:33–35.
556. MacLean AD. Spinal changes in a case of infantile scurvy. *Br J Radiol* 1968;41:385–387.
557. McLean S, McIntosh R. Healing in infantile scurvy as shown by x-ray. *Am J Dis Child* 1928;36:875–930.
558. McKibbin B, Porter RW. The incidence of vitamin C deficiency in meningomyelocoele. *Dev Med Child Neuro* 1967;9:338–344.
559. Nerubay J, Pilderwasser D. Spontaneous bilateral distal femoral physiolysis due to scurvy. *Acta Orthop Scand* 1984;55:18–20.
560. Ossosky HJ. Infantile scurvy. *Am J Dis Child* 1965;109:173–176.
561. Quiles M, Sanz TA. Epiphyseal separation in scurvy. *J Pediatr Orthop* 1988;8:223–225.
562. Ramar S, Sivaramakrishnan V, Manoharan K. Scurvy—a forgotten disease. *Arch Phys Med Rehabil* 1993;74:92–95.
563. Scott W. Epiphyseal dislocations in scurvy. *J Bone Joint Surg Am* 1941;23:314–322.
564. Silverman FM. Recovery from epiphyseal invagination sequel to an unusual complication of scurvy. *J Bone Joint Surg Am* 1970;52:384–390.
565. Silverman FM. An unusual osseous sequel to infantile scurvy. *J Bone Joint Surg Am* 1953;35:215–220.

#### Copper Deficiency and Scurvy-Like Syndrome

566. Botash AS, Nasca J, Dubowy R, et al. Zinc-induced copper deficiency in an infant. *Am J Dis Child* 1992;146:709–711.
567. Cordano A, Baertl JM, Graham GG. Copper deficiency in infancy. *Pediatrics* 1964;43:324–336.
568. Grunebaum M, Horodniceanu C, Steinherz R. The radiographic manifestations of bone changes in copper deficiency. *Pediatr Radio* 1980;9:101–104.
569. Heller RM, Kirchner SG, O'Neill JA, et al. Skeletal changes of copper deficiency in infants receiving prolonged total parenteral nutrition. *J Pediatr* 1978;92:947–949.

#### Cerebral Palsy

570. Fry K, Hoffer MM, Brink J. Femoral shaft fractures in brain-injured children. *J Trauma* 1976;16:371–373.

571. Heinrich SD, Drvaric DM, Darr K, MacEwen GD. The operative stabilization of pediatric diaphyseal femur fractures with flexible intramedullary nails: a prospective analysis. *J Pediatr Orthop* 1994;14:501-507.
572. Kaye JJ, Freiburger RH. Fragmentation of the lower pole of the patella in spastic lower extremities. *Radiology* 1971;101:97-100.
573. Lee JJK, Lyne, ED. Pathologic fractures in severely handicapped children and young adults. *J Pediatr Orthop* 1990;10:497-500.
574. Lloyd-Roberts GC, Jackson AM, Albert JS. Avulsion of the distal pole of the patella in cerebral palsy: a cause for deteriorating gait. *J Bone Joint Surg Br* 1985;67:252-254.
575. McIvor WC, Samilson RL. Fractures in patients with cerebral palsy. *J Bone Joint Surg Am* 1966;48:858-866.
576. Miller F, Dias RC, Dabney KW, et al. Soft-tissue release for spastic hip subluxation in cerebral palsy. *J Pediatr Orthop* 1997;17:571-584.
577. Miller F, Girardi H, Lipton G, et al. Reconstruction of the dysplastic spastic hip with peri-iliac pelvic and femoral osteotomy followed by immediate mobilization. *J Pediatr Orthop* 1997;17:592-602.
578. Miller PR, Glazer DA. Spontaneous fractures in the brain-crippled, bedridden patient. *Clin Orthop* 1976;120:134-137.
579. Rosenthal RK, Levine DB. Fragmentation of the distal pole of the patella in spastic cerebral palsy. *J Bone Joint Surg Am* 1977;59:934-939.
580. Sherk HH. Indications for orthopaedic surgery in mentally retarded patient. *Clin Orthop* 1973;90:174-177.

#### Myelomeningocele

581. Anderson TE, Drummond DS, Breed AL, Taylor CA. Malignant hyperthermia in myelomeningocele: a previously unreported association. *J Pediatr Orthop* 1981;1:401-403.
582. Anschuetz RH, Freehafer AA, Shaffer JW, Dixon MS Jr. Severe fracture complications in myelodysplasia. *J Pediatr Orthop* 1984;4:22-24.
583. Boytim MJ, Davidson RS, Charney E, Melchionni JB. Neonatal fractures in myelomeningocele patients. *J Pediatr Orthop* 1991;11:28-30.
584. Dormans JP, Templeton J, Schreiner MS, Delfico AJ. Intraoperative latex anaphylaxis in children: classification and prophylaxis of patient at risk. *J Pediatr Orthop* 1997;17:622-625.
585. Drabu KJ, Walker G. Stiffness after fractures around the knee in spina bifida. *J Bone Joint Surg Br* 1985;67:266-267.
586. Drennan JC, Freehafer AA. Fracture of the lower extremities in paraplegic children. *Clin Orthop* 1971;77:211-217.
587. Drummond DS, Moreau N, Cruss RL. The results and complications of surgery for the paralytic hip and spine in myelomeningocele. *J Bone Joint Surg Br* 1980;62:49-53.
588. Drummond DS, Moreau N, Cruss RL. Post-operative neuropathic fractures in patients with myelomeningocele. *Dev Med Child Neuro* 1981;23:147-150.
589. FDA Allergic Reactions to Latex Containing Medical Devices: *FDA Medical Alert*, 1991.
590. Edvardsen P. Physeo-epiphyseal injuries of the lower extremities in myelomeningocele. *Acta Orthop Scand* 1972;43:550-557.
591. Eichenholtz SN. Management of long-bone fractures in paraplegic patients. *J Bone Joint Surg Am* 1963;45:299-310.
592. Feiwell E, Sakai D, Blatt T. The effect of hip reduction on function in patients with myelomeningocele. *J Bone Joint Surg Am* 1978; 60:169-173.
593. Gyepes MT, Newbern DH, Neuhauser EBD. Metaphyseal and physeal injuries in children with spina bifida and myelomeningoceles. *Am J Roentgeno*. 1965;95:168-177.
594. James CCM. Fractures of the lower limbs in spina bifida cystica: a survey of 44 fractures in 122 children. *Dev Med Child Neuro*. 1970;22:88-93.
595. Kumar SJ, Cowell HR, Townsend P. Physeal, metaphyseal, and diaphyseal injuries of the lower extremities in children with myelomeningocele. *J Pediatr Orthop* 1984;4:25-27.
596. Laidlaw AT, Loder RT, Hensinger RN. Telescoping intramedullary rodding with Bailey-Dubow nails for recurrent pathologic fractures in children without osteogenesis imperfecta. *J Pediatr Orthop* 1998;18:4-8.
597. Lock TR, Aronson DD. Fractures in patients who have myelomeningocele. *J Bone Joint Surg Am* 1989;71:1153-1157.
598. Meeropol E, Frost J, Pugh L, et al. Latex allergy in children with myelodysplasia: a survey of Shriners Hospitals. *J Pediatr Orthop* 1993;13:1-4.
599. Norton PL, Foley JJ. Paraplegia in children. *J Bone Joint Surg Am* 1959;41:1291-1309.
600. Quillis AN. Fractures in children with myelomeningocele: a report of 15 cases and a review of the literature. *Acta Orthop Scand* 1974;45:883-897.
601. Rodgers WB, Schwend RM, Jaramillo D, et al. Chronic physeal fractures in myelodysplasia: magnetic resonance analysis, histologic description, treatment, and outcome. *J Pediatr Orthop* 1997;17:615-621.
602. Townsend PF, Cowell HR, Stegf NL. Lower extremity fractures simulating infection of myelomeningocele. *Clin Orthop* 1979;144:255-259.
603. Wenger DR, Jeffcoat BT, Herring JA. The guarded prognosis of physeal injury in paraplegic children. *J Bone Joint Surg Am* 1980;62:241-246.

#### Muscular Dystrophy

604. Epstein BS, Abramson JL. Roentgenological changes in the bones in cases of pseudohypertrophic muscular dystrophy. *Arch Neurol Psychiatry* 1941;46:868-876.
605. Hsu JD. Extremity fractures in children with neuromuscular disease. *Johns Hopkins Med J* 1979;145:89-93.
606. Hsu JD, Garda-Ariz M. Fracture of the femur in Duchenne's muscular dystrophy patient. *J Pediatr Orthop* 1981;1:203-207.
607. Maybarduk PK, Levine M. Osseous atrophy associated with progressive muscular dystrophy. *Am J Dis Child* 1941;61:565-576.
608. Siegel IM. Fractures of long bones in Duchenne's muscular dystrophy. *J Trauma* 1977;17:219-222.
609. Vignos PJ Jr, Archibald KC. Maintenance of ambulation in childhood muscular dystrophy. *J Chronic Dis* 1960;12:273-290.

#### Arthrogyposis and Poliomyelitis

610. Diamond LS, Alegado R. Perinatal fractures in arthrogyposis multiplex congenita. *J Pediatr Orthop* 1981;1:189-192.
611. Neuhauser EBD. Arthrogyposis multiplex congenita: susceptibility to injury. *Postgrad Med* 1969;46:61-63.
612. Robin GC. Fractures in poliomyelitis in children. *J Bone Joint Surg Am* 1966;48:1048-1054.
613. Sodergard J, Ryppy S. The knee in arthrogyposis multiplex congenita. *J Pediatr Orthop* 1990;10:177-182.
614. Williams P. The management of arthrogyposis. *Orthop Clin North Am* 1978;9:67-88.

#### Spinal Cord Injury

615. Abramson AS. Bone disturbances in injuries to the spinal cord and cauda equina (paraplegia): their prevention by ambulation. *J Bone Joint Surg Am* 1948;30:982-987.
616. Comarr AE, Hutchinson RH, Bors E. Extremity fractures of patients with spinal cord injuries. *Am J Surg* 1962;103:732-739.
617. Drennan JC, Freehafer AA. Fractures of the lower extremities in paraplegic children. *Clin Orthop* 1971;77:211-217.
618. Eichenholtz SN. Management of long-bone fractures in paraplegic patients. *J Bone Joint Surg Am* 1963;45:299-310.
619. Freehafer AA, Mast WA. Lower extremity fractures in patients with spinal-cord injury. *J Bone Joint Surg Am* 1965;47:683-694.
620. Hulth A, Olerud S. The healing in fractures in denervated limbs: an experimental study using sensory and motor rhizotomy and peripheral denervation. *J Trauma* 1965;5:571-579.
621. Katz JF. Spontaneous fractures in paraplegic children. *J Bone Joint Surg Am* 1953;35:220-226.
622. Robin GC. Fracture in childhood paraplegia. *Paraplegia* 3:165-170, 1965.
623. Schneider R, Goldman AB, Bohne WHO. Neuropathic injuries to the lower extremities in children. *Radiology* 1978;128:713-718.

# 7 CHILD ABUSE

[ROBERT M. CAMPBELL, JR.](#)

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## EPIDEMIOLOGY

Child abuse is maltreatment of the child by either parents or caretakers, and includes physical, sexual, and emotional abuse, as well as emotional and physical neglect. The epidemic of child abuse continues to worsen in the United States, with approximately 2.9 million reports now filed annually compared with 1.2 million in 1982 (151). The incidence of physical abuse nationally is estimated to be 4.9 children per thousand, and one of every 1,000 abused children die (76). In Texas alone, 50,746 children were confirmed victims of child abuse or neglect in fiscal year 1995, with 101 deaths recorded (152). The types of abuse these children were subjected to included physical abuse in 31.5%, sexual abuse in 15.6%, emotional abuse in 6.8%, abandonment in 2%, medical neglect in 4.7%, physical neglect in 15.4%, and neglectful supervision in 40.3%. The minimal annual cost of child abuse in the United States is estimated to be 9 billion dollars (151). The long-term social costs of child abuse are impossible to estimate: one third of the victims of child abuse grow up to be seriously inept, neglectful, or abusive parents; one third are at high risk for eventually becoming abusive parents; and only one third do not repeat the destructive patterns they were exposed to as children (120). The orthopaedist becomes involved in the care of 30% to 50% of abused children (2). Early recognition of these abused patients by the orthopaedist is critical, because the children who are returned to their homes after an unrecognized episode of child abuse have a 25% risk of serious reinjury and a 5% risk of death (132).

## HISTORICAL OVERVIEW

In 1946, Caffey (28) described six infants with long bone fractures, chronic subdural hematomas, and intraocular bleeding without a history of trauma to explain the injuries. Although his work is often cited as the first report in the English literature of child abuse, Caffey did not speculate about the etiology of the children's injuries. In 1953, Silverman (139) characterized the unique metaphyseal fractures found in abused children in the pediatric literature and clearly emphasized that these were due to nonaccidental trauma. Altman and Smith (8) in 1960 published the first series in the orthopaedic literature of injuries caused by child abuse. General public awareness of child abuse increased with the 1962 publication of a report by Kempe et al. (79) characterizing the problem as *the battered child syndrome*. In 1974, Caffey introduced the term *whiplash-shaken infant syndrome* to the literature to emphasize the etiology of subdural hematomas in infants caused by shaking episodes (29). In 1974, Congress acknowledged the national importance of the prevention of child abuse by the passage of the Child Abuse Prevention and Treatment Act.

## THE HOMES AT RISK

In assessing where abuse of children may occur, households in turmoil from marital separation, job loss, divorce, family death, housing difficulties, or money problems are more likely to have abusive episodes (49). Families with two unplanned births are 2.8 times more likely to have an episode of child abuse than families with no unplanned births (164). In these homes at risk, stepparents, babysitters, boyfriends, relatives, and even larger siblings frequently are abusers (1,69,121). The parents of battered children may themselves have been abused when they were children (63). Parental substance abuse, whether alcohol or other drugs, makes child abuse more likely (67). The risk of physical child abuse is fivefold more likely with maternal cocaine use (158). Young, unmarried mothers are more likely to have their infants die from intentional injury, with a peak incidence of 10.5 intentional deaths per 10,000 live births in one study (136). Violence in the home is not directed solely toward the child; in one study (30) of families with substantiated child abuse, 30% of the mothers had been battered. Although the youngest, poorest, most socially isolated and economically frustrated caretakers are the most likely to act violently toward their children (161), any adult from any social or economic level may be guilty of abusing a child (1).

## THE CHILDREN AT RISK

Certain children of all ages are more likely to experience abuse, and younger children are particularly at risk. Most reported cases of child abuse involve children younger than age 3 years (61). In one report (18) of child abuse 78% of all fractures reported were in children less than 3 years of age and 50% of all fractures occurred in children younger than 1 year of age. Infants younger than 1 year are especially at risk for the most severe form of child abuse: infant homicide (44,86). With severe shaking injuries by caretakers, 30% of infants are disabled and another 30% die (25). The problem probably is more widespread than suspected. There is evidence to suggest that in the United Kingdom, 10% to 20% of children diagnosed as having sudden infant death syndrome may be intentional infant homicides (56). In one report (131), covert video recordings of adults attending their children who were hospitalized for suspicious illness documented 14 separate instances of caretaker attempts at upper airway obstruction. First-born children (18), premature infants, stepchildren, and handicapped children are at a greater risk for child abuse (2). Benedict et al. (19), in a longitudinal study of 500 handicapped children followed from birth to age 10 years, documented a 4.6% incidence of physical abuse. Surprisingly, they found that the most severely involved children were less likely to be abused, whereas the marginally functioning children were at greater risk, with parental frustration possibly being a factor.

Children who are persistently presented by parents for medical assessment of vague illness and have a history of multiple diagnostic or therapeutic procedures with unclear outcome are at risk for having a form of child abuse known as Munchausen's syndrome by proxy (14,111). Munchausen's syndrome is named for Baron von Munchausen, an 18th century mercenary whose exaggerated tales of adventure were viewed with great suspicion. Asher, in 1951, described Munchausen's syndrome in adults who presented with apparent acute illness accompanied by dramatic, untruthful medical histories to gain attention (14). In Munchausen's syndrome by proxy, children become the victims of this adult behavior when parents with a misguided sense of purpose fabricate a wide range of childhood illnesses for their children, often subjecting them to needless diagnostic work-ups and treatment (111). Symptoms of the child's "illness" are based on an imaginary medical history given by the



had scalp or facial hematomas and 12% had lacerations about the head. There was only one nondisplaced skull fracture and one long bone fracture in a patient with osteogenesis imperfecta.

Additional important information about the child and the family may be obtained by a review of past medical records or by contacting social workers who may have been involved with the family. Conferring with the family's primary health care provider also may be extremely helpful. A medical release from the family is necessary before these sources of information can be explored (49). The physician or social worker should be asked if there have been prior concerns regarding abuse or neglect; whether there has been a suspicious pattern of injury, illness, ingestion of objects, or medications or noncompliance with health care recommendations; whether the family is receiving counseling or other support from any community groups; and whether the family has any previous involvement with child protective services or the police (49).

## Documentation

Careful documentation is critical. Chart notes may later be needed in court as evidence for either custodial hearings or criminal trial (101), and defending inaccurate or partial chart notes in court can be extremely embarrassing. Each account should be recorded in as much detail as possible, using quotation marks for exact quotes and specifying who is giving the history. Particularly with crucial answers, the exact question preceding the response should be documented. In addition, the general emotional state of the individual providing the account, as well as the individual's reaction to emotionally charged questions should be documented to assist in later evaluation of the credibility of the account. If the family wishes to change their story after the initial account, no changes should be made to the earlier record, but an addendum should be placed detailing the new account. The completed record should include several specific items (Table 7-2). If child protective services recommends emergency transfer of custody of the child to a foster home or a shelter, then the orthopaedist should summarize chart documentation in a separate notarized narrative, which is preferred by most courts.

- Mechanism of injury
- Family social history
- Past medical history
- Family history of diseases such as osteogenesis imperfecta or other metabolic disease
- Physical examination findings (positive and negative)
- X-ray findings
- Laboratory results
- Photographs of soft tissue injuries
- Results of consultations
- Diagnosis of child abuse
- Treatment recommendations
- Acute injuries
- Investigation by child protective services

TABLE 7-2. DOCUMENTATION OF CHILD ABUSE

## PHYSICAL EXAMINATION

After the initial musculoskeletal evaluation for acute fracture assessment, an extremely thorough physical examination of the patient should follow, systematically going from the head to the toes, to detect any signs of additional acute or chronic injury. Additional acute fractures may cause local tenderness and swelling, whereas chronic fractures may produce swelling from the presence of callus and clinical deformity from malunion. Specific radiographs should detect clinical fractures, with skeletal surveys showing most occult fractures. Other body areas commonly involved in child abuse include the central nervous system, the skin, the abdomen, and the genitalia. These areas should be carefully evaluated for signs of acute and especially chronic injury, because 50% of verified abuse cases show evidence of prior abuse (63).

### Neurologic Injuries

Head injuries are the leading cause of death in child abuse (21). When an infant with musculoskeletal injury presents with altered mental status, whiplash-shaken infant syndrome should be suspected. Violent shaking of a small child whiplashes the relatively large head back and forth over the thorax with possible development of subdural hematomas (Fig. 7-1). Some authors (50) believe these head injuries actually occur when the child's head is slammed onto a soft surface such as a mattress. On impact, deceleration forces approaching 400 Gs may be seen, tearing the bridging vessels between the skull and the brain, and producing intracranial hemorrhage and cerebral edema. Skull fractures, though, are rare unless the child is thrown onto a hard object. Simple linear skull fractures usually are due to a fall of less than 4 feet and may be accidental, but complex skull fractures without a history of significant trauma, including comminuted, diastatic (separated sutures), displaced fractures, and fractures crossing suture lines, are suspicious for abuse (21). Children with head injuries caused by direct impact trauma are described as having shaken impact syndrome (50). Children with head injuries have fevers, bulging fontanelles, and sometimes enlargement of the head. A pattern of paresis may be present, and reflexes may be increased (29). Skull fractures generally are uncommon, and either ultrasound or computed tomography (CT) scans are used to diagnose subdural hematomas. Cerebral edema may be lethal (37), and emergency neurosurgical consultation is needed. Funduscopic examination by an ophthalmologist is indicated because concurrent retinal hemorrhage is common (29). The examiner should be aware, however, that retinal hemorrhages due to normal vaginal birth are present in 40% of newborns, but these injuries resolve by 3 to 4 weeks of age (53). Shaken baby syndrome has a high mortality rate: Thirty percent of infants die of their injuries (25).



FIGURE 7-1. Violent shaking of a small child held by the chest whiplashes the head back and forth over the thorax, with the possible development of subdural hematomas as well as rib fractures. Infants held by the extremities are also at risk for long bone fracture.

Fatal cerebral injury also occurs in the abusive tin ear syndrome: a clinical triad of unilateral ear bruising, CT scan evidence of ipsilateral cerebral edema, and hemorrhagic retinopathy, with a 100% mortality rate in one series (66).

Spinal fractures occur in child abuse when a child is forcibly slammed onto a countertop with hyperflexion of the spine (1). Although neurologic deficit with this type of injury is uncommon (41), children should be carefully evaluated for signs of spinal cord injury secondary to spinal fracture when abuse is suspected.

### Soft Tissue Injuries

In addition to examination of the soft tissue around the acute fracture site for swelling and bruising, the patient's entire body should be carefully and systematically evaluated to detect acute and chronic soft tissue trauma. Deliberate soft tissue injuries are present in 81% to 92% of abused patients (61,109). The types of skin

lesions commonly encountered in child abuse include ecchymosis, welts, abrasions, lacerations, scars, and burns.

The typical toddler often has multiple bruises over bony prominences such as the chin, brow, knees, and the shins ( 1,132). Bruises on the back of the head, neck ( 1), buttocks, abdomen, legs, arms, cheeks, or genitalia may be suspicious for abuse, although accidental bruises can occur in all these locations ( 132). Accidental bruising of the head or face, though, is much less common. In a study of 400 nonabused children, Robertson ( 128) found an overall incidence of just 7% for accidental soft tissue injuries of the face and head, with the peak incidence of 17% seen in toddlers. Soft tissue injuries were present on the lower extremities and buttocks in only 31% of children and on the upper extremities of only 9%. The configuration of the bruise due to abuse may resemble the implement used to inflict the injury ( Fig. 7-2), but usually the soft tissue injuries of abuse are “weapon” specific in fewer than 10% of patients ( 109). Although bruises often are concentrated on the trunk and buttocks, they also can be present on the head and proximal extremities. The weapons used to abuse children can be almost any common object, but they often include belt buckles, staplers, ropes, switches, coat hangers, ironing cords, and the open or closed human hand ( 77,149). Bruises inflicted by an open hand may appear on the face or a flat area of skin from a slap and grasp marks may appear as ovoid lesions when the fingertips are deeply embedded in the extremities or the shoulders of the child during extreme shaking ( 72). The injury pattern and the severity of the ecchymosis depends on the amount of force used, how directly the instrument made contact, and the specific type of implement used to strike the child ( 72). Welts are more complex skin lesions in which swelling accompanies bruising from injury through lashing or whipping.

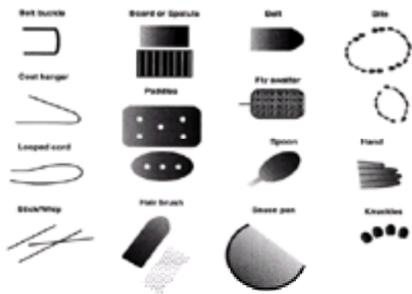


FIGURE 7-2. Healing soft tissue injuries may resemble the instrument used to inflict the lesion.

The type of soft tissue injuries in child abuse may depend on the age of the child ( Table 7-3). In the pediatric dentistry literature, in a series of 266 children suspected of being abused, Jesse and Rieger ( 74) also found that bruises were the most common soft tissue injury, with the most common facial injury ecchymosis of the cheek, forehead, periorbital area, or the lip.

Age	Bruises	Abrasions	Lacerations	Scars	Burns	Headed injury
<1 mo	7%	8%	-	-	1%	3%
1-24 mo	64%	13%	4%	4%	12%	3%
>24 mo	6%	2%	4%	11%	3%	3%

Data from McMillan P, Swanson W, Gaffney M, and Daniels C. Soft tissue injury as an indicator of child abuse. *J Bone Joint Surg Am* 77: 1173-1182.

TABLE 7-3. DISTRIBUTION OF SOFT TISSUE INJURIES IN 371 ABUSED CHILDREN

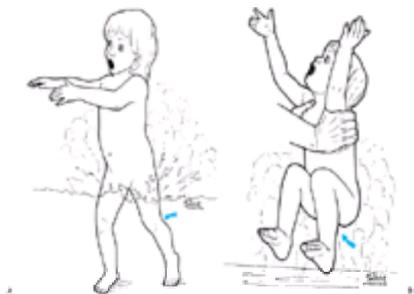
The age of a cutaneous contusion reportedly can be estimated by a change in its color over 2 to 4 weeks after injury, with fading of the lesions beginning at the periphery. An acute contusion is blue or reddish purple; it gradually changes to green, then to yellow, with final resolution as a brownish stain as the hemoglobin is finally broken down ( 159). Langlois and Gresham ( 97) noted that a yellowish bruise must be older than 18 hours; a red, purple, blue, or black coloration of the bruise may be present from 1 hour after injury to resolution; red is always present in bruises regardless of the age; and bruises of identical age and etiology on the same person may be of different appearances and may change at different rates. Although the age of a superficial contusion may be approximately dated by its appearance, a deep contusion may take some time to rise to the skin surface because of fascial planes and thus delay visible ecchymosis.

Natural skin lesions should not be mistaken for bruises. Mongolian spots are naturally occurring deep-blue pigmented areas that are present on the lower back at birth, usually just proximal to the buttocks. They occur more commonly in black and Asian infants than in white infants ( 15). Unlike traumatic bruises, they do not change in color but gradually resolve as the child matures ( 72). Cultural differences should always be considered when unusual skin lesions are noted. Vietnamese children may be subjected to a folklore medical practice known as cao-gio, which causes suspicious scratches and bruises on the back and may be mistaken for child abuse ( 13).

Acute lacerations in various stages of healing and chronic scarring may be present in an abused child. Like bruises, the configuration of the injury can resemble the weapon used to inflict the injury on the child. Although minor lacerations around the eye are fairly common, multiple scars due to either lacerations or burns ( 125) are suspicious for abuse.

### Burns

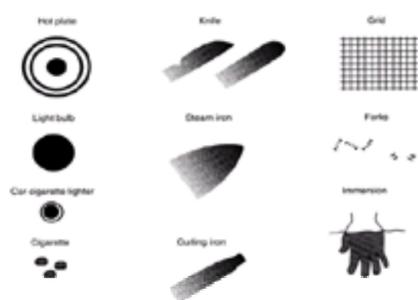
Burns are found in approximately 20% of abused patients ( 61) and are most likely to be found in patients younger than 3 years of age ( 109). Burn evaluation should include configuration, approximate percentage of body surface area, location, distribution, uniformity, length of time the child was in contact with the burning agent, temperature of the burning agent, and presence or absence of splash marks when hot liquids are involved ( 72). Most self-inflicted accidental pour or spill burns occur anteriorly on the child, but accidental burns can also occur on the back. In accidental burns, the injury usually has an arrowhead configuration in which the burn becomes shallower and more narrow as it moves downward, and there may be splash marks surrounding the lesion ( 72). Abuse should be suspected when deep second- or third-degree burns are seen well demarcated with circumferential definition. In accidental hot water immersion, an indistinct stocking or glove configuration may be seen with varying burn depths and indistinct margins. In deliberate immersion burns, the burn demarcation shows uniform depth and a well-demarcated water line ( 72). Particularly severe burns occur when a child's buttocks are immersed in hot tap water as abusive punishment. The central aspect of the buttocks may be spared, giving a doughnut-like appearance to the burn. Galleno and Oppenheim ( 61) noted that in accidental hot water immersion, the child is uniformly scalded about the lower extremities as the legs are quickly extended by the child to climb out of the water, but in deliberate, abusive immersion the children are lowered into the water, so they instinctively pull up their legs to avoid the scalding hot water, and knee flexion may protect the popliteal areas from burn ( Fig. 7-3). The average child abused by scalding burns is an undernourished 2-year-old child with burns involving 15% to 20% of the body, usually the buttocks; these children have a 10% to 15% mortality rate from burn owing to sepsis ( 127).



**FIGURE 7-3. A:** In accidental hot water immersion, the child is uniformly scalded about the lower extremities as the legs are extended quickly by the child to climb out of the water, with burns occurring behind the knee (*curved arrow*). **B:** In nonaccidental immersion, the child instinctively pulls up the legs to avoid the hot water, and knee flexion may protect the popliteal areas from burn (*curved arrow*)

Burns also can be inflicted by many objects commonly found in the household. Sometimes the object can be identified by the configuration of the burn. Intentional burns by cigarettes are circular, deeply excavated, and sometimes repetitive, usually about 8 mm in diameter (72). Isolated cigarette burns may be accidental, and in such cases, they are usually superficial, pointing downward, and often appearing triangular. Lesions of impetigo resemble those of cigarette burns, but they are much more superficial.

Contact with heated objects may cause burns of unique shape that allow identification of their etiology (Fig. 7-4). Children accidentally grasping curling irons sustain burns of the palms, whereas burns on the dorsum of the hands are more suspicious for abuse (76). Hair dryers can be used to inflict burns on children, and full-thickness skin burns can result from contact with the grill of a dryer up to 2 minutes after it has been turned off (124). Such burns can resemble those caused by hot water or could have a grill pattern caused by direct contact with the dryer. Abuse burns have also been inflicted by stun guns (59). These devices deliver a high-voltage impulse of up to 100,000 volts at 3 to 4 mA, incapacitating the individual and leaving hypopigmented burn scars on the skin 0.5 cm apart. Rope burns, sustained when children are restrained about the wrists for beatings, may be seen as circular scars about the wrists (76). Full-thickness skin burns have been reported in small children who were placed in microwave ovens (7).



**FIGURE 7-4.** Contact burns by heated objects may cause burns of unique shape that allow identification of their cause.

Certain folklore practices may cause lesions simulating abusive burns. Round burns on the abdomen, buttock, or between the thumb and forefinger of Southeast Asian children may be due to a variant on the Chinese medical practice of moxibustion. Folk medical practitioners burn balls of the moxa herb on the surface of the skin for therapeutic purposes, and both cigarettes and yarn have been similarly used in refugee camps. The knowledge of these practices may help to avoid inappropriate accusations of child abuse (57). Infants may sustain second-degree burns when they accidentally come in contact with the hot vinyl upholstery of a car (133).

### Abdominal Injuries

Trauma to the abdomen is the second most common reason for death from child abuse (21). Careful evaluation is needed to rule out occult abdominal injury in the abused child. These are injuries due to beatings with the hand or fist and also can occur when the child is thrown into a fixed object. External abdominal bruising is present in only 12% to 15% of cases of major abdominal injuries (72). Patients with abdominal injury due to child abuse may have fever, vomiting, and anemia, with abdominal distention, localized involuntary spasm, and absent bowel sounds (121). One of the most common abdominal injuries is a ruptured liver or spleen, and the hypovolemic shock from blood loss can be fatal (156). Blunt trauma to the abdomen also may cause intestinal perforation, usually involving the large intestine, and the physical examination suggests peritonitis with free air seen on abdominal radiograph. Intramural duodenal hematoma may cause obstruction and projectile vomiting (72). More severe trauma may cause duodenal avulsion or transection with nausea, vomiting, and clinical acute abdomen (155). Severe blunt trauma to the abdomen or a blow to the back may cause renal or bladder contusion with hematuria. Blunt trauma to the abdomen also may result in shearing of the mesenteric vessels, leading to hypovolemic shock due to blood loss. Pancreatic transection can occur where the pancreas overlies the vertebral column, and a pseudocyst may form (72).

Liver function tests are useful in detecting abdominal injury due to occult liver injury in child abuse. In one study (39), elevated aspartate aminotransferase, alanine aminotransferase, and lactic dehydrogenase enzyme levels were useful markers for occult liver lacerations in abused children who had false-negative abdominal examinations. A CT scan is used to define liver injury. If a bone scan is obtained to diagnose occult fracture, there may be abnormal patterns of isotope uptake in the abdomen due to intestinal injury, renal contusion, or muscle trauma of the abdominal wall (71).

When abdominal injury is suspected in an abused child, the hematocrit and hemoglobin levels are checked, the child is typed and crossmatched for blood, and an intravenous line is placed to provide replacement fluids. Emergency general surgery consultation should be obtained. The overall mortality rate associated with visceral injury in child abuse is 40% to 50% (39).

### Genital Injuries

Sexual abuse should always be considered when evaluating a physically abused child. Children who have been sexually abused can have symptoms of bed wetting, fecal incontinence, painful defecation, pelvic pain, abdominal pain, vaginal itching and bleeding, sexually transmitted diseases, and pregnancy in postmenarchal women. Types of sexually transmitted diseases found in abused children include gonorrhea, syphilis, chlamydia, trichomoniasis, and lymphogranuloma venereum. Although the percentage of sexually assaulted children with obvious physical trauma to the genitalia is low, failure to document such findings is a serious matter because sexual abuse is always a criminal offense and must be reported to legal authorities.

The genitalia should always be examined in a chaperoned setting. Infant and toddler girls are placed in a supine frog-leg position, and boys are placed in either a prone knee-chest position or in a lateral decubitus position (140). Patterns of injury that suggest sexually motivated assault include bruises, scratches, and burns around the lower trunk, genitalia, thighs, buttocks, and upper legs, including the knees. Pinch or grip marks may be found where the child was held. Attempted or achieved penetration may involve the mouth, vagina, or anus (70). Sexually abused boys may have poor rectal sphincter tone, perianal scarring, or urethral discharge. Female genital examination findings that are consistent with, but not diagnostic of, sexual abuse include chafing, abrasion, or bruising of the inner thighs or genitalia; distortion of the hymen; decreased or absent hymen; scarring of the external genitalia; and enlargement of the hymenal opening (10). The examination of the female genitalia can be normal even when there has been penetration, because hymenal tissue is elastic and there can be rapid healing of trauma. There also is a wide variability of appearance of normal female genitalia (31,55), but posterior hymen disruption is rare and is suspicious for abuse (20). If the sexual assault occurred

within 72 hours of evaluation, then a rape kit must be used by the evaluating physician or nurse examiner to provide medical evidence of the attack ( 96).

The physical signs of sexual abuse, including genital trauma, sexually transmitted diseases, or presence of sperm are present in only 3% to 16% of verified sexual assaults (17,140), but even this minority of patients will be undiagnosed if sexual abuse is not considered when a child presents with musculoskeletal injury due to abuse. The orthopaedist or a consultant such as a pediatrician or a gynecologist must perform and document the genital examination in children with physical abuse.

## FRACTURES IN CHILD ABUSE

Fractures documented on plain x-ray studies are present in 11% to 55% of abused children and are most common in children younger than 3 years of age ( 1,43,63). Fractures due to abuse should be suspected in a young child if the caretaker brings in the child for evaluation, reporting no accident, but does report a change in the child such as extremity swelling or decreased movement of the limb (99). Infants in the first year of life with a fracture of the skull or the extremities have an equal risk of the etiology being either accident or abuse ( 107). Femoral fractures are especially suspicious for child abuse in younger children. One study ( 12) found that 79% of patients younger than 2 years of age with femoral fractures were battered, and of those, two thirds had femoral fractures as their only sign of abuse. Accidental femoral fractures can occur in children old enough to stand or run who fall with a twisting injury to the lower extremities, but femoral fractures in children younger than 1 year of age are most likely due to abuse ( 154). Fractures of both the lateral clavicle and the scapula are suggestive of abuse in young children ( 94). Infants may normally have a separate ossification center adjacent to the tip of the acromion, simulating a fracture ( 92), but a true fracture has sharp, demarcated edges; may be positive on bone scan; and will show callus on healing ( Fig. 7-5). Fractures of the hands and feet are most commonly due to accidental trauma in older children ( 113) but are suspicious for abuse in infants. Nimkin et al. ( 117) reviewed 11 hand and foot fractures in abused children younger than 10 months of age and found mostly torus fractures either of the metacarpals or the proximal phalanges of the hand and similar fractures of the first metatarsals of the feet. Clinical signs of fracture were present in only one patient, and bones scans were insensitive to the presence of the fractures in all patients.



**FIGURE 7-5. A:** A 7-week-old girl was seen in the emergency department with swelling of the left shoulder. History revealed her being roughly grabbed by the shoulder by one of her parents. The anteroposterior x-ray study shows a transverse lucency of the acromion, which was thought to represent either a congenital lesion or a traumatic fracture (arrow). **B:** A technetium bone scan was obtained. Although the delayed anterior image was normal, the posterior view showed increased uptake in the left shoulder in the area of the acromion (arrow) and a fracture was diagnosed. The incident was investigated. **C:** A follow-up x-ray study 9 days later showed callus bridging the fracture site of the acromion (arrow).

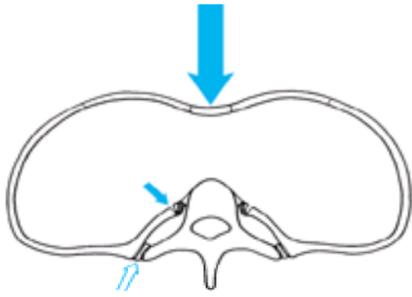
All types of fractures have been reported in the child abuse literature. In one of the largest series, King et al. ( 80) reported 429 fractures in 189 abused children. Fifty percent of these patients had a single fracture, and 17% had more than three fractures. Approximately 60% of fractures were found in roughly equal numbers in the humerus, femur, and tibia. Fractures also occurred in the radius, skull, spine, ribs, ulna, and fibula in order of decreasing frequency. Another study ( 121) found a similar incidence of fractures of the humerus, femur, and tibia in abused children, with skull fractures seen in 14% of patients ( Fig. 7-6). In contrast, Akbarnia et al. ( 3) found that rib fractures in abused patients were twice as prevalent as fractures of any one long bone; the next most frequently fractured bone was the humerus, followed by the femur and the tibia. Nearly a third of their patients had skull fractures. Loder and Bookout ( 102) found the tibia to be the bone most commonly fractured in their series of abused children, followed by the humerus, the femur, the radius, and the ulna. Skull fractures were the most commonly reported fracture in one series (109), but only 10% of their patients underwent x-ray evaluation.



**FIGURE 7-6. A:** A 4-month-old male without history of trauma was brought into the emergency department by his parents with a history of decreased use of the arm. This distal humeral shaft fracture was seen on x-ray study. **B:** Skeletal survey disclosed a posterior skull fracture (arrow), and the injuries were investigated.

## Rib Fractures

Rib fractures are uncommon in childhood accidents, so their presence is very suspicious for child abuse, especially when other long bone fractures are present ( 147). Abusive rib fractures may be caused by squeezing of the chest by a caretaker ( 29), hitting the child from behind, or stepping on the chest ( 90,143). Kleinman et al. (90) postulated that severe shaking of an infant (see Fig. 7-1) can cause front-to-back chest compression, which levers the posterior rib over the transverse process of the vertebral body, causing fractures of the posterior rib shaft at the transverse process and of the rib head adjacent to the vertebra ( Fig. 7-7). These posterior rib fractures are difficult to diagnose acutely because of a lack of displacement. Even with healing, the callus on x-ray study may be obscured by the overlying transverse process (86), and a bone scan may be needed to make the diagnosis (Fig. 7-8). Posterior rib fractures are believed to be most common in child abuse, but fractures may occur anywhere along the arc of the rib, including disruption of the anterior costochondral junction. Posterior paraspinal rib fractures tend to occur between T4 and T9, rib fractures in the axillary line occur mostly in the lower rib cage, and disruption of the anterior costochondral junction usually occurs from the second to the ninth ribs (40).



**FIGURE 7-7.** With anterior compression of the chest, the posterior rib is levered over the transverse process of the vertebral body ( *open arrow*), causing fractures of the rib head adjacent to the vertebra ( *shaded arrow*) as well as avulsion fractures of the rib at the transverse process.

Lateral rib fractures, both acute and chronic, may be difficult to see on standard anteroposterior (AP) x-rays, so oblique views may be necessary. Healing fractures show early callus, but healed fractures may be subtle, with only a fusiform thickening of the rib (see Fig. 7-8). Old fractures of the ribs in child abuse may form lytic, expansile lesions that mimic a tumor (104). Fractures along the lateral arc of the rib are likely due to AP compression of the chest ( Fig. 7-9A), while lateral compression of the chest likely causes fractures of the rib along its posterior arc lateral to the transverse process as well as disruptions of the costochondral junction (Fig. 7-9B).



**FIGURE 7-8. A:** This 5-week-old infant was presented by her parents with a complaint of irritability when her left leg was handled. Her mother stated the infant's 18-month-old sibling may have "kicked" her in the leg. X-ray studies showed an acute fracture of the proximal tibia. **B:** Skeletal survey showed a healing fracture of the left distal clavicle ( *arrow*), a healed fracture of the anterior left 5th rib ( *arrow*), and question of healing posterior right 6th, 7th, 8th, and 9th ribs ( *arrow*). **C:** The posterior image of the delayed phase of the bone scan confirmed the presence of healing posterior rib fractures ( *arrow*).



**FIGURE 7-9. A:** Anteroposterior compression of the infant chest causes fractures both at the rib head and adjacent to the transverse processes ( *open arrow*), but fractures along the lateral arc of the rib are also possible because of the acute bending of the ribs at this point by compression ( *shaded arrow*). **B:** Lateral compression of the chest likely causes fractures of the rib along its posterior arc lateral to the transverse process, as well as possible disruption of the costochondral junction ( *open arrows*).

Acute anterior costochondral separations of the ribs may be difficult to see on x-ray study, but ultrasound can show costochondral dislocation ( 141) and, with healing, the anterior end of the osseous rib becomes widened and clubbed on x-ray study ( 94,121).

In the rare instances when rib fractures are discovered in abused infants undergoing resuscitation for cardiac arrest, there may be confusion about the etiology of the fractures (42), but the elasticity of the infant chest seems to enable it to tolerate compressions, with only 2% of 94 nonabused infants resuscitated having rib fractures in one series (23). In addition to rib fractures, abused infants can sustain severe lung contusion and respiratory distress from chest wall trauma ( 108), with fatal fat embolus reported (115). Necklace calcifications may be present in strangulation cases ( 33).

In the case of infant fatalities of suspicious origin, postmortem high-detail skeletal surveys and specimen radiography can be helpful in diagnosing child abuse ( 83). In a postmortem study of 31 infants who died of inflicted skeletal injury, Kleinman et al. ( 88) found a total of 165 fractures (51% rib fractures, 39% metaphyseal long bone fractures, 5% long bone shaft fractures, 4% fractures of the hands and feet, 1% clavicular fractures, and less than 1% spinal fractures). It is important to obtain skeletal radiography before autopsy to avoid artifact (P. Kleinman, Personal communication, 1998).

### Spinal Fractures

Spinal fractures in child abuse are rare but can occur when a child is forcibly slammed onto a flat surface with hyperflexion of the spine ( 1). Based on autopsy findings (84), spinal fractures of fatally abused children generally involved 25% or less compression of the vertebrae. Half of the fractures involved the anterosuperior end plate associated with a compression deformity, 30% had pure compression fractures, and 20% had fractures of the superior end plate without significant compression. In another study of fractures of the cervical spine, prevertebral soft tissue edema on x-ray study was the only sign of cervical injury, because spontaneous reduction of the cervical vertebrae after dislocation was common ( 150). Thomas et al. (153) reported a 9-week-old boy with spinal cord injury due to cervical spine fracture who presented as a floppy infant. Although routine cervical radiographs were normal, magnetic resonance imaging (MRI) studies showed retropulsion of a fragment of the primarily cartilaginous C3 vertebrae into the spinal canal. Carrion et al. ( 32) reported circumferential physal fractures of the spine associated with child abuse that required open reduction. Bone scans using single photon emission computed tomography can be helpful for diagnosing occult compression fractures of the thoracic spine (40). Although neurologic injury in spinal fractures due to child abuse is uncommon ( 41), any patient with abusive spinal injury should undergo complete neurologic examination.

### ADDITIONAL IMAGING STUDIES

In addition to standard x-ray studies of the acute injury, a skeletal survey is used to detect the presence of additional fractures in battered children. The skeletal

survey should include separate AP and lateral views of the skull and the entire spine, and an AP view of the chest and extremities that includes the shoulders, pelvis, extremities, feet and hands (98). Oblique x-ray studies of the hand are recommended to detect subtle torus fractures of the metacarpals and the phalanges (117). A single AP x-ray study of an entire infant, the so-called baby gram, is not adequate because the obliquity of the angle at which the x-ray transverses the skeleton may obscure subtle fractures (40). The indications for skeletal survey are not completely clear. Merten et al. (112) recommended skeletal survey in infants 1 year of age or younger when there is evidence of neglect and in children age 2 years or younger when clinical abuse is evident. The American Academy of Pediatrics Section on Radiology (11) considered a skeletal survey mandatory in all cases of suspected physical abuse in children younger than 2 years of age, but the sensitivity of skeletal surveys was unclear in older patients. The cost-effectiveness of such skeletal surveys appears to be low because in one study of 331 children, only eight patients without overt physical signs of child abuse had occult fractures revealed by the survey (54), but the use of the skeletal survey in these eight patients possibly prevented both reinjury and death. Perhaps when the death of a child is the ultimate risk of the misdiagnosis of child abuse, we should not play the odds but use every medical test at our disposal to identify the child at risk for abuse so we can try to protect him or her from further possible fatal abuse.

Sty and Starshak (145) reported a false-negative rate of 12.3% in skeletal surveys of abused children, and suggested that a technetium bone scan is the best screening test for occult fractures. Technetium bone scans, however, may fail to diagnose either epiphyseal or metaphyseal fractures and often fail to show skull fractures (113,145). Jaudes (73) found that when results of either a bone scan or a skeletal survey were normal in an abused child, the use of both tests often revealed additional occult fractures. Technetium bone scans are especially useful in the diagnosis of occult rib fractures (40,143), but consistent interpretation is difficult in children younger than 18 months of age. Technetium scans are not useful for dating fractures, because increased isotope uptake may occur at a fracture as early as 24 hours after injury and scan abnormalities may persist for years (58). Kleinman et al. (91) reported that a follow-up skeletal survey 2 weeks after the initial series detected 27% more fractures and provided assistance in dating in 20% of previously detected fractures.

Other imaging techniques may be useful in investigating suspicious x-ray study or bone scan findings. Markowitz and co-workers (105) found that sagittal and coronal sonograms of injured knees in abused children were helpful in diagnosing epiphyseal fractures when compared with the normal side. They cautioned that epiphyseal fractures due to rickets in neonates can be confused with child abuse on ultrasound evaluation. Rogers and Poznanski (130) pointed out that acute interruption of the physis in trauma of any etiology can be seen on MRI. They recommended multiplanar gradient-echo imaging with a repetition time of 50 to 700 ms, an echo time of 200 ms, and a flip angle of 20 degrees for optimal imaging of the physis.

## INTERPRETING IMAGING STUDIES IN CHILD ABUSE

There is no predominant pattern of diaphyseal fracture in child abuse. Traditionally, a mid-shaft spiral fracture is believed to be caused by a violent twisting injury to the extremity of the child. In a study of 23 long-bone fractures in battered children, Herndon (69) found spiral fractures in 78%. He suggested that in children younger than 3 years of age, a spiral fracture of a long bone is highly suggestive of child abuse. Other authors (61), however, found that 71% of diaphyseal fractures were transverse in abused children. In another study of 34 patients in the first year of life, there was no difference in fracture patterns between those injured by trauma and those injured by abuse. Loder and Bookout (102) reviewed 69 long bone fractures in abused children and found that 56% were transverse, 36% oblique, and only 8% spiral. In another study of 429 fractures (80), 48% of fractures were transverse and 26% were spiral. Most of these long bone fractures were in either the middle or distal third of the shaft. Transverse fractures are most commonly associated with either a violent bending force or a direct blow to the extremity, whereas spiral or oblique fractures of the long bones are due to twisting injuries or a fall. Significant rotational force is required, however, to produce a spiral fracture with a fall (1). Another author (154) emphasized that children old enough to run can fall and fracture their femurs if there is a significant twisting motion at the time of injury. In delayed follow-up, long bone fractures may show exuberant callus because of a lack of immobilization, and multiple fractures may be present in different stages of healing (2). Juxtacortical calcification may be seen without fracture when there is diaphyseal periosteal separation due to tractional or torsional force when the limb is grasped or pulled along the shaft of the bone (112).

Metaphyseal and epiphyseal fractures of the long bones are classically associated with child abuse (28,139). In toddlers, these fractures can occur when the child is violently shaken by the extremities (Fig. 7-10) with direct violent traction on or rotation of the extremity (113). Metaphyseal fractures may be characterized by impaction into the epiphysis, with profound production of new periosteal bone. Buckle fractures may be present in multiple sites, and these injuries seldom produce exuberant callus. Repeated injury causes irregular metaphyseal deformities. Periosteal avulsion produces new bone formation within 2 to 3 weeks of injury and can be confused with osteomyelitis (2). New bone formation may be delayed, however, in children with malnutrition. Metaphyseal fractures composed 40% of fractures in one series (61) but less than 15% in another (94).



**FIGURE 7-10.** When a toddler is violently shaken by the extremities, long bone fractures can occur through either direct violent traction or rotation of the extremity. These children are also at risk for closed-head injury because of violent motion of the head during the shaking episode (arrow).

Kleinman (81) ranked the specificity of skeletal trauma for abuse (Table 7-4). Distinguishing between an accident and child abuse is based on both location and type of fracture. He cautioned that both moderate- and low-specificity x-ray findings of child abuse become more highly specific when there is an inadequate explanation for the injury.

<b>High Specificity</b>
■ Any metaphyseal lesion
■ Posterior rib fracture
■ Scapular fracture
■ Spinous process fracture
■ Sternal fracture
<b>Moderate Specificity</b>
■ Multiple fractures, especially bilateral
■ Fractures of different ages
■ Epiphyseal separation
■ Vertebral body fracture or subluxation
■ Digital fracture
■ Complex skull fracture
<b>Low Specificity</b>
■ Clavicular fracture
■ Long bone shaft fracture
■ Linear skull fracture

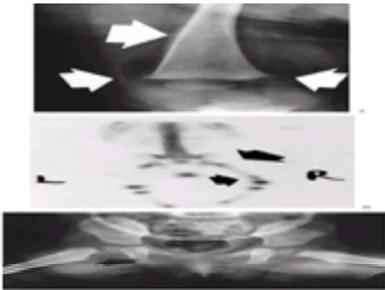
Data from Kleinman PK, ed. Diagnostic imaging of child abuse. Baltimore: Williams & Wilkins, 1987.

**TABLE 7-4. SPECIFICITY OF SKELETAL TRAUMA FOR ABUSE**

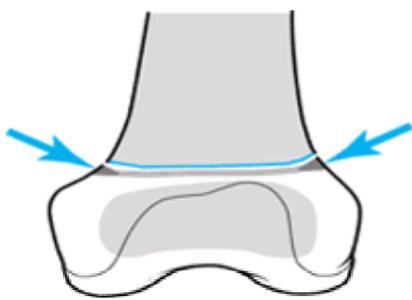
### The Corner Fracture of Child Abuse

The so-called pathognomonic fracture of child abuse is the corner or bucket-handle fracture of the metaphysis (1,2). On x-ray study, there is no gross displacement of the epiphysis, but a corner or chip fracture is seen at the edge of the involved metaphysis (Fig. 7-11). If a significant portion of the metaphyseal rim is involved, a bucket-handle fracture pattern is produced. Another fracture pattern that may be seen is a faint metaphyseal lucency just adjacent to the physis. Classically, these

fractures have been believed to result from avulsion of metaphyseal fragments through the periosteal attachment of the epiphysis ( 28,98). Kleinman et al. (85,87) challenged this view based on their histopathologic study at autopsy of metaphyseal fractures of abused infants. They found that in bucket-handle or corner fractures, there was actually a full-thickness metaphyseal fracture extending through the primary spongiosa of bone just above the zone of provisional calcification. This fracture corresponded to the zone of metaphyseal lucency occasionally seen on x-ray. Centrally, the amount of metaphysis remaining attached to the physis was thin, but peripherally, the fracture line curved away from the physis so that a substantial metaphyseal rim remained attached to the physis. On x-ray study, this metaphyseal rim formed the basis for both corner and bucket-handle fractures ( Fig. 7-12). In healing fractures, biopsy specimens showed metaphyseal extension of hypertrophied chondrocytes (89,122). These fractures are most likely caused by either violent shaking or traction injuries to the extremity and are specifically suggestive of child abuse (1) (see Fig. 7-10); however, they are not the most common fractures in abused children, with their incidence in large series ranging from 15% to 32% (61,80,94,102). Subepiphyseal-metaphyseal lucency can also be caused by systemic diseases such as rickets and leukemia.



**FIGURE 7-11.** A: An 11-month-old girl with a history of right thigh swelling was seen. X-ray studies of the right femur show corner or “chip” fractures of the distal femur (bottom arrows). Faint periosteal calcification is also visible along the lateral cortex ( top arrow). Such fractures are suspicious for child abuse. B: A technetium bone scan showed increased uptake of isotope in the distal and proximal femur ( arrows). C: A healing epiphyseal plate fracture of the proximal femur is seen ( arrow).



**FIGURE 7-12.** In the corner or bucket-handle fracture, full-thickness metaphyseal fracture extends through the primary spongiosa just above the zone of provisional calcification. Centrally, the amount of metaphysis remaining attached to the physis is thin, but peripherally, the fracture line curves away from the physis so a substantial metaphyseal rim remains attached to the epiphyseal plate ( arrows). On x-ray study, this metaphyseal rim (dark shaded area) forms the basis for both the corner and bucket-handle fractures.

## DATING FRACTURES

X-ray proof of unexplained fractures in various stages of healing is believed to be strong evidence of child abuse ( 3) ( Fig. 7-13). The orthopaedist often is asked to estimate the age of fractures with some certainty to corroborate a history of injury given by caretakers ( Fig. 7-14). Whereas most experienced orthopaedists can roughly estimate the age of fractures based on x-ray appearance, specific guidelines have been established for estimating the age of fractures in children ( 118) ( Table 7-5). Both age and nutritional status can affect the appearance of callus around healing fractures ( 47). Growth lines present on radiographs may be helpful in dating fractures (52). In general, fractures on x-ray study are acute until callus appears, then their age is based on the presence of either soft or hard callus some weeks later ( Fig. 7-15). The most difficult fractures to date are those that are completely healed with substantial remodeling, and often the only sign of fracturing is a thickened cortex ( Fig. 7-16).



**FIGURE 7-13.** A: A 7-month-old boy presented with a nondisplaced left distal tibial fracture with periosteal calcification extending up the tibial shaft ( arrows). B: A skeletal survey revealed dense, sclerotic cortical thickening of the contralateral tibia consistent with healed fracture ( arrow). The presence of multiple fractures in different stages of healing is very suggestive of child abuse.



**FIGURE 7-14.** A 3-year-old female was brought to the clinic with a swollen elbow. The mother stated that the child had fell only a week ago, but x-ray studies of the elbow showed a healing supracondylar fracture of the distal humerus that was likely 2 to 3 weeks old based on presence of soft callus ( arrows). Child protective services was contacted and a full examination including skeletal survey was performed. No other signs or risk factors for child abuse were detected. The mother then admitted that the child had indeed fallen 3 weeks ago. She was allowed to take her child home after treatment and was counseled about the need for prompt medical

care when her children were injured.

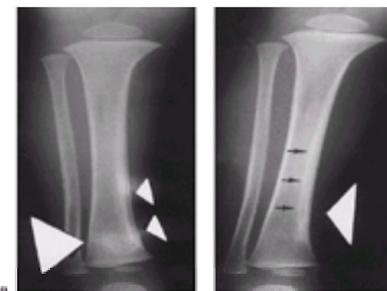
X-Ray Appearance	Peak Changes
Resolution of soft tissue swelling	4–10 days
New periosteal bone	10–14 days
Loss of definition of fracture line	14–21 days
Presence of soft callus	14–21 days
Presence of hard callus	21–42 days
Remodeling of fracture	1 year (early—3 months; late—2 years to maturity)

After O'Connor, JF, Cohen J. Dating fractures. In: Kleinman PK, ed. Diagnostic imaging of child abuse. Baltimore: Williams & Wilkins, 1987.

**TABLE 7-5. GUIDELINES FOR ESTIMATING THE AGE OF FRACTURES IN CHILDREN**



**FIGURE 7-15. A:** A 6-year-old patient presents with a nondisplaced transverse fracture of the distal radius ( *arrow*). **B:** At 4-week follow-up, soft callus is seen enveloping the fracture site ( *arrow*). **C:** At 6-week follow-up, hard callus is seen and early remodeling is occurring at the fracture site.



**FIGURE 7-16. A:** An 18-month-old child presents with a 1-month-old nondisplaced fracture of the distal tibia ( *large arrow*). The fracture line is beginning to fade, but periosteal calcification is seen medially ( *small arrows*). **B:** At 5 months after injury, the fracture shows almost complete remodeling ( *large arrow*) with the only sign of past injury a thickened medial cortex of the tibia ( *small black arrows*). Subtle signs of past fracture are best noted by comparing the x-rays of the injured side to the contralateral uninjured side.

## LABORATORY STUDIES AND CONSULTATIONS

An abused child should have a complete blood cell count with sedimentation rate, liver function studies, and urinalysis. Clotting studies should be performed routinely, especially in patients with ecchymosis, to rule out a blood disorder as a cause for the bruises. If there is any suspicion of substance abuse by any family member, a toxicology screen should also be performed on the patient ( 61). Any significant nonorthopaedic injury should prompt appropriate consultations by neurosurgery, general surgery, plastic surgery, or ophthalmology ( 2). Any female patients who require pelvic examination to rule out sexual abuse should have a gynecologic consultation.

## THE DIFFERENTIAL DIAGNOSIS

Although it is extremely important not to miss the diagnosis of child abuse, it is equally important not to make the diagnosis in error. Kaplan pointed out that overdiagnosing battered child syndrome can be harmful to the family, with the parents being placed at risk of losing custody of their child and also facing criminal charges ( 78). Even direct allegations of child abuse may turn out to be false. Bemet ( 22) pointed out that patients or family friends may make false statements about an abuse situation through misinterpretation, confabulation, fantasy, delusions, and other mechanisms. The American Academy of Child and Adolescent Psychiatry ( 9) has published guidelines for the evaluation of abuse that state that the possibility of false allegations needs to be considered, particularly if allegations are coming from the parent rather than the child, if the parents are engaged in a dispute over custody or visitation, and/or the child is a preschooler.

Normal metaphyseal radiologic appearance should not be confused with child abuse. The radiologic metaphyseal variants include acute angulation of the ossified peripheral tip of the metaphysis adjacent to the physis (most commonly seen in the proximal tibia, distal femur, proximal fibula, distal radius, and distal ulna), which is bilateral in 41% of individuals. A bony beak may be seen medially in the proximal humerus and tibia in rare cases and is bilateral in 77% of individuals. Cortical irregularity in the medial proximal tibia may be seen in 4% of individuals and is bilateral in 25%. Spurs may extend beyond the metaphyseal margins in both the distal radius and lateral aspect of the distal femur, with bilateral variants in 25% of individuals ( 82).

The signs of child abuse found on x-ray study also can be mimicked by systemic diseases such as scurvy, osteogenesis imperfecta, Caffey's disease, osteomyelitis, septic arthritis, fatigue fracture, osteoid osteoma and other tumors, rickets, leukemia, hypophosphatasia, neuromuscular disease, metastatic neuroblastoma, congenital indifference to pain, osteopetrosis, kinky hair syndrome, and prostaglandin therapy ( 2,21). Fractures due to both vitamin D deficiency rickets and drug-induced rickets have been mistaken for child abuse ( 163). There has been an increase in the incidence of syphilis in females of childbearing age, and congenital syphilis can mimic fractures of child abuse with diaphysitis, metaphysitis, and multiple pathologic fractures in different stages of healing ( 100). McClain et al. ( 106) reported a 2-year-old child who died of undiagnosed acute lymphoblastic leukemia. The child had been reported as a possible victim of child abuse because of ecchymosis on the back and extremities. They emphasized that the clinical signs of leukemia, including fever, pallor, petechia, purpura, adenopathy, hepatosplenomegaly, and bone pain, should be sought in children with bruising of unknown origin. Physiologic periostitis ( Fig. 7-17), in contrast to lesions from child abuse, is usually bilateral with no excessive uptake of isotope on bone scan ( 40). The presence of metabolic disease and pathologic fractures does not exclude the possibility of child abuse. Duncan and Chandry ( 51) reported a 3-month-old girl with multiple fractures associated with rickets who died suddenly at 5 months of age. Child abuse was suspected but not proven. Three years later, evidence of child abuse was found in a subsequent sibling in the same family. Several diseases are

commonly brought up in custodial hearings as alternative possibilities to child abuse to explain trauma findings, and these diseases should be carefully explored in the differential diagnosis.



**FIGURE 7-17.** A 10-month-old infant with physiologic periostitis of the femurs (*arrows*). This type of x-ray appearance may be confused with fractures of child abuse, but this periostitis tends to be bilateral with a uniform appearance in contrast to multiple fractures in different stages of healing seen in battered children.

## OSTEOGENESIS IMPERFECTA

Undiagnosed osteogenesis imperfecta should always be considered when a child presents with multiple fractures of unknown etiology, but it may be a very difficult diagnosis clinically. Osteogenesis imperfecta due to spontaneous mutation can occur without a family history (123). The so-called hallmark of osteogenesis imperfecta is an intensely blue sclerae, but this feature is consistently present only in type I (137) and may be completely absent in patients with type IV osteogenesis imperfecta (123). Sillence and Butler (138) noted that patients with either type II or III osteogenesis imperfecta may have blue sclerae at birth but the sclerae can become normal by adolescence. The rare type II osteogenesis imperfecta has normal sclerae, but bone abnormalities and osteopenia are severe and early death is likely (123). Blue sclerae may be present in normal young children and can be misleading as a sign of pathologic bone fragility. The presence of abnormal teeth, known as dentinogenesis imperfecta, may be helpful in a diagnosis of osteogenesis imperfecta if the child is old enough for teeth to have erupted. Plain x-ray studies, however, may show long bones of normal density in both types I and IV osteogenesis imperfecta. Another radiographic sign of osteogenesis imperfecta, wormian bones of the skull, is consistently present only in type III and is often absent in types I and IV (123). Some authors believe that the presence of metaphyseal fracture is pathognomonic for child abuse and, therefore, helpful to distinguish abuse from osteogenesis imperfecta (5), but other authors (45,123) believe that there is no particular fracture pattern that renders the diagnosis of osteogenesis imperfecta likely. Patients with osteogenesis imperfecta tend to bruise to excess, which simulates lesions of child abuse (135), and sudden infant death has also been recorded in patients with undiagnosed osteogenesis imperfecta (119).

Sometimes, when the diagnosis of osteogenesis imperfecta cannot be made on clinical grounds, the diagnosis can be made by biochemical assay. Gahagan and Rimsza (60) stated that 87% of patients with osteogenesis imperfecta have abnormal procollagen that can be detected by current techniques. A skin biopsy is performed for fibroblast culture, and fibroblasts are assayed for both abnormally low levels of procollagen and primary abnormal procollagen (27). Steiner et al. (146) reported that over a 4-year period, 48 patients were referred to them for collagen analysis to rule out the presence of osteogenesis imperfecta in cases of suspected child abuse. Only six of these children had abnormal collagen test results, and in five of those six patients, the diagnosis of osteogenesis imperfecta could have been made on clinical and radiographic grounds. They concluded that routine collagen biochemical testing for osteogenesis imperfecta is unwarranted in these children and collagen analysis should be reserved for the rare instances when diagnostic uncertainty persists in cases of suspected child abuse.

Even when a child has osteogenesis imperfecta, fractures may be due to abuse. Knight and Bennett (93) reported on a young child with osteogenesis imperfecta whose abuse could not be proved until linear bruising of the face suggestive of slapping was documented.

### Temporary Brittle Bone Disease

In 1993, Patterson et al. (123) described 39 patients with a variant of osteogenesis imperfecta that they described as a temporary brittle-bone disease in which fractures were limited to the first year of life and then there was spontaneous improvement. These patients presented with vomiting, followed by diarrhea, anemia, hepatomegaly, episodes of apnea, neutropenia, and edema. The most common x-ray findings were metaphyseal corner fractures, rib fractures, diaphyseal fractures, periosteal reaction of long bones, expanded costochondral junctions, and delayed bone age. Only 31% of patients had osteopenia on x-ray study. They suggested that a self-limiting period of copper deficiency was the cause of this problem, but limited serum copper assays were inconclusive. Other authors (4,6,36) doubt the existence of “temporary” brittle-bone disease because of the rarity of fractures associated with proven copper deficiency syndrome. Judicial authorities (34,103,160) have commented that although one patient in the series of Patterson et al. had injuries due to child abuse, this fact was not included in the report, and they had concerns that assumptions proposed by medical experts that injuries may be considered solely due to disease may inhibit full investigation of such injuries by civil authorities and place children at risk for further abuse.

### Sudden Infant Death Syndrome

In sudden infant death syndrome (SIDS), there is a distinct possibility of child abuse (56), but other causes of sudden death must be excluded. Byard et al. (26) reported a 5-month-old girl who died suddenly because of spontaneous subarachnoid hemorrhage from undiagnosed Ehlers-Danlos syndrome. They recommended collagen analysis in patients with unexplained multifocal spontaneous hemorrhages to exclude this rare syndrome. Sperry and Pfalzgraf (144) reported a 9-month-old infant whose diagnosis of sudden infant death syndrome became uncertain when postmortem x-rays showed healing symmetric clavicular fractures and a healing left medial humeral epicondyle fracture. Subsequent investigation showed that the child had undergone “chiropractic” manipulation 4 weeks before death by an unlicensed therapist to correct “shoulder dislocations,” and the parents were exonerated of abuse charges.

### Accidental Trauma

In considering the differential diagnosis of child abuse, accidental trauma should always be considered. The orthopaedist, however, should be comfortable with the diagnosis of accidental trauma only when the acute injury is brought promptly to medical attention and has a plausible mechanism of injury and there are no risk factors for child abuse.

## TREATMENT AND LEGAL REPORTING REQUIREMENTS

Once child abuse is recognized, the first step in treatment is hospital admission. This is therapeutic in that it places the child in a safe, protected environment and provides the opportunity for additional diagnostic work-up and, more importantly, investigation of the family's social situation by appropriate personnel. In university settings, multidisciplinary teams often are available to evaluate and treat such children, but in other circumstances, the orthopaedist may be primarily responsible for coordinating both evaluation and treatment. Court custody may be required for children of uncooperative families who refuse admission, and hospitalization should be continued until a full investigation is completed by the appropriate child protective services. In the United States, the physician is required by law to report all suspected child abuse to appropriate child protective services or legal authorities. When the reporting is done in good faith, the physician has immunity against criminal or civil liability for these actions, but only in three states—Ohio, California, and Alabama—is this protection extended to include absolute immunity (38). The distinction is critical. Absolute immunity means that the physician who reports suspected child abuse cannot ever be held for damages sought by families for allegedly inappropriate reports of child abuse or neglect. The granting of absolute immunity, even for physicians, is not encouraged by the American legal system because in theory it would protect individuals who make false reports of child abuse in order to harass families and would deprive the injured parties their legal right to seek damages for harmful actions. In contrast, physician immunity based on good faith reporting of suspected child abuse is contingent on the physician having a reasonable belief that abuse or neglect has occurred. Although in theory this protection seems to be quite adequate, recently there has been a dramatic rise in the number of lawsuits filed by families seeking damages for alleged, unfounded reports of child abuse and neglect. Although it is true that by the time these lawsuits are eventually resolved, physicians have almost never been held liable for good faith reports of child abuse; in a substantial number of these cases, the physicians first lost at trial level before eventually prevailing at appeal. Considerable expense, frustration, and loss of time can be experienced by the physician in defending against

such allegations as the families and their attorneys pursue multiple forms of legal theories in court in an attempt to evade the immunity provisions ( 38). On the other hand, failure to report suspected child abuse may expose the physician to charges of malpractice ( 1). All states require physicians to report not only cases of definitive child abuse or neglect but also cases when abuse is just suspected or is considered a possibility. Physicians have been held liable for damages for their negligence in failing to diagnose child abuse when the child subsequently was reinjured by more abuse, and ironically, the parents also may be able to collect additional compensation for losses due to medical expenses. In order for families to be successful in these lawsuits, they must be able to prove that the failure to make the diagnosis of child abuse was negligent and that, had the diagnosis been made, steps would have been taken to protect the child from additional abuse. Although the probability of a physician being held liable under such circumstances is low, the amount of damages can be high if the family does prevail when the child has suffered permanent sequelae (38).

After admission, the orthopaedist proceeds with care of the child's musculoskeletal injuries and coordinates various medical consultations. There should be frequent communications with child protective services to stay current with the results of their investigations. Recommendations for disposition of the child after completion of medical treatment may be a group decision through a multidisciplinary team or, more often, the decision of the primary physician, who may be the orthopaedist. Final disposition choices may include return to the family, return to a family member who does not live in the child's home, or placement in a shelter or a foster home setting. The risk of reinjury and death is significant if the abused child is returned to the unsafe home, so the orthopaedist must strongly support child protective services in custodial actions when it is believed that the child's injury truly occurred from abuse at home. Not only must the definitive diagnosis of child abuse be documented in the chart but a separate notarized affidavit may be necessary. Commonly, custodial actions by child protective services are reviewed in a court hearing in a matter of weeks, and the physician is likely called to testify in the hearing. Criminal charges also may be brought against the perpetrator of the child abuse, and the physician likely also serves as a witness in these proceedings.

## THE COURTROOM

The orthopaedist fills a dual role in the courtroom in child abuse proceedings. First, he or she serves as a material witness whose testimony is confined to the physician's personal involvement in the legal matter. The testimony may include clarification to the court of information contained in progress notes in the chart or of other past documentation. As a material witness, the physician, like the layman, cannot render opinions about the facts as stated during his or her testimony. In addition, however, the physician may also be sworn in as an expert witness ( 64). This is an individual considered by the court to have special knowledge and experience that qualifies him or her to render opinions about certain facts presented in the courtroom. The limits of the physician's expertise are usually defined by the attorneys in court before the testimony of the expert witness.

Physicians usually are reluctant to testify in court for many reasons. The courtroom is an unfamiliar setting for almost all physicians, and the adversarial nature of the American law system makes it a hostile environment. In the courtroom, the perception of truth is just as important as the truth itself, and opposing attorneys will search for inconsistencies in the testimony or unfamiliarity with the record to discredit the physician witness. To avoid being a poor witness, the orthopaedist must meticulously prepare to give testimony.

The orthopaedist preparing to testify in a child abuse case should begin with a thorough review of the child's medical records and a review of recent medical literature on the subject of child abuse (64). Often, there is a pretestimony discussion with child protective services counsel in family court cases or the district attorney's office in criminal cases. Such meetings should preferably be in person, and the orthopaedist's professional training and expertise are examined to determine whether he or she may serve as a material witness, an expert witness, or both. The attorney should be provided the orthopaedist's curriculum vitae, and another copy should be made available to the court. If the orthopaedist is to serve as a material witness, the factual information of the case as well as the limitations of the physicians' knowledge are discussed, as are questions that may be posed during testimony. Orthopaedists functioning as expert witnesses should indicate relevant information that should be provided through questioning during testimony. In addition, anticipated testimony from any opposing expert witness and cross-examination questions from the opposing attorney should be discussed. The opposing attorney also may request an informal pretestimony meeting. The orthopaedist should request a list of questions that will be asked in this session ahead of time and request that both the prosecution attorney and the opposing attorney be present during the session, which is often recorded. The next step may be a deposition in which both attorneys question the witness under oath to "discover" the testimony that the witness will provide in court. The primary purpose for a deposition in the discovery process is to keep attorneys from later being surprised in court by testimony of witnesses ( 35). Preparations for depositions should be meticulous. Any testimony the physician gives during the deposition will be recorded, and later in court, any inconsistencies between testimony and prior depositions will be vigorously attacked by attorneys in cross-examination. Depositions are rarely used in criminal prosecutions ( 64).

A subpoena is issued requiring a physician witness to appear at the courtroom at a certain time, but often there may be hours of delay before the testimony actually begins. Through prior arrangements with the attorney, the orthopaedist may be placed "on call" if he or she works within a reasonable distance of the courtroom and can be available a short time before their actual testimony is needed. The physician has no legal right to such treatment and must be prepared to honor the exact conditions of the subpoena if alternative arrangements cannot be made. If significant delays are encountered to giving testimony and the attorneys are not responsive to physician hardship, then the orthopaedist should contact the judge directly to remedy the situation ( 35). In the courtroom, the orthopaedist should be conservatively dressed and appear attentive, competent, poised, and at ease ( 35,64).

Once called to the stand, the orthopaedist is sworn in and identified. Next follows qualification, direct examination, and then cross-examination. In the qualification process, the attorney asks the physician fairly detailed questions about the orthopaedist's training and background to establish whether he or she is a credible witness (64). The attorney wishes to impress the judge or jury with the orthopaedist's qualifications as a witness, whereas the opposing attorney may challenge the witness with questions to cast doubt on his or her expertise ( 35). During this phase, the attorneys also may establish the limits of the physician's expertise as an expert medical witness. Next, the attorney will proceed with direct examination. A series of questions are asked that aim at developing a logical and progressive line of thought leading to a conclusion ( 35). In child abuse cases, in particular, the testimony will lead to the fact that the abuse has occurred and that it has been appropriately diagnosed. In addition, the physician expert witness may be asked to give an opinion of the risk for subsequent abuse if the child returns to the home where the alleged abuse occurred. Almost never will the physician witness be asked about the guilt or innocence of the caretaker accused of abuse, but the orthopaedist in certain circumstances will come close to answering the "ultimate question" ( 35), by testifying about a child's statement of history if it identifies the abuser. Some states, however, restrict such testimony. In Maryland, a physician may not testify regarding any disclosures made by a child abuse victim unless the disclosure is admissible under a recognized exception to the rule prohibiting hearsay evidence ( 46). The orthopaedist should ask about any possible restrictions on his or her testimony with the attorney in pretrial discussion. In testimony, the orthopaedist will want to use the courtroom setting to advocate for the safety and well-being of the child ( 64). Questions regarding medical findings will often be prefaced in the courtroom by the words "reasonable medical certainty," a term that is poorly understood by most physicians. Chadwick ( 35) offered a definition of reasonable medical certainty as "certain as a physician should be in order to recommend and carry out treatment for a given medical condition." He offered an example that the certainty for the diagnosis and treatment of leukemia must be much higher than that for diagnosis and treatment of a viral upper respiratory infection.

During testimony, the orthopaedist's words should be carefully chosen and should be understandable by a lay jury. Testimony should be objective, honest, and thorough (64). Attorneys may frame questions in ways that are difficult to understand, and the orthopaedist should not hesitate to ask the attorney to clarify a question (35). Answers should be brief, without volunteering extra information, but the perception listeners will have of the answers should be carefully considered by the orthopaedist. In particular, attorneys may phrase yes or no questions that could place misleading words in the mouth of the orthopaedist. In such situations, when neither response is appropriate, the orthopaedist should answer in a sentence that provides an accurate answer ( 64). Physicians are considered expert to the court because they have more information than is usual and customary, and they are to provide the judge and jury with an unbiased expert opinion ( 64). Language should be straightforward, and visual aids may be used in providing clear testimony. The expert should use testimony as an educational process for the court, in which the common experience and knowledge of the jury is used to build understanding with common sense explanations of medical findings ( 35).

Cross-examination by the opposing attorney follows direct examination. The opposing attorney's role is to challenge the material presented by the physician witness to protect the defendant (64). This may involve an attempt to bring into question the physician's credibility, the medical record, the physician's training or expertise, or the physician's objectivity or composure and clarity of thought before the jury ( 64). Attorneys may accomplish this by finding inconsistencies with prior statements, asking leading questions as well as questions that allow only certain desired answers, and minimizing physician qualifications ( 35). The attorney may frame a question that contains certain elements that the physician agrees with and others that are misleading, and the question will often end with "Isn't that so, doctor?" The physician witness should be firm in answering such questions, clearly stating what in the question they agree with and what they do not. It is also common to encounter questions from attorneys based on hypotheses that are extremely unlikely, and the physician needs to point out that unlikelihood ( 35). Part of the strategy of aggressive cross-examination is to provoke the physician into arguments or unprofessional behavior that could discredit the physician or his or her testimony before the court. In particular, juries will allow aggression on the part of the attorney, but they expect physician witnesses to respond professionally, even under extreme duress (64). Inexperienced potential physician witnesses can prepare themselves by either watching trials or participating in mock trials ( 35). Brent (24) assembled an excellent series of vignettes of expert medical witness case studies in court and provided detailed instructions with regard to the responsibilities of such experts. Both

redirect examination and recross-examination may follow cross-examination at the discretion of attorneys, but usually these procedures are very short ( 35).

## Disposition Following Custody Hearings

After a hearing or trial, the child historically either remained in the protective custody of the state or was returned to the home, but the danger of further abuse exists in both situations. In a study of 206 care and protection petitions brought to the Boston juvenile courts ( 114), 31% were dismissed with return of the child to the parents. During a 2-year follow-up of these dismissed cases, 29% had reports of further mistreatment and 16% were returned to court under another care and protection petition. One risk factor identified by the study was a previous appearance in court. Half of dismissed cases with this risk factor returned to court again. Of the children ordered permanently removed from parental custody by the court, 6% returned to court with evidence of further abuse by another caretaker. Another alternative pathway of custody is gaining popularity with the court systems in which the abused child is released to the custody of a relative of the family with consideration given to the wishes of the parents or other prior custodians of the child. Although in theory this approach may help preserve the integrity of the family unit, the child may still be in danger in this sort of arrangement. Handy et al. ( 65) of the Pediatric Forensic Medicine Program of the Kentucky State Medical Examiner's Office noted evidence of recurrent abuse 2 to 9 months after the original injury in six patients out of 316 referrals to the program. They emphasized that in two of these cases, the child was in protective custody of a family relative when the original perpetrator was allowed unsupervised access to the child in violation of court order. One 6-month-old infant placed in the custody of a grandmother was bent backwards by the original perpetrator until his head touched his feet, sustaining a thoracic spinal fracture with fatal outcome. Another 4-year-old was in the custody of her grandmother when the child's mother gained unauthorized access to her and the child was reinjured with a burn on the face with an iron. It is possible that such reinjuries occur because either the close relatives of child abuser may not believe that the caretaker committed the original abuse or the relatives are under emotional pressure to allow the caretaker to have access to the child in spite of court order. It is hoped that the court systems can strike a balance between the need to preserve the family unit and the need to protect the child from further abuse.

## PREVENTION OF CHILD ABUSE

Prevention of child abuse lies in early intervention. Home visitor programs can contact a mother immediately after the birth of her child and arrange for a visit in which the mother's parenting strengths are assessed. Parents requiring additional support are linked to community agencies and family resources ( 134,142). Such support seems to enhance parent and child interactions, and mothers report a diminished need to punish or restrict their children. Antivictimization programs teach children certain concepts believed to facilitate self-protection, such as identification of strangers, types of touching, saying "no" to inappropriate advances, and telling someone about inappropriate behavior. Parenting education offers instruction in specific parenting skills such as discipline methods, basic child care, infant stimulation, child development, education, and familiarity with local support services and introduction to other new parents in the community ( 156). Continuing abuse can be prevented by the orthopaedist's prompt recognition of child abuse in the emergency department and appropriate intervention.

The most important issue in dealing with child abuse is to help both the child and the family through early recognition of the problem and appropriate therapeutic measures by all health personnel. The failure to diagnose child abuse may result in serious injury to or death of the child.

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## CHAPTER REFERENCES

1. Akbarnia BA. The role of the orthopaedic surgeon in child abuse. In: Morrissy RM, ed. *Lovell and Winter's pediatric Orthopaedics*. Philadelphia: J.B. Lippincott, 1996:1315.
2. Akbarnia BA, Akbarnia NO. The role of the orthopedist in child abuse and neglect. *Orthop Clin North Am* 1976;7:733-742.
3. Akbarnia B, Torg JS, Kirkpatrick J, Sussman S. Manifestations of battered-child syndrome. *J Bone Joint Surg Am* 1974;56:1159-1166.
4. Albin D. Osteogenesis imperfecta: a review. *Can Assoc Radiol J* 1998;49:110-123.
5. Albin DS, Greenspan A, Reinhart M, Grix A. Differentiation of child abuse from osteogenesis imperfecta. *Am J Roentgenol* 1990;154:1035-1046.
6. Ablin DS, Sane SM. Non-accidental injury: confusion with temporary brittle bone disease and mild osteogenesis imperfecta. *Pediatr Radio*. 1997;27:111-113.
7. Alexander RC, Surrall JA, Cohle SD. Microwave oven burns to children: an unusual manifestation of child abuse. *Pediatrics* 1987;79:255-260.
8. Altman DH, Smith RL. Unrecognized trauma in infants and children. *J Bone Joint Surg Am* 1960;42:407-413.
9. American Academy of Child and Adolescent Psychiatry. *Policy statement: guidelines for the clinical evaluation of child and adolescent sexual abuse*. Washington, DC: American Academy of Child and Adolescent Psychiatry, 1990.
10. American Academy of Pediatrics. Guidelines for the evaluation of sexual abuse of children. *Pediatrics* 1991;87:254-260.
11. American Academy of Pediatrics Section on Radiology. Diagnostic imaging of child abuse. *Pediatrics* 1991;87:262-264.
12. Ander WA. The significance of femoral fractures in children. *Ann Emerg Med* 1982;11:174-177.
13. Anh NT. "Pseudo-battered child" syndrome. *JAMA* 1976;236:2288.
14. Asher R. Munchausen syndrome. *Lancet* 1951;1:339.
15. Asnes RS. Buttock bruises = mongolian spot [Letter]. *Pediatrics* 1984;74:321.
16. Babcock J, Hartman K, Pedersen A, et al. Rodenticide-induced coagulopathy in a young child: a case of Munchausen syndrome by proxy. *Am J Pediatr Hematol Oncol* 15:126-130, 1993.
17. Bays J, Chadwick D. Medical diagnosis of the sexually abused child. *Child Abuse Negl* 1993;17:91-110.
18. Beals RK, Tufts E. Fractured femur in infancy: the role of child abuse. *J Pediatr Orthop* 1983;3:583-586.
19. Benedict RB, Wulff LM, Hall BJ. Reported maltreatment in children with multiple disabilities. *Child Abuse Neglect* 1990;14:207-217.
20. Berenson AB, Heger AH, Hayes JM, et al. Appearance of the hymen in prepubescent girls. *Pediatrics* 1992;89:387-394.
21. Berkowitz CD. Pediatric abuse: new patterns of injury. *Emerg Med Clin North Am* 1995;13:321-341.
22. Bernet W. False statements and the differential diagnosis of abuse allegations. *J Am Acad Child Adolesc Psychiatry* 1993;32:903-910.
23. Betz P, Liebhardt E. Rib fractures in children—resuscitation or child abuse? *Int J Legal Med* 1994;106:215-218.
24. Brent RL. The irresponsible expert witness: a failure of biomedical graduate education and professional accountability. *Pediatrics* 1982;7:754-762.
25. Bruce DA, Schut L, Bruno LA, Wood JH, Sutton LN. Outcome following severe head injuries in children. *J Neurosurg* 1978;48:679-688.
26. Byard RW, Keeley FW, Smith CR. Type IV Ehlers-Danlos syndrome presenting as sudden infant death. *Am J Clin Pathol* 1990;93:579-582.
27. Byers PH. Disorders of collagen biosynthesis and structure. In: Scriver CR, Beaudet AL, Sly WS, Valle D. *The metabolic basis of inherited disease*. New York: McGraw-Hill, 1989:2814-2824.
28. Caffey J. Multiple fractures in long bones of infants suffering from chronic subdural hematoma. *Am J Roentgenol* 1946;56:163-173.
29. Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics* 1974;54:396-403.
30. Campbell JC. Child abuse and wife abuse. *Md Med J* 1994;43:349-350.
31. Cantwell HB. Vaginal inspection as it relates to child sexual abuse in girls under thirteen. *Child Abuse Negl* 1983;7:171-176.
32. Carrion WV, Dormans JP, Drummond DS, Christofersen MR. Circumferential growth plate fracture of the thoracolumbar spine from child abuse. *J Pediatr Orthop* 1996;16:210-214.
33. Carty H. Case report: child abuse—necklace calcification—of strangulation. *Br J Radiol* 1993;66:1186-1188.
34. Cazalet J. Note Re R (A Minor) (Expert Evidence) [1991] 1 FLR 291.
35. Chadwick DL. Preparation for court testimony in child abuse cases. *Pediatr Clin North Am* 1990;37:955-970.
36. Chapman S, Hall CM. Non-accidental injury, or brittle bones. *Pediatr Radio*. 1997;27:106-110.
37. Cho D-R, Wang Y-C, Chi C-S. Decompression craniotomy for acute shaken/impact baby syndrome. *Pediatr Neurosurg* 1995;23:192-198.
38. Clayton EW. Potential liability in cases of child abuse and neglect. *Pediatr Ann* 1997;26:173-177.
39. Coant PN, Kornberg AE, Brody AS, Edwards-Holmes K. Markers for occult liver injury in cases of physical abuse in children. *Pediatrics* 1992;89:274-278.
40. Conway JJ, Collins M, Tanz RR, et al. The role of bone scintigraphy in detecting child abuse. *Semin Nucl Med* 1993;23:321-333.
41. Cullen JC. Spinal lesions in battered babies. *J Bone Joint Surg Br* 1975;57:364-366.
42. Cumming WA. Neonatal skeletal fractures: birth trauma or child abuse? *J Can Assoc Radiol* 1979;30:30-33.
43. Dalton HJ, Slovis T, Helfer RE, et al. Undiagnosed abuse in children younger than 3 years with femoral fracture. *Am J Dis Child* 1990;140:875-878.
44. Desilva S, Oates RK. Child homicide—the extreme of child abuse. *Med J Aust* 1993;158:300-301.
45. Dent JA, Patterson CR. Fractures in early childhood: osteogenesis imperfecta or child abuse? *J Pediatr Orthop* 1991;11:184-186.
46. Drake JA. Physicians and attorneys: a partnership on behalf of the youngest victims of family violence. *Md Med J* 1994;43:365-367.
47. Dreizen S, Spirakis CN, Stone RE. The influence of age and nutrition on "bone scar" formation in the distal end of the growing femur. *Am J Phys Anthropol* 1964;22:295-306.
48. Dubowitz H, Black M. Teaching pediatric residents about child maltreatment. *Dev Behav Pediatr* 1991;12:305-307.
49. Dubowitz H, Bross DC. The pediatrician's documentation of child maltreatment. *Am J Dis Child* 1992;146:596-599.

50. Duhaime AC, Gennarelli TA, Thibault LE, et al. The shaken baby syndrome: a clinical, pathological, and biomechanical study. *J Neurosurg* 1987;66:409–415.
51. Duncan AA, Chandry J. Case report: multiple neonatal fractures—dietary or deliberate? *Clin Radio*. 1993;48:137–139.
52. Edwards DK. Skeletal growth lines seen on radiographs of newborn infants: prevalence and possible association with obstetric abnormalities. *Am J Roentgenol* 1993;161:141–145.
53. Eller AW, Brown, GC. Retinal disorders of childhood. *Pediatr Clin North Am* 1983;30:187–201.
54. Ellerstein NS, Norris KJ. Value of radiologic skeletal survey in assessment of abused children. *Pediatrics* 1984;74:1075–1078.
55. Emans SJ, Woods ER, Flagg NT, Freeman A. Genital findings in sexually abused, symptomatic and asymptomatic girls. *Pediatrics* 1987;79:778–785.
56. Emery JL. Child abuse, sudden infant death syndrome, and unexpected infant death. *Am J Dis Child* 1993;147:1097–1100.
57. Feldman KW. Pseudoabusive burns in asian refugees. *Am J Dis Child* 1984;138:768–769.
58. Fordham EW, Ramachandran PC. Radionuclide imaging of osseous trauma. *Semin Nucl Med* 1974;4:411–429.
59. Frechette A, Rimsza ME. Stun gun injury: a new presentation of the battered child syndrome. *Pediatrics* 1992;89:898–901.
60. Gahagan S, Rimsza ME. Child abuse or osteogenesis imperfecta: how can we tell? *Pediatrics* 1991;88:987–991.
61. Galleno H, Oppenheim WL. The battered child syndrome revisited. *Clin Orthop* 1982;62:11–19.
62. Goss PW, McDougall PN. Munchausen syndrome by proxy—a cause of preterm delivery. *Med J Aust* 1992;157:814–817.
63. Green FC. Child abuse and neglect: a priority problem for the private physician. *Pediatr Clin North Am* 1975;22:329–339.
64. Halverson KC, Elliott BA, Rubin MS, Chadwick DL. Legal considerations in cases of child abuse. *Primary Care* 1993;20:407–415.
65. Handy TC, Nichols GR II, Smock WS. Repeat visitors to a pediatric forensic medicine program. *J Forensic Sci* 1996;41:841–844.
66. Hanigan WC, Peterson RA, Njus G. Tin ear syndrome: rotational acceleration in pediatric head injuries. *Pediatrics* 1987;80:618–622.
67. Helfer RE. The epidemiology of child abuse and neglect. *Pediatr Ann* 1984;13:745–751.
68. Helfer RE, Slovis TL, Black M. Injuries resulting when small children fall out of bed. *Pediatrics* 1977;60:533–535.
69. Herndon WA. Child abuse in a military population. *J Pediatr Orthop* 1983;3:73–76.
70. Hobbs CJ, Wynne JM. The sexually abused battered child. *Arch Dis Child* 1990;65:423–427.
71. Howard JL, Barron BJ, Smith GG. Bone scintigraphy in the evaluation of extraskkeletal injuries from child abuse. *Radiographics* 1990;10:67–81.
72. Hyden PW, Gallagher TA. Child abuse intervention in the emergency room. *Pediatr Clin North Am* 1992;39:1053–1081.
73. Jaudes PK. Comparison of radiography and radionuclide bone scanning and the detection of child abuse. *Pediatrics* 1984;73:166–168.
74. Jessee SA, Rieger M. Physical abuse. A study of age-related variables among physically abused children. *Journal of Dentistry for Children* 1996;14:275–280.
75. Joffe M, Ludwig S. Stairway injuries in children. *Pediatrics* 1988;83(Part 2):457–461.
76. Johnson CF. Inflicted injury versus accidental injury. *Pediatr Clin North Am* 1990;37:791–814.
77. Johnson CF, Kaufman KL. The hand as a target organ in child abuse. *Clin Pediatr* 1990;29:66–72.
78. Kaplan JM. Pseudoabuse—the misdiagnosis of child abuse. *J Forens Sci* 1986;31:1420–1428.
79. Kempe CH, Silberman FN, Steele BF, et al. The battered-child syndrome. *JAMA* 1962;181:105–112.
80. King J, Diefendorf D, Apthorp J, et al. Analysis of 429 fractures in 189 battered children. *J Pediatr Orthop* 1988;8:585–589.
81. Kleinman PK, ed. *Diagnostic imaging of child abuse*. Baltimore: Williams & Wilkins, 1987.
82. Kleinman PK, Belanger PL, Karellas A, Spevak MR. Normal metaphyseal radiologic variants not to be confused with findings of infant abuse. *Am J Roentgenol* 1991;156:781–783.
83. Kleinman PK, Blackbourne BD, Marks SC, et al. Radiologic contributions to the investigation and prosecution of cases of fatal infant abuse. *N Engl J Med* 1989;320:507–511.
84. Kleinman PK, Marks SC. Vertebral body fractures in child abuse: radiologic-histopathologic correlates. *Invest Radio*. 1992;27:715–722.
85. Kleinman PK, Marks SC. Relationship of the subperiosteal bone collar to metaphyseal lesions in abused infants. *J Bone Joint Surg Am* 1995;77:1471–1476.
86. Kleinman PK, Marks SC, Adams VI, Blackbourne BD. Factors affecting visualization of posterior rib fractures in abused infants. *Am J Roentgenol*. 1988;150:635–638.
87. Kleinman PK, Marks SC, Blackbourne B. The metaphyseal lesion in abused infants: a radiologic-histopathologic study. *Am J Roentgenol* 1986;146:895–905.
88. Kleinman PK, Marks SC, Richmond JM, Blackbourne BD. Inflicted skeletal injury: a postmortem radiologic—histopathologic study in 31 infants. *Am J Roentgenol*. 1995;165:647–650.
89. Kleinman PK, Marks SC Jr, Spevak MR, et al. Extension of growth-plate cartilage into the metaphysis: a sign of healing fracture in abused infants. *Am J Roentgenol* 1991;156:775–779.
90. Kleinman PK, Marks SC Jr, Spevak MR, Richmond JM. Fractures of the rib head in abused infants. *Radiology* 1992;185:119–123.
91. Kleinman PK, Nimkin K, Spevak MR, et al. Follow-up skeletal surveys in suspected child abuse. *Am J Roentgenol* 1996;167:893–896.
92. Kleinman PK, Spevak MR. Variations in acromial ossification simulating infant abuse in victims of sudden infant death syndrome. *Radiology* 1991;180:185–187.
93. Knight DJ, Bennet GC. Nonaccidental injury in osteogenesis imperfecta: a case report. *J Pediatr Orthop* 1990;10:542–544.
94. Kogutt MS, Swischuk LE, Fagan CJ. Patterns of injury and significance of uncommon fractures in the battered child syndrome. *Radiology* 1974;121:143–149.
95. Kravitz H, Driessen G, Gomberg R, Korach A. Accidental falls from elevated surfaces in infants from birth to one year of age. *Pediatrics* 1969;44:869–876.
96. Krugman RD. Recognition of sexual abuse in children. *Pediatr Rev* 1986;8:25–30.
97. Langlois, NEI, Gresham GA. The age of bruises: a review and study of the color changes with time. *Forensic Sci Int* 1991;50:227–238.
98. Leonidas JC. Skeletal trauma in the child abuse syndrome. *Pediatr Ann* 1983;12:875–881.
99. Leventhal JM, Thomas SA, Rosenfield NS, Markowitz RI. Fractures in young children: distinguishing child abuse from unintentional injuries. *Am J Dis Child* 1993;147:87–92.
100. Lim HK, Smith WL, Sato Y, Choi J; Congenital syphilis mimicking child abuse. *Pediatr Radio*. 1995;25:560–561.
101. Limbos MAP. Documentation of child abuse: How far have we come? *Pediatrics* 1998;102:53–58.
102. Loder RT, Bookout C. Fracture patterns in battered children. *J Orthop Trauma* 1991;5:428–433.
103. Lynch MA. A judicial comment on temporary brittle bone disease [Letter]. *Arch Dis Child* 1995;73:379.
104. Magid N, Glass T. A “hole in a rib” as a sign of child abuse. *Pediatr Radio*. 1990;20:334–336.
105. Markowitz RI, Hubbard AM, Harty MP, et al. Sonography of the knee in normal and abused infants. *Pediatr Radio*. 1993;23:264–267.
106. McClain JL, Clark MA, Sandusky GE. Undiagnosed, untreated acute lymphoblastic leukemia presenting as suspected child abuse. *J Forensic Sci* 1990;35:735–739.
107. McClelland CQ, Heiple KG. Fractures in the first year of life: a diagnostic dilemma? *Am J Dis Child* 1982;136:26–29.
108. McEniery J, Hanson R, Grigor W, Horowitz A. Lung injury resulting from a nonaccidental crush injury to the chest. *Pediatr Emerg Care* 1991;7:166–168.
109. McMahan P, Grossman W, Gaffney M, Stanitski C. Soft-tissue injury as an indication of child abuse. *J Bone Joint Surg Am* 1995;77:1179–1183.
110. Meadow SR. Non-accidental salt poisoning. *Arch Dis Child* 1993;68:448–452.
111. Mehl AL, Coble L, Johnson S. Munchausen syndrome by proxy: a family affair. *Child Abuse Neglect* 1990;14:577–585.
112. Merten DF, Carpenter BLM. Radiologic imaging of inflicted injury in the child abuse syndrome. *Pediatr Clin North Am* 1990;37:815–837.
113. Merter LDF, Radkowski MA, Leonidas JC. The abused child: a radiological reappraisal. *Radiology* 1983;146:377–381.
114. Murphy JM, Bishop SJ, Jellinek MS, Quinn SD. What happens after the care and protection petition? Reabuse in a court sample. *Child Abuse Negl* 1992;16:485–493.
115. Nichols GR II, Corey TS, Davis GJ. Case report: non-fracture-associated fatal fat embolism in a case of child abuse. *J Forensic Sci* 1990;35:493–499.
116. Nimityongskul P, Anderson LD. The likelihood of injuries when children fall out of bed. *J Pediatr Orthop* 1987;7:184–186.
117. Nimkin K, Spevak MR, Kleinman PK. Fractures of the hands and feet in child abuse: imaging and pathological features. *Radiology* 1997;203:233–236.
118. O'Connor JF, Cohen J. Dating fractures. In: Kleinman PK, ed. *Diagnostic imaging of child abuse*. Baltimore: Williams & Wilkins, 1987:103.
119. Ojima K, Matsumoto H, Hayase T, Fukui Y. An autopsy case of osteogenesis imperfecta initially suspected as child abuse. *Forensic Sci Int* 1994;65:97–104.
120. Oliver JE. Intergenerational transmission of child abuse: rates, research, and clinical implications. *Am J Psychiatry* 1993;150:1315–1324.
121. O'Neill JA Jr, Meacham WF, Griffin PP, Sawyer JL. Patterns in injury in battered child syndrome. *J Trauma* 1973;13:332–339.
122. Osier LK, Marks SC Jr, Kleinman PK. Metaphyseal extensions of hypertrophied chondrocytes in abused infants indicate healing fractures. *J Pediatr Orthop* 1993;13:249–254.
123. Patterson CR, Burns J, McAlton SJ. Osteogenesis imperfecta: the distinction from child abuse and the recognition of a variant form. *Am J Med Genet* 1993;45:187–192.
124. Prescott PR. Hair dryer burns in children. *Pediatrics* 1990;86:692–697.
125. Purdue GF, Hunt JL, Prescott PR. Child abuse by burning—an index of suspicion. *J Trauma* 1988;28:221–224.
126. Reece RM. Unusual manifestations of child abuse. *Pediatr Clin North Am* 1990;37:905–912.
127. Renz BM, Sherman R. Abusive scald burns in infants and children: a prospective study. *Am Surgeon*. 1993;59:329–334.
128. Robertson DM, Barbor P. Unusual injury? Recent injury in normal children and children with suspected non-accidental trauma. *Br Med J* 1982;285:1399–1401.
129. Rosenberg DA. Web of deceit: a literature review of Munchausen syndrome by proxy. *Child Abuse Negl* 1987;11:547–563.
130. Rogers LF, Poznanski AK. Imaging of epiphyseal injuries. *Radiology* 1994;191:297–308.
131. Samuels MP, McCloughlin W, Jacobson RR, et al. Fourteen cases of imposed upper airway obstruction. *Arch Dis Child* 1992;67:162–170.
132. Schmitt BD. Child abuse. In: Green M, Haggerty RJ, eds. *Pediatrics*. Philadelphia: WB Saunders, 1984:111.
133. Schmitt BD, Gray JD, Britton HL. Car seat burns in infants: avoiding confusion with inflicted burns. *Pediatrics* 1978;62:607–609.
134. Section on child abuse and neglect. *A guide to references and resources in child abuse and neglect*. Elk Grove Village, Ill.: American Academy of Pediatrics, 1994:107–190.
135. Shoenfeld Y, Fried A, Ehrenfeld NE. Osteogenesis imperfecta: review of the literature with presentation of 29 cases. *Am J Dis Child* 1975;129:679–687.
136. Siegel CD, Graves P, Maloney K, et al. Mortality from intentional and unintentional injury among infants of young mothers in Colorado, 1986 to 1992. *Arch Pediatr Adolesc Med* 1996;150:1077–1083.
137. Sillence D. Osteogenesis imperfecta: an expanding panorama of variants. *Clin Orthop* 1981;159:11–25.
138. Sillence D, Butler B, Latham M, Barlow K. Natural history of blue sclerae in osteogenesis imperfecta. *Am J Med Genet* 1993;45:183–186.
139. Silverman FN. The roentgen manifestations of unrecognized skeletal trauma in infants. *Am J Roentgenol* 1953;69:413–427.
140. Sinal SH. Sexual abuse of children and adolescents. *South Med J* 1994;87:1242–1258.
141. Smects AJ, Robben SGF, Meradji M. Sonographically detected costo-chondral dislocation in an abused child. *Pediatr Radio*. 1990;20:566–567.
142. Smith PB, Poertner J, Fields JD. Preventing child abuse and neglect in Texas. *Texas Med* 1990;86:44–45.

143. Smith FW, Gilday DL, Ash JM, Green MD. Unsuspected costo-vertebral fractures demonstrated by bone scanning in the child abuse syndrome. *Pediatr Radiol* 1980;10:103-106.
144. Sperry K, Pfalzgraf R. Inadvertent clavicular fractures caused by "chiropractic" manipulations in an infant: an unusual form of pseudoabuse. *J Forensic Sci* 1995;35:1211-1216.
145. Sly JR, Starshak RJ. The role of bone scintigraphy in the evaluation of the suspected abused child. *Radiology* 1983;146:369-375.
146. Steiner RD, Pepin M, Beyers PH. Studies of collagen synthesis and structure in the differentiation of child abuse from osteogenesis imperfecta. *J Pediatr* 1996;128:542-547.
147. Strouse PJ, Owings CL. Fractures of the first rib in child abuse. *Radiology* 1995;197:763-765.
148. Sullivan PM, Brookhouser PE, Scanlan JM, et al. Patterns of physical and sexual abuse of communicatively handicapped children. *Ann Otol Rhinol Laryngol* 1991;100:188-427.
149. Sussman SJ. Skin manifestations of the battered-child syndrome. *J Pediatr* 1968;72:99-101.
150. Swischuk LE. Spine and spinal cord trauma in the battered child syndrome. *Radiology* 1969;93:733-738.
151. Texas Department of Health. *Children's health and safety initiative, building blocks for healthy children*. 1996:24.
152. Texas Medical Association Foundation, A Report by the Blue Ribbon Panel on Family Violence.
153. Thomas NH, Robinson L, Evans A, Bullock P. The floppy infant: a new manifestation of nonaccidental injury. *Pediatr Neurosurg* 1995;23:188-191.
154. Thomas SA, Rosenfield NS, Leventhal JM, Markowitz RI. Long-bone fractures in young children: distinguishing accidental injuries from child abuse. *Pediatrics* 1991;88:1-476.
155. Tracy T Jr, O'Connor TP, Weber TR. Battered children with duodenal avulsion and transection. *Am Surgeon* 1993;59:342-345.
156. Touloukian RJ. Abdominal visceral injuries in battered children. *Pediatrics* 1968;42:642-646.
157. Wall J. Re AB (Child Abuse; Expert Evidence.) [1995] 1 FLR 181.
158. Wasserman DR, Leventhal JM. Maltreatment of children born to cocaine-dependent mothers. *Am J Dis Child* 1993;147:1324-1328.
159. Wilson FF. Estimation of age of continuous contusions in child abuse. *Pediatrics* 1977;60:750-752.
160. Wissow LS. Child abuse and neglect. *N Engl J Med* 1995;332:1425-1431.
161. Wolfner GD, Gelles RJ. A profile of violence toward children: a national study. *Child Abuse Negl* 1993;17:197-212.
162. Young WC. Sadistic ritual abuse: an overview in detection and management. *Primary Care* 1993;20:447-458.
163. Zeiss J, Wycliffe ND, Cullen BJ, et al. Radiological case of the month. *Am J Dis Child* 1988;142:1367-1368.
164. Zuravin SJ. Unplanned childbearing and family size: their relationship to child neglect and abuse. *Fam Planning Perspect* 1991;23:155-161.

## FRACTURES AND DISLOCATIONS OF THE HAND AND CARPUS IN CHILDREN

THOMAS J. GRAHAM  
PETER M. WATERS

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### OVERVIEW OF PEDIATRIC HAND INJURIES

For the growing child, the hand is the primary instrument of protection, social interaction, and discovery. Therefore, it is not surprising that it remains one of the most frequently injured parts of the body in children. The recognition and appropriate treatment of these injuries have immediate and long-ranging effects on the child's ability to participate in society.

#### Epidemiology

##### Overall Incidence

Several factors combine to make the pediatric hand vulnerable to injury. Among these are the usage pattern of this relatively exposed appendage and the child's curiosity about the surrounding world. Youngsters are often profoundly ignorant of the dangers in their environment, even at home, where most injuries occur (18,104,234,235). This is why injuries of the hand and wrist are among the most common in the skeletally immature population (88,104,232). They account for up to 25% of fractures recorded in pediatric populations (Table 8-1).

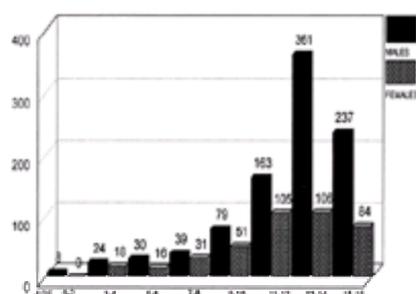
Peak age: 13 years  
 Annual incidence: 26.4 per 10,000 children  
 Percentage of all pediatric emergency patients: 1.7%  
 Right side incidence equals left  
 Male incidence is greater than female incidence  
 Most common areas  
   Nonphyseal: distal phalanx (crush)  
   Physeal: proximal phalanx  
 the index and small fingers are more commonly fractured

TABLE 8-1. INCIDENCE OF PEDIATRIC HAND INJURIES

#### Biphasic Distribution of Pediatric Hand Fractures

Fractures of the hand have a peak incidence in the adolescent age group. The most common mechanism of injury in this age group is from participation in sports. The second peak is in the toddler age group secondary to crush injuries (12,70,104,127,234,235).

The prevalence of these injuries increases sharply after the eighth year (104,234,235). This may be due to the changing balance between injury-resistant cartilage and bone in the maturing hand, and the fact that children participate more aggressively in contact sports around this age. The number of hand fractures in children peaks around age 13, which coincides with adolescents in organized contact sports (Fig. 8-1).



**FIGURE 8-1.** Total number of fractures of the hand, 1970–1975, Malmo, Sweden. (Reprinted from Landin LA. Fracture patterns in children. *Acta Orthop Scand Suppl* 1983;202:54; with permission.)

### **At-Risk Population**

Worlock and Stower (235) conducted a true incidence study of pediatric hand fractures in Nottingham, England, by carefully defining the at-risk population. They projected an annual incidence of 26.4 fractures per 10,000 children. Bhende et al. (18) reported their experience in a busy pediatric emergency department in Pittsburgh, Pennsylvania. A total of 27,294 patients were seen in a 6-month period at their center, of which 464 (1.7%) had a final diagnosis of “hand injury.” Eighty-seven patients sustained 92 fractures. Those sustaining skeletal trauma were significantly older than the rest (11.5 years vs. 8.25 years).

### **Incidence of Specific Fractures**

There is disagreement about which is the most common fracture in the child's hand. Some researchers cite the distal phalanx crush injury as the most frequent (88); others recognize the Salter-Harris (S-H) II fracture of the proximal phalanx base (104,234,235). Many pediatric hand injuries occur about the metacarpophalangeal (MCP) joint (50,214). Dislocations of the pediatric hand are relatively uncommon injuries. The MCP joint is the most commonly dislocated joint in the immature hand (84,130).

Fractures of the phalanges outnumber metacarpal fractures. The right and left hands are equally affected by skeletal trauma. The border digits (index and small fingers) are the most commonly injured rays (18,104,234,235). More boys than girls present with hand fractures.

### **Incidence of Physeal Fractures**

Pathologic forces may be transmitted through the physis in a child's hand, because this often is the path of least resistance. Injuries involving the physis are less common than extraphyseal fractures. The prevalence of physeal injuries in the hand varies from 10% to almost 40% in the reported series (104,127,138,198,234,235). S-H II fractures predominate in all reports. The proximal phalanx is the most commonly injured single bone, involved in about 70% of patients. The small and thumb rays are the most commonly injured (roughly 40% and 30%, respectively) (104).

### **Athletic Injuries**

Second to the home, playing fields and school settings are the most common sites of injuries for children. American football is the predominant contributor to athletic hand injuries. Skiing is well known for causing ulnar collateral ligament (UCL) injuries, or the bony equivalent, at the thumb MCP joint (21,37,76). Pediatric hand injuries have been reported in almost every sport, including boxing, swimming, soccer, and gymnastics (40,76,85,104,138,182,226,240).

### **Practical Anatomy of the Immature Hand**

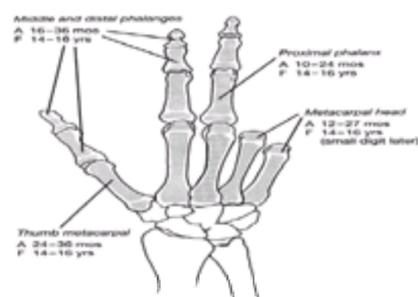
Adults and children have different patterns of hand injury because of different usage patterns and the unique character of the child's skeleton and soft tissues. Knowledge of physeal architecture, soft tissue origins and insertions, and the influence of the strong ligaments and the periosteum is helpful in recognizing and treating children's hand fractures. In this section, the skeletal articular anatomy of the immature hand is described, with special emphasis on structural relationships and their influence on mechanics. In later parts of this chapter, the relevant pathologic anatomy is covered as specific injuries are discussed.

### **Osseous Anatomy**

Phylogenetically, there are potential epiphyses at both the proximal and distal ends of all the tubular hand bones. However, secondary ossification centers develop only at the distal ends of the metacarpals of the index, long, and small rays, and proximally in the thumb. Conversely, the secondary centers of ossification are manifested only at the proximal ends in all digits (95,211).

### **Secondary Ossification Centers**

In boys, the proximal phalangeal secondary ossification center appears at 15 to 24 months and fuses at 16 years (Fig. 8-2) (95,211). In girls, the appearance and fusion occur earlier, at 10 to 15 months and 14 years, respectively. The appearance of the secondary ossification centers of the middle and distal phalanges lags behind by 6 to 8 months, but fusion occurs at the same time as that of the proximal phalanx. The epiphyses of the small finger phalanges appear later than those of the other triphalangeal digits (index, long, and ring fingers).



**FIGURE 8-2.** Appearance of secondary ossification centers (A). Fusion of secondary centers to the primary centers (F).

At the metacarpal level, the secondary ossification centers appear distally in the index, long, ring, and small rays at 18 to 27 months in boys and at 12 to 17 months in girls. The proximally located thumb metacarpal secondary ossification center appears 6 to 12 months after its counterparts in the fingers. These secondary centers fuse at the metacarpal and phalangeal levels at the same time, about 14 to 16 years.

### **Physeal Anatomy**

The microscopic anatomy of the physis and its influence on fracture geometry have been the focus of intense study. The zones of the physis have distinct morphology and molecular/chemical composition that contribute to the unique biomechanics of fracture propagation in the skeletally immature patient (31). Although there is an expanded discussion in Chapter 5, some characteristics of physeal injury specific to the hand are presented here.

The zone of chondrocyte hypertrophy (zone III) is the least resistant to mechanical stresses. It is devoid of the inherent stabilizing properties of collagen maintained in juxtametaphyseal zones I and II or the calcium present in zone IV (88,215). Therefore, the fracture often involves zone III as the path of least resistance.

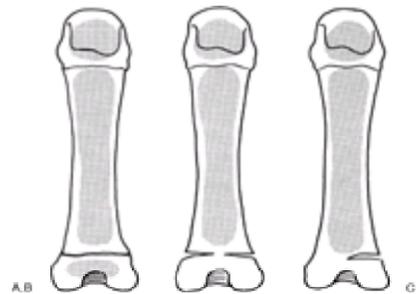
Near skeletal maturity, the irregularity of the physeal zones increases (31). Thus, a fracture line may actually be transmitted through several zones. This variable path through irregular topography may contribute to the phenomenon of partial growth arrest seen with fractures involving the physis.

Furthermore, the pattern of physeal injuries appears to be somewhat age dependent. Intraarticular epiphyseal fractures (S-H III or IV) are more prevalent as the child nears skeletal maturity; S-H I and II fractures that leave the epiphysis intact are more likely to occur in younger patients ( [90,232](#)).

The high level of metabolic activity about the physis is fueled by a rich blood supply that is particularly well suited to resist injury and combat future growth disturbance. The generous perfusion of the physis and epiphysis from both periosteal and endochondral vessels probably contributes to the maintenance of normal growth patterns after fracture. The ability of these vessels to maintain blood supply even after significant displacement of fractures has been consistently demonstrated.

### **Pseudoepiphyses and Double Epiphyses**

A persistent expression of the distal epiphysis of the thumb metacarpal is called a pseudoepiphysis ( [97](#)). The pseudoepiphysis appears earlier than its standard counterparts, then fuses rapidly. By the sixth or seventh year, it has been incorporated and is inconspicuous. Pseudoepiphyses also have been noted at the proximal ends of the finger metacarpals, usually of the index ray. Its only clinical significance is that it must be differentiated from an acute fracture ( [Fig. 8-3](#)).



**FIGURE 8-3.** Abnormal epiphyseal appearance. **A:** Double epiphysis. **B:** Pseudoepiphysis. **C:** Notched epiphysis.

A “double epiphysis” can be seen in any bone of the hand, but these anomalies are more common in the metacarpals of the index finger and thumb. There are variable expressions of the double epiphysis, but the true entity must be considered only when a fully developed growth mechanism is present on both ends of a tubular bone. The double epiphysis must be delineated from the pseudoepiphysis or metaphyseal “notching” ( [57,97,221,233](#)).

The double epiphysis is usually seen in children with other congenital anomalies, but its presence does not appear to influence bone growth appreciably. When fractures occur in bones with a double epiphysis, growth of the involved bone appears to be accelerated ( [233](#)). Periphyseal notching can be confused with trauma or the double epiphysis. The location of the notches can coincide with the physis or may be slightly more distant from the epiphysis. Notching is a benign condition that should not influence the structural properties of the bone to a clinically significant level ( [233](#)).

### **Soft Tissue Anatomy**

The material strength of the child's soft tissues to withstand tensile forces often exceeds that of the adjacent physis and epiphysis ( [156](#)). For this reason, ligament ruptures and tendon avulsions occur less often in children than physeal or epiphyseal fractures.

Knowledge of the relationships between the bony elements and supporting soft tissues in the pediatric hand has been enhanced by cadaver dissections of skeletally immature specimens ( [24,102](#)). The collateral ligaments, volar plate, and mechanically weak extensors provide little protection for the physis, because of the relative paucity of supporting structures aligned to resist these forces. The physes are unprotected by the collateral ligaments, volar plate, and mechanically weak extensors. The flexor tendons provide only a modicum of protection, and also may propagate the fracture through the physis, especially in the terminal phalanx ( [196](#)).

### **Tendons**

As a rule, extensor tendons insert onto epiphyses. The terminal tendon of the digital extensor mechanism and the extensor pollicis longus both insert on the terminal phalangeal epiphyses of their respective rays. The central slip of the extensor digitorum communis terminates onto the dorsal aspect of the epiphysis of the middle phalanx. Likewise, the extensor pollicis brevis inserts onto the epiphysis of the proximal phalanx of the thumb.

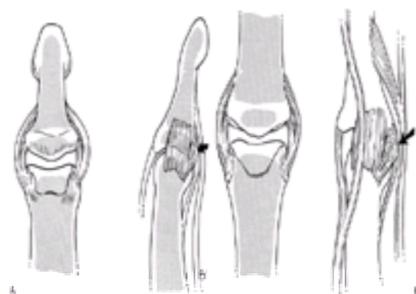
The thumb abductors—the abductor pollicis longus and brevis—have more broad-based insertions onto the epiphysis and metaphysis of the first metacarpal. The multiple slips of the abductor pollicis longus vary in number and have insertions onto the bone, capsule, and fascia of the thenar eminence.

The digital flexor tendons—the flexor digitorum profundus and the flexor pollicis longus—insert at the metadiaphyseal region of their respective terminal phalanges. The flexor digitorum superficialis inserts onto the central three fifths of the middle phalanx.

### **Collateral Ligaments**

#### **Interphalangeal**

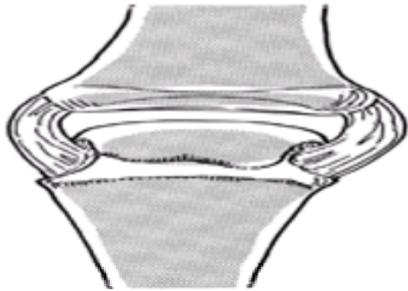
At the interphalangeal joint level, the collateral ligaments originate from the collateral recesses of the phalangeal head, span the physis, and insert onto both the metaphysis and epiphysis of the middle and distal phalanges ( [Fig. 8-4](#)). This accounts for the rarity of lateral S-H III injuries at the interphalangeal joints. The collaterals also insert onto the volar plate, which also spans the interphalangeal joints. This three-sided box protects the physes and epiphyses of the interphalangeal joints from laterally directed forces ( [50](#)).



**FIGURE 8-4.** The anatomy of the collateral ligaments at the distal (**A**) and proximal (**B**) interphalangeal joints. The collateral ligaments at the interphalangeal joints originate in the collateral recesses of the juxtaepiphyseal region and insert into both the metaphyses and epiphyses of their respective middle and distal phalanges. Additional insertion into the volar plane ( *arrows*) is seen at these levels.

## Metacarpophalangeal Joint

At the MCP joint of the fingers, the collateral ligaments originate from the metacarpal epiphysis and insert almost exclusively onto the epiphysis of the proximal phalanx (Fig. 8-5). This anatomic arrangement accounts for the frequency of lateral S-H III injuries at the MCP level. Some fibers may originate from the distal metaphysis of the metacarpal, but this represents only a minor and variable component of the ligament's substance. The ligamentous anatomy about the thumb MCP joint most closely resembles that of the interphalangeal joints because of its growth center arrangement.



**FIGURE 8-5.** The collateral ligaments at the metacarpophalangeal joint both originate and insert almost exclusively on the epiphyseal regions of the metacarpal and the proximal phalanx.

The anatomy of the supporting structures at the MCP level accounts for the high percentage of S-H II and III fractures at this level. The physis is relatively unprotected by soft tissue restraints, and therefore must rely almost solely on the inherent stability of the physis to resist fracture forces.

### **Other Soft Tissues**

#### **Volar Plate**

The volar plate, a stout stabilizer of the interphalangeal joint and MCP joint, is particularly well suited to resist hyperextension forces. Its origin is from the volar metaphysis of the respective proximal digital segment (Fig. 8-4B). The volar plate inserts onto the epiphysis of the distal segment. The plate itself receives insertional fibers from the accessory collateral ligaments, which are best developed at the proximal interphalangeal (PIP) joint.

#### **Periosteum**

Little had been written about the specific role of the periosteum in the stabilization of fractures in the tubular bones of the hand. Because it is so well developed in the child's hand, it can be a significant asset or liability in fracture management. It can minimize displacement of torsional fractures of the diaphysis and may aid in maintaining the stability of other reduced fractures. Conversely, the thick periosteum can become interposed between displaced fracture fragments, thus preventing an effective closed reduction.

## **Evaluation of Pediatric Hand Injuries**

### **Unique Challenges**

Many factors make the injured child uncomfortable, including pain, the novel surroundings, and "white coat" fear. He or she may temporarily refuse to offer either the injured or uninjured hand for examination. Patience and minimizing the contributors to anxiety are essential. The child's hand posture and movements can be observed to gain clues about the location and severity of the injuries by watching the child interact with the environment. In addition to performing a standard, comprehensive examination of the upper extremity, nontraditional methods of assessment may be needed in children, such as an assessment of tendon function by having the child grasp the examiner's pen. It often is helpful to examine the uninjured hand first to familiarize the child with the techniques of examination.

Many factors may be at work to make interaction with the involved adults another obstacle to effective examination. These range from guilt over the injury to the rare child abuse issues. Calming the adults pays dividends: more information can be gained, and a cooperative parent can help the physician communicate with the child.

### **Tissues Are Unique**

#### **Small Structures**

The hands of the child are much smaller than those of the examiner, making precise palpation and some diagnostic maneuvers difficult.

#### **Loose Areolar Tissue**

The loose areolar tissues and generous fat of the child's hand, especially on the dorsal aspect, can hide significant deformity and swelling.

#### **Physiologic Hyperelasticity**

Many children maintain a physiologic hyperelasticity that would be judged pathologic in most adults. However, joint stability and proper tracking of congruous articulations should not be affected by this. Therefore, it is important to conduct a comparison examination of the contralateral side when injury to a stabilizing structure is being considered.

### **P>Clinical Examination**

The examination begins immediately on entering the room, when the child's level of comfort, hand posture, and extremity use pattern has not been altered by examination.

#### **Basic Examination**

The physician should look for swelling, ecchymosis, deformity, and obvious open injuries. The uninjured hand has a normal cascade of semi-flexed digits; deviation from this posture may indicate injury. For examples, digital flexor tendon lacerations or malrotated fractures interrupt the cascade.

Palpation for tenderness should be anatomically specific to increase diagnostic accuracy. Another valuable diagnostic tool is osseous percussion to assess for fractures. However, it is best to defer these potentially painful maneuvers to the later stages of the examination.

#### **Dynamic Evaluation**

**Range of Motion.** Range of motion should be measured and recorded. The integrity of extrinsic flexor and extensor musculotendinous units can be assessed by

observing the excursion of the digits with wrist flexion and extension (the tenodesis effect). The digits should extend with wrist flexion and flex as the wrist is extended.

Persistent limitation of active motion in a child usually means that significant bony or soft tissue injury is present. Stability of each joint in question should be assessed in a systematic fashion. Collateral ligament integrity should be checked by stressing the MCP and interphalangeal joints in both 30 degrees of flexion and full extension. If the amount of deviation resulting from stress on a particular joint is markedly different from the adjacent digit or the same digit on the contralateral side, the stabilizing structures may be injured. Likewise, the quality of the end point determined on stress examination can be an important clue to the degree of tissue disruption.

**Volar Plate Integrity.** Integrity of the volar plate is assessed by hyperextension of the joint. A significant increase in passive extension compared with an adjacent or the contralateral digit usually indicates a volar plate injury that requires immobilization in flexion.

**Stress Testing of a Dislocated Joint.** Stress testing of the relocated joint also is important in determining further treatment. A functional relocation test can be performed to determine the range of motion through which a relocated joint is stable. Active range of motion of the digit is performed by the patient, and the congruence of the reduction can be assessed clinically and radiographically. Motion of the joint should be painless if the anesthetic block has been successful. If the digit is prone to subluxation or redislocation in a narrow range of active motion, the need for operative intervention or long-term immobilization must be considered.

**Neurovascular Injury.** One clue to possible nerve injury is excessive bleeding at the time of the original injury or during evaluation in the emergency department. Because of the proximity of the digital artery to the digital nerve, there is a high concordance between extensive pulsatile bleeding and laceration of the digital nerve.

**Neurologic Evaluation.** It is particularly difficult to determine sensory function in a young child. Meaningful objective data are difficult to obtain, and gross observations, such as withdrawal to stimuli, are inadequate grounds on which to base therapeutic decisions. A helpful examination maneuver to assess nerve integrity is the wrinkle test. Immersion of a normally innervated digit in warm water for up to 5 minutes results in corrugation or wrinkling of the volar skin of the tuft. This finding is absent in a denervated digit. The wrinkle test also can be performed simply by observing the digit as it soaks in the solution as part of the preoperative preparation.

Objective sensibility testing requires patient cooperation and maturity. For example, two-point discrimination is a learned test. Techniques to increase its validity would be performance of repeated trials, testing of multiple uninjured digits, and use of the test only in children over 5 years of age. There is no role for pin-prick or sharp sensation testing in a young child.

**Sedation and Anesthesia.** After nerve function has been adequately assessed and documented, conscious sedation or local anesthesia may be worthwhile to perform reduction maneuvers, assess joint laxity or tendon integrity, explore wounds, and apply splints. If the patient is relaxed and pain free, the opportunity to effect good fracture care is enhanced. The specific protocols for using analgesic agents, conscious sedation, and local or regional anesthesia are discussed in [Chapter 3](#).

### **Radiographic Examination**

#### **Special Considerations**

A careful clinical evaluation is the prerequisite for conducting proper radiographic examination. This is especially true in the multiply injured hand, in which careful localization of areas of tenderness or deformity can direct a thorough radiographic assessment. Several imaging factors can make it difficult to obtain information necessary for a correct diagnosis and effective treatment.

#### **Lack of Osseous Detail**

Knowledge of the age-specific morphology of the hand and wrist bones is essential. In addition, an appreciation of the relationships of the ossific nuclei to each other can allow recognition of physeal fractures or soft tissue injuries that may produce only subtle widening of the interosseous spaces or small angular variations.

#### **Normal Variants**

Irregularities and normal variants are relatively common in the young hand. Imaging of the contralateral, presumably uninjured, side usually is the most effective way to resolve many of the questions that arise in interpretation of the radiographs of a traumatic injury. A familiarity with some of the better recognized variations also helps to determine whether skeletal injury is present. The examiner should not hesitate to consult an atlas of child development and normal radiographic variants ([95,211](#)).

#### **Plain Radiographs**

Anteroposterior (AP), lateral, and oblique radiographs usually are needed to evaluate the injured hand or digit completely. Oblique views are particularly useful in assessing intraarticular fractures. Specialized views for detailing particular anatomic areas can compliment the standard hand and wrist series.

A common pitfall in treating children's finger fractures is failing to obtain a true lateral film of the injured digit. Overlap of the adjacent fingers must be overcome by isolating the digit or splinting the fingers.

Stress views are now rarely used for evaluation of fractures. With the advent of low-radiation mini-fluoroscopy units, real-time assessment of articular congruity and joint stability throughout a motion arc can be conducted.

#### **Differential Diagnosis**

Nontraumatic entities are often interpreted as acute injuries. These entities are rare but may cause swelling, deformity, and limited range of motion.

#### **Congenital**

In 1927, Kirner ([116](#)) described palmar and radial curving of the terminal phalanx of the small digit distal phalanx that occurred spontaneously between 8 and 14 years of age ([Fig. 8-6](#)). This entity can be confused with an acute fracture or epiphyseal separation, but the history, the clubbed appearance of the affected digit, and bilaterality would argue against an acute cause ([63](#)). Trigger thumbs in young children are sometimes mistaken for interphalangeal joint dislocations because of the fixed flexion posture of the interphalangeal joint. Attempted reductions are painful and do not resolve the problem. The key diagnostic feature of the trigger thumb is the palpable nodule at the A1 pulley region.



**FIGURE 8-6. A and B:** A 12-year-old boy presented with incurving of the tip of the right small finger. The anteroposterior and lateral radiographs show radial and palmar incurving of the distal phalanx, characteristic of Kirner's deformity. **C:** Two years later, healing has occurred, but the deformity has persisted.

## Thermal Injury

Thermal injury to the growing hand (frostbite, burns from flame or radiation) can cause bizarre deformities from altered appositional and interstitial bone growth. An ischemic necrosis of the physes and epiphyses can result (Fig. 8-7). The clinical result can be manifested by bone width, length, or angulatory growth disturbance due to the unpredictable effect on the growing elements (98,159).



**FIGURE 8-7.** An 11-year-old girl sustained a frostbite injury to the right hand at age 5 when she lost her mitten. There has been premature fusion of the physis of the middle and proximal phalanges and widening and irregularity of the bases of the shortened phalanges. Although not demonstrated in this photograph, the thumb has been spared, because it is usually enclosed in the hand while the child is exposed to the elements.

## Osteochondrosis (Thiemann's Disease)

Epiphyseal narrowing and fragmentation can be a characteristic of Thiemann's disease, thought to be an osteochondrosis of the phalangeal epiphyses. This hereditary entity usually involves the middle and distal phalanges and typically resolves without aggressive treatment, although some permanent joint deformity has been reported in some patients (52,191).

## Tumors

Benign ganglia of the wrist and hand may cause limited motion, pain, and obvious swelling. Generally, clinical examination of the ganglion of the affected joint or cyst of tendon sheath is diagnostic. Radiographs are typically normal except for the soft tissue changes.

The rare bone, cartilage, or muscle malignancies of the hand have a much firmer consistency to palpation. Radiographs reveal the bony changes in an osteogenic sarcoma and may demonstrate periosteal reaction in an adjacent rhabdomyosarcoma.

## Inflammatory and Infectious Processes

Dactylitis from sickle cell anemia can masquerade as either an infectious or traumatic process. Historical evidence of sickle cell disease in the patient or family and the characteristic fusiform digital swelling indicate this pathology.

The inflammatory arthropathies (juvenile rheumatoid arthritis, scleroderma, systemic lupus) may cause joint effusion or tenosynovitis that can be confused with trauma.

An infectious process often can be mistaken for a traumatic one, but local and systemic evaluation usually makes this distinction evident.

## General Principles of Treatment

### Nonoperative Management

Most children's hand fractures heal without complications. Many do not require reduction, and brief immobilization is sufficient. Minimally displaced fractures without malrotation can be treated with closed reduction and immobilization for 3 to 4 weeks.

### Pain Control

Conscious sedation, careful regional anesthesia, or even general anesthesia should be considered to facilitate reduction and application of immobilization. The choice of anesthetic technique is age dependent. Digital block administered at the level of the metacarpal head frequently is used for fracture reduction in children and adolescents. Axillary blocks, hematoma blocks, and rapid fracture manipulation without anesthesia should be avoided in young children.

### Traction

Skin or skeletal traction has little role in the treatment of children's hand fractures. Application of external devices such as finger traps is poorly tolerated in children. Furthermore, it is difficult to find a size match that permits adequate distraction. Skeletal traction is rarely useful in treating fractures of the hand in a child.

### Gentle and Prompt Manipulation

The likelihood of iatrogenic physal injury substantially increases with repeated, forceful manipulations. Likewise, late (more than 5–7 days) manipulation of a periphyseal fracture should be avoided. Prompt reduction is advisable unless anesthesia is contraindicated. In the acute period, swelling is at its minimum, reduction is more easily performed, and immobilization can be more effectively applied. Swelling itself is not an indication to postpone fracture care; fracture reduction is typically the best way to control swelling and deformity.

### Postreduction Management

#### Types of Immobilization

Immobilization is best applied when the child is under the influence of the sedative or anesthetic used for fracture reduction. An appropriate amount of cast padding material should be applied. Too much padding renders the splint or cast ineffective in controlling the reduction, but too little may lead to skin compromise from thermal injury or pressure. The risk of compartment syndrome may be decreased by the use of splints rather than circumferential casts.

For fractures of the phalanges and metacarpals, immobilization of the injured digit with at least one of the adjacent digits is advocated. The axiom "Never immobilize a single digit" should be followed. Below-elbow (short arm) immobilization is acceptable, provided cooperation is reasonable. For more proximal fractures or fractures in very young children (under age 3–5), long arm immobilization is necessary. Children will attempt to "escape," so long arm immobilization in a "boxing glove" or "mitten" cast is important to prevent loss of immobilization. The use of rigid materials other than accepted casting materials (e.g., tongue blades, aluminum splints,

arm boards) or wrapping the hand and digits around rolls of tape or gauze should be discouraged.

### **The Safe Position**

Like the adult hand, the child's hand should be placed in the correct position for immobilization, the "safe position." Maximal MCP flexion with extension of the interphalangeal joints places the collateral ligaments at their maximal length and helps prevent contracture. However, the likelihood of MCP joint contracture is small in children.

### **Follow-up Frequency**

Nondisplaced fractures that were simply immobilized can be reevaluated at 3 to 4 weeks when the cast or splint is discontinued. Fractures that required reduction or specialized immobilization should be evaluated weekly to ensure that reduction has not been lost. Because children's hand fractures heal rapidly, close follow-up, especially in the first 7 to 14 days, is necessary to detect displacement before it is too late for closed correction.

### **Surgical Management**

#### **Meticulous Soft Tissue Handling**

As in all hand surgery, gentle tissue handling is mandatory. Careful handling of the thick periosteal layer is important, because creation of periosteal flaps, later sutured back anatomically, enhances healing and remodeling. The periosteal layer also provides an excellent cover for implants and is a good sliding surface, allowing smooth tendon excursion.

#### **Preservation of Growth Potential**

Careful manipulation of and around the physis is of obvious importance. Excessive tissue stripping at this level should be avoided. Thoughtful consideration of whether the physis needs to be exposed to effect fracture reduction, and whether it should be crossed by an implant, is warranted. The smallest diameter nonthreaded wire that effectively holds the fragments should be used. Wires are left in only 3 to 4 weeks, so they should be left protruding (but protected) for easy removal.

Implant choices must be individualized to the fracture pattern and to the patient's size. Smooth wires, tension bands, and mini-screws are preferred over larger implants in children. For most fractures requiring fixation, smooth wires are the implant of choice.

#### **Postfracture Care**

Formalized therapy rarely is necessary in children. Simple liberation from immobilization and instructions to the patient and parents regarding range of motion, strengthening, and activity return usually are sufficient. In rare circumstances (complicated fractures, multiple trauma, or failure to achieve desired functional results in an appropriate time period), formal hand therapy may be indicated. If corrective splinting is to be used, the child and caregivers must be comfortable with its application and use. Serial static splinting may be preferred to patient-adjustable dynamic splints.

### **Complications**

The clinician should not be nonchalant about any aspect of the child's treatment simply because the pediatric hand is so forgiving in its ability to remodel and regain motion. Recognition of the potential pitfalls is important, as is the development of a systematic plan for rectifying complications if they occur.

#### **Pitfalls to Avoid**

##### **Failure to Appreciate the Severity of Injury**

A complete set of high-quality radiographs should be obtained; with the surgeon accompanying the child to the radiographic suite if necessary. Contralateral views should be obtained as needed, and normal variants identified.

Specific problem fracture patterns (phalangeal neck, intercondylar, osteochondral "slice," or any malrotated fracture of the tubular bones) need to be recognized to select appropriate treatment.

##### **Inadequate Postreduction Alignment**

Radiographs after reduction must be scrutinized for subtle hints of malalignment or associated injuries. The reduced digits should be checked for malrotation by evaluating the plane of the fingernails with the fingers semiflexed or with the tenodesis maneuvers previously described. Restoration of length, particularly in fractures of the metacarpals, is best evaluated with radiographs, but clinical foreshortening of the ray may be the first clue to malreduction.

##### **Overdependence on Remodeling**

Although remodeling in the immature skeleton is extensive, it is not a substitute for inadequate treatment ([162](#)). Bony remodeling occurs in the AP plane, especially in younger patients and injuries close to the physis. Correction of medial-lateral malalignment is much more limited, except at the MCP and carpometacarpal (CMC) joints, where multiplanar motion exists. Little, if any, rotational remodeling should be performed.

## **SPECIFIC FRACTURES OF THE PEDIATRIC HAND**

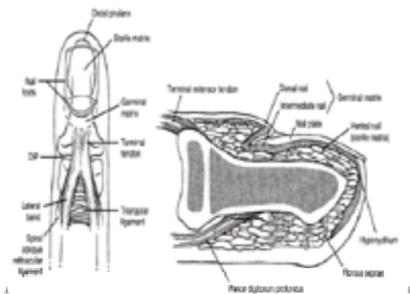
### **Fractures of the Phalanges**

#### ***Injuries of the Distal Phalanx***

The terminal aspect of the digit is among the most frequent sites of injury in children. The distal phalanx and its accompanying soft tissues are relatively unprotected and are offered by the curious child as a first contact with the environment.

#### ***Surgical Anatomy***

The skin, nail elements, soft tissues, and bone of the distal digit are closely related ([Fig. 8-8](#)). The dorsal periosteum of the distal phalanx is the underlying nutritional and structural support for the sterile matrix or ventral nail. The germinal matrix, made up of the dorsal and intermediate nail, is responsible for generating the nail plate. The volar aspect of the distal phalanx anchors the pulp through tough, fibrous septae that stabilize the skin against shear forces. The terminal extensor tendon inserts onto the epiphysis of the distal phalanx. The flexor digitorum profundus bypasses the physis to insert onto the metadiaphysis of the distal phalanx.

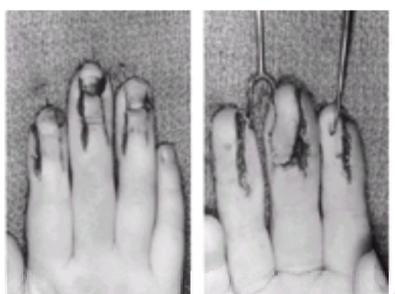


**FIGURE 8-8.** Anatomy about the distal phalanx. **A:** The skin, nail, and extensor apparatus share a close relationship with the bone of the distal phalanx. Specific anatomic structures at the terminal aspect of the digit are labeled. **B:** This lateral view of the nail demonstrates the tendon insertions and the specific anatomy of the specialized nail tissues.

### Mechanism of Injury

The two primary mechanisms of injury about the distal phalanx are crush trauma and hyperflexion force. In a crushed fingertip, the soft tissues are injured and the bone is fractured. Conversely, if violent forced flexion is exerted on the extended distal phalanx, there is a greater potential for bony injury of the dorsal epiphysis.

The spectrum of injury produced by both these mechanisms varies. Many crush injuries result only in minor tissue disruption and need little or no intervention ( [Fig. 8-9](#)), whereas other injuries may require bony fixation, meticulous nail bed repair, and skin coverage.



**FIGURE 8-9. A and B:** Crush injury to the fingers of a 4-year-old in which the lacerations were closed by primary repair; only minimal nail bed repair was necessary. Of concern is the depth of the laceration about the germinal matrix tissue. Meticulous repair must be undertaken in this area, as well as at the sterile matrix level.

Mallet injuries result from an axial loading or flexion force applied to the extended tip of the finger. Although the underlying pathology can be variable, the digit usually is flexed at the distal interphalangeal (DIP) joint and active extension is impossible.

### Fracture Patterns

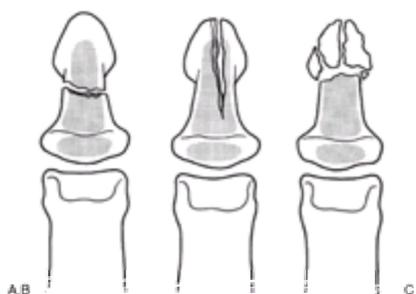
Fractures of the distal phalanx can be divided into those that are extraphyseal and those that are physeal ( [Table 8-2](#)).

- Extraphyseal (see Fig. 8-10)
  - Transverse diaphysis
  - Longitudinal splitting
  - Comminuted separations
- Physeal
  - Dorsal mallet injuries (see Fig. 8-13)
    - Salter-Harris I or II
    - Salter-Harris III or IV
    - Salter-Harris I or II joint dislocation
    - Avulsion of extensor + Salter-Harris fracture
  - Reverse mallet injuries (see Fig. 8-14)
    - Avulsion of flexor with bone (jersey finger)

**TABLE 8-2. CLASSIFICATION OF FRACTURES OF THE DISTAL PHALANX**

### Extraphyseal Fractures

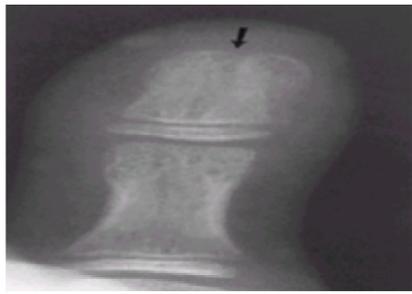
Extraphyseal fractures of the distal phalanx are common and can take the form of a simple distal tuft fracture or an unstable diaphyseal fracture underlying a nail bed laceration. They can be divided into three types ( [Fig. 8-10](#)).



**FIGURE 8-10.** Three types of extraepiphyseal fractures of the distal phalanx. **A:** Transverse diaphyseal fracture. **B:** Cloven-hoof longitudinal splitting fracture. **C:** Comminuted distal tuft fracture with radial fracture lines.

A transverse fracture ([Fig. 8-10A](#)) may occur either at the distal extent of the terminal phalanx or through the diaphysis. Distal fractures are separations between the diaphysis and the most distal extent of the phalanx and also can include a series of radial fracture lines propagating through the distal tuft. Transverse fractures through the diaphysis are almost always associated with a significant nail bed injury.

A longitudinal splitting of the nail tissues and bone ([Fig. 8-10B](#)) is much less common. This pattern is the result of excessive hoop stress when the tubular distal phalanx is subjected to crushing forces. The “cloven-hoof” appearance of the fracture is characteristic ([Fig. 8-11](#)). The fracture can be completely contained within the shaft or can continue to be propagated through the physis and even into the joint ([12](#)).



**FIGURE 8-11.** Extraepiphyseal fracture of the distal phalanx, the cloven-hoof longitudinal splitting fracture. In this patient, the fracture line ( *arrow*) does not appear to extend across the physis.

Comminuted fractures take the form of semilunar separations at the distal tuft, or multifragment fractures of the distal diaphysis ([Fig. 8-10C](#) and [Fig. 8-12](#)). Usually sustained as a result of a crush injury, these fractures are often accompanied by significant injury to surrounding soft tissues of the distal tip.



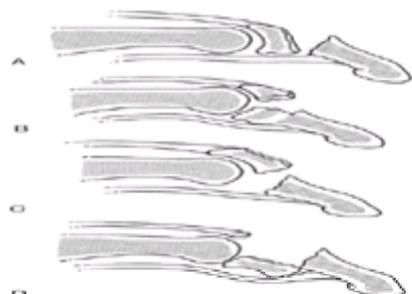
**FIGURE 8-12.** Extraepiphyseal distal phalanx fracture. The fracture line is slightly distal to the insertion of the flexor digitorum profundus tendon. The accompanying soft tissue disruption (*arrow*) can be appreciated on the radiograph.

A rare but clinically important fracture, sometimes called a reverse mallet injury, can occur in the distal phalanx. This is an avulsion fracture of the volar metaphysis caused by traction on the flexor digitorum profundus tendon. These fractures occasionally extend into the physis. The DIP joint usually is held in neutral or slight dorsiflexion.

### **Physeal Fractures**

The clinical appearance of a physeal fracture resembles that of an adult mallet finger. Four basic fracture patterns involve the physis, and all result in the characteristic flexed posture of the DIP joint.

Fractures often cause the flexed posture of a mallet finger in the child. However, a “typical” mallet injury, avulsion or laceration of the terminal tendon at the DIP or distal phalanx level, can occur in a patient of any age. The mallet equivalent fractures are of four types ([Fig. 8-13](#)). An open S-H I or II fracture with flexion of the distal fragment occurs predominantly in young patients (under 12 years of age). The characteristic flexed posture is adopted because of the unopposed flexor digitorum profundus force acting on the distal fragment. Because this fracture is almost always accompanied by nail bed laceration, there is a high risk for infolding or incarceration of the germinal or sterile matrix in the fracture site. This constellation of clinical findings has been labeled a Seymour fracture ([195](#)).



**FIGURE 8-13.** The mallet-equivalent fractures.

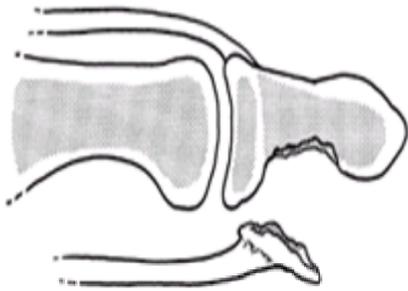
In teenagers, a true bony mallet can occur with a displaced S-H III or IV fracture. Instead of an avulsion of the terminal tendon from its distal phalanx insertion, a fracture results in inability to actively extend the DIP joint.

Rarely, an S-H I or II fracture causes extrusion of the epiphyseal fragment ([146](#)). This “epiphyseal dislocation” is clinically challenging to diagnose because the distal fragment can remain relatively colinear with the axis of the digit, whereas the displaced epiphysis is dorsally dislocated by the traction of the extensor mechanism.

Even more rarely, the epiphysis separates or is fractured simultaneously, with an avulsion of the terminal extensor from the fragment. This variant may leave the DIP joint unreduced, or the free epiphysis may settle into a reasonable articular relationship at the DIP joint ([190](#)). These injuries may be unrecognized initially when they occur before the secondary ossification center appears.

## Jersey Finger

This rare injury causes an inability to actively flex the DIP joint. Forced extension of the flexed DIP joint can result in either a bony injury or a soft tissue disruption involving the insertion of the flexor digitorum profundus ( [Fig. 8-14](#) ) ( [125,227](#) ). If a fracture occurs at the tendon insertion, the flexor digitorum profundus is sometimes prevented from retraction by tethering on the A5 or A4 pulley. The location of the bony fragment on radiography will identify the level of tendon retraction. Soft tissue avulsion of the flexor digitorum profundus usually retracts to the level of the palm.

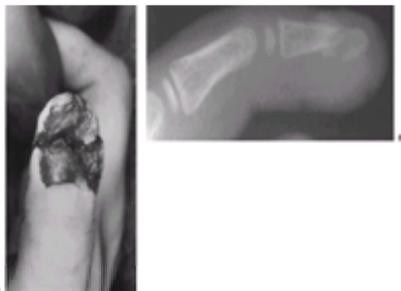


**FIGURE 8-14.** A flexor digitorum profundus avulsion fracture of the distal phalanx, the jersey finger.

## Diagnosis

### **Signs and Symptoms.**

*Status of the Nail and Nail Bed.* Because almost half of patients with nail bed injuries seen in an emergency department have a concomitant fracture of the distal phalanx, a high index of suspicion for bony injury must be maintained when an obvious open nail bed injury or a subungual hematoma is present ( [Fig. 8-15](#) ) ( [241](#) ). A subungual hematoma that involves more than 50% of the nail plate often indicates a distal phalangeal fracture.



**FIGURE 8-15. A:** A crush injury to the thumb of a 4-year-old with a stellate nail bed laceration and fracture of the tuft. **B:** Avulsion of the nail plate after crushing trauma is accompanied by a distal phalanx fracture. This must be treated as an open fracture.

Occasionally, smaller matrix lacerations can bleed enough to cause pain simply from the pressure developed under the nail plate. More often, the nail plate is lifted from under the folds, which may decompress any underlying hematoma. Rarely, the nail is completely avulsed without an accompanying soft tissue injury to the sterile or germinal matrix.

*Tissue Loss.* The distal aspect of the fingers and thumb are at risk for significant trauma. Partial or complete terminal amputations occur in patients of all ages. Most of these injuries are incomplete amputations, through a level that includes variable amounts of the specialized nail tissues. Complete amputations at the distal phalangeal level generally are distal to the physis.

**Radiographic Findings.** A thorough series of films focusing on the distal phalanx and DIP joint is necessary to evaluate the injury completely. Specialized imaging, such as computed tomography (CT) scanning or magnetic resonance imaging (MRI), is not needed to evaluate this region.

### **Treatment Options**

The fractures associated with nail bed lacerations are true open injuries and require attention to both the osseous and soft tissue elements. Any open fracture requires mechanical debridement, irrigation, and appropriate antibiotics. Most of these injuries occur in children under 5 years of age and do not require treatment beyond initial repair. The stability of the phalangeal fracture is evaluated, and if the phalanx is not stable enough to act as a foundation for nail bed repair, pinning is performed.

The repair or reconstruction of the soft tissues surrounding the distal phalanx is just as critical as the osseous repair. When indicated, meticulous nail bed repair, neuroorrhaphy, tenorrhaphy, and skin coverage must be performed.

### **Extraphyseal Fractures.**

*Stabilization.* Options for stabilizing extraphyseal fractures include closed splinting, percutaneous fixation, or open reduction. Most fractures are stable, and require only simple splinting. However, a smooth Kirschner wire may be inserted either into the phalanx from the tip or occasionally across the DIP joint in fractures with marked comminution or very proximal fractures. A hypodermic needle is a good substitute for the standard smooth wires ( [145](#) ).

*Hematoma Evacuation.* If a hematoma is the only outward manifestation of underlying injury, observation or simple evacuation may be sufficient. Indications for a hematoma evacuation include significant subungual hematoma (>50% of the nail plate), painful pressure under the nail, or evidence of infection. The decompression can be performed with a hypodermic needle used as a drill. A heated paper clip or cautery tip can be effective, but a margin of safety is necessary to avoid further tissue injury. DaCruz et al. ( [53](#) ) reported a high incidence of late nail deformity if the hematoma was not decompressed.

*Nail Bed Repair.* If nail bed repair is required, the nail is removed with a blunt septal or Freer elevator. Partial nail removal is seldom appropriate, because the extent of the injury may not be appreciated. Further exposure of the proximal nail tissue (germinal matrix or dorsal/intermediate nail) by incisions in the eponychial fold may be necessary. The nail bed is repaired with interrupted absorbable 6-0 or 7-0 sutures under loupe magnification. The likelihood of nail deformity is minimized by this surgical intervention.

After careful approximation, the nail bed is stented to avoid scarring that could hinder subsequent nail growth. If the nail has not been too badly damaged, it can be used to stent the nail folds after it has been denuded of all soft tissue remnants and has been perforated centrally to allow free drainage of subungual fluids. Other

substitutes, such as a plastic stent fashioned from a culturette tube, a foil suture pack, or a commercially available stent, also can be used. The use of the nail itself or some inert foreign material has been controversial ([68,189,241](#)). The nail itself is thought to be more stable and may assist in “molding” the repair, but the risk of infection must be considered if the preparation before insertion is inadequate. Sandzen and Oakey ([184](#)) argued that this risk is lower in children than in adults. Suturing of the replaced nail may help prevent migration from underneath the nail folds. Anchoring the stent with a suture has been described, but this may cause incidental injury to the sterile or germinal matrices.

### Physeal Fractures.

*Nonoperative Management.* Nonoperative treatment of a mallet finger in a child, as in an adult, is overwhelmingly favored. When the injury is closed, there is seldom an indication for operative intervention in a typical bony mallet. Even when there is some displacement of the dorsal epiphyseal fragment, good results can be obtained with splinting ([Fig. 8-16](#)).



**FIGURE 8-16. A:** A very unstable fracture that necessitated extrication of the nail bed with subsequent repair. **B:** Stabilization of the bony elements with a longitudinal smooth pin.

Amputation at the distal tip can involve skin, nail tissue, and bone. These injuries are often worrisome to parents of a young child because of the potential loss of digital length. However, it is important to remember that the growth for the distal phalanx comes from the physis that often is proximal to the level of the amputation, and long-term digital shortening is rare.

Support for nail growth is a primary consideration. When less than 50% to 60% of the distal phalanx remains, the likelihood of nail deformity in the form of a hooked or “parrot’s beak” nail is high. Often, lesser injuries can be treated with simple dressing changes or allowed to heal by secondary intention. Skin grafting at the distal tip often is unsatisfying because the durability, sensibility, and appearance are suboptimal.

If most of the distal phalanx is spared, only a relatively transverse nail bed and soft tissue laceration exists. Simply allowing the tissue to heal, with or without a small amount of bony resection, is an option. In addition, results are often surprisingly good with healing by secondary intention over a small amount of exposed bone.

The spectrum of injury can range from a minimal nail bed laceration, manifesting as a minor subungual hematoma, to the significant laceration and unstable periphyseal injury ([Fig. 8-17](#)). Campbell ([36](#)) advocates an aggressive approach for open bony mallet fingers, even with minimally displaced fractures. Late complications such as tendon imbalance, nail growth or adherence problems, premature physeal closure, and even chronic osteomyelitis can occur with relatively innocuous trauma.

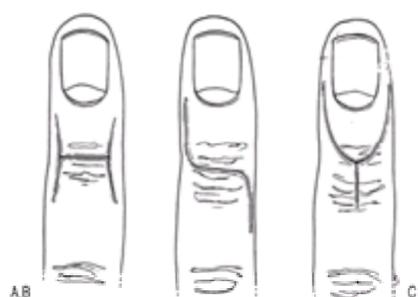


**FIGURE 8-17. A:** Although mallet fractures of this pattern and amount of displacement often can be treated by closed methods, the extreme deformity and dorsal prominence was of concern to the patient and his parents. It was therefore elected to open the fracture to reduce and stabilize the fragment. **B:** This intraoperative photograph demonstrates the amount of articular surface involved and the attachment of the terminal tendon.

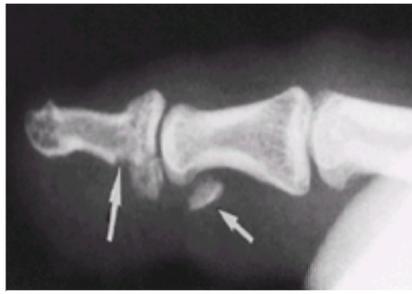
*Operative Management.* When an S-H IV bony mallet fracture is displaced and adequate reduction cannot be obtained by closed means, operative stabilization should be considered. Although most of these fractures can be treated closed, a large dorsal fragment (typically >50%) or significant DIP joint subluxation may require operative intervention ([50,198](#)). Closed manipulation and percutaneous pin stabilization of the reduced joint are generally used.

The Seymour fracture is an absolute indication for open reduction and stabilization. The sterile matrix must be extricated from the fracture at the physeal level. Not until the soft tissue is removed will the fracture reduce appropriately. Those rare fractures with epiphyseal dislocation require operative intervention to restore joint congruity and to reestablish continuity of the extensor apparatus.

*Surgical Procedures.* When a complex injury cannot be reduced or stabilized using a closed technique, a dorsal approach is necessary to expose the fragment and reduce the fracture ([Fig. 8-18](#)). It may be necessary to take down the collateral ligament to obtain reduction. Although we prefer to avoid it, fixation of the fragment itself is advocated by some clinicians ([88,92,104,117,162,175](#)). When the epiphysis is completely displaced from its articular contact and its relationship with the remaining distal phalanx, open reduction through a dorsal approach is necessary. Several different approaches for exposure of the DIP joint have been described, primarily for use in the treatment of arthrosis and mucous cyst excision ([48,65](#)), for example, an H- or S-shaped incision or an extended Y flap ([Fig. 8-19](#)).



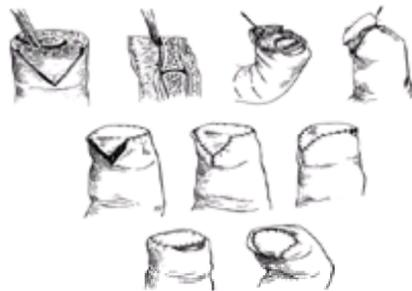
**FIGURE 8-18.** Three exposures of the distal interphalangeal joint. **A:** H-type flap with the transverse limb over the distal interphalangeal joint. **B:** S-shaped exposure of the distal interphalangeal joint. **C:** An extended Y exposure of the DIP joint; the apex of the V part of the incision is at the distal interphalangeal joint. In all these exposures, care is taken not to injure the germinal matrix or its blood supply, which is located in the sulcus just proximal to the nail fold.



**FIGURE 8-19.** In this volar fracture in a 17-year-old athlete, a fracture through the epiphysis extends into the joint ( *large arrow*). The flexor digitorum profundus with an attached fragment of the metadiaphysis has retracted to the level of the A4 pulley ( *small arrow*).

Once the joint is exposed and the fragment with its attached terminal tendon is retrieved (or the free fragment and avulsed tendon), repair can be performed with a smooth wire, pullout wire, tension band, or heavy suture (99). Pinning of the DIP joint is necessary to maintain joint and physeal congruity.

A jersey finger avulsion of the flexor digitorum profundus requires open repair. The profundus tendon needs to be identified surgically at the level of retraction. This may be at the A2 or A4 pulley of the flexor tendon sheath (Fig. 8-20), or it may have retracted to the lumbrical origin off the flexor digitorum profundus in the palm. If the vincular blood supply and synovial bathing from the fibroosseous sheath are both lost, the repair should be immediate. Ideally, a primary repair under little or no tension should be performed within a week. Treatment of chronic flexor digitorum profundus avulsions may include no repair, flexor digitorum profundus tenodesis, DIP joint fusion, and delayed repair.



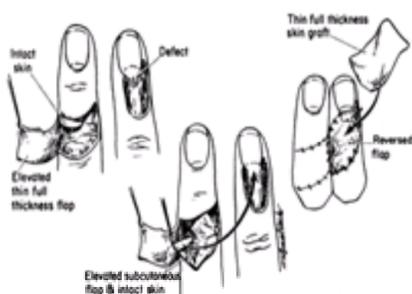
**FIGURE 8-20.** The reversed cross-finger subcutaneous flap. A full-thickness skin flap is elevated from the donor digit based on the side opposite the injured digit. The subcutaneous tissues are then elevated from the epitenon, based on the side of the injured digit. The subcutaneous tissues are reflected in a reversed fashion into the defect, and a thin full-thickness skin graft is applied to the recipient. The full-thickness skin flap from the donor is then replaced over the vascular epitenon. Division of the flap can take place at 2 weeks. (Reprinted from Atasoy E. Reversed cross-finger subcutaneous flap. *J Hand Surg* 1982;7:481–483; with permission.)

**Soft Tissue Coverage of Amputations.** Soft tissue coverage over the dorsal aspect of the distal phalanx and reconstruction of the nail folds are among the most difficult challenges in hand surgery. The surgical approach for the dorsal injury must be tissue specific. Simple healing by primary closure is preferred. In the rare complete avulsion of the nail bed with exposed distal phalanx dorsally in a young child (under 5 years), the amputated nail bed and pulp can be replaced with minimal defatting. In addition to simple nail bed repair or nonvascular replacement of the amputated part, composite grafts of skin and subcutaneous tissue from local or distant sites (e.g., the adjacent digits, the toes) have been used (42,196,242).

The simplicity and predictability of bony recession, traction neurectomy, and primary closure must not be forgotten in the midst of more elaborate techniques. A well-performed primary closure of a traumatic amputation can hasten a return to high-level function with minimal discomfort and disability. Ablation of the germinal matrix, when indicated for significant nail loss, and neurectomy minimize the need for future revision surgeries. Of course, amputation in a child's hand should be reserved for only the most significant tissue disturbances that are not reconstructable in other ways.

**Reconstruction Options.** Options for reconstructing the distal tip injury include V-Y volar advancement, a thenar flap, a cross-finger flap, a pedicled flap, and a neurovascular island flap.

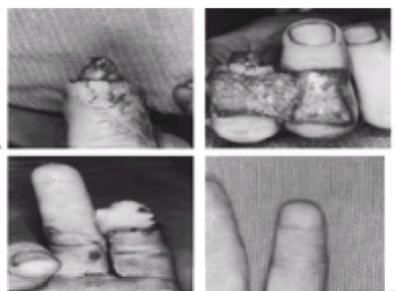
The triangular V-Y volar advancement flap was described in 1970 by Atasoy et al. (8). Adequate skin on the volar and lateral sides is needed. The flap is designed so that the apex of the triangle proximally is at the DIP crease, and the base of the triangle is the amputation margin. The triangle of skin is liberated carefully with sharp dissection of the fibrous septae that tether it proximally, thus allowing it to advance distally to cover the defect. The void left behind is closed, converting the V into a Y (Fig. 8-21).



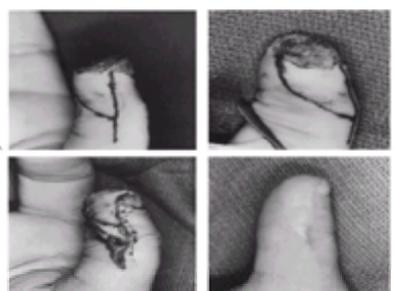
**FIGURE 8-21.** V advancement flap. This triangular volar flap is elevated to cover a distal defect in the author's original description. A V is formed with its apex at the distal interphalangeal joint flexion crease. The base of the triangular flap is the margin of the amputation. The fiber septae are elevated from the distal phalanx. Care is taken to advance the volar skin by sequential division of small fibrous tethers to the volar side. When adequate immobilization of the flap is realized, it can be sutured in place over the tip. (Reprinted from Atasoy E, Ioakimidis E, Kasdan ML, et al. Reconstruction of the amputated finger tip with a triangular volar flap. *J Bone Joint Surg [Am]* 1970;52:921–926; with permission.)

The thenar flap was first described by Gatewood in 1926 (78). This innovative procedure buries the amputated tip into the tissue about the thenar eminence. The disadvantage of this flap is the extreme flexion that must be maintained for the duration of the attachment. Other clinicians (145) have described flexion contractures with this flap, and they have suggested that the candidates for this seldom-needed procedure be young. I also advocate the thenar flap only in patients under 20 years of age. If it is selected for coverage, the U- or H-shaped flap of skin and subcutaneous tissue must be located as close to the thumb MCP flexion crease as possible to minimize PIP joint flexion.

The cross-finger flap is one of the most versatile reconstructive procedures used about the distal tip (113). If tissue is inadequate for primary closure or local advancement, a cross-finger flap permits coverage without the need for a distant flap (e.g., groin, cross-chest). This flap requires a nontraumatized adjacent donor. Like the reverse cross-finger flap (Fig. 8-22), the donor flap is based on the side of the injured digit, but instead of using only the subcutaneous fat as a flap applied to the dorsum, the cross-finger flap brings skin and subcutaneous tissue to the volar side (Fig. 8-23).

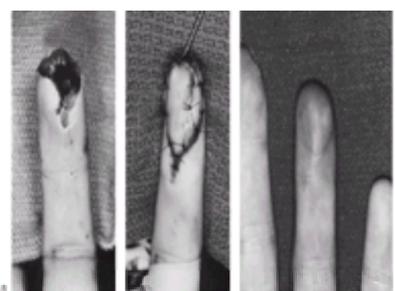


**FIGURE 8-22.** The cross-finger flap. This 17-year-old boy was working on his car when his distal tip was caught in the area of the fanbelt. **A:** Extensive volar and distal loss was appreciated, but there is only minimal injury to the bony elements and laceration of the nail bed. The patient and parents were strongly against amputation and therefore were offered the option of a cross-finger flap. The most proximal level of injury is at the distal interphalangeal joint flexion crease. **B:** A flap of skin and subcutaneous tissue is elevated from the dorsal aspect of the adjacent donor digit based on the side of the index finger. The vascular epitenon is left behind to nourish the donor digit and support a graft. The flap is then brought to the volar aspect and sewn into the recipient. Care is taken to contour the flap to recapitulate the appearance of a normal digital tip. **C:** An excellent functional and cosmetic result was realized in this case. **D:** More than 50% of the nail was supported by the distal phalanx, and this can be maintained by performing good nail hygiene to avoid any type of hooked-nail deformity.



**FIGURE 8-23.** A pedicle neurovascular island flap for distal thumb coverage. **A and B:** This 16-year-old guitarist sustained a sharp distal tip amputation of the thumb. Because of the importance of this digit to his musical abilities, he strongly wanted this to be resurfaced with a durable, sensible, and contoured flap. A neurovascular island flap was based on the radial digital nerve and large accompanying vessel. The flap was designed in a V-Y fashion. **C:** After the flap was mobilized, it was brought up to cover the distal tip, and care was taken to contour the flap. **D:** Excellent contour of the distal thumb was realized with intact sensibility. The patient was able to play the guitar without decrement in his skills. (Courtesy of Richard S. Idler, M.D.)

A pedicled neurovascular island flap is an appropriate alternative when durable sensible tissue is needed. This flap can be designed and advanced on a local neurovascular bundle or transferred from a remote location, and it is particularly useful in the thumb for unilateral volar tissue loss (Fig. 8-24).



**FIGURE 8-24.** Volar V-Y advancement flap for coverage of volar tissue loss. **A:** A volar oblique tissue loss is a particularly good indication for an advancement flap when there is adequate tissue remaining between the distal interphalangeal joint crease and the level of the injury. In this case, the nail tissues were uninjured. **B:** The flap is fashioned with its apex at the distal interphalangeal joint and mobilized to cover the tip. The defect is closed behind the flap, thus creating the Y. **C:** The result is a very cosmetic-appearing digit that has good durability and sensibility.

The more autonomous volar and dorsal blood supply of the thumb is the basis of Moberg's volar advancement flap for coverage of tissue loss at the distal thumb (Fig. 8-25) (155). Bilateral mid-lateral incisions permit advancement of the volar skin to cover an acral defect. Coverage is made easier by flexing the interphalangeal joint of the thumb. Transverse division of the more proximal skin can sometimes be performed, with skin grafting into the defect. In a child, the skin usually is more pliable and permits primary closure.



**FIGURE 8-25.** A thenar flap was chosen in this case. An H-flap technique was used. Another common technique is use of a composite graft from the toe pulp.

## AUTHORS' PREFERRED METHOD OF TREATING DISTAL PHALANGEAL INJURIES

### Extraphyseal Fractures

For distal phalangeal fractures that do not involve the physis, the decision concerning treatment is based on the stability of the fracture and the status of the nail bed. Most simple closed fractures are treated with immobilization. In a young child, this may involve a mitten cast; in an adolescent, splinting of the tip and DIP joint, leaving the PIP free for motion, may be all that is necessary. Gentle manipulation of some fractures may improve slight angulation. Immobilization is worn for 3 to 4 weeks at most, until clinical union has brought comfort. Protection during contact activity can be continued if the digit is at risk.

For fractures accompanied by a nail bed laceration, adequate local anesthesia (a metacarpal block) is instilled with or without conscious sedation. A Penrose drain is inserted at the base of the digit, the nail plate is completely removed with a Freer elevator, and meticulous mechanical debridement and copious irrigation are performed. In the rare unstable distal phalangeal bony injury, the fracture is pinned.

Pinning of the phalanx is best performed with image intensification with a low-radiation fluoroscopy unit. We use 0.028- or 0.035-inch smooth wire, depending on the patient's size. A 22-gauge hypodermic needle can be used if other resources are unavailable. The wire or needle is left protruding from the distal end of the phalanx, to make later removal easier. The DIP joint is crossed if sufficient stability is not achieved by pinning the fragments of the terminal phalanx, but this is avoided if possible. The pin is maintained for about 3 to 4 weeks.

With a stable platform established, sterile germinal matrix repair proceeds. Using 6-0 or 7-0 absorbable on a spatulated needle minimizes damage to the friable tissues of the nail bed. The injured nail is discarded, and petroleum- or antibiotic-impregnated gauze is placed under the nail folds. This covering of the repaired matrix simply falls off in several weeks.

A bandage that protects the distal phalanx usually is all that is necessary after nail bed repair. Leaving the PIP free is appropriate in older, cooperative patients, but younger patients may need immobilization up to and including a long arm dressing for this very distal injury. The parents and patient are told that it may take several cycles of nail growth (3–6 months) before the final morphology of the nail is known.

### Physeal Fractures

#### *Dorsal Mallet Injuries*

We attempt to reduce the fracture in almost all closed mallet injuries. The reduction maneuver recreates the deformity by gentle flexion. The distal fragment is then extended to restore the anatomy. Reduction is held while the splint is carefully applied. We apply a splint (either volar or dorsal) then obtain radiographs to evaluate the reduction in the immobilized state ([Fig. 8-26](#)).

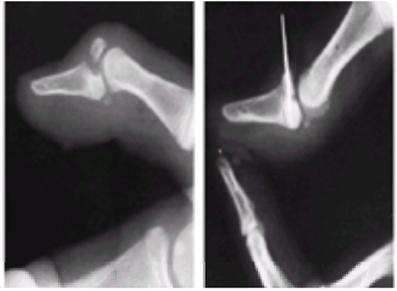


**FIGURE 8-26. A:** A 13-year-old boy sustained an open type II fracture when a fellow football player stepped on his hand. **B:** After the wound was cleansed, a closed reduction was performed, preserving the nail. Normal function resulted.

Our normal splinting regimen is 24-hour wear for no less than 4 weeks, and typically for 6 weeks. This time frame can be adjusted depending on the amount of bony apposition and age of the patient. A cooperative patient can be furnished with both volar and dorsal splints, if it is understood that any flexion of the DIP during changing of the splint will prolong the immobilization period. The two-splint regimen has almost eliminated the infrequent skin problems seen with dorsal splinting alone and the transient sensory disturbance with prolonged volar protection. The finger is held in neutral to 15 degrees of extension; extreme hyperextension is contraindicated, because it may cause skin hypoperfusion and necrosis ([178](#)). Loss of flexion can occur with prolonged hyperextension but is rare with closed treatment.

The patient and parent should check the skin at least daily to prevent skin necrosis from splint pressure. A follow-up visit at 5 to 7 days will reveal problems if the patient does not understand the instructions. Radiographs are taken weekly for the first 2 weeks, then every 2 weeks to monitor for loss of reduction and volar subluxation ([188](#)).

The presence or absence of an open injury and the fracture configuration dictate the need for operative intervention. If unacceptable reduction of the fracture fragments or subluxation or dislocation of the DIP joint remains after closed manipulation, then surgery is performed to reduce the fragment and DIP joint. We prefer to perform this percutaneously. The dorsal fragment can be pinned with a 0.028-inch smooth Kirschner wire driven into the epiphysis in a parallel direction ([Fig. 8-27](#)). The wire is left protruding from the skin for later removal. Pinning of the DIP joint also often is necessary. If this is not feasible, a dorsal approach is used to expose the dorsal fragment in these mallet equivalent injuries ([Fig. 8-19](#)). Care is taken to avoid the vessels that nourish the germinal matrix, which are located in the shallow sulcus proximal to the nail fold. This area can be appreciated by viewing the distal digit from the lateral side. This averts interruption of tissue perfusion and prevents the major complication of hematoma.



**FIGURE 8-27. A:** A 14-year-old boy sustained a displaced type III fracture of the distal phalanx when he was struck on the end of the thumb with a ball. **B:** Open reduction and Kirschner wire fixation were required when manipulation failed to reduce the fracture because of the displacement caused by the action of the extensor pollicis longus tendon on the fracture fragment.

If the patient is near skeletal maturity, a tension band, pullout wire, or suture anchor can be used for fixation. Although a neutral position or slight (10 degrees or more) hyperextension is preferred for closed treatment, the joint should not be repaired open in hyperextension because this position tends to persist. Neutral alignment of the DIP joint is favored if the fracture is open.

### **Reverse Mallet Injuries**

For reverse mallet, flexor digitorum profundus avulsion injuries, we prefer bone-to-bone fixation whenever possible. If the fragment is too small or comminuted, repair of the tendon to the fracture bed is adequate. Pinning of the joint is avoided so that passive motion can be started early. In patients under 15 years of age, 4 weeks of immobilization yields results equivalent to those of protected motion protocols in adults.

### **Soft Tissue Management**

For terminal loss of skin and subcutaneous tissue only, we again stress the necessity of wound cleansing and dressing changes to all injuries left to heal by secondary intention. This method of treatment is not benign neglect, but rather a reasonable approach that usually results in superior functional and cosmetic results. Skin grafts from the groin, antecubital fossa, or hypothenar glabrous border rarely are used for coverage. Donor morbidity, hyperpigmentation, lack of sensitivity, and appearance are potential drawbacks of skin-grafting procedures.

Dorsal loss is the most difficult to reconstruct. We attempt tension-free closure first, then consider local tissue relaxation and advancement before opting for a reverse cross-finger flap or distant flap. Distal tip and volar loss on the remaining bone, the status of the nail tissues, and the availability of sufficient volar skin distal to the DIP flexion crease must be considered. We have found the volar V-Y advancement, cross-finger flap, and thenar flaps to be good options.

We have not detailed microsurgical reconstruction methods or many of the innovative but arduous flaps reported frequently in the literature. Instead, we have provided a basic list of options that will suffice in most patients. Knowledge of the indications for and the technical aspects of these procedures permits the surgeon to approach most distal tip reconstructive challenges in a logical fashion.

### **Postoperative Care and Rehabilitation**

The immobilization used for an extraphyseal fracture depends on the stability of the fracture and the age of the patient. Young children with fractures that require reduction are immobilized with long arm mitten casts. As the child ages, or with stable injuries, the degree of immobilization is decreased. For an adolescent, this may mean that only the digital tip is splinted to the mid-middle phalangeal level, thus allowing PIP motion.

Treatment of physeal fractures is similar to that described for extraphyseal fractures. If there is a persistent extensor lag, continued splinting even after pin removal can effect improvement. Removal of the pin at 3 weeks and full-time splinting for an additional 2 to 3 weeks is reasonable. This is particularly useful when a pin was placed across the DIP joint to gain stability.

Antibiotic use is advocated with open fractures, but in only the most complex, contaminated injuries should a patient be admitted for intravenous antibiotic administration. A seven-day course of oral agents usually suffices. Antibiotics do not replace the need for thorough mechanical debridement.

If the distal phalangeal fracture was pinned with a smooth wire or hypodermic needle, the implant can be removed at 3 or 4 weeks, usually in the office setting. There is no need for sedation or local anesthesia.

The covering of the nail bed, whether native nail plate or foreign material, falls off with dressing changes or activities of daily living. Monitoring of the soft tissue healing often is monitored for several weeks after injury. Waiting several months to evaluate the status of nail growth and adherence is imperative.

No formal hand therapy is required, because the child's own use pattern returns the DIP joint to good function in a matter of weeks. In some patients, DIP joint motion is more limited, especially when there has been an arthrotomy for articular restoration. The patient can be taught an exercise program that specifically includes middle phalanx blocking exercises to restore DIP flexion.

### **Prognosis**

Functional and cosmetic results are generally favorable. Even a small amount of extensor lag or a minor longitudinal nail ridge are well tolerated by most patients. During the early dialogue with parents, the potential for long-term impact of the injury should be emphasized. They must understand that any injury in a skeletally immature patient may interfere with longitudinal or angular growth, and that articular injury may accelerate the degenerative process later in life. However, these problems are rare. The appearance of the nail is important to most parents, and the possibility of growth failure, plate deformity, and nonadherence should be described. Although DeCruz cited a high rate of nail deformity with subungual hematomas and distal phalanx fractures, this has not been the norm in our institutions.

### **Complications**

**Inability to Maintain a Stable Bony Foundation.** Pinning the distal phalanx, with or without crossing the DIP joint, provides stability and improves the chances for healing of the dorsal aspect of the distal phalanx.

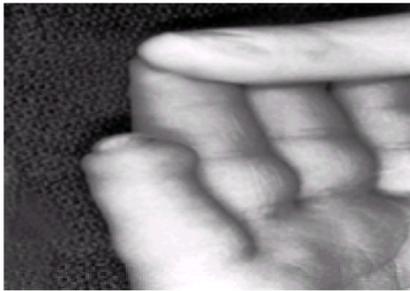
**Osteomyelitis.** Osteomyelitis is best prevented by meticulous debridement and irrigation of the fracture and soft tissue injury at the initial evaluation. If infection appears later in the treatment course, the usual principles for skeletal infection must be followed. The infected tissue must be thoroughly debrided, weighing the options of skin coverage or shortening amputation. The amount of bony support of the nail is a key issue in the retention of the germinal matrix, because a hook-nail may result when a significant amount (>50%) of the distal phalanx is lost.

**Impaired Nail Growth.** The problem can be one of poor growth or adherence. If it is a growth problem, the germinal matrix has been injured, and the absence can be complete or partial. One option for treatment is to resect the area of a "normal nail" and replace it with a full-thickness or split-thickness skin graft (42); nail bed grafting from the adjacent bed, another digit, or toe is a sound alternative. These grafts can be vascularized or nonvascularized. The results in children have been superior to those in adults (119,196,197,242).

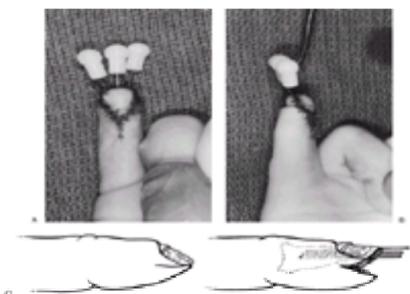
If the problem is nail lift-off or lack of adherence, then similar resection and grafting techniques can be used to contour the sterile matrix. Partial-thickness grafting

from the adjacent sterile matrix of the injured digit can be used. In the rare instance that an underlying deformity of the distal phalanx is causing the problem, splitting the sterile matrix for exposure and smoothing the dorsal aspect of the phalanx can help.

One specific complication of lost nail support is a hook-nail or "parrot's beak" nail, in which the nail plate takes a significant volar curve over the end of an abbreviated distal phalanx (Fig. 8-28). This is a potential hygiene and cosmetic problem and may prevent the tip from being functional. Atasoy et al. (9) described an "antenna" procedure for reconstructing the hook-nail. Elevating the sterile matrix and supporting the nail tissue with a volar advancement flap, thenar flap, composite graft from the toe, or a cross-finger flap can significantly improve the appearance and eliminate marginal and distal skin compromise. The name *antenna* comes from the multiple (usually three) small wires used to support the matrix postoperatively (Fig. 8-29).



**FIGURE 8-28.** A patient with a distal fingertip amputation that has gone on to develop a hook-nail deformity.



**FIGURE 8-29. A and B:** Postoperative photograph views of the patient shown in Fig. 8-28 who had the antenna procedure. This involved a volar V-Y advancement flap chosen to cover the distal tip. The sterile matrix of the nail was elevated and is supported by the three smooth wires and transferred flap tissue. **C:** Line drawings demonstrating technique of elevation and support of the sterile matrix with wires. (A and B Courtesy of William B. Kleinman, M.D. C reprinted from Atasoy E, Godfrey A, Kalisman M. "Antenna" procedure for the "hook-nail" deformity. *J Hand Surg* 1983;8:55; with permission.)

**Extensor Lag.** Some extensor lag (10 degrees or less) is not uncommon after appropriate treatment of a mallet injury. Continued extension splinting can be tried but loses efficacy after the third or fourth month. A digital cast may be an excellent way to avoid a potential lapse in splint wear that is required over a long period. Terminal tendon reconstruction with a dermodesis procedure has been described for this situation. A spiral oblique retinacular ligament reconstruction has been used to correct "chronic mallet," but the procedure is technically demanding (214). Release of the central slip insertion into the middle phalanx (Fowler procedure) can lessen the DIP extensor lag and the PIP secondary swan neck deformity. If the amount of lag is unacceptable and is accompanied by the rare occurrence of arthrosis at the DIP joint due to premature physeal closure, the only realistic options may be DIP fusion or interposition arthroplasty. These are radical steps in children, and we have not used them. If the impairment does not severely compromise function or limit future vocational avenues (e.g., musician), then the patient can make the decision later in life.

### Proximal and Middle Phalanges

The proximal and middle phalanges have similar characteristics with respect to their osteology and soft tissue anatomy. Other factors, such as the tendon balance around each segment, contribute to the unique fracture characteristics seen at the individual members of the osteoarticular column.

### Surgical Anatomy

Some pertinent aspects of the unique anatomy of the child's hand affect the pattern of fractures about the phalanges. These concepts are integral to understanding the radiograph and the clinical manifestations of skeletal trauma children.

The physes are located proximally in the phalanges. The physis of the thumb metacarpal also is located proximally; those of the other four metacarpals are distal (Fig. 8-2). The collateral ligaments at the PIP and DIP joints originate from the collateral recesses of the proximal bone and insert onto both the epiphysis and metaphysis of the distal bone. The ligament also inserts on the volar plate (Fig. 8-4). The collateral ligaments at the MCP joints of the fingers originate and insert almost exclusively onto the epiphyses of the opposing bones. The thumb MCP collateral ligaments most closely resemble those of the interphalangeal joints, having epiphyseal and metaphyseal insertions distally (Fig. 8-5).

The volar plate has a metaphyseal origin from a recessed part of the phalangeal neck and inserts distally onto the epiphysis. It receives fibers from the collateral ligaments (Fig. 8-4).

The extensor tendons insert onto the dorsal aspect of the epiphysis of the middle and distal phalanges. At the middle phalanx, the flexor digitorum superficialis inserts over about two thirds of the central portion of the middle phalanx onto the sublimis ridge. The flexor digitorum profundus has a metaphyseal insertion onto the distal phalanx.

The periosteum is important in fracture stability, reduction maneuvers, and potential remodeling capability. It is a well-developed structure that may occasionally block reduction when it is interposed in the fracture. The periosteal hinge may be a useful adjunct in fracture reduction.

### Diagnosis

**Anatomic Considerations.** The epiphysis and physis at the MCP level are relatively unprotected for two reasons: the collateral ligaments and volar plate insert exclusively proximal to the physis, and there are two epiphyses opposed at this joint. Conversely, the periphyseal bone of the middle phalanx is stabilized by the insertion of the collaterals onto both sides of the physis.

**Fracture Forces.** Pure torsion or lateral bending forces are rare: a combination is typically responsible for fractures of the phalanges. These fractures can affect the periarticular areas or the shaft. Forces resulting from a violent lateral bending moment or a significant injury directed in the AP (flexion/extension) plane can cause a phalangeal neck fracture. This fracture propagates through the subcondylar fossa and the distal diaphysis. Fractures caused by hyperextension forces are found predominately about the PIP joint and, to a lesser extent, at the MCP joint.

**Injury Mechanisms.** Most fractures of the proximal and middle phalanges result from axial loads, torsional or angular forces, such as catching balls or collisions in sports. Crush injuries include direct injuries from implements such as hammers or closing doors.

### Classification

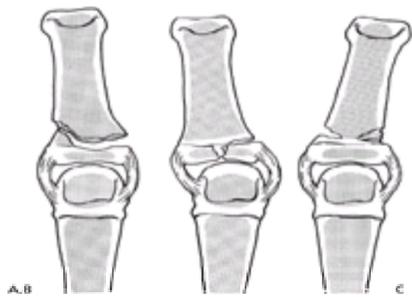
Four patterns of phalangeal fractures can be recognized: fractures involving the physis, the shaft of the phalanx, the unique fracture anatomy about the phalangeal neck, and the condylar area ([Table 8-3](#)).

Physeal  
Shaft  
Phalangeal neck  
Intraarticular (condylar)

**TABLE 8-3. CLASSIFICATION OF PROXIMAL AND MIDDLE PHALANX FRACTURES**

**Physeal Fractures.** Hastings and Simmons ([104](#)) reported that the proximal phalanx was the most commonly injured bone in their series of 354 pediatric hand fractures, and this has been corroborated by other clinicians ([12,90,127,215](#)). This was due in part to the large number of S-H II fractures of the base of the proximal phalanx. They also observed that physeal fractures occurred in the hand 34% more often than elsewhere in the appendicular skeleton. Leonard and Dubravcik ([127](#)) cited a 41% incidence of physeal fractures among their 276 pediatric hand fractures.

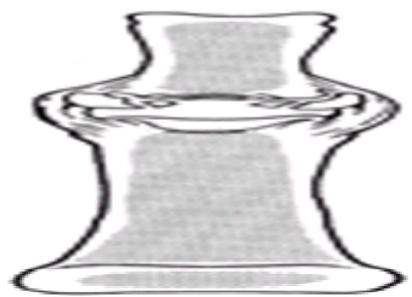
**Two Variations.** Physeal fractures of the proximal phalanx are of two varieties: S-H type II extraarticular fractures and S-H types III and IV intraarticular fractures ([Fig. 8-30](#)). Both of these patterns can be caused by lateral bending moments, but the more prevalent S-H II results from additional rotational forces.



**FIGURE 8-30.** Type A fractures of the proximal phalanx. **A:** Classic Salter-Harris type II fracture with the Thurston-Holland fragment. **B:** The intraarticular Salter-Harris type III or type IV fracture pattern. **C:** The extra-octave Salter-Harris II type of fracture with metaphyseal buckling.

Physeal fractures about the middle phalanx are rare. A lateral avulsion mechanism can cause an S-H III or IV fracture, but the entire epiphysis rarely separates through the physis, or in an S-H II pattern. A dorsal avulsion of bone by the central slip can be caused by forced flexion against an extended digit, producing a dorsal S-H III fracture. Furthermore, the lateral band can subluxate volar to the axis of rotation of the PIP joint, creating a boutonniere deformity.

Children rarely sustain comminuted intraarticular fractures of the PIP joint, considered “pilon” fractures or fracture–dislocations ([207](#)). These injuries usually involve the proximal aspect of the middle phalanx. They often occur in adolescent athletes and usually result from an axial load sustained while catching a ball or contacting an opponent. Fracture fragments from the volar side may have attached volar plate; the dorsal fragment is likely to have the central slip attached. The central aspect of the joint may be depressed, and comminution is frequent ([Fig. 8-31](#)).



**FIGURE 8-31.** Pilon fracture of the middle phalanx, in which there is comminution or central joint depression of the epiphysis.

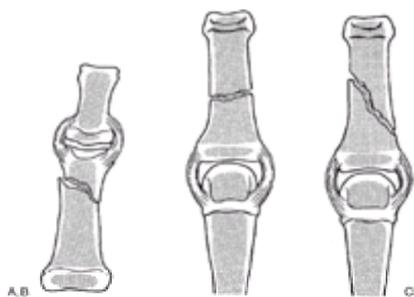
**Gamekeeper's Injuries.** A UCL avulsion injury at the base of the thumb proximal phalanx is similar to the adult gamekeeper's or skier's thumb. The fracture itself usually is an S-H III injury ([Fig. 8-32](#)). Despite the obvious fracture deformity, the focus is on the ligament that may be attached to the small fragment. Rarely, the ligament tears away from the bone, in addition to the fracture of the epiphysis. If the UCL (with or without a bony attachment) is excluded from its bed by the adductor aponeurosis, then open repair is necessary. This injury is called a Stener lesion after the clinician who described the exclusion of the UCL from its proximal phalangeal insertion by the adductor aponeurosis ([206](#)). In his 1962 account, Stener described this lesion in 25 of 39 patients with complete ruptures of the thumb MCP joint UCL. This derangement is significant because healing of the ruptured collateral ligament back to its original bed cannot occur owing to adductor interposition. The purely ligamentous injury is discussed in the later section of this chapter on Dislocations of the Hand and Carpus.



**FIGURE 8-32.** The spectrum of presentation of ulnar collateral ligament injury of the thumb. **A and B:** On stress examination, a widening of the physis is seen. No true intraarticular fracture can be appreciated, yet instability at this level is inferred by this radiographic finding. The arrow demonstrates the direction of force and location of the pathology. Varying sizes of fragments (**B and C**) can be associated with ulnar collateral ligament avulsion fractures (arrows). The size of the fragment is important with respect to the congruity of the metacarpophalangeal joint.

**Shaft Fractures.** Fractures of the phalangeal shaft are not as common as those around the joints, perhaps because of the stabilizing influences of the flexor sheath and thick periosteum, and the relatively short lever arm of the digit.

Phalangeal fractures may be transverse, spiral, or spiral-oblique (Fig. 8-33). Any of these types can be comminuted. The characteristic displacement is an apex volar pattern. The proximal fragment is pulled into flexion by the intrinsic influence, and the distal fragment is extended by the force of the central slip and lateral band insertions (Fig. 8-34).



**FIGURE 8-33.** Metacarpal shaft fractures. Fracture in the distal shaft (**A**). Transverse (**B**) or spiral oblique (**C**) fractures.



**FIGURE 8-34. A and B:** Two views of a transverse mid-diaphyseal fracture of the proximal phalanx that demonstrates the characteristic apex volar deformity. The volar flexion of the proximal fragment and dorsiflexion of the distal fragment are secondary to the tendon forces acting across the fracture site. The arrows demonstrate the actual direction of displacement.

With an oblique fracture, the deformity involves shortening and rotation. The displacement seen on radiography usually is minimal, but careful clinical evaluation of rotational alignment is critical. If a spiral-oblique fracture appears widely displaced, soft tissue interposition (extensor mechanism or periosteum) may be an impediment to reduction. Direct trauma to the phalanx, or a high-energy bending or torsional injury, can result in comminution (Fig. 8-35).



**FIGURE 8-35. A:** A crushing mechanism resulted in a complex comminuted fracture of the proximal phalanx in this child. Longitudinal splitting elements seemed to involve the proximal phalangeal growth mechanism. Excellent alignment was maintained and healing progressed. **B:** There was little disturbance of growth, but the subcondylar fossa (arrow) was somewhat obliterated by callus. There was only minor loss of flexion in this digit, and this will be allowed to mature before decisions are made concerning subcondylar fossa reconstruction.

**Neck Fractures.** Extraarticular fractures of the distal aspect of the phalanges are often located at the phalangeal neck. Displaced neck fractures, also referred to as subcondylar fractures, can occur in young children as a result of getting a finger trapped in a closing door with a subsequent attempt to withdraw it. In fractures of the phalangeal neck, the head fragment remains attached to the collateral ligaments and tends to rotate into extension (59). This displacement through the phalangeal neck disrupts the architecture of the subcondylar fossa. Because this region accommodates the infolded volar plate and volar phalangeal lip during interphalangeal flexion, malunited neck fractures result in a mechanical block to interphalangeal flexion.

**Intraarticular (Condylar) Fractures.** Phalangeal fractures that involve the joint can result from combined longitudinal and angulatory forces, and osteochondral fractures may result from shearing stress. Fractures can be associated with subluxations or dislocations of the joint. Despite heightened awareness of these fractures, they are difficult to identify and hard to treat ( 104,127).

Fracture patterns include small lateral avulsion fractures, unicondylar or intracondylar fractures, bicondylar or transcondylar fractures, and a rare shearing injury in which nearly the entire articular surface and its underlying subchondral bone are separated from the distal aspect of the phalanx. A T or Y condylar pattern can be caused at the interphalangeal joints by an axial loading injury but is uncommon in children.

### Signs and Symptoms

Aside from swelling and minor ecchymosis, many of these deformities are not clinically obvious. The child generally refuses to actively move the digit, and attempts at passive motion are resisted. Rarely, there is a subtle deviation of the phalangeal segment that reflects the depression of the condylar support.

Every fracture of the proximal and middle phalanges must be carefully examined for clinical malrotation. Malrotation of the digit can be detected by deviation of the plane of the nails with the fingers semiflexed or by an abnormal cascade with wrist tenodesis. This may result from deformity at any level in the osteoarticular column. Fracture at the middle or proximal phalanx can result in significant rotational deformity.

### Radiographic Findings

True AP and lateral radiographic views are mandatory in the assessment of phalangeal fractures, and oblique radiographs of the digits in question should be obtained.

One of the most significant factors influencing the long-term results in many phalangeal fractures is failure to recognize the extent of injury on the original radiographs. This is especially true in unicondylar and bicondylar fractures. The presence of the physes proximally and the complex periarticular architecture distally add to the difficulty of diagnosing subtle injuries.

Physeal and diaphyseal fractures usually can be detected by clinical and radiographic examination. However, a proximal shaft fracture of the proximal phalanx with volar angulation can be obscured by overlap of the digits on radiography. If there is a questionable finding on standard radiographs, special views or tomograms may assist in making a definitive diagnosis ( 130). Examination under fluoroscopy can be especially helpful when trying to characterize proximal phalangeal shaft fractures near the physis.

**Straight-Line Method.** Injuries about the MCP joint are common, but sometimes the relationships in this region where two epiphyses meet can be confusing. One radiographic technique particularly helpful in assessing alignment about the MCP joint is Campbell's straight-line method ( Fig. 8-36) (36). Although best for discovering MCP dislocations, it also can assist in evaluation of fractures about this joint that may be associated with displacement or joint subluxation. The phalangeal line is drawn down the centers of the phalanges and metacarpals. In the uninjured digit, these lines are colinear, but fracture deformity or dislocation about the MCP joint can skew this relationship.



**FIGURE 8-36.** The straight-line method of assessing alignment about the metacarpophalangeal joint. The long axes of the metacarpal and proximal phalanx should align, as they do in this normal hand (A). If there is a fracture in the proximal phalanx, as in this patient's opposite or injured hand (B and C), the axes will not be colinear (arrows). (Courtesy of Robert M. Campbell, Jr., M.D.)

**Double Density Sign.** The most difficult fracture to recognize on radiography is the partial condylar or unicondylar pattern. The AP films may look completely normal, and there is only a slight overlap of the subchondral surfaces on the true lateral projection. This small inconsistency is often overlooked as poor technique, but the "double density" shadow is made by the offset of the displaced condyle ( Fig. 8-37). A rotated unicondylar fracture is best identified on the oblique view.



**FIGURE 8-37.** A 12-year-old girl injured her left proximal interphalangeal joint playing volleyball. She was treated for a sprained finger because no fracture was appreciated in the initial radiographs. The first film (A) that was accepted as an anteroposterior view was actually rotated. On the lateral projection (B), the medial and lateral condyles are at distinctly different levels (arrows). This represents the double density shadow made by the offset of the displaced condyle. Six weeks later, the patient had a significant angular deformity secondary to a malunited single condyle fracture (C). (Reprinted from O'Brien ET. Fractures of the hand. In: Green DP, ed. *Operative hand surgery*, 2nd ed. New York: Churchill Livingstone, 1988:737; with permission.)

**"Distal Epiphysis" Means Fracture.** In phalangeal neck fractures, the rotated head can be confused with an epiphysis. However, there is no epiphysis at the distal end of the phalanx; this represents a pathologic condition. The adequacy of the subcondylar fossa must be evaluated on the lateral view.

### Treatment

Most phalangeal fractures can be treated nonoperatively. Outcomes are uniformly good for most of these fractures. Despite the favorable biology of youth, certain fractures require a more aggressive approach.

**Physeal Fractures.** Most phalangeal fractures are fractures of the proximal phalangeal base. One of the most common fractures has a readily recognizable name that is both descriptive and memorable. Rang (175) coined the term *extra-octave fracture* to describe the S-H II fracture of the ulnar aspect of the proximal phalangeal base. The digit has the clinical appearance of supraphysiologic deviation in an ulnar direction. Such an arrangement may be beneficial to the span of a pianist ( Fig. 8-38). Most of these fractures can be treated with closed reduction and immobilization for about 3 weeks.



**FIGURE 8-38. A:** An extra-octave fracture in a 12-year-old girl. **B:** The deformity was corrected with the metacarpophalangeal joint in full flexion.

**Closed Methods.** If displacement is minimal, simple splinting in the safe position for 3 weeks is indicated. If the fracture is displaced, manipulation can be performed with local anesthesia or conscious sedation. Placing a hard object (such as a pencil) in the web space and using it as a fulcrum to assist reduction has been perpetuated in orthopaedic training (232), but this manipulation technique may create a transient nerve injury from a focal crush.

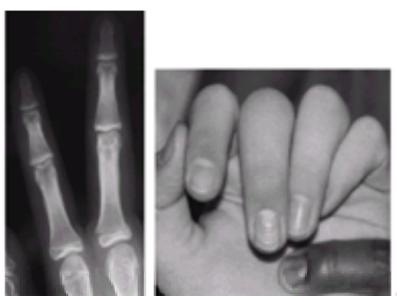
The reduction maneuver requires maximal MCP joint flexion, which relaxes the skin of the web space and the intrinsic muscles. Then the digit is manipulated into the opposite direction of the deformity. Temporary immobilization (light plaster or loose “buddy taping”) should be used while radiographs are obtained after reduction to minimize the chance of redisplacement during positioning for the radiographs.

**Operative Indications.** Despite the high rate of success with closed treatment of phalangeal base fractures, irreducible fractures have been reported (12,47,103,127). Reduction was prevented by various tissues, including periosteum and flexor or extensor tendons. In significantly displaced fractures, the distal fragment can herniate through the extensor apparatus (Fig. 8-39).



**FIGURE 8-39.** This displaced type I phalangeal base fracture was irreducible by closed means. The distal fragment can herniate through a rent in the sagittal band, periosteum, or extensor mechanism to become incarcerated in the fracture.

Some S-H II fractures may be reducible but unstable after reduction. These tend to be higher energy injuries with more internal disruption of the supporting soft tissues. Insertion of a smooth percutaneous pin after reduction may be warranted. Rarely is there any long-term functional compromise or growth disturbance ( Fig. 8-40) (103,193).



**FIGURE 8-40. A:** Anteroposterior radiograph of a Salter-Harris II fracture at the base of the middle finger. The radiograph reveals slight angulation and can look relatively benign. However, it is imperative that a clinical examination be performed with assessment of the digital cascade for malrotation. **B:** Tenodesis of the wrist with passive extension results in relatively painless digital flexion. In this case, the unacceptable malrotation is evident by the degree of overlap of the middle finger on the ring finger and the widening of the interspace between the index and middle fingers.

A displaced S-H III fracture of the proximal phalangeal base is particularly difficult to treat. The fracture fragments can be sizable and may involve more than one fourth of the joint surface, affecting the stability of the articulation. If a significant amount (>25%) of the articular surface is involved, or if residual displacement exceeds 1.5 mm, most clinicians advocate open reduction and internal fixation (104,193).

If open reduction and internal fixation are required, the alternatives are intraepiphyseal Kirschner wires or small screws that are inserted parallel to the joint surface (35,73). The latter have little use in the small phalanges of children.

Comminuted fracture–dislocations of the middle phalangeal epiphysis are rare in children, so the recommendations for treatment are extrapolated from experience in adults. Displacement of the fragments, compression of the central aspect, and instability of the joint all complicate this injury. The alternatives for treatment are open reduction with internal fixation and closed dynamic traction.

Anatomic restoration of these pilon fractures (209) is desirable, but the morbidity of opening the PIP joint is well known. If the fragments are large, then open reduction and smooth wire fixation may be possible. This can be accomplished by elevating the lateral bands, taking care to protect the central slip insertion on the dorsal

fracture fragment. A central depression of the epiphysis may require elevation. The implications for potential future growth are ominous.

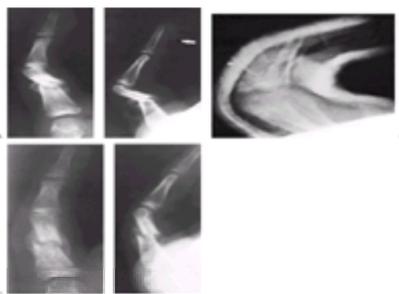
The other alternative, dynamic traction, avoids open manipulation of the PIP joint ( 1,192). This may take one of several forms, but the concepts of ligamentotaxis and early motion for articular remodeling are the basis for this treatment. Agee's force-couple concept is innovative but somewhat arduous to apply and wear ( 1). Likewise, Schenck's large outrigger device has demonstrated remarkable results but is cumbersome ( 192). The use of distraction devices is evolving, and development of new external fixators and lower profile distractors with articulating elements may provide better options.

Primary arthrodesis and volar plate arthroplasty have no role in children's phalangeal fractures. These are salvage procedures that should be contemplated only after skeletal maturity.

**Shaft Fractures.** Nondisplaced and stable fractures can be treated successfully with simple immobilization. Fractures that are unstable after reduction or cannot be reduced by closed methods require operative intervention. The fracture orientation, amount of comminution, success of closed manipulation, and stability all influence the treatment decisions for phalangeal shaft fractures. The stabilizing influence of the flexor sheath and periosteum help keep reduced fractures in place. Safe position splinting for 3 to 4 weeks should be adequate for clinical union ( 46).

Phalangeal fractures in the proximal third of the shaft can have significant volar angulation because of tendon forces. They can be difficult to see on radiography, and malunion may result from inadequate treatment. If significant remodeling does not occur, a claw hand can be caused by imbalance of the extensor mechanism ( 13).

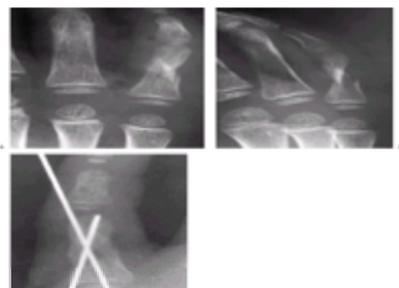
**Limits of Remodeling.** Failure of closed reduction also can cause persistent malrotation, instability of a transverse mid-shaft fracture, or unacceptable angulation ( Fig. 8-41). Simmons and Lovullo (198) reported that angulation of up to 20 to 25 degrees in the plane of motion may be acceptable in children under 10 years of age, but older children have difficulty tolerating more than 10 to 15 degrees of deformity. Coonrad and Pohlman ( 46) concluded that angulations of 30 and 20 degrees respectively will be remodeled in these groups. Favorable factors for remodeling are young age, juxtaphyseal location, and deformity within the plane of motion of the involved joint.



**FIGURE 8-41. A:** A 12-year-old boy presented with this healing angulated fracture of the shaft of the proximal phalanx of the little finger 2½ weeks after injury. **B:** Closed osteoclasis corrected the deformity, and the finger was immobilized in flexion in an ulnar gutter splint. **C:** The splint was discontinued 4 weeks later when these radiographs were made. Nearly normal function had returned when the patient was seen 3 months after injury.

Late clinical malunion is a frequent problem. Prompt referral, accurate clinical and radiographic assessment, and appropriate fracture stabilization will prevent this problem.

**Surgical Management.** Stein (205) advised closed reduction and crossed Kirschner wire fixation of transverse phalangeal shaft fractures that could not be maintained in less than 20 degrees of angulation in a well-molded splint ( Fig. 8-42). Closed pinning also is an option for spiral and oblique fracture patterns. If comminution is significant or reduction cannot be obtained or maintained, open treatment is indicated.

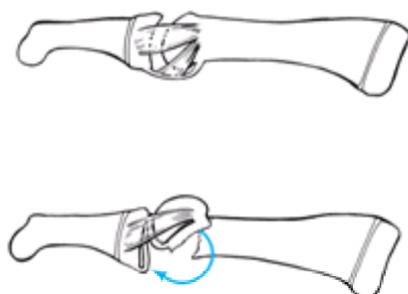


**FIGURE 8-42. A and B:** A minimally comminuted transverse fracture of the proximal phalanx with a short oblique component was difficult to control by closed methods. **C:** Crossed Kirschner wires were used to stabilize the fracture. The distal-to-proximal wire exits dorsal to the physis and therefore does not jeopardize the growth potential.

Open reduction can be performed through a dorsal incision. Splitting of the extensor tendon provides excellent exposure of fractures of the proximal phalanx, and the lateral bands can be elevated to gain access to the fracture. The choice of implant is smooth wires or screws. In adolescent athletes, the goal of early motion and protected return to activity may influence implant choice.

For fractures of the distal third of the proximal and middle phalanges, the extensor tendon should not be split, but instead deviated to the radioulnar side for access to the fracture. To permit this, part of the distal aspect of the transverse retinacular ligament must sometimes be incised at its insertion onto the dorsal apparatus.

**Phalangeal Neck Fractures.** Some clinicians have reported successful closed treatment of fractures of the phalangeal neck. However, this is difficult because these fractures tend to be unstable and displace due to the persistent attachment of the distal fragment on the collateral ligaments ( Fig. 8-43).



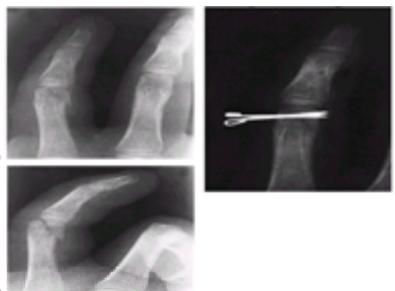
**FIGURE 8-43.** Pericondylar or subcondylar phalangeal fractures tend to rotate due to their ligamentous attachments. These fractures are difficult to reduce and control by closed means because of the interposition of the volar plate and ligamentous forces. (Reprinted from Wood BE. Fractures of the hand in children. *Orthop Clin North Am* 1976;7:527–534; with permission.)

Closed manipulation is by digital distraction and volar-directed pressure on the distal fragment with hyperflexion of the PIP joint. Percutaneous pinning usually is necessary to maintain the reduced position (Fig. 8-44) (59). This is difficult because of the small size of the fragment. Under fluoroscopy, one or two small Kirschner wires are inserted through the collateral recess and across the fracture. These wires should engage the contralateral cortex proximal to the fracture site. An alternative is to insert the pins through the articular surface of the proximal phalanx in a longitudinal fashion, crossing the fracture to engage the proximal fragment. This carries the risk of growth disturbance, because the pins cross the physis of the proximal phalanx.



**FIGURE 8-44.** Displaced phalangeal neck fracture of the proximal phalanx revealing loss of subchondral fossa at the proximal interphalangeal joint. If this is not corrected to anatomic alignment, there will be a mechanical block to flexion.

**Intraarticular Fractures.** The treatment options for displaced intraarticular fractures of the phalanges are closed reduction with percutaneous pinning and open reduction with internal fixation (20,104,193). The percutaneous technique involves the use of a towel clip or reduction clamp to effect and maintain reduction while percutaneous pins are inserted. If this is not feasible, open reduction and internal fixation is indicated through a dorsal or lateral incision with reduction of the fragment under direct vision. Care should be taken to preserve the blood supply of the fracture fragments entering through the collateral ligaments. Fracture stabilization is by either Kirschner wires or mini-screws (Fig. 8-45).



**FIGURE 8-45. A:** A 10-year-old boy sustained a displaced radial condylar fracture of the proximal phalanx when he injured his ring finger playing football. **B:** The oblique view is most helpful in determining the extent of displacement. **C:** Open reduction and internal fixation the day after injury resulted in normal function.

Rarely, severe metaphyseal comminution leaves a void after anatomic alignment of the articular fragments. This may require supplemental bone grafting and more extensive fracture stabilization with a condylar blade plate.

## AUTHORS' PREFERRED METHOD OF TREATING PROXIMAL AND MIDDLE PHALANGEAL FRACTURES

### Physeal Fractures

All nondisplaced fractures are treated with simple immobilization until pain free. Most displaced S-H I and II fractures about the hand can be treated with closed reduction (Fig. 8-46). This should be performed under comfortable conditions for the patient, including local anesthesia and conscious sedation. Simply flexing the digit maximally at the MCP joint and applying the appropriate maneuver (e.g., radial deviation for extra-octave fractures) successfully reduce the fracture. Checking the clinical alignment and rotation, as well as the radiographs, is imperative. We place the patient in a safe position splint for 3 to 4 weeks. One interval radiograph is obtained at 5 to 7 days to ensure that the reduction is being held. The rapidity of healing limits the ability to remanipulate the fracture if reduction has been lost. The remodeling potential of fractures angulated in the flexion-extension plane is significant because of their proximity to the active physis. Remodeling in the medial-lateral plane is more limited because of the minor adduction-abduction range of motion at the MCP joint.



**FIGURE 8-46. A:** Salter-Harris II fracture of the proximal phalanx of the thumb. **B:** Gentle closed reduction, performed under fluoroscopic control, yielded an anatomic reduction. The large surface area contact about the physis helped stabilize the fracture without the use of implants. Rapid healing and excellent function resulted.

## Operative Indications

Unstable fractures can be percutaneously pinned after reduction. Small (0.028-inch) smooth wires that hold the epiphysis reduced can be placed through the metaphyseal flag or the Thurston-Holland fragment, or the physis can be crossed if necessary.

If closed reduction is not possible, a dorsal exposure of the fracture allows extrication of the impediment and fixation with smooth wires. This is rarely necessary.

Salter-Harris III fractures of both the middle and proximal phalanges are difficult to reduce and maintain by closed methods. The rare dorsal S-H III fracture of the middle phalanx base can sometimes be treated by closed reduction and splinting in extension. If the fragment does not reduce, open reduction and fixation are warranted to avoid the later development of a boutonniere deformity. A dorsal approach, with an incision between the central tendon and the lateral band, is preferred. The dorsal fragment can be excised if it is small and the patient is near skeletal maturity, and the central slip can be repaired back to bone. The fragment usually should be pinned so the epiphysis is restored. The PIP joint may need to be pinned for up to 3 weeks to permit healing.

If the epiphyseal fragment is displaced more than 1.5 mm or involves more than 25% of the articular surface, or if joint stability is compromised, then open reduction and internal fixation are indicated. This is more common in the proximal phalanx at the MCP joint. The fracture is exposed through a dorsal incision between the sagittal band and the extensor tendon. The joint capsule is incised dorsally while preserving the collateral ligaments. If there is an associated collateral ligament or soft tissue disruption, this should be repaired simultaneously. Smooth wires are driven parallel to the joint to reduce and stabilize the epiphysis. It rarely is necessary to pin the fragment to the shaft. Mini-screws can be used in larger patients and larger fragments but add little stability. Motion can be started at 7 to 10 days, and the pins are removed at 4 to 6 weeks. However, intraarticular fractures typically take longer to heal.

## Rare Intraarticular Fracture–Dislocations

The very rare pilon fracture or intraarticular fracture–dislocation at the proximal phalanx base presents a management dilemma in children and adults ( 209). The treatment alternatives are open reduction with anatomic restoration and dynamic traction ( 1,192). Opening the PIP joint is worthwhile when a large enough fragment exists and the epiphysis has not been too distorted. If the child is in later adolescence, treating the complex fracture–dislocation with elevation of the epiphyseal bone (with bone grafting) is reasonable.

Outstanding results with the use of the traction method in adults have been reported, and the benefits of early motion to both the bone and soft tissue present a strong argument against open reduction. If the child is not within 1 to 2 years of maturity, a significant physeal injury at this level has growth consequences. Open reduction may increase that risk. Although the utility of dynamic traction methods in skeletally immature patients is unproven, the healing and remodeling potential of children makes them particularly good candidates for minimal intervention in these complex injuries.

## Shaft Fractures

### Closed Reduction Technique

After satisfactory anesthesia is obtained, displaced shaft fractures are reduced by longitudinal traction followed by correction of the angular deformity with flexion or the rotatory deformity with appropriate pronation or supination. For reduction of a proximal phalangeal fracture, the MCP joint is maximally flexed to relax the intrinsic muscle pull and to stabilize the proximal fragment. Recreation of the deformity with exaggerated dorsiflexion of the distal fragment disengages the fracture fragments and allows the periosteum to assist in guiding fracture alignment.

Checking the reduction with respect to length and displacement in the coronal, sagittal, and rotatory planes is essential. Three or four plaster strips are carefully applied anteriorly and posteriorly while molding the fractured digit and one adjacent digit into the safe position. Despite best efforts, it is difficult to obtain more than 75 to 80 degrees of MCP flexion. The lighter plaster dressing is stable enough to permit radiographs to be taken; if alignment is adequate, the splint is reinforced.

### Postreduction Care

Fractures of the middle phalanx should be immobilized in more of a functional position, with moderate flexion of the PIP and DIP joints added to maximal MCP flexion. For fractures of both segments, immobilization should be for 3 to 4 weeks. Tenderness usually has abated at this time, and many children show evidence of bony healing at this interval. Buddy taping can be used for an additional 2 weeks. Formal therapy or dynamic splinting usually is not required to regain motion. Protection during sporting activities should be continued until motion and strength are fully restored.

### Percutaneous Pinning

Unstable spiral-oblique fractures of the proximal and middle phalanges require closed reduction with pinning ( 91). The fracture orientation dictates the angle of pin insertion. Ideally the pins should be placed at 90 degrees to the fracture line. Traction through fingertraps or an assistant and the aid of image intensification are indispensable. Placement of the pins in the mid-axial line prevents iatrogenic injury of the more volar neurovascular structures in the mid-lateral line.

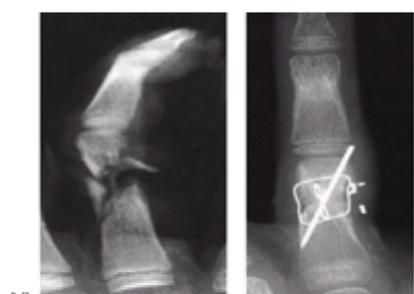
### Open Reduction

Open reduction rarely is required except in older adolescents or for irreducible fractures in younger children. The surgical approach for phalangeal shaft fractures can be dorsal or mid-lateral. The dorsal approach with tendon splitting is better for fractures of the proximal two thirds of the phalanx. The extensor tendon is split longitudinally in the center, with care to avoid injuring the underlying periosteum. The periosteum can be incised to one side of the phalanx so that the tendon and periosteal incisions do not coincide. Repair of the two layers after anatomic reduction should keep knots away from the sliding surface between them; thus, the knots in the periosteum should be buried close to the bone, and the tendon sutures should be tied so the knots face the subcutaneous tissue.

Fractures of the distal third of the proximal and middle phalanges are best approached through elevation of the lateral bands or deviation of the extensor apparatus. Access can be gained through a dorsal skin incision or a mid-lateral incision; however, the latter might be preferable to keep the skin incision away from the tendon. The insertion of the central slip on the epiphysis of the middle phalanx should not be detached, because this could result in a late boutonniere deformity.

## Complex Injuries

Combined injuries that affect several tissue systems are common in the digits. Because of their curiosity and desire to explore, children are vulnerable to injury from machinery and household equipment. Skin, tendon, neurovascular structures, and bone can all be injured in the same digit ( Fig. 8-47). Anatomic reduction and stable fixation are the goals in older children with these complex injuries so that early motion can be achieved.



**FIGURE 8-47. A:** 14-year-old boy sustained a near-amputation of his ring digit while using a bandsaw. **B:** 90-90 intraosseous wiring was supplemented with Kirschner

wire fixation to provide a stable base for soft tissue repair. Good healing was achieved in the 2 intervening months since the original operation.

Open fracture care is of paramount importance, followed by establishment of a stable bony foundation. Markedly comminuted fractures or injuries with bone loss may require staged bony reconstruction, with stabilization in the acute setting by minimal internal or external means. Again, stable fixation is the absolute goal so that early motion can be achieved. Bone grafting of defects can be performed at the time of secondary closure or skin coverage.

Vascular reconstruction, as necessary, is performed next, then the nerve status is assessed. Primary neuroorrhaphy usually is favored, but later nerve grafting may be considered for large segmental nerve defects in untidy wounds. Tendon surgery principles are the same for children and adults. A viable bed must be established, and the status of the tendon and the fibroosseous theca must be assessed. Repair of the tendon by an experienced surgeon usually is best. Tendon grafting or other salvage procedures have a very limited role in children. Flexor tendon rehabilitation in a child sometimes can be complicated due to lack of cooperation, so individualizing the protocol may be necessary. In patients under 15 years of age, cast immobilization and protected motion protocols yield the same end results.

## Neck Fractures

### Pin Fixation

If recognized early, minimally displaced subcondylar fractures can be treated with gentle manipulation and immobilization ( [Fig. 8-48](#)). Because the distal fragment is dorsally displaced, flexion of the fragment with thumb pressure may reestablish the anatomic relationships, and immobilization in flexion can maintain reduction. However, most of these fractures are more displaced and unstable. If the fracture is reducible but unstable, cross-pinning can be performed. This is best performed by flexing the PIP or DIP joint and placing small (0.028-inch) wires through the collateral recesses to engage the proximal fragment in a crossed fashion. Wires can be removed at 4 weeks and motion started soon thereafter. If an acute subcondylar fracture cannot be reduced by closed manipulation, open reduction and similar percutaneous pinning are indicated. Elevating the lateral bands to gain access would be the choice of approach to the proximal phalanx; deviating the extensor mechanism to the radial or ulnar side for middle phalangeal subcondylar fractures would yield the best access. Care is taken to maintain the blood supply to the small distal fragment to lessen the risk of avascular necrosis.



**FIGURE 8-48.** This minimally displaced type III fracture was amenable to closed reduction. Although the anatomy of the subcondylar fossa was altered by the fracture, excellent active and passive motion of the PIP joint was maintained. Excellent function was realized after 3 weeks of splinting and an early aggressive motion program.

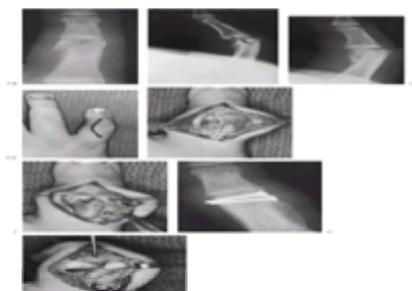
### Limited Remodeling Potential

If the fracture is unrecognized or neglected, significant loss of flexion may result. Rarely younger children may remodel at this end of the bone even though it is distant from the physis. Usually the residual deformity is functionally limiting. If the patient presents when there is still a slight fracture line present in the healing, malaligned bone, percutaneous pin osteoclasis can be performed. Using fluoroscopy, one or two smooth pins are inserted into the fracture and used to “joystick” the distal fragment into a reduced position. The fracture is then stabilized with percutaneous pins. This approach may lessen the risk of avascular necrosis with late open reduction. Rarely these fractures can be opened as late as 2 to 3 weeks by taking down the early callus with a combination of sharp dissection and blunt dissection with a Freer elevator. Again, fixation with crossed pins is the best way to stabilize the injury.

## Intraarticular Fractures

### Open Reduction Usually Necessary

Intraarticular fractures of the phalanges are likely to require either percutaneous reduction and pin stabilization or open reduction and internal fixation. [Figure 8-49](#) illustrates an intraarticular fracture of the proximal phalanx treated by open reduction through a dorsal incision. A mid-lateral incision can be used to approach an intraarticular fracture at the DIP joint, and a volar incision can be used to expose the PIP joint. The same concepts of careful tendon and periosteal handling described above hold true. Longitudinal division of the transverse retinacular band on the side of a single condyle fracture is preferred. Rarely, the extensor tendon/lateral band interval must be exploited on both sides of the digit to inspect and manipulate the fragment. Exposure of the fragment with minimal soft tissue stripping is important. This may be difficult, because an abundant amount of early callus forms in these fractures, and clearing this tissue can be arduous. Flexion of the PIP or DIP joint provides better access to the fracture.



**FIGURE 8-49.** Authors' treatment of a complex unicondylar fracture of the proximal phalanx. **A:** Anteroposterior film demonstrating the intraarticular component of fracture and comminution on the radial aspect. **B:** The double density sign (*arrows*). **C:** Oblique film better characterizes the displaced rotated condylar fracture fragment. The step-by-step operative approach follows. **D:** Dorsal approach to the fracture fragment. **E:** Incision between the central tendon and lateral band. **F:** Careful handling of the periosteal flap (*open triangle*). The fracture fragment is exposed and debrided of the early callus. **G:** Flexion of the digit shows how the displacement is exacerbated (*arrows*) because of the pull of the collateral ligaments. **H:** A mini-screw is applied after reduction of the fragments. This can be preceded by provisional smooth wire fixation. The reduction must be judged by the articular surface and not the area in the metaphysis, because part of this may have been reabsorbed or debrided. Two screws were selected because of comminution of the fracture, but care was taken not to hinder motion by incarcerating the collateral ligament. Great care must be taken not to tether the ligament when placing definitive fixation. By 6 weeks' follow-up, excellent motion had been obtained.

Sometimes the fragment has been slightly comminuted or plastic deformation has occurred, making a perfect fit all around the fragment impossible. Therefore, reduction must be judged at the articular surface. Often the subcondylar fossa must be cleared of bone, and failure to perform this step results in a flexion block despite anatomic joint reduction. Provisional or permanent fixation can be obtained with one or two smooth wires. The direction of these wires is dictated by the fracture configuration. Oblique cross-pinning is best for subcondylar and some transcondylar fractures, but small avulsions and unicondylar fractures may be stabilized better by pinning parallel to the joint. Rotational control of the fragment may require multiple implants. Pinning or screw placement through the collateral ligament should be avoided so that there is no tethering of the soft tissues.

Full motion should be verified before closing the wound. The decision to leave the pins as retained implants or to bring them out of the skin for later removal must be individualized. In younger children, the pins are left out of the skin, and a sterile dressing and cast are left intact until removal. In cooperative adolescents, treatment is more adultlike, with early motion protocols. In this case, the pins are buried to lessen the risk of skin irritation and superficial or deep infection. Countersunk 1.5- or 2-mm mini-screws are not too prominent to become troublesome later. Late removal of these implants at this level rarely is necessary.

For a simple collateral ligament avulsion fracture, protected early motion usually is satisfactory treatment. The digit should be buddy taped to the adjacent digit to prevent recurrent angulatory or rotatory stress on the healing ligament avulsion. Rarely, interphalangeal or metacarpophalangeal joint instability requires open repair. The surgical approach can be through a mid-axial incision, and arthrotomy can be avoided. Secure fixation allows early motion and less risk of long-term contracture (Fig. 8-50).



**FIGURE 8-50. A:** An avulsion fracture of the proximal phalanx with its attached collateral ligament destabilized the digit. **B:** It was approached extraarticularly through a mid-axial incision and secured with a mini-screw, permitting early motion.

Certain intraarticular fractures present even greater challenges because of their pattern or orientation ( Fig. 8-51). Intraarticular shear fractures can be treated with arthrotomy and small Kirschner wire fixation. Supplemental bone graft may be necessary. This osteochondral slice fracture can be problematic despite appropriate treatment. It is even more complicated when it is missed or its severity underappreciated in the acute setting. When multiple injuries about the interphalangeal joints occur (Fig. 8-52), the treatment approach must maximize fixation of all injuries.



**FIGURE 8-51.** This intraarticular shearing fracture of the proximal phalanx is one of the more difficult ones to treat. There is little bony support for the articular cartilage, which makes fixation difficult.



**FIGURE 8-52. A:** A 4-year-old with a significant crush injury to the long digit resulting in a metadiaphyseal and subcondylar fracture of the proximal phalanx with a combined paraepiphyseal fracture of the middle phalanx. The PIP joint appears reduced. **B:** Reduction was followed by longitudinal wire fixation. Healing of the fractures with minimal deformity was realized. **C:** There appears to be a possibility of partial growth arrest ( arrows) of the middle phalanx physis, which presented little clinical problem.

Pathologic fractures of the phalanges are rare. The most common is secondary to enchondromas and other benign tumors of bone. Rarely, they occur with remote or recent osteomyelitis. An interesting pericondylar fracture in a child with a congenital deformity is shown in Fig. 8-53.



**FIGURE 8-53. A:** A subcondylar fracture (*arrows*) in an 8-year-old boy with complete complex syndactyly of the two ulnar digits. **B:** Because closed reduction and splinting failed to control it adequately, it was stabilized with a single longitudinal pin. Recovery was complete.

### Postoperative Care and Rehabilitation

The duration of immobilization after surgical intervention for phalangeal fractures is usually 3 to 4 weeks. Percutaneous pins are removed at the same time as the immobilization. For periarticular fractures in an older child, an interval of protected motion or further immobilization may be considered. For example, pins can be removed at 3 weeks and motion started at 4 weeks. Periarticular fractures must be monitored more closely than others for the progress of motion recovery. Although little formal hand therapy is needed, the child must reestablish a usage pattern of the digit for the soft tissues and joints to improve their flexibility. The border digits, index and small, seem to be the most difficult to activate after fracture surgery. If full motion and strength are not achieved at home, then formal hand therapy may be necessary.

Patients who have more extensive surgery, such as for complex fractures or replantations, are more prone to develop motion limitations. Flexion can be lost from the mechanical block of scar or bone in the subcondylar fossa. Extension can be limited after surgical manipulation of the extensor apparatus. Attention must be directed to both flexion and extension in the postoperative regimen. Static or dynamic splinting may be instituted if progress is slow.

### Prognosis

Considering the frequency with which most surgeons care for these injuries, the number of true complications and functional impairments is low. Despite appropriate treatment, however, some patients have motion loss, malunion, and growth disturbance.

### Factors Affecting Remodeling

The factors that influence the remodeling of fractures are the patient's age at the time of injury and the amount of growth remaining, the location of the fracture with respect to the physis, and the magnitude and the plane of malalignment (23).

**Age.** The younger the patient, the more time and potential exist for remodeling. However, several clinicians argue that significant remodeling can still occur when as little as 2 years of growth remains (88,175).

**Fracture Location.** Fractures near an open physis have a greater potential to remodel. This is why so few of the prevalent proximal phalanx base fractures cause later deformity, and why there is even greater emphasis on anatomic restoration of pericondylar injuries, which are so distant from the physis.

**Malalignment.** Angular deformity usually is not strictly limited to the coronal or sagittal plane, but most of the displacement may be present in one of these directions. Deformity in the flexion-extension plane is thought to remodel reliably. Several clinicians have observed remodeling in the range of 20 to 30 degrees in the sagittal plane in children under 10 years of age and about 10 to 20 degrees of remodeling in older children (46,198).

**Limits of Remodeling.** Deformity in the coronal or adduction-abduction plane is considerably less. This is rarely quantified but is probably 50% or less than remodeling in the sagittal plane. The ability of border digits to withstand more coronal plane angulation before overlap occurs may dictate different recommendations for acceptable residual angulation. Likewise, the potential of the second and fifth rays to remodel is higher due to their relative freedom of motion in abduction. Radial abduction deformity of the index finger and ulnar abduction deformity of the small digit at the proximal phalanx level may be tolerated up to 20 degrees. It is likely that only 10 degrees will remodel to an acceptable amount in the other digits. No significant amount of coronal remodeling (5 degrees or more) occurs at the middle phalanx.

We agree with Green that little remodeling occurs at the distal aspect of the phalanges (90). However, in one report a displaced phalangeal neck fracture demonstrated striking remodeling in a 7-year-old boy. Dramatic improvement in the radiographic appearance and the motion of the digit occurred 14 months after the injury without surgical intervention (151). Perhaps very young children can reconstitute their anatomy, but early anatomic reduction remains the treatment of choice for displaced phalangeal neck (subcondylar) fractures.

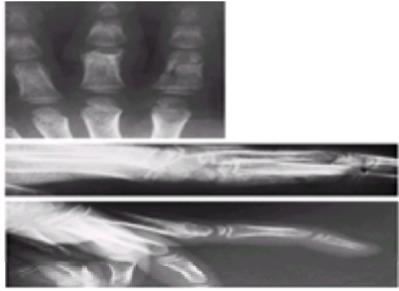
### Late Deformities

Posttraumatic degenerative joint disease is rare in children, but intraarticular injury and sepsis are two factors that may predispose to these changes. As Rank and Wakefield stated, "Unless the joint itself has been the subject of direct injury, we need never fear secondary joint changes in children to the extent that we do in adults" (176). In the rare occurrence of traumatic arthrosis, emphasis should be on the patient, not on the radiographs: minimal pain and excellent function often accompany a significant architectural disturbance. Nonsurgical treatment options are few, so the timing of intervention is likely to be the most important factor. Only when pain and functional limitation compromise the patient's daily activities or when the deformity threatens to affect future vocational choices should reconstruction be considered.

Reconstruction options include osteotomy, vascularized joint transfer, interposition or distraction arthroplasty, prosthetic joint replacement, and arthrodesis (199). Amputation may be appropriate for a completely dysfunctional digit. All these interventions are considered salvage surgery.

### Complications

**Failure to Recognize a Displaced Fracture.** Recognition of a displaced intraarticular fracture is difficult, and the impact of an unrecognized injury is functionally significant (Fig. 8-54). There usually is a suggestion of pathology on the lateral film, but it is subtle. The AP film is notorious for concealing the true nature of this injury. The oblique film most effectively shows the fracture configuration. Fluoroscopy can be useful in examining the small joints.



**FIGURE 8-54. A:** A 3-year-old girl sustained a fracture of the neck of the proximal phalanx of the index and middle fingers when they were caught in a door. The displaced fracture in the middle finger gives the appearance of an epiphysis at the distal end of the phalanx. **B:** Unfortunately, no true lateral radiograph of the injured finger was obtained. Close scrutiny of this lateral hand radiograph shows a dorsally displaced neck fracture, rotated almost 90 degrees ( *arrow*). No reduction was performed because the displacement was not appreciated. **C:** Eighteen months later, remodeling is not correcting the deformity. The proximal interphalangeal joint is hyperextended, and the patient had a significant loss of flexion.

Unfortunately, too often displaced phalangeal neck fractures are not recognized. The fracture may be confused with an epiphysis, or a minor avulsion fracture, or it may be believed to be nondisplaced. Unfortunately, many children with these fractures present late with a malunion and loss of interphalangeal flexion.

Malrotated fractures also are often missed until healed. The radiographs can be benign in appearance but the clinical appearance profound. Every patient with an acute phalangeal shaft or physeal fracture should be examined for malrotation.

**Redisplacement.** Although most phalangeal fractures are successfully treated by closed means, some fractures are difficult to control ( [Fig. 8-55](#)). In general, the closer the patient is to skeletal maturity, the more likely it is that reduction will be lost, especially with transverse or short oblique fractures of the proximal phalanx. Because of surrounding tendon forces, the fracture tends to angulate with the apex volar. The physician must guard against this when applying immobilization, and frequent follow-up is suggested.



**FIGURE 8-55. A and B:** An 8½-year-old girl with a mildly displaced fracture of the neck of the middle phalanx. **C:** Closed reduction was successful on the day of injury. A plaster splint was applied. **D:** Two weeks later, the fracture had redisplaced and the deformity was worse than it was originally. Closed pin fixation could have prevented this problem.

If the fracture is unstable in the first 7 to 10 days after injury, closed reduction with percutaneous smooth wire fixation is an excellent option. The reduction must be carefully assessed and ensured before proceeding with fixation. If the fracture cannot be reduced, then soft tissue or bony interposition may necessitate open reduction. Percutaneous pinning is still favored, but mini-screw fixation in patients close to maturity can be considered for some fracture patterns.

**Malunion.** Because children's fractures heal so quickly, a significant amount of callus is sometimes encountered in patients who were not referred in a timely manner, or in those who lost reduction in the interval since the last assessment ( [Fig. 8-56](#)). This complication can be prevented by examining the child within 24 to 48 hours of the injury, and frequent clinical and radiographic examinations should be performed in the first 7 to 10 days. Rereduction and pinning or internal fixation is always easier than reconstructing a complex malunion.



**FIGURE 8-56. A:** Deformity in the ring finger of a 13-year-old. **B and C:** Radiographs revealed a malunion of the radial condyle with intraarticular incongruity. It is difficult to appreciate this from the lateral film, but the hint of a double density shadow is seen ( *arrow*). The lateral alignment and flexor function in the digit was essentially normal. Accelerated arthrosis is possible, but no reconstructive options should be offered because of normal function.

**Late Osteotomies.** If the deformity is significant, the malunion should be allowed to mature biologically, while regaining maximal motion, strength, and tissue elasticity. Osteotomy should be performed through the site of deformity or, if feasible, at a site where good fixation can be obtained and the bone has a realistic chance of proceeding to union. For malunions of the diaphysis of the phalanges, osteotomy usually is performed at the basilar metaphysis. It is easier to make the correction at this level because the tissues are more substantial and the bones are larger for holding fixation.

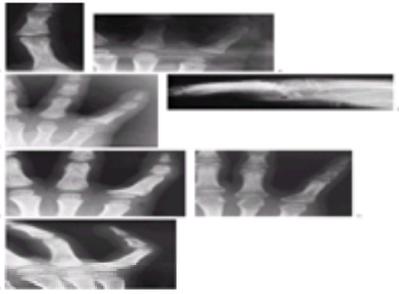
If the malunion is at the subcondylar or intraarticular level, the osteotomy needs to be at the site of deformity. If the fracture is mature, then realistic reconstructive goals must be established. Imaging studies such as CT scanning, tomography, or dynamic fluoroscopy can be used to assess the effect of the articular incongruity and the "extra" bone in the subcondylar fossa that may be impinging on the volar lip of the flexing phalanx. Motion can be restored by simply removing the mature bone from the subcondylar fossa, but this does little to improve joint congruity. When the child is still young, it may be more reasonable to accept the intraarticular deformity and perform the osteotomy of the fossa only for motion recovery.

Intercondylar osteotomy is exceedingly difficult, but it is the only way to improve or restore articular anatomy. This procedure is seldom performed, and there is little information about it in the literature. It may be best to follow the child and consider a salvage procedure, such as fusion or arthroplasty, only if significant symptoms

persist.

**Fracture Take-Down.** Juxtaarticular malunions are among the most challenging problems facing the reconstructive surgeon. If the child is under 10 years of age and the problem is recognized in the first 2 weeks after injury, callus take-down and anatomic restoration either by percutaneous osteoclasts or open repair can be successful. The status of the joint must be evaluated and any fracture callus in the subcondylar fossa that may inhibit flexion must be removed. The interval of time from injury to reconstruction can be extended to 3 weeks in children up to their mid-teens. There are no physes on the distal ends of the phalanges that would permit significant remodeling.

**Growth Disturbance.** If the original injury involved the physis, growth disturbance can occur, resulting in stunted longitudinal growth or angulatory deformity ( [Fig. 8-57](#)). Fortunately, this is rare. It is more likely when there is a vascular injury or infection.



**FIGURE 8-57.** Types of growth disturbances after fracture in the skeletally immature patient. **A:** Partial growth arrest. Seven months after a Salter-Harris type IV fracture of the middle phalanx, there is mild angular deformity and a bony bar crossing the physis. **B:** Recovery and remodeling. In this 3-year-old boy, the true extent of the cause of his swelling little finger was not appreciated. Anteroposterior view shows a rounded appearance of the epiphysis of the proximal phalanx. **C and D:** Six weeks later, the anteroposterior view reveals some bone formation adjacent to the displaced epiphysis, and a true lateral view shows the epiphysis as rotated 90 degrees (*arrow*). **E and F:** Twelve months later, anteroposterior and oblique views show a persistently distorted epiphysis. **G:** Appearance at age 18 (15 years after injury) shows complete correction. He also had normal clinical appearance and function.

## Fractures of the Metacarpals

### Fractures of the Finger Metacarpals

Unlike the phalanges, the metacarpals are relatively protected by their position within the hand and the stout soft tissues that surround them. There is significant variation in the relative mobility of the metacarpals. The stability of the second and third rays at the CMC joint is in contrast to the significant motion of the thumb and medial two rays.

### Surgical Anatomy

Fractures of the metacarpals occur at the epiphyseal or periphyseal region, the neck, the shaft, or the base. Because the collateral ligaments at the MCP joint level originate and insert almost exclusively on the epiphyses, the physes are relatively unprotected from injury ( [Fig. 8-5](#)). This is especially true when the forces have a combined axial load with a rotational component. Failure at the physis or an intraarticular fracture can be caused by any combination of forces. S-H II, III, and IV fractures of the metacarpals have all been reported ( [131,142,162,188](#)).

The metacarpal neck is the most common site of fracture in this bone. The geometry of the metacarpal and the way the hand is used in a punching motion make the metacarpal neck susceptible to fracture. The cortical bone of the subcondylar fossa is relatively thin and supports a broad metaphyseal area. The angulation of the metacarpal as it approaches the articular region also may play a role.

Fracture patterns of the metacarpal diaphysis are similar to those in the phalanges. Transverse fractures, resulting from a direct blow to the hand, are relatively uncommon. Most metacarpal fractures resulting from torsion have a spiral or oblique pattern. Fractures of the central rays are stabilized by the transverse metacarpal and intervolar plate ligaments, making significant displacement uncommon. Diaphyseal fractures of the border digits (index and small) displace more readily.

### Role of Periosteum

Each metacarpal has a thick periosteal layer that provides an excellent gliding surface between it and the undersurface of the extensor tendons. This layer can either aid or hinder the closed management of fractures in this area. The periosteum can serve as a hinge for reduction (particularly in neck fractures) or can prevent significant displacement. Conversely, the periosteum can become interposed between fracture fragments (especially in spiral fractures) and prevent reduction. If open treatment is necessary, the periosteal layer must be respected, and careful handling improves healing and motion potential.

### Avascular Necrosis

McElfresh and Dobyns ( [142](#)) and others ( [174](#)) recognized that a tense effusion can develop after intraarticular fracture of the metacarpal epiphysis. The pressure can tamponade the vessels that enter the epiphysis circumferentially adjacent to the physis ( [51](#)). Synovial vessels also may be disrupted by trauma. These vascular injuries can leave the physis devoid of blood supply and susceptible to necrosis and growth arrest.

### Mechanism of Injury

Direct trauma, rotational forces, and axial loading may all cause fractures at the metacarpal level. All these forces can be generated by striking an object or participating in contact sports, the two etiologies that produce most metacarpal fractures in children.

### Physeal Injuries

Epiphyseal and physeal fractures of the metacarpal head are common in the fifth ray but are rare in the index, long, and ring digits ( [13,36,104](#)). Light and Ogden ( [131](#)) reported five patients who sustained metacarpal epiphyseal fractures of the central three rays. Longitudinal growth disturbance resulted after axial loading mechanisms in these patients, but the prevalence of growth disturbance after metacarpal fracture is probably minimized because most of these injuries to the physis occur in older children (12–16 years of age). Fractures of the metacarpal neck are more common than physeal injuries.

### Pediatric Boxer Fracture

The mechanism of metacarpal neck fracture in the child is the same as that seen in the adult “boxer’s fracture.” Bending moments concentrated at the metacarpal neck can be generated by a force concentrated more dorsally on the epiphysis. These fractures have a component of axial loading and an element of tangential force, and the inherently weaker architecture of the neck fails under these combined forces.

### Direct and Torsional Forces

Torsional forces that cause oblique and spiral fractures in other tubular bones also may act on the metacarpals. Although the border digits are more susceptible to this

injury, the interior rays can just as easily be grasped and torqued in a wrestling match or bicycle accident. The other way in which the diaphysis can fail is by exposure to direct trauma. A transverse fracture can result by three-point bending when the bone is stabilized on its proximal and distal end and force is directed to the mid-diaphysis. A classic example of this mode of failure would be when the hand is stabilized on the ground and is stepped on, as in a football pile-up.

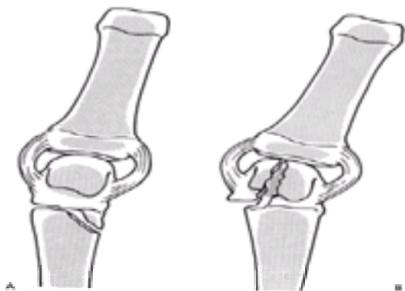
### Patterns of Fracture in the Finger Metacarpals

[Table 8-4](#) lists the types of finger metacarpal fractures.

Epiphyseal and physeal fractures  
 Neck fractures  
 Shaft fractures  
 Metacarpal base fractures

**TABLE 8-4. CLASSIFICATION OF FINGER METACARPAL FRACTURES**

**Epiphyseal and Physeal Fractures.** Although S-H II fractures of the fifth metacarpal are common among patients 12 to 16 years of age, fractures that involve the epiphyseal and physeal regions of the other digits are rare (13,36,104). Intraarticular, head-splitting fractures at the metacarpal epiphysis and physis consistent with S-H II, III, and IV patterns seldom occur at the metacarpal level (Fig. 8-58).

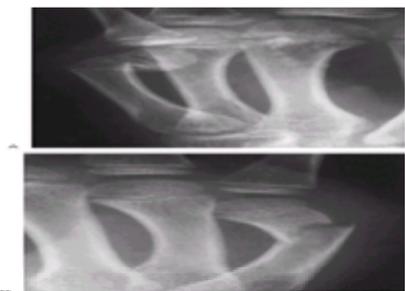


**FIGURE 8-58. A:** A Salter-Harris type II fracture of the metacarpal head. **B:** A head-splitting fracture of the metacarpal epiphysis.

In their report of five head-splitting fractures in the central three digits (131), Light and Ogden emphasized the role of open reduction and internal fixation. They postulated that fractures resulting from an axial loading injury were more likely to develop longitudinal growth disturbance.

The rich blood supply of the epiphyseal and periphyseal areas has already been described, but special consideration of intracapsular bleeding in metacarpal fractures is warranted. McElfresh and Dobyns (142) and Prosser and Irvine (174) emphasized the possible pathogenesis of avascular necrosis: it may be caused by vascular tamponade resulting from intracapsular bleeding associated with distal metacarpal fractures. Although the influence of the fracture itself on the development of avascular necrosis cannot be ruled out, these clinicians advocated joint aspiration as a preventive measure in the management of these complex injuries.

**Metacarpal Neck Fractures.** The metacarpal neck is the most frequent site of metacarpal fracture in children. It is unknown why neck fractures are so much more common than physeal fractures. Neck fractures in children are analogous to boxer's fractures in adults (Fig. 8-59). They are more common in the fifth and fourth rays than in the radial triphalangeal digits. This is why the term *boxer's fracture* is a misnomer, because trained pugilists take the punching impact on the second and third rays (those with a stable CMC joint), not the medial two rays.



**FIGURE 8-59. A:** A true boxer's fracture of the metacarpal neck of the fifth ray. **B:** This fracture occurs more in the diaphysis and should not be considered a boxer's fracture.

**Metacarpal Shaft Fractures.** Patterns of metacarpal shaft fractures are transverse, oblique, or spiral. These injuries tend to occur in older children. Like fractures in other tubular bones, the pattern of shaft fracture yields clues to the pathologic forces that caused the fracture, and indicates the mechanism of reduction. Aside from recognition of the pattern, the amount of shortening, angular displacement, and rotation is critical to making treatment decisions.

**Metacarpal Base Fractures.** The CMC joint is protected from injury by its proximal location in the hand and the stability afforded by the bony congruence and soft tissue restraints. The fourth and fifth CMC joints are more mobile than the second and third rays. Injury at this level is uncommon in children and usually results from high-energy trauma.

Fracture through the metaphyseal region of the base can occur as the result of a direct blow or an axial load (often delivered while punching). These are typically small compression injuries that usually are stable. Significant injury at this level often results from a fall from a height or a crushing mechanism, and is associated with fracture-dislocation at the CMC level.

## Signs and Symptoms

Deformity and significant swelling accompanying a metacarpal fracture can be hidden in the dorsal hand. In young children, the capacious areolar tissue can accommodate a large amount of blood and edema.

Any fractured element of the osteoarticular column can cause rotational and length deformity of the entire ray. In metacarpal fractures, the border digits (index and small) are more prone to displace significantly because they have only minimal stabilizing influence from the transverse metacarpal ligaments ( 88). A small amount of displacement at the metacarpal level can translate into significant clinical malrotation at the more distal digit.

**Radiographic Findings.** Metacarpal fractures usually are not difficult to identify on radiography. A metacarpal head-splitting fracture or some periepiphyseal fractures can be occult, however, and these may have the greatest impact on long-term outcome.

The Brewerton view is helpful in evaluating the metacarpal head and is taken with the dorsum of the hand against the cassette and the MCP joints flexed about 65 degrees. The central beam is angled 15 degrees to the ulnar side of the hand ( 122). This projection focuses on the metacarpal heads of the supinated and slightly dorsiflexed hand and may highlight subtle bony detail in this area. It demonstrates better the relationship at the MCP joint. An oblique film also may disclose a coronal fracture pattern.

It is sometimes difficult to measure the amount of displacement or angulation of a metacarpal neck fracture on a standard lateral radiograph. Therefore, it is helpful to mentally subtract superimposed images of the adjacent metacarpals to appreciate the position of the fragments. Oblique films also can help in this evaluation.

If a fracture involves the base of the metacarpal, the standard radiographic series should be supplemented with a 30 degree pronated or supinated oblique view. These show the extent of joint involvement and demonstrate any associated joint subluxation or dislocation.

The most gracile of the metacarpals is the ring or fourth ray; this is a consistent finding in children and adults. Oblique nutrient arteries that can be confused with fractures usually are seen on the volar radial aspect of the metacarpals. These lucent lines usually appear to affect only one cortex, and there is no interruption in the outer contour of the cortex.

## Treatment

### Epiphyseal and Physeal Fractures.

**Closed Reduction.** If the fracture pattern is consistent with a collateral ligament avulsion fracture, management can be based on the amount of displacement and fracture stability (Fig. 8-60). Many of these fractures can be treated by closed methods. Gentle reduction under metacarpal or wrist block should be followed by careful application of a safe position splint ( Fig. 8-61).



**FIGURE 8-60. A:** A small avulsion fracture from the collateral recess in a child near skeletal maturity can be treated by closed means ( arrow). **B:** In another child, close to growth plate fusion, this fracture propagated through the vestige of the physis ( arrow). Likewise, this can be treated by closed means. In both of these fractures, aspiration should be contemplated.



**FIGURE 8-61. A:** A 12-year-old boy with a displaced Salter-Harris type II fracture of the second metacarpal and an undisplaced fracture of the proximal shaft of the third metacarpal. **B:** Reduction was incomplete, but good remodeling is noted in this radiograph made 4 months later. **C:** Eighteen months later, remodeling is complete and there is a full range of motion in the index metacarpophalangeal joint. (Courtesy of Sigurd C. Sandzen, Jr., M.D.)

**Pin Fixation.** If the fracture is reducible but unstable, percutaneous pin fixation is recommended. If the metaphyseal flag (the Thurston-Holland fragment) is large enough, the wire can gain purchase through it, but usually the pin must cross the physis. If this is the case, the use of small-diameter smooth wires is advocated. Care must be taken to minimize the number of passes that may further damage the physis.

**Open Reduction.** Displaced intraarticular head-splitting fractures require open reduction and internal fixation. Immediate aspiration of intraarticular hematoma, which may compromise the physeal and epiphyseal blood supply, also should be considered ( 142,174).

Mini-screw fixation may be preferred over smooth wire fixation for greater stability, although either implant is acceptable. The goals of surgical treatment are anatomic reduction and stable fixation, permitting early motion.

### Neck Fractures.

**Nonoperative Procedures.** Metacarpal neck fractures are most often treated successfully by closed methods. Adequate anesthesia, closed reduction with the Jahss maneuver, and application of a safe position splint are normally the definitive treatment.

The Jahss maneuver is performed by flexing the MCP joint to 90 degrees, to relax the deforming force of the intrinsic muscles and the tightened collateral ligaments (110a). The proximal phalanx exerts upward pressure on the metacarpal head as the physician applies pressure on the dorsal aspect of the proximal metacarpal

fracture, forcing it toward the volar side to place upward pressure on the proximal phalanx. Jahss suggested immobilization in a position of MCP and PIP flexion, but we do not advocate this type of immobilization because of central stiffness and skin compromise. Immobilization in the intrinsic plus or safe position in the appropriate splint, with the PIP joints free, usually suffices. Recently, we have been limiting the immobilization to a level just distal to the MCP flexion crease and encouraging early motion of the liberated segments.

**Operative Procedures.** A few children near skeletal maturity have metacarpal neck fractures that are difficult to reduce or maintain. Options for treatment are percutaneous pinning and open reduction. As the child matures, less angulation can be accepted at the metacarpal neck. For fractures at the index and long metacarpal neck, closed reduction may be successful in reestablishing anatomic relationships, but continued instability may necessitate percutaneous pinning to maintain the reduction. Open reduction may guarantee anatomic restoration, but the dissection and manipulation may further compromise the physeal blood supply ([Fig. 8-62](#)). Stabilization with percutaneous pins is typically elected. Minifragment screws are seldom used in this region.



**FIGURE 8-62. A and B:** A dorsally angulated fracture of the second metacarpal in a 14-year-old boy. Closed reduction was unstable. **C:** Closed reduction and percutaneous Kirschner wire fixation was performed. Because of the lack of mobility of the second carpometacarpal joint, a proximal transfixation pin is usually unnecessary when the fracture involves the second metacarpal. (Reprinted from O'Brien ET. Fractures of the hand. In: Green DP, ed. *Operative hand surgery*, 2nd ed. New York: Churchill Livingstone, 1988:715–716; with permission.)

Intramedullary (bouquet) pinning has been used for metacarpal shaft and neck fractures in adults and adolescents ([72,87](#)). This technique should be reserved only for patients at or near physeal closure. Through a small incision near the metacarpal base, access to the intramedullary canal is made with a drill or awl. Prebent Kirschner wires, with the sharp tip removed, are introduced in an antegrade fashion. The wires assist in fracture reduction and stabilization. Several wires are stacked in the canal for immediate stability. The wires are cut flush with the proximal cortex. This technique is particularly well suited for the small and index metacarpals, but fractures in all the triphalangeal rays can be treated in this fashion. Because intramedullary wires are likely to cause growth disturbance in an active physis, this technique should be reserved for special situations in which immediate stability and early motion are needed for patients at or near skeletal maturity.

#### **Shaft Fractures.**

**Closed Reduction Usually Adequate.** Treatment of fractures of the metacarpal shafts is similar to that of phalangeal fractures. Closed reduction and splint immobilization suffice for most metacarpal fractures. An unstable shaft fracture can be managed by closed reduction and percutaneous pinning. Open reduction and internal fixation of a metacarpal shaft fracture are rarely indicated in children; one exception may be multiple adjacent displaced metacarpal fractures.

**Pin Fixation Options.** Some transverse or spiral oblique fractures may be difficult to control by closed methods. Pinning of the fracture fragments to the adjacent stable metacarpal or percutaneous pin fixation of the fracture itself may hold fragments in a favorable position ([Fig. 8-63](#)). If a long spiral-oblique fracture is associated with significant malrotation, percutaneous pinning or mini-screw fixation may be considered. The latter may permit early motion, which may minimize tendon adhesions. Another option for stabilization of transverse and some oblique fractures is intraosseous wires combined with smooth Kirschner wire fixation, but the magnitude of the dissection and volume of hardware argue against this option.



**FIGURE 8-63.** Radiographs of ring and small finger diaphyseal metacarpal fractures initially treated with closed reduction and splinting. The malalignment with a flexion deformity is evident on the lateral view. Multiple diaphyseal level metacarpal fractures are usually unstable with a high rate of loss of reduction with closed treatment. Percutaneous pin treatment is preferred.

**Open Reduction.** Although this procedure is rarely indicated in children, the capacity of the dorsal metacarpal area to accommodate a screw or a plate is greater than that in the digit. For long spiral oblique fractures, interfragmentary screws can be an excellent option. Anatomic reduction can be obtained, the screws can be countersunk so as not to interfere with tendon gliding, and the excellent stability can permit early motion.

For a severely comminuted fracture, especially if there is bone loss, plate fixation can be used. Bone grafting should be considered if there has been bone loss. Plating requires more extensive soft tissue dissection and occupies more space than wires or screws. Tendon adhesions are a significant concern with plate fixation. If plating is used, rigid fixation must be ensured so that early motion can combat these potential complications.

#### **Metacarpal Base Fractures.**

**Associated Injuries.** Fractures of the metacarpal base or fracture–dislocations at the CMC joint are high-energy injuries with significant tissue disruption. Palpation of the muscle compartments for signs of evolving compartment syndrome and careful neurovascular assessment are always indicated.

**Pin Fixation Techniques.** Sandzen ([187](#)) advocated closed reduction and percutaneous pinning for isolated dislocations of the small ray CMC joint. Closed treatment is aided by longitudinal traction and careful manipulation. A palpable and audible reduction may accompany successful manipulation. If the reduction is unstable (which is the rule rather than the exception), consideration can be given to percutaneous pinning. The pins can be placed transversely between the metacarpals and through the base of the fractured or dislocated metacarpal, or they can be placed through the collateral recess, across the medullary canal, and across the CMC joint. The latter pattern is more technically difficult, and care must be taken to avoid the physis. If the medullary canal can be accessed through the collateral recess, the pin can be driven from distal to proximal, crossing the reduced CMC joint.

**Open Reduction.** Open reduction may be necessary to effect reduction and ensure stable fixation ([Fig. 8-64](#)) ([118,230](#)). Various incisions can be used to gain access to the CMC joint. Longitudinal incisions adjacent to the affected ray permit investigation of the fracture–dislocation and allow fasciotomies if necessary. Percutaneous

pinning can be performed in any of the ways described previously. Regardless of the pinning method chosen, the physes should be avoided.



**FIGURE 8-64. A and B:** An 11-year-old boy sustained a fracture of the proximal shaft of the second metacarpal and a fracture–dislocation of the third carpometacarpal joint when his hand was caught in a cyclone fence he was attempting to climb. **C and D:** Closed manipulation reduced the third metacarpal joint, but buttonholing of the second metacarpal in the volar soft tissues necessitated open reduction and Kirschner wire fixation. Normal function resulted.

## AUTHORS' PREFERRED TREATMENT OF FINGER METACARPAL FRACTURES

### Role of Closed Manipulation

There is a limited role for closed manipulation and immobilization in the treatment of displaced physeal and epiphyseal fractures. It is difficult to gain access to the head-splitting variant and very difficult to control these highly unstable intraarticular fractures. When the patient is seen soon after the injury, the likelihood of successful closed manipulation may be greater. Aspirating the hematoma from the joint is a prerequisite to successful fracture manipulation.

Fractures of the metacarpal neck are particularly suited to closed treatment. Again, it is best to perform these reduction maneuvers early after the injury so that swelling is at a minimum. The Jahss maneuver (previously described) is our treatment of choice for closed reduction of metacarpal neck fractures. Under wrist block and conscious sedation as necessary, an appropriate reduction maneuver often aligns the fracture, improves comfort, and provides information about the “personality” of the fracture. We can then determine the difficulty of the reduction, the presence of interposed tissue, and the instability of the fracture. We advocate the more low-profile splint that permits motion with most of these fractures that have been reduced and are believed to be stable.

The fracture pattern and amount of comminution usually determine if closed reduction of metacarpal shaft fractures is possible. Some long spiral-oblique fractures can be successfully treated with closed methods because their fracture surfaces can be readily approximated, and the large surface area can help stabilize the fracture and promote healing. Other oblique fractures demonstrate a tendency to shorten and rotate, and may require stabilization with pins (younger patient) or screws (adolescent).

It is sometimes difficult to effect or maintain reduction in transverse fractures of the mid-shaft, but we attempt closed treatment of single metacarpal fractures. We have more success in reducing and holding fractures of the central rays (long and ring digits) than with fractures of the border digits. If the transverse fracture can be reduced, but is unstable, percutaneous intramedullary pinning through the collateral recess or tangentially from the base is used. We try to cross the fracture with a pin that is as close to perpendicular to the transverse fracture as possible.

Fractures and fracture–dislocations of the CMC joint can be reduced closed as described previously. We attempt to perform a closed reduction, but recognize the tendency of these injuries to redislocate if they are not stabilized. Our assessment of stability after the reduction dictates the need for fixation, but pinning across the CMC joint is almost always required. Additional pins to adjacent metacarpals can be used to add stability.

### Techniques for Operative Management

Despite early treatment, expert reduction maneuvers, and careful application of immobilization, some fracture patterns cannot be adequately closed.

#### *Epiphyseal and Physeal Fractures*

For epiphyseal and physeal fractures, the joint is approached through a gently curved or longitudinal incision just to the side of the metacarpal head. The arthrotomy can be made in a tendon-splitting and tendon-sparing manner; both choices are reasonable and typically do not affect later extensor function. The tendon-splitting approach is performed by incising in the mid-portion of the tendon without disturbing the underlying periosteal layer. We prefer the tendon-sparing approach. A longitudinal incision 2 or 3 mm lateral to the extensor tendon, through the sagittal band, permits elevation. Another option is to create a T-shaped incision in which the longitudinal peritendinous aspect is joined by a perpendicular incision in line with the fibers of the sagittal band. In either approach, the extensor mechanism is then elevated to a limited extent by dorsal retraction on the hood. The periosteum is carefully handled, although very little of it needs to be reflected when approaching intraarticular fractures. The capsule itself can be incised either transversely or in a T-shaped fashion, depending on the intraarticular fracture pattern. This is the least traumatic and most easily reconstructable approach into the MCP joint.

The joint capsule can be entered with a T- or L-shaped incision to expose a head-splitting fracture. The arthrotomy decompresses any hematoma in the joint. Direct manipulation of the fragments with a dental pick (respecting the hyaline cartilage) aligns the fracture. The use of reduction clamps or a transverse smooth wire can provide provisional stabilization while mini-screws are placed. We prefer mini-screw fixation, but 0.035-inch wires can be used if the fragments are too small or if multiple fractures are present.

Salter-Harris II fractures seldom require open reduction. They typically occur in adolescents, but enough remodeling is left about the physis that up to 30 to 35 degrees of angulation probably can be accepted. If the reduction can keep the angulation in the AP plane within 25 or 30 degrees and there is no rotational deformity, casting for 3 to 4 weeks is usually adequate, with functional bracing and guarded motion started at that time.

If the fracture is unstable, percutaneous pinning with a single smooth Kirschner wire is best ( [Fig. 8-65](#)). Engaging a large metaphyseal fragment is difficult but feasible. Avoiding the extensor mechanism with the pin is important and is best done with a lateral approach of the pin. The pin can cross the physis near its center and rest in the head. Penetration into the joint must be avoided.



**FIGURE 8-65. A and B:** This markedly comminuted and displaced transphyseal fracture of the fourth metacarpal ray presented in this 7-year-old boy. **C:** Effective

closed reduction, followed by percutaneous pinning, stabilized the metaphyseal aspect of the fracture and resulted in near-anatomic reduction and an excellent final result. The Salter-Harris type II fracture of the small finger metacarpal required no additional treatment aside from the splint used for the pinned ring finger ray. This secondary injury healed uneventfully.

## ***Metacarpal Neck Fractures***

### **Reduction Techniques**

Fractures at the metacarpal neck are treated with closed reduction and safe position splinting. Rarely, a fracture is quite unstable and difficult to control by closed means. After careful assessment of sensation, anesthesia in the form of a wrist block (with the possible addition of conscious sedation) can be used. If the metacarpal neck of the small or ring digit is fractured, an ulnar nerve block usually suffices. A median nerve block is necessary for comfortable reduction of fractures in the index and long metacarpals.

We advocate the Jahss maneuver described previously for reduction of these fractures. Sometimes, additional manipulation with palmar pressure to the metacarpal head can effect further reduction of the neck fracture. The reduction can be checked with fluoroscopy before applying the splint, or careful splinting can be performed and plain radiographs obtained.

### **Postreduction Immobilization**

We recommend the following measures for the application of immobilization:

1. An adequate but not excessive amount of cast padding should be applied. Skin integrity is important, but too much cast padding diminishes the ability of the plaster to hold the fracture reduction.
2. Using plaster strips in the anterior and posterior aspects of the hand and wrist permits good molding. Applying two or three thicknesses of splints on the anterior and posterior sides, followed by careful molding of the fractured rays into a safe position, can temporarily hold the reduction. Repeating this sequence two or three times over already hardened underlayers offers the best chance to stabilize fractures that are difficult to maintain.
3. Before applying too many thicknesses of plaster, it is prudent to wrap a single layer of cast padding around five or six thicknesses (on each of the volar and dorsal sides) and check the radiographs. This permits evaluation of the reduction without obscuring by plaster.
4. Careful molding with application of the splints is performed by using palmar pressure at the metacarpal head level and continued dorsal pressure on the proximal metacarpal. To avoid leaving finger impressions that could cause areas of skin damage, continuous smoothing of the plaster to eliminate areas of depression and air bubbles is advisable.
5. If the reduction is adequate, more plaster can be applied to make the splint as rigid as possible.
6. For immobilization, the safe position at the MCP and PIP joints could be used, but we have had success with a more extended MCP joint and a free PIP joint in most of our patients. If the safe position is elected, the wrist should be extended about 10 to 15 degrees and the MCP joint flexed maximally. Because children rarely develop significant stiffness, it may be advisable to extend the splint past the PIP.
7. The typical period of immobilization for metacarpal neck fractures is 3 to 4 weeks for most children.
8. We change the initial splint to a cast of similar nature at 2 weeks if needed, depending on the integrity of the cast. We typically assess the fracture reduction at 5 to 7 days to ensure that rare remanipulation is not necessary. If the integrity of the original splint is good and the reduction is being maintained, we continue the original immobilization for the entire treatment course.

### ***Unstable Fractures***

If the fracture of the metacarpal neck is reducible but unstable, we consider percutaneous pinning. This is important in unstable neck fractures in the index and long metacarpals. We have a fluoroscopy unit available so that the adequacy of the reduction and the location of the pinning can be determined intraoperatively. One limiting factor to the use of percutaneous pinning is the amount of bony purchase that can be achieved. We avoid tethering the collateral ligaments with the pin, which is why we pin in maximal MCP flexion. We seek the collateral recess as the entry site for the pin and often perform cross-pinning, meaning two collateral recess pins from opposite sides that cross near mid-shaft. Sometimes the pin simply slides down the intramedullary canal and has good purchase; this is perfectly acceptable. This method also requires 3 to 4 weeks of immobilization.

We make sure that the pins that penetrate the skin do not have any direct contact with the cast padding or plaster, because motion of the splint could cause pin track problems. This is why we pad the pins adequately. We also bend the pins to prevent migration.

### ***Shaft Fractures***

#### **Uncomplicated Fractures**

We usually are successful in treating most metacarpal shaft fractures with closed reduction and immobilization for 3 to 4 weeks. We mold the splint to provide adequate three-point bending. Downward pressure at the apex-dorsal fracture site should be balanced with upward pressure at the head and the proximal shaft. Correct rotation and acceptable length must be verified before applying the splint to keep the MCP joint flexed about 70 degrees. The PIP joint can be free, thus avoiding even transient stiffness. Because the ability of the metacarpal shaft to remodel is small compared with that of the metacarpal neck, rotation and angulation must be carefully assessed. Careful assessment of the appearance of the hand and angulation in the AP and lateral planes on radiography ensures the success of reduction.

#### **Multiple or Unstable Fractures**

Occasionally, shaft fractures are unstable or accompanied by adjacent shaft fractures. Options for stabilizing these fractures include percutaneous pinning and open reduction and internal fixation. Some of these fractures can be stabilized with cross-pinning technique or other pinning constructs that traverse the intramedullary canal. Checking the rotation of each ray after stabilization is key.

Another option is to pin the reduced distal fragment to the adjacent metacarpal with two parallel transverse wires (0.035- or 0.045-inch). An additional proximal pin placed parallel to the distal pins helps stabilize the construct. We avoid flattening of the hand: attempts to recreate the longitudinal and transverse arches of the hand are important when dealing with adjacent metacarpal fractures.

If the metacarpal shafts of the ring and small digits are involved, additional stabilization of the proximal fragment to the adjacent metacarpal is required. If the fracture is quite proximal, we consider pinning the CMC joint to avoid further influence of CMC motion.

The pins are bent and cut off, and the sharp ends of the pins are covered with a prefabricated cover for cast padding. An additional safe position splint is then applied. Fracture healing in the shaft of the metacarpal may be slower than in the areas about the physis, so we usually keep these pins in for 5 to 6 weeks. Careful assessment of clinical union is important because with excellent reduction, very little fracture callus may be seen on radiographs. When the patient is nontender to palpation about the fracture site and radiographs show evidence of fracture union after the fourth or fifth week, the splinting is discontinued.

#### ***Open Reduction***

Open reduction and internal fixation of metacarpal shaft fractures are rarely indicated. Multiple adjacent fractures, fractures with interposed soft tissue blocking reduction, or open comminuted fractures may be exceptions. Most of these patients are pre- or true adolescents. We occasionally use interfragmentary screw fixation for long spiral-oblique fractures with good bony apposition. After careful reduction and stabilization of the fracture in anatomic position, screws can be inserted, typically 1.1, 1.5, or 2.0 mm in diameter. We overdrill the proximal cortex to effect compression with interfragmentary screw fixation. Countersinking the heads of the

screws is advisable to minimize prominence and potential adhesions.

### **Surgical Approach**

Whichever method of internal fixation is chosen, the approach is similar. The metacarpal shaft is approached through an incision centered over the dorsum of the web spaces adjacent to the fracture. To minimize potential extensor adhesions, we prefer to use this interval rather than a direct incision over the bone. The incision can be extended proximally if necessary. The extensor tendon is retracted to gain access to the shaft. The thick periosteum should be meticulously respected and repaired if possible. The periosteum usually cannot be repaired after plate fixation. The interfragmentary screws often can be covered by the periosteal layer, especially if countersunk. Because the fractures should be crossed at 90 degrees with the interfragmentary screws, some of the screw heads could be in the plane of the metacarpals and would not be problematic on the dorsum.

With multiple metacarpal fractures, an approach through an incision in the intermetacarpal space is particularly helpful. We usually can reach adjacent metacarpals easily through a single incision between them. If all four metacarpals are fractured, making one incision in the intermetacarpal space on the dorsum between the index and long finger and a second between the ring and small digits permits access. If needed for developing compartment syndrome, fasciotomies of the interosseous muscles also can be performed through these incisions.

### **Plate Fixation**

If mini-plate fixation is chosen, options are a flat plate or a semitubular plate. The plate that conforms to the bone architecture and offers the best stability should be chosen. Usually, a five-hole or seven-hole plate is selected, and the hole directly over the fracture site often is left unfilled. The engagement of at least four cortices proximal and distal to the fracture is advisable.

### **Metacarpal Base Fractures: Closed Procedures**

We usually gently manipulate basilar fractures of the metacarpal after a regional block of the ulnar nerve and its dorsal sensory branch, or a Bier block. The fracture can then be immobilized in a short arm ulnar gutter splint, leaving the PIP joint free to move for about 3 to 4 weeks, or until nontender.

### **Complex Fracture–Dislocations**

Fracture–dislocations at the CMC base are more challenging. Few reports in the literature address this subject, and the patterns of presentation and treatments vary. Researchers have reported isolated ([187](#)), multiple ([118,230](#)), dorsal ([187,230](#)), and volar ([118](#)) dislocations. The treatments have varied from closed manipulation and immobilization to open treatment ([118,187](#)). Initial closed treatment failed in a complex case of fracture–dislocation of the medial four CMC joints, and later operative treatment was necessary ([230](#)). We advocate an initial attempt at closed reduction to diminish the pain and deformity in the area of the injury. Regional (wrist) or Bier block and conscious sedation usually provide adequate anesthesia. We use fingertraps for distraction. An audible and palpable “clunk” accompanies a successful reduction, but we remain concerned about instability. We question the adequacy of simple closed treatment for this injury. An isolated fifth ray fracture–dislocation can be stabilized with a well-padded splint, but this is not our choice; stability must be ensured by pinning.

### **Percutaneous Pinning**

Percutaneous pinning of the reduced fracture–dislocation can be performed with oblique and transverse pins between the metacarpals and across the CMC joint in single- or double-ray injuries. We also use longitudinal Kirschner wires inserted through the collateral recesses and down the canal to eventually cross the CMC joint ([118](#)). However, inserting these pins can be difficult, and the physis is at greater risk for pin penetration. An easier arrangement is to place oblique and transverse pins through the bases of the metacarpals, then perform intermetacarpal transverse pinning, respecting the transverse arches of the hand.

Radiographs (including appropriate oblique views) are needed to verify the reduction and pin placement. Very unstable ulnar-sided dislocations tend to slip if not pinned after reduction. Furthermore, fragments can rotate (up to 180 degrees) and block reduction. If the surgeon is not completely comfortable with the reduction, then proceeding to open reduction is advocated.

### **Open Reduction**

If reduction cannot be achieved by closed means, or if there are multiple irreducible CMC dislocations (especially if the CMC dislocation is volar), two dorsal incisions in the II-III and IV-V web areas at the CMC level are necessary. These incisions allow access to the bony injuries at the two adjacent rays and permit volar and dorsal fasciotomies if needed for developing compartment syndrome. Bony reduction is performed, ensuring that the articular fragments are not rotated or blocking reduction at the CMC joint. Either pinning method described can be used.

### **Postoperative Care and Rehabilitation**

For fractures treated by closed methods, no formal therapy is needed after casting. In very active children and young athletes, a light splint can be worn for protection from direct trauma and as a signal to the patient and his or her peers that the hand is not quite back to normal. Free motion of the digits and wrist is permitted while the patient is in a controlled environment, and the splint is weaned off by 7 or 10 days.

Epiphyseal and physeal fractures successfully stabilized by mini-screw fixation should be aggressively rehabilitated. After 3 to 5 days, a removable splint is applied. Active-assisted motion is performed six to eight times a day. Interval splinting is discontinued after 3 to 4 weeks. Dynamic splinting rarely is required.

Neck fractures treated with percutaneous pinning are maintained in a safe position splint with the PIP joint free for 3 to 4 weeks. The pins are removed at the end of this period, and more aggressive mobilization of the MCP joint with active and active-assisted motion is begun. If the intramedullary technique was used, unrestricted active motion is started after 3 to 5 days of hand rest. A removable splint can be furnished for interval wear. The intramedullary pins need not be removed.

For shaft fractures, the postoperative care is determined by the method of fixation used. Percutaneous pin fixation should be protected for 4 to 6 weeks in a splint. Because dorsally angulated shaft fractures may redisplace if fixation is removed too soon, pins should be removed after radiographs demonstrate bridging callus. After pin removal, a motion program can commence. If a spiral-oblique fracture was fixed with screws, motion can safely start within a week of surgery, depending on the status of the wound.

Percutaneous pins stabilizing fracture–dislocations of the metacarpal base should be left in place for at least 5 or 6 weeks. It is important to assess the patient clinically and with a complete radiographic series to determine the progress of healing. When the pins are removed, active and active-assisted motion of the adjacent joints can begin.

### **Prognosis**

Most metacarpal fractures of all types heal well and leave little residual deformity or functional limitation. The factors that may hinder the eventual outcome are summarized for each type of fracture in [Table 8-5](#).

Epiphyseal and physeal fractures  
 Avascular necrosis, malreduction/malunion  
 Neck Fractures  
 Excessive apex dorsal angulation, malrotation  
 Shaft fractures  
 Malrotation, soft tissue interposition, nonunion  
 Metacarpal base fractures  
 Loss of reduction, malreduction of articular fragments, late instability

**TABLE 8-5. ADVERSE FACTORS FOR FINGER METACARPAL FRACTURES**

### Complications

**Avascular Necrosis of the Metacarpal Head.** Avascular necrosis of part or all of the intraarticular metacarpal head can occur after fracture and may be influenced by the direct injury and also by the intracapsular pressure caused by the contained hematoma ( Fig. 8-66). Avascular necrosis may result in significant irregularity of the articular surface, but symptoms rarely are significant enough to warrant surgical intervention.



**FIGURE 8-66. A:** A 12-year-old boy sustained a minimally displaced Salter-Harris type II fracture of the proximal phalanx in a fall. **B:** One year later, the patient presented with mild pain in the metacarpophalangeal joint. The metacarpal head shows considerable deformity secondary to avascular necrosis. (Courtesy of James H. Dobyns, M.D.)

If the insult occurs in a growing child, remarkable remodeling of the adjacent articular surface may still result in a functional joint. Part-time splint protection while the joint is symptomatic is all that is needed in most patients. Rarely, significant joint incongruity as a residual of avascular necrosis may require reconstructive surgery. There are several reconstructive options for incongruous joints with early arthrosis, but none are very satisfactory. The most predictable treatment is arthrodesis; arthroplasty (interposition, perichondral resurfacing, distraction, or implant) and vascularized joint transfer rarely are appropriate in children ( 199).

**Rotational Malalignment.** Even a small amount (<10 degrees) of rotational malalignment of the metacarpals can cause overlap of the digits in flexion and functional disturbance (Fig. 8-67). If rotation was not corrected and maintained by closed reduction and percutaneous pinning, decisions about the timing and type of treatment are important. The correction can be performed electively several months after the original injury. This permits the fracture to mature and allows the tissues to stabilize.



**FIGURE 8-67. A and B:** A 15-year-old boy presented with severe overlapping of the ring finger on the little finger when he made a fist. Four months earlier, he had a spiral fracture of the metacarpal that had healed with a severe rotatory malunion. **C:** Distal osteotomy through the deformity stabilized with pin fixation corrected the malrotation. Basilar rotational osteotomy is another treatment alternative. (Reprinted from O'Brien ET. Fractures of the hand. In: Green DP, ed. *Operative hand surgery*, 2nd ed. New York: Churchill Livingstone, 1988:731; with permission.)

We advocate proximal shaft or basilar osteotomy to correct malrotation of even a distal deformity (in contrast to performing an osteotomy at the site of the deformity to correct angulation). Performing the correction away from the site of original fracture has many advantages. The bone in this area heals well, and the relatively large cancellous surfaces permit correction of rotational malalignment. Kirschner wire fixation usually is satisfactory, but a small T plate applied to the tension side of the bone is an acceptable alternative.

**Nonunion.** Experience with metacarpal nonunion is limited. Although treatment approaches have varied, the results have all been satisfactory. Ireland and Taleisnik (110) reported on two patients, noting that their report of this entity was the first in the literature. A 10-year-old girl underwent bone grafting and pinning of painless but clinically deformed second and third metacarpal nonunions 7 months after injury. She healed uneventfully. A second metacarpal nonunion in a 3-year-old boy healed spontaneously after 2 years of follow-up. Ogden ( 162) successfully treated nonunions of the second and third metacarpals of a child by reduction and pinning without bone grafting. These patients all appeared to be relatively asymptomatic. Unless clinical deformity is present, following these “nonunions” (perhaps until maturity) may be a reasonable alternative. When reconstruction is performed, open reduction and bone grafting are the standard. Fixation choices are percutaneous pins or small plates.

### Fractures of the Thumb Metacarpal

Fractures of the thumb metacarpal neck and head are uncommon and usually result from direct injury. Fractures of the metacarpal shaft are common, and typically closed reduction is adequate. Metaphyseal and physeal fractures of the thumb metacarpal have received most of the attention in the literature because of their frequency and potential impact on function.

## Anatomic Considerations

**Muscle Forces.** The muscles that originate or insert on or distal to the metacarpal may influence fracture geometry. Depending on the vector of pull, these musculotendinous units act as stabilizers or deformers of particular fracture patterns. The other soft tissues (collateral ligaments, volar plate, periosteum) also influence the pathoanatomy. Their relative positions in the thumb ray were described earlier in this chapter.

**Musculotendinous Units Inserting Distal to the Thumb Metacarpal.** Like the tendon insertions on the terminal phalanges of the fingers, the flexor pollicis longus inserts on the metaphysis and the extensor pollicis longus on the epiphysis, on their respective palmar and dorsal surfaces. The distal two thirds of the proximal phalanx is devoid of tendinous insertion. The base of the proximal phalanx and its intimate structures (the volar plate and sesamoid bones) are the site of multiple attachments that play a role in fracture mechanics. The adductor pollicis has insertion into the extensor apparatus and onto the medial aspect of the proximal phalanx. The aponeurotic expansion plays a role in the pathologic anatomy of thumb UCL ruptures by excluding the torn UCL from its original bed (the Stener lesion) ( 206). The flexor pollicis brevis has two insertions: the fibers insert onto the volar medial aspect of the proximal phalangeal base and into the medial sesamoid and volar plate tissues. Most of the abductor pollicis brevis insertion is onto the sesamoids and volar plate.

All three of these structures (adductor pollicis, abductor pollicis brevis, flexor pollicis brevis) can deform fractures occurring at the metacarpal level. Their direction of displacement or activity in fracture stabilization is dictated by their relative positions and directions of pull. The fiber orientation and the point of insertion determine the degree and pattern of fracture deformity.

**Musculoskeletal Units Inserting on the Thumb Metacarpal.** The opponens pollicis has a very broad insertion over the central metacarpal shaft and base. It plays an integral role in the concerted motions that allow thumb opposition. The opponens pollicis also may participate in fracture situations by pulling the distal fragment into relative adduction and flexion, along with the units that insert distally on the flexor surface.

At the metacarpal base, the abductor pollicis longus has one of its multiple potential insertion sites. The abductor pollicis longus can have insertional slips to the fascia of the thenar eminence, the trapezium, and the CMC capsule. It also is the prime deforming force in some fracture–dislocations or subluxations ( 15) about the thumb CMC joint. In addition, the flexor pollicis brevis can have part of its origin from the more medial aspect of the metacarpal base. This may add to the flexion force and apex-dorsal angulation in some fractures of the metacarpal shaft.

**Bony Architecture at the MCP joint.** The “double saddle” architecture of the articular surfaces of the thumb metacarpal and distal trapezium is uniquely well suited to provide mobility for positioning the thumb in space. It also locks into a stable arrangement when pinch force is initiated. When the metacarpal is fractured, the mobility present at the CMC joint can make reduction difficult. There is no volar plate or well-developed collateral ligament system to provide reinforcement. When the base becomes untethered from the rest of the column, it can be difficult to balance the remaining distal metacarpal fragment onto this moving target.

## Classification

**Head and Shaft Fractures.** Fractures of the thumb metacarpal head and shaft are similar to those of the triphalangeal digits ( Table 8-6). There is little need to subclassify these injuries, because of this similarity and because they are treated in a similar manner to their medial counterparts. The focus of this section is on the variable patterns of metacarpal base fractures, including fractures that involve the physis and epiphysis ( Fig. 8-68).

Fractures of the head  
Fractures of the shaft  
Fractures of the thumb metacarpal base  
Fractures distal to the physis  
Salter-Harris II fractures—metaphyseal medial  
Salter-Harris II fractures—metaphyseal lateral  
Intraarticular Salter-Harris III or IV fractures

TABLE 8-6. CLASSIFICATION OF THUMB METACARPAL FRACTURES

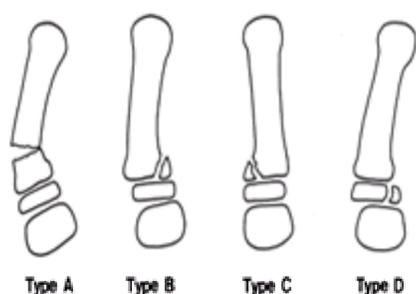


FIGURE 8-68. Classification of thumb metacarpal fractures. **A:** Metaphyseal fracture. **B and C:** Salter-Harris type II physeal fractures with lateral or medial angulation. **D:** Salter-Harris type III fracture (pediatric Bennett's fracture).

**Thumb Metacarpal Base Fractures.** Type A fractures occur between the physis and the junction of the proximal and middle thirds of the bone. They often are transverse or slightly oblique and angulated apex-lateral, with an element of medial impaction ( Fig. 8-69).

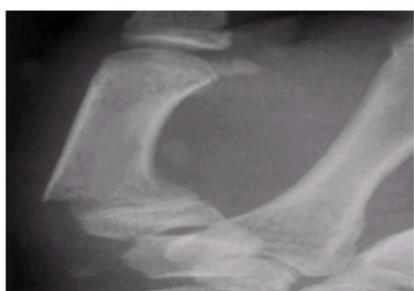


FIGURE 8-69. Thumb metacarpal fracture. This transverse fracture does not involve the growth plate. It was successfully reduced and went on to heal nicely.

Salter-Harris II fractures are prevalent at the thumb metacarpal base. Most of the patterns have the metaphyseal flag on the medial side (type B) and the shaft fragment angulated laterally and slightly proximally from the pull of the abductor pollicis longus. In addition, the adductor pollicis may adduct the shaft. Although this common pattern resembles Bennett's fracture with respect to the deforming forces, it does not have an intraarticular extension.

The less common type C thumb metacarpal base fracture has the reverse pattern, with the metaphyseal fragment on the lateral side and the shaft displacement medial. This may result from more significant trauma and usually is more difficult to treat by closed methods.

The type D fracture is an S-H III or IV fracture of the thumb base that most closely resembles the adult Bennett's fracture. Several clinicians have related anecdotal experience with this injury ([23,79,88,193](#)) and have underscored the rarity of this pattern. The deforming forces are similar to those for type B injuries, but the intraarticular component of the fracture permits the base-shaft fragment to subluxate laterally at the CMC joint.

### Signs and Symptoms

Swelling and ecchymosis are obvious signs of significant injury to the metacarpal. Most of the swelling appears to be in the thenar eminence. Depending on the level of injury, the thumb can be malrotated or angulated. Active thumb motion usually is limited.

The planes of motion for the thumb are different from those of the rest of the hand. When conducting the physical examination, motion of the thumb in radial and palmar abduction should be assessed carefully. The integrated motion of opposition also should be scrutinized. Malrotation is more challenging to judge, but the perpendicular relationship of the thumb's nail plate to those of the other digits provides a good guide.

### Radiographic Findings

A standard set of hand films can be supplemented by a hyperpronated view of the thumb that accentuates the detail at the CMC joint. Biplanar images of the thumb are a must. Type B fractures often appear well reduced on one view (the lateral view of the thumb) but are significantly displaced on the AP radiograph.

### Fracture Eponyms

We avoid the use of eponyms to describe fracture patterns about the metacarpal base. The use of terms such as *children's Bennett's fracture* or *baby Bennett's* do little to assist the evaluator in developing a plan of treatment. This is why we discourage the use of the terms *Bennett's fracture* ([15](#)) and *Rolando's fracture*. Terms to characterize the size, location, intraarticular or physeal extension, degree of comminution, and displacement of this complex set of fractures are more descriptive.

### Treatment

#### Head and Shaft Fractures

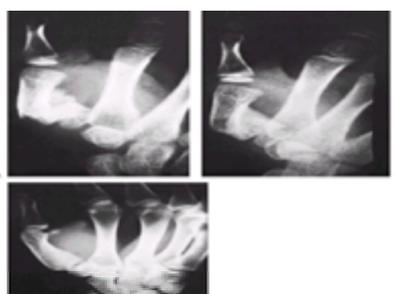
The need for anatomic restoration of intraarticular or peri-articular fractures of the distal metacarpal has already been emphasized in other sections. Fractures of the thumb metacarpal shaft can be treated much the same as those of the medial four rays.

#### Thumb Metacarpal Base Fractures

##### Type A.

*Closed Reduction.* Type A fractures usually can be treated by closed methods. Although swelling about the thenar eminence limits manipulation of the fracture and diminishes the effectiveness of immobilization, most fractures can still be treated successfully by closed reduction and immobilization.

The maneuver is performed with local anesthesia and is accomplished by exerting an extension force on the metacarpal head; hyperextension of the MCP joint should be avoided. Pressure is applied to the apex of the fracture to effect reduction. With the proximity of the fracture to the physis, exact reduction may not be required, because remodeling is rapid in this region of the bone ([Fig. 8-70](#)). Ogden ([162](#)) and others ([104,117](#)) have shown that lateral angulation of up to 30 degrees can adequately remodel.



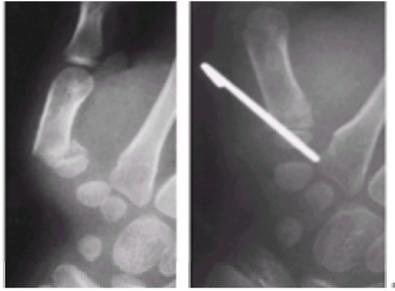
**FIGURE 8-70. A:** A 10-year-old boy with a severe head injury was first noted to have this markedly displaced type A fracture of the first metacarpal 3 weeks after injury. **B:** One month later, the radiograph shows union of the unreduced fracture with some early remodeling. **C:** Seven years later, the radiograph shows that complete remodeling has occurred. Functioning was normal. (Remodeling around this multiaxial joint can be compared with that seen in fractures around the proximal humerus.) (Courtesy of Sigurd C. Sandzen, Jr., M.D.)

*Percutaneous Pins.* If there is interposed tissue or if the reduction is too unstable to maintain in a splint, percutaneous pinning can be used. Because of the fracture pattern, it is highly likely that the physis will need to be crossed by the smooth wires. Pinning across the CMC joint can even be performed, but it is usually reserved for more proximal fracture patterns.

##### Types B and C.

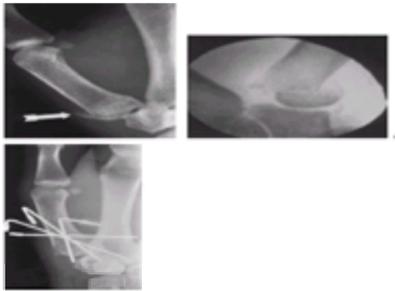
*Closed Reduction.* Type B and C fractures deserve an attempt at closed reduction, but the mobility of the metacarpal base and the swelling about the ray make this difficult. Comminution and soft tissue interposition (or transperiosteal "buttonholing" in type C fractures) ([123,232](#)) may further complicate the situation. Regardless of the method used to obtain reduction, anatomic restoration of this injury, which involves the physis, is important and has been emphasized by Smith and Peimer ([201](#)). Other clinicians have documented significant remodeling in S-H II fractures in this area ([13,117](#)). If closed reduction is accomplished and maintained in a short arm thumb spica splint, radiographs should be obtained after splint application and again in the first 5 to 7 days to reassess the status of the reduction.

*Percutaneous Pins.* If closed reduction is possible but the resulting relationship is unstable, percutaneous pinning is an excellent alternative ([Fig. 8-71](#)). Three options for pin configuration are direct fixation of the reduced fragments (however, this is prohibitively difficult because of the small size of the metaphyseal flag); pinning across the CMC joint in a position where the fragments are reduced; and pinning from the first to the second metacarpal to stabilize the larger shaft-base fragment. Combinations of these configurations may need to be used for more complicated or unstable fractures.



**FIGURE 8-71. A:** The reduction of this type B fracture was unstable. **B:** A single percutaneous pin was necessary to maintain alignment. An excellent functional result followed.

*Open Reduction.* Open reduction must be performed if the epiphyseal fragments cannot be aligned and maintained by closed means. Butt (35) observed that medially displaced type C fractures require open reduction (Fig. 8-72). This often is due to the interposed periosteum blocking the reduction (232).



**FIGURE 8-72. A:** The small metaphyseal flag on the lateral aspect of the thumb base looks fairly innocuous in one projection (arrow). However, it was a herald of more marked displacement of the distal fragment in a medial direction. **B:** This was well imaged with the aid of fluoroscopy when closed reduction was attempted. Closed reduction maneuvers were unsuccessful, because the physeal fracture appeared to have interposed tissue and was difficult to manipulate. **C:** Open reduction was performed through a curvilinear incision over the carpometacarpal joint. The fracture was stabilized by Kirschner wires that crossed the fracture site, the physis, and the carpometacarpal joint. An additional pin was placed between the first and second metacarpals to keep the thumb in good position. Near-anatomic reduction was realized with rapid healing and excellent function.

#### **Type D Fractures.**

*Nonoperative Treatment.* Most of the experience with treating type D injuries has been in adults. Numerous treatment methods have been described, including splinting, traction devices, pinning, and special screw fixation (208). For type D fractures in children, closed reduction has been tried with variable results by Gedda (79). He reported 105 patients with fracture–dislocations of the thumb trapeziometacarpal joint, only two of whom had open physes. One patient did well with 4 weeks of immobilization; the other, who required repeated reduction attempts, had significant loss of mobility, early physeal closure, and persistent subluxation at a 4-year follow-up. Griffiths (96) reported two childhood Bennett's fractures, both treated by closed means. Healing of the fracture was delayed in the first patient, but the outcome was good at 1 year. In the second patient, significant clinical and radiographic deformity was present at follow-up, but no pain or motion loss resulted.

*Open Reduction.* The best results have been reported with operative means to restore anatomy. Blount (23) used skeletal traction through the head or shaft of the proximal phalanx to treat this injury in an adolescent. Percutaneous pinning of the reduced fracture, including trans-CMC pinning, was successful in a report by Rang (175). Segmüller and Schönenberger (193) recommended open treatment as a way to ensure anatomic reduction and fracture stability. The implant choice can be individualized, but smooth wires are favored to minimize potential injury to the physis and articular cartilage (Fig. 8-73) (88,193).



**FIGURE 8-73. A:** A 14-year-old boy sustained this Salter-Harris type III fracture of the proximal thumb metacarpal when he fell out of a pickup truck. There is slight lateral subluxation of the carpometacarpal joint. **B:** Uneventful healing and normal function followed open reduction and Kirschner wire fixation. (Reprinted from O'Brien ET. Fractures of the hand. In: Green DP, ed. *Operative hand surgery*, 2nd ed. New York: Churchill Livingstone, 1988:769; with permission.)

*Traction.* Although not an initial choice for the treatment of intraarticular metacarpal base fractures, oblique skeletal traction is an alternative treatment that may have a role in some complex injuries. Spanberg and Thoren (203) described this method, which has the advantage of minimal tissue dissection but offers no direct control of the fragments. More severe comminution or skin compromise overlying the thenar area are possible indications for this method.

*External Fixation.* In a severe open injury with potential bone loss, there is another alternative that we have used extensively in our adult patients with crush injuries to the hand from farming or industrial accidents. Büchler et al. described a quadrilateral external fixator system between the first and second rays that keeps the column out to length, permits soft tissue management, and maintains the first web space span (32). Staged reconstruction can then be performed, including bone grafting and skin coverage.

## **AUTHORS' PREFERRED TREATMENT OF THUMB METACARPAL FRACTURES**

### **Closed Reduction**

For most thumb metacarpal fractures, closed manipulation and splinting is the appropriate and definitive treatment. Intraarticular fractures of the head and complex

shaft fractures in the distal aspect of the bone with soft tissue interposition may be the exceptions to this rule.

Base fractures usually can be managed by closed methods. Type A and B fractures can be reduced under adequate conscious sedation supplemented by radial and median nerve blocks. The maneuver for reduction consists of palmar-directed pressure exerted over the apex of the fracture, with counterpressure applied to the metacarpal head. Pressure applied distal to the head will only extend the MCP joint and affects the fracture minimally. A well-molded splint can then be applied and postreduction radiographs obtained.

### Acceptable Angulation

The amount of residual angulation considered acceptable in basilar metacarpal fractures is based on our experience and the reports in the literature ( [104,118,162](#)). Because the motion at the adjacent CMC joint is multiplanar and the fractures are near (or involve) the physis, the remodeling potential is great. Malangulation of about 20 degrees or less remodels quite predictably; even angulation of 30 degrees appears to exhibit significant remodeling, but the improvement in radiographic appearance is less predictable. Clinical deformity and functional loss are still unlikely, even at that degree.

### Pin Fixation

If closed methods are successful but the fracture remains unstable, it should be pinned with smooth wires (0.028- or 0.035-inch). Driving the pins in a lateral-to-medial direction is usually a reliable way to secure the larger fragments. Pinning across the CMC joint or to the adjacent metacarpal can be performed to increase stability. We have not encountered growth disturbance when the physis and joint have been crossed by a single smooth wire.

### Open Reduction

Displaced S-H III and IV fractures require open reduction and internal fixation. We prefer to perform an open reduction through an L-shaped incision overlying the CMC joint at the glabrous border of the skin. We carefully avoid injury to the small terminal branches of the dorsal sensory branch of the radial nerve, the palmar cutaneous branch of the median nerve, and the lateral antebrachial cutaneous nerve. All these branches may innervate this area. The origins of the thenar eminence muscles can be reflected medially and the capsule entered. The joint is inspected for loose fragments and cartilage damage, and the fracture is reduced and pinned.

We prefer to avoid multiple pins (or attempts) and minimize the number of pins crossing the physis. In some larger fragment fractures, parallel pinning can minimize the potential injury to the physis. Pinning of the first metacarpal to the second is a sound practice that stabilizes the entire construct and avoids transarticular or transphyseal pinning.

Type C fractures can be particularly difficult to manage. They are often more widely displaced, and the distal fragment may be buttonholed through the thick periosteum. We still attempt closed reduction, but the threshold to progress to open reduction is low. The same concepts for the open treatment of type A and B fractures hold true in the treatment of this variant.

Type D fractures require open reduction and internal fixation. Pinning across the CMC joint sometimes is needed to maintain the congruity of the joint that has been subluxated. This added stability allows the pericapsular structures time to heal. Pinning across to the second metacarpal sometimes can accomplish the same stability.

### Postoperative Care and Rehabilitation

Immobilization of 4 to 6 weeks usually is adequate, depending on the fracture severity and rate of healing. Pins usually are removed before 6 weeks. Original stiffness usually subsides quickly. Attention should be focused on the interphalangeal joint of the thumb in all phases of rehabilitation. If there is some limitation of motion at the CMC joint due to the primary pathology, additional motion may be required from the adjacent articulations.

**Return to Sports.** Return to contact sports and hand-intensive activities is a complex issue that involves the patient, parents, and coach, as well as the physician. The decision is based on multiple factors such as the nature of the injury, the temporal relationship to the season of play, and the level of participation required. Many patients with less severe injuries who are involved in minimally hand-intensive aspects of sports may be candidates for early return (within 1 or 2 weeks to permit wound healing). Protective orthoses can be worn, and a removable splint and systematic motion program can be conducted when not participating. For example, a football lineman with a central metacarpal fracture may receive a “playing cast” for practice and games and can be protected with a thermoplastic safe position splint between games ([17](#)). If the injury was more severe or if greater dexterity is needed for adequate participation, the player must regain a full range of painless motion before returning to the sport. The interval between clinical union and regaining the proper dexterity for athletic endeavors usually is 2 to 4 weeks.

There are no stock answers to these questions, which are being faced with increasing frequency in a society that places such emphasis on athletic achievement. Recommendations must be based on the surgeon's preference and experience. Recalling the admonition to “do no harm” is an excellent guiding principle when trying to reach an important decision. However, it is sometimes difficult to convince the patient, parents, and coach of the relative importance of present sporting involvement versus long-term hand function.

### Prognosis

The remodeling capabilities of fractures near or involving the physis are extensive. This is especially true about the basilar thumb joint, where multiple planes of motion encourage remodeling. Even residual deformity that exists in the metacarpal of the thumb can be concealed by excellent function. The other elements of the osteoarticular column adapt positively to limitation at one link. Even malrotation is tolerated better by the thumb than any of the triphalangeal digits. Malreduction predictably hinders long-term function only when the fracture involves a significant portion of the joint. Arthrosis is accelerated if the joint remains significantly incongruous.

### Complications

The complications described for the other tubular bones of the hand—particularly those involving the finger metacarpals—can be seen in the thumb ray. Nonunion, malunion, and aseptic necrosis can all complicate the treatment course of thumb metacarpal fractures.

For the rare nonunion, extended immobilization may result in late consolidation ([96](#)), but bone grafting and rigid fixation can be performed if the nonunion is recalcitrant. Symptomatic malunion is uncommon, but basilar osteotomy and fixation can restore the relationships needed for improved function.

Intraarticular incongruity is a potential short- and long-term problem: pain, motion limitation, and accelerated arthrosis may all be sequelae. Surgical intervention should be delayed as long as possible, and the available options discussed with the patient and parents. Salvage procedures (e.g., fusion, interposition, or implant arthroplasty) have already been described. Surprisingly good function can accompany a poor radiographic appearance, so the surgeon must remember to treat the patient, not simply the radiographs, in a child with degenerative arthrosis.

## CARPAL INJURIES IN CHILDREN

### General Principles

#### Epidemiology

Fractures and dislocations about the child's wrist are rare, and injuries to the immature carpus often present diagnostic and therapeutic dilemmas because of the difficulties in examining an injured child and the limited ability of radiographs to detail the immature skeleton ([13,158](#)).

The neighboring physis of the distal radius is among the body's most frequently injured ([49](#)). When the typical history of a fall on an outstretched hand is elicited and wrist pain is the presenting complaint, it is likely that the forces were transmitted through the distal radial physis, not the carpus ([218](#)). Nonetheless, the impact of a

highly mechanized society and the increased level of participation in athletics has made the wrist a focus of both acute and chronic injury in the child ( [183](#)).

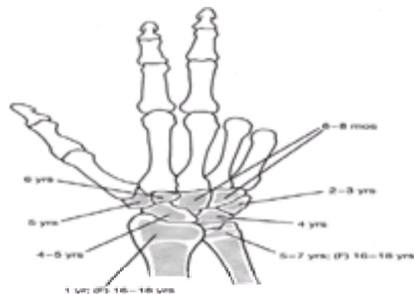
### **Anatomy**

The unique chondroosseous composition of the bones of the maturing carpus make them relatively immune to fracture throughout early development. Likewise, the inherent hyperelasticity of young tissue protects the ligamentous anatomy about the carpus.

### **Ossification of the Carpus**

The human wrist begins as a single cartilaginous mass, but by the 10th week the carpus transforms into eight distinct entities with definable intercarpal separations. Although there are some minor changes in contour, these precursors greatly resemble the individual carpal bones in their mature form ( [129](#)). It is distinctly unusual for the capitate not to demonstrate an ossification center by the sixth month, and failure to ossify by the first year may indicate a congenital anomaly.

The carpal bones ossify in a predictable pattern, with only slight variations ( [Fig. 8-74](#)) ( [95,211](#)). The appearance of the hamate on radiography closely follows that of the capitate at about 4 months. The ossification center for the triquetrum appears during the second year and is almost always present by 3 years of age. The lunate begins ossification around the fourth year. The scaphoid begins to ossify in the fifth year, usually slightly predating the appearance of the trapezium. Scaphoid ossification begins distally and progresses proximally; this factor affects certain pathologic processes that are discussed later ( [162](#)). The trapezium and trapezoid demonstrate ossification centers on radiography in the fifth year, with the trapezoid lagging slightly behind. The pattern of ossification usually concludes with the bony development of the pisiform at the ninth or tenth year.



**FIGURE 8-74.** The age at the time of appearance of the ossific nucleus of the carpal bones and distal radius and ulna are shown. The ossific nucleus of the pisiform (not shown) appears at about 6 to 8 years of age.

Within each chondral mass, ossification occurs around a defined centrum in an eccentric, centrifugal fashion ( [170](#)). The scaphoid, trapezoid, lunate, trapezium, and pisiform may demonstrate multiple centers of ossification ( [136,162](#)). Although these variations are well recognized, they may be confusing in the setting of acute trauma to the wrist region.

### **Anatomic Relationships to Carpal Fracture Patterns**

The ossific nucleus remains cloaked in its cartilaginous cover during development. This is thought to provide a unique protection that makes fracture in the immature carpus extremely rare ( [13,88](#)). This observation is supported by epidemiologic studies of fractures of the scaphoid that show them to be infrequent in children under 7 years of age but more common through the teenage years ( [93,124](#)). A critical ratio of cartilage to bone at which fractures become more prevalent has never been determined. In addition, biomechanical studies of fracture propagation in the immature carpus are lacking.

Although this section focuses on injuries to the wrist, the surgeon must appreciate the frequency and mechanics of fracture about the distal radius, because this provides the platform for surgery on the carpal bones. An in-depth discussion of fractures about the distal radius in children appears in [Chapter 9](#).

Fractures about the radial platform account for almost half of the pediatric fractures in some series ( [23,49,58,234](#)). A significant number of these fractures involve the distal radial physis, placing it at or near the top in frequency of physeal injuries ( [126,154](#)). Concomitant injury of the radius and carpus should always be suspected in children, as in adults.

### **Force Transmission During Carpal Injury**

It is important to understand the force transmission characteristics about the distal radius and wrist, because simultaneous fractures of the radial platform and carpus have been reported in children ( [3,45,94,158,216](#)). Examples of these combined injuries are given here and in [Chapter 9](#). Although these combined injuries are rare, they must be recognized and treated appropriately to maximize functional outcome.

### **Specific Carpal Fractures**

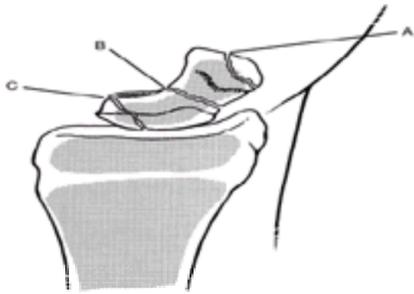
#### **Scaphoid Fractures: Epidemiology**

The scaphoid is the most frequently injured carpal bone in children ( [13,41,93](#)). This is also true in adults, one of the few similarities between wrist injuries in skeletally mature and immature patients. The incidence of scaphoid fracture in the entire population peaks in the late teens to mid-twenties ( [62,88](#)). In skeletally immature patients, the peak is at about age 15 ( [198](#)). This peak comes after a steady increase, paralleling the evolving ossification of the scaphoid from its cartilaginous precursor.

During the first decade of life, fractures to the scaphoid are extremely rare but have been reported ( [22,93,124,182,198,202,216](#)). The rarity of scaphoid fractures in very young children is somewhat comforting because of the difficulties in evaluating them. However, fractures have been reported in children as young as 4 years ( [22](#)), and there has even been a report of bilateral scaphoid fractures in an 8-year-old child ( [75](#)).

#### **Mechanism of Injury: Differences in Children**

The mechanisms and pathoanatomy of scaphoid fractures vary considerably between adults and children ( [Fig. 8-75](#)). The familiar mid-waist fracture in adults who fall on an outstretched hand is uncommon in childhood ( [135,223](#)).



**FIGURE 8-75.** The three types of scaphoid fractures. (A) Distal third. (B) Middle third. (C) Proximal pole.

Fracture of the distal third of the scaphoid, often an extraarticular injury, is the most common injury (216). Many scaphoid fractures that occur in teenagers and younger children result from direct trauma to the bone itself, not from the usual hyperdorsiflexion injury (22,124).

Fractures at the scaphoid waist are becoming more common in older adolescents as rigorous participation in contact athletics increases. These adult-like injuries carry with them the same risks of nonunion and avascular necrosis as their counterparts in skeletally mature patients (153).

Proximal pole fractures are distinctly rare in children and often represent a ligament avulsion fracture of the scapholunate ligament. The scaphoid also can be fractured as a component of a greater arc injury (140).

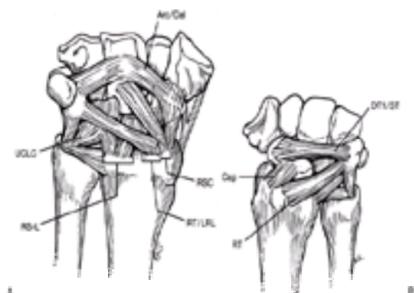
### **Regional Mechanisms Vary**

#### **Distal Scaphoid**

Distal pole fractures usually have a dorsoradial or dorsovolar fragment resulting from an avulsion injury. The strong scaphotrapezial ligaments can direct force through the distal scaphoid, and the capsular attachments on both sides may contribute additional force. The ligament/bone interface typically remains intact, with mechanical failure through the bone substance. The fracture line can skirt the periphery of the ossific nucleus, resulting in a chondral injury alone or a small osteochondral fragment that may be discernible on radiography.

A systematic study of fracture patterns of maturing bones is needed, but the observation that immature bones fail before their surrounding soft tissue structures is well accepted. There is speculation that recently ossified areas are weaker than more mature ones (46).

The different fracture patterns in the distal scaphoid may indicate a vulnerability of the bone to tension forces from several surrounding soft tissues, including the radioscaphoid ligament, radioscaphocapitate ligament, scaphotrapezial ligaments, and capsular attachments, as well as the dorsal radiocarpal or intercarpal ligaments (Fig. 8-76) (44,216).



**FIGURE 8-76.** The volar and dorsal extrinsic and intrinsic carpal ligaments. **A:** Selected volar ligaments about the wrist. *RS-L*, radioscaphoid-lunate; *RT/LRL*, radiotriquetral/long radiolunate; *RSC*, radioscaphocapitate ligament; *Arc/Del*, arcuate or deltoid ligament; *UCLC*, ulnocarpal ligament complex, including ulnocarpal volar ligament, disc-triangular and disc-lunate ligaments. **B:** Dorsal ligaments about the wrist. *Cap*, capsular attachments; *RT*, radiotriquetral ligament; *DTI/ST*, dorsal transverse intercarpal ligament or scaphotrapezial ligament.

#### **Middle Third**

Fractures at the middle third of the scaphoid account for about a third of those in immature patients. These fractures occur closer to the mid-teenage years and usually are more recognizable on radiography. Because they are encountered with advancing maturity and in situations where trauma has been significant, it is advisable to keep a high index of suspicion for other injuries about the carpus (241,75,205).

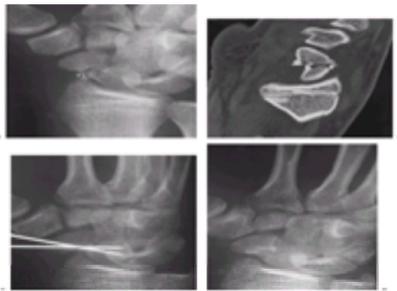
Despite the frequency with which these injuries are seen in both children and adults, there is still debate about the mechanism of fracture. The direction of force, its magnitude, and the position of the intercalated segments of the hand-forearm unit all exert influence.

#### **Influence of Dorsiflexion**

Cadaver studies by Frykman demonstrated that greater degrees of wrist dorsiflexion correlate with increasingly more distal fractures in the upper extremity (74). This work was reproduced by Weber and Chao (223), and it is generally agreed that extreme dorsiflexion (more than 90 to 95 degrees) is necessary to cause volar, tension-side scaphoid fractures at the mid-waist. Whether the fracture occurs in the proximal or distal part of the middle third is determined by the extent of radial or ulnar deviation at the time of impact.

#### **Tension Versus Compression Forces**

Some clinicians argue that the fracture is caused by tension or bending loads concentrated on the volar side at the distal pole (74,223). In this scenario, the proximal pole is stabilized and protected by the concavity of the radius and the radiocarpal ligaments while the exposed distal pole is subjected to force applied on the radial half of the palm. Other clinicians postulate a compression mechanism for midwaist fractures (43). The compressive forces may be exerted from the capitate onto the concave acetabulum of the scaphoid, or possibly from the dorsal lip of the distal radius. This may explain some of the volar radial comminution in a pattern resembling "butterfly" fragments (Fig. 8-77).

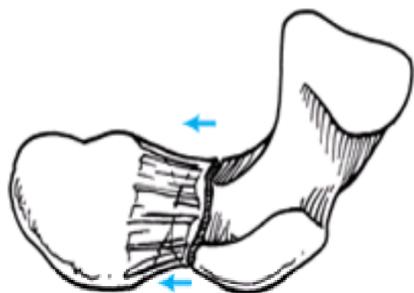


**FIGURE 8-77.** **A:** A displaced mid-waist scaphoid fracture with comminution, including a butterfly fragment (*arrow*) from the volar radial aspect. **B:** Computed tomographic scan demonstrates the comminution. **C:** Open reduction with internal fixation was performed with two smooth wires and bone graft from the distal radius. **D:** Normal healing and function resulted.

Strict deviation in the sagittal or coronal plane is not commonly encountered outside the laboratory. Torsional forces also influence the types of fractures seen ( [71](#)). The unique shape of the scaphoid at its mid-section also may play a role in the pattern of fracture propagation through this region.

### Proximal Pole

Fractures at the proximal pole of the scaphoid are rare in children. Because the pattern of ossification follows the distally derived blood supply ( [124](#)), a fracture at the proximal end of the scaphoid may propagate through the interface between newly ossified tissue and the cartilaginous anlagen, or the injury may be strictly transchondral. Proximal pole fractures can occur at the margin of the radioscapohocapitate ligament or slightly proximal to that area. More proximal fractures may represent destabilization of the scapholunate joint, because the interosseous ligament may avulse a small fragment of bone from the proximal pole rather than a mid-substance rupture ( [Fig. 8-78](#)).



**FIGURE 8-78.** A proximal pole scaphoid fracture can destabilize the scapholunate articulation. The scapholunate interosseous ligament can avulse a fragment of bone from the proximal pole (*arrows*). Small proximal pole fractures, therefore, are not innocuous and may herald significant disturbance in wrist mechanics.

### Classification

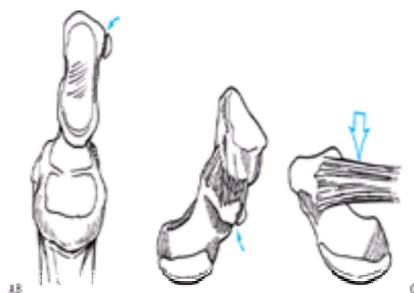
[Table 8-7](#) lists the types of scaphoid fractures.

- Fractures of the distal pole
  - Extraarticular distal pole fractures
  - Intraarticular distal pole fractures
- Fractures of the mid-waist
- Fractures of the proximal pole

**TABLE 8-7. CLASSIFICATION OF SCAPHOID FRACTURES**

### Type A: Fractures of the Distal Pole

Fractures of the distal pole are the most frequent scaphoid fractures in children. The multiple soft tissue attachments in this area can lead to variations in fracture pattern, but the most important factor affecting prognosis is whether the fracture involves the articulations of the scaphoid with adjacent bones ( [Fig. 8-79](#)).



**FIGURE 8-79.** **A and B:** Small extraarticular distal fractures of the scaphoid (*arrows*) are likely to be the result of traction forces by the capsular condensations on the volar aspect, or the radial leaf of the scaphotrapezial ligament. **C:** Extraarticular dorsal fractures can likewise be the result of capsular attachment, but the dorsal transverse intercarpal ligament (*open arrow*) also may play a role in their propagation.

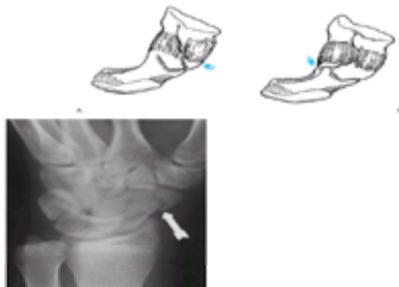
### Type A1: Extraarticular Distal Pole Fractures

Fractures of the distal pole can be either volar or dorsal avulsions; rarely, a direct volar impaction injury occurs. The stout scaphotrapezial ligaments can contribute the tensile force that accounts for most of the fractures. The volar and dorsal capsules also can be a factor. Most extraarticular fractures are volar fractures that probably result from the combined influence of the radial leaf of the scaphotrapezial ligament and the volar capsule acting in tension. The fragments vary in size, and their radiographic appearance is age dependent. The dorsal transverse intercarpal ligament attaches to the distal scaphoid and may contribute tensile forces with wrist hyperflexion. This ligament, usually implicated in dorsal triquetral avulsion fractures, also can cause a fracture of the distal scaphoid.

The tissue condensation on the radial side that has been considered the “radial collateral ligament” may be implicated in more radial distal pole fractures. These tissues are taut in wrist extension and ulnar deviation and may provide an additional force in the development of the volar extraarticular fracture.

### Type A2: Intraarticular Distal Pole Fractures

These fractures may be simply larger fragment counterparts to type IA fractures with an intraarticular component ( [Fig. 8-80](#)). The ulnar leaf of the scaphotrapezial ligament is typically more stout than the radial. It is more difficult to generate the pure tensile forces in this ligament because of its location and the orientation of the insertion to the more sloping articular surface of the distal ulnar scaphoid. If there is more of an element of straight dorsiflexion, or a radial-directed vector force on the thenar area, this complex can be put on tension.



**FIGURE 8-80.** Two variations of a type IB intraarticular fracture of the distal pole of the scaphoid. **A:** The more prevalent type is on the radial aspect of the volar distal scaphoid. This fragment has attached to it the radial leaf of the scaphotrapezial ligament ( *arrow*). **B:** It is rare to have an intraarticular fracture slanted to the ulnar aspect of the scaphoid, but the stout ulnar leaf of the scaphotrapezial ligament may be attached to a fracture fragment ( *arrow*). **C:** Radiograph of an intraarticular distal pole scaphoid fracture. Closed reduction produced an excellent result.

### Type B: Mid-waist Fractures

The waist of the scaphoid can be defined either by dividing the bone into thirds or considering the area bounded by the radioscapocapitate ligament. In any case, fractures of the midwaist can take many forms in children ( [Fig. 8-81](#)). Many of these fractures appear incomplete or at a minimum nondisplaced. Comminuted fractures are rare but can occur with higher energy ( [Fig. 8-77](#)).



**FIGURE 8-81.** Mid-waist scaphoid fractures can differ in their location, orientation, and amount of comminution. All these factors influence the stability and potential healing of these injuries.

Displacement and comminution are other important factors to consider. Significant fracture fragment displacement is rare in isolated scaphoid trauma. When a scaphoid fracture is associated with more extensive carpal injury, such as a fracture–dislocation, displacement is more likely.

Not all mid-waist fractures can be lumped into the same category. An appreciation for the location of the fracture as it relates to the radial and capitate articular surfaces is important in treatment planning ( [Fig. 8-82](#)).



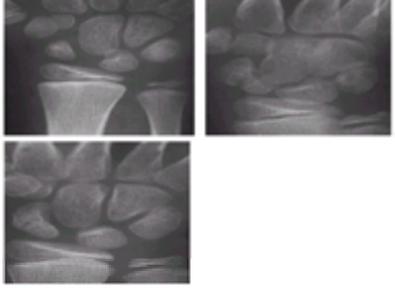
**FIGURE 8-82.** **A:** A barely detectable fracture line is located in the mid-waist of the scaphoid ( *arrow*). **B:** In contrast, this mid-waist scaphoid fracture has occurred at the junction of the middle and distal thirds. Articulation between the scaphoid and capitate has been rendered incongruous by displacement of the distal pole ( *open arrow*). There is mild comminution in the radial aspect ( *closed arrow*).

### Type C: Proximal Fractures

Fractures of the proximal third of the scaphoid and proximal pole present diagnostic and therapeutic dilemmas. Because this area is the last to ossify, radiographs may be difficult to interpret. The tenuous blood supply of this region presents the same problems in children as it does in adults.

## Bipartate Scaphoid Controversy: Traumatic versus Developmental

Louis et al. (136) argue that the concept of the bipartite scaphoid should be dismissed in favor of a traumatic etiology for the radiographic appearance of a dissociated proximal pole (Fig. 8-83). In contrast, Doman and Marcus (60) demonstrated bilateral bipartite scaphoids by MRI in a child with no history of antecedent trauma. These clinicians argued that congenital bipartite scaphoids can exist apart from syndromic conditions, but are less prevalent than Pfitzner's estimate of 0.5% (168). There is also speculation that the abnormal morphology associated with many of the preaxial dysplasias may predispose the usually gracile scaphoid to fracture and possible avascular necrosis (129). The debate continues concerning the existence of a congenital bipartite scaphoid. Bunnell (30) enumerated five criteria that must be met to classify such a variant as a congenital bipartite scaphoid: (a) similar appearance of a bipartite scaphoid bilaterally, (b) absence of historical or clinical evidence of antecedent trauma, (c) equal size and uniform density of each component, (d) absence of degenerative change between the scaphoid components or elsewhere in the carpus, and (e) smooth, rounded architecture of each scaphoid component.



**FIGURE 8-83. A:** This 9-year-old boy fell on an outstretched hand and had significant radial-sided pain and tenderness, but original radiographs failed to reveal any bony abnormalities. It is difficult to judge the scapholunate relationship in the immature carpus. **B:** About 1.5 years later, the patient still had radial-sided wrist pain, and radiographs demonstrated a mid-waist scaphoid nonunion. This would not be considered a bipartite scaphoid, but instead an injury that was sustained while the cartilaginous anlagen persisted. Later ossification demonstrated the bony injury. **C:** After 2 months of casting, the fracture appears to be on its way to union.

### Signs and Symptoms

#### Snuff Box Tenderness

As in adult scaphoid fractures, the primary cause of delayed union or nonunion in a child is delayed diagnosis. Recognition of this fracture is even more challenging in children. Swelling commonly obliterates the anatomic snuff box when the scaphoid is fractured. Tenderness to palpation in this area has long been recognized as a sign of scaphoid fracture. Painful, limited wrist motion accompanies this injury.

#### Trapping the Scaphoid

Another diagnostic maneuver that may prove helpful in isolating injury to the scaphoid is “trapping” the scaphoid between the examiner's thumbs. By placing one thumb in the anatomic snuff box and the other on the distal tubercle of the scaphoid, pain with scaphoid motion can be better assessed. Because many scaphoid fractures occur in the distal region in children, palpation of the volar radial tubercle is especially important.

### Radiographic Findings

Study of radiographs must be complete and meticulous. Malformations of the maturing carpus have been described with certain congenital anomalies (173), and often the scaphoid has more than one potential center of ossification (202). AP, lateral, oblique, and dedicated scaphoid views make up the standard series. Fractures of the mid-waist may be appreciated on all views. Distal pole fractures are typically best imaged on the lateral or pronated oblique view. CT scanning of acute fractures or potential nonunions or malunions of the scaphoid furnishes detailed information about scaphoid morphology (186). The scans are made in the longitudinal axial plane of the scaphoid, and three-dimensional reconstructions can be made if necessary.

### Treatment

#### Presumed Fracture

Even if radiographs of the acutely injured wrist appear normal, application of a thumb spica cast is advocated if the mechanism of injury and clinical examination raise the suspicion of a scaphoid fracture. Occult fractures of the scaphoid occurred in 12% of patients in one series (41).

A long arm thumb spica cast in the initial 2 weeks after wrist injury optimally immobilizes the area and prevents cast slippage or damage in children, known for their propensity to escape immobilization. At clinical follow-up in about 2 weeks, radiographs are obtained out of plaster to assess the presence of a fracture line or the position of fracture fragments. If pain is markedly diminished and there remains no radiographic evidence for a scaphoid fracture, immobilization may be discontinued or changed to a removable orthosis. Gradual motion programs need not be formalized for most children.

If there is persistent pain with normal radiographs, replacement in a cast and repeat examination and radiography can be performed 2 weeks later. A bone scan can be considered; it is a sensitive test but lacks specificity. Bone scans are particularly difficult to interpret in growing children because of the increased uptake about the physes. Furthermore, the invasive nature of the test and the prolonged periods of scanning may be difficult for the child to endure. MRI provides a noninvasive alternative, but motion artifact is again a problem.

#### Confirmed Fracture

If a scaphoid fracture is appreciated at the first clinical follow-up, immobilization can be continued. The debate over whether long arm or short arm immobilization is best continues. Considering a child's activity level and potential lack of cooperation, long arm immobilization may be best. However, if treatment is begun promptly, most scaphoid fractures in children heal within 6 to 8 weeks (41) if immobilized in a short arm cast. Longer periods of immobilization—8 weeks to 4 months—should be considered for fractures that occur in later adolescence or fractures that were initially unrecognized.

Recommended casting positions have included radial deviation with or without flexion (6,223,236). Conversely, ulnar deviation opens the fracture gap in mid-waist fractures. The position of the cast and the joints immobilized are a matter of individual preference and experience with particular techniques. Because most of these different casts furnish roughly the same union rates (around 90%), the importance of the final casted position is probably not paramount. We recommend a thumb spica cast that permits interphalangeal joint motion. Positioning the wrist in slight flexion and radial deviation maximally coapts the fragments.

Including other joints in the scaphoid cast has been suggested. Inclusion of the elbow is controversial. Eliminating forearm rotation is thought by some to neutralize the forces that displace scaphoid fractures (212). However, other investigators considered long arm immobilization unnecessary (4). This issue was largely resolved with the prospective, randomized work of Gellman et al. on the treatment of nondisplaced scaphoid waist fractures in adults (80). This group randomized stable fractures to either long or short arm casting, then changed the cast to a short arm cast at 6 weeks. The long arm group had 100% union, but two of the 23 fractures treated with short arm immobilization failed to heal. The healing time was protracted in patients with short arm casts (9.5 vs. 12.7 weeks).

In children, long arm immobilization is unlikely to cause significant functional debility (or impose restraints that may affect occupation, thus causing economic

hardship), so long arm thumb spica immobilization is a sound initial treatment for these fractures. It is also a much more secure construct.

Inclusion of the thumb interphalangeal joint or other digits (namely the index and long fingers in the three-fingered chuck cast) is seldom practiced today. This is an effective method for eliminating forces about the scaphoid but is cumbersome and difficult to apply.

In one study of adult scaphoid fractures, good results were recorded with dynamic splinting ( [26](#)), but there would be little if any indication for this flexible splint in children.

### **Fractures Presenting Late**

Vahvanen and Westerlund ([216](#)) reported three children with scaphoid fractures recognized late. At the time, these children had bone resorption at the fracture site, although all achieved union after lengthy immobilization (7, 12, and 14 weeks). Segmuller and Schonenberger ( [193](#)) reported three children with delayed union of the scaphoid and significant bone cyst formation, in whom union was obtained with immobilization for several months. Healing of delayed unions in the pediatric scaphoid with lengthy immobilization has been verified by other clinicians and should be the first alternative used for a pediatric scaphoid fracture that is recognized late ( [93](#)).

The favorable biology for bone healing in children is probably the reason for the success of closed treatment of scaphoid fractures, whether initiated in acute fractures or in those that present late. The fact that most scaphoid fractures in children are minimally displaced or nondisplaced enhances the ability to treat these fractures by closed means.

### **Displaced Scaphoid Fracture**

#### **Closed Reduction and Casting.**

There are no reports detailing specific experience with closed treatment of significantly displaced fractures of the scaphoid in children. In the rare displaced pediatric scaphoid fracture, closed reduction should be attempted. Several researchers have described maneuvers to reduce the displaced scaphoid fracture ( [115,132](#)). Traction and ulnar deviation usually extend the distal pole that is probably displaced into a flexed posture, but holding the reduction when the wrist is returned to a more anatomic position with a molded cast is difficult, and there is some risk of skin compromise over the distal pole. McLaughlin ( [144](#)) emphasized the difficulty in obtaining anatomic reduction in his observations of intraoperative fragment relationships with different wrist positions.

Placing percutaneous smooth wires under fluoroscopy may be a more realistic method for closed treatment if reduction is to be effected. The possible collapse pattern of the lunate must be corrected to achieve maximal results.

#### **Open Reduction and Internal Fixation**

If displacement exceeds 1 mm or if there is significant angulation (10 degrees or more) on any of the images during a treatment course for fracture of the scaphoid, open reduction and internal fixation should be considered. There is no role for open reduction without internal fixation ( [144](#)). The implant choice must be individualized, and size is a significant consideration. Smooth wires are very effective in the treatment of scaphoid fractures, including those in children. Smooth Kirschner wires of 0.035 or 0.045 inch are relatively easy to insert and provide good stability. Inserting a Herbert screw or a similar implant is more technically demanding. The ability to use such an implant is based on the size of the fragments and the surgeon's experience.

Mintzer and Waters ([152](#)) reported open reduction and internal fixation of an acute displaced scaphoid fracture in a 9-year-old girl. They elected to operate because of the initial displacement at the fracture site and the flexion (humpback) deformity of the distal fragment. Through the usual volar approach, a Herbert screw was used to stabilize the fracture, which went on to uneventful union; good wrist function resulted.

## **AUTHORS' PREFERRED METHOD OF TREATMENT OF SCAPHOID FRACTURES**

We treat almost all nondisplaced or minimally displaced scaphoid fractures with cast immobilization when there is no associated injury. The indications for open reduction in adults include any fracture displaced more than 1 mm or 10 degrees, and some minimally displaced fractures in special circumstances (e.g., professional athletes, surgeons) ([152](#)). We are not as aggressive in children, preferring instead to treat nondisplaced and minimally displaced complete fractures with casting.

Fractures with more than 1 mm of displacement or angular deformity of more than 10 degrees are considered for more aggressive treatment. Acceptable displacement depends on the level of the fracture and the maturity of the child's bones. The most accepted indications for open reduction and internal fixation of acute fractures of the scaphoid are significant displacement and association with additional carpal injury.

### **Role of Closed Treatment**

For scaphoid fracture treatment, we favor long arm thumb spica immobilization for the initial 4 to 6 weeks, followed by short arm immobilization until clinical and radiographic union occurs. We use long arm immobilization even in presumed scaphoid fractures as an initial treatment: it protects the patient and underscores the potential gravity of the injury.

Radiographs should be obtained in the first 7 to 10 days, then monthly. Clinical examination yields similarly important information about the progress toward union. We have rarely needed tomograms or CT scanning to assess the relative position of fragments, but they may be considered when loss of reduction is questioned.

### **Open Reduction and Internal Fixation**

If we opt to proceed with surgical treatment, fractures of the middle and distal thirds are exposed through a volar approach. The tendon sheath of the flexor carpi radialis is divided on its radial side to protect the palmar cutaneous branch of the median nerve. The tendon is retracted ulnarward as the incision is taken down to the scaphoid through the floor of the sheath. The radioscapocapitate ligament and part of the long radiolunate ligaments are carefully divided and tagged for later repair. The scaphoid fracture is assessed for fragment position, reducibility, and stability after reduction. Bone grafting may be considered if there is extensive comminution. We prefer to obtain this graft from the volar or dorsal distal radius, avoiding the morbidity of iliac surgery.

Once the scaphoid is reduced, the method of fixation can be chosen. Smooth wires (0.035-, 0.045-, or 0.062-inch) are preferred in pediatric scaphoid fractures. At least two wires should be placed to stabilize the fracture and minimize rotational displacement. If the patient is near skeletal maturity, a Herbert screw can be inserted as described by its originator ( [106,107](#)). Pins, if used, can be left under the skin of the thenar eminence or allowed to penetrate the skin. A short arm thumb spica splint is used if the patient is cooperative and the fracture was well stabilized. If a bone screw was used, early motion can be started at the second to fourth week, with interval splinting for protection. When smooth wires stabilize the fracture, rigid immobilization is continued until healing is demonstrated on radiography.

Rare proximal pole fractures can be approached through the same volar approach or a dorsal incision based over the anatomic snuff box. The scaphoid can be delivered into the interval between the first and third dorsal compartments by ulnar deviation of the wrist. The radial artery and its branches are vulnerable on this approach and must be protected ([Fig. 8-84](#)).



**FIGURE 8-84. A:** A 15-year-old male athlete sustained a proximal pole scaphoid fracture that was minimally displaced. **B:** After long discussion with the family, it was decided to perform open reduction and internal fixation. This was performed through a dorsal approach with Herbert screw fixation. Excellent results were obtained. (Courtesy of James E. Culver, Jr., M.D.)

The dorsal approach is particularly useful in surgery for proximal pole fractures and nonunions. We have expanded the use of this approach to include selected patients with more distal fractures that are minimally displaced. The morbidity from the dorsal exposure can sometimes be less than that of the volar route; experience with patient selection and the geometry of the scaphoid are prerequisites for using this approach.

#### Follow-up Care and Rehabilitation

The protocols for immobilization with closed and open treatment have already been detailed. After the cast is removed, a program may be started that emphasizes recovery of a full range of painless motion before embarking on any strengthening. For young patients immobilized less than 2 months, it usually is unnecessary to use formal therapy modalities. For some adolescents, and if an operatively treated fracture was accompanied by other injuries about the carpus, some formal rehabilitation may be needed. There is little role for dynamic splinting.

Continued follow-up with radiographs and clinical examinations ensures that no carpal collapse pattern is present and that avascular necrosis of the scaphoid has not complicated the fracture course.

If the child or adolescent is amenable, we remove the smooth wires in the office under local anesthesia. We have not found it necessary to remove a Herbert screw in any patient who has a united fracture, but the implication of long-term indwelling metal is unknown.

#### Complications

##### Nonunion

**Nonoperative Treatment.** Scaphoid nonunion is distinctly rare in children, but reports of this complex entity do exist ([41,55,134,139,163,170,202](#)). About 20 nonunions have been reported in the world literature, with the largest series detailing eight cases. This paucity of material emphasizes the rarity of this complication and also limits our understanding of treatment methods. There is convincing evidence that immobilization, lengthy if necessary, is the cornerstone of treatment of scaphoid nonunions in children ([Fig. 8-85](#)). The evaluation of serial clinical and radiographic parameters must be thorough, and tenderness about the scaphoid and continued radiolucency may persist even after union has been achieved ([124](#)).



**FIGURE 8-85. A:** A 10-year-old boy was first seen after a fall on the outstretched hand. The radiograph shows an old fracture of the waist of the scaphoid. There was no definite history of an old injury. **B:** The fracture went on to solid healing after 9 months of immobilization. Motion and strength of the wrist were normal. (Courtesy of Norman H. Higgins, M.D.)

**Operative Treatment.** Open reduction, bone grafting, and internal fixation are the standard procedures for treatment of scaphoid nonunions in adults and also have been used in children ([134,139,170,202](#)). The volar approach is typically used, along with autogenous bone graft and internal fixation ([Fig. 8-77](#)). The concepts of operative scaphoid nonunion treatment in children are similar to those in adults and are familiar to practitioners who do scaphoid surgery ([Fig. 8-86](#)). However, the technical demands of the procedure may be amplified because of the patient's small bone size. This may be a factor in the choice of implant, and smooth wires usually are better suited than a scaphoid screw.



**FIGURE 8-86. A:** A 15-year-old boy developed a nonunion of the scaphoid. A thumb spica cast for a period of months failed to bring about union. The cyst at the mid-waist grew. **B:** Formal takedown of the nonunion via a volar approach plus intercalary bone grafting with Herbert screw fixation permitted union and had excellent function.

**Sequelae.** The altered kinematics that predispose the adult wrist to accelerated degenerative change after scaphoid nonunion, scapholunate ligament injury, or scapholunate advanced collapse (the SLAC wrist) ([137,222](#)) have never been demonstrated in the pediatric wrist. Studies detailing bipartite scaphoid when

radiographic union was not demonstrated after fracture suggest that only minimal clinical symptoms have been present at long-term follow-up ( [124,136](#)). Therefore, it is unknown how aggressively scaphoid nonunion should be treated in children.

Most patients have tenderness to palpation about the scaphoid in the snuff box or over the volar tubercle. Motion between the fragments is likely to be demonstrated on fluoroscopy. However, sometimes there is a strong cartilaginous bridge between two ossification centers that were separated by a traumatic injury. This may permit normal motion of the scaphoid in daily activities and possibly with even higher demand occupations. If this is true, the morbidity of bone grafting and open reduction must be balanced against the potential for improved results. Our bias is decidedly against leaving the child with an established nonunion.

We probably will never have sufficient data to provide meaningful conclusions. Natural history studies would be necessary to resolve this issue, and the rarity of scaphoid nonunion probably makes this unfeasible. With the refinement of MRI as a diagnostic tool, fractures in the immature carpus may be better diagnosed ( [124](#)), and we may see a greater number of defined chondral injuries that can be studied.

### **Pathologic Fracture**

Nonunion of a pathologic fracture has been reported in an adolescent girl. As a 7-year-old, she had osteomyelitis of the scaphoid that was treated by operative drainage and intravenous antibiotics. She was asymptomatic for 6 years, and then wrist pain recurred with activity. Nine years after the original episode, she presented with a frank nonunion of the scaphoid. After two operations to gain union, without the complication of infection in either, the patient went on to have a good result. In retrospect, evaluation of the initial radiographs demonstrated a loss of normal density of the scaphoid with disturbance of the normal trabeculation.

### **Avascular Necrosis**

Idiopathic avascular necrosis of the scaphoid, similar to Preiser's disease of the adult carpus, appears to have no counterpart in children. One child with radial hypoplasia demonstrated changes consistent with avascular necrosis in a very gracile scaphoid that was thought to fail under normal compressive loads ( [129](#)). The patient had significant pain and was treated with scaphoid excision and interposition arthroplasty.

### **Capitate Fractures**

Impaction or compression fractures of the capitate have been reported by several researchers ( [129,217](#)). A hyperdorsiflexion type of mechanism causes abutment of the capitate waist against the lunate or dorsal aspect of the radius. Isolated fractures of the capitate are rare and usually result from high-energy trauma ( [82,239](#)). They can be diagnostic challenges: the proximal fragment of the capitate can rotate 180 degrees, thus presenting a confusing radiographic picture in the immature wrist. In an adult, it usually is easier to determine that such a displacement has occurred, but in a child whose ossification is incomplete, the malposition may be more difficult to ascertain. Radiographs of the contralateral side are needed when attempting to evaluate some of the more complex injury patterns about the pediatric wrist.

### **Naviculocapitate Syndrome**

The naviculocapitate syndrome is well described in adults ( [217](#)), and this same combination, originally described by Gouldsbrough ( [86](#)), also has been seen in children. Anderson treated two 13-year-old patients with scaphoid-capitate fractures in combination with distal radial fractures ( [7](#)).

### **Associated Fractures Common**

More commonly, fracture of the capitate occurs with fractures of other carpal bones ( [Fig. 8-87](#)). Typically, the scaphoid is injured by forces transmitted in a dorsiflexion, ulnar deviation, intercarpal direction through the radial column ( [7,86](#)).



**FIGURE 8-87.** A 12-year-old boy sustained multiple carpal fractures when a heavy weight crushed his wrist. Sixteen months later, he had an established nonunion of the capitate that required bone grafting to obtain union. (Courtesy of James H. Dobyms, M.D.)

### **Treatment**

Treatment of a capitate fracture depends on the character of the particular injury, as well as the trauma sustained by surrounding tissues. Obtaining a film of the wrist in distraction might be one of the most valuable methods for determining whether an injury to the intercarpus has occurred. Before full ossification has been completed, small osteochondral fragments in the mid-carpal region may indicate a greater arc injury or isolated capitate fracture. MRI may be a useful adjunct in these complex injuries.

### **Nonoperative Treatment**

If the displacement of the capitate, or even the capitate and the scaphoid, is minimal, 6 to 8 weeks of immobilization is an effective means of treatment ( [7](#)). In one report of simultaneous scaphoid and capitate fracture, closed reduction and immobilization proved to be acceptable treatment for the bony injuries, but the position of radial deviation and extreme flexion necessary to maintain this reduction caused a compression neuropathy of the median nerve in a child, requiring decompression ( [45](#)).

### **Operative Treatment**

If displacement of the proximal pole of the capitate is significant, open reduction with or without internal fixation should be considered. These anatomic relationships must be restored for the mechanics of the carpus and to avoid future problems of ischemic necrosis of the proximal pole. Nonunion of the capitate has been reported in a significant crush injury to the hand of a 13-year-old girl. Bone grafting was performed about 1.5 years later with good success ( [149](#)). We are aware of no literature detailing avascular necrosis after capitate fracture in children.

### **Triquetral Fractures**

Small triquetral avulsion fractures are common in adults. It is likely that they are so commonly seen by practitioners dealing with hand injuries that a true incidence or prevalence could never be determined. This injury can happen in the maturing carpus, especially as ossification nears completion. The probable mechanism is an avulsion force from the dorsal ligament structures ( [Fig. 8-88](#)).



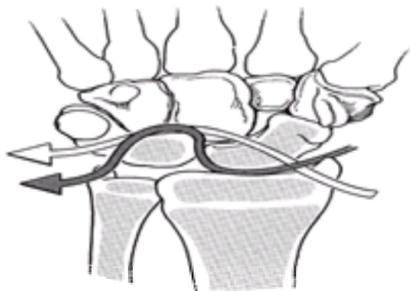
**FIGURE 8-88.** A minimally displaced dorsal triquetral avulsion fracture ( *arrow*). These can be treated with short-term immobilization.

#### **Extremely Rare Injury**

Although there are no published papers specifically reporting this injury in children, its treatment should be conservative, as in the adult wrist. A short period of immobilization with icing is likely to be enough symptomatic treatment to permit early motion. O'Brien (E.T. O'Brien, personal communication) observed a triquetral body fracture in a child that required open reduction and internal fixation.

#### **Associated with Global Injuries**

A significant fracture to the triquetral body also can occur with a more global carpal disruption. The path of the greater arc ( [140](#)) can go through the triquetrum ([Fig. 8-89](#)) ([45,124](#)). If a transtriquetral fracture is seen, attention must be directed toward the radial styloid, scaphoid, and mid-carpal joint as well. Again, distraction films may be helpful. Closed treatment for some of these complex disruptions, including the triquetrum, is effective.



**FIGURE 8-89.** The greater arc (*white arrow*) is associated with fractures of the carpal bones, which may include the scaphoid, lunate, capitate, hamate, and triquetrum. In this depiction, the radial styloid also has been fractured, creating a trans-styloid pattern. The dark arrow depicts the lesser arc, in which forces are transmitted through soft tissue structures only, resulting in progressive perilunar instability. Numerous varieties of injuries associated with these patterns of forced transmission can be seen. (Reprinted from Mayfield JK, Johnson RP, Kilcoyn RK. Carpal dislocations: pathomechanics and progressive perilunar instability. *J Bone Joint Surg [Amj]* 1980;5:226–241; with permission.)

#### **Hamate Fractures**

Ali ([5](#)) reported on a 16-year-old boy with a fracture of the hamate body and a pisotriquetral dislocation. The patient also had an evolving compartment syndrome of the volar forearm from the significant crush injury. The patient underwent open reduction and internal fixation for the bony lesions, but an ulnar nerve palsy persisted 3 months after the surgery.

There are no specific articles detailing fractures of the hamulus in children, but such fractures can occur in someone nearing skeletal maturity. Those who play racquet sports or golf would be specifically susceptible. The evaluation and treatment of this entity in children may differ from those in adults. Early excision may be too aggressive in a skeletally immature patient, and closed treatment should be favored initially.

#### **Pisiform Fractures**

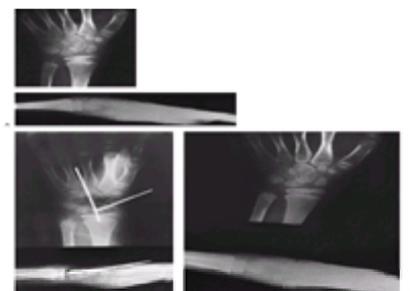
There are no specific discussions of pisiform fractures in children in the literature. Direct trauma causing a comminuted fracture or a flexor carpi ulnaris avulsion injury may occur in late adolescence. Most of these fractures can be treated symptomatically, as in adults.

#### **Lunate Fractures**

Blount described a fracture–dislocation of the lunate in association with a crushing type of physeal fracture of the distal radius in a 12-year-old boy ([23](#)). Open reduction of the lunate through a volar approach (without internal 7 necessary), as a growth arrest of the radial physis resulted from this complex fracture mechanism.

#### **Perilunar Injuries**

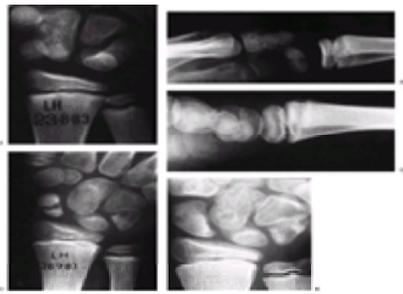
Perilunar injuries with or without fractures of the carpal bones have been reported by several researchers ([Fig. 8-90](#)) ([41,45,166](#)). Because of the unique chondroosseous nature of the maturing lunate, a small osteocartilaginous fracture may go unrecognized on radiography and heal uneventfully. Otherwise, lunate fractures in children are decidedly rare.



**FIGURE 8-90. A:** This 6-year-old boy was first seen with a dorsal perilunar dislocation 6 months after falling downstairs. He had been treated with steroids by a pediatrician because of a presumptive diagnosis of juvenile rheumatoid arthritis. **B:** Open reduction and pin fixation was performed through a dorsal incision 6 months after injury. **C:** Seven months later, most of the displacement had recurred, but motion was quite good and the patient had no pain. (Courtesy of William F. Benson,

M.D.)

The pattern of progressive perilunar instability can occur in a child ( [140](#)). In some of these patients, a scaphoid fracture is the focus, and an appreciation for the force transmission through the wrist will be lost. In these trans-scaphoid injuries, soft tissue injury to the intrinsic ligaments must be suspected and appropriate diagnostic and therapeutic interventions instituted ( [Fig. 8-91](#)). Transcarpal dislocations are discussed later in this chapter.



**FIGURE 8-91.** **A and B:** A 9½-year-old boy sustained a trans-scaphoid perilunate dislocation of the wrist when he fell 60 feet off a viaduct. **C and D:** Closed reduction was performed 8 days after injury because the correct diagnosis had initially been unrecognized. Plaster immobilization was maintained for 4½ months because of transient avascular necrosis of the proximal pole and delayed healing. **E:** Three years after injury, there was slight irregularity of the proximal pole, but the patient had normal function. (Case courtesy of C.L. Colton.)

### Lunatomalacia or Kienböck's Disease

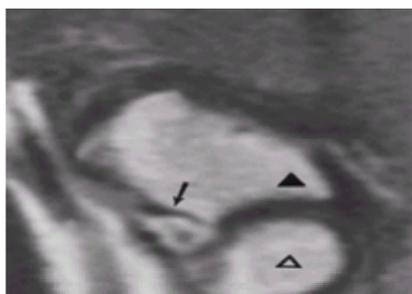
An analogue to the adult Kienböck's disease or lunatomalacia has been observed in children under 10 years of age ( [Fig. 8-92](#)) ([16,177](#)). It was unclear in these reports whether a history of trauma preceded the clinical picture. The symptoms were vague and of low grade, and radiographs revealed only mildly increased density of the lunate with no change in external morphology. Advanced imaging studies such as tomography, MRI, or scintigraphy were not used to evaluate these patients. Immobilization for up to 1 year has been used with success to treat this Kienböck's variant in children. The lunate itself has remodeled in satisfactory fashion; furthermore, if there is altered lunate architecture, usually there are few symptoms, and good motion recovery is expected.



**FIGURE 8-92.** A 12-year-old boy sustained a wrist injury and developed a small area of segmental rarefaction in the lunate, consistent with avascular necrosis (*arrow*). This may represent a pediatric form of adult Kienböck's lunatomalacia. (Courtesy of Greg Goldsmith, M.D.)

### Trapezium Fractures

A fracture of the trapezium is extraordinarily rare in adults or children. Direct palmar trauma may cause a trapezium ridge fracture. A dorsal impaction fracture may result from a hyperextension injury ( [Fig. 8-93](#)), or a small ligament avulsion can be seen. In general, immobilization and symptomatic treatment are adequate.



**FIGURE 8-93.** A 15-year-old girl sustained a nondisplaced scaphoid fracture that healed uneventfully with closed treatment. She continued to complain of dorsal wrist pain 10 months after the injury. Bone scan revealed increased uptake in the area of the distal scaphoid and proximal triquetrum. No plain radiograph abnormalities could be appreciated. A limited magnetic resonance imaging scan demonstrated a nonunion of an intraarticular fracture of the trapezium (*arrow*). The scaphoid is identified by an open triangle, the triquetrum by a closed triangle. Excision of the fragment brought pain relief, and normal function returned.

### Soft Tissue Injuries About the Carpus

#### Ligamentous Injuries

Ligamentous injuries about the pediatric wrist are even more uncommon than osseous injuries. The special viscoelastic properties of the carpal structures probably provide protection from catastrophic ligament injuries in the immature wrist. Also, the difficulties with physical examination and radiographic interpretation may disguise some injuries.

Fracture–dislocations of the pediatric wrist have been described ( [2,166](#)). These were not subtle injuries: they were easily detectable and resulted from high-force mechanisms. These children, 10 and 12 years old, probably had bony and soft tissue makeup approximating that of an adult wrist and also sustained enough trauma to manifest this combined injury. Another combined injury about the wrist was reported by Giddins and Shaw ( [83](#)). In this case, lunate subluxation was associated with

a distal radius physeal fracture. Instability of the lunate was initially recognized and treated surgically 6 weeks after the injury by repair of the dorsal ligament and pin fixation of the lunate.

There are a few reports of isolated ligamentous injuries of a child's wrist. Gerard (81) reported a 7-year-old girl who sustained only low-energy injuries from a fall from a standing posture at 3 months of age. The parents related that the child excluded the affected hand from normal activity, and a painful limitation and clinical deformity of the wrist were present at the time of evaluation at 7 years. A volar intercalated type of instability with carpal collapse was present when the radiographs were reviewed. The distinct pattern was absent on the contralateral uninjured side. Operative reconstruction was chosen, using an extensor carpi radialis graft and pinning. Although follow-up was brief, clinical and radiographic results were considered excellent.

Hyperelasticity syndromes may predispose patients to aberrant carpal mechanics. Atraumatic bilateral perilunar dislocation patterns have been observed in association with Marfan's syndrome (167). A complete examination of the patient to determine whether global hyperlaxity is present would be useful in evaluating wrist findings that seem out of the ordinary. Films of the contralateral extremity also can provide vital information. True intercarpal and radiocarpal ligamentous injuries are rare in children, but the diagnosis should still be considered if clinical findings suggest it. Despite the infrequency of these injuries, clinicians may find themselves questioning an apparent subtle widening or malalignment in the carpus. An appropriate index of suspicion, experience, and judicious use of ancillary studies (e.g., arthrography, stress radiography, and real-time fluoroscopic evaluation) are advocated.

If several factors in the history and examination point to potential ligamentous injury, short-term immobilization and reexamination are appropriate. Often there is a marked diminution in clinical symptoms, and uncomfortable intervention is avoided.

The goals of a mobile, painless wrist and the prevention of accelerated degeneration within the carpus are most often met by a systematic evaluation and treatment process. If aggressive intervention is necessary, the timing may have to be considered.

It is difficult to make a general recommendation about treatment for these extremely rare injuries. Some difficult decisions would have to be made in, for instance, a late adolescent athlete with an acute scapholunate injury. We would probably treat these patients as adults, with anatomic reduction, repair of soft tissues, and internal and external immobilization as necessary until healing occurs. Some formal rehabilitation may be needed to recover motion and strength. If ligamentous disruptions occur in an extremely young patient, it is not unreasonable to permit the remodeling potential of the immature carpus and the innate ability of children to regain useful and painless range of motion to work in the child's favor.

### **Distal Radioulnar Joint Disruption**

Dislocation of the distal radioulnar joint also is rare in children, although with higher levels of athletic participation, we may start to see more open soft tissue destabilizing injuries (100). Fracture through the ulnar physis occurs with some frequency, and this route of force transmission may be somewhat protective to the distal radioulnar joint and triangular fibrocartilage complex (120,128). The pediatric Galeazzi equivalent is a fracture of the radius accompanied by a fracture of the distal ulnar physis that destabilizes the distal radioulnar joint. Galeazzi fractures and their equivalents are discussed in Chapter 9.

Acute lesions of the triangular fibrocartilage complex are rare in childhood. If there is a high index of clinical suspicion for such an injury in a child, arthrography may be used to confirm the diagnosis. Cadaver dissections by Mikic (147) failed to demonstrate triangular fibrocartilage changes of a degenerative nature in any specimen under 27 years of age. Although this information may not be directly applicable, it does lower the likelihood of a significant triangular fibrocartilage complex injury, with or without distal radioulnar joint disruption.

A radiocarpal dissociation from the distal ulna usually is a catastrophic injury that results from high-energy mechanisms. Subtle injuries also can occur; comparison radiographs and clinical examination are important steps in evaluation. Many of these less significant injuries can be treated with immobilization in full supination, which reduces the distal radioulnar joint.

### **Athletic Injuries of the Immature Wrist**

Injuries to the upper extremity have been largely neglected in sports medicine because of the focus on catastrophic, high-profile lower extremity injuries. However, as our young patients participate with greater sophistication at earlier ages, the number of wrist injuries is increasing. This is especially true in high-velocity, hand-intensive sports, where the demands on the hand and wrist are the greatest (17,56).

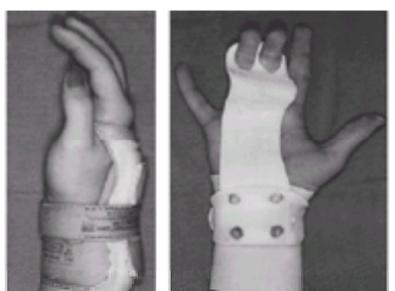
Significant injuries to the wrist are underrecognized and can be undertreated unless the physician is familiar with these pathologies. In addition, the health-care professional attending athletic contests or evaluating the injured player in the early period must temper the athlete's and coaches' desire for him or her to continue play if the extent of the injuries may make this unfeasible. Acute injuries to the wrist can occur as a result of direct contact from a ball, high-energy collisions, and attempts to brace against opponents or falls onto the turf. Fractures of the carpal bones, ligamentous injuries, or a combination injury may result.

Conversely, there is increasing recognition of the effect of long-term, repetitive exposure to particular activities specifically involving the wrist. The stresses that high-performance gymnastic maneuvers place on the radial physis have been discussed and characterized in the literature (38,179,183,184). Similar physeal injuries have been reported at the distal ulna as a result of gymnastics (238). The clinical and radiographic manifestation is not unlike an "acquired Madelung deformity," as reported in one patient by Vender and Watson (219). Management is described in Chapter 9.

Whether a sports injury to the wrist is acute or secondary to repetitive loading, a sensible approach that includes input from the patient, parents, and coaches is advocated. We strongly recommend using the traditional and tested methods of treating injuries and try to disregard any outside influences that may be at work forcing a more aggressive approach to these young athletes. The definitions of *aggressive* and *conservative* are sometimes clouded in this population. Some argue that internal fixation of a minimally displaced scaphoid fracture in a high-performance athlete is a safe and predictable method that brings about rapid union and returns the patient to the previous level of athletic involvement. However, we do not advocate such an approach in children.

There may be some debate about scholarship candidates who have little growth remaining before their final season of high school sports. These decisions must be individualized, and the patient's desires and the particular aspects of the sport are important factors to be considered.

Physicians, parents, and coaches can work together to effect significant change in the realm of education and safety. It is important to have proper coaching, conditioning, and equipment when teaching young athletes. This applies to both team activities and individual sports. Proper use of safety equipment is necessary and beneficial. Such equipment includes materials for preventing injury to the upper extremity, such as the wrist guards used in in-line skating or the hand and wrist protectors used in gymnastics (Fig. 8-94). Innovative types of immobilization and protection that are approved by rules committees of the particular level of play, whether it be the local school board all the way up to the NCAA or NFL, are used. The concept of the playing cast has been well studied and is an excellent adjunct to return athletes safely to the field or court while minimizing the danger to themselves and other participants.



**FIGURE 8-94.** Wrist guards for gymnastics. **A:** The "lion's paw" protector used mainly for vault. **B:** Hand and wrist protectors used primarily for the uneven parallel bars.

## AUTHORS' PREFERRED METHOD OF TREATMENT

Distal pole fractures occurring in preadolescents are treated symptomatically. Short-term (2–3 weeks) splinting or casting is usually adequate.

Our experience with teenaged athletes has forced us to contemplate an emerging subset of carpal injuries—the “typical” scaphoid waist fracture in a skeletally immature patient. If displaced (1 mm or more of fracture gap or 10 degrees of angulation), open reduction and internal fixation are indicated. We favor a variable pitch screw inserted through a volar approach, after anatomic reduction. For some fractures in the proximal third, a dorsal approach for insertion can be used.

If there is a mismatch between the size of the scaphoid, or the fracture fragment, and the prospective implant, then smooth wires can be used for stabilization. The same concepts of approach and implant insertion hold.

## DISLOCATIONS OF THE HAND AND CARPUS

### Dislocations of the Interphalangeal Joints

In children, the soft tissue stabilizers of the interphalangeal joints (the collateral ligaments, the inserting tendons, and volar plate) exceed the physeal tissue in strength and ability to withstand pathologic forces. It is for this reason that failure through the bony elements is much more common in this age group. Occasionally, dislocations do occur, as do fracture–dislocations ([Fig. 8-95](#)).



**FIGURE 8-95. A:** A 15-year-old boy sustained a dorsal dislocation of the proximal interphalangeal joint with a Salter-Harris type II fracture of the middle phalanx when he jammed his little finger playing baseball. **B:** Closed manipulation successfully reduced the dislocation and the fracture.

The specific anatomy and interrelationships of the bones and soft tissues in the pediatric hand have been extensively discussed earlier in this chapter. We refer the reader to these sections for a thorough description of the insertions of the volar plate, collateral ligaments, and musculotendinous units about the interphalangeal joints.

### *Interphalangeal Joint Dislocations*

#### The Distal Interphalangeal Joint

A hyperextension force or axial load on a slightly flexed terminal phalanx can result in dislocation of the DIP joint without fracture. Most soft tissue disruption is volar and lateral, resulting in the distal phalanx coming to rest in a position dorsal to the middle phalanx. The collateral ligaments and volar plate typically fail at their middle phalangeal origin.

Most of these pure dislocations can be easily relocated by longitudinal traction and recreation of the hyperextension force, followed by the terminal reduction maneuver. The DIP joint congruity is assessed clinically and with radiographs. Two to three weeks of splinting, similar to the splinting used for mallet injuries, will allow the ligaments to heal. Motion recovery usually is full, and late instability is rare.

Irreducible or complex dislocations of the DIP occur almost exclusively in adults, but some clinicians have included immature patients in their series ([164,171,185,194](#)). The reports that have included younger patients have lacked specific details of operative findings, and have mixed pure soft tissue injuries with fracture–dislocations ([210](#)). However, some important facts have been common to most reports:

1. The distal phalanx is dorsal and blocked from reduction by the interposed volar plate, having been avulsed from the middle phalanx. Other tissues that may be interposed are the collateral ligaments and the flexor digitorum profundus ([211](#)).
2. Relocation of the complex dislocation, performed through a dorsal approach, usually requires division of at least one collateral ligament, removal of the interposed volar plate, and relocation of the flexor digitorum profundus.
3. Pinning of the joint is an option for a period of 3 weeks until soft tissue healing is adequate to start early motion. No late instability was reported, and motion recovery was uniformly satisfactory.

#### The Proximal Interphalangeal Joint

Injuries of the PIP joint are relatively rare in children, in comparison with their occurrence in adults. Bony injuries about the PIP joint garner the most attention because of their potentially disastrous results, but injuries to the soft tissues often lead to persistent swelling, instability or stiffness, and tendon imbalance.

**The Jammed Finger.** The jammed finger or “coach's finger” is a popular term in the lay community used to describe the swollen digit resulting from an axial loading injury with variable angulatory and rotational forces. The pain and deformity usually are localized to the PIP joint, and flexion is difficult. The pediatric patient often has trouble localizing the discomfort to the volar, lateral, or dorsal aspect of the joint. By the time the physician has seen the patient, several coaches or parents have already tried to “pull it back in place.”

At first evaluation, little more is known about the pathoanatomy than it is primarily focused at the PIP joint. The history should include questions about the mechanism of injury, and whether there was a perceived deformity of the digit that was reduced by manipulation.

Radiographs will reveal a fracture, if present, but often are unclear with respect to the skeleton. Diffuse soft tissue swelling is appreciated. Care must be taken to study the congruity of the reduction at the PIP joint. A loss of concentricity can be quite subtle, evidenced by a dorsal V space instead of a smooth articular mating.

Evaluation under metacarpal block anesthesia is helpful in determining the integrity of the soft tissue structures about the PIP joint. A stress examination of the collateral ligaments can be conducted. Passive range of motion can be recorded painlessly, and active motion can be assessed.

A closed boutonniere deformity is inferred when active extension from a flexed posture is impossible, but extension can be maintained if the digit is passively placed at neutral. Other provocative maneuvers for diagnosis of an extensor mechanism injury include a test of central slip continuity described by Elson ([66](#)). With the patient's digit flexed 90 degrees at the PIP joint over a table edge, inability to extend the PIP joint coupled with fixed extension of the DIP joint indicates a central slip

rupture.

For many of these presentations, no specific pathologic entity is defined. The soft tissue swelling and vague pain can persist for nearly a year, and the affected PIP joint may always be slightly more voluminous. Radiographs typically show no bony sequelae or accelerated degeneration, although cartilage or bone bruise is a likely part of the injury.

Treatment of the “jammed finger” consists of initial icing and elevation for 72 hours, with immobilization in a hand-based splint to permit rest. Edema reduction and motion should then follow, and warm soaks may bring some relief. Buddy taping to the adjacent digit protects the injured member and promotes earlier motion recovery.

A documented central slip rupture is treated with extension splinting for four weeks, followed by dynamic extension splinting for 2 to 4 weeks. The splint permits motion, while preventing volar subluxation of the lateral bands. Rarely, operative intervention is required for closed extensor mechanism injuries. Primary tendon repair, with or without joint pinning, usually is reserved for special circumstances. The greater strength of the flexor surface muscles usually allows recovery of flexion, even after several weeks of extension splinting.

**True Pip Dislocations.** PIP dislocations can be dorsal, volar, or lateral. Although a significant number of these injuries are incomplete ligament avulsions or intrasubstance tears, combined with a spectrum of injuries to the volar plate, some may present as irreducible joint dislocations.

**Dorsal PIP Dislocations.** Dislocations about the PIP joint in which the middle phalanx is displaced dorsal to the proximal phalanx are the most common ( [Fig. 8-96](#)). Failure of the collateral ligaments and the volar plate are all a part of the pathogenesis of the dislocation. These structures may even block attempts at reduction, creating a rare irreducible dorsal dislocation.



**FIGURE 8-96. A:** Lateral radiograph of a volar plate avulsion injury. There is a small fracture fragment volarly from the epiphysis of the middle phalanx. Note the associated soft tissue swelling. These fractures should not be treated with prolonged immobilization. Early buddy taping and motion lessen the risk of a flexion contracture. **B:** Dorsal dislocation of the proximal interphalangeal joint in a 10-year-old girl.

Many dorsal dislocations are better termed subluxations or sprains, because the injury to the fibers of the collateral ligaments does not result in frank loss of joint congruity or cause instability of the articulation through a normal arc of motion. In fact, many of these injuries either go unseen by the physician or are seen after they are reduced by the patient or other attendant. Many of these reductions take place on the playing field or gymnasium, and the patient returns to an athletic contest.

With a dorsal dislocation, neurovascular status typically is normal, but pain and deformity are obvious. Fracture is ruled out with standard radiographs, with the addition of an oblique film to better assess the condylar area. After adequate anesthesia, the dislocation is reduced with longitudinal traction and hyperextension, followed by gentle flexion of the middle phalanx back onto the proximal phalanx articular surface.

The quality of the reduction and the stability of the joint must be assessed. Radiographs should demonstrate a concentric reduction. Most dislocations are stable throughout the normal range of motion, but others may redislocate as the PIP joint is brought into more extension. Eaton ( [65](#)) described the active stability test to determine the adequacy of reduction and the need to potentially treat by operative means. Simply asking the blocked and reduced patient to perform active flexion and extension to determine stability is the cornerstone. If the joint remains stable in this arc, then 2 to 3 weeks of buddy taping are adequate.

Extension-block splinting, as advocated by McElfresh et al. ( [142](#)), may be needed for fracture–dislocations. Our regimen for splinting of injuries that are reducible and stable in at least some range is detailed below:

1. For a completely stable joint with no lateral instability or volar plate laxity, we allow early motion with buddy taping to the side of the greatest lateral tenderness.
2. For a closed reduction that is stable, yet associated with a degree of lateral instability (<20 degrees) or volar plate laxity (<20 degrees hyperextension), splinting of the PIP joint for 2 to 3 weeks in about 30 degrees of flexion is the initial treatment. Buddy taping can continue for 2 to 3 additional weeks.
3. For significant volar plate injury without lateral instability, a greater degree of PIP joint flexion is used for about 3 weeks. We typically splint at 5 degrees less than the maximal extension at which the joint starts to dislocate; this is usually between 45 and 60 degrees. The joint is extended slowly every 7 to 10 days until full extension is realized at the third or fourth week. The joint has been left free to flex throughout this treatment course. Bowers ( [29](#)) has reported development of a swan neck pattern in two children with volar plate rupture after dorsal dislocation, and both needed volar plate repair. More aggressive early dorsal block splinting may permit healing of the injured plate and prevent such a deformity.

There is considerable disagreement concerning primary repair of torn collateral ligaments associated with PIP joint dislocations in adults ( [135,141,180,188](#)). With the paucity of experience or reports of this pure ligamentous injury in children, even less of a consensus exists. Redler and Williams ( [180](#)) did include four skeletally immature patients in their series of 14 complete collateral ligament ruptures. They reported complete functional recovery. Blount ( [23](#)) and Vicar ( [220](#)) described a patient in whom the collateral ligament was interposed in the PIP joint and had to be extracted to permit reduction. In Vicar's patient, the injury was several weeks old, but the open repair was successful.

Other researchers ( [28,232](#)) described a tumescence at the site of the collateral ligament avulsion (from its proximal phalangeal origin) that has been bothersome to patients and may cause functional limitation. These fibrocartilagenous or fibroosseous masses have been excised with uniform success. Another peculiar injury was reported by Whipple et al. ( [228](#)) in which the volar cartilage surface of the middle phalanx was folded into the joint, in a dorsal fracture–dislocation. The cartilage flap was easily restored, and excellent function was realized within a month.

We have been very pleased with simple closed reduction, coupled with either short-term immobilization in an extension block splint or simple buddy taping. If the dorsal dislocation cannot be reduced, evacuation of interposed tissue is necessary. When the joint is to be opened for reduction, we primarily repair the collateral ligament(s) with nonabsorbable suture. However, we reserve this treatment for unstable joints, because we have found that postreduction instability is rare, because most of these patients tend toward stiffness.

The “gray area” between open and closed treatment of these injuries is when reduction can be accomplished, but significant instability is present. In this young population, we still make an attempt at closed treatment if the joint can be reduced. If the joint needs to be splinted in more than 60 degrees of flexion to remain stable, we advocate primary ligament repair. We approach the digit through a mid-axial incision on the side of the collateral ligament rupture. Secure primary repair or suture to bone is accomplished. Pinning of the joint is avoided if possible. Motion is begun at the second week.

Dynamic splinting may be necessary for terminal motion recovery. The fibrous masses that have been described above usually present little impediment to motion recovery. If the mass is troublesome, it can be excised after the other tissues have stabilized. Waiting about 6 months may allow the surgeon to combine a capsulotomy or tenolysis, if needed, with the excision.

**Volar PIP Dislocations.** Although volar PIP dislocations are uncommon in the pediatric population, there are common themes that can be appreciated from the few reports ([111](#),[160](#),[165](#)):

1. These injuries often go undetected. The delay in diagnosis ranged from 1 to 52 months in one series ([165](#)), and the single patient reported in another series was 3 weeks old ([160](#)).
2. Interposition of soft tissues (the volar plate, lateral band) can hamper attempts at closed reduction. Likewise, fracture fragments can render the dislocation irreducible by closed means ([111](#)).
3. Long-term results are often suboptimal. It is difficult to ascertain whether the late treatment is the ultimate cause, or contributing factors like the violence of the initial trauma or extent of tissue involvement also may play a role.

Volar dislocation is an extensor-side injury. The interval between the lateral band and the central tendon may be the site for herniation of the proximal phalangeal head, or the central slip may rupture from its middle phalangeal insertion. Thompson and Eaton ([213](#)) recommend 3 weeks of extension splinting followed by intermittent dynamic splinting for patients in whom a congruous reduction is obtained. If the reduction is blocked by soft tissue, exploration may be necessary. We approach the digit from the dorsum, and exploit the injury interval to evacuate the joint. Careful reconstruction of the extensor tendon is performed, and splinting in extension is continuous for 3 to 4 weeks. The flexion typically is regained without too much difficulty over the course of the next 6 weeks. Some dynamic splinting can be used to assist or accelerate motion recovery.

**Lateral PIP Dislocation.** Garroway et al. ([77](#)) emphasized the role of torsional forces in the development of complex dislocations about the PIP joint. Although his five classes spanned volar and dorsal dislocations, and no specific mention of these injuries in children was made, the concept of multiaxial force in the pathogenesis of PIP dislocations was underscored. It is rare to have forces directed purely in one plane, and simply labeling the injury as a dorsal or volar tissue disruption is practical but incomplete. The tissue that is likely damaged in all these injuries, to an extent, is the collateral ligament.

Kieffhaber and co-workers ([114](#)) focused attention on the collateral ligament system and related displacement of the digit in the coronal plane to tissue pathology. More than 20 degrees of lateral deviation to gentle stress reliably indicated complete ligamentous disruption. The order of failure was also elucidated in this study: first, the collateral ligament is detached from the proximal phalangeal origin; then the accessory and proper collaterals are separated as forces move distally; the final area of failure is the distal volar plate insertion from the middle phalanx.

Treatment of the collateral ligament in the setting of a volar and dorsal dislocation has already been described. Protection of the collateral ligament can be accomplished with rigid splinting or buddy taping. The extent of the injury, evidenced by the amount of lateral deviation to stress and the quality of the end point, may mitigate for more aggressive treatment in some patients. If the joint can be dislocated by applying lateral stress, then consideration should be given to collateral repair. If a fracture is present in conjunction with the lateral stress injury, consideration could be stronger for opening the injury through a mid-axial approach and stabilizing the bone and repairing the soft tissues ([47](#)).

## AUTHORS' PREFERRED METHOD OF TREATMENT

The variables that must be considered when planning a treatment course for PIP level injuries in a child include extent and anatomic location of soft tissue disruption, presence or absence of fracture, reducibility, and stability after reduction. Almost all PIP dislocations deserve an attempt at closed reduction and stability assessment. Indeed, the standard protocol of anesthetic block, reduction, active and passive motion testing, and radiographic assessment suffice in most circumstances.

### Dorsal Dislocation

If the reduction was easily obtained and good motion arc and stability are present, we prescribe a short period (less than a week) of dorsal blocking protection. This can be followed by active motion with buddy taping.

If there is a proclivity for the joint to redislocate at 30 degrees of flexion or more, we exercise more of a standard dorsal blocking splint regimen: 45 to 60 degrees for 5 to 7 days, followed by 30 to 45 degrees for 5 to 7 days, followed by 15 to 30 degrees or buddy taping until swelling is decreased and stable motion is realized. The entire course usually lasts 2 to 3 weeks.

If surgical intervention is indicated in the rare severe intraarticular fracture, irreducible dislocation, or a combination, the ultimate goal of stable fixation to permit early motion must be observed. These problem fractures typically occur in adolescents, so "adult principles" govern.

### Volar Dislocation

This is a rare injury, but can occur in children. One of the classic mechanisms seems to be catching the digit in the spokes of a bicycle or exercise equipment. This injury reflects a disruption of the dorsal apparatus, and this must be respected. Therefore, splinting is in extension for up to 4 weeks, followed by a spring-loaded extension splint.

A dorsal epiphyseal fracture may indicate a central slip avulsion fracture that may require open treatment.

### Lateral Dislocations

Injury to the collateral ligaments is a likely component of all these dislocations, regardless of true "direction." Conversely, isolated rupture, or pure lateral dislocation, is probably rare. An incompetent collateral ligament is best treated by closed means. The exception to this is when there has been an incarceration of a lateral band in the dislocated joint.

### Metacarpophalangeal Joint Dislocations

The most common site for dislocation in the child's hand is the MCP joint ([84](#),[130](#),[200](#)). The thumb MCP joint dislocates most often as a result of a hyperextension force. The gamekeeper's or skier's thumb can be thought of as a momentary dislocation or subluxation. A small epiphyseal fragment may accompany the UCL, or a pure soft tissue injury can result. The most common complex or irreducible dislocation is at the index MCP joint.

### Dorsal Dislocations of the Triphalangeal Digits

The most common dislocation of the MCP joint is dorsal dislocation of the index digit ([Fig. 8-97](#)). The injury results from a hyperextension force that causes a rupture or avulsion of the volar plate ([33](#)). When the proximal phalanx becomes situated dorsal to the metacarpal, there is little question of the diagnosis. The digit is supinated, ulnarly deviated, and shortened. The interphalangeal joints are slightly flexed. Most patients have volar skin dimpling over the metacarpal head.



**FIGURE 8-97. A:** A 3-year-old boy with a complete complex dislocation of the index finger metacarpal joint. **B:** Note the parallelism in this lateral view. Open reduction was performed through a volar approach, and normal function resulted.

Radiographs can confirm the diagnosis, especially when there is gross malalignment of the bony elements or the sesamoids are seen in the joint. The position of the volar plate in children over 10 years of age can be inferred by sesamoid position in the index and thumb rays (211). Rarely, other digits have visible sesamoids, and this same diagnostic technique can be used. It is often suggested that the presence of the sesamoids in the joint connotes a complex dislocation, but this finding is not absolute.

Other clues to dislocation at the MCP joint can be gleaned from radiographs. Robins (181) observed simple narrowing of the MCP joint space in MCP dislocation. Campbell (36) described the concept of the “phalangeal line” as a guide to determining the presence of fracture or dislocation at the MCP joint. He showed that a line drawn down the axis of the proximal phalanx should pass through the axis of rotation of the metacarpal head in the uninjured digit. When a fracture or dislocation was present, the phalangeal line became skewed (36). The concept of “parallelism” of the metacarpal and the phalanx is another expression of Campbell's concept. This finding is not universally seen in the complex dislocation. The surgeon should be especially cognizant of the infrequent occurrence of adjacent MCP dislocations. These most often occur with more violent trauma and can be difficult to diagnose clinically because of hand swelling. Radiographs may be difficult to interpret, because projections may obscure adjacent MCP joints.

To explain the pathoanatomy of an irreducible or complex dislocation, Kaplan (112) described the position of the metacarpal head with respect to the surrounding soft tissues. The metacarpal head becomes “picture-framed” by the flexor tendon to the ulnar side and the lumbrical to the radial side. The superficial transverse metacarpal ligament and the natatory ligaments can settle dorsally around the metacarpal neck, and the palmar aponeurosis captures the metacarpal on the volar side. The collar is tightened by longitudinal traction, and this common reduction maneuver can actually convert a reducible dislocation to an irreducible one. The volar plate can become interposed between the dorsal metacarpal head and the dislocated proximal phalanx, further blocking reduction.

Reduction is effected by further hyperextending the joint, then attempting to capture the volar plate with the dorsal lip of the proximal phalanx. Intraarticular infiltration of anesthetic fluid can assist reduction by “floating” the volar plate out of its displaced position. The success rate for closed reduction of complex dislocation is low. Operative reduction and clearing of the joint usually are required. If a reasonable attempt at closed reduction has failed, open reduction is warranted.

**Volar Approach.** When closed reduction is unsuccessful, open methods are needed. Many surgeons prefer the volar approach for reduction of the MCP dislocation (Fig. 8-97). The volar approach affords excellent exposure of the metacarpal head and the structures that incarcerate it (11,14,23,84,92,112,130,143). The authors that prefer this approach accept the risk of potential injury to the digital nerves that are draped over the articular surface of the metacarpal head. Kaplan and others advocate division of multiple structures, including the A1 pulley, the distal edge of the palmar aponeurosis, the superficial transverse metacarpal ligament, and the natatory ligaments. Other clinicians believe that division of the A1 pulley alone (130) or the transverse metacarpal ligament alone (104) is sufficient.

McLaughlin modified the volar approach to the MCP joint by incorporating it with a mid-lateral incision into the digit, choosing to extend the incision along the radial mid-lateral line (143). He used a skin hook to extract the volar plate from its position in the dislocated joint. Reduction was accomplished without the need for additional soft tissue releases.

Metacarpophalangeal dislocations in the other triphalangeal joints are uncommon (Fig. 8-98). Baldwin et al. (10) described an irreducible fifth ray MCP dislocation in which the metacarpal head was entrapped by the abductor digiti quinti and flexor digiti minimi on the medial side and the long flexor tendons to the lateral side. Open reduction with incision of the volar plate was necessary to correct the deformity.



**FIGURE 8-98.** A relatively rare dorsal dislocation of the long finger was irreducible by closed means. A dorsal approach permitted visualization of the joint and extrication of the volar plate. Excellent function ensued after early motion was started.

Metacarpophalangeal dislocations in the central rays are rare (Fig. 8-99) (161). Lateral fracture–dislocations at the MCP joint sometimes occur, and are often S-H III fractures of the phalangeal base. Open reduction usually is necessary to treat the fracture.



**FIGURE 8-99. A:** A 9-year-old girl sustained this radial fracture–dislocation of the middle finger in a fall on the stairs. **B:** Six weeks after closed reduction and immobilization in a radial gutter splint for 3 weeks, there was full motion and normal stability.

The optimal treatment is prompt recognition and reduction of the MCP dislocation. The window of opportunity for successful reduction is difficult to define. Although reasonable results have been obtained when the dislocation is reduced within the first 3 to 4 months, this is still suboptimal treatment. After 6 months, results are less predictable, and accelerated arthrosis may necessitate an early salvage procedure.

**Dorsal Approach.** Most surgeons use a dorsal exposure for reduction of complex MCP joint dislocations. Described by Becton et al. (14), this simplified technique avoids the catastrophic complication of digital nerve transection, while still permitting adequate joint access.

By relaxing the flexor pull by simply flexing the wrist and digits, the joint can be approached through a tendon-splitting incision. Either a transverse or hockey stick

capsulotomy can be used, but the injury may have stripped the capsule to an extensive degree. Use of a Freer or septal elevator defines the joint, and may sweep away the soft tissue that was interposed. Throughout the procedure, protection of the metacarpal head articular surface is paramount.

Pushing the volar plate back into position may be the only treatment needed for the joint. At times, a flexor tendon is wrapped around the condyle. If the volar plate is difficult to reduce, it can be split from the dorsum in a longitudinal fashion. This maneuver relaxes the plate, and permits it to slip back to anatomic position. The volar plate need not be repaired or sutured back to surrounding tissues after its reduction.

**Postoperative Therapy.** Regardless of the approach used, there is universal agreement that early motion is necessary. The minimal amount of tissue dissection facilitates this, and a minimal soft dressing is all that is needed in the acute postoperative period. Rarely, a dorsal blocking splint may be needed to prevent hyperextension that may lead to redislocation. There exists no report of redislocation at the MCP level after open reduction.

**Missed MCP Dislocations.** Early reduction of the MCP dislocation has been stressed by Hunt et al. (109). It is universally agreed that early reduction and an early motion program should be accomplished within the first 7 days (112,143). Despite appropriate radiographs and clinical examination, inexperience with these injuries can lead to a missed dislocation.

Significant motion limitation and deformity characterize this entity, although pain is typically minimal. Operative intervention is the only reasonable alternative, thus we caution against vigorous attempts at closed reduction when a dislocation has been present more than 2 or 3 weeks.

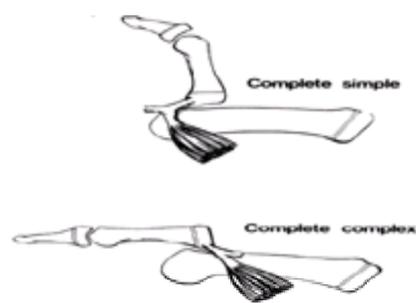
Murphy and Stark (157) discussed the treatment of missed dislocations. They performed open reduction of MCP dislocations that had been neglected for 3 weeks to 3 months. Both volar and dorsal approaches were used, and the UCL had to be resected in some patients. Transarticular pinning was maintained for 3 weeks. There was decreased motion at the MCP joint, but it was still thought to be in the useful range. Late complications included partial growth arrest. Barenfeld (11) achieved normal motion in a patient whose dislocation was reduced 3½ months after injury. Lipscomb and Janes (133) reported a 20-year follow-up of an unreduced thumb MCP dislocation. They related that a “new metacarpal head, joint, and ligament apparently formed spontaneously.” It is likely that the exceptional remodeling about the adjacent epiphyses adapted to the new forces contributed by the bones in their new position.

We agree that surgical reduction will likely require both volar and dorsal approaches. The multiple structures that can incarcerate the metacarpal head have contracted, and release of any tight structure is indicated. Dorsal clearing of the joint often is needed to remove the fibrous tissue that has occupied the void left by the dislocation.

### Dorsal Dislocation of the Thumb Ray

In many ways, MCP joint dislocations of the thumb are similar to those of the triphalangeal digits. The hyperextension mechanism is the same, as is the proclivity of the dislocation to be irreducible. Because of the thumb's differing anatomy and functional importance, it is considered separately.

The pathophysiology was elucidated by Farabeuf in 1876 (69). His classification system is complete and quite useful. It describes tissue injury and explains the implications for and likelihood of reduction. The focus of the system is the integrity and position of the volar plate, the status of the collateral ligaments, and the relative position of the metacarpal and proximal phalanx. The components of the classification are incomplete dislocation, simple complete dislocation, and complete complex dislocation (Fig. 8-100).



**FIGURE 8-100.** Simple and complex dorsal dislocation of the thumb metacarpophalangeal joint.

**Incomplete Thumb MCP Dislocations.** Incomplete dislocations occur when the volar plate ruptures, but the collateral ligaments remain intact. In its most benign form, an intrasubstance injury or partial avulsion of the volar plate occurs, which may result in supraphysiologic extension at the MCP joint. Often, the volar plate rupture is complete, and the MCP joint subluxates as the proximal phalanx perches on the dorsum of the metacarpal.

A period of immobilization usually is adequate treatment. Three weeks is sufficient for more significant injuries, and discontinuation of the splint when tenderness subsides is reasonable for low-level trauma. If a child desires to return to athletic participation, protection with a hand-based thumb spica orthosis is recommended. A pure ligamentous injury at the ulnar collateral can be classified as an incomplete dislocation. This is best shown by stress radiographs and examination.

**Simple Complete Thumb MCP Dislocation.** The simple complete dislocation of the thumb MCP is the most clinically obvious. The proximal phalanx is dorsally displaced, and its long axis is 90 degrees to that of the metacarpal.

Many of these dislocations can be reduced by closed means (Fig. 8-101). The reduction maneuver previously described for interphalangeal joint reduction is particularly useful, including insufflation of the joint with lidocaine. This maneuver resembles that described by Burton and Eaton (34), who recommended extension of the thumb interphalangeal joint to lift the flexor pollicis longus and volar plate away from their position potentially blocking the reduction. Many clinicians have warned against excessive longitudinal traction when attempting reduction. The resulting force may convert a reducible injury into a complex dislocation (92,143).



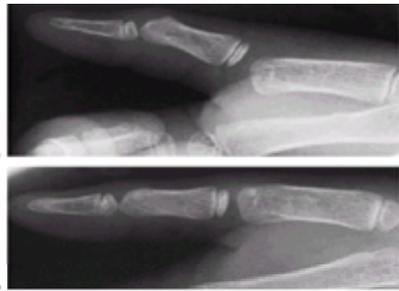
**FIGURE 8-101.** Complete simple dislocation. A 9-year-old boy with a dorsal dislocation of the thumb metacarpophalangeal joint. The dislocation lacks parallelism, and closed reduction was successful.

If reduction is successful, thumb spica casting for 3 weeks usually is sufficient to permit healing of the volar plate and collateral ligaments. Care must be taken during cast application so as not to deviate the thumb in a radial or ulnar direction, because of the potential of concomitant collateral ligament rupture.

**Complete Complex Thumb MCP Dislocation.** The dislocation that represents the most tissue disturbance, and the one that normally requires open reduction, appears as the most clinically inconspicuous. In a complete complex dislocation, the long axes of the proximal phalanx and metacarpal can be parallel. The thumb is shortened and swollen about the MCP joint.

It is difficult to obtain closed reduction of a complete complex dislocation, although occasionally closed manipulation can reduce the joint ( 200). Even if closed reduction is successful, some surgeons advocate open ligament repair if significant lateral instability is present.

As a rule, this injury must be treated by open reduction (Fig. 8-102). Farabeuf (69), Weeks (225), and Bohart (25) recommended a dorsal approach to the joint, but a volar approach usually is advocated to reliably extricate the volar plate.



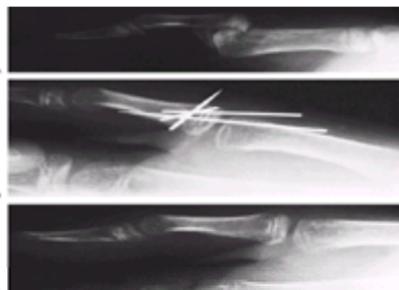
**FIGURE 8-102.** Complete complex dislocation. **A:** Closed reduction attempts failed to reduce this dorsal metacarpophalangeal dislocation in a 7-year-old boy. Note the relative parallelism between the dislocated thumb and metacarpal. **B:** After open reduction through a volar incision. Normal function resulted.

The volar approach is made through a Brunner incision. The digital nerves of the thumb are central in an uninjured thumb, and become dangerously tented over the metacarpal head in a complete dislocation. Great care must be exercised to avoid their injury. The flexor pollicis brevis muscle is followed down to the volar plate, then the plate can be removed from the displaced position. Repair of the volar plate back to the metacarpal neck restores anatomy ( 92,207), but the value of this procedure must be weighed against the potential of undue stiffness. It seldom is necessary to repair or reconstruct the collateral ligaments.

Three weeks of immobilization in a thumb spica cast is followed by a gradual range-of-motion program. Protection during athletics is also recommended for complete complex dislocations.

#### Fracture–Dislocation

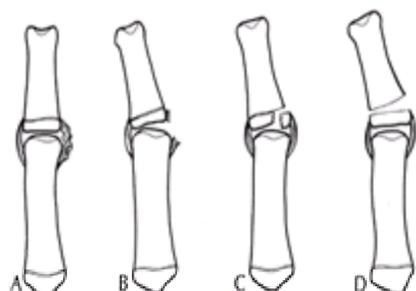
Dorsal fracture–dislocation of the thumb MCP joint is a rare injury that also usually requires treatment ( Fig. 8-103).



**FIGURE 8-103.** **A:** A 12-year-old boy sustained a dorsal fracture–dislocation of the metacarpophalangeal joint when his thumb was struck by a shotgun bolt. Note that a posteroanterior view of the thumb metacarpal appears on the same image as a lateral view of the phalanges. **B:** Closed attempts to reduce the fracture–dislocation were unsuccessful, and open reduction and internal fixation with Kirschner wires were performed. **C:** Four months after surgery, he had only a 10 degree loss of joint motion.

#### Thumb MCP UCL Injury (Gamekeeper's Thumb)

**Four Types in Children** Ulnar collateral ligament injury of the child's thumb MCP is not given much attention in the voluminous literature on gamekeeper's thumb. Forced abduction stress at the child's thumb MCP joint results in four types of injury: (a) a simple sprain of the UCL; (b) a rupture or avulsion of the insertion or origin (229) of the ligament; (c) a simple S-H I or II fracture of the proximal physis; or (d) an avulsion fracture (S-H III) of the ulnar one fourth to one third of the epiphysis of the proximal phalanx (Fig. 8-104).



**FIGURE 8-104.** Ulnar instability of the thumb metacarpal joint. **A:** Simple sprain. **B:** Rupture of the ligament. **C:** Avulsion fracture (Salter-Harris type III). **D:** Pseudo-gamekeeper's injury resulting from a Salter-Harris type I or II fracture of the proximal phalanx.

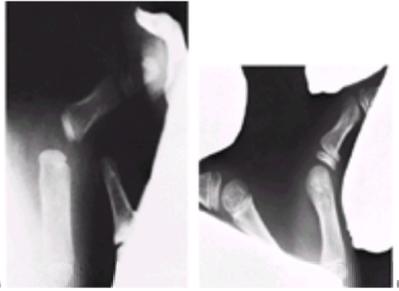
**An Adolescent Injury.** The injury is usually encountered in the preadolescent or adolescent. Clinically, the thumb is quite swollen, particularly in the area of the MCP

joint, and ecchymosis often extends out into the skin of the first web. If routine radiographs show no bony injury, the joint must be stressed by applying lateral force with the joint in full extension first, then in full flexion. Preliminary infiltration of the joint with lidocaine or blocking of the median and superficial radial nerves is usually necessary. If the ligament is completely torn, there will be no exact end point on stressing the ligament, particularly in flexion.

**Breakdancer's Thumb.** Winslet and associates (231) reported on three teenagers with a displaced fracture of the ulnar corner of the base of the proximal phalanx of the thumb with ulnar instability after breakdancing. All three had open reduction and internal fixation. They coined the name *breakdancer's thumb* for the injury.

#### Treatment.

**Cast Immobilization.** Healing in a cast is unlikely because the ligament, which is usually avulsed from its distal insertion, is often caught over the edge of the extensor aponeurosis when the joint reduces spontaneously (206,207). The ligament cannot heal, and chronic lateral instability with a weakened pinch is likely. A false impression of instability can result from an S-H I or II fracture of the proximal phalanx that is undisplaced on the original radiograph and becomes obvious only on the stress radiograph (Fig. 8-104D and Fig. 8-105). Cast immobilization is all that is required for this injury. Incomplete tears (<45 degrees difference on stress testing) heal in a thumb spica cast worn for 4 weeks.



**FIGURE 8-105. A:** Even with gentle stress, a complete ulnar collateral ligament incompetence is easily detected. This 8-year-old girl was treated with early ligament repair, and normal stability resulted. **B:** In this 10-year-old boy, a pseudo-gamekeeper's thumb is the result of a Salter-Harris type II fracture of the proximal phalanx. (B courtesy of James H. Dobyns, M.D.)

**Operative Indications.** If the stress radiograph, made with the thumb extended, shows that the joint opens 45 degrees more than the normal side, a complete tear has occurred, and open repair is indicated (Fig. 8-105) (200). An S-H III fracture of the ulnar corner of the epiphysis of the proximal phalanx is by far the most common childhood gamekeeper's injury. The significantly displaced fracture (fragment rotated and displaced >1.5 mm) requires open reduction and internal fixation to restore the integrity of the UCL and to obtain a congruous joint surface (Fig. 8-106).



**FIGURE 8-106. A:** An ulnar collateral ligament Salter-Harris type III avulsion fracture in a 12-year-old girl. **B:** After open reduction and internal fixation.

**Missed Thumb MCP Dislocations.** Like some MCP dislocations in the triphalangeal digits, thumb MCP dislocations also can go undetected or neglected (133). Depending on many factors (length of time since original injury, age of the patient, current level of function), the surgeon must decide how aggressively to treat the problem once recognized.

If detected in the first 6 to 8 weeks, we recommend an attempt at reduction through a volar or combined approach. Transarticular pinning can be considered. Depending on the tissue quality, amount of symptoms, and functional status, it is not unreasonable to attempt reduction up to 4 to 6 months after injury.

Some late injuries may be best left to remodel to their maximal function, then a salvage procedure such as soft tissue arthroplasty or fusion (199) can be performed.

#### AUTHORS' PREFERRED METHOD OF TREATMENT

Management of MCP level dislocations should be stepwise and logical. We believe that essentially every MCP dislocation deserves an attempt at closed reduction. After insufflating the joint with anesthetic, recreation of the deformity with hyperextension and performing a reduction maneuver can result in reestablishment of the articular relationship. This should be performed under optimized conditions, which may include conscious sedation and even an operating room setting. The latter is advocated in case the surgeon must proceed to open reduction.

We advocate the dorsal approach for most MCP dislocations requiring open treatment. The volar plate is the typical tissue blocking reduction, and it usually can be removed by sweeping the joint with a septal elevator in most cases. In some patients, the interposed volar plate needs to be incised in a longitudinal fashion to promote its exit from the joint. There is no indication for volar plate repair.

If the surgeon is more experienced or more confident with the volar approach, it cannot be overemphasized that the digital nerve is draped over the prominent metacarpal head. Utmost care must be exercised in liberating the incarcerated metacarpal from the surrounding soft tissues and reducing the joint.

#### Dislocations of the Carpometacarpal Joints

Dislocations of the CMC joint, with or without small fracture fragments from the metacarpal base, are rare injuries. The decision whether to open the dislocation sight is based primarily on the stability after closed reduction. We suggest stabilization with percutaneous pins in almost all these injuries because of the proclivity to redislocate.

The fractures that accompany the dislocations can range from small avulsions that accompany the ligament disruption to large fragments comprising a significant percentage of the articular surface (Fig. 8-64). Often these fragments are displaced and rotated. Some large articular fractures can rotate 180 degrees while the fracture dislocation is in its maximum displacement. It is important to recognize that fracture fragments can block reduction and be the source of continued pain and

potential nonunion.

Although isolated dislocations at the CMC level do occur, their pathogenesis and treatment are the same as disruptions in which fractures occur. (See previous section on [metacarpal fractures](#).)

## Dislocations of the Carpus

### *Rare and Often Unrecognized*

Intercarpal dislocations are uncommon in children. Due to the rarity of the injury and the relative smallness of the child's carpal bone, this injury is likely to go unrecognized ([Fig. 8-90](#) and [Fig. 8-91](#)). A 10-year-old child with an acute dorsal trans-scaphoid perilunate dislocation was reported by Peiro et al. ([166](#)). Sixteen weeks after closed reduction and cast immobilization, he had a normal wrist. Ogden ([162](#)) published the radiograph of a young child with a trans-scaphoid perilunar dislocation but gave no details. Christodoulou and Colton ([41](#)) mentioned a 9-year-old boy who had a trans-scaphoid perilunar dislocation, but no details were given. Light ([129](#)) described an 11-year-old child who had open reduction and Kirschner wire fixation of a trans-scaphoid perilunar dislocation with a fracture through the lunate. Pennes et al. ([167](#)) reported a 14-year-old girl with Marfan's syndrome who had bilateral dorsal perilunate dislocations, unrelated to trauma, secondary to ligamentous laxity.

### *Salvage Treatment Needed*

The principles of treatment do not differ from those for adults with dislocations of the carpus. Reconstructive surgery may be required for a chronic dislocation that has gone unrecognized ([Fig. 8-107](#)). Gerard ([81](#)) reported the only child in the literature with a chronic posttraumatic carpal instability. The 7-year-old girl had pain and limitation of wrist motion secondary to a scapholunate dissociation with a palmar flexion collapse deformity of the lunocapitate joint. Open reduction and pin fixation, with reconstruction of the scapholunate ligament with a portion of extensor carpi radialis longus tendon, corrected the instability. Light ([129](#)) recommended the use of arthrography and MRI to demonstrate scapholunate dissociation in the immature carpus.



**FIGURE 8-107. A and B:** An 11-year-old girl sustained this injury 7 months previously. She had been radiographed several times, but the correct diagnosis of volar trans-scaphoid perilunate dislocation was not made. **C:** Intercarpal fusion was performed, and 1 year after injury the range of motion was as follows: dorsiflexion 30 degrees, palmar flexion 30 degrees, radial deviation 10 degrees, and ulnar deviation 25 degrees. She had minimal discomfort with strenuous use.

One of us (T.J.G.) has reported a skeletally immature teenage girl with hemiatrophy who presented with the extremely rare pattern of a palmar mid-carpal dislocation ([89](#)). Although the patient recalled no antecedent trauma, the wrist had apparently been dislocated for a lengthy period, judging from the radiographic and clinical appearance. Symptoms and function improved after reduction and mid-carpal fusion.

## Dislocation of the Distal Radioulnar Joint

### *More Common in the Older Child*

Dislocation of the distal radioulnar joint may occur with ([148](#)) or without a fracture of the radius. An isolated dislocation is uncommon in young children, but may occur near the end of the growth period. The dislocation may be in a dorsal or volar direction and is likely to go unrecognized.

### *Computed Tomographic Imaging Most Useful*

Computed tomographic scanning has proved superior to plain radiographs in diagnosing a dislocation or subluxation of the distal radioulnar joint ([61,150,224](#)). The scan is especially useful in determining through a cast whether the dislocation is reduced.

### *Anatomic Considerations*

Many clinicians ([54,105](#)) have correctly pointed out that the radiocarpal complex dislocates from the ulna, which, because of its fixed proximal articulation with the humerus, cannot dislocate. Common terminology, however, still refers to the ulna as being dislocated.

The head of the ulna articulates with the sigmoid notch of the radius. The stability of the distal radioulnar joint is maintained by the triangular fibrocartilage and the dorsal and volar radioulnar ligaments. The triangular fibrocartilage, extending from the distal margin of the radial sigmoid notch to the base of the styloid process of the ulna, acts primarily to prevent lateral displacement of the ulna ([100,105](#)). The dorsal and volar radioulnar ligaments are the prime stabilizers of the distal radioulnar joint. The dorsal radioulnar ligament becomes taut on pronation, and the volar radioulnar ligament becomes taut on supination.

### *Mechanisms Vary*

Hyperpronation results in a tear of the dorsal ligament and triangular fibrocartilage and a dorsal dislocation of the distal ulna. Hypersupination tears the volar radioulnar ligament and triangular fibrocartilage, producing a volar dislocation of the distal ulna.

### *Clinical and Radiographic Findings*

If the dislocation is dorsal, there is a marked dorsal prominence of the distal end of the ulna, and any attempt to supinate the pronated forearm is resisted. The AP view shows an abnormal separation at the distal radioulnar joint, and the ulna is dorsally displaced on the true lateral view. If the dislocation is volar, the wrist appears narrower than normal because the normal dorsal prominence of the distal ulna is missing. The forearm is fixed in supination. The ulna and radius are overlapped on the AP view, and the ulna is volarly displaced on the true lateral view.

### *Treatment*

#### **Nonoperative Usually Satisfactory**

If the diagnosis is made acutely, closed reduction usually is successful. A volarly displaced ulna is reduced in full pronation, and a dorsal displacement is corrected by direct pressure over the ulna and full supination. A long arm cast in the corrected position is worn for 4 to 6 weeks. A 4-year-old girl with a dorsal dislocation of the distal ulna, successfully treated with a long arm cast in supination for 4 weeks, was reported by Heiple and Freehafer ([105](#)). Birch-Jensen ([19](#)) reported a successful

result in a 14-year-old boy with a volar dislocation of the distal ulna reduced by slight pressure and immobilized for only a week with a dorsal splint.

## Operative Indications

The dislocation is occasionally irreducible because of an interposed extensor carpi ulnaris tendon ( 10,39). Chronic or recurrent dislocation is likely to follow an undiagnosed or inadequately treated acute dislocation. Attempts to restabilize the distal radioulnar joint in children using soft tissue procedures have met with some success. Dameron (54) advised excision of the distal end of the ulna if the dislocation was untreated for 2 months or longer. Excision of the distal ulna, if indicated, should of course be postponed until growth is complete. Dislocation of the distal ulna secondary to premature cessation of radial growth is best treated by ulnar shortening.

## Association with Fractures of the Radius

Dislocation or disruption of the distal radioulnar joint with a fracture of the shaft or distal radius is referred to as a Galeazzi fracture (actually a fracture–dislocation complex). This entity is discussed in more detail in [Chapter 9](#).

## ACKNOWLEDGMENT

We would like to recognize the unparalleled contribution of Eugene T. “Tom” O'Brien, M.D., to the clinical understanding and surgical science of treating children's hand and wrist injuries. His experience and insight was brought to us through the first four editions of this text, and the influence of his dedication to this topic guides this and future writings.

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## CHAPTER REFERENCES

1. Agee JM. Unstable fracture dislocations of the PIP joint of the fingers: a preliminary report of a new treatment technique. *J Hand Surg [Am]* 1978;3:386–389.
2. Aggarwal AK, Sangwan SS, Siwach RC. Trans-scaphoid perilunate dislocation in a child. *Contemp Orthop* 1993;26:172–174.
3. Albert MC, Barre PS. A scaphoid fracture associated with a displaced distal radial fracture in a child. *Clin Orthop* 1989;240:232–235.
4. Alho A, Kankaanpaa U. Management of fractured scaphoid bone: a prospective study of 100 fractures. *Acta Orthop Scand* 1975;46:737–743.
5. Ali MA. Fracture of the body of the hamate bone associated with compartment syndrome and dorsal decompression of the carpal tunnel. *J Hand Surg* 1986;11:207–210.
6. Amadio PC, Berquist TH, Smith DK, et al. Scaphoid malunion. *J Hand Surg [Am]* 1989;14:679–687.
7. Anderson WJ. Simultaneous fracture of the scaphoid and capitate in a child. *J Hand Surg [Am]* 1987;12:271–273.
8. Atasoy E, Ioakimidis E, Kasclan ML, et al. Reconstruction of the amputated finger with a triangular volar flap: a new surgical procedure. *J Bone Joint Surg [Am]* 1970;52:921–926.
9. Atasoy E, Godfrey A, Kalisman M. The “antenna” procedure for the “hook-nail” deformity. *J Hand Surg* 1983;8:55–58.
10. Baldwin LW, Miller DL, Lockhart LD, et al. MCP joint dislocations of the fingers: a comparison of the pathological anatomy of index and little finger dislocations. *J Bone Joint Surg [Am]* 1967;49:1587–1590.
11. Barenfeld PA, Weseley MS. Dorsal dislocation of the MCP joint of the index finger treated by late open reduction. *J Bone Joint Surg [Am]* 1972;54:1311–1313.
12. Barton NJ. Fractures of the phalanges of the hand in children. *Hand* 1979;2:134–143.
13. Beatty E, Light TR, Belsole RJ, et al. Wrist and hand skeletal injuries in children. *Hand Clin* 1990;6:723–738.
14. Becton JL, Christian JD, Goodwin HN, et al. A simplified technique for treating the complex dislocation of the index MCP joint. *J Bone Joint Surg [Am]* 1975;57:698–700.
15. Bennett EH. Fractures of the metacarpal bones. *Dublin J Med Sci* 1982;73:72–75.
16. Benz HJ, Blenche BA. Restitution Einer Lunatamnekrose Bein Kind. *Z Orthop* 1976;114:819–821.
17. Bergfeld JA, Weiker GG, Andrish JT. Soft-playing splint for protection of significant hand and wrist injuries in sports. *Am J Sports Med* 1982;10:293–299.
18. Bhende MS, Dandrea LA, Davis HW. Hand injuries in children presenting to a pediatric emergency department. *Ann Emerg Med* 1993;22:1519–23.
19. Birch-Jensen A. Luxation of the distal radioulnar joint. *Acta Chir Scand* 1951;101:312–317.
20. Blair WF, Marcus NA. Extrusion of the PIP joint—case report. *J Hand Surg* 1981;6:146–147.
21. Blitzer CM, Johnson RJ, Ettlinger CF, et al. Downhill skiing injuries in children. *Am J Sports Med* 1984;12:142–147.
22. Bloem JJA. Fracture of the carpal scaphoid in a child aged 4. *Arch Chir Neerlandicum* 1971;23:91–94.
23. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955:112–128.
24. Bogumill GP. A morphologic study of the relationship of collateral ligaments to growth plates in the digits. *J Hand Surg* 1983;8:74–79.
25. Bohart PG, Gelberman RH, Vandell RF, et al. Complex dislocations of the MCP joint. *Clin Orthop* 1982;164:208–210, 1982.
26. Bora FW Jr, Culp RW, Osterman AL. A flexible wrist splint. *J Hand Surg [Am]* 1989;14:574–575.
27. Bora FW Jr, Didizian NH. The treatment of injuries to the carpometacarpal joint of the little finger. *J Bone Joint Surg [Am]* 1974;56:1459–1463.
28. Borde J, Lefort J. Injuries of the wrist and hand in children. In: Tubiana R, ed. *The hand*. Vol. 2. Philadelphia: WB Saunders, 1985:844–868.
29. Bowers WH. The PIP joint volar plate. II: A clinical study of hyperextension injury. *J Hand Surg* 1981;6:77–81.
30. Boyes JH. *Bunnell's surgery of the hand*. Philadelphia: JB Lippincott, 1970:592.
31. Brighton CT. Clinical problems in epiphyseal plate growth and development. *Instr Course Lect* 1974;3:105–122.
32. Büchler U, McCollam SM, Oppikofer C. Comminuted fractures of the basilar joint of the thumb: combined treatment by external fixation, limited internal fixation, and bone grafting. *J Hand Surg [Am]* 1991;16:556–560.
33. Burman M. Irreducible hyperextension dislocation of the MCP joint in a finger. *Bull Hosp Joint Dis* 1953;14:290–291.
34. Burton RI, Eaton RG. Common hand injuries in the athlete. *Orthop Clin North Am* 1973;4:809–838.
35. Butt WD. Rigid wire fixation of fractures of the hand. *Henry Ford Hosp Med J* 1956;4:134–143.
36. Campbell RM. Operative treatment of fractures and dislocations of the hand and wrist region in children. *Orthop Clin North Am* 1990;21:217–243.
37. Carr CR, Johnson RJ, Pope MH. Upper extremity injuries in skiing. *Am J Sports Med* 1981;9:378–383.
38. Carter SR, Aldridge MJ, Fitzgerald R, et al. Stress changes of the wrist in adolescent gymnasts. *Br J Radiol* 1988;61:109–112.
39. Cetti MNE. An unusual cause of blocked reduction of the Galeazzi injury. *Injury* 1977;9:56–61.
40. Chambers RB. Orthopedic injuries in athletes (ages 6 to 17). *Am J Sports Med* 1979;7:195–197.
41. Christodoulou AG, Colton CL. Scaphoid fractures in children. *J Pediatr Orthop* 1986;6:37–39.
42. Clayburgh RH, Wood MB, Cooney WP. Nail bed repair and reconstruction by reverse dermal grafts. *J Hand Surg* 1983;8:594–599.
43. Cobey MC, White RK. An operation for nonunions of the carpal navicular. *J Bone Joint Surg* 1946;28:757–764.
44. Cockshott WP. Distal avulsion fracture of the scaphoid. *Br J Radiol* 1980;53:1037–1040.
45. Compson JP. Transcarpal injuries associated with distal radial fractures in children: a series of three cases. *J Hand Surg [Br]* 1992;17:311–314.
46. Coonrad RW, Pohlman MH. Impacted fractures of the proximal portion of the proximal phalanx of the finger. *J Bone Joint Surg [Am]* 1969;51A:129–1296.
47. Cowen NJ, Kranik AD. An irreducible juxtaepiphyseal fracture of the proximal phalanx. *Clin Orthop* 1975;110:42–44.
48. Crawford AH, Gupta A, Risitano G, et al. Mucous cyst of the distal interphalangeal joint: treatment by simple excision or excision and rotation flap. *J Hand Surg [Br]* 1990;15:113–114.
49. Crawford AH. Pitfalls and complications of fractures of the distal radius and ulna in childhood. *Hand Clin* 1988;4:403–413.
50. Crick JC, Franco RS, Connors JJ. Fractures about the interphalangeal joints in children. *J Orthop Trauma* 1988;1:318–325.
51. Crock HV, Chari PR, Crock MC. The blood supply of the wrist and hand bones in man. In: Tubiana R, ed. *The hand*. Vol. 1. Philadelphia: WB Saunders, 1981:335–349.
52. Cullen JC. Thiemann's disease—osteochondrosis juvenilis of the basal epiphyses of the phalanges of the hand—report of two cases. *J Bone Joint Surg [Br]* 1970;52:532–534.
53. DaCruz DJ, Slade RJ, Malone W. Fractures of the distal phalanges. *J Hand Surg [Br]* 1988;13:350–352.
54. Dameron TB. Traumatic dislocation of the distal radioulnar joint. *Clin Orthop* 1972;83:55–63.
55. DeBoeck H, Van Wellen P, Haentjens P. Nonunion of a carpal scaphoid fracture in a child. *J Orthop Trauma* 1991;5:370–372.
56. DeHaven KE, Lintner DM. Athletic injuries: comparison by age, sport, and gender. *Am J Sports Med* 1986;14:218–224.
57. de Iturriza JR, Tanner JM. Cone-shaped epiphyses and other minor anomalies in the hands of normal British children. *J Pediatr* 1969;75:265–272.
58. Dicke TE, Nunley JA. Distal forearm fractures in children. *Orthop Clin North Am* 1993;24:333–340.
59. Dixon GL, Moon NF. Rotational supracondylar fractures of the proximal phalanx in children. *Clin Orthop* 1972;83:151–156.
60. Doman AN, Marcus NW. Congenital bipartite scaphoid. *J Hand Surg [Am]* 1990;15:869–873.
61. Drewniany JJ, Palmer AK. Injuries to the distal radioulnar joint. *Orthop Clin North Am* 1986;17:451–459.
62. Dunn AW. Fractures and dislocations of the carpus. *Surg Clin North Am* 1972;52:1513–1538.
63. Dykes RG. Kirner's deformity of the little finger. *J Bone Joint Surg [Br]* 1978;60:58–60.
64. Eaton RG. *Joint injuries of the hand*. Springfield, IL: Charles C Thomas, 1971.

65. Eaton RG, Dobranski AI, Littler JW. Marginal osteophyte excision in treatment of mucous cysts. *J Bone Joint Surg [Am]* 1972;55:570–574.
66. Elson RA. Rupture of the central slip of the extensor hood of the finger. *J Bone Joint Surg [Br]* 1986;68:229–231.
67. Enber WD, Clancy WG. Traumatic avulsion of the fingernail associated with injury to the phalangeal epiphyseal plate. *J Bone Joint Surg [Am]* 1978;60:713–714.
68. Ersek RA, Galaria U, Denton DR. Nail bed avulsions treated with porcine xenografts. *J Hand Surg [Am]* 1985;10:152–153.
69. Farabeuf LHF (as quoted by Barnard HL). Dorsal dislocation of the first phalanx of the little finger. Reduction by farabeuf's dorsal incision. *Lancet* 1901;1:88–90.
70. Fischer MD, McElfresh EC. Physeal and periphyseal injuries of the hand. Patterns of injury and results of treatment. *Hand Clin* 1994;10:287–301.
71. Fisk GR. Carpal instability and the fractured scaphoid. *Ann R Coll Surg Eng*; 1970;46:63–76.
72. Foucher G. "Bouquet" osteosynthesis in metacarpal neck fracture: a series of 66 patients. *J Hand Surg [Am]* 1995;20(suppl):86–90.
73. Freeland AE, Barret GR, Wheelless GS. Correction of abduction deformity of the small finger caused by avulsion. *Am J Sports Med* 1985;13:273–276.
74. Frykman G. Fracture of the distal radius including sequelae—shoulder-hand-finger syndrome, disturbance in the distal radioulnar joint and impairment of nerve function. A clinical and experimental study. *Acta Orthop Scand Suppl* 1967;108:3.
75. Gamble JG, Simmons SC. Bilateral scaphoid fractures in a child. *Clin Orthop* 1982;162:125–128.
76. Garrick JG, Regua RK. Injuries in high school sports. *Pediatrics* 1978;61:465–469.
77. Garroway RY, Hurtst LC, Leppard J, Dick HM. Complex dislocations of the PIP joint. A pathoanatomic classification of the injury. *Orthop Rev* 1984;13:21–28.
78. Gatewood. A plastic repair of finger defects without hospitalization. *JAMA* 1926;87:1479.
79. Gedda KO. Studies in Bennett's fracture: anatomy, roentgenology, and therapy. *Acta Chir Scand* 1954;5:193.
80. Gellman H, Caputo RJ, Carter V, et al. Comparison of short and long thumb spica casts for nondisplaced fractures of the carpal scaphoid. *J Bone Joint Surg [Am]* 1989;71:354–357.
81. Gerard FM. Post-traumatic carpal instability in a young child. *J Bone Joint Surg [Am]* 1980;62:131–133.
82. Gibbon WW, Jackson A. An isolated capitate fracture in a 9-year-old boy. *Br J Radiol* 1989;62:487–488.
83. Giddins GE, Shaw DG. Lunate subluxation associated with a Salter-Harris type II fracture of the distal radius. *J Hand Surg [Br]* 1994;19:193–194.
84. Gilbert A. Dislocation of the MCP joints in children. In: Tubiana R, ed. *The hand*. Vol. 2. Philadelphia: WB Saunders, 1985:922–925.
85. Goldberg B, Rosenthal PP, Robertson LS. Injuries in youth football. *Pediatrics* 1988;81:255–261.
86. Goulesbrough C. A case of fractured scaphoid and os magnum in a bone 10 years old. *Lancet* 1916;2:792.
87. Gonzales MH, Igram CM, Hall RF. Flexible intramedullary nailing for metacarpal fractures. *J Hand Surg [Am]* 1995;20:382–387.
88. Grad JB. Children's skeletal injuries. *Orthop Clin North Am* 1986;3:437–449.
89. Graham TJ, Jacobson PA. Atraumatic palmar midcarpal dislocation in a skeletally immature adolescent with hemiatrophy. *J Hand Surg [Am]* 1999;24:1281–1285.
90. Green DP. Hand injuries in children. *Pediatr Clin North Am* 1977;24:903–918.
91. Green DP, Anderson JR. Closed reduction and percutaneous pin fixation of fractured phalanges. *J Bone Joint Surg [Am]* 1973;55:1651–1653.
92. Green DP, Terry GC. Complex dislocation of the MCP joint. *J Bone Joint Surg. [Am]* 1973;55:1480–1486.
93. Greene MH, Hadies AM, LaMont RL. Scaphoid fractures in children. *J Hand Surg [Am]* 1984;9:536–541.
94. Greene WB, Anderson WJ. Simultaneous fracture of the scaphoid and radius in a child. *J Pediatr Orthop* 1982;2:191–194.
95. Greulich WW, Pyle SI. *Radiographic atlas of skeletal development of the hand and wrist*, 2nd ed. Stanford, CA: Stanford University Press, 1959.
96. Griffiths JC. Bennett's fracture in childhood. *Br J Clin Pract* 1966;20:582–583.
97. Haines RW. The pseudoepiphysis of the first metacarpal of man. *J Anat* 1974;117:145–158.
98. Hakstian RW. Cold-induced digital epiphyseal necrosis in childhood (symmetric focal ischemic necrosis). *Can J Surg* 1972;15:168–178.
99. Hamas RS, Horrell ED, Pierret GP. Treatment of mallet finger due to intraarticular fracture of the distal phalanx. *J Hand Surg [Am]* 1978;3:361–363.
100. Hamlin C. Traumatic disruption of the distal radioulnar joint. *Am J Sports Med* 1977;5:93–96.
101. Hanel DP, Scheid DK. Irreducible fracture–dislocation of the distal radioulnar joint secondary to entrapment of the extensor carpi ulnaris tendon. *Clin Orthop* 1988;234:56–60.
102. Hankin FM, Janda DH. Tendon and ligament attachments in relationship to growth plates in a child's hand. *J Hand Surg [Br]* 1989;14:315–318.
103. Harryman DT II, Jordon TF III. Physeal phalangeal fracture with flexor tendon entrapment. *Clin Orthop* 1990;250:194–196.
104. Hastings H II, Simmons BP. Hand fractures in children. *Clin Orthop* 1984;188:120–130.
105. Heiple KG, Freehafer AA. Isolated traumatic dislocation of the distal end of the ulna or distal radioulnar joint. *J Bone Joint Surg [Am]* 1962;44:1387–1394.
106. Herbert TJ. Use of the Herbert bone screw in surgery of the wrist. *Clin Orthop* 1986;202:79–92.
107. Herbert TJ, Fisher WE. Management of the fractured scaphoid using a new bone screw. *J Bone Joint Surg [Br]* 1984;66:114–123.
108. Herndon JH, ed. *Scaphoid fractures and complications*. AAOS Monograph Series. Park Ridge, IL: American Academy of Orthopaedic Surgeons, 1993:27.
109. Hunt JC, Watts HB, Glasgow JD. Dorsal dislocation of the MCP joint of the index finger with particular reference to open dislocation. *J Bone Joint Surg [Am]* 1967;49:1572–1578.
110. Ireland MD, Taleisnik J. Nonunion of metacarpal extraarticular fractures in children: report of two cases and review of the literature. *J Pediatr Orthop* 1986;6:352–355.
  
- 110a. Jahss SA. Fractures of the metacarpals: a new method of reduction and immobilization. *J Bone Joint Surg* 1938;20:178–186.
  
111. Jones NF, Jupiter JB. Irreducible palmar dislocation of the PIP joint associated with an epiphyseal fracture of the middle phalanx. *J Hand Surg [Am]* 1985;10:261–264.
112. Kaplan EB. Dorsal dislocation of the MCP joint of the index finger. *J Bone Joint Surg [Am]* 1957;39:1081–1086.
113. Kappel DA, Burech JG. The cross-finger flap: an established reconstructive procedure. *Hand Clin* 1985;1:677–684.
114. Kiefhaber TR, Stern PJ. Fracture dislocations of the proximal interphalangeal joint. *J Hand Surg [Am]* 1998;23:368–380.
115. King RJ, MacKenney RP, Elnur S. Suggested method for closed treatment of fractures of the carpal scaphoid: hypothesis supported by dissection and clinical practice. *J R Soc Med* 1982;75:860–867.
116. Kirner J. Doppelseitige Verdrummung des Kleinfingr-grundgleides als Selbständiges Krankheitsbild. *Fortschr Rontgenst* 1927;36:804.
117. Kleinman WB, Bowers WH. Fractures, ligamentous injuries to the hand. In: Bora FW Jr, ed. *The pediatric upper extremity: diagnosis and management*. Philadelphia: WB Saunders, 1988.
118. Kleinman WB, Grantham SA. Multiple volar carpometacarpal joint dislocation. Case report of traumatic volar dislocation of the medial four carpometacarpal joints in a child and review of the literature. *J Hand Surg [Am]* 1978;3:377–382.
119. Koshima I, Soeda S, Takase T, et al. Free vascularized nail grafts. *J Hand Surg [Am]* 1988;13:29–32.
120. Landfried MJ, Stenclik M, Susi JG. Variant of Galeazzi fracture–dislocation in children. *J Pediatr Orthop* 1991;11:332–335.
121. Landin LA. Fracture patterns in children. *Acta Orthop Scand* 1983;202:1–109.
122. Lane CG. Detecting occult fractures of the metacarpal head: the Brewerton view. *J Hand Surg* 1977;2:131–133.
123. Langenskiöld A. Surgical treatment of partial closure of the growth plate. *J Pediatr Orthop* 1981;1:3–11.
124. Larson B, Light TR, Ogden JA. Fracture and ischemic necrosis of the immature scaphoid. *J Hand Surg [Am]* 1987;12A:122–127.
125. Leddy JP, Packer JQ. Avulsion of the profundus tendon insertion in athletes. *J Hand Surg [Am]* 1977;2:66–69.
126. Lee BS, Esterhai JL, Das M. Fracture of the distal radial epiphysis. Characteristics and surgical treatment of premature, post-traumatic epiphyseal closure. *Clin Orthop* 1984;185:90–96.
127. Leonard MH, Dubravcik P. Management of fractured fingers in the child. *Clin Orthop* 1970;73:160–168.
128. Letts M, Rowhani N. Galeazzi-equivalent injuries of the wrist in children. *J Pediatr Orthop* 1993;13:561–566.
129. Light TR. Injury to the immature carpus. *Hand Clin* 1988;4:415–424.
130. Light TR, Ogden JA. Complex dislocation of the index MCP joint in children. *J Pediatr Orthop* 1988;8:300–305.
131. Light TR, Ogden JA. Metacarpal epiphyseal fractures. *J Hand Surg [Am]* 1987;12:460–464.
132. Linscheid RL, Dobyns JH, Beabout W, et al. Traumatic instability of the wrist: diagnosis, classification, and pathomechanics. *J Bone Joint Surg [Am]* 1972;54:1612–1632.
133. Lipscomb PR, Janes JM. 20-Year follow-up on an unreduced dislocation of the first MCP joint in a child. *J Bone Joint Surg [Am]* 1969;51:1216–1218.
134. Littlefield WG, Friedman RL, Urbaniak JR. Bilateral nonunion of the carpal scaphoid in a child: a case report. *J Bone Joint Surg [Am]* 1995;77:124–125.
135. London PS. Sprains and fractures involving the interphalangeal joints. *Hand* 1971;3:155–158.
136. Louis DS, Calhoun TP, Garn SM, et al. Congenital bipartite scaphoid—fact or fiction? *J Bone Joint Surg [Am]* 1976;58:1108–1112.
137. Mack GR, Bosse MJ, Gelberman RH, et al. The natural history of scaphoid nonunion. *J Bone Joint Surg [Am]* 1984;66:504–509.
138. Markiewicz AD, Andrich JT. Hand and wrist injuries in the preadolescent and adolescent athlete. *Clin Sports Med* 1992;11:203–233.
139. Maxted MJ, Owen R. Two cases of nonunion of carpal scaphoid fractures in children. *Injury* 1982;13:441–443.
140. Mayfield JK, Johnson RP, Kilcoyne RK. Carpal dislocations: pathomechanics and progressive perilunar instability. *J Bone Joint Surg [Am]* 1980;5:226–241.
141. McCue FC, Honner R, Johnson MC, et al. Athletic injuries of the PIP joint requiring surgical treatment. *J Bone Joint Surg [Am]* 1970;52:937–956.
142. McElfresh EC, Dobyns JH. Intraarticular metacarpal head fractures. *J Hand Surg [Am]* 1983;8:383–393.
143. McLaughlin HL. Complex "locked" dislocation of the MCP joints. *J Trauma* 1965;5:683–688.
144. McLaughlin HL. Fracture of the carpal navicular (scaphoid) bone: some observations based on treatment by open reduction and internal fixation. *J Bone Joint Surg [Am]* 1954;36:765–774.
145. Melone CP Jr, Grad JB. Primary care of fingernail injuries. *Emerg Med Clin North Am* 1985;3:255–261.
146. Michelinakis E, Vourexaki H. Displaced epiphyseal plate of the terminal phalanx in a child. *Hanc* 1980;12:51–53.
147. Mikic ZD. Age changes in the triangular fibrocartilage of the wrist joint. *J Anat* 1978;126:367–384.
148. Mikic Z. Galeazzi fracture–dislocations. *J Bone Joint Surg [Am]* 1975;57:1071–1080.
149. Minami M, Yamazaki J, Chisaka N, et al. Nonunion of the capitate. *J Hand Surg [Am]* 1987;12:1089–1091.
150. Mino DE, Palmer AK, Levinsohn EM. The role of radiography and computerized tomography in the diagnosis of subluxation and dislocation of the distal radioulnar joint. *J Hand Surg* 1983;8:23–31.
151. Mintzer CM, Waters PM, Brown DJ. Remodelling of a displaced phalangeal neck fracture. *J Hand Surg [Br]* 1994;19:594–596.

152. Mintzer CM, Waters PM. Acute open reduction of a displaced scaphoid fracture in a child. *J Hand Surg [Am]* 1994;19:760–761.
153. Mintzer CM, Waters PM. Surgical treatment of pediatric scaphoid fracture nonunions. *J Pediatr Orthop* 1999;19: 236–239.
154. Mizuta T, Benson WM, Foster BK, et al. Statistical analysis of the incidence of physeal injuries. *J Pediatr Orthop* 1987;7:518–523.
155. Moberg E. Aspects of sensation in reconstructive surgery of the upper extremity. *J Bone Joint Surg [Am]* 1954;46:817–825.
156. Morscher E. Strength and morphology of growth cartilage—hormonal influence of puberty. *Reconstr Surg Traumatol* 1968;10:3–104.
157. Murphy AF, Stark HH. Closed dislocation of the MCP joint of the index finger. *J Bone Joint Surg [Am]* 1967;49:1579–1586.
158. Nafie SAA. Fractures of the carpal bones in children. *Injury* 1987;18:117–19.
159. Nakazato T, Ogino T. Epiphyseal destruction of children's hands after frostbite: a report of 2 cases. *J Hand Surg [Am]* 1986;11:289–292.
160. Neviasser RJ, Wilson JN. Interposition of the extensor tendon resulting in persistent subluxation of the PIP joint of the finger. *Clin Orthop* 1972;83:118–120.
161. Nussbaum R, Sadler AH. An isolated, closed, complex dislocation of the MCP joint of the long finger: a unique case. *J Hand Surg [Am]* 1986;11:558–561.
162. Ogden JA. *Skeletal injury in the child*. Philadelphia: WB Saunders, 1990.
163. Onuba O, Ireland J. Two cases of nonunion of fractures of the scaphoid in children. *Injury* 1984;15:109.
164. Palmer AK, Linscheid RL. Irreducible dorsal dislocation of the distal interphalangeal joint of the finger. *J Hand Surg* 1977;2:406–408.
165. Peimer CA, Sullivan DJ, Wild DR. Palmar dislocation of the PIP joint. *J Hand Surg [Am]* 1984;9:39–48.
166. Peiro A, Martos F, Mut T, et al. Trans-scaphoid perilunate dislocation in a child. A case report. *Acta Orthop Scand* 1981;52:31–34.
167. Pennes DR, Braunstein EM, Shirazi KK. Carpal ligamentous laxity with bilateral perilunate dislocation in Marfan syndrome. *Skel Radio* 1985;13:62–64.
168. Pfitzner W. Beiträge zur Kenntniss des Menschlichen Extremitätenskelets. VII Die Morphologischen Elemente des Menschlichen Hand-Skelets. *Z Morphol* 1900;2:77–157.
169. Phillips JH. Irreducible dislocation of a distal interphalangeal joint: case report and review of literature. *Clin Orthop* 1981;154:188–190.
170. Pick RY, Segal D. Carpal scaphoid fracture and nonunion in an 8-year-old child. *J Bone Joint Surg [Am]* 1983;65:1188–1189.
171. Pohl AL. Irreducible dislocation of a distal interphalangeal joint. *Br J Plast Surg* 1976;29:227–229.
172. Pollen AG. *Fractures and dislocations in children*. Baltimore: Williams & Wilkins, 1973.
173. Poznanski AL, Holt JF. The carpals in congenital malformation syndromes. *Am J Roentgenol* 1971;112:443–459.
174. Prosser AJ, Irvine GB. Epiphyseal fracture of the metacarpal head. *Injury* 1988;19:34–47.
175. Rang M. *Children's fractures*, 2nd ed. Philadelphia: JB Lippincott, 1983:221–232.
176. Rank BK, Wakefield AR. *Surgery of repair as applied to hand injuries*, 2nd ed. Edinburgh: Livingstone, 1960:268–269.
177. Rasmussen F, Schantz K. Lunatomalacia in a child. *Acta Orthop Scand* 1986;57:82–84.
178. Rayan GM, Mullins PT. Skin necrosis complicating finger splinting and vascularity of the distal interphalangeal joint overlying skin. *J Hand Surg [Am]* 1987;12:548–552.
179. Read MT. Stress fractures of the distal radius in adolescent gymnasts. *Br J Sports Med* 1981;15:272–276.
180. Redler I, Williams JT. Rupture of a collateral ligament of the PIP joint of the fingers. *J Bone Joint Surg [Am]* 1967;49:322–326.
181. Robins RHC. Injuries of the MCP joints. *Hand* 1971;3:159–163.
182. Roser LA, Clawson DK. Football injuries in the very young athlete. *Clin Orthop* 1970;69:219–223.
183. Roy S, Caine D, Singer KM. Stress changes of the distal radial epiphysis in young gymnasts. A report of 21 cases and a review of the literature. *Am J Sports Med* 1985;13:301–308.
184. Ruggles D, Peterson H, Scott S. Radial growth plate injury in a female gymnast: a case study. *Med Sci Sports Exerc* 1991;23:393–396.
185. Salamon PB, Gelberman RH. Irreducible dislocation of the interphalangeal joint of the thumb. *J Bone Joint Surg [Am]* 1978;60:400–401.
186. Sanders WE. Evaluation of the humpback scaphoid by computed tomography in the longitudinal axial plane of the scaphoid. *J Hand Surg [Am]* 1988;13:182–187.
187. Sandzen SC. Fracture of the fifth metacarpal resembling Bennett's fracture. *Hand* 1973;5:49–51.
188. Sandzen SC. *Atlas of wrist and hand fractures*. Littleton, MA: PSG, 1979.
189. Sandzen SC, Oakey RS. Crushing injury of the fingertip. *Hand* 1972;4:253–256.
190. Savage R. Complete detachment of the epiphysis of the distal phalanx. *J Hand Surg [Br]* 1990;15:126–128.
191. Schantz K, Rasmussen F. Thiemann's finger or toe disease. *Acta Orthop Scand* 1986;57:91–93.
192. Schenck RR. Dynamic traction and early passive motion for fractures of the PIP joint. *J Hand Surg [Am]* 1986;11:850–858.
193. Segmüller G, Schönenberger F. Fracture of the hand. In: Weber BG, Brunner C, Freuler F, eds. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980:218–225.
194. Selig S, Schein A. Irreducible buttonhole dislocations of the fingers. *J Bone Joint Surg* 1940;22:436–441.
195. Seymour N. Juxtaepiphyseal fractures of the terminal phalanx of the finger. *J Bone Joint Surg [Br]* 1966;48:347–349.
196. Shepard GH. Nail grafts for reconstruction. *Hand Clin* 1990;6:79–102.
197. Shibata M, Seki T, Yoshizu T, et al. Microsurgical toenail transfer to the hand. *Plast Reconstr Surg* 1991;88:102–109.
198. Simmons BP, Lovallo JL. Hand and wrist injuries in children. *Clin Sports Med* 1988;7:495–511.
199. Simmons BP, Stirrat CR. Treatment of traumatic arthritis in children. *Hand Clin* 1987;3:611–625.
200. Smith RJ. Post-traumatic instability of the MCP joint of the thumb. *J Bone Joint Surg [Am]* 1977;59:14–21.
201. Smith RJ, Peimer CA. Injuries to the metacarpal bones and joints. *Adv Surg* 1977;2:341–374.
202. Southcott R, Rosman MA. Nonunion of the carpal scaphoid fractures in children. *J Bone Joint Surg [Br]* 1977;59:20–23.
203. Spanberg O, Thoren L. Bennett's fracture: a new method of treatment with oblique traction. *J Bone Joint Surg [Br]* 1963;45:732–736.
204. Spira E, Farin I. The vascular supply to the epiphyseal plate under normal and pathological conditions. *Acta Orthop Scand* 1967;38:1–22.
205. Stein F. Skeletal injuries of the hand in children. *Clin Plast Surg* 1981;8:65–81.
206. Stener B. Displacement of the ruptured ulnar collateral ligament of the MCP joint of the thumb. A clinical and anatomical study. *J Bone Joint Surg [Br]* 1962;44:869–879.
207. Stener B. Hyperextension injuries of the MCP joint of the thumb—rupture of ligaments, fracture of sesamoid bones, rupture of flexor pollicis brevis. An anatomical and clinical study. *Acta Chir Scand* 1963;125:275–293.
208. Stern PJ. Fractures of the metacarpals and phalanges. In: Green DP, ed. *Operative hand surgery*. New York: Churchill Livingstone, 1993:748.
209. Stern PJ, Roman RJ, Kieffhaber TR, et al. Pilon fractures of the PIP joint. *J Hand Surg [Am]* 1991;16:844–850.
210. Stripling WD. Displaced intraarticular osteochondral fracture—cause for irreducible dislocation of the distal interphalangeal joint. *J Hand Surg* 1982;7:77–78.
211. Stuart HC, Pyle SI, Cornon J, et al. Onsets, completions, and spans of ossification in the 29 bone-growth centers of the hand and wrist. *Pediatrics* 1962;29:237–249.
212. Thomaidis VT. Elbow-wrist-thumb immobilization in treatment of fractures of the carpal scaphoid. *Acta Orthop Scand* 1973;44:679–689.
213. Thompson JS, Eaton RG. Volar dislocation of the PIP joint. *J Hand Surg* 1977;2:232.
214. Thompson JW, Littler JW, Upton J. The spiral oblique retinacular ligament. *J Hand Surg* 1978;3:482–487.
215. Torre BA. Epiphyseal injuries in the small joints of the hand. *Hand Clin* 1988;4:113–120.
216. Vahvanen V, Westerlund M. Fractures of the carpal scaphoid in children. *Acta Orthop Scand* 1980;51:909–913.
217. Vance RM, Gelberman RH, Evans EF. Scaphocapitate fractures. Patterns of dislocation, mechanisms of injury, and preliminary results of treatment. *J Bone Joint Surg [Am]* 1980;62:271–276.
218. Van Herpe LB. Fractures of the forearm and wrist. *Orthop Clin North Am* 1976;7:543–556.
219. Vender M, Watson H. Acquired Madelung-like deformity in a gymnast. *J Hand Surg [Am]* 1988;13:19–21.
220. Vicar AJ. PIP joint dislocations without fractures. *Hand Clin* 1988;4:5–13.
221. Wakeley CPG. Bilateral epiphysis at the basal end of the second metacarpal. *J Anat* 1974;58:340–345.
222. Watson HK, Ballet FL. The SLAC wrist: scapholunate advanced collapse pattern of degenerative arthritis. *J Hand Surg [Am]* 1984;9:358–365.
223. Weber ER, Chao EY. An experimental approach to the mechanism of scaphoid waist fractures. *J Hand Surg* 1978;3:142–148.
224. Wechsler RJ, Wehbe MA, Rifkin MD, et al. Computed tomography diagnosis of distal radioulnar subluxation. *Skel Radio* 1987;16:1–5.
225. Weeks PM. *Acute bone and joint injuries of the hand and wrist. A clinical guide to management*. St. Louis: CV Mosby, 1981.
226. Weiker GG. Hand and wrist problems in the gymnast. *Clin Sports Med* 1992;11:189–201.
227. Wenger DR. Avulsion of the profundus tendon insertion in football players. *Arch Surg* 1973;106:145–149.
228. Whipple TL, Evans JP, Urbaniak JR. Irreducible dislocation of a finger joint in a child. *J Bone Joint Surg [Am]* 1980;62:832–833.
229. White GM. Ligamentous avulsion of the ulnar collateral ligament of the thumb in a child. *J Hand Surg [Am]* 1986;11:669–672.
230. Whitson RO. Carpometacarpal dislocation. A case report. *Clin Orthop* 1955;6:189–195.
231. Winslet MC, Clark NMP, Mulligan PG. Break-dancer's thumb—partial rupture of the ulnar collateral ligament with a fracture of the proximal phalanx of the thumb. *Injury* 1986;17:201–202.
232. Wood VE. Fractures of the hand in children. *Orthop Clin North Am* 1976;7:527–542.
233. Wood VE, Hannah MD, Stilson W. What happens to the double epiphysis in the hand. *J Hand Surg [Am]* 1994;19A:353–360.
234. Worlock PH, Stower MJ. The incidence and pattern of hand fractures in children. *J Hand Surg [Br]* 1986;11:198–200.
235. Worlock P, Stower MJ. Fracture patterns in Nottingham children. *J Pediatr Orthop* 1986;6:656–660.
236. Yanni D, Lieppins P, Laurence M. Fractures of the carpal scaphoid: a critical study of the standard splint. *J Bone Joint Surg [Br]* 1991;73:600–602.
237. Yellin JA, Towbin RB, Kaufman RA. Stubbed finger osteomyelitis. *J Trauma* 1985;25:808–809.
238. Yong-Hing K, Wedge J, Bowen CV. Chronic injury to the distal ulnar and radial growth plates in an adolescent gymnast: a case report. *J Bone Joint Surg [Am]* 1988;70:1087–1089.
239. Young TB. Isolated fracture of the capitate in a 10-year-old boy. *Injury* 1986;17:133–134.
240. Zaricznyj B, Shattuck LJ, Mast TA. Sports-related injuries in school-aged children. *Am J Sports Med* 1980;8:318–324.
241. Zook EG, Guy RJ, Russell RC. A study of nail bed injuries: causes, treatment, and prognosis. *J Hand Surg [Am]* 1984;9:247–252.
242. Zook EG, Russell RC. Reconstruction of a functional and esthetic nail. *Hand Clin* 1990;6:59–68.

## DISTAL RADIUS AND ULNA FRACTURES

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Forearm fractures in children are the most common long bone fractures, comprising about 40% of all pediatric fractures ( [19,20,32](#)). The distal aspect of the radius and ulna is the most common site of fracture in the forearm ( [5,18,19,26,36](#) ). These fractures have been reported to be three times more common in boys; however, the increased participation in athletics by girls at a young age may be changing this ratio. Although these fractures occur at any age, they are most frequent during the adolescent growth spurt ( [2](#)). A direct fall is the usual mechanism of injury. With the wrist and hand extended to protect the child, a fracture occurs if the mechanical force is sufficient. Regardless of the type, these fractures cause pain in the distal forearm, tenderness directly over the fracture site, and limited motion of the wrist and hand. Deformity depends on the degree of fracture displacement. Standard radiographs are diagnostic of fracture type and displacement. Metaphyseal fractures are most common, followed by physeal fractures ( [13,23,32](#)); the distal fragment in either usually is extended. Associated fractures of the hand and elbow regions are rare. Occasionally a direct blow or a fall onto a flexed wrist and hand causes volar displacement or angulation of the distal fragment.

Repetitive loading of the wrist can lead to physeal stress injuries of the distal radius and, less commonly, the ulna. These injuries are rare, and occur most frequently in gymnasts ( [1,4,6,9,21,27,33](#)). Any patient with chronic physeal region wrist pain who participates in an activity with repetitive axial loading of the wrist, such as gymnastics or break dancing ( [15](#)), should be examined for a stress injury.

The pediatric Galeazzi injury usually involves a distal radial metaphyseal fracture and a distal ulnar physeal fracture. These injuries are rare, but need to be identified acutely for proper management. The specifics of injury mechanisms and fracture patterns for individual fracture types are discussed in separate sections of this chapter.

### CLASSIFICATION

Distal radial and ulnar fractures are defined by their anatomic relationship to the physis. Transphyseal injuries are classified by the widely accepted Salter-Harris system ( [88](#)). Metaphyseal injuries may be torus or buckle fractures, greenstick or incomplete fractures, or complete injuries. Pediatric equivalents of adult Galeazzi fracture–dislocations involve a distal radial fracture and either a soft tissue disruption of the distal radioulnar joint (DRUJ) or a transphyseal fracture of the ulna ( [Table 9-1](#)). In contrast to adults, skeletally immature patients rarely sustain intraarticular fractures of the distal radius. On occasion, a Salter-Harris type III fracture, a triplane fracture ( [42](#)), or an adolescent intraarticular Colles' fracture occurs.

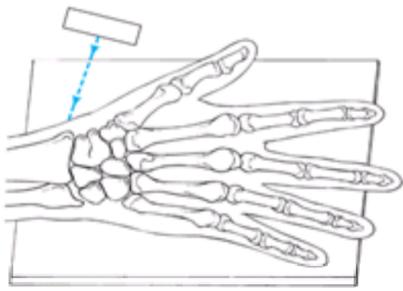
**Physeal fractures**  
   Distal radius  
   Distal ulna  
**Distal metaphyseal (radius or ulna)**  
   Torus  
   Greenstick  
   Complete fractures  
**Galeazzi fracture–dislocations**  
   Dorsal displaced  
   Volar displaced

**TABLE 9-1. DISTAL FOREARM FRACTURES: GENERAL CLASSIFICATION**

Distal radial fracture stability has been more clearly defined in adults ( [35](#)) than in children. At present, an unstable fracture in a child is often defined as one that cannot be reduced closed. Pediatric classification systems have yet to more precisely define fracture stability, but this issue is critical in determining proper treatment

management.

Fractures also are defined by the degree of displacement and angulation. Static anteroposterior (AP) and lateral radiographs can be diagnostic of the fracture type and degree of deformity (Fig. 9-1). In adults, the distal radial articular alignment averages 22 degrees on the AP view and 11 degrees on the lateral view (17,22,24,28,34). Radial inclination is a goniometric measurement of the angle between the distal radial articular surface and a line perpendicular to the radial shaft on the AP radiograph. Palmar tilt is measured by a line across the distal articular surface and a line perpendicular to the radial shaft on the lateral view. Pediatric values for radial inclination tend to be less, depending on the degree of skeletal maturity of the patient. Palmar tilt tends to be more consistent regardless of the age of the patient.



**FIGURE 9-1.** Radiographic angulation of the distal radius. The correct position for a lateral view of the distal radius. The wrist is positioned as for the standard lateral radiograph, but the x-ray beam is directed 15 degrees cephalad. (Redrawn from Johnson PG, Szabo RM. Angle measurements of the distal radius: a cadaver study. *Skel Radio*. 1993;22:243; with permission.)

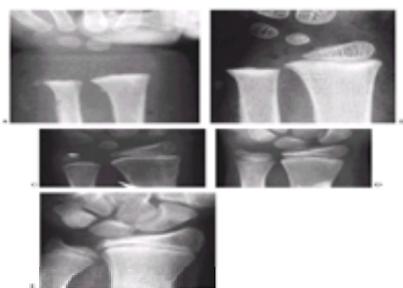
Rarely, tomographic views are necessary to assess intraarticular involvement or displacement ( Fig. 9-2). This can be by AP and lateral tomograms, computerized tomographic (CT) scans, or magnetic resonance imaging (MRI). Dynamic motion studies with fluoroscopy can provide important information on fracture stability and the success of various treatment options (31). Dynamic fluoroscopy requires adequate pain relief and has been used more often in adult patients with distal radial fractures.



**FIGURE 9-2. A:** Computed tomography scan of displaced Salter-Harris type IV fracture. **B:** Surgical correction included external fixation distraction, arthroscopically assisted reduction, and smooth pin fixation.

## ANATOMY

The distal radial epiphysis normally appears between 0.5 and 2.3 years in boys and 0.4 and 1.7 years in girls ( 12). Initially transverse in appearance, it rapidly becomes more adultlike with its triangular shape. The contour of the radial styloid progressively elongates with advancing skeletal maturity. The secondary center of ossification for the distal ulna appears at around age 7. Similar to the radius, the ulnar styloid appears with the adolescent growth spurt. It also becomes more elongated and adultlike until physeal closure. On average, the ulnar physis closes at age 16 in girls and age 17 in boys, whereas the radial physis closes on average 6 months later than the ulnar physis ( 16,30). The distal radial and ulnar physes contribute approximately 75% to 80% of the growth of the forearm and 40% of the growth of the upper extremity (75) (Fig. 9-3).



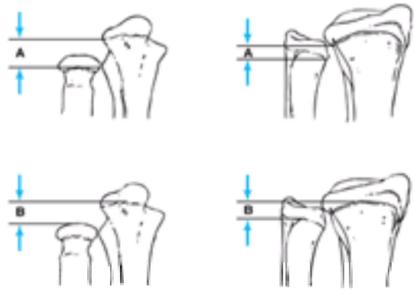
**FIGURE 9-3.** Ossification of the distal radius. **A:** Preossification distal radius with transverse ossification front in a 15-month-old boy. **B:** The triangular secondary ossification center of the distal radius in a 2-year-old girl. The initial ossification center of the styloid in this 7-year-old girl progresses radially ( arrow). **D:** Extension of the ulnar ossification center into the styloid process of an 11-year-old. **E:** The styloid is fully ossified and the epiphyses have capped their relative metaphyses in this 13-year-old boy.

The distal radius articulates with the distal ulna at the DRUJ. Both the radius and ulna articulate with the carpus, serving as the support for the hand. The radial joint surface has three concavities for its articulations: the scaphoid and lunate fossi for the carpus and the sigmoid notch for the ulnar head. These joints are stabilized by a complex series of volar and dorsal radiocarpal, ulnocarpal, and radioulnar ligaments. The volar ligaments are the major stabilizers. Starting radially at the radial styloid, the radial collateral, radioscapohcapitate, radiolunotriquetral, and radioscapohunate ligaments volarly stabilize the radiocarpal joint. The dorsal radioscapohoid and radial triquetral ligaments are less important stabilizers.

The triangular fibrocartilage complex (TFCC) is the primary stabilizer of the ulnocarpal and radioulnar articulations. It extends from the sigmoid notch of the radius across the DRUJ and inserts into the base of the ulnar styloid. It also extends distally as the ulnolunate, ulnotriquetral, and ulnar collateral ligaments and inserts into the ulnar carpus and base of the fifth metacarpal ( 10). The interosseous ligament helps stabilize the radius and ulna more proximally in the diaphysis of the forearm. The ulna remains relatively immobile as the radius rotates around it. The complex structure of ligaments stabilize the radius, ulna, and carpus through the normal wrist.

motion of 120 degrees of flexion and extension, 50 degrees of radial and ulnar deviation, and 150 degrees of forearm rotation ( 10).

The length relationship between the distal radius and ulna is defined as ulnar variance. In adults, this is measured by the relationship of the radial corner of the distal ulnar articular surface to the ulnar corner of the radial articular surface ( 56). However, measurement of ulnar variance in children requires modifications of this technique. Hafner ( 53) described measuring from the ulnar metaphysis to the radial metaphysis to lessen the measurement inaccuracies related to epiphyseal size and shape (Fig. 9-4). If the ulna and radius are of equal lengths, there is a neutral variance. If the ulna is longer, there is a positive variance, and if the ulna is shorter, there is a negative variance.

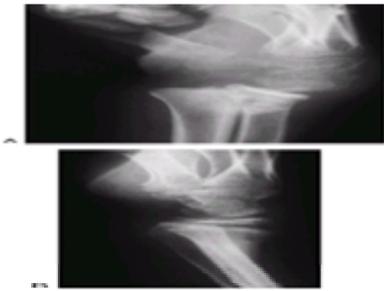


**FIGURE 9-4.** Hafner's technique to measure ulnar variance. **A:** The distance from the most proximal point of the ulnar metaphysis to the most proximal point of the radial metaphysis. **B:** The distance from the most distal point of the ulnar metaphysis to the most distal point of the radial metaphysis. (From Hafner R, Poznanski AK, Donovan JM. Ulnar variance in children. Standard measurements for evaluation of ulnar shortening in childhood. *Skel Radio*. 1989;18:514; with permission.)

Variance is not dependent on the length of the ulnar styloid ( 3), but the measurement is dependent on forearm positioning and radiographic technique ( 8,11,29). Radiographs of the wrist to determine ulnar variance should be standardized with the hand and wrist pronated on the cassette, the elbow flexed 90 degrees, and the shoulder abducted 90 degrees. The importance of ulnar variance relates to the force transmission across the wrist with axial loading. Normally the radiocarpal joint bears approximately 80% of the axial load and the ulnocarpal joint bears 20%. Changes in the length relationship of the radius and ulna alter respective load bearing. Biomechanical and clinical studies have shown that this load distribution is important in fractures, TFCC tears, and Kienbock's disease ( 9,14,25).

## PHYSEAL INJURIES

Distal radial physeal injuries were described more than 100 years ago ( 43,85), and these early descriptions raised concerns regarding permanent deformity from this injury. In the 1930s, however, Aitken ( 37,38) concluded from his observations at the Boston City Hospital outpatient clinic that permanent deformity was rare. Instead, he emphasized the remodeling potential of distal radial physeal fractures, even when not reduced. The observations of Aitken have been confirmed throughout the twentieth century (Fig. 9-5). Most researchers agree that as long as there is sufficient growth remaining, a distal radial extension deformity from a malunited fracture has the potential to remodel. Permanent deformity can occur in malunited fractures near the end of growth or fractures that cause distal radial growth arrest.



**FIGURE 9-5. A:** A 13-year-old boy presented 1 month after injury with a displaced and healed Salter-Harris type II distal radius fracture with obvious clinical deformity. **B:** Over the next 6 months the patient grew 4 inches and the deformity remodeled without intervention.

## Diagnosis

Distal radial physeal fractures are far more common than distal ulnar physeal fractures ( 61,71,79,84,91). The nondominant arm in boys is most commonly injured. The peak incidence is in the preadolescent growth spurt ( 36,61). More than 50% of distal radial physeal fractures have an associated ulnar fracture. This usually is an ulnar styloid fracture but can be a distal ulnar plastic deformation, greenstick, or complete fracture ( 40,65,67). The mechanism of injury generally is a fall on an outstretched hand and wrist. The distal fragment usually displaces dorsally, creating an extension deformity that is usually clinically apparent. Patients have pain and tenderness at the fracture site, and the range of motion at the wrist and hand usually is limited by pain. Neurovascular compromise is uncommon but can occur ( 95). When present, it usually consists of median nerve irritability or dysfunction caused by direct trauma to the nerve at the time of injury or ongoing ischemic compression from the displaced fracture. Thenar muscle function and discriminatory sensibility (two-point discrimination) should be tested before reduction in the emergency setting. Acute carpal tunnel syndrome or forearm compartment syndrome can occur, but more often is caused by marked volar forearm and wrist swelling that occurs after reduction and application of a well-molded, tight cast ( 46,89,95). Open physeal fractures are rare, but the local skin should be examined closely for penetration.

Plain AP and lateral radiographs are diagnostic of the fracture type and deformity. Classification is by the Salter-Harris system for physeal fractures ( 88). Most are Salter-Harris type II fractures. The dorsal displacement of the distal fragment of the epiphysis and dorsal Thurston-Holland metaphyseal fragment is evident on the lateral view (Fig. 9-6). Salter-Harris type I fractures also usually displace dorsally. Volar displacement of either a Salter-Harris type I or II fracture is less common ( Fig. 9-7). Nondisplaced Salter-Harris type I fractures may be indicated only by a displaced pronator fat pad sign ( Fig. 9-8 and Fig. 9-9) ( 90,100) or tenderness over the involved physis ( 40,86). A scaphoid fat pad sign may indicate a scaphoid fracture ( Fig. 9-10). If the acute fracture is unrecognized, a late-appearing periosteal reaction may indicate the fracture.

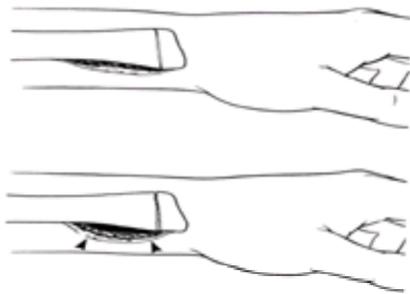


**FIGURE 9-6.** Dorsally displaced physeal fracture (type A). The distal epiphysis with a small metaphyseal fragment is displaced dorsally ( curved arrow) in relation to

the proximal metaphyseal fragment.



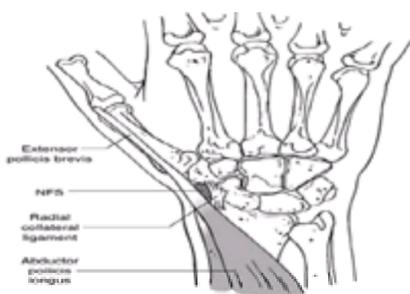
**FIGURE 9-7.** Volarly displaced physeal fracture (type B). Distal epiphysis with a large volar metaphyseal fragment is displaced in a volar direction ( *curved arrow*). (Reprinted from Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:21; with permission.)



**FIGURE 9-8.** Subperiosteal hemorrhage from an occult fracture of the distal radius causes an anterior displacement of the normal pronator quadratus fat pad ( *arrows*).



**FIGURE 9-9.** (A) A 13-year-old girl with tenderness over the distal radius after a fall. The only radiograph finding is an anterior displacement of the normal pronator quadratus fat pad (*arrow*). (B) The opposite normal side (arrow indicates normal fat pad). (C) Two weeks later, there is a small area of periosteal new bone formation (*arrow*) anteriorly, substantiating that bony injury has occurred.

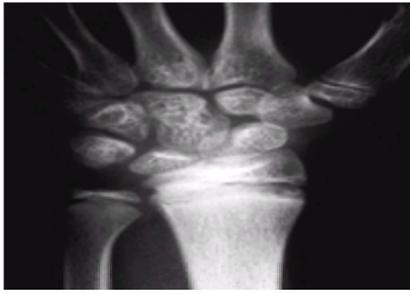


**FIGURE 9-10.** Anatomic relationships of the navicular fat stripe ( *NFS*). The NFS, shaded black, is located between the combined tendons of the abductor pollicis longus and extensor pollicis brevis, and the lateral surface of the carpal navicular. (Reprinted from Terry DW, Ramen JE. The navicular fat stripe. *Ham J Roent Rad Ther Nucl Med* 1975;124:25; with permission.)

Salter-Harris type III fractures are rare and may be caused by a compression injury or an avulsion of the radial origin of the volar radiocarpal ligaments ( [39,65](#)) ([Fig. 9-11](#)). Triplane equivalent fractures ([81](#),–[83](#)), a combination of Salter-Harris type II and III fractures in different planes, are rare. CT scans may be necessary to define the fracture pattern and degree of intraarticular displacement ( [Fig. 9-2](#)). Stress injuries to the physis occur most commonly in competitive gymnasts ([Fig. 9-12](#)).



**FIGURE 9-11.** Anteroposterior radiograph of Salter-Harris type III fracture of the distal radius.



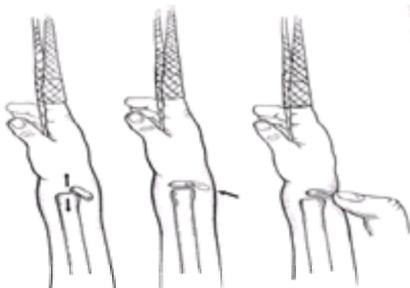
**FIGURE 9-12.** Stress changes in a female gymnast with widening of the distal radial physis from long-standing high-level performance.

### Treatment Options

As for most fractures, treatment options include no reduction, closed reduction and cast immobilization, closed reduction and pin fixation, and open reduction. Nondisplaced fractures are immobilized until appropriate healing and pain resolution have been achieved (40,86). If there is a question of fracture stability, these fractures should be treated with a long arm cast and monitored closely during the first 3 weeks of healing to be certain that there is no loss of alignment. Most displaced Salter-Harris type I and II fractures can be treated successfully in the acute care setting with gentle closed reduction and cast immobilization. Closed reduction and percutaneous pin fixation are performed in patients with neurovascular compromise and displaced physeal fractures (95) to lessen the risk of development of a compartment syndrome in the carpal tunnel or forearm. Open reduction is indicated for irreducible fractures, open fractures, displaced Salter-Harris type III and IV fractures, and triplane equivalent fractures. Irreducible fractures are usually due to an entrapped periosteum or pronator quadratus (70). Internal fixation usually is with smooth pins to lessen the risk of growth arrest. Plates and screws are rarely used unless the patient is near skeletal maturity because of concerns about further physeal injury. In the rare displaced intraarticular Salter-Harris type III or IV fracture, internal fixation can be intraepiphyseal without violating the physis. If it is necessary to cross the physis, then smooth pins should be used to lessen the risk of iatrogenic physeal injury. Extraarticular external fixation also can be used to stabilize and align the fracture.

### Closed Reduction

Most displaced Salter-Harris I and II fractures are treated with closed reduction and cast stabilization. Closed manipulation of the displaced fracture is performed with appropriate sedation, analgesia, or anesthesia to achieve pain relief and an atraumatic reduction (40,58,86). Most of these fractures involve dorsal and proximal displacement of the epiphysis with an apex–volar extension deformity. Manipulative reduction is by gentle distraction and flexion of the distal epiphysis, carpus, and hand over the proximal metaphysis (Fig. 9-13 and Fig. 9-14). The intact dorsal periosteum is used as a tension band to aid in reduction and stabilization of the fracture. Unlike similar fractures in adults, finger trap distraction with pulley weights is often counterproductive. However, finger traps can help stabilize the hand, wrist, and arm for manipulative reduction and casting by applying a few pounds of weight for balance. Otherwise an assistant is helpful to support the extremity in the proper position for casting.



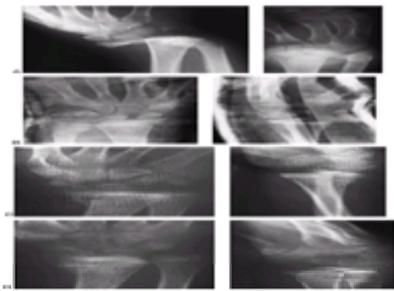
**FIGURE 9-13.** Acceptable method of closed reduction of distal physeal fractures of the radius. **A:** Position of the fracture fragments as finger trap traction with countertraction is applied (arrows). **B:** Often with traction alone the fracture will reduce without external pressure (arrows). **C:** If the reduction is incomplete, simply applying direct pressure over the fracture site in a distal and volar direction with the thumb often completes the reduction while maintaining traction. This technique theoretically decreases the shear forces across the physis during the reduction process.



**FIGURE 9-14.** **A:** Lateral radiograph of dorsally displaced Salter-Harris type II fracture. **B:** Lateral radiograph after closed reduction and cast application. **C:** Reduction of the volar displaced fracture shown in Fig. 9-5. The forearm was in supination with three-point molding anterior over the distal epiphysis and proximal shaft (white arrows). The third point is placed dorsally over the distal metaphysis (open arrow). (The dorsal surface of the cast is oriented toward the bottom of this figure.) (Reprinted from Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:17; with permission.)

If portable fluoroscopy is available, immediate radiographic assessment of the reduction is obtained. Otherwise, a long arm cast is applied and appropriate AP and lateral radiographs are obtained to assess the reduction. The cast should provide three-point molding over the distal radius to lessen the risk of fracture displacement (Fig. 9-13 and Fig. 9-14). The distal dorsal mold should not impair venous outflow from the hand, which can occur if the mold is placed too distal and too deep so as to obstruct the dorsal veins. Postcasting instructions for elevation and close monitoring of swelling and the neurovascular status of the extremity are critical.

The fracture also should be monitored closely with serial radiographs for the first 3 weeks to be certain that there is no loss of anatomic alignment ( [Fig. 9-15](#)). Generally these fractures are stable after closed reduction and cast immobilization. If there is loss of reduction after 7 days, the surgeon should be wary of repeat reduction because of the risk of physeal arrest ( [40,88](#)). Fortunately, remodeling of an extension deformity with growth is common if the patient has greater than 2 years of growth remaining and the deformity is less than 20 degrees ( [Fig. 9-5](#)).



**FIGURE 9-15.** **A:** Anteroposterior and lateral radiographs of severely displaced Salter-Harris type II fracture of the distal radius. **B:** Closed reduction shows marked improvement but not anatomic reduction. The cast had to be bivalved due to excessive swelling. **C:** Unfortunately, the patient lost reduction after a new fiberglass cast was applied. **D:** Out of cast radiographs show a healed malunion in a similar position to the preradiation radiographs.

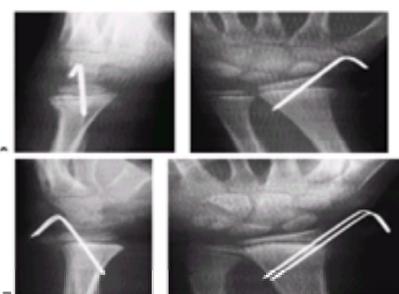
### **Closed Reduction and Percutaneous Pinning**

The indications for percutaneous pinning of distal radial physeal fractures are controversial. The best indication is a displaced radial physeal fracture with median neuropathy and significant volar soft tissue swelling ( [95](#)) ( [Fig. 9-16](#)). These patients are at risk for development of an acute carpal tunnel syndrome or forearm compartment syndrome with closed reduction and cast immobilization ( [46,54,89,95](#)). The torn periosteum volarly allows the fracture bleeding to dissect into the volar forearm compartments and carpal tunnel. If a tight cast is applied with a volar mold over that area, compartment pressures can increase dangerously. Percutaneous pin fixation allows the application of a loose dressing, splint, or cast without the risk of loss of fracture reduction ( [Fig. 9-16](#)).



**FIGURE 9-16.** **A:** Clinical photograph of patient with a displaced Salter-Harris type II fracture of the distal radius. The patient has marked swelling volarly with hematoma and fracture displacement. The patient had a median neuropathy upon presentation. **B:** Lateral radiograph of the displaced fracture. **C:** Lateral radiograph in postoperative splint after percutaneous pinning to lessen the risk of neurovascular compromise in a cast.

Pin fixation can either be single or double ( [Fig. 9-17](#)). Fluoroscopy is used to guide proper fracture reduction and pin placement. Anesthesia is used for adequate pain relief and to lessen the risk of further physeal injury. The fracture is manipulated into anatomic alignment and the initial, and often only, pin is placed from the distal epiphysis of the radial styloid obliquely across the physis into the more proximal ulnar aspect of the radial metaphysis ( [Fig. 9-17](#)). A sufficient skin incision should be made with pin placement to be certain there is no iatrogenic injury to the radial sensory nerve or extensor tendons. Stability of the fracture should be evaluated with flexion/extension and rotatory stress under fluoroscopy. Often in children and adolescents a single pin and the reduced periosteum provide sufficient stability to prevent redisplacement of the fracture. If fracture stability is questionable with a single pin, a second pin should be placed. The second pin can either parallel the first pin or, to create cross-pin stability, can be placed distally from the ulnar corner of the radial epiphysis between the fourth and fifth dorsal compartments and passed obliquely to the proximal radial portion of the metaphysis ( [10,66,92](#)). Again, the skin incisions for pin placement should be sufficient to avoid iatrogenic injury to the extensor tendons.



**FIGURE 9-17.** **A:** Anteroposterior and lateral radiographs of displaced Salter-Harris type II fracture pinned with a single pin. **B:** After reduction and pinning with parallel pins.

The pins are bent, left out of the skin, and covered with a sterile dressing. Splint or cast immobilization is used but does not need to be tight because fracture stability is provided by the pins. The pins are left in until there is adequate fracture healing, usually 4 weeks. The pins can be removed in the office without sedation or anesthesia.

One of the arguments against pin fixation is the risk of additional injury to the physis by a pin ( [42](#)), but this has not been documented. The risk of physeal arrest is more from the displaced fracture than from a short-term, smooth pin. As a precaution, smooth, small-diameter pins should be used, insertion should be as atraumatic as possible, and removal should be executed as soon as there is sufficient fracture healing for fracture stability in a cast or splint alone.

### **Open Reduction**

The main indication for open reduction of a displaced distal radial Salter-Harris type II physeal fracture is irreducibility ( [Fig. 9-18](#)). Most often this is caused by

interposed periosteum or, less likely, pronator quadratus (57,70,99). Open reduction is performed via a volar approach to the distal radial physis. The interval between the radial artery and the flexor carpi radialis is used. This dissection also can proceed directly through the flexor carpi radialis sheath to protect the artery. The pronator quadratus is isolated and elevated from radial to ulnar. Although this muscle can be interposed in the fracture site, the volar periosteum is more commonly interposed. This is evident with elevation of the pronator quadratus. The periosteum is extracted from the physis with care to minimize further injury to the physis. The fracture can then be easily reduced. Cast immobilization is possible, but usually a percutaneous smooth pin is used for stabilization of the reduction. The method of pin insertion is the same as after closed reduction.



**FIGURE 9-18.** Irreducible fracture. This 13-year-old sustained severe trauma producing widely displaced fracture fragments (arrows). Closed reduction could not be performed because of interposed median nerve and deep flexor tendons: open reduction was required. (Courtesy of Earl A. Stanley, M.D.)

Open physeal fractures are rare but do require open reduction. The open wound and fracture site require irrigation and debridement. Care should be taken with mechanical debridement of the physeal cartilage to avoid further risk of growth arrest. Cultures should be taken at the time of operative debridement, and appropriate antibiotics are used to lessen the risk of deep space infection.

The rare Salter-Harris type III or IV fracture or triplane fracture (83) may require open reduction if the joint or physis cannot be anatomically reduced closed. The articular and physeal alignment can be evaluated by radiographic tomograms (trispiral or CT), MRI scans, or wrist arthroscopy (Fig. 9-2). If anatomic alignment of the physis and articular surface is not present, the risk of growth arrest, long-term deformity, or limited function is great (Fig. 9-15). Even minimal displacement (>1 mm) should not be accepted in this situation. Arthroscopically assisted reduction is helpful to align and stabilize these rare physeal fractures (47,50).

## AUTHOR'S PREFERRED METHOD OF TREATMENT

Most Salter-Harris type I and II fractures are reduced closed under conscious sedation with the assistance of portable fluoroscopy. A long arm cast with appropriate three-point molding is applied. This is changed to a short arm cast when there is sufficient healing for fracture stability, usually after 3 to 4 weeks. Cast immobilization is discontinued when there is clinical and radiographic evidence of fracture healing, generally 4 and 6 weeks after fracture. Range-of-motion and strengthening exercises are begun with a home program. When the child achieves full motion and strength, he or she can return to full activity, including competitive sports. Follow-up radiographs are obtained at 6 to 12 months after fracture to be certain there is no growth arrest.

A patient with a displaced Salter-Harris type I or II physeal fracture, significant soft tissue swelling volarly, and median neuropathy (Fig. 9-16) with ipsilateral elbow and radial fractures (Fig. 9-19) is treated with closed reduction and percutaneous pinning. This avoids the increased risk of compartment syndrome in the carpal canal or volar forearm that is present if a well-molded, tight cast is applied. In addition, acute percutaneous pinning of the fracture prevents increased swelling, cast splitting, loss of reduction, and concerns about malunion or growth arrest with repeat reduction (Fig. 9-17). Acute pinning of the fracture with one or two smooth pins through the radial epiphysis provides fracture stability without a compressive cast. The risk of growth arrest from a narrow-diameter, smooth pin left in place for 3 to 4 weeks is exceedingly small.



**FIGURE 9-19. A:** Ipsilateral distal radial physeal and supracondylar fractures. This 6-year-old sustained both a dorsally displaced distal radial physeal fracture (closed arrow) and a type II displaced supracondylar fracture of the humerus (open arrows). **B:** Similar case treated with percutaneous pinning of radial physeal fracture and supracondylar humeral fracture.

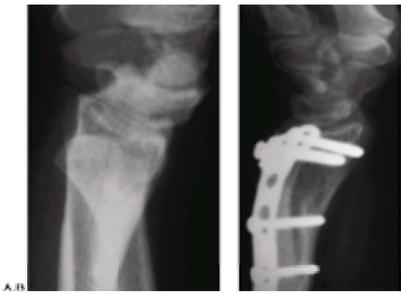
Open reduction is reserved for irreducible Salter-Harris type I and II fractures, open fractures, fractures with associated acute carpal tunnel or forearm compartment syndrome, displaced (>1 mm) Salter-Harris type III or IV fractures, or triplane equivalent fractures. For an irreducible Salter-Harris type I or II fracture, exposure is from the side of the torn periosteum. Because these fractures usually are displaced dorsally, a volar exposure is used. Smooth pins are used for stabilization and are left in for 3 to 4 weeks. Open fractures are exposed through the open wound with proximal and distal extension for adequate debridement. All open debridements are performed in the operating room under general anesthesia. Acute compartment syndromes are treated with immediate appropriate release of the transverse carpal ligament or forearm fascia. The transverse carpal ligament is released in a Z-plasty fashion to lengthen the ligament and prevent volar bow-stringing and scarring of the median nerve against the palmar skin. Displaced intraarticular fractures are best treated with arthroscopically assisted reduction and fixation, but this is an equipment-intensive operation requiring arthroscopic instruments, camera, and monitor, along with fluoroscopy. Distraction across the joint can be achieved with application of an external fixator or finger traps. Standard dorsal portals (3/4 and 4/5) are used for viewing the intraarticular aspect of the fracture and alignment of the reduction (Geisling). In addition, direct observation through the arthroscope can aid in safe placement of the intraepiphyseal pins. Fluoroscopy is used to evaluate the extraarticular aspects of the fracture (triplane equivalent and type IV fractures), the reduction, and placement of fixation pins (Fig. 9-2).

## Complications

### Malunion

Complications from physeal fractures are relatively rare. The most frequent problem is malunion. Fortunately, these fractures often occur in children with significant growth remaining. The deformity from a Salter-Harris type I or II fracture is within the plane of motion of the wrist joint and, therefore, will remodel with ensuing growth (40,58,86) (Fig. 9-5). Repeat reduction should not be performed more than 7 days after fracture because of the risk of growth arrest. The malunited fracture should be monitored over the next 6 to 12 months for remodeling. If the fracture does not remodel, persistent extension deformity of the distal radial articular surface puts the patient at risk for developing mid-carpal instability (96) or degenerative arthritis of the wrist. Corrective osteotomy with bone grafting and internal fixation is required (49) (Fig. 9-20). An opening-wedge dorsal osteotomy is made, iliac crest bone of appropriate trapezoidal shape to correct the deformity is inserted, and either plate or

external fixator is used to maintain correction until healing.

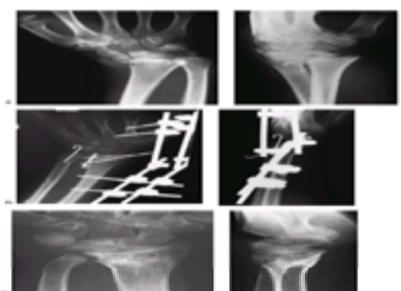


**FIGURE 9-20.** **A:** Radial metaphyseal fracture that did not remodel in a now 16-year-old skeletally mature boy. **B:** Corrective osteotomy with iliac crest bone graft and internal fixation was performed.

Intraarticular malunion is more worrisome ([Fig. 9-21](#) and [Fig. 9-22](#)) because of the risk of development of degenerative arthritis if the articular step-off is more than 2 mm ([59](#)). MRI or CT scans can be useful in preoperative evaluations. Arthroscopy allows direct examination of the deformity and areas of impingement or potential degeneration. Intraarticular osteotomy with bone grafting in the metaphysis to support the reconstructed articular surface is controversial and risky. However, it has the potential of restoring anatomic alignment to the joint and preventing serious long-term complications. This problem fortunately is uncommon in children because of the rarity of the injury and this type of malunion.



**FIGURE 9-21.** **A:** Anteroposterior and lateral radiographs of a 15-year-old skeletally mature boy with a displaced intraarticular fracture. **B:** This fracture needs to be treated like an adult's, with open reduction and internal fixation with a volar buttress plate.



**FIGURE 9-22.** **A:** Anteroposterior and lateral radiographs of a 14-year-old girl who fell from a height. There is extensive intraarticular comminution. **B:** Open reduction, internal fixation, and external fixation was performed. **C:** One year follow-up radiographs reveal early arthrosis. The patient has mild pain and near full range of motion.

### Physeal Arrest

Distal radial physeal arrest can occur from either the trauma of the original injury ([Fig. 9-23](#)) ([55,65,94](#)) or late (>7 days) reduction of a displaced fracture. The exact incidence of radial growth arrest is unknown, but has been estimated to be 7% of all displaced radial physeal fractures. ([65](#)). The trauma to the physeal cartilage from displacement and compression is a significant risk factor for growth arrest. However, a correlation between the risk of growth arrest and the degree of displacement, type of fracture, or type of reduction has yet to be defined. Similarly, the risk of further compromising the physis with late reduction at various time intervals is still unclear. The current recommendation is for an atraumatic reduction of a displaced physeal fracture less than 7 days after injury.



**FIGURE 9-23.** Physeal arrest in a Peterson type I fracture. **A:** Injury film showing what appears to be a benign metaphyseal fracture. Fracture line extends into the physis (*arrows*). **B:** Two years postinjury, a central arrest (*open arrow*) has developed, with resultant shortening of the radius. (Reprinted from Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:21; with permission.)

When a growth arrest develops, the consequences depend on the severity of the arrest and the amount of growth remaining. A complete arrest of the distal radial physis in a skeletally immature patient can be a serious problem. The continued growth of the ulna with cessation of radial growth can lead to incongruity of the DRUJ, ulnocarpal impaction, and development of a TFCC tear ([Fig. 9-24](#)). The radial deviation deformity at the wrist can be severe enough to cause limitation of wrist and

forearm motion (Fig. 9-25). Pain and clicking can develop at the ulnocarpal or radioulnar joints, indicative of ulnocarpal impaction or a TFCC tear. The deformity will progress until the end of growth. Pain and limited motion and function will be present until forearm length is rebalanced; until the radiocarpal, ulnocarpal, and radioulnar joints are restored; and until the TFCC tear and areas of chondromalacia are repaired or debrided ( 72,77,93).



**FIGURE 9-24. A:** Anteroposterior radiograph of ulnar carpal impaction secondary to growth arrest in the distal radius. **B:** Anteroposterior and lateral radiographs after Z-shortening of the ulna to a negative ulnar variance.



**FIGURE 9-25. A:** Anteroposterior radiograph of radial growth arrest and ulnar overgrowth after physeal fracture. Patient complained of ulnar-sided wrist pain and clicking. **B:** Clinical photograph of ulnar overgrowth and radial deviation deformity.

Ideally, physeal arrest of the distal radius will be discovered early before the consequences of unbalanced growth develop. Radiographic screening 6 to 12 months after injury can identify the early arrest. A small area of growth arrest in a patient near skeletal maturity may be clinically inconsequential. However, a large area of arrest in a patient with marked growth remaining can lead to ulnocarpal impaction and forearm deformity if intervention is not performed early. An MRI scan can map the area of arrest (80) (Fig. 9-26). If it is less than 45% of the physis, a bar resection can be attempted ( 63,64). This may restore radial growth and prevent future problems (Fig. 9-27). If the bar is larger than 45% of the physis, an ulnar epiphysiodesis will prevent growth imbalance of the forearm ( 72). The growth discrepancy between forearms in most patients with fractures is minor and does not require treatment.



**FIGURE 9-26. A:** Anteroposterior radiograph of growth arrest with open ulnar physis. **B:** Magnetic resonance imaging scan of large area of growth arrest that was not deemed resectable by mapping.

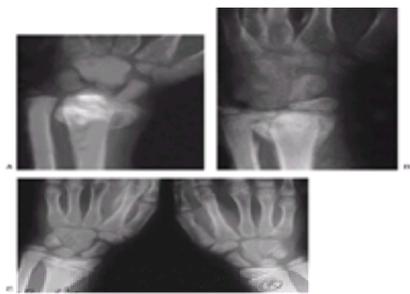


**FIGURE 9-27. Osseous bridge resection. A:** This 10-year-old had sustained a distal radial physeal injury 3 years previously and now complained of prominence of the distal ulna with decreased supination and pronation. **B:** Polytomes revealed a well-defined central osseous bridge involving about 25% of the total diameter of the physis. **C:** The bridge was resected, and autogenous fat was inserted into the defect. Growth resumed with resumption of the normal ulnar variance. Epiphysiodesis of the distal ulna was postponed for 6 months. **D:** Unfortunately, the radius slowed its growth, and a symptomatic positive ulnar variance developed. **E:** This was treated with an epiphysiodesis (*open arrow*) and surgical shortening of the ulna. The clinical appearance and range of motion of the forearm returned to essentially normal.

### ***Ulnocarpal Impaction Syndrome***

The growth discrepancy between the radius and ulna can lead to relative radial shortening and ulnar overgrowth ( Fig. 9-28). The distal ulna can impinge on the lunate and triquetrum and cause pain with ulnar deviation, extension, and compression activities ( 41). Loading the ulnocarpal joint in ulnar deviation and compression will recreate the pain. Radiographs show the radial arrest, ulnar overgrowth, and distal ulnocarpal impingement. The ulnocarpal impaction may be caused by a hypertrophic ulnar styloid fracture union ( Fig. 9-29) or an ulnar styloid nonunion (44,68) (Fig. 9-30). An MRI scan may reveal chondromalacia of the lunate or

triquetrum, a tear of the TFCC, and the extent of the distal radial physeal arrest.



**FIGURE 9-28.** **A:** A markedly displaced Salter-Harris type IV fracture of the distal radius in an 11-year-old boy who fell from a horse. **B:** Film taken 3 weeks after closed reduction demonstrates displacement of the comminuted fragments. **C:** Eighteen months postinjury, there was 15 mm of radial shortening, and the patient had a pronounced radial deviation deformity of the wrist.



**FIGURE 9-29.** Anteroposterior radiograph revealing hypertrophic ulnar styloid healing as the source of the ulnar carpal impaction pain in this patient.



**FIGURE 9-30.** **A:** Anteroposterior radiograph of distal radial growth arrest, ulnar overgrowth, and an ulnar styloid nonunion. Wrist arthroscopy revealed an intact triangular fibrocartilage complex. **B:** Anteroposterior and lateral radiographs after ulnar shortening osteotomy.

Treatment should correct all components of the problem. The ulnar overgrowth is corrected by either an ulnar shortening osteotomy or radial lengthening. Most often a marked degree of positive ulnar variance requires ulnar shortening to neutral or negative variance. If the ulnar physis is still open, a simultaneous arrest should be performed to prevent recurrent deformity. If the degree of radial deformity is marked, this should be corrected by a realignment or lengthening osteotomy. Criteria for radial correction is debatable, but I have used radial inclination of less than 11 degrees on the AP radiograph ([Fig. 9-31](#)) ([72](#)). In the rare case of complete arrest in a very young patient, radial lengthening is preferable to ulnar shortening.



**FIGURE 9-31.** **A:** More severe ulnar overgrowth with dislocation of the distal radioulnar joint and flattening of the radial articular surface. **B:** Intraoperative fluoroscopic view of ulnar shortening and radial osteotomy to corrective deformities.

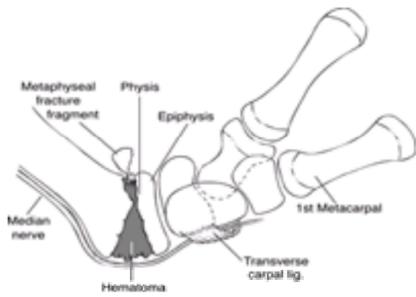
### **Triangular Fibrocartilage Complex Tears**

Triangular fibrocartilage complex tears should be repaired. The presence of an ulnar styloid nonunion is often indicative of an associated peripheral tear of the TFCC ([72,93](#)). The symptomatic ulnar styloid nonunion is excised ([44,68,76](#)) and any TFCC repaired. If physical examination or preoperative MRI scan indicates a TFCC tear in the absence of an ulnar styloid nonunion, an initial arthroscopic examination can define the lesion and appropriate treatment. Peripheral tears (Palmer type B) are the most common TFCC tears in children and adolescents and can be repaired arthroscopically by an outside-in suture technique. Central tears (Palmer type A) are rare in children and can be debrided arthroscopically. Tears off the sigmoid notch (Palmer type D) can be repaired open or arthroscopically. Distal volar tears (Palmer type C) are repaired open, at times with ligament reconstruction.

### **Neuropathy**

Median neuropathy can occur from direct trauma from the initial displacement of the fracture, traction ischemia from a persistently displaced fracture, or the development of a compartment syndrome in the carpal canal or volar forearm ([Fig. 9-32](#)) ([95](#)). All patients with displaced distal radial fractures should undergo a

Careful motor-sensory examination upon presentation to an acute care facility. The flexor pollicis longus, index flexor digitorum profundus, and abductor pollicis brevis muscles should be tested. Light touch and two-point discrimination sensibility of the thumb and index finger should be tested in any child over 5 years of age with a displaced Salter-Harris type I or II fracture. The presence of median neuropathy and marked volar soft tissue swelling are indications for percutaneous pin stabilization of the fracture to lessen the risk of compartment syndrome in a cast.



**FIGURE 9-32.** Volar forearm anatomy outlining the potential compression of the median nerve between the metaphysis of the radius and dorsally displaced physeal fracture. The taut volar transverse carpal ligament and fracture hematoma are also contributing factors. (Redrawn from Waters PM, Kolettis GJ, Schwend R. Acute median neuropathy following physeal fractures of the distal radius. *J Pediatr Orthop* 1994;14:173–177; with permission.)

Median neuropathy caused by direct trauma or traction ischemia generally resolves after fracture reduction. The degree of neural injury will determine the length of time to recovery. Recovery can be monitored with an advancing Tinel's sign along the median nerve. Motor-sensory testing can define progressive return of neural function.

### **Carpal Tunnel Syndrome**

Median neuropathy caused by a carpal tunnel syndrome will not recover until the carpal tunnel is decompressed. After anatomic fracture reduction and pin stabilization, volar forearm and carpal tunnel pressures are measured. Gelberman (51) recommended waiting 20 minutes or more to allow for pressure-volume equilibration before measuring pressures. If the pressures are elevated beyond 40 mm Hg or the difference between the diastolic pressure and the compartment pressure is less than 30 mm Hg (58), an immediate release of the affected compartments should be performed. The carpal tunnel is released through a palmar incision in line with the fourth ray, with care to avoid injuring the palmar vascular arch and the ulnar nerves exiting Guyon's canal. The transverse carpal ligament is released with a Z-plasty to prevent late bow-stringing of the nerve against the palmar skin. The volar forearm fascia is released in the standard fashion.

### **RADIAL PHYSEAL STRESS FRACTURES**

Repetitive axial loading of the wrist in dorsiflexion can lead to physeal stress injuries (Fig. 9-12), almost always involving the radius. Competitive gymnastics is by far the most common cause (6,7,21,33,45,69,87). Other activities reported to cause radial physeal stress fractures include break dancing (15). Factors that predispose to this injury include excessive training, poor techniques, and attempts to advance too quickly in competitive level. Proper coaching is important in preventing these injuries.

A child with a radial physeal stress fracture has recurring, activity-related wrist pain, usually aching and diffuse, in the region of the distal radial metaphysis and physis. Extremes of dorsiflexion and palmar flexion reproduce the pain. There is local tenderness over the dorsal, distal radial physis. Resistive contracture strength testing of the wrist dorsiflexors often reproduces the pain. There may be fusiform swelling about the wrist if there is reactive bone formation. The differential diagnosis includes physeal stress injury, ganglion, ligamentous or TFCC injury, tendonitis or muscle-tendon tear, fracture such as a scaphoid fracture, and avascular necrosis of the scaphoid (Preiser's disease) or lunate (Kienbock's disease). Radiographs may be diagnostic. Physeal widening and reactive bone formation are indicative of chronic physeal stress fracture. Premature physeal closure indicates long-standing stress (27,98). In this situation, continued ulnar growth leads to an ulnar positive variance and pain from ulnocarpal impaction or a TFCC tear (1,33). Normal radiographs may not show an early physeal stress fracture. If the diagnosis is suggested clinically, a bone scan or MRI scan is indicated. Bone scans are sensitive but nonspecific. MRI scans usually are diagnostic.

Treatment first and foremost involves rest. This may be difficult depending on the skill level of the athlete and the desires of the child, coach, and parents. Short arm cast immobilization for several weeks may be the only way to restrict stress to the radial physis in some patients. Splint protection is appropriate in cooperative patients. Protection should continue until there is resolution of pain with examination and activity. The athlete can maintain cardiovascular fitness, strength, and flexibility while protecting the injured wrist. Once the acute physeal injury has healed, return to weight-bearing activities should be gradual. This requires the cooperation of the coach and parents. Adjustment of techniques and training methods often is necessary to prevent recurrence. The major concern is development of a radial growth arrest in a skeletally immature patient. This is an avoidable complication with well-trained coaches and athletes.

If a radial growth arrest has already occurred upon presentation, treatment depends on the degree of deformity and the patient's symptoms. Physeal bar resection usually is not possible because the arrest is usually too diffuse in stress injuries. If there is no significant ulnar overgrowth, a distal ulnar epiphysiodesis will prevent the development of an ulnocarpal impaction syndrome. For ulnar overgrowth and ulnocarpal pain, an ulnar shortening osteotomy is indicated. Techniques include transverse, oblique, and Z-shortening osteotomies. Transverse osteotomy has a higher risk of nonunion than either oblique or Z-shortening and should be avoided. The status of the TFCC also should be evaluated by MRI scan or wrist arthroscopy. If there is an associated TFCC tear, it should be debrided or repaired as appropriate.

### **ULNAR PHYSEAL FRACTURES**

Isolated ulnar physeal fractures are rare injuries. Most ulnar physeal fractures occur in association with radial metaphyseal or physeal fractures. Physeal separations are classified by the standard Salter-Harris criteria. These injuries include types I, II, III, and rarely IV fractures. The rare pediatric Galeazzi injury usually involves an ulnar physeal fracture rather than a soft tissue disruption of the distal radioulnar joint. Another ulnar physeal fracture is an avulsion fracture off the distal aspect of the ulnar styloid (91). Although an ulnar styloid injury is an epiphyseal avulsion, it usually is associated with soft tissue injuries of the TFCC and ulnocarpal joint and does not cause growth-related complications.

Physeal growth arrest is frequent with distal ulnar physeal fractures. The incidence has been cited from 21% (73) to 55% (52). It is unclear why the distal ulna has a higher incidence of growth arrest after fracture than does the radius.

### **Treatment**

Treatment options are similar to those for radial physeal fractures: immobilization alone, closed reduction and cast immobilization, closed reduction and percutaneous pinning, and open reduction. Often these fractures are minimally displaced or nondisplaced. Immobilization until fracture healing at 3 to 6 weeks is standard treatment. Closed reduction is indicated for displaced fractures with more than 50% translation or 20 degrees angulation. Most ulnar physeal fractures reduce to a near anatomic alignment with reduction of the radial fracture. Failure to obtain a reduction of the ulnar fracture may indicate that there is soft tissue interposed in the fracture site. This is an indication for open reduction. Exposure should be from the side of the torn periosteum. The interposed soft tissue (periosteum, extensor tendons, and flexor tendons (48,60,74)) must be extracted from the fracture site. If reduction is not stable, a small-diameter smooth pin can be used to maintain alignment until healing at 3 to 4 weeks. Further injury to the physis should be avoided during operative exposure and reduction because of the high risk of growth arrest (Fig. 9-33, Fig. 9-34 and Fig. 9-35).



**FIGURE 9-33. A and B:** A 10-year-old boy sustained a closed Salter-Harris type I separation of the distal ulnar physis ( *arrows*), combined with a fracture of the distal radial metaphysis. **C:** An excellent closed reduction was achieved atraumatically. **D:** Long-term growth arrest of the distal ulna occurred.



**FIGURE 9-34. A:** The appearance of the distal ulna in the patient seen in [Fig. 9-21](#) 3 years after injury, demonstrating premature fusion of the distal ulnar physis with 3.2 cm of shortening. The distal radius is secondarily deformed, with tilting and translocation toward the ulna. **B:** In the patient in [Fig. 9-21](#) with distal ulnar physal arrest, a lengthening of the distal ulna was performed using a small unipolar distracting device. The ulna was slightly overlengthened to compensate for some subsequent growth of the distal radius. **C:** Six months after the lengthening osteotomy, there is some deformity of the distal ulna, but good restoration of length has been achieved. The distal radial epiphyseal tilt has corrected somewhat, and the patient has asymptomatic supination and pronation to 75 degrees.



**FIGURE 9-35.** Similar case to [Fig. 9-34](#), but with more progressive distal radial deformity treated with corrective osteotomy and epiphysiodesis of the distal radius.

## ULNAR STYLOID FRACTURES

Ulnar styloid avulsion fractures are common in association with radial fractures ([91](#)) and represent a soft tissue avulsion of the attachment of the TFCC or ulnocarpal ligaments. Treatment consists of immobilization and monitoring of long-term outcome, and most heal without sequelae ([62](#)). However, an acute displaced fracture of the base of the styloid represents a disruption of the TFCC. Most of these injuries occur in adolescents with high-velocity trauma at or near skeletal maturity. Treatment should be by tension band reinsertion of the styloid to the metaphysis and repair of the TFCC ([Fig. 9-36](#)). The tension band wire is removed at 3 to 6 weeks.



**FIGURE 9-36. A:** An adolescent with an open, comminuted fracture of the distal radius metaphysis and a base of ulnar styloid fracture with disruption of the triangular fibrocartilage complex (TFCC). **B:** After thorough irrigation and debridement of the radius, internal fixation of the radius was performed with care taken to not violate the physis with hardware. The ulna styloid base fracture and TFCC was repaired with an open tension band technique with a suture and smooth wire.

Some ulnar styloid fractures result in nonunion ([Fig. 9-30](#)) or hypertrophic union ([Fig. 9-29](#)) ([44,68,76,93](#)). Nonunion may be associated with TFCC tears or ulnocarpal impaction. The hypertrophic healing represents an ulnar positive variance and ulnocarpal impaction. Both cause ulnar-sided wrist pain. Compression of the lunate or triquetrum on the distal ulna reproduces the pain. Clicking with ulnocarpal compression or forearm rotation represents either a TFCC tear or chondromalacia of the lunate or triquetrum. Surgical excision of the nonunion or hypertrophic union with repair of the TFCC to the base of the styloid is the treatment of choice. Postoperative immobilization for 4 weeks in a long arm cast followed by 2 weeks in a short arm cast protects the TFCC repair.

### Complications

#### Growth Arrest

The most common complication of distal ulnar physal fractures is growth arrest. Golz ([52](#)) described 18 such fractures, with growth arrest in 10%. If the patient is

young enough, continued growth of the radius will lead to deformity and dysfunction. The distal ulnar aspect of the radial physis and epiphysis appears to be tethered by the foreshortened ulna (Fig. 9-34 and Fig. 9-35). The radial articular surface develops increased inclination toward the foreshortened ulna. This is similar to the deformity Peinado (78) created experimentally with arrest of the distal ulna in rabbits' forelimbs. The distal ulna loses its normal articulation in the sigmoid notch of the distal radius. The metaphyseal-diaphyseal region of the radius often becomes notched from its articulation with the distal ulna during forearm rotation. Frequently, these patients have pain and limitation of motion with pronation and supination (41).

Ideally, this problem is identified before the development of marked ulnar foreshortening and subsequent radial deformity. Because it is well known that distal ulnar physeal fractures have a high incidence of growth arrest, these patients should have serial radiographs to identify growth arrest early. Unfortunately, in the distal ulnar physis, physeal bar resection generally is unsuccessful. Surgical arrest of the radial physis can prevent radial deformity. Usually this occurs late enough in growth that the forearm length discrepancy is not a problem.

Most often these patients present late with established deformity. Treatment then involves rebalancing the length of the radius and ulna. The options include hemiphyseal arrest of the radius, corrective closing wedge osteotomy of the radius, ulnar lengthening (41,52,73), or a combination of these procedures (Fig. 9-34 and Fig. 9-35). The painful impingement of the radius and ulna with forearm rotation can be corrected with reconstitution of the DRUJ. If the radial physis has significant growth remaining, a radial physeal arrest should be performed at the same time as the surgical rebalancing of the radius and ulna. Treatment is individualized depending on the age of the patient, degree of deformity, and level of pain and dysfunction.

## METAPHYSEAL FRACTURES

The metaphysis of the distal radius is the most common site of forearm fracture in children and adolescents (19,32,154). They occur most commonly in boys in the nondominant arm (168). These fractures have a peak incidence during the adolescent growth spurt, which in girls is age 11 to 12 years and in boys is 12 to 13 years (2). During this time of extensive bone remodeling, there is relative osteoporosis of the distal radial metaphysis, which makes this area more susceptible to fracture with a fall.

### Diagnosis

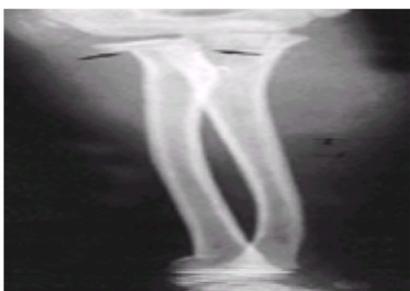
The mechanism of injury is generally a fall on an outstretched hand. The usual dorsiflexion position of the wrist leads to tension failure on the volar side. Fracture type and degree of displacement depend on the height and velocity of the fall (154). These fractures can be nondisplaced torus or buckle injuries (common in younger children with a minimal fall) or dorsally displaced fractures with apex volar angulation (more common in older children with higher velocity injuries) (Fig. 9-37). Displacement may be severe enough to cause foreshortening and bayonet apposition (Fig. 9-38). Rarely, a mechanism such as a fall from a height can cause a distal radial fracture associated with a more proximal fracture of the forearm or elbow (97,143,155) (Fig. 9-39). A fall with a palmar flexed wrist can produce a volarly displaced fracture with apex dorsal angulation (Fig. 9-38, Table 9-2).



**FIGURE 9-37.** Metaphyseal biomechanical patterns. **A:** Torus fracture. Simple bulging of the thin cortex (arrow). **B:** Compression greenstick fracture. Angulation of the dorsal cortex (large curved arrow). The volar cortex is intact but slightly plastically deformed (small white arrows). **C:** Tension failure greenstick fracture. The dorsal cortex is plastically deformed (white arrow) and the volar cortex is complete and separated (black arrows). **D:** Complete length maintained. Both cortices are completely fractured, but the length of the radius has been maintained. (Reprinted from Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:24; with permission.)



**FIGURE 9-38.** Complete fractures; bayonet apposition. **A:** Dorsal bayonet. **B:** Volar bayonet.

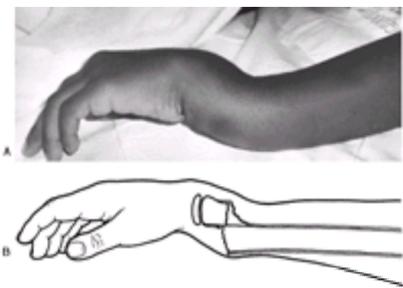


**FIGURE 9-39.** A 10-year-old girl with an innocuous-appearing distal radius fracture associated with an ipsilateral angulated radial neck fracture (arrows).

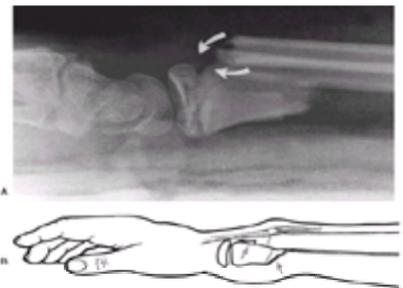
Directional displacement  
 Dorsal  
 Volar  
 Fracture combinations  
 Isolated radius  
 Radius with ulna  
 Ulnar styloid  
 Ulnar physis  
 Ulnar metaphysis, incomplete  
 Ulnar metaphysis, complete  
 Biomechanical patterns  
 Torus  
 Greenstick  
 One cortex  
 Two cortices  
 Complete fracture  
 Length maintained  
 Bayonet apposition

**TABLE 9-2. CLASSIFICATION: DISTAL METAPHYSEAL FRACTURES**

Children with distal radial fractures present with pain, swelling, and deformity of the distal forearm ( Fig. 9-40 and Fig. 9-41). The clinical signs depend on the degree of fracture displacement. With a nondisplaced torus fracture in a young child, medical attention may not be sought until several days after injury, because the intact periosteum is protective in this situation, lessening pain and the child's restriction of activities. Most distal radius fractures, however, will present acutely after the fall with an obvious deformity. Physical examination is limited by the patient's pain and anxiety. It is imperative to obtain an accurate examination of the motor and sensory components of the radial, median, and ulnar nerves before treatment. Median nerve motor function is evaluated by testing the abductor pollicis brevis (intrinsic) and flexor pollicis longus (extrinsic) muscles. Ulnar nerve motor evaluation includes testing the first dorsal interosseous (intrinsic), abductor digit quinti (intrinsic), and flexor digitorum profundus to the small finger (extrinsic) muscles. Radial nerve evaluation involves testing the common digital extensors for metacarpophalangeal joint extension. Sensibility to light touch and two-point discrimination should be tested. Normal two-point discrimination is less than 5 mm but is not present until age 5 to 7 years. Pin-prick sensibility testing will only hurt and scare the already anxious child and should be avoided. A recent prospective study indicated an 8% incidence of nerve injury in children with distal radial fractures ( 168).

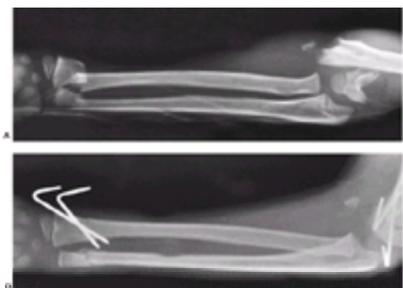


**FIGURE 9-40.** Dorsal bayonet deformity. **A:** Typical distal metaphyseal fracture with dorsal bayonet showing a dorsal angulation of the distal forearm. **B:** Usually the periosteum is intact on the dorsal side and disrupted on the volar side.



**FIGURE 9-41.** Reverse bayonet. **A:** Typical volar bayonet fracture. Often the distal end of the proximal fragment is buttonholed through the extensor tendons ( arrows). (Reprinted from Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:27; with permission.) **B:** Intact volar periosteum and disrupted dorsal periosteum ( arrows). The extensor tendons are displaced to either side of the proximal fragment.

The ipsilateral extremity should be carefully examined for fractures of the carpus, forearm, or elbow ( 107,128,129,133,147,155,160–162) because 3% to 13% of distal radial fractures have associated ipsilateral extremity fractures ( Fig. 9-42) (120,155), increasing the risk of neurovascular compromise and compartment syndrome (Papavasiliou, Staninski, ring).



**FIGURE 9-42.** Ipsilateral fractures. **A:** Markedly displaced ipsilateral distal radial and supracondylar fractures. **B:** Both fractures were reduced and stabilized with pins placed percutaneously. (Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:29; with permission.)

Radiographs are diagnostic of the fracture type and degree of displacement. Standard AP and lateral radiographs usually are sufficient. Complete wrist, forearm, and elbow views are necessary for high-velocity injuries or when there is clinical tenderness. More extensive radiographic studies (CT scan, tomograms) usually are not necessary unless there is intraarticular extension of the metaphyseal fracture in a skeletally mature adolescent.

## Classification

These fractures are classified by fracture pattern, type of associated ulnar fracture, and direction of displacement. Fracture displacement is broadly classified as dorsal or volar. Most distal radial metaphyseal fractures are displaced dorsally with apex volar angulation ( 32) Volar displacement with apex dorsal angulation can occur with palmar flexion injuries.

Metaphyseal fracture patterns are either torus, incomplete or greenstick, or complete fractures. Torus fractures are axial compression injuries. The site of cortical failure is the transition from metaphysis to diaphysis ( 138). These injuries are stable because of the intact periosteum. On rare occasion, they may extend into the physis, putting them at risk for growth impairment ( 80,81). Incomplete or greenstick fractures occur with a combination of compressive and rotatory forces, generally a dorsiflexion force and supination deforming force. This leads to a volar tension side failure and a dorsal compression injury. The degree of force determines the amount of plastic deformation, dorsal comminution, and fracture angulation and rotation. If the force is sufficient, a complete fracture occurs with disruption of both the volar and dorsal cortices. Length may be maintained with apposition of the proximal and distal fragments. Frequently, the distal fragment lies proximal and dorsal to the proximal fragment in bayonet apposition (Table 9-2).

The ulnar fracture often associated with radial metaphyseal fracture can be metaphyseal or physeal, or an ulnar styloid avulsion. Similar to radial metaphyseal fractures, the ulnar fracture can be complete or incomplete.

Distal radial fractures also can occur in conjunction with more proximal forearm fractures ( 101), Monteggia fracture–dislocations ( 102), supracondylar distal humeral fractures (Reis, ring, Staninski), or carpal fractures ( 102,128,129,133,161,162). The combination of a displaced supracondylar distal humeral fracture and a displaced distal radial metaphyseal fracture has been called the pediatric floating elbow. This injury combination is unstable and has an increased risk for malunion and neurovascular compromise.

Pediatric distal radial metaphyseal fractures are not classified by degree of instability. Unstable fractures have been predominately identified retrospectively by the failure to maintain a successful closed reduction (Fig. 9-43). This occurs in approximately 30% of complete distal radial metaphyseal fractures ( 139,146,167). This high percentage of loss of alignment has been tolerated because of the remodeling potential of the distal radius. Anatomic remodeling is possible because the extension deformity is in the plane of motion of the wrist joint, the metaphyseal fracture is juxtaphyseal, and most of these fractures occur while there is still significant growth remaining. However, concern has increased about the high failure rate of closed reduction to maintain anatomic alignment of these fractures. Factors that have been identified as increasing the risk of loss of reduction with closed manipulation and casting include poor casting, bayonet apposition, translation greater than 50% the diameter of the radius, apex volar angulation greater than 30 degrees, isolated radial fractures, and radial and ulnar metaphyseal fractures at the same level (139,146,167). These factors define in general terms unstable fractures.



**FIGURE 9-43.** Results of angulation. **A:** Significant apex volar angulation of the distal fragment. **B:** The appearance was not as apparent cosmetically as in another patient with less angulation that was directed apex dorsally. (Reprinted from Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:27; with permission.)

## Treatment

Treatment options are similar to those for radial physeal fractures: immobilization alone, closed reduction and cast immobilization, closed reduction and percutaneous pinning, and open reduction. The fracture type, degree of fracture instability, associated soft tissue or skeletal trauma, and the age of the patient all influence choice of treatment.

### Torus Fractures

Torus fractures are compression injuries with minimal cortical disruption. If only one cortex is violated, the injury is stable. Treatment should consist of protected immobilization to prevent further injury and relieve pain. Once the patient is comfortable, range-of-motion exercises and nontraumatic activities can begin. Fracture healing usually occurs in 2 to 4 weeks ( 20,40,68,74). Simple torus fractures usually heal without long-term sequelae.

Bicortical disruption on both the AP and lateral views indicates a more severe injury than a stable torus fracture. Splint or limited immobilization in this situation puts the child at risk for displacement. More prolonged immobilization, long arm cast protection in a young patient, and closer follow-up are generally recommended to lessen the risk of malunion. These fractures generally heal in 3 to 6 weeks.

### Incomplete/Greenstick Fractures

#### Immobilization Alone

Treatment of incomplete distal radial and ulnar fractures depends on the age of the patient, the degree and direction of fracture displacement and angulation, the surgeon's biases regarding remodeling, and the surgeon's and community's biases regarding deformity. In younger patients, the remodeling potential of an acute distal radial malunion is extremely high. Acceptable sagittal plane angulation of an acute distal radial metaphyseal fracture has been reported to be from 10 to 35 degrees in patients under 5 years of age ( 74,103,108,141,145,153,168). Similarly, in patients under 10 years of age, the degree of acceptable angulation has ranged from 10 to 25 degrees ( 74,103,108,141,153,168). In patients over 10 years of age, acceptable malunion has ranged from 5 to 20 degrees depending on the skeletal maturity of the patient ( 74,103,108,112,141,153,167,168) (Table 9-3).

Age (yr)	Sagittal Plane		Frontal Plane
	Boys	Girls	Boys & Girls
4-9	20	15	15
9-11	15	10	5
11-13	10	10	0
>13	5	0	0

Acceptable residual angulation is that which will result in total radiographic and functional correction.  
Courtesy B. deCourtivron, M.D., Centre Hospitalier Universitaire de Tours, Tours, France.

**TABLE 9-3. ACCEPTABLE ANGULAR CORRECTIONS IN DEGREES**

The high potential for remodeling of a distal radial metaphyseal malunion has led some researchers to recommend immobilization alone. As mentioned, the range of accepted malalignment has been broad as well as age and researcher dependent ([Fig. 9-44](#) and [Fig. 9-45](#)).



**FIGURE 9-44.** Extensive remodeling. **A:** Injury film of a 7-year-old with a tension failure greenstick fracture. **B and C:** Lateral and anteroposterior views of the same patient taken 1 month later showing development of 45 degrees angulation in the sagittal plane and 40 degrees in the coronal plane. **D and E:** True appearance taken 4 years later shows only residual angulation of 10 degrees in the sagittal plane and full correction of radial angulation in the coronal plane. The patient had a range of forearm motion equal to that of the opposite extremity and was asymptomatic.



**FIGURE 9-45.** Bayonet remodeling. **A:** After numerous attempts at closed reduction, the best alignment that could be obtained was dorsal bayonet apposition in this 8-year-old. **B:** Three months postfracture, there is good healing and early remodeling. **C and D:** Five years after the injury (age 13), remodeling was complete and the patient had normal appearance and forearm motion.

Acceptable frontal plane deformity has been more uniform. The fracture tends to displace radially with an apex ulnar angulation. This does have the potential to remodel ([144](#)), but less so than sagittal plane deformity. Most researchers agree that only 10 degrees or less of acute malalignment in the frontal plane should be accepted. More malalignment than this may not remodel and may result in loss of forearm rotation because of the loss of interosseous space between the radius and ulna ([169](#)) ([Table 9-3](#)).

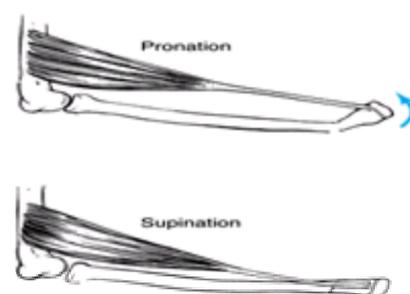
**Closed Reduction**

Most researchers agree that displaced and malaligned incomplete fractures should be reduced closed. The areas of controversy are the degree of acceptable deformity, whether the intact cortex should be fractured, and the position of immobilization.

Controversies about acceptable angulation of the fracture after closed reduction involve the same differences discussed in the immobilization section. As mentioned, more malalignment can be accepted in younger patients, in those with sagittal plane deformity, and in those without marked cosmetic deformity. Malaligned apex volar incomplete fractures are less obvious than the less common apex dorsal fractures.

As Evans ([115](#)) and Rang ([86](#)) emphasized, incomplete forearm fractures have a rotatory component to their malalignment. The more common apex volar fractures represent a supination deformity, whereas the less common apex dorsal fractures are malrotated in pronation. Correction of the malrotation is necessary to achieve anatomic alignment. Controversy exists regarding completion of greenstick fractures ([18,86,111,118,151](#)). Most researchers advocate completion of the fracture to reduce the risk of subsequent loss of reduction from the intact periosteum and concave deformity acting as a tension band ([157,168](#)) to redisplace the fracture. Completing the fracture increases the risk of instability and malunion.

The position and type of immobilization also have been controversial. Recommendations for the position of postreduction immobilization include supination, neutral, and pronation. The rationale for immobilization in pronation is that reduction of the more common apex volar fractures requires correction of the supination deformity ([115](#)). Following this rationale, apex dorsal fractures should be reduced and immobilized in supination. Pollen ([145](#)) believed that the brachioradialis was a deforming force in pronation and was relaxed in supination ([Fig. 9-46](#)) and advocated immobilization in supination for all displaced distal radial fractures. Kasser ([58](#)) recommended immobilization in slight supination to allow better molding of the volar distal radius. Some researchers advocate immobilization in a neutral position, believing this is best at maintaining the interosseous space and has the least risk of disabling loss of forearm rotation in the long term ([113,141,158](#)). Davis and Green ([111](#)) and Ogden ([74](#)) advocate that each fracture seek its own preferred position of stability. Gupta and Danielsson ([130](#)) randomized immobilization of distal radial metaphyseal greenstick fractures in neutral, supination, or pronation to try to determine the best position of immobilization. Their study showed a statistical improvement in final healing with immobilization in supination.



**FIGURE 9-46.** The brachioradialis is relaxed in supination but may become a deforming force in pronation. (Reprinted from Pollen AG. *Fractures and dislocations in*

Another area of controversy is whether long arm or short arm cast immobilization is better. Most publications on pediatric distal radial fracture treatment advocate long arm cast treatment for the first 3 to 4 weeks of healing ([20,40,58,74,103,158](#)). The rationale is that elbow flexion reduces the muscle forces acting to displace the fracture. In addition, a long-arm cast may further restrict the child's activity and therefore decrease the risk of displacement. However, Chess ([104,105](#)) published reported redisplacement and reduction rates with well-molded short arm casts similar to those with long arm casts. They used a cast index (sagittal diameter divided by coronal diameter at the fracture site) of 0.7 or less to indicate a well-molded cast. Wilkins achieved similar results with short-arm cast treatment ([168](#)). However, in most centers, the standard is still long arm cast immobilization ([20,40,58,103,158](#)).

## COMPLETE FRACTURES

Complete fractures of the distal radius, with or without an associated displaced ulnar fracture, are unstable fractures. Generally these fractures are displaced dorsally, tearing the volar periosteum and soft tissues. The distal fragment of epiphysis and metaphysis often is in bayonet apposition with the proximal fragment ([Fig. 9-38](#)). Concomitant radial and ulnar fractures at the same level may be more unstable than isolated fractures ([167](#)) ([Fig. 9-47](#)). However, Gibbons reported loss of reduction in 91% of isolated radial fractures after closed reduction. Although a rare fracture with bayonet apposition in a very young patient may remodel ([168](#)), the standard treatment for completely displaced fractures is reduction and stabilization. The current controversy is whether cast immobilization alone is adequate stabilization or whether percutaneous pin fixation is more appropriate for displaced, complete, distal radial metaphyseal fractures.



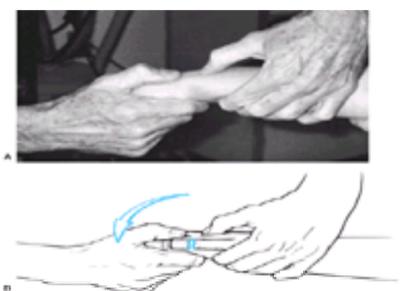
**FIGURE 9-47.** Radial deviation constricts the interosseous space, which may decrease forearm rotation. (Reprinted from Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:28; with permission.)

## Reduction Techniques

Techniques of reduction have included initial distraction with finger traps ([111,157](#)) followed by manipulation and direct manipulation of the fracture by accentuating the deformity ([Fig. 9-48](#) and [Fig. 9-49](#)). Both Rang ([86](#)) and Fernandez ([118](#)) expressed concern about the success of finger trap distraction because the intact dorsal periosteum will not stretch adequately to allow reduction. They advocated sequential reduction maneuvers: initial manipulation of the distal fragment dorsally to accentuate the deformity ([Fig. 9-48](#)), thumb pressure on the relaxed dorsal edge of the distal fragment to correct the overriding, and reduction of the fracture by forceable application of distal and volar pressure ([Fig. 9-49](#)). Anatomic reduction may require repetitive "toggling" of the distal fragment volarly.



**FIGURE 9-48. A and B:** Use of the thumb to push the distal fragment hyperdorsiflexed 90 degrees (*solid arrow*) until length is reestablished. Countertraction is applied in the opposite direction (*open arrows*).

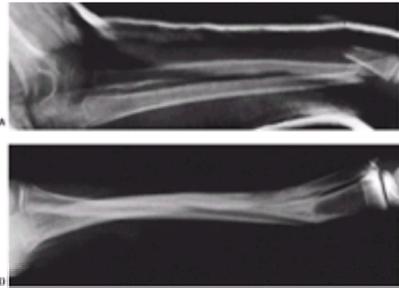


**FIGURE 9-49. A and B:** Once length has been reestablished, the distal fragment is flexed into the correct position. Alignment is checked by determining the position of the fragments with the thumb and forefingers of each hand.

There is considerable controversy about what constitutes an acceptable reduction ([76,110,111,121,123,131,137,148,149,163](#)). This is clearly age dependent, because the younger the patient, the greater the potential for remodeling ([Fig. 9-50](#)). Volar-dorsal malalignment has the greatest potential for remodeling because this is in the plane of predominant motion of the joint. Marked radioulnar malalignment is less likely to remodel. Malrotation will not remodel ([Fig. 9-51](#)). The ranges for acceptable reduction according to age are given in the immobilization section on incomplete fractures and apply to complete fractures as well.



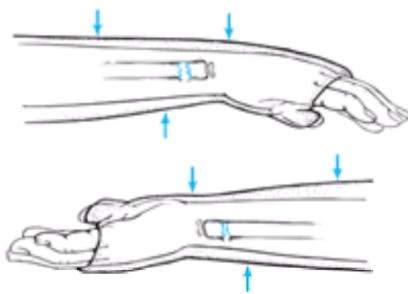
**FIGURE 9-50. A:** Appearance 6 weeks after closed reduction of a distal forearm fracture in an 8½-year-old boy. The radius was reduced, and the ulnar fracture remained overriding. **B:** Eighteen months after injury the ulnar fracture had remodeled completely with symmetric distal radioulnar joints.



**FIGURE 9-51. A:** This 7-year-old with bayonet apposition was immobilized in a long arm cast in full pronation. **B:** One year later, there was still considerable angulation with significant loss of forearm rotation and an unacceptable cosmetic appearance.

### Cast Immobilization

As discussed earlier there is some disagreement regarding short arm or long arm cast immobilization ([104,105,168](#)). However, regardless of the length of the cast, it is imperative to have a well-molded cast over the fracture site ([Fig. 9-52](#)). After reduction of a dorsally displaced fracture, three-point fixation is used with dorsal pressure proximal and distal to the fracture site and volar pressure over the reduced fracture. Excessive swelling should be monitored. If there is any concern regarding impending compartment syndrome, the cast and webril should be immediately bivalved and the patient's clinical status monitored closely.

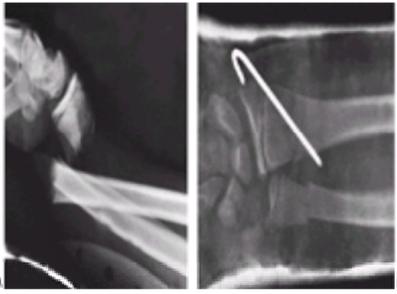


**FIGURE 9-52. Three-point molding. Top:** Three-point molding for dorsally angulated (apex volar) fractures, with the proximal and distal points on the dorsal aspect of the cast and the middle point on the volar aspect just proximal to the fracture site. **Bottom:** For volar angulated fractures, where the periosteum is intact volarly and disrupted on the dorsal surface, three-point molding is performed with the proximal and distal points on the volar surface of the cast and the middle point just proximal to the fracture site on the dorsal aspect of the cast.

The primary problem with closed reduction and cast immobilization is loss of reduction ([Fig. 9-53](#)). Mani et al. ([139](#)) and Proctor et al. ([146](#)) described remanipulation rates of 21.3% and 23.5%, respectively. Mani et al. ([139](#)) concluded that initial displacement of the radial shaft of over 50% was the single most reliable predictor of failure of reduction. Proctor et al. ([146](#)) found that complete initial displacement resulted in a 52% incidence of redisplacement of distal radial fractures in children. Gibbons et al. ([126](#)) noted that completely displaced distal radius fractures with intact ulnas had a remanipulation rate of 91% after closed reduction and cast immobilization alone versus a 0% rate of remanipulation when the same fractures were treated with closed reduction, Kirschner wire fixation, and cast immobilization. All three researchers strongly advocated percutaneous pinning of distal radial fractures at risk of redisplacement ([Fig. 9-54](#)). Widmann and Waters ([167](#)) prospectively studied all distal radial fractures in children requiring reduction over the course of 1 year (POSNA). Of the 86 distal radial metaphyseal fractures, 31% lost reduction and required further intervention with repeat reduction, casting, or pinning. In patients over 10 years of age with angulation of more than 30 degrees, the remanipulation rate was 75%. These findings led to a more recent prospective, randomized study by Waters et al. ([166](#)) of distal radial metaphyseal fractures treated by either closed reduction and cast immobilization or closed reduction and percutaneous pinning. Selection criteria were a closed metaphyseal fracture angulated more than 30 degrees in a skeletally immature patient over 10 years of age. To maximize the outcome of the cast immobilization group, these patients were treated by a member of the Pediatric Orthopedic Society of North America with expertise in trauma care. General anesthesia, fluoroscopic control, and a long arm cast were used. Despite these optimal conditions, 7 of 18 patients in the cast immobilization group lost reduction and required remanipulation.



**FIGURE 9-53. Serial radiographs at 3 days (A) and 10 days (B) revealing slow loss of reduction that is commonplace after distal radial metaphyseal fracture closed reduction.**



**FIGURE 9-54.** Severe swelling. **A:** An 11-year-old with marked displacement and severe swelling from a high-energy injury. **B:** Once reduced, the fragment was secured with an oblique percutaneous pin across the fracture site. (Reprinted from Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:29, with permission.)

The results of all of these studies indicate that distal radial metaphyseal fractures with initial displacement of more than 30 degrees are inherently unstable. Loss of reduction is common, with the risk in the 30% to 40% range. Incomplete reduction ([139,146](#)) and poor casting techniques ([104,105,168](#)) increase the risk of loss of reduction. In addition, the risk of loss of reduction increases with the age of the patient and the degree of initial displacement.

Loss of reduction requires repeat manipulation or it will result in a malunion. Although the rate of malunion is frequent after these fractures ([40,46,54,58,67,89,104,105,109,111,165](#)), because of the potential for remodeling in skeletally immature patients, it has not been considered a serious problem ([17,37,38,103,121–124](#)). Distal radial fractures are juxtaphyseal, the malunion often is in the plane of motion of the wrist joint (dorsal displacement with apex volar angulation), and the distal radius accounts for 60% to 80% of the growth of the radius. All these factors favor remodeling of a malunion. However, deCourtivron et al. ([112](#)) reported that of 602 distal radial fractures, 14% had an initial malunion of more than 5 degrees. Of these, 78% corrected the frontal plane deformity and only 53% remodeled completely in the sagittal plane. In addition, 37% had loss of forearm rotation.

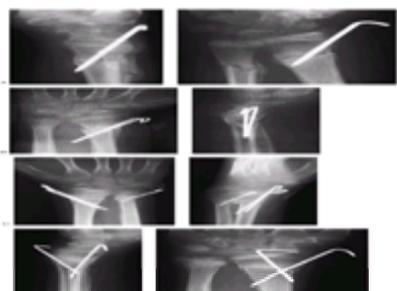
### Closed Reduction and Percutaneous Pinning

In the past 10 years, closed reduction and percutaneous pinning have become more common as the primary treatment of distal radial metaphyseal fractures in children and adolescents ([125,139,146,166,167](#)). The indications cited include fracture instability and high risk of loss of reduction ([125,139,146](#)), excessive local swelling that increases the risk of neurovascular compromise ([95,166,168](#)), ipsilateral fractures of the distal radius and elbow region (floating elbow) that increase the risk of compartment syndrome ([Fig. 9-43](#)) ([155,169](#)), and any remanipulation ([Fig. 9-55](#)) ([167,168](#)).



**FIGURE 9-55.** Remanipulation. **A and B:** Two weeks after what appeared to be initially an undisplaced greenstick fracture, a 14-year-old boy was found to have developed late angulation of 30 degrees in both the coronal and sagittal planes. **C:** Because this was beyond the limits of remodeling, a remanipulation was performed. To prevent reangulation, the fracture was secured with a pin placed percutaneously obliquely through the dorsal cortex.

Pinning usually is done from distal to proximal under fluoroscopic guidance. When possible, the physis is avoided. Adequate exposure should be obtained to avoid radial sensory nerve or extensor tendon injury. Smooth Kirschner or **C**-wires are used. In younger patients, a single pin with supplemental cast immobilization may be adequate fixation. Crossed pins are more stable ([Fig. 9-56](#)). The first pin, or single pin, enters from the radial side distal to the fracture and passes obliquely to the ulnar aspect of the radius proximal to the fracture. The second pin enters the radius distal to the fracture between the fourth and fifth compartments and passes obliquely across the fracture into the proximal radial side of the radius. The pins are left out through the skin to allow easy removal in the ambulatory setting. A supplemental, loose-fitting cast is applied. The advantage of pin fixation is that a tight, well-molded cast is not necessary to maintain reduction. This lessens the risk of neurovascular compromise with associated excessive swelling or ipsilateral fractures. Obviously, pin fixation avoids the risk of loss of reduction in an unstable fracture. Pinning does have the risk of infection and concerns regarding growth injury.



**FIGURE 9-56.** Different pinning techniques for unstable distal radius and ulnar metaphyseal fractures. **A:** A single oblique pin in the radius. **B:** Two parallel pins in the radius. **C:** Two parallel pins in the radius and a single pin in the ulna. **D:** Crossed pins in the radius.

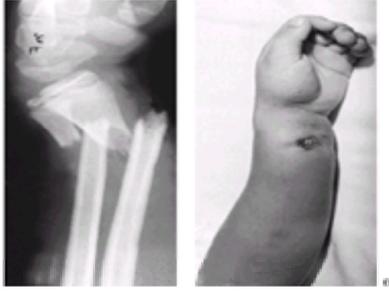
### External Fixation

Unlike distal radial fractures in adults, external fixation rarely is indicated in skeletally immature patients. Although it can be used successfully ([152,165](#)), the success rates of both closed reduction and percutaneous pinning techniques make it unnecessary for uncomplicated distal radial fractures in children. The best indication is severe associated soft tissue injuries. A severe crush injury, open fracture, or replantation after amputation that requires extensive soft tissue care and surgery are all indications for the use of external fixation. Supplemental external fixation also may be necessary for severely comminuted fractures to maintain length and provide additional stability to pin fixation ([Fig. 9-22](#)). Standard application of the specific fixator chosen is performed with care to avoid injury to the adjacent sensory nerves

and extensor tendons.

### Open Reduction

Open reduction is indicated for open or irreducible fractures ( [Fig. 9-36](#), [Fig. 9-57](#), and [Fig. 9-58](#)). Open fractures constitute approximately 1% of all distal radial metaphyseal fractures. All open fractures, regardless of grade of soft tissue injury, should be irrigated and debrided in the operating room. The open wound should be enlarged adequately to debride the contaminated and nonviable tissues and protect the adjacent neurovascular structures. After thorough irrigation and debridement, the fracture should be anatomically reduced and stabilized, usually with two smooth pins. If the soft tissue injury is severe, supplemental external fixation allows for observation and treatment of the wound without jeopardizing the fracture reduction. The original open wound should not be closed primarily. Appropriate prophylactic antibiotics should be used depending on the severity of the open fracture.



**FIGURE 9-57.** Open fractures. Radiograph (A) and clinical photo (B) of an open fracture of the distal radius. This patient needs formal irrigation and debridement in the operating room.



**FIGURE 9-58.** A 10-year-old girl with a markedly displaced closed fracture of the distal radius with an angulated ulnar fracture. Note the wide separation between the radial fragments. Dimpling of the skin was noted when longitudinal traction was applied and reduction was impossible. At open reduction, the proximal fragment was buttonholed through the forearm fascia and located between the median nerve and finger flexor tendons. The pronator quadratus muscle was also interposed between the two fragments.

Irreducible fractures are rare ([Fig. 9-58](#)) and generally are secondary to interposed soft tissues. With dorsally displaced fractures, the interposed structure usually is the volar periosteum or pronator quadratus ([132](#)) and rarely the flexor tendons or neurovascular structures. In volarly displaced fractures, the periosteum or extensor tendons may be interposed. The fracture should be approached in a standard fashion opposite the side of displacement (i.e., volar approach for an irreducible dorsal fracture). The adjacent neurovascular and tendinous structures are protected and the offending soft tissue is extracted from the fracture site. Pin stabilization is recommended to prevent problems with postoperative swelling or loss of reduction in cast.

Closed reduction rarely fails if there is no interposed soft tissue. However, occasionally multiple attempts at reduction of a bayonet apposition fracture can lead to significant swelling that makes closed reduction impossible. If the patient is too old to remodel bayonet apposition, open reduction is appropriate. Pin fixation without violating the physis is recommended.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

### Nondisplaced Fractures

Nondisplaced metaphyseal compression fractures, including torus and unicortical compression greenstick fractures, are inherently stable. These include torus and unicortical compression greenstick fractures. Immobilization is used until resolution of pain and radiographic evidence of healing, generally about 3 weeks. Depending on the activity level of the patient, a volar wrist splint or a short arm cast can be used. Immobilization provides comfort from pain during healing and protects against displacement with secondary injury. It is important that an unstable bicortical fracture not be unrecognized on radiograph. Bicortical fractures need more protection, longer restriction of activity, and closer follow-up to avoid displacement and malunion. A well-molded long arm cast is applied and radiographs obtained every 7 to 10 days until evidence of early radiographic healing. A short-arm cast is then worn until clinical and radiographic healing is complete. Any loss of reduction is treated with repeat reduction. Return to contact sports is restricted until the patient regains full motion and strength.

### Minimally Displaced Fractures

Displaced greenstick fractures that are reduced are at risk for redisplacement. If left unreduced or poorly immobilized, a mild deformity can become severe during the course of healing. Therefore, closed anatomic reduction is performed in all bicortical fractures with more than 10 degrees of malalignment. Generally these fractures have apex volar angulation and dorsal displacement. Conscious sedation is used with portable fluoroscopy in the emergency care setting. The distal fragment and hand are distracted and then reduced volarly. With isolated distal radial fractures, it is imperative to reduce the DRUJ with appropriate forearm rotation. For apex volar fractures, this usually is with pronation. If the fracture is apex dorsal with volar displacement, the reduction forces are the opposite. A long arm cast with three-point molding is used for 3 to 4 weeks. Radiographs are obtained every 7 to 10 days until there is sufficient callus formation. A short arm cast or volar wrist splint is used until full healing, generally at 4 to 6 weeks after fracture reduction. The patient is then restricted from contact sports until full motion and strength are regained, which may take up to 3 weeks after cast removal. Formal therapy rarely is required. The patient and parents should be warned at the start of treatment of the risk of redisplacement of the fracture.

### Bayonet Apposition

Marked displacement of distal radial metaphyseal fractures usually results in foreshortening and dorsal overlap of the distal fragment on the proximal fragment. This often is associated with a same-level ulnar metaphyseal fracture, similarly in bayonet apposition. Rarely, the distal fragment is in volar bayonet apposition. Both of these situations require more skill of reduction and complete analgesia at the fracture site. At our institution, we reduce this fracture in the operating room with general anesthesia or in the emergency room with conscious sedation and supplemental local hematoma block. In either situation, portable fluoroscopy is used. The fracture usually is reduced in the emergency room in young patients with minimal swelling and no neurovascular compromise, and in whom cast treatment will be sufficient. Reduction with general anesthesia is preferred for older patients and for those with marked displacement, swelling, or associated neurovascular compromise in whom

percutaneous pin treatment is chosen.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

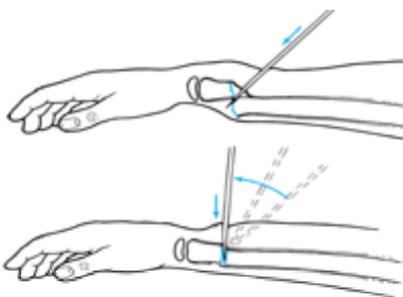
The reduction maneuver is the same regardless of anesthesia type or stabilization method. As opposed to a Colles' fracture in an adult, traction alone will not reduce the fracture because the dorsal periosteum acts as a tension band that does not respond to increasing linear traction with weights. Finger traps with minimal weight (less than 10 pounds) can be used to balance the hand and help with rotational alignment (the "steel resident") ( [Fig. 9-59](#)). However, applying progressive weight will only distract the carpus and will not alter the fracture alignment.



**FIGURE 9-59.** Once reduced, the fracture is maintained with finger trap traction and countertraction on the arm. The quality of reduction is assessed quickly with the image intensifier.

After applying preliminary traction with either light-weight finger traps or hand traction, a hyperdorsiflexion maneuver is performed ( [Fig. 9-48](#)). The initial deformity is accentuated and the distal fragment is brought into marked dorsiflexion. The dorsum of the hand should be brought more than 90 degrees, and at times parallel to the dorsum of the forearm to lessen the tension on the dorsal forearm. Thumb pressure is used on the distal fragment while still in this deformed position ( [Fig. 9-48](#)) to restore length by bringing the distal fragment beyond the proximal fragment. Reduction is then obtained by flexing the distal fragment while maintaining length ( [Fig. 9-49](#)). Often this initial reduction maneuver restores length and alignment, but translational reduction is incomplete. The fracture should be completely reduced by toggling the distal fragment all the way volarly by repetitive slight dorsiflexion positioning of the distal fragment followed by volar pressure with the thumbs. It is important to anatomically reduce the fracture. Loss of reduction with cast immobilization is more likely if the fracture remains translated or malaligned.

If the patient presents late with marked swelling and the reduction is difficult, it is useful to try to lever the proximal fragment distally with a percutaneous smooth wire ( [Fig. 9-60](#)). This may prevent an unnecessary open reduction. Percutaneous pin fixation is used after reduction.



**FIGURE 9-60.** Pin leverage. **A:** If a bayonet is irreducible, after sterile preparation, a chisel-point Steinmann pin can be inserted between the fracture fragments from a dorsal approach. Care must be taken not to penetrate too deeply past the dorsal cortex of the proximal fragment. **B:** Once the chisel is across the fracture site, it is levered into position and supplementary pressure is placed on the dorsum of the distal fragment ( *arrow*) to slide it down the skin into place. This procedure is usually performed with an image intensifier.

### Cast Treatment

If the patient is under 10 years of age, has no prereluction signs or symptoms of neurovascular impairment, or has minimal swelling, then cast immobilization is used ( [Fig. 9-61](#) and [Fig. 9-62](#)). I use a long arm cast. The cast is applied with the aid either of an assistant or finger traps and balancing counter weights on the upper arm ( [Fig. 9-59](#)). The advantage of the finger trap steel resident is that there is no risk of muscle fatigue, mental distraction, or failure to maintain elbow flexion at 90 degrees that can occur with a human assistant. The cast is applied with the elbow flexed 90 degrees, the wrist in slight palmar flexion, and the forearm in the desired rotation for stability and alignment. This varies with each fracture and each surgeon. My preference is slight supination (20–30 degrees) unless the fracture dictates differently. This allows excellent molding against the volar aspect of the distal radius at the fracture site.



**FIGURE 9-61.** **A:** Lateral radiograph of displaced metaphyseal radius and ulna fractures. **B:** Anteroposterior and lateral healed radiographs with anatomic alignment after closed reduction.



**FIGURE 9-62.** Anteroposterior and lateral radiographs of anatomic alignment with closed reduction of a distal radius metaphyseal fracture.

One of the most important elements in a successful long arm cast for a forearm fracture is the application of the webriil. The webriil should be applied in a continuous roll with overlap of one third to one half its width. Extra padding is applied over the olecranon, along the volar and dorsal forearm where the cast may have to be split, and at the ends of the cast to prevent irritation from fraying. Plaster of paris is used for the cast to obtain the best mold possible. Initially a single layer is applied, followed by splints five layers thick along the volar and dorsal forearm and the extension region of the elbow. This lessens the bulk of the cast and still allows deep molds. The cast is completed with plaster of paris rolls over the splints. A three-point mold is applied at the fracture site ( [Fig. 9-61](#) ) as the cast hardens. In addition, molds are applied to maintain a straight ulnar border, the interosseous space, and straight posterior humeral line. This creates the classic “box” long arm cast rather than the all too frequent “banana” cast that allows for displacement. Final radiographs are obtained, and if the reduction is anatomic, the cast is overwrapped with fiberglass to lessen the weight, increase patient satisfaction, and prevent cast breakdown that could lead to loss of reduction.

Patients are either discharged or admitted to the hospital depending on the degree of concern regarding risk of excessive swelling, neurovascular compromise, and patient and parental reliability. If there is any doubt, the patient is admitted for observation. The cast is split anytime there are signs of neurovascular compromise. The patient is instructed to maintain elevation for at least 48 to 72 hours after discharge and return immediately if excessive swelling or neurovascular compromise occurs. The patient and family are warned of the risk of loss of reduction and the need for close follow-up. We inform our patients and parents that the risk of return to the day surgery unit for repeat reduction is approximately 30% during the first 3 weeks.

Follow-up examinations and radiographs are obtained every 7 to 10 days for 3 weeks. If there is loss of reduction, I individualize treatment depending on the patient's age, degree of deformity, time since fracture, and remodeling potential ( [Fig. 9-53](#) ). If restoration of alignment with growth occurs, I reassure the family that the child should achieve anatomic alignment over time with growth. If the child is older, there is risk of further displacement, or the deformity is marked, repeat reduction in the day surgery unit with fluoroscopy is performed. Most often, a percutaneous pin is used for the second reduction ( [Fig. 9-55](#) ).

Cast immobilization usually is for 4 to 6 weeks. The long arm cast is changed to a short arm cast at 3 to 4 weeks. With clinical and radiographic healing ( [Fig. 9-61](#) and [Fig. 9-62](#) ), a protective volar splint is used and activities are restricted until the patient regains full motion and strength, usually in 1 to 3 weeks after cast removal. As with other distal radial fractures, formal physical therapy is rarely required.

#### Percutaneous Pin Fixation

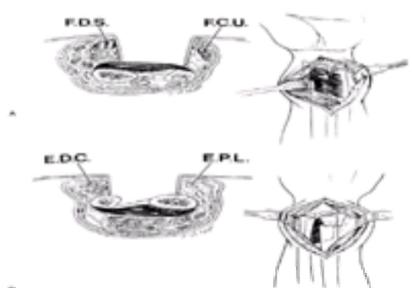
Percutaneous pinning of distal radial metaphyseal fractures is most often used in patients with excessive swelling or signs of neurologic injury. In these situations, the patient is at risk for development of a forearm or carpal tunnel compartment syndrome with a well-molded, tight-fitting cast. Similarly, concurrent displaced supracondylar and distal radial fractures are treated with percutaneous fixation of both fractures to lessen the risk of neurovascular compromise. Older patients near the end of growth with bayonet apposition fractures also are treated with percutaneous pin fixation because they have less ability to remodel and their fractures are very unstable with a high risk of displacement. Finally, open fractures usually are treated with pin fixation.

The pinning technique for the radius is either a single radial-sided pin or crossed radial- and ulnar-sided pins. Fixation of the ulna rarely is necessary ( [Fig. 9-56](#) ). Stability with a single pin is checked with fluoroscopy, and if further fixation is needed, a second pin is added. The physis is avoided if possible. A small incision is made for the insertion of each pin to protect the radial sensory nerve and adjacent extensor tendons. Smooth pins are used and are removed in the office as soon as there is sufficient healing to make the fracture stable in a cast or splint, usually at 4 weeks. Rehabilitation is similar to that for cast-treated fractures.

#### Open Reduction

The two most common indications for open reduction are an open fracture (Haasbeck) and an irreducible fracture. All open fractures are irrigated and debrided in the operating room. The initial open wound is extended adequately to inspect and cleanse the open fracture site. After thorough irrigation and debridement, the fracture is reduced and stabilized. A cast is rarely applied in this situation because of concern about fracture stability, soft tissue care, and excessive swelling. Crossed pin fixation often is used with Gustib grade 1 or 2 open fractures. More severe soft tissue injuries usually require external fixation with a unilateral frame, with care taken to avoid soft tissue impingement during pin placement. If flap coverage is necessary for the soft tissue wounds, the fixator pins should be placed in consultation with the microvascular surgeon planning the soft tissue coverage.

Irreducible fractures are usually secondary to soft tissue entrapment. With dorsal displacement, this is most often either the volar periosteum or pronator quadratus, and open reduction through a volar approach is necessary to extract the interposed soft tissues and reduce the fracture ( [Fig. 9-63](#) ). Percutaneous pin fixation usually is used to stabilize the fracture in patients with open physes. If plate fixation is used, it should avoid violation of the physis ( [Fig. 9-36](#) ). Displaced intraarticular injuries in skeletally immature patients are adultlike and require standard treatment such as open reduction and internal fixation ( [Fig. 9-21](#) ) or combination treatment with internal and external fixation ( [Fig. 9-22](#) ).



**FIGURE 9-63. A:** Volar approach through the interval between the digital flexors and ulnar neurovascular bundle. *FDS*, flexor digitorum superficialis; *FCU*, flexor carpi ulnaris. **B:** Dorsal approach between the third and fourth dorsal compartments. *EDC*, extensor digitorum communis; *EPL*, extensor pollicis longus. (Reprinted from Holmes JR, Luis DS. Entrapment of pronator quadratus in pediatric distal-radius fractures: recognition and treatment. *J Pediatr Orthop* 1994;14:498—500; with permission.)

#### Complications

Distal radial metaphyseal fractures have complications similar to physeal fractures but with different frequencies. Loss of reduction and malunion are the most

common problems, and growth-related complications are infrequent. Neurovascular compromise does occur and should be considered in the acute management of this fracture.

### **Malunion**

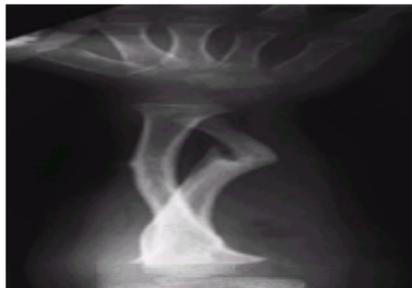
Loss of reduction is a common complication of distal radial metaphyseal fractures treated with cast immobilization. Because this complication occurs in at least 30% of bayonet apposition fractures ([126,139,146,166,167](#)), many surgeons treat this fracture primarily with pin fixation to avoid the problems that can occur with malunion ([Fig. 9-51](#)). Otherwise it is clear that patients treated with cast immobilization need to be monitored closely ([172](#)).

Fortunately, many angular malunions of the distal radius will remodel ([13,74,76,103,111,121,122](#) and [123,137,144,149](#)), probably because of asymmetric physal growth ([136,137](#)). The younger the patient, the less the deformity, and the closer the fracture is to the physis, the greater the potential for remodeling. It is unclear whether there is any capacity for rotational malunion remodeling ([124,150](#)). Angular and rotational malunion that does not remodel can lead to loss of motion. The degree and plane of loss of motion, as well as the individual affected, determine if this is functionally significant ([172](#)). Cadaveric studies indicate that residual malangulation of more than 20 degrees of the radius or ulna will lead to loss of forearm rotation ([140,158,159](#)). Less than 10 degrees of malangulation did not alter forearm rotation significantly in either study. Distal third malunion affected rotation less than middle or proximal third malunion. Radioulnar malunion affected forearm rotation more than volar-dorsal malunion. Malangulation may lead to a loss of rotation at a 1 : 2 degree ratio, whereas malrotation may lead to rotational loss at only a 1 : 1 degree loss ([86](#)). The functional loss associated with rotational motion loss is difficult to predict. This has led some clinicians to recommend no treatment ([110,111](#)), arguing that most of these fractures will remodel, and those that do not remodel will not cause a functional problem ([134](#)). However, a significant functional problem is present if shoulder motion cannot compensate for loss of supination.

I prefer to reduce these fractures as anatomically as possible and lessen the risk of malunion. No element of malrotation is accepted in the reduction. As indicated in the treatment sections, fractures at high risk of loss of reduction and malunion are treated with anatomic reduction and pin fixation. Fractures treated in a cast are followed closely and re-reduced for any loss of alignment of more than 10 degrees. Although loss of rotation can occur with anatomic reduction ([32,142,168](#)), it is less likely than with malunions.

### **Nonunion**

Nonunion of a closed radial or ulnar fracture is rare. In children, nonunion has been universally related to a pathologic condition of the bone or vascularity ([168](#)). Congenital pseudarthrosis or neurofibromatosis ([135](#)) ([Fig. 9-64](#)) should be suspected in a patient with a nonunion after a benign fracture. This occurs most often in an isolated ulnar fracture. The distal bone is often narrowed, sclerotic, and plastically deformed. These fractures rarely heal with immobilization. Vascularized fibular bone grafting usually is necessary for healing of a nonunion associated with neurofibromatosis or congenital pseudarthrosis. If the patient is very young, this may include a vascularized epiphyseal transfer to restore distal growth.



**FIGURE 9-64.** This 3-year-old presented to the emergency room with pain after an acute fall on his arm. The ulna is clearly pathologic with thinning and deformity prior to this injury. This represents neurofibromatosis.

Vascular impairment also can lead to nonunion. Distal radial nonunion has been reported in a child with an ipsilateral supracondylar fracture with brachial artery occlusion. Revascularization of the limb led to eventual union of the fracture. Nonunion also can occur with osteomyelitis and bone loss ([44](#)). Debridement of the necrotic bone and either traditional bone grafting, osteoclast lengthening, vascularized bone grafting, or creation of a single-bone forearm are surgical options. The best choice depends on the individual patient.

### **Cross-Union**

Cross-union is a rare complication of pediatric distal radial and ulnar fractures. It has been described after high-energy trauma and internal fixation ([164](#)). A single pin crossing both bones increases the risk of cross-union ([164](#)). Synostosis take-down can be performed, but the results usually are less than full restoration of motion. It is important to determine if there is an element of rotational malunion with the cross-union because this will affect the surgical outcome.

Soft tissue contraction across both bones also has been described ([116](#)). Contracture release resulted in restoration of forearm motion.

### **Refracture**

Fortunately, refractures after metaphyseal radial fractures are rare and much less common than after diaphyseal level radial and ulnar fractures. Most commonly, refracture occurs with premature discontinuation of immobilization or early return to potentially traumatic activities. It is advisable to protectively immobilize the wrist until full radiographic and clinical healing (usually 6 weeks) and to restrict activities until full motion and strength are regained (usually an additional 1–3 weeks). Individuals involved in high-risk activities, such as downhill ski racing, snowboarding, or skateboarding, should be protected with a splint during those activities for much longer.

### **Growth Disturbance**

Growth arrest of the distal radius after metaphyseal fracture is rare. Abram and Connolly each reported one patient with physal arrest after nondisplaced torus fractures. Two additional patients were reported in a series of 150 distal radial metaphyseal fractures ([119](#)). Wilkins and O'Brien ([168](#)) proposed that these arrests may be in fractures that extend from the metaphysis to the physis. This coincides with a Peterson type I fracture ([81,82](#)) ([Fig. 9-65](#)) and in essence is a physal fracture. These fractures should be monitored for growth arrest.

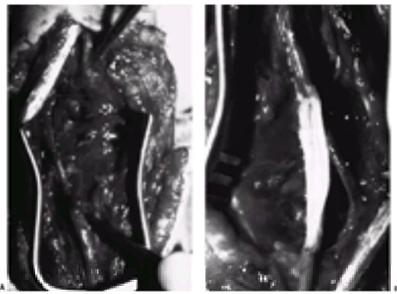


**FIGURE 9-65.** Peterson type I physeal injury. **A:** A comminuted distal metaphyseal fracture that extends to the physis (*open arrow*). **B:** Six weeks after the fracture, the callus also extends up to the physis (*open arrow*).

Both undergrowth and overgrowth of the distal radius after fracture has been described by DePablos ( 114). The average difference in growth was 3 mm, a range of —5 to +10 mm of growth disturbance compared with the contralateral radius. Maximal overgrowth occurred in the 9- to 12-year-old age group. As long as the patient is asymptomatic, under- or overgrowth is not a problem. If ulnocarpal impaction or DRUJ disruption occurs, then surgical rebalancing of the radius and ulna may be necessary.

### Neurovascular Injuries

Both the median and ulnar ( 106,163) nerves are less commonly injured in metaphyseal fractures than in physeal fractures. The mechanisms of neural injury in a metaphyseal fracture include direct contusion from the displaced fragment, traction ischemia from tenting of the nerve over the proximal fragment ( 144), entrapment of the nerve in the fracture site ( 40,170), rare laceration of the nerve ( Fig. 9-66), and the development of an acute compartment syndrome. If signs or symptoms of neuropathy are present, a prompt closed reduction should be performed. Extreme positions of immobilization should be avoided because this can lead to persistent traction or compression ischemia and increase the risk of compartment syndrome. If there is marked swelling, it is better to percutaneously pin the fracture than to apply a constrictive cast. If there is concern about compartment syndrome, the forearm and carpal canal pressures should be measured immediately. If pressures are markedly elevated, appropriate fasciotomies and compartment releases should be performed immediately. Finally, if the nerve was intact before reduction and is out after reduction, neural entrapment should be considered, and surgical exploration and decompression may be required. Fortunately, most median and ulnar nerve injuries recover after anatomic reduction of the fracture.



**FIGURE 9-66.** A grade III open fracture of the radius resulted in complete disruption of the ulnar nerve. Intraoperative photographs of the nerve deficit between the operative jeweler's forceps (**A**) and sural nerve grafting (**B**) after the wound was clean enough to allow for nerve reconstruction.

### Infection

Infection after distal radial fractures is rare and is associated with open fractures or surgical intervention. Fee et al. ( 117) described the development of gas gangrene in four children after minor puncture wounds or lacerations associated with distal radial fractures. Treatment involved only local cleansing of the wound in all four and wound closure in one. All four developed life-threatening clostridial infections. Three of the four required upper limb amputations, and the fourth underwent multiple soft tissue and bony procedures for coverage and treatment of osteomyelitis.

Infections related to surgical intervention also are rare. Superficial pin site infections can occur and should be treated with pin removal and antibiotics. Deep-space infections from percutaneous pinning of the radius has not been described, but it is only reasonable to think that it will occur at some point. All deep-space infections should be treated with appropriate surgical debridement, antibiotics, and wound management.

### PEDIATRIC GALEAZZI FRACTURES

Fractures of the distal radius associated with DRUJ disruption have been called Galeazzi fracture–dislocations. Although Sir Ashley Cooper is credited with the first description of this injury in 1824, Riccardo Galeazzi ( 177,188) ( Fig. 9-67) gave this fracture–dislocation its name with his 1934 report of 18 such injuries. In children, this injury may involve either disruption of the DRUJ ligaments or, more commonly, a distal ulnar physeal fracture. The former is called a true Galeazzi lesion and the latter is a Galeazzi equivalent lesion ( Fig. 9-68) ( 60,181,182,187,188).



**FIGURE 9-67.** Riccardo Galeazzi, 1866–1952. (Reprinted from *J Bone Joint Surg [Br]* 1953;35:680; with permission.)

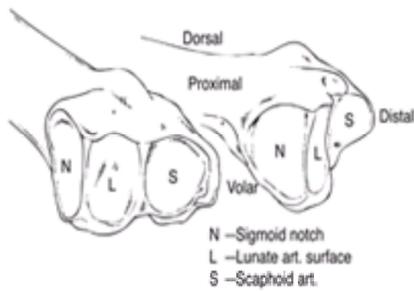


**FIGURE 9-68.** Galeazzi fracture–dislocation variant. Interposed periosteum can block reduction of the distal ulnar physis (*arrow*). This destabilizes the distal radial metaphyseal fracture. (Reprinted from Lanfried MJ, Stenlik M, Susi JG. Variant of Galeazzi fracture–dislocation in children. *J Pediatr Orthop* 1991;11:333; with permission.)

permission.)

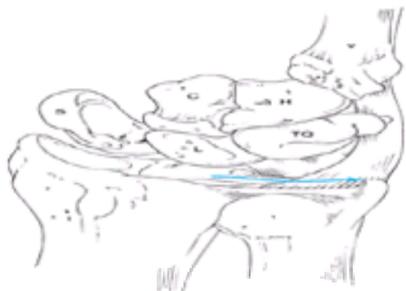
## Anatomy

The radius normally rotates around the relatively stationary ulna. The two bones of the forearm articulate at the proximal and distal radioulnar joints. In addition, proximally the radius and ulna articulate with the distal humerus and distally with the carpus. These articulations are responsible for forearm pronation and supination, as well as elbow and wrist flexion and extension. At the DRUJ, the concave sigmoid notch of the radius incompletely matches the convex, asymmetric, semicylindrical shape of the distal ulnar head (Fig. 9-69) (3). This allows some translation at the DRUJ with rotatory movements. The ligamentous structures are critical in stabilizing the radius as it rotates about the ulna.

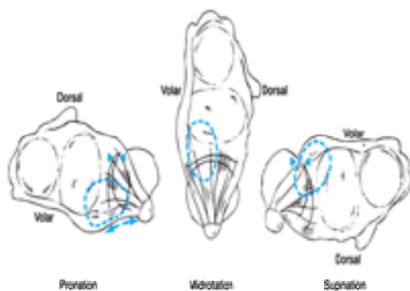


**FIGURE 9-69.** (Redrawn from Bowers WH. *Green's Operative Hand Surgery*. New York: Churchill–Livingstone, 1993:988.)

The DRUJ includes multiple soft tissue attachments, the most important of which is the TFCC. The TFCC includes the volar and dorsal ligamentous attachments of the distal ulna to the radial sigmoid notch, as well as the distal extension to the ulnar styloid, carpus, and base of the fifth metacarpal. The volar ulnocarpal ligaments (V ligament) from the ulna to the lunate and triquetrum are important ulnocarpal stabilizers (3,190,196). The central portion of the TFCC is the articular disk (Fig. 9-70). The interaction between the bony articulation and the soft tissue attachments accounts for stability of the DRUJ during pronation and supination. At the extremes of rotation, the joint is most stable. The compression loads between the radius and ulna are aided by the tensile loads of the TFCC to maintain stability throughout rotation (Fig. 9-71).

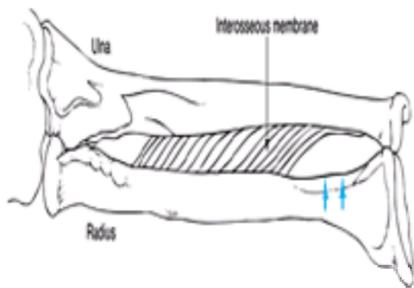


**FIGURE 9-70.** Diagrammatic drawing of the meniscal reflection and the prestyloid recess. The meniscal reflection runs from the dorsoulnar radius to the ulnovolar carpus. The arrow denotes access under the reflection to the tip of the styloid—the so-called prestyloid recess. (Redrawn from Bowers WH. *Green's Operative Hand Surgery*. New York: Churchill–Livingstone, 1993.)



**FIGURE 9-71.** Distal radioulnar joint stability in pronation (left) is dependent on (1) tension developed in the volar margin of the triangular fibrocartilage ( TFC, *small arrows*) and (2) compression between the contact areas of the radius and ulna (volar surface of ulnar articular head and dorsal margin of the sigmoid notch, *large arrows*). Disruption of the volar TFC would therefore allow dorsal displacement of the ulna in pronation. The reverse is true in supination, where disruption of the dorsal margin of the TFC would allow volar displacement of the ulna relative to the radius as this rotational extreme is reached. The dark area of the TFC emphasizes the portion of the TFC that is not supported by the ulnar dome. The dotted circle is the arc of load transmission (lunate to TFC) in that position. (Redrawn from Bowers WH. *Green's Operative Hand Surgery*. New York: Churchill–Livingstone, 1993.)

Throughout the mid-forearm, the interosseous ligament (Fig. 9-72) connects the radius to the ulna. It passes obliquely from the proximal radius to the distal ulna. However, the interosseous ligament is not present in the distal radius. Moore et al. found that injuries to the TFCC and interosseous ligament were responsible for progressive shortening of the radius with fracture in a cadaveric study. The soft tissue component to the injury is a major factor in the deformity and instability in a Galeazzi fracture–dislocation.



**FIGURE 9-72.** The attachment and the fibers of the interosseous membrane are such that there is no attachment to the distal radius. (Redrawn from Kraus B, Horne G. Galeazzi fractures. *J Trauma* 1985;25:1094; with permission.)

## Diagnosis

Galeazzi fracture–dislocations are relatively rare injuries in children. Walsh and McLaren ( 194) cited an incidence of 3% of pediatric distal radial fractures in their study. Most series of Galeazzi fracture contain a relatively small number of pediatric patients ( 81,182,183,194).

The mechanism of injury is axial loading in combination with extremes of forearm rotation ( 175,187,189,191). In adults, the mechanism of injury usually is an axially loading fall with hyperpronation. This results in a distal radius fracture with a dorsal ulnar dislocation. However, in children, both supination (apex volar) and pronation (apex dorsal) deforming forces have been described ( 181,182,194). The mechanism of injury is most obvious when the radial fracture is an incomplete fracture. With an apex volar (supination) radial fracture, the distal ulna is displaced volarly, whereas with an apex dorsal (pronation) radial fracture, the distal ulna is displaced dorsally (Fig. 9-68 and Fig. 9-69). This is evident both on clinical and radiographic examinations. In addition, the radius is foreshortened in a complete fracture, causing more radial deviation of the hand and wrist ( Fig. 9-70). A child with a Galeazzi injury has pain and limitation of forearm rotation and wrist flexion and extension. Neurovascular impairment is rare.

The radial fracture is evident on radiographs, and concurrent injuries to the ulna or DRUJ should be identified. A true lateral view is necessary to identify the direction of displacement, which is imperative to determine the method of reduction. Rarely are special radiographs, such as a CT scan, necessary.

## Classification

Galeazzi fracture–dislocations are most commonly described by direction of displacement of either the distal ulnar dislocation or the radial fracture. Letts ( 181,182) preferred to describe the direction of the ulna: volar or dorsal. Walsh and McLaren ( 194) classified pediatric Galeazzi injuries by the direction of displacement of the distal radial fracture. Dorsal displacement (apex volar) fractures were more common than volar displacement (apex dorsal) fractures in their series ( Fig. 9-73). Wilkins and O'Brien (168) modified the Walsh and McLaren method by classifying radial fractures as incomplete and complete fractures and ulnar injuries as true dislocations and physeal fractures (Table 9-4). DRUJ dislocations are called true Galeazzi lesions and distal ulnar physeal fractures are called Galeazzi equivalent lesions (60,178,181,182).



**FIGURE 9-73.** Walsh's classification. **A:** The most common pattern, in which there is dorsal displacement with supination of the distal radius ( open arrow). The distal ulna (black arrow) lies volar to the dorsally displaced distal radius. **B:** The least common pronation pattern. There is volar or anterior displacement of the distal radius (open arrow), and the distal ulna lies dorsal (black arrow). (Reprinted from Walsh HPJ, McLaren CANP. Galeazzi fractures in children. *J Bone Joint Surg [Br]* 1987;69:730–733; with permission.)

Type I: Dorsal (apex volar) displacement of distal radius
Radius fracture pattern
Greenstick
Complete
Distal ulna physis
Intact
Disrupted (equivalent)
Type II: Volar (apex dorsal) displacement of distal radius
Radius fracture pattern
Greenstick
Complete
Distal ulna physis
Intact
Disrupted

Reprinted from Walsh HPJ, McLaren CANP, Owen R. Galeazzi fractures in children. *J Bone Joint Surg [Br]* 1987;69:730–733.

**TABLE 9-4. CLASSIFICATION: GALEAZZI FRACTURES IN CHILDREN**

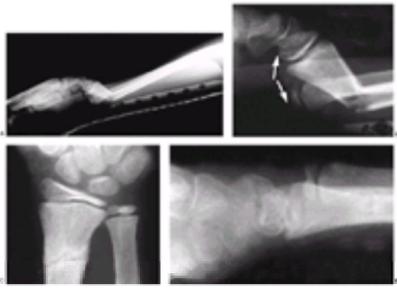
## Treatment

Pediatric Galeazzi fractures have a higher success rate with nonoperative treatment than similar injuries in adults ( 183,187). In adults, it is imperative to anatomically reduce and internally fix the distal radial fracture ( 180,183,184,185,187). Generally the DRUJ is reduced with reduction and fixation of the radius. In pediatric patients, the distal radial fracture often is a greenstick type that is stable after reduction and cast immobilization is sufficient ( 183,194). However, adolescents with complete fractures should be treated with internal fixation similar to adults.

### Closed Reduction

The method of reduction for greenstick radial fractures depends on the type of displacement. With apex volar dorsally displaced fractures of the radius, the rotatory deformity is supination. Pronating the radius and applying dorsal-to-volar reduction force should align the fracture and reduce the DRUJ ( Fig. 9-74). Similarly, if the incomplete radial fracture is an apex dorsal volar displaced fracture, the rotatory deformity is pronation ( Fig. 9-75). Supinating the forearm and applying volar-to-dorsal force should reduce the incomplete fracture of the radius and the DRUJ dislocation ( 168,181,182,193). In both these situations, portable fluoroscopy

can be used to evaluate the fracture–dislocation reduction and to test the stability of the distal ulna. If anatomically reduced and stable, a long arm cast is applied with appropriate rotation and three-point molds. The cast is left in place for 6 weeks to allow the soft tissue injuries to heal.



**FIGURE 9-74.** Supination-type Galeazzi fracture. **A:** View of the entire forearm of an 11-year-old boy with a Galeazzi fracture–dislocation. **B:** Close-up of the distal forearm shows that there has been disruption of the distal radioulnar joint ( *arrows*). The distal radial fragment is dorsally displaced (apex volar), making this a supination type of mechanism. Note that the distal ulna is volar to the distal radius. **C and D:** The fracture was reduced by pronating the distal fragment. Because the distal radius was partially intact by its greenstick nature, the length was easily maintained, reestablishing the congruity of the distal radioulnar joint. The patient was immobilized in supination for 6 weeks, after which full forearm rotation and function returned.



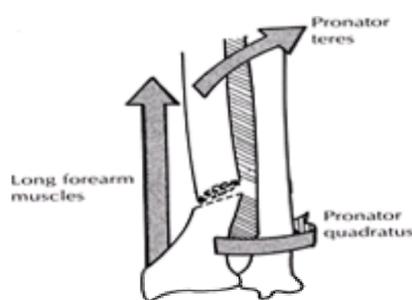
**FIGURE 9-75.** Pronation Galeazzi. This 8-year-old sustained a pronation Galeazzi fracture. **A:** The anteroposterior view shows some shortening of the distal radius (*arrow*) in relation to the distal ulna, which has a small greenstick component. **B:** The pronation component (*arrow*) is better appreciated on this lateral view. The distal ulna lies dorsal to the distal radius (*open arrow*).

In a Galeazzi equivalent injury with a radial fracture and an ulnar physal fracture, both bones should be reduced. Usually this can be accomplished with the same methods of reduction if the radial fracture is incomplete. The distal ulnar physis can remodel a nonanatomic reduction if there is sufficient growth remaining and the ulnar physis continues to grow normally. Unfortunately, the risk of ulnar growth arrest after a Galeazzi equivalent has been reported to be as high as 55% ( [52](#)).

Complete fractures of the distal radius have a higher rate of loss of reduction after closed treatment ( [168](#)). If not monitored closely and re-reduced if necessary, loss of reduction can lead to malunion with loss of motion and function. These injuries may be best treated with open reduction as in adults.

### Open Reduction

The indication for open reduction of the radial fracture is failure to obtain or maintain fracture reduction. This most often occurs with unstable complete fractures ( [Fig. 9-76](#)). Open reduction and internal fixation of the radius are performed via an anterior approach ( [Fig. 9-77](#)). Standard compression plating is preferred to intramedullary or cross-pinning techniques. Stable, anatomic reduction of the radius almost always leads to stable reduction of the DRUJ dislocation. A long arm cast is used for 6 weeks to allow fracture and soft tissue healing.

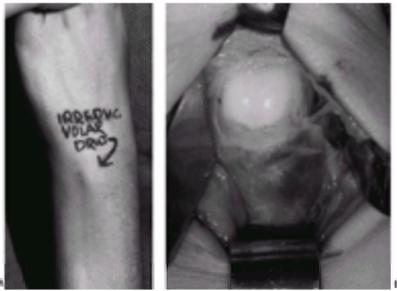


**FIGURE 9-76.** Fractures of the distal radius are angulated toward the ulna due to the pull of the long forearm muscles and the pronator quadratus. (Redrawn from Cruess RL. The management of forearm injuries. *Orthop Clin North Am* 1973;4:969–982; with permission.)



**FIGURE 9-77.** **A:** The patient with the pronation injury shown in [Fig. 9-69](#) had a closed reduction and attempted fixation with pins placed percutaneously across the fracture site. However, this was inadequate in maintaining the alignment and length of the fracture of the distal radius. **B:** The length of the radius and the distal radioulnar relationship were best reestablished after internal fixation of the distal radius with a plate placed on the volar surface. The true amount of shortening present on the original injury film (see [Fig. 9-69A](#)) is not really appreciated until the fracture of the distal radius is fully reduced. (Reprinted from Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:34; with permission.)

Occasionally, the DRUJ dislocation cannot be reduced ([Fig. 9-78](#)) because of interposed soft tissues, most commonly the periosteum, extensor tendons (extensor carpi ulnaris, extensor digiti quinti), TFCC, or other ligamentous structures ([173,174,179,182](#)). The easiest approach for open reduction of the DRUJ is an extended ulnar approach. Care should be taken to avoid injury to the ulnar sensory nerve. This approach allows for exposure both volarly and dorsally to extract the interposed soft tissues and repair the torn structures. Smooth pin fixation of the DRUJ can be used to maintain reduction and allow application of a loose-fitting cast. The pin is removed in the office at four weeks with continuation of the cast for 6 weeks.



**FIGURE 9-78.** An adolescent girl presented 4 weeks postinjury with a painful, stiff wrist. **A:** By examination, she was noted to have a volar distal radioulnar dislocation that was irreducible even under general anesthesia. **B:** At the time of surgery the distal ulna was noted to have buttonholed out of the capsule, and there was entrapped triangular fibrocartilage and periosteum in the joint.

Similarly, the ulnar physeal fracture can be irreducible in a Galeazzi equivalent injury. This also has been reported to be secondary to interposed periosteum ([60,187](#)), extensor tendons ([48,57,103](#)), or joint capsule ([176](#)). Open reduction must be executed with care to avoid further violating the physis.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

### Incomplete Fractures

Incomplete fractures of the distal radius with either a true dislocation of the DRUJ or an ulnar physeal fracture are treated with closed reduction and long arm cast immobilization. This can be performed in the emergency setting with conscious sedation or in the operating room with general anesthesia. Portable fluoroscopy is used. If the fracture is apex volar with dorsal displacement of the radius and volar dislocation of the DRUJ, then pronation and volar-to-dorsal force on the radial fracture is used for reduction. If the fracture is apex dorsal with volar displacement of the radius and dorsal dislocation of the DRUJ, then supination and dorsal-to-volar force is applied to the distal radius for reduction. The reduction and stability of the fracture and DRUJ dislocation are checked on dynamic fluoroscopy before long arm cast immobilization. If both are anatomically reduced and stable, the cast is used for 6 weeks to allow soft tissue and fracture healing. In a Galeazzi equivalent injury, there is potential for remodeling of the physeal fracture if sufficient growth remains. As long as the DRUJ is reduced, malalignment of less than 10 degrees can remodel in a young child. The risk of physeal growth arrest is high with this physeal injury, and operative exposure may increase the of growth impairment. If the fracture is severely malaligned, the DRUJ cannot be reduced, or the patient is older and remodeling is unlikely, open reduction and smooth pin fixation are indicated ([168](#)).

### Complete Fractures

Open reduction and internal fixation with an anterior plate and screws are used for complete Galeazzi fractures of the radius. The DRUJ usually reduces anatomically and is stable with reduction and fixation of the radius. The patient is immobilized in a long arm cast for 4 weeks and a short arm cast for 2 more weeks. Return to unrestricted activities and sports depends on restoration of full motion and strength.

### Irreducible Dislocations

Irreducible dislocations are treated with open reduction. Extensile exposure is necessary to define the pathologic anatomy and carefully reduce the DRUJ. The interposed soft tissues are extracted and repaired. Depending on the stability of the reduction and repair, a supplemental smooth pin may be used across the DRUJ for 4 weeks to maintain the joint reduction. This is particularly true if the patient presents late.

### Complications

#### Malunion and DRUJ Subluxation

Malunion of the radius can lead to subluxation of the DRUJ, limited forearm rotation, and pain, usually secondary to persistent shortening and malrotation of the radial fracture. Most often this occurs when complete fractures are treated with closed reduction and there is failure to either obtain or maintain reduction of the radial fracture. The ulna remains subluxed and heals with an incongruent joint. Treatment for this requires proper recognition and corrective osteotomy. If physical examination is not definitive for diagnosis, then a CT scan in pronation, neutral rotation, or supination may be helpful. An MRI scan or wrist arthroscopy will aid in the diagnosis and management of associated ligamentous, chondral, or TFCC injuries that will benefit from debridement or repair. It is important to understand that if the DRUJ subluxation is caused by a radial malunion, a soft tissue reconstruction of the DRUJ alone will fail ([3](#)).

#### Ulnar Physeal Arrest

Golz and Ogden ([52](#)) cited a 55% incidence of ulnar physeal arrest with Galeazzi equivalent fractures. If the patient is young enough, this ulnar growth arrest in the presence of ongoing radial growth will lead to deformity. Initially there will be ulnar shortening. Over time, the foreshortened ulna can act as a tether, causing asymmetric growth of the radius. There will be increased radial articular inclination on the AP radiograph and subluxation of the DRUJ. Operative choices include ulnar lengthening, radial closing wedge osteotomy, radial epiphysiodesis, and a combination of the above procedures that is appropriate for the individual patient's age, deformity, and disability.

#### Nerve Injury

Injuries to the ulnar nerve ([183](#)) and anterior interosseous nerve ([192,195](#)) have been described with Galeazzi fracture–dislocations. These injuries have had spontaneous recovery. Moore et al. ([186](#)) described an 8% incidence of injury to the radial nerve with operative exposure of the radius for internal fixation in their series. Careful surgical exposure, dissection, and retraction can decrease this risk.

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## CHAPTER REFERENCES

1. Albanese SA, Palmer AK, Kerr DR, et al. Wrist pain and distal growth plate closure of the radius in gymnasts. *J Pediatr Orthop* 1987;9:23–28.
2. Bailey DA, Wedge JH, McCulloch RG, et al. Epidemiology of fractures of the distal end of the radius in children associated with growth. *J Bone Joint Surg [Am]* 1989;71:1225–1231.
3. Bowers W. The distal radioulnar joint. In: Green D, Hotchkiss R, Pederson W, eds. *Green's operative hand surgery*, 4th ed. New York: Churchill-Livingstone, 1999:986–1032.
4. Caine D, Roy S, Singer KM, et al. Stress changes of the distal radial growth plate. a radiographic survey and review of the literature. *Am J Sports Med* 1992;20:290–298.
5. Cheng JC, Shen WY. Limb fracture pattern in different pediatric age groups: a study of 3,350 children. *J Orthop Trauma* 1993;7:15.
6. De Smet L, Claessens A, Lefevre J, et al. Gymnast wrist: an epidemiologic survey of ulnar variance and stress changes of the radial physis in elite female gymnasts. *Am J Sports Med* 1994;22:846–850.
7. Dobyys J, Gabel G. Gymnast's wrist. *Hand Clin* 1990;6:493.
8. Epner R, Bowers W, Guilford W. Ulnar variance: the effect of wrist positioning and roentgen filming technique. *J Hand Surg [Am]* 1982;7:298–305.
9. Ekenstrom F. The anatomy of the distal radioulnar joint. *Clin Orthop Rel Res* 1992;275:14–18.
10. Fernandez D, Palmer A. Fractures of the distal radius. In: Green D, Hotchkiss R, Pederson W, eds. *Green's operative hand surgery*, 4th ed. New York: Churchill-Livingstone, 1999:929–985.
11. Friedman S, Palmer A, Short W, et al. Changes in ulnar variance with grip. *J Hand Surg [Am]* 1993;18:713–716.
12. Gam S, Rohmann C, Silverman F. Radiographic standards for post-natal ossification and tooth calcification. *Med Radiogr Photogr* 1967;43:45–66.
13. Gandhi RK, Wilson P, Brown JM, et al. Spontaneous correction of deformity following fractures of the forearm in children. *Br J Surg* 1962;50:5–10.
14. Gelberman R, Salamon P, Jurist J, et al. Ulnar variance in Kienbock's disease. *J Bone Joint Surg [Am]* 1975;57:674–676.
15. Gerber SD, Griffing PP, Simmons BP. Breakdancer's wrist. *J Pediatr Orthop* 1986;6:98–99.
16. Greulich W, Pyle S. *Radiographic atlas of skeletal development of the hand and wrist*, 2nd ed. Stanford, CA: Stanford University Press, 1959.
17. Hughston JC. Fractures of the forearm. *J Bone Joint Surg [Am]* 1962;44:1664–1667.
18. Johnson PG, Szabo RM. Angle measurements of the distal radius: a cadaver study. *Skel Radio*. 1993;22:243–246.
19. Landin LA. Fracture patterns in children: analysis of 8682 fractures with special reference to incidence, etiology and secular changes in a Swedish urban population, 1950–1979. *Acta Chir Scand Suppl* 1983;202.
20. Lawton L. In: Letts M, ed. *Fractures of the distal radius and ulna in management of pediatric fractures*. New York: Churchill-Livingstone, 1994:345–368.
21. Meeusen R, Borms J. Gymnastic injuries. *Sports Med* 1992;13:337.
22. Mino D, Palmer A, Levinshon E. The role of radiology and computerized tomography in the diagnosis of incongruity of the distal radioulnar joint. *J Bone and Joint Surg [Am]* 1985;67:247–252.
23. Mizuta T, Benson WM, Foster BK, et al. Statistical analysis of the incidence of physeal injuries. *J Pediatr Orthop* 1987;7:518.
24. Morton R. A radiographic survey of 170 clinically diagnosed as "Colles' fracture." *Lancet* 1907;1:731–732.
25. Palmer A, Werner F. The triangular fibrocartilage complex of the wrist. Anatomy and function. *J Hand Surg* 1981;6:153–161.
26. Reed MH. Fractures and dislocations of the extremities in children. *J Trauma* 1977;17:35.
27. Roy S, Caine D, Singer KM. Stress changes of the distal radial epiphysis in young gymnasts. A report of 21 cases and a review of the literature. *Am J Sports Med* 1985;13:301–308.
28. Short W, Palmer A, Werner F, et al. A biomechanical study of the distal radius. *J Hand Surg [Am]* 1897;12:529–534.
29. Steyers C, Blair W. Measuring ulnar variance: a comparison of techniques. *J Hand Surg [Am]* 1989;14:607–612.
30. Stuart H, Pyle S, Comoni J, et al. Onsets, completions, and spans of ossification in the 29 growth centers of and wrist. *Pediatrics* 1962;29:237.
31. Waters P, Hipp J, Taylor B, et al. Do radiographic classification systems predict distal radius fracture instability? Presented at the Association of Bone and Joint Annual Meeting, Phoenix: April 1997.
32. Thomas EM, Tuson KWR, Browne PSH. Fractures of the radius and ulna in children. *Injury* 1975;7:120–124.
33. Tolat AR, Sanderson PL, Desmet L, et al. The gymnast's wrist: acquired positive ulnar variance following chronic epiphyseal injury. *J Bone Joint Surg [Br]* 1992;17:678–681.
34. Van der Linden W, Ericson R. Colles' fracture: how should its displacement be measured and how should it be immobilized? *J Bone Joint Surg [Am]* 1981;63:1285–1288.
35. Waters PM, Mintzer CM, Hipp JA, et al. Noninvasive measurement of distal radius instability. *J Hand Surg [Am]* 1997;22:572–579.
36. Worlock P, Stower M. Fracture patterns in Nottingham children. *J Pediatr Orthop* 1986;6:656–660.

## Physeal Injuries of the Distal Radius and Ulna

37. Aitken AP. Further observations on the fractured distal radial epiphysis. *J Bone Joint Surg* 1935;17:922–927.
38. Aitken AP. The end results of the fractured distal radial epiphysis. *J Bone Joint Surg* 1935;17:302–308.
39. Arima J, Uchida Y, Miura H, et al. Osteochondral fracture in the distal end of the radius. *J Hand Surg [Am]* 1993;18:489–491.
40. Armstrong P, Joughlin J, Clarke H. In: Green N, Swiontkowski M, eds. *Pediatric fractures of the forearm, wrist and hand in skeletal trauma in children*, 2nd ed. Philadelphia: WB Saunders, 1994:161–257.
41. Bell MJ, Hill RJ, McMurtry RY. Ulnar impingement syndrome. *J Bone Joint Surg [Br]* 1985;67:126–129.
42. Boyden EM, Peterson HA. Partial premature closure of the distal radial physis associated with Kirschner wire fixation. *Orthopaedics* 14:585–588, 1991.
43. Bruns [cited by Cotton FS]. *Dislocations in joint fractures*, 2nd ed. Philadelphia: WB Saunders, 1924:370.
44. Burgess RC, Watson HK. Hypertrophic ulnar styloid nonunions. *Clin Orthop Rel Res* 1988;228:215–217.
45. Caine D, Roy S, Singer KM, Broekhoff J. Stress changes of the distal radial growth plate. A radiographic survey and review of the literature. *Am J Sports Med* 1992;20:290–298.
46. Crawford AH. Pitfalls and complications of fractures of the distal radius and ulna in childhood. *Hand Clin* 1988;4:403–413.
47. Doi K, Hattori Y, Otsuka K, et al. Intra-articular fractures of the distal aspect of the radius: arthroscopically assisted reduction compared with open reduction and internal fixation. *J Bone Joint Surg [Am]* 1999;81:1093–1110.
48. Evans DL, Stauber M, Frykman GK. Irreducible epiphyseal plate fracture of the distal ulna due to interposition of the extensor carpi ulnaris tendon. A case report. *Clin Orthop Rel Res* 1990;251:162–165.
49. Fernandez D. Correction of post-traumatic wrist deformity by osteotomy, bone grafting and internal fixation. *J Bone Joint Surg [Am]* 1988;64:1538–1551.
50. Geissler W, Freeland A, Weiss A-P, et al. Techniques of wrist arthroscopy. Instructional course lecture. *J Bone Joint Surg [Am]* 1999;81:1184–1197.
51. Gelberman RJ. Acute carpal tunnel syndrome. In: Gelber AN ed. *Operative Nerve Repair and Reconstruction*. Philadelphia. JB Lippincott. 1991:937–948.
52. Golz RJ, Grogan DP, Greene TL, et al. Distal ulnar physeal injury. *J Pediatr Orthop* 1991;11:318–326.
53. Hafner R, Poznanski AK, Donovan JM. Ulnar variance in children = mstandard measurements for evaluation of ulnar shortening in juvenile rheumatoid arthritis, hereditary multiple exostosis and other bone or joint disorders in childhood. *Skel Radio*. 1989;18:513–516.
54. Hernandez J Jr, Peterson HA. Fracture of the distal radial physis complicated by compartment syndrome and premature physeal closure. *J Pediatr Orthop* 1986;6:627–630.
55. Horii E, Tamura Y, Nakamura R, et al. Premature closure of the distal radial physis. *J Hand Surg [Br]* 1993;18:11–16.
56. Hulten O. Uber Anatomische Variationen Der Hand-Gelenkknochen. *Acta Radiol* 1928;9:155–168.
57. Karlsson J, Appelqvist R. Irreducible fracture of the wrist in a child. *Acta Chir Scand* 1987;58:280–281.
58. Kasser JR. Forearm fractures. In: MacEwen GD, et al., eds. *A practical approach to assessment and treatment*. Baltimore: Williams & Wilkins, 1993:165–190.
59. Knirk J, Jupiter J. Late results of intra-articular fractures of the distal end of the radius in young adults. *J Bone Joint Surg [Am]* 1985;68:647–659.
60. Landfried MJ, Stenlik M, Susi JG. Variant of Galeazzi fracture–dislocation in children. *J Pediatr Orthop* 1991;11:332–335.
61. Landin LA. Fracture patterns in children: analysis of 8682 fractures with special reference to incidence, etiology and secular changes in a Swedish urban population, 1950–1979. *Acta Chir Scand Suppl* 1983;202.
62. Langenberg R. Fracture of the ulnar styloid process. Effect on wrist function in the presence of distal radius fracture. *Zentralbl Chir* 1989;114:1006–1011.
63. Langenskiold A. Surgical treatment of partial closure of the growth plate. *J Pediatr Orthop* 1981;1:3–11.
64. Langenskiold A, Osterman K. Surgical treatment of partial closure of the epiphyseal plate. *Reconstr Surg Traumatol* 1979;17:48–64.
65. Lee BS, Esterhai JL, Das M. Fracture of the distal radial epiphysis. Characteristics and surgical treatment of premature, post-traumatic epiphyseal closure. *Clin Orthop* 1984;185:90–96.
66. Lenoble E, Dumontier C, Goultallier, et al. Fracture of the distal radius. A prospective comparison between trans-styloid and Kapandji fixations. *J Bone Joint Surg [Br]* 1995;77:562–567.
67. Lesko PD, Georgis T, Slabaugh P. Irreducible Salter-Harris II fracture of the distal radial epiphysis. *J Pediatr Orthop* 1987;7:719–721.
68. Maffulli N. Painful hypertrophic nonunion of the ulnar styloid. *J Hand Surg [Br]* 1990;15:355–357.
69. Mandelbaum BR, Bartiolozzi AR, Davis CA, et al. Wrist pain syndrome in the gymnast: pathogenesis, diagnostic, and therapeutic considerations. *Am J Sports Med* 1989;17:305–317.
70. Manoli A. Irreducible fracture–separation of the distal radial epiphysis. Report of a case. *J Bone Joint Surg [Am]* 1982;64:1095–1096.
71. Mizuta T, Benson WM, Foster BK, et al. Statistical analysis of the incidence of physeal injuries. *J Pediatr Orthop* 1987;7:518–523.
72. Waters P, Bae D, Montgomery K. The management of post-traumatic distal radius growth arrest. Presented at PUSNA Speciality Day, AAOS Annual Meeting, San Francisco, March 3, 2001.
73. Nelson DA, Buchanan JR, Harrison CS. Distal ulnar growth arrest. *J Hand Surg [Am]* 1984;9:164–171.
74. Ogden JA. *Skeletal injury in the child*. Philadelphia: WB Saunders, 1990.
75. Ogden JA, Beall JK, Conlogue GJ, et al. Radiology of postnatal skeletal development. IV. Distal radius and ulna. *Skel Radio*. 1981;6:255–266.
76. Onne L, Sandblom PH. Late results in fractures of the forearm in children. *Acta Chir Scand* 1949;98:549–567.
77. Palmer AK, Glisson RR, Werner FW. Relationship between ulnar variance and triangular fibrocartilage complex thickness. *J Hand Surg [Am]* 1984;9:681–683.
78. Peinado A. Distal radial epiphyseal displacement after impaired distal ulnar growth. *J Bone Joint Surg [Am]* 1979;61:88–92.
79. Peterson CA, Peterson HA. Analysis of the incidence of injuries to the epiphyseal growth plate. *J Trauma* 1972;12:275–281.
80. Peterson HA. Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258.

81. Peterson HA. Physeal fractures: part 3, classification. *J Pediatr Orthop* 1994;14:439–448.
82. Peterson HA. Physeal fractures: part 2, two previously unclassified types. *J Pediatr Orthop* 1994;14:431–438.
83. Peterson H. Triplane fracture of the distal radius: case report. *J Pediatr Orthop* 1996;16:192–194.
84. Peterson HA, Madhok R, Benson JT, et al. Physeal fractures: Part I, epidemiology in Olmstead County, Minnesota, 1979 & 1988. *J Pediatr Orthop* 1994;41:423–430.
85. Poland J. *Traumatic separation of the epiphysis*. London: Smith, Elder & Co., 1898.
86. Rang M. *Children's fractures*. Philadelphia: JB Lippincott, 1983.
87. Ruggles DL, Peterson HA, Scott SG. Radial growth plate injury in a female gymnast. *Med Sci Sports Exerc* 1991;23:393–396.
88. Salter RB, Harris WR. Injuries involving the epiphyseal plate. *J Bone Joint Surg [Am]* 1963;45:587–622.
89. Santoro V, Mara J. Compartment syndrome complicating Salter-Harris type II distal radius fracture. *Clin Orthop* 1988;233:226–229.
90. Sasaki Y, Sugioka Y. The pronator quadratus sign: its classification and diagnostic usefulness for injury and inflammation of the wrist. *J Hand Surg [Br]* 1989;14:80–83.
91. Stansberry SD, Seischuk LE, Swischuk JL, et al. Significance of ulnar styloid fractures in childhood. *Pediatr Emerg Care* 1990;6:99–103.
92. Stein A, Katz S. Stabilization of comminuted fractures of the distal inch of the radius. Percutaneous pinning. *Clin Orthop Rel Res* 1975;108:174–181.
93. Terry C, Waters P. Triangular fibrocartilage injuries in pediatric and adolescent patients. *J Hand Surg [Am]* 1998;23:626.
94. Valverde J, Albinana J, Certucha J. Early posttraumatic physeal arrest in distal radius after a compression injury. *J Pediatr Orthop* 1996;5:57–60.
95. Waters PM, Koletis GJ, Schwend R. Acute median neuropathy following physeal fractures of the distal radius. *J Pediatr Orthop* 1994;14:13–177.
96. Watson K, Talesnik J. Midcarpal instability caused by malunited fracture of the distal radius. *J Hand Surg [Am]* 1984:350.
97. Weiker GG. Hand and wrist problems in the gymnast. *Clin Sports Med* 1992;11:189–202.
98. Young-Hing K, Wedge JH, Bowen CVA. Chronic injury to the distal ulnar and radial growth plates in an adolescent gymnast. A case report. *J Bone Joint Surg [Am]* 1988;70:1087–1089.
99. Young TB. Irreducible displacement of the distal radial epiphysis complicating a fracture of the lower radius and ulna. *Injury* 1984;16:166–168.
100. Zammit-Maempel I, Bisset RAL, Morris J, et al. The value of soft-tissue signs in wrist trauma. *Clin Radio* 1988;39:664–668.

## Fractures of the Metaphysis

101. Banas MP, Dalldorf PG, Marquardt JD. Skateboard and in-line skate fractures: a report of one summer's experience. *J Orthop Trauma* 1992;6:301–305.
102. Biyani A. Ipsilateral Monteggia equivalent injury and distal radial and ulnar fracture in a child. *J Orthop Trauma* 1994;8:431–433.
103. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955.
104. Chess DG, Hyndman JC, Leahey JL. Short-arm plaster for paediatric distal forearm fractures. *J Bone Joint Surg [Br]* 1987;69:506.
105. Chess DG, Hyndman JC, Leahey JL, et al. Short-arm plaster cast for distal pediatric forearm fractures. *J Pediatr Orthop* 1994;14:211–213.
106. Clarke AC, Spencer RF. Ulnar nerve palsy following fractures of the distal radius: clinical and anatomical studies. *J Hand Surg [Br]* 1991;16:438–440.
107. Compson JP. Transcarpal injuries associated with distal radial fractures in children: a series of three cases. *J Hand Surg [Br]* 1992;17:311–314.
108. Cooper RR. Management of common forearm fractures in children. *J Iowa Med Soc* 1964;54:689–698.
109. Creasman C, Zaleske DJ, Ehrlich MG. Analyzing forearm fractures in children. The more subtle signs of impending problems. *Clin Orthop* 1984;188:40–53.
110. Daruwalla JS. A study of radioulnar movements following fractures of the forearm in children. *Clin Orthop* 1979;139:114–120.
111. Davis DR, Green DP. Forearm fractures in children. Pitfalls and complications. *Clin Orthop* 1976;120:172–184.
112. De Courtivron B. Spontaneous correction of the distal forearm fractures in children. Scientific presentation. European Pediatric Orthopaedic Society Annual Meeting, Brussels, Belgium, April 1995.
113. Deffer PA, Schonholtz G, Litchman HM. Displaced forearm fractures in children. *Bull Hosp Joint Dis* 1963;24:42–47.
114. DePablos J, Franzreb M, Barrios C. Longitudinal growth pattern of the radius after forearm fractures conservatively treated in children. *J Pediatr Orthop* 1994;14:492–495.
115. Evans EM. Fractures of the radius and ulna. *J Bone Joint Surg [Br]* 1951;33:548–561.
116. Fatti JF, Mosher JF. An unusual complication of fracture of both bones of the forearm in a child. *J Bone Joint Surg [Am]* 1986;68:451–453.
117. Fee NF, Dobranski A, Bisla RS. Gas gangrene complicating open forearm fractures. Report of five cases. *J Bone Joint Surg [Am]* 1977;59:135–138.
118. Fernandez DL. Conservative treatment of forearm fractures in children. In: Chapchal G, ed. *Fractures in children*. New York: Thieme-Stratton, 1981.
119. Fodden DI. A study of wrist injuries in children: the incidence of various injuries and of premature closure of the distal radial growth plate. *Arch Emerg Med* 1992;9:9–13.
120. Fowles JC, Kassab MT. Displaced supracondylar fractures of the elbow in children: a report on the fixation of extension and flexion fractures by two lateral percutaneous pins. *J Bone Joint Surg [Br]* 1974;56:490–500.
121. Friberg KSI. Remodeling after distal forearm fractures in children. The effect of residual angulation on the spatial orientation of the epiphyseal plates. *Acta Chir Scand* 1979;50:537–546.
122. Friberg KSI. Remodeling after distal forearm fractures in children. Correction of residual angulation in fractures of the radius. *Acta Chir Scand* 1979;50:741–749.
123. Friberg KSI. Remodeling after distal forearm fractures in children. The final orientation of the distal and proximal epiphyseal plates of the radius. *Acta Chir Scand* 1979;50:731–739.
124. Fuller DJ, McCullough CJ. Malunited fractures of the forearm in children. *J Bone Joint Surg [Br]* 1982;64:364–367.
125. Guero S. Fractures and epiphyseal fracture separation of the distal bones of the forearm in children. In: Saffar P, Cooney WP, eds. *Fractures of the distal radius*. Philadelphia: JB Lippincott, 1995:280.
126. Gibbons CL, Woods DA, Pailthorpe C, et al. The management of isolated distal radius fractures in children. *J Pediatr Orthop* 1994;14:207–210.
127. Glatzner RL, Perlman RD, Michaels G, et al. Fractures of both bones of the distal forearm in children. *Bull Hosp Joint Dis* 1967;28:14–25.
128. Greene WB, Anderson WJ. Simultaneous fracture of the scaphoid and radius in a child. *J Pediatr Orthop* 1982;2:191–194.
129. Grundy M. Fractures of the carpal scaphoid in children. A series of eight cases. *Br J Surg* 1969;56:523–524.
130. Gupta RP, Danielsson LG. Dorsally angulated solitary metaphyseal greenstick fractures in the distal radius. *J Pediatr Orthop* 1990;10:90–92.
131. Hogstrom H, Nilsson BE, Willner S. Correction with growth following diaphyseal forearm fracture. *Acta Chir Scand* 1976;47:229–303.
132. Holmes JR, Luis DS. Entrapment of pronator quadratus in pediatric distal-radius fractures: recognition and treatment. *J Pediatr Orthop* 1994;14:498–500.
133. Hove LM. Simultaneous scaphoid and distal radial fractures. *J Hand Surg [Br]* 1993;18:11–16.
134. Johari A, Sinha M. Remodeling of forearm fractures in children. *J Pediatr Orthop [Br]* 1999;8:84–87.
135. Kameyama O, Ogawa R. Case report. Pseudarthrosis of the radius associated with neurofibromatosis: report of a case and review of the literature. *J Pediatr Orthop* 1990;10:128–131.
136. Karaharju EO, Ryoppy SA, Makinen RJ. Remodeling by asymmetrical epiphyseal growth. *J Bone Joint Surg [Br]* 1976;58:122–126.
137. Larsen E, Vittas D, Torp-Pedersen S. Remodeling of angulated distal forearm fracture in children. *Clin Orthop* 1988;237:190–195.
138. Light TR, Ogden DA, Ogden JA. The anatomy of metaphyseal torus fracture. *Clin Orthop* 1984;188:103–111.
139. Mani GV, Hui PW, Cheng JC. Translation of the radius as a predictor of outcome in distal radial fractures of children. *J Bone Joint Surg* 1993;75:808–811.
140. Matthews LS, Kaufner H, Gaver DF, et al. The effect of supination-pronation of angular malalignment of fractures of both bones of the forearm. *J Bone Joint Surg [Am]* 1982;64:14–17.
141. McLaughlin HL. *Trauma*. Philadelphia: WB Saunders, 1959.
142. Nilsson BE, Obrant K. The range of motion following fracture of the shaft of the forearm in children. *Acta Chir Scand* 1977;48:600–602.
143. Papavasiliou V, Nenopoulos S. Ipsilateral injuries of the elbow and forearm in children. *J Pediatr Orthop* 1986;6:58–60.
144. Perona PG, Light TR. A case report and review of the literature. Remodeling of the skeletally immature distal radius. *J Orthop Trauma* 1990;4:356–361.
145. Pollen AG. *Fractures and dislocations in children*. Baltimore: Williams & Wilkins, 1973.
146. Proctor MT, Moore DJ, Paterson JMH. Redisplacement after manipulation of distal radial fractures in children. *J Bone Joint Surg [Br]* 1993;75:453–454.
147. Reis M, Molena M, Chambers H, et al. The floating forearm: supracondylar fractures of the humerus associated with ipsilateral forearm fractures in children. 1996 (submitted for publication).
148. Roberts JA. Angulation of the radius in children's fractures. *J Bone Joint Surg [Br]* 1986;68:751–754.
149. Roy DR. Completely displaced distal radius fractures with intact ulnas in children. *Orthopaedics* 1989;12:1089–1092.
150. Ryoppy S, Karaharju EO. Alteration of epiphyseal growth by an experimentally produced angular deformity. *Acta Chir Scand* 1974;45:490–498.
151. Schranz PJ, Fagg PS. Undisplaced fractures of the distal third of the radius in children: an innocent fracture? *Injury* 1992;23:165–167.
152. Schuind F, Cooney WP III, Burny F, et al. Small external fixation devices for the hand and wrist. *Clin Orthop Rel Res* 1993;293:77–82.
153. Sharrard WJW. *Paediatric orthopaedics and fractures*. Oxford: Blackwell Scientific Publications, 1971.
154. Skillern PG Jr. Complete fracture of the lower third of the radius in childhood with greenstick fracture of the ulna. *Ann Surg* 1915;61:209–225.
155. Stanitski CL, Micheli LS. Simultaneous ipsilateral fractures of the arm and forearm in children. *Clin Orthop* 1980;153:218–221.
156. Stockley I, Harvey IA, Getty CJM. Acute volar compartment syndrome of the forearm secondary to fractures of the distal radius. *Injury* 1986;18:101–104.
157. Stuhmer KG. Fractures of the distal forearm. In: Weber BG, Bruner C, Freuler F, eds. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980:203–217.
158. Tachdjian MO. *Pediatric orthopaedics*. Philadelphia: WB Saunders, 1990.
159. Tarr RR, Garfinkel AI, Sarmiento A. The effects of angular and rotational deformities of both bones of the forearm. An *in vitro* study. *J Bone Joint Surg [Am]* 1984;66:65–70.
160. Templeton PA, Graham HK. The floating elbow in children. *J Bone Joint Surg [Br]* 1995;77:791–796.
161. Trumble EE, Benirschke SK, Vedder NB. Ipsilateral fractures of the scaphoid and radius. *J Hand Surg [Am]* 1993;18:8–14.
162. Vahvanen V, Westerlund M. Fracture of the carpal scaphoid in children. *Acta Orthop Scand* 1980;51:909–913.
163. Vance RM, Gelberman RH. Acute ulnar neuropathy with fractures at the wrist. *J Bone Joint Surg [Am]* 1978;60:962–965.
164. Vince KG, Miller JE. Cross-union complicating fracture of the forearm, part ii: children. *J Bone Joint Surg [Am]* 1987;69:654–660.
165. Voto SJ, Weiner DS, Leighley B. Redisplacement after closed reduction of forearm fractures in children. *J Pediatr Orthop* 1990;10:79–84.
166. Waters P, Miller B, Taylor B, et al. Prospective study of displaced radius fractures in adolescents treated with casting vs. percutaneous pinning. Presented at AAOS Annual Meeting, 2000.
167. Widmann R, Waters P, Reeves S. Complications of closed treatment of distal radius fractures in children. Presented at PUSNA Annual Meeting, Miami, May 1995.
168. Wilkins K, O'Brien E. Distal radius and ulna fractures. In: Rockwood and Green.

169. Wilkins KE. Operative management of upper extremity fractures in children. Rosemont, Illinois. American Academy of Orthopaedic Surgeons, 1994.
170. Wolfe JS, Eyring EJ. Median-nerve entrapment within a greenstick fracture. A case report. *J Bone Joint Surg [Am]* 1974;56:1270-1272.
171. Woodbury DF, Fischer B. An overriding radius fracture in a child with an intact ulna: management considerations. *Orthopaedics* 1985;8:763-765.
172. Younger A, Treadwell S, McKenzie W, et al. Accurate prediction of outcome after pediatric forearm fractures. *J Pediatr Orthop* 1994;14: 200-206.

### Galeazzi Fractures in Children

173. Biyani A, Bhan S. Dual extensor tendon entrapment in Galeazzi fracture-dislocation: a case report. *J Trauma* 1989;29:1295-1297.
174. Cetti MNE. An unusual cause of blocked reduction of the Galeazzi injury. *Injury* 1977;9:59-61.
175. Dameron TB. Traumatic dislocation of the distal radio-ulnar joint. *Clin Orthop* 1972;83:55-63.
176. Engber WD, Keene JS. Irreducible fracture-separation of the distal ulnar epiphysis. Report of a case. *J Bone Joint Surg [Am]* 1985;67:1130-1132.
177. Galeazzi R. Di una particolare sindrome, traumatica delle scheletro dell'avambraccio. *Attie Mem Soc Lombardi Chir* 1934;2:12.
178. Homans J, Smith JA. Fracture of the lower end of the radius associated with fracture or dislocation of the lower end of the ulna. *Boston Med Surg J* 1922;187:401-407.
179. Itoh Y, Horiuchi Y, Takahashi M, et al. Extensor tendon involvement in Smith's and Galeazzi's fractures. *J Hand Surg [Am]* 1987;12:535-540.
180. Kraus B, Horne G. Galeazzi fractures. *J Trauma* 1985;25:1093-1095.
181. Letts M, Rowhani N. Galeazzi-equivalent injuries of the wrist in children. *J Bone Joint Surg* 1993;13:561-566.
182. Letts RM. Monteggia and Galeazzi fractures. In: Letts RM, ed. *Management of pediatric fractures*. New York: Churchill Livingstone, 1994:313-321.
183. Mikic Z. Galeazzi fracture-dislocations. *J Bone Joint Surg [Am]* 1975;57:1071-1080.
184. Mohan K, Gupta AK, Sharma J, et al. Internal fixation in 50 cases of Galeazzi fracture. *Acta Orthop Scand* 1988;59:318-320.
185. Moore TM, Klein JP, Patzakis MJ, et al. Results of compression-plating of closed Galeazzi fractures. *J Bone Joint Surg [Am]* 1985;67:1015-1021.
186. Moore TM, Lester DK, Sarmiento A. The stabilizing of soft-tissue constraints in artificial Galeazzi fractures. *Clin Orthop Rel Res* 1985;194:189-194.
187. Reckling FW, Cordell LD. Unstable fracture-dislocations of the forearm. The Monteggia and Galeazzi lesions. *Arch Surg* 1968;96:999-1007.
188. Reckling FW, Peltier LF. Riccardo Galeazzi and Galeazzi's fracture. *Surgery* 1956;58:453-459.
189. Rose-Innes AP. Anterior dislocation of the ulna at the inferior radio-ulnar joint. *J Bone Joint Surg [Br]* 1960;42:515-521.
190. Schuind F, An K, Bergland L, et al. The distal radioulnar ligaments. *J Hand Surg [Am]* 1991;16:1106-1114.
191. Snook GA, Chrisman OD, Wilson TC, et al. Subluxation of the distal radio-ulnar joint by hyperpronation. *J Bone Joint Surg [Am]* 1969;51A:1315-1323.
192. Stahl.
193. Vesely DG. The distal radio-ulnar joint. *Clin Orthop Rel Res* 1967;51:75-91.
194. Walsh HPJ, McLaren CAN. Galeazzi fractures in children. *J Bone Joint Surg [Br]* 1987;69:730-733.
195. Warren JD. Anterior interosseous nerve palsy as a complication of forearm fractures. *J Bone Joint Surg [Br]* 1963;45:511-512.
196. Werner F, Palmer A, Fortino M, et al. Force transmission through the distal ulna.

CHARLES T. PRICE  
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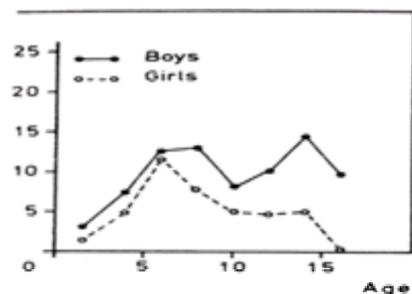
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## FRACTURES OF THE SHAFTS OF THE RADIUS AND ULNA

Fractures of the radius and ulna shafts account for only 3% to 6% of all children's fractures ( 64,65) (Fig. 0-1). In boys, there is a bimodal peak, the first at approximately age 9 years and the second at approximately 13 or 14 years of age. Girls show a single peak at approximately age 5 or 6 years ( 65). Approximately 75% of fractures of the shafts of the radius and ulna are in the distal third, 15% in the middle third, and 5% in the proximal third. Monteggia fracture–dislocations and complex injuries account for the remaining 5% (25,38,123).

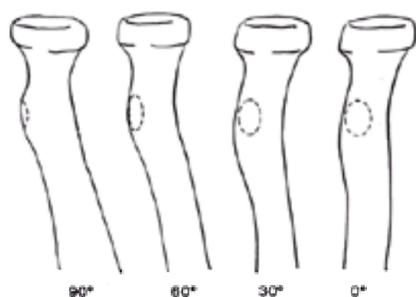


**FIGURE 10-1.** The annual incidence per 10,000 children of fractures of the shaft of the radius and ulna for the various age groups. Note the bimodal peaks for boys and single peak for girls. (From Landin LA. Fracture patterns in children: analysis of 8,682 fractures with special reference to incidence, etiology and secular changes in a Swedish urban population, 1950–1979. *Acta Orthop Scand Suppl* 1983;202:65; with permission.)

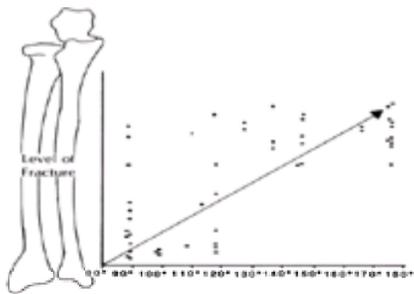
Traditional teaching ( 30,42) developed a concept of postreduction positioning based on which third of the radial and ulnar shafts was fractured:

1. If the fracture of the shafts was *proximal* to the insertion of the pronator teres, the forearm should be held in *supination*.
2. If the fracture was in the *middle third*, *mid-position* was advised.
3. If the fracture was in the *distal third*, *pronation* was the position of choice.

This concept was challenged by Evans, who recognized two groups of fractures: those in which the shaft of the radius remained in continuity and those in which it was completely displaced. He advocated pronation or supination of greenstick, Monteggia, and Galeazzi fractures based on the mechanism of injury ( 29,30). For complete fractures, Evans recommended determining the position of the bicipital tuberosity of the radius on an anteroposterior (AP) radiograph, then placing the distal fragment in the same amount of rotation to effect reduction (Fig. 10-2). He found that all such fractures usually needed to be immobilized in some degree of supination ( Fig. 10-3).



**FIGURE 10-2.** The normal bicipital tuberosity from full supination (90 degrees) to mid-position (0 degrees). In children, these characteristics are less clearly defined. (Redrawn from Evans EM. Fractures of the radius and ulna. *J Bone Joint Surg Br* 1951;33:548; with permission.)

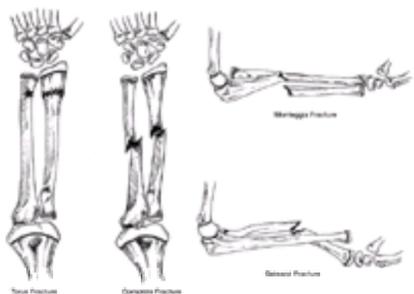


**FIGURE 10-3.** The rotational position of the upper radial fragment in 50 cases, plotted against the level of fracture. Each case is represented by a black dot. (Redrawn from Evans E. M. Fractures of the radius and ulna. *J Bone Joint Surg Br* 1951;33:548; with permission.)

## Diagnosis

### Mechanism of Injuries

Because cortical bone is strong, it often requires greater trauma to produce failure of the diaphysis than the metaphysis. As is typical of most fractures in the upper extremity, fractures of the radial and ulnar shafts usually are caused by indirect forces, such as landing on the outstretched upper extremity ( [Fig. 10-4](#)). When the child falls, the main deforming force is transmitted to the radius. The radius usually fails first, followed by the ulna ( [128](#)). Shaft fractures are more common in younger children because the cortical bone is more porous and the transition from diaphysis to metaphysis is less distinct than in older children ( [128](#)).



**FIGURE 10-4.** A fall on the outstretched hand can produce any of these shaft fractures: torus fracture (A); complete fracture (B); Monteggia fracture–dislocation (C); Galeazzi fracture–dislocation (D).

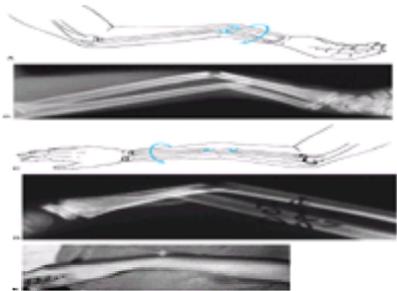
### Signs and Symptoms

Pain, swelling, crepitus, and deformity make the diagnosis obvious. However, plastic deformation injuries present primarily with deformity, mild tenderness, and limited forearm rotation. Pain and swelling may be minimal and crepitus is absent in plastic deformation injuries and minor greenstick fractures.

### Radiographic Findings

#### Rotational Alignment

Anteroposterior and lateral radiographs that include both the proximal and distal radioulnar joints are essential for an accurate analysis of rotation of the fracture fragments. The radius is a curved bone and malrotation can be recognized by a break in the smooth curve of the radius and a sudden change in the width of the cortex ( [17,68,76,82](#)). Radiographs should also reveal whether fractures are complete or incomplete. Incomplete, or greenstick, fractures with angulation usually have a rotational deformity ( [Fig. 10-5](#)). If the fracture is complete, the distal fragment may be in any position, but the position of the proximal fragment is determined by muscle pull.



**FIGURE 10-5.** Angulation plus rotation. **A and B:** Forced supination of the forearm produces a fracture pattern with apex volar angulation in addition to dorsal displacement with supination of the distal fragment. **C and D:** Forced pronation of the forearm can result in a fracture pattern with apex dorsal angulation, in addition to pronation and volar displacement of the distal fragment. **E:** Clinically, the apex dorsal angulation (*arrow*) in pronation injuries is much more prominent.

For complete fractures, it is necessary to determine the position of the proximal fragment so that the distal fragment can be positioned to align with the same amount of rotation as the proximal fragment. This can be determined by the position of the bicipital tuberosity in the fractured radius. The bicipital tuberosity is medial when the forearm is supinated, posterior when in mid-position, and lateral when pronated ( [Fig. 10-2](#)). If in doubt, radiographs of the opposite normal forearm should be obtained in supination, mid-position, and pronation to compare the position of the bicipital tuberosity.

Milch proposed the following anatomic criteria to determine proper torsional alignment of the forearm bones, regardless of the degree of rotation ( [76](#)):

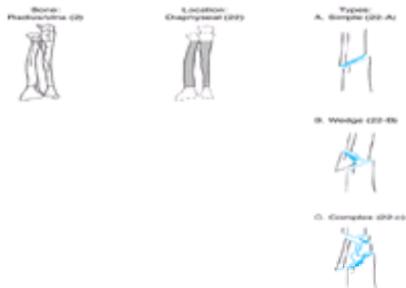
1. The coronoid process of the ulna points anteriorly and the styloid process of the ulna points posteriorly in the lateral projection. The radial styloid is not seen in the normal lateral projection with the forearm supinated.
2. In the normal AP view with the forearm supinated, the coronoid and styloid processes of the ulna are hidden but the radial styloid and bicipital tuberosity are seen. For example, in a completely supinated normal forearm, an AP view shows both the bicipital tuberosity and the radial styloid, but neither the coronoid process nor the ulnar styloid is seen. The lateral view shows both the coronoid process and the ulnar styloid, but neither prominence of the radius is seen. Thus, if the styloid process of the ulna is seen on the AP view and is absent on the lateral view, a torsional deformity of the ulna is present. If the bicipital tuberosity of

the radius projects medially in the AP view and the radial styloid is not seen, torsional deformity of the radius is present.

3. In the mid-position no bony prominences are seen in the AP projection, but all four prominences are seen on the lateral view. Angulation usually is associated with rotation (Fig. 10-5).

### Classification

The Orthopaedic Trauma Association (OTA) has classified fractures of the radius and ulna with the numeric designation of "2." Diaphyseal fractures are classified with a second number "2." Thus, all shaft fractures of the radius and ulna are designated "22." Simple fractures are given the letter "A," wedge fractures are "B," and complex fractures are "C" (Fig. 10-6). Wedge and complex fractures are uncommon in children, so most pediatric shaft fractures are designated 22-A. When only the ulna is fractured, this is identified with an additional numeral "1" (22-A1). An isolated simple radial fracture is identified with an additional "2" (22-A2). The most common fracture is a simple fracture of both bones. Simple both-bone shaft fractures are identified by adding the numeral "3" (22-A3) (Fig. 10-7). Subgroups are identified by placing a decimal and using additional numerals. For example, a simple fracture of the ulnar shaft with radial head dislocation, Monteggia fracture-dislocation, is identified as 22-B1.3 (Fig. 10-8). More complex fractures have additional alphanumeric classifications to allow for standardization of research and communication (92).



**FIGURE 10-6.** Orthopaedic Trauma Association Classification. Fractures of the radius and ulna are designated by the numeral "2." Diaphyseal fractures are identified by a second numeral "2." Simple fractures are given the letter "A," wedge fractures are "B," and complex fractures are "C." (Redrawn from Orthopaedic Trauma Association Committee for Coding and Classification. Fracture and dislocation compendium. *J Orthop Trauma* 1996;10[Suppl 1]:1; with permission.)



**FIGURE 10-7.** Orthopaedic Trauma Association Classification. When only the ulna is fractured, this is identified by the additional numeral "1." Isolated radius fracture is identified with an additional "2." Both bone fractures are identified by adding the numeral "3." (Redrawn from Orthopaedic Trauma Association Committee for Coding and Classification. Fracture and dislocation compendium. *J Orthop Trauma* 1996;10[Suppl 1]:1; with permission.)

### 3. With dislocation of radial head (Monteggia) (22-B1.3)



**FIGURE 10-8.** Orthopaedic Trauma Association Classification. Subgroups of forearm fractures are identified by the use of a decimal and additional numerals. Monteggia fracture dislocation is classified 22-B1.3. (Redrawn from Orthopaedic Trauma Association Committee for Coding and Classification. Fracture and dislocation compendium. *J Orthop Trauma* 1996;10[Suppl 1]:1; with permission.)

The OTA Classification System has some deficiencies when applied to pediatric and adolescent forearm fractures. Greenstick fractures and plastic deformation are not classified separately. Comminution is rare in children and often has little clinical significance. Treatment decisions in the pediatric age group rarely depend on specific classification. Closed reductions are common in the pediatric age group and clinical decisions are based on degree of completion and level of the fracture, combined with severity and direction of deformity.

The traditional classification of shaft fractures of the radius and ulna is outlined in the following sections.

### Joints Intact

Shaft fractures in which the proximal and distal radioulnar joints remain intact can be subcategorized by their degree of completion, direction of deformity, or level of fracture (Table 10-1):

**Radioulnar Joint Intact**  
 Degree of completeness  
 Plastic deformation  
 Greenstick fracture  
 Complete disruption  
 Direction of deformity (greenstick)  
 Apex volar (supination injury)  
 Apex dorsal (pronation injury)  
 Level of fracture  
 Proximal third  
 Middle third  
 Distal third  
**Radioulnar joints disrupted**  
 Proximal—Monteggia fracture–dislocation complex  
 Distal—Galeazzi fracture

**TABLE 10-1. CLASSIFICATION: SHAFT FRACTURES**

*Degree of completion:* The degree of completion usually is defined as plastic deformation, greenstick, or complete. Proper identification is important because each of these types is managed differently.

*Direction of deformity:* The direction of the deformity can be identified as either apex volar (supination injury) or apex dorsal (pronation injury). This distinction is particularly important for management of greenstick fractures.

*Level of fracture:* The level of fracture is identified as occurring in the proximal, middle, or distal third.

**Joints Disrupted**

These types of forearm shaft fractures include Monteggia fracture–dislocation, when the ulnar shaft fracture is associated with radial head dislocation proximally, and Galeazzi fracture–dislocation, when the radial shaft fracture is associated with dislocation of the distal radioulnar joint. Monteggia and Galeazzi fractures are discussed elsewhere in this textbook, but the clinician must always be cognizant of the possibility of occult or missed joint disruption whenever a forearm fracture is identified.

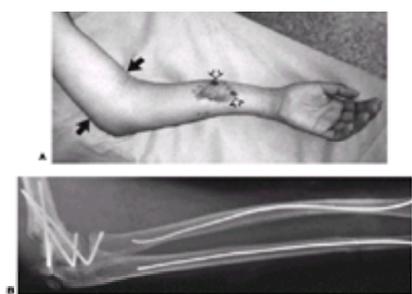
**Unusual Fracture Patterns**

Plastic deformation injuries also may be difficult to detect and require special management techniques.

The so-called floating elbow (Fig. 10-9) is a special variant that combines a fracture of the humerus with a forearm fracture. Ipsilateral forearm fractures are seen in 3% to 13% of supracondylar fractures (99,119,149). The supracondylar fracture usually is displaced and requires closed reduction with pinning before the forearm fracture can be treated. Nondisplaced supracondylar fractures may not require pinning (149). After stabilization of the supracondylar fracture, the forearm fracture can be treated with closed reduction and plaster immobilization if the forearm fracture is stable or minimally displaced (99,119,149). However, when the forearm fracture is unstable or open, or if there is extensive soft tissue injury, plate or intramedullary fixation may be a more appropriate method to maintain reduction (Fig. 10-10).



**FIGURE 10-9.** “Floating elbow” injury. **A and B:** A 5-year-old with ipsilateral grade II open fracture of the radius and ulna and a type III, closed supracondylar humerus fracture. **C and D:** Irrigation and debridement of the forearm wound followed by intramedullary stabilization of the ulna. The supracondylar fracture was treated by open reduction and pinning. **E and F:** All fractures healed at 4 weeks after surgery before pin removal in the office. **G and H:** Forearm and elbow at 1 year after surgery are virtually anatomic.



**FIGURE 10-10.** Ipsilateral supracondylar fractures. **A:** A 10-year-old with grade I open fractures of the radius and ulna (open arrows). An ipsilateral supracondylar fracture is manifest by considerable swelling at the elbow (large black arrows). **B:** The supracondylar fracture was first manipulated and reduced and then secured with pins placed percutaneously. The forearm fracture was then cleaned and debrided, and secured with intramedullary pins inserted distally.

Segmental, comminuted, or open forearm fractures usually indicate high-energy trauma. More aggressive management usually is warranted for these types of injuries.

**Anatomy**

The radius bows laterally immediately distal to the bicipital tuberosity. The bicipital tuberosity is distal to the neck on the medial aspect of the shaft and slightly anterior. The radius has one distinct border for the attachment of the interosseous membrane. The proximal radius is cylindrical, changing to a triangular shape at the junction of the proximal and middle thirds and flattening at its lower end. The radius, like the ulna, is a thick-walled bone throughout the greater part of the shaft.

The radius is bound to the ulna by the interosseous membrane and annular ligament at the proximal radioulnar joint. The triangular articular disk binds the radius to the ulna at the distal radioulnar joint. These structures allow pronation and supination movements of the forearm. The interosseous distance is greater distally than proximally because of the curve of the radius. In pronation, the radius rotates diagonally across the ulna and the palm faces posteriorly. The crossover point of the two bones is in the proximal forearm. Walker demonstrated that longitudinal forces of less than 9.8 kg are transmitted by the interosseous membrane to the ulnohumeral

joint and loads of greater magnitude are transmitted to the radiohumeral joint ( [142](#)).

### Treatment Principles

The objective of successful treatment of greenstick or complete fractures is full recovery of forearm rotation without cosmetic deformity. Delayed unions and nonunions are rare in children. Thus, closed reduction and cast immobilization is often successful and should be attempted in most fractures.

Cadaver studies ( [76,122](#) ) have demonstrated that 10 degrees of residual fixed angular deformity in the middle or distal third of the radius or ulna does not cause functional loss of forearm rotation. Rotatory deformities produced losses of pronation and supination equal to the degree of rotational deformity ( [122](#) ). Sarmiento et al. compared experimental results from cadaver studies with clinical radiographic findings in 105 patients with residual deformities and concluded that limitation of rotation is within clinically acceptable ranges with residual angulation of 10 degrees or less ( [113](#) ).

Spontaneous correction of residual angulation can occur in children. The amount of correction depends on age, the distance from the physis, the severity of deformity, and the direction of angulation. Greater degrees of correction can be expected in younger children and in more distal fractures ( [11,24,38,54,89,139,153](#) ). Onne and Sandblom observed that during the first decade of life, the shafts of the forearms have an excellent capacity of correcting angulation up to 20 degrees spontaneously ( [89](#) ). Others have confirmed this remodeling potential ( [38,50,79,95,96,123,139](#) ). The capacity for remodeling diminishes after the age of 10 years. Angulation of more than 10 degrees after the age of 10 years is unlikely to remodel ( [35,50,54,89,95,96,139](#) ).

Malrotation is difficult to measure and may or may not remodel ( [21,35,42,95,96](#) ). Fuller and McCullough observed that malrotation of the fracture limited movement to the same degree as the rotational deformity ( [35](#) ). Grant and Weiss observed that malrotation does not correct with growth, but noted that 30 degrees of malrotation may be accepted without functional deficit ( [42](#) ).

Determining acceptable limits for angulation at the time of reduction is complicated by the fact that malunion does not always correlate with loss of forearm rotation. Daruwalla observed that it is difficult to predict the extent of limitation of forearm movement with varying degrees of angulation ( [24](#) ). Hogstrom et al. observed that the correlation between final angulation and range of pronation and supination is weak ( [50](#) ). This observation has been confirmed by others ( [42,51,84,95,96,153](#) ).

From a functional standpoint, Carey et al. ( [15](#) ) noted that patients older than 10 years of age might have residual changes on radiographs without a commensurate loss in range of motion. They reported on nine patients, 11 to 15 years of age, with an average angulation of 13 degrees (range, 5–30 degrees). Five of the nine patients lost forearm rotation ranging from 20 to 35 degrees, but none had functional deficits. They concluded that none of these patients would have been better off with open reduction. Thomas et al. ( [123](#) ) reviewed 65 malunions in children up to age 15 years and concluded that up to 15 degrees of angular deformity is acceptable because the final loss of function is negligible. When treating forearm fractures, the clinician should consider the statement by Hey Groves that “art should secure supination and nature be trusted to secure pronation” ( [48](#) ). A supination loss cannot be compensated well by adduction at the shoulder, but a pronation loss can be masked by abduction at the shoulder.

Nilsson and Obrant examined 18 adults who had sustained displaced forearm fractures as children ( [84](#) ). All had been reduced and maintained in good position without displacement or angulation. The average loss of pronation–supination motion was 19 degrees, even in the absence of malunion. Thus, factors other than residual angulation may also be responsible for loss of forearm rotation ( [84,122](#) ).

Another difficulty in predicting functional outcome is that some younger patients lose forearm rotation regardless of remodeling ( [95,96](#) ). Younger et al. noted that the mean age of patients with restricted movement was 7.2 years ( [153](#) ). Holdsworth and Sloan noted that functional improvement is complete by 3 years after injury, but radiographic appearances may continue to improve beyond that time ( [51](#) ). As a rule, older patients are at greater risk for loss of pronation–supination motion, but this is not always the case ( [24,35,105](#) ).

Length discrepancy between the radius and ulna is uncommon after shaft fracture of one or both bones ( [21,27,95,96,139](#) ). Limb overgrowth or shortening does not occur if the physis is undamaged ( [15,27](#) ). This suggests that remodeling can occur with regard to length as well as angulation.

Overriding of fracture fragments is another consideration. Blount and Johnson stated that overriding with bayonet apposition is acceptable if the angular alignment is satisfactory ( [11](#) ) ( [Fig. 10-11](#) ). This observation has been confirmed by others ( [41,95,96,98](#) ).



**FIGURE 10-11.** Bayonet apposition. **A:** An 11-year-old sustained a fracture of the radial and ulnar shafts with bayonet apposition but excellent alignment. **B:** Four weeks after the fracture, there is early callus with maintenance of the bayonet apposition but satisfactory linear alignment. **C and D:** Five years postinjury (age 16 years), there is excellent linear alignment and full supination and pronation.

The direction of angulation may influence loss of forearm rotation. Daruwalla ( [24](#) ) observed that angulation of both bones, with the apex of the angle toward the interosseous space, resulted in loss of motion. Roberts ( [104](#) ) confirmed this finding, but Price et al. ( [95,96](#) ) stated that interosseous encroachment is an unpredictable indicator of loss of motion.

Location of the fracture is another factor that influences outcome. Numerous researchers have noted that proximal shaft fractures have a worse prognosis than distal shaft fractures ( [10,21,24,38,95,96,123,130,153](#) ).

Recommendations for acceptable reduction vary. Moesner and Ostergaard ( [79](#) ) stated that children younger than 9 years of age with angulation of less than 20 degrees will regain full range of motion and 90% remodeling. This conclusion has been supported by Carey et al. ( [15](#) ) and others ( [54,89,39](#) ). However, Blount ( [10](#) ) cautioned, “fractures of the middle third of the forearm should not be allowed to remain angulated to any appreciable degree except in very young children.” Daruwalla ( [24](#) ) concluded that after age 6 years, remodeling is unlikely to correct a deformity of more than 10 degrees. Daruwalla believed that angulation of 15 degrees is acceptable in children younger than 5 years. Hogstrom et al. ( [50](#) ) noted that young children have a good chance of achieving correction of angular deformity, but concluded that all deformities exceeding 10 degrees should be corrected because it is impossible to predict remodeling. Price et al. ( [95,96](#) ) concluded that 10 degrees of angulation, complete displacement, and loss of radial bow can be accepted rather than resorting to open reduction ( [Table 10-2](#) ).

Age	Angulation	Malrotation	Displacement	Loss of Radial Bow
Age <3 yr	15 degrees	45 degrees	Complete	Yes
Age ≥3 yr	10 degrees	30 degrees	Complete	Partial

**TABLE 10-2. LIMITS OF ALIGNMENT**

## Treatment Options

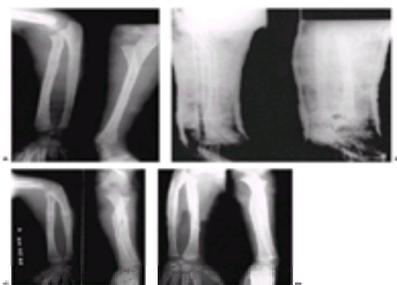
### Closed Management

#### Greenstick Fractures

Skillful closed management usually is successful for this injury. Greenstick fractures may appear to be angulated and may also have a rotational element. Holdsworth and Sloan compared this fracture with a cardboard tube that, when twisted, tends to bend; unbending it is possible only if it is untwisted ( 51). When the apex is toward the dorsum of the hand (apex dorsal-pronation injury), the forearm should be supinated to achieve reduction ( Fig. 10-12). When the apex is toward the palm of the hand (apex volar-supination injury), a pronation force must be applied to secure reduction ( Fig. 10-13).



**FIGURE 10-12. A:** Greenstick fracture of both shafts of the forearm at the distal third with pronation of the distal forearm and apex dorsal angulation at the fracture site. **B and C:** The fracture was manipulated into supination and placed in a long arm cast, correcting the angular and rotational malalignment.



**FIGURE 10-13. A:** Greenstick fracture of the proximal third of the radius and ulna, with apex volar angulation of the radius. **B:** Postreduction view in long arm cast shows 40-degree angulation of the radius. This original reduction was in neutral but should have been in full pronation. As a result, there is some residual angulation at the fracture site. **C:** At 1 month, there was 15-degree residual angulation of the proximal radius, which was thought would remodel to some degree. **D:** At 8 months postinjury, angulation of the radius persisted, with supination of 45 degrees and pronation to 70 degrees. Despite the residual angulation, the patient does not complain of any functional impairment.

Should greenstick fractures be made complete? There appears to be no unanimity of opinion. Advocates for breaking the intact cortex believe that angulation will recur if it is not done ( 11,43,53,98,146). Others believe it is unnecessary to break the intact cortex ( 1,25,30,33,123). Instead, the intact cortex is used as an aid to reduction. Advocates for leaving the hinged cortex intact emphasize the need for full pronation for apex volar (supination) greenstick fractures and full supination for apex dorsal (pronation) greenstick fractures ( 1,25,33) ( Fig. 10-12 and Fig. 10-13).

Another reason to break the intact cortex was proposed by Gruber ( 43), who noted that greenstick forearm fractures have the highest risk of refracture of all pediatric fractures. He postulated that the intact cortex heals by primary bone healing with little callus. Simultaneously, a resorption zone develops on the side of the broken cortex. Thus, the weakness of the bone persists beyond the initial healing phase. He recommended initial fracturing of the intact cortex to reduce the risk of fracture recurrence.

## AUTHORS' PREFERRED METHOD OF TREATMENT

Even with minimally angulated greenstick fractures, the elbow should be evaluated carefully to ensure that a Monteggia type IV lesion with an ipsilateral radial head dislocation is not present.

Minimal angulation can be accompanied by significant rotational deformity. Any angulation should be reduced if a family member or other observer has made specific comments that the forearm appears deformed. The deformity present immediately after injury often is the final position into which the fracture tends to drift when the swelling has subsided.

Pain relief for fracture reduction may be achieved by a variety of methods ( 5,20,31,46,55,57,72,88,133,145,147). The American Academy of Pediatrics has published guidelines for the monitoring and management of children during and after sedation ( 2) (see also Chapter 3). For greenstick fractures, I prefer a minidose Bier block as described by Juliano ( 57), or intravenous sedation as described by Varela et al. ( 133). Intravenous sedation provides adequate analgesia for one reduction attempt, which usually is sufficient. A Bier block provides more prolonged analgesia.

Supinating and extending the wrist while gently correcting the angulation ( Fig. 10-12) reduces apex dorsal angulated greenstick fractures (pronation injuries). Volar

angulated greenstick fractures (supination injuries) are reduced by pronating and flexing the wrist while gently correcting the angulation. I do not intentionally fracture the opposite cortex, but a crack often is heard as the reduction is completed. Full pronation or full supination maintains the reduction if the opposite cortex remains intact. A sugar-tong splint is then applied, with the elbow flexed to a right angle.

Approximately 1 week later, the splint is removed and a well-molded long arm cast is applied. Radiographs should be made at that time to determine whether reduction has been maintained. Cast immobilization is continued for 6 weeks. I recommend a Velcro splint for an additional 6 weeks because of the high incidence of refracture with this injury.

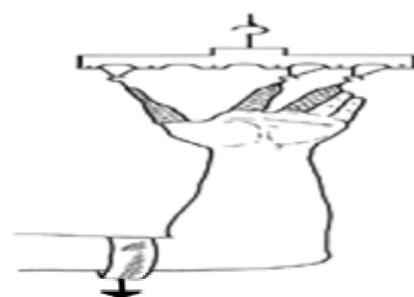
### Complete Fractures

If the fracture is complete, an entirely different situation exists. The distal fragment may be in any position, but muscle pull determines the position of the proximal fragment (Fig. 10-14). Thus, it becomes necessary to determine the position of the proximal fragment so that the distal fragment can be aligned with it.



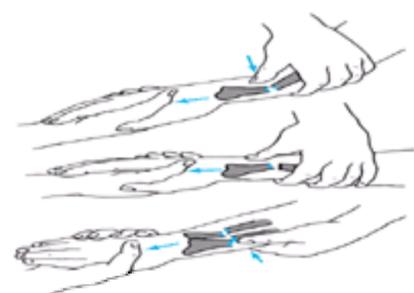
**FIGURE 10-14.** Muscle forces that deform fractures of the radius above the level of the insertion of the pronator teres. The proximal fragment supinates because of the unopposed pull of the supinator and biceps muscles. (Redrawn from Cruess RL. The management of forearm injuries. *Orthop Clin North Am* 1973;4:969; with permission.)

If both fractures are complete and overriding, Davis and Green (25) advocated fingertrap traction of 10 to 15 pounds to bring out to length and to let the fracture seek its own correct rotational alignment. Traction through fingertraps with countertraction on the arm tends to stretch an intact periosteum to allow bone ends to oppose (Fig. 10-15).

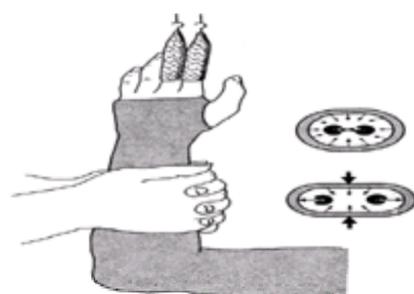


**FIGURE 10-15.** Application of traction-countertraction using fingertraps. (Redrawn from Weber BG, Brunner C, Freuler F. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980; with permission.)

Alternatively, it is possible to increase the deformity and toggle one bone at a time by thumb pressure, and then immobilize in a long arm cast in correct rotation with the elbow flexed to 90 degrees (Fig. 10-16). The cast should be molded into an oval (Fig. 10-17). If the reduction is unsatisfactory, remanipulation is necessary.



**FIGURE 10-16. Top:** Traction and countertraction of the thumb is used to increase the deformity. **Center:** With traction still maintained, the thumb slips farther distally to correct the angulation. It is best to avoid disrupting the periosteum, but on occasion this is necessary. **Bottom:** Ulnar or radial deviation can also be corrected by traction and thumb pressure. (Redrawn from Weber BG, Brunner C, Freuler F. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980; with permission.)

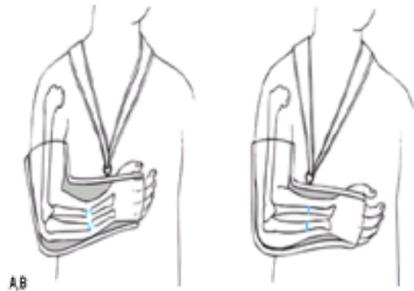


**FIGURE 10-17.** While the cast hardens, it is pressed together by both hands to form an oval. This increases the width of the interosseous space. Traction should be released gradually while this is done. (Redrawn from Weber BG, Brunner C, Freuler F. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag,

1980; with permission.)

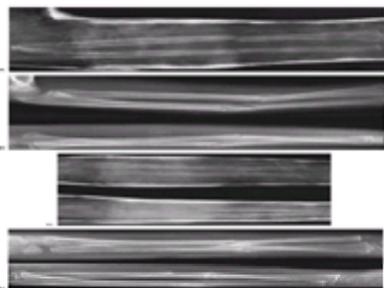
The proper position for immobilization depends on the fracture. Evans recommended that all complete both-bone fractures should be immobilized in some degree of supination (29). Carey et al. (15) advocated immobilization of all fractures in neutral position without extremes of pronation or supination. Proximal fractures are more likely to require supination for maintenance of reduction (11,29,30).

One of the major difficulties in treating fractures of the shafts of the radius and ulna is the development of late angulation because of slippage of the forearm proximally in the long arm cast when the elbow is flexed to 90 degrees (Fig. 10-18). In an effort to prevent this, Rang (98) originally placed a ring proximally on the cast to counteract this migration (Fig. 10-18). Another option is to immobilize the forearm with the elbow in extension.



**FIGURE 10-18.** Movement within the cast. **A:** As the swelling subsides, the forearm migrates proximally in the cast, causing it to angulate. **B:** This migration can be prevented to some extent by attaching the suspension eyelet proximally. (Redrawn from Rang M. *Children's fractures*, 2nd ed. Philadelphia: JB Lippincott, 1983:207; with permission.)

The elbow usually is immobilized in flexion, but this is not always necessary or desirable. The use of a long arm cast in extension was originally described in the 1940s by Thorndike and Dimmler (126). It was also advocated by Watson-Jones (144) in the 1950s. In a series of 130 pediatric forearm fractures, Gainor and Hardy (37) found it necessary to treat eight patients in elbow extension because reduction was unstable in flexion. Walker and Rang (142) reported satisfactory results in 15 patients who required management with the elbow in extension. They noted that most forearm fractures, which are stable in flexion, should still be treated in a conventional cast, but unstable fractures can safely be treated with the elbow in extension (Fig. 10-19 and Fig. 10-20).



**FIGURE 10-19.** Long arm extension cast. **A:** Postreduction film in an 8-year-old boy showing satisfactory alignment with elbow in 90 degrees of flexion and the forearm in mid-position. **B:** Radiographs taken 4 weeks postfracture show 30 degrees of angulation at the fracture site. **C:** The forearm was remanipulated and placed in a long arm cast in extension. **D:** Six weeks after remanipulation, there is satisfactory alignment of both fracture fragments. Subsequently, the patient regained full supination and pronation of the forearm. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:36; with permission.)



**FIGURE 10-20.** **A:** An extension cast on a 7-year-old with fractures of the shafts of the radius and ulna. **B:** The thumb must be incorporated in the cast, with extra padding placed over the base of the thenar eminence (arrow) to prevent the development of pressure areas from the distal migration of the cast.

Radiographs should be made at intervals for up to 3 weeks after reduction. Minor loss of alignment at this time is observed. Major loss of alignment requires remanipulation under anesthesia. Voto et al. (140) noted that 7% of their pediatric forearm fractures required remanipulation. Jones and Weiner (56) advocate closed management with pins and plaster technique as a salvage technique when casting alone is insufficient.

## AUTHORS' PREFERRED METHOD

When fractures of both bones are complete, the muscle pull on the fragments becomes important. It is necessary to determine the rotation of the proximal fragment and then place the distal fragments in the same amount of rotation.

For these fractures, I prefer prolonged and complete pain relief, because more than one attempt may be required. Regional anesthetic techniques such as Bier or axillary blocks or general anesthesia are often better for this injury than hematoma block or intravenous sedation.

Initially, I attempt a manual closed reduction. An assistant applies countertraction at the elbow. I use one hand to apply traction while the other manipulates the fracture site. If necessary, I increase the deformity by thumb pressure and hitch one bone at a time. The forearm is then placed in the selected position of rotation.

This position is almost always neutral to supination and rarely requires any degree of pronation. The elbow usually is flexed to 90 degrees and a well-molded sugar-tong splint is applied.

If the initial or subsequent attempts at manual reduction fail, then traction is applied by fingertraps ( [Fig. 10-15](#)). The arm is allowed to seek its own position of rotation, as described by Carey et al. ( [16](#)). Plain radiographs or image intensification is used to check alignment with the arm in traction. A long arm cast is then applied with careful molding to maintain the interosseous space and the alignment ( [Fig. 10-17](#)). When the cast hardens, the traction is released and further radiographs are obtained. The cast is split on one side in the recovery room. I leave the padding intact unless problems with swelling are likely.

I tend to use the ulna as the primary indicator of angulation and the radius as the indicator of rotation. Initially, I accept overriding but I try not to accept angulation; angulation tends to lead to increasing deformity on follow-up. In a child younger than 9 years of age, I accept 15 degrees of angulation, 45 degrees of malrotation, complete displacement, and straightening of the radius. From ages 9 to 14 years in girls and 9 to 15 years in boys, I accept 10 degrees of angulation, 30 degrees of malrotation, complete displacement, and some loss of radial bow ( [Table 10-2](#)).

Radiographs are made at 1 week, 2 weeks, and occasionally at 3 weeks after reduction. It is necessary to change the cast as swelling subsides after reduction. Changing the splint or cast at 1 week is painful and often unnecessary. I prefer to wait until 10 to 14 days after injury for the first cast change. At that time, the fracture is "sticky" and gentle molding can be performed to correct any mild loss of alignment. Severe loss of alignment requires remanipulation under anesthesia with or without surgical stabilization. Remanipulation is an effective means to manage reangulation up to 21 days after initial management.



## OPERATIVE TREATMENT: OPEN REDUCTION AND INTERNAL FIXATION

### Indications

Operative indications are controversial for radial and ulnar shaft fractures in children. Holdsworth and Sloan ( [51](#)), in a report of 51 children with malunions of proximal shaft fractures, noted only three unsatisfactory results and concluded, "there is little place for internal fixation of proximal forearm fractures in children under the age of 12 years." There is no argument that open fractures require surgical debridement and good evidence that internal fixation of the fracture facilitates management of the soft tissue component of the injury ( [44,86,152](#)). For similar reasons, a compartment syndrome or soft tissue interposition can be a primary indication for surgical intervention ( [36,39,40,45,62,100,108,150](#)). Open reduction and internal fixation may also be indicated as the primary treatment after refracture of the radius or ulna ( [4,51](#)). In this group of fractures, callus and soft tissue scarring often impede satisfactory reduction, and osteopenia caused by immobilization of the original fracture contributes to prolonged healing. Fractures that occur shortly before the cessation of growth often require internal fixation to achieve an acceptable result ( [146](#)).

Considerable controversy exists regarding the indications for surgery when alignment is difficult to maintain after closed reduction. Many researchers have noted that open reduction with internal fixation is rarely necessary for forearm fractures in children. Mild to moderate deformity is well tolerated even in older children ( [15,24,35,51,95,96,123](#)). However, Fuller and McCullough ( [35](#)) recommended surgical intervention for children 8 years of age or older if the fracture defies all attempts at closed reduction. Kay et al. ( [59](#)) recommended open reduction and internal fixation for patients older than 10 years of age if the fracture proves unstable by closed methods. Many other investigators ( [1,10,33,51,83,86,118,130,141](#)) have also recommended surgical intervention in older children when adequate reduction cannot be obtained by closed means.

Modern surgical and anesthetic techniques, prophylactic antibiotics, and intraoperative fluoroscopy have improved the results of operative management. Thus, there has been a trend toward more aggressive operative treatment of these fractures ( [23,34,52,69,91,102,132,152,154](#)). Frequent indications for operative treatment of forearm shaft fractures in children are listed in [Table 10-3](#).

Unstable fracture after attempted closed reduction  
 Unacceptable closed reduction (Table 10-2)  
 Open fracture  
 Multiple trauma—floating elbow  
 Refracture with displacement  
 Compartment syndrome  
 Segmental or comminuted fractures (these usually are unstable  
 but can sometimes be managed closed)  
 Age  
 Female  $\geq 14$  yr  
 Male  $\geq 15$  yr

TABLE 10-3. INDICATIONS FOR OPERATIVE TREATMENT OF FOREARM SHAFT FRACTURES IN CHILDREN

### Specific Operative Techniques

Several methods of surgical stabilization have been used in children ( [3,52,66,83,91,93,107,130,132,141,152](#)), including compression plates and screws, intramedullary rods, and external fixation with pins and plaster or with external fixation devices. Oblique cross-pin fixation usually is inadequate for diaphyseal forearm fractures because the bone diameter is small ( [148](#)).

**Compression Plates.** Several clinicians ( [1,18,59,83,124,130,152](#)) have reported the occasional use of plate and screw osteosynthesis for pediatric forearm fractures. Nielsen and Simonsen ( [83](#)) reported results of plate fixation in 29 children with forearm fractures. They emphasized that internal fixation was used for only 8% of the displaced forearm fractures when closed reduction was unsuccessful. One of their 29 patients contracted a deep infection, 1 had a refracture after plate removal, 2 reported slight sensory disturbance, and 8 had scars exceeding 5 mm in width. All fractures healed, and none lost more than 20 degrees of forearm rotation. Vainionpaa et al. ( [130](#)) also reported open reduction with plate fixation when closed management was unsuccessful. Nine percent of forearm fractures at their institution required surgical intervention. Thirteen of 14 patients regained satisfactory forearm rotation. There was one refracture and one unsightly scar. They concluded that the results of open reduction and internal fixation were satisfactory, considering the severity of the fractures.

Compression plate fixation should be considered in patients who have sustained refractures of original diaphyseal fractures ( [Fig. 10-21](#)) because in many cases poor results have followed closed management. These patients often have an intramedullary canal full of old callus or bone septa from the previous fractures that makes passage of intramedullary devices difficult. Price et al. ( [96](#)) and Creasman et al. ( [21](#)) recommended that refractures in these patients should be stabilized internally with compression plates.



**FIGURE 10-21. Refractures.** **A:** This 6-year-old boy sustained displaced greenstick fractures of the mid-shaft radius and ulna. He underwent satisfactory closed reduction and long arm cast application for 8 weeks with good healing. **B:** Two months later, he fell and sustained refractures through the old fracture sites with minimal displacement. **C:** After reimmobilizing for 6 weeks, satisfactory healing was achieved. **D:** Seven years later, he sustained a third fracture in essentially the same area. Closed reduction and long arm casting failed to obtain a satisfactory alignment. **E:** Because of the previous two fractures, compression plate fixation was used instead of intramedullary pin fixation. Subsequent healing was uneventful, with complete resumption of normal forearm motion.

Although there has been a reluctance to use plates in open fractures, in the series reported by Moed et al. (78), Haasbeek and Cole (43), and Wyrsh et al. (152), the use of compression plates in open fractures did not increase the problems of wound or fracture healing, and in fractures with more extensive soft tissue injury, it was thought that plate fixation facilitated wound care and fracture healing. The principles and techniques of open reduction and plate fixation are the same in children as in adults, except that smaller plates may be used in children. (81,105). A volar approach is preferred for fractures of the distal and middle thirds of the radius. For fractures in the proximal third of the radius, a dorsal approach is usually better. When both bones require stabilization, two incisions should be used to reduce the risk of synostosis. The ulna is easily accessed through a direct approach. (137,138). Accurate contouring of the radial plate is necessary to avoid loss of radial bow.

The main advantage of compression plate fixation is that it produces an accurate reduction with sufficient stability to begin early motion. However, supplemental immobilization is recommended in children because they are active (83,130,152).

Disadvantages include the soft tissue exposure, which can alter healing at the fracture site, scarring and problems with cosmesis, and the possibility of infection. (132)

Subsequent plate removal requires a second operation, which is not always a benign procedure. Refractures and neurapraxia have been reported in children after forearm plate removal (83,130). The complications after routine metal removal have caused some surgeons to question the value of this practice (6,58,74,114). We do not recommend routine plate removal unless the plate is causing discomfort or the child anticipates participating in activities that place him or her at unusual risk for refracture.

**Intramedullary Fixation.** Intramedullary nails have been used to stabilize adult forearm fractures since 1913, when Schone (115) first described the use of silver rods in the radius and ulna. Various types of nail or wire implants have been used since then (13,28,61,66,70,109,111,121). However, supplemental immobilization is often required, and nonunion rates in adults have ranged from 6% to 20% (61,70,90,111,117,121). In children, nonunions are rare, and minimal intramedullary fixation can maintain acceptable alignment until fracture healing occurs. Thus, intramedullary nailing of forearm fractures is more practical in children than in adults. The advent of image intensification has made it easier to stabilize a closed reduction with intramedullary devices inserted through percutaneous routes. The relative simplicity and low morbidity of intramedullary fixation have popularized this technique for pediatric forearm fractures in which the reduction may be difficult to maintain by cast immobilization alone (3,34,66,69,86,91,93,94,102,107,135,152,154).

Amit et al. (3) reported excellent results with no complications in a series of 20 adolescent forearm fractures managed by closed reduction and intramedullary fixation. Intramedullary fixation using elastic nails proved to be stable enough that postoperative immobilization was eliminated in the series reported by Verstreken et al. (135) and Lascombes et al. (66). Verstreken et al. reported excellent results with no complications in all 57 children in their series. Lascombes et al. reported 85 patients ranging in age from 4 to 16 years, 92% of whom regained full range of motion: only 1 patient lost more than 30 degrees of forearm rotation. There was one case of sensory neuropathy, nine instances of skin irritation over the end of the nail, three refractures, and four secondary angulations of less than 10 degrees, which were believed to be due to inadequate anchorage of the nail. There were no infections, nonunions, or cross-unions. Because Lascombes et al. did not accept theories of remodeling in malunited diaphyseal fractures, they advocated elastic stable intramedullary nailing of displaced forearm shaft fractures in all children older than 10 years of age and after failed conservative management in younger children.

More recently, other investigators have reported equally impressive results with flexible intramedullary nailing of forearm fractures in children. Richter et al. (102) reported excellent (24 fractures) or good (5 fractures) functional results in 29 of 30 children with "unstable" proximal- or middle-third forearm fractures treated by elastic stable intramedullary nailing. Luhman et al. (69) reported excellent or good results with intramedullary fixation of all 25 patients in their series with unstable diaphyseal forearm fractures. There were no infections, delayed unions or malunions, or synostoses. Based on this experience, they concluded that intramedullary nailing is a safe, effective, and user-friendly technique for management of unstable forearm fractures in children. Flynn and Waters (34) described excellent results in 16 children with both-bone forearm fractures using a modification of the technique in which fixation of only one bone is done and the other is treated closed.

Cullen et al. (23) reported 18 complications in 10 of 20 patients who had undergone intramedullary fixation of their forearm fractures. Problems included hardware migration, infection, loss of reduction, reoperation, nerve injury, loss of motion, synostosis, muscle entrapment, and delayed union. In spite of these complications, 19 of the 20 patients had excellent (n = 17) or good (n = 2) outcomes. Similarly, Shoemaker et al. (116) reported 9 complications in 8 of 32 patients with unstable forearm fractures treated with intramedullary fixation. In spite of these complications, 31 of the 32 patients had excellent results, and 1 had a good result. Complications in both series were more frequent when only one bone was stabilized or when immobilization was discontinued prematurely.

Van de Reis et al. (132) compared results of intramedullary nailing to plate fixation for treatment of forearm fractures in children. Results were excellent in 78% of the patients in either treatment group. Functional outcome, rate of union, and number of complications were statistically similar for the two groups. The advantages of intramedullary nailing included shorter operative time, better cosmesis, less soft tissue dissection, and easier hardware removal.

**Techniques of Intramedullary Fixation.** Contoured Kirschner wires or elastic nails can be introduced proximally or distally in the ulna but are always introduced distally in the radius (Fig. 10-22). Amit et al. (3) recommended pinning the ulna first through a 1-cm incision over the olecranon apophysis. Lascombes et al. (66) entered the ulna just distal and lateral to the olecranon apophysis. The radius is approached radially just proximal to the distal physis with protection of the superficial radial nerve. A 45-degree oblique hole is drilled in the lateral cortex of the metaphysis to allow nail insertion. Blunt-ended 1.5- to 2.5-mm diameter stainless steel or titanium nails are used. The nails are curved slightly to allow three-point fixation. The tip is bent 30 to 40 degrees to allow manipulation in the medullary canal. The bent tip should not exceed 5 mm in length. The nail is manipulated across the fracture site and advanced to the cancellous bone of the metaphysis. Rotation of the curved nail allows correction of angulation and restoration of radial bow. The nail is then bent at the insertion site and cut short enough to avoid skin irritation. Alternatively, the nail can be left percutaneously. The latter technique allows the implant to be removed in the office, although it may be associated with a slightly higher incidence of soft tissue problems. Approximately 15% to 20% of the fractures require a limited, direct exposure of the fracture site to achieve reduction (148,154).



**FIGURE 10-22. Intramedullary fixation.** **A:** In the Marote technique, a wire is inserted through a small opening in the distal radial metaphysis and through another small opening in the olecranon. (From Parsch K. The Marote wiring in proximal and midshaft fractures of the child. *Operat Orthop Traumatol* 1990;2:249; with permission.) **B:** This 10-year-old sustained a grade I open fracture midshaft to the ulna with a closed fracture of the radius. **C:** After debridement, fixation was achieved using wires inserted distally in the radius and proximally in the ulna.

The advantages of intramedullary nailing include minimal dissection at the fracture site, small incisions, low refracture rate, and easier removal than plates and screws. Elastic nail fixation has also been successfully used for open fractures in children ( [66](#)).

Disadvantages include the possibility of infection when closed fractures are managed surgically, the need for a subsequent procedure to remove the implants, skin irritation from the nail tip, and the risk of injury to the superficial radial nerve. Rigid immobilization of the fracture is difficult with elastic intramedullary fixation. Postoperative immobilization is recommended ( [3](#) ) but is not always necessary ( [66,135](#) ). Stabilization with compression plates is recommended when early mobilization is desirable. Compression plate fixation may be preferred for correction of malunions or when stabilization is required after re-fracture.

**Pins and Plaster, or External Fixation.** Voto et al. ( [141](#) ) reported the use of pins and plaster in 20 pediatric forearm fractures when a satisfactory reduction could not be managed by closed means alone. One Steinmann pin was placed through the first or second metacarpal neck and another transversely through the proximal ulna. Traction was then applied for reduction and was released after the pins were incorporated in a plaster cast. All patients obtained subjective satisfactory results without complications. A subsequent series by Jones and Weiner ( [56](#) ) confirmed the usefulness of this technique when closed management fails.

External fixators are rarely used for pediatric forearm fractures ( [95,115](#) ). The indications for use in children are rare and are similar to the indications for use in adults. These include extensive comminution, segmental bone loss, or grossly inadequate soft tissue coverage ( [97](#) ).

## AUTHORS' PREFERRED METHOD

Some investigators ( [59,66,83,130,135](#) ) reporting internal fixation of pediatric forearm fractures have indicated that 8% to 31% of midshaft fractures require surgery. ( [86](#) ) Voto et al. ( [140](#) ) reported surgical stabilization of only 1.5% of all pediatric forearm fractures.

Greenstick fractures rarely require internal fixation. Displaced forearm fractures almost always require operative intervention. The incidence of operative treatment should be less than 10% with proper closed management.

**Intramedullary Fixation.** When stabilization is required, I prefer intramedullary nailing with Kirschner wires or titanium rods. The tip must be blunt to prevent it from impaling the inner cortex. Because of their flexibility, the newer intramedullary nails or pins usually allow reconstruction of the normal bow of the radius. If a Steinmann or large straight pin is used, it may alter the normal bow of the radius, which may in turn affect forearm rotation ( [Fig. 10-23](#) ). A limited surgical exposure of the fracture site occasionally is needed to facilitate reduction. Reduction with small wires may not be anatomic but usually is adequate for healing and restoration of function. Fixation of only one bone (when both are fractured) is a less invasive modification of the technique that facilitates treatment by allowing the other fracture to be rotated into reduction. Although Flynn and Waters ( [34](#) ) have reported good results with this technique, others have cited a slightly higher risk of loss of reduction with single-bone fixation ( [23](#) ).

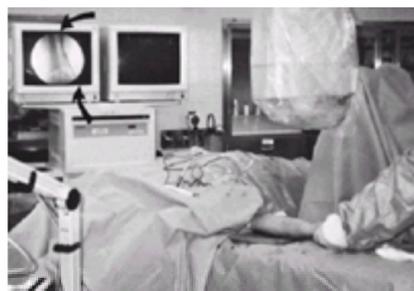


**FIGURE 10-23.** The natural bow of the radius may be lost, thus inhibiting forearm rotation, if a solid, straight intramedullary pin is used for internal fixation. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:41; with permission.)

The fracture is manipulated under the image intensifier and an attempt is made to obtain as nearly anatomic a reduction as possible. There should be at least 50% apposition of the bone ends at the fracture site. With less reduction, it is difficult to pass the wire across the fracture site. Passage is done with the patient supine and the forearm on a radiolucent arm board ( [Fig. 10-24](#) and [Fig. 10-25](#) ).



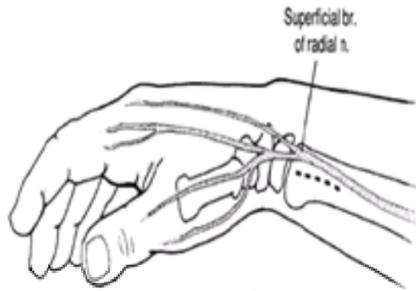
**FIGURE 10-24.** Correct positioning of the forearm on a radiolucent arm board. The image intensifier must be perpendicular to the long axis of the forearm and away from the operating surgeon.



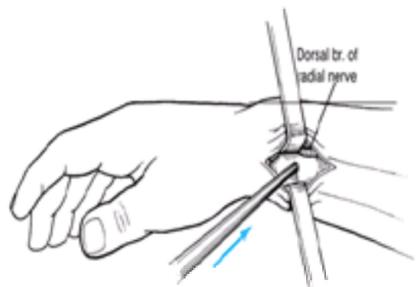
**FIGURE 10-25.** The image monitor ( *arrows* ) must be in the direct line of vision of the operating surgeon so that he or she can visualize pin migration at all times without turning his or her head.

If the fracture is open, I first do a standard debridement to decrease contamination. The preferred technique of treating open fractures is outlined in [Chapter 4](#). The fracture is then reduced by direct or indirect means.

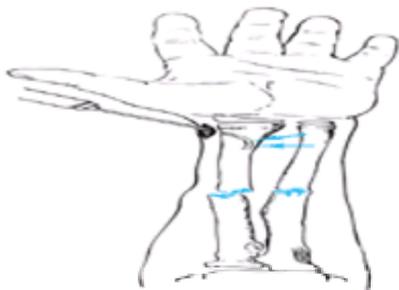
I follow the technique outlined by Klaus Parsch ([93](#)), who has used this procedure extensively in Europe. I try to reduce the radius first. The incision for introducing the pin into the distal radial metaphysis is made just proximal to the radial styloid. A small incision is made to allow visualization of the two branches of the superficial radial nerve ([Fig. 10-26](#)). Once the metaphysis of the distal radius is exposed, a drill hole (usually 2.5 mm in diameter) is made through the metaphysis, directing it somewhat craniad ([Fig. 10-27](#)). To allow the insertion of the pin and passage proximally up the shaft of the radius, the pin needs two bends in the distal portion. In the terminal 5 mm, a 30-degree bend is made in the rod, which eases its transfer across the fracture site. The second bend, a gradual curve approximately 1 to 1.5 cm proximal, allows the rod to bounce off the opposite internal cortex ([Fig. 10-28](#)). The rod is repeatedly rotated in each direction to facilitate its passage up the medullary canal; it can also be tapped with a small mallet.



**FIGURE 10-26.** Entrance of the radial pin. The incision is made over the radial aspect of the distal metaphysis, just below the two branches of the superficial radial nerve.

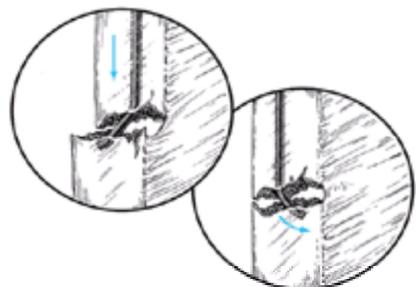


**FIGURE 10-27.** The entrance hole is drilled craniad under direct vision through the exposed distal radial metaphysis.



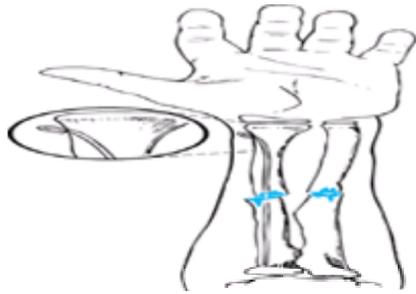
**FIGURE 10-28.** A 0.062<sup>2</sup> Kirschner wire is inserted through the drill hole into the metaphysis. The long curve (*large arrow*) of the distal pin facilitates passing it up the medullary canal. The 30-degree terminal bend (*small arrow*) is used to facilitate reduction.

When it reaches the fracture site, the second gentle curve in the rod usually has disappeared. The portion that remains is a small 30-degree bend in the tip of the rod, which facilitates its crossing the fracture site. Often, if the fracture is offset a bit, this bend can be set to engage the opposite cortex. The rod tip is then rotated to obtain reduction and passage up into the medullary canal of the proximal fragment ([Fig. 10-29](#)).



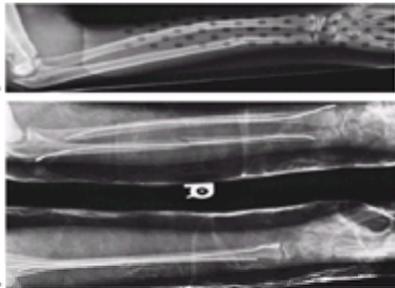
**FIGURE 10-29.** **Left:** The distal 30-degree angulation is used to facilitate entering the proximal fragment. **Right:** Once the proximal fragment is entered, the rod can be twisted or turned so that the correct alignment is achieved. The pin is then passed up the proximal fragment.

Once the reduction is achieved and the rod enters the proximal fragment, it is then passed up to the level of the radial tuberosity. The distal end of the rod is cut off and bent slightly to allow it to lie just outside the metaphyseal cortex ([Fig. 10-30](#)).

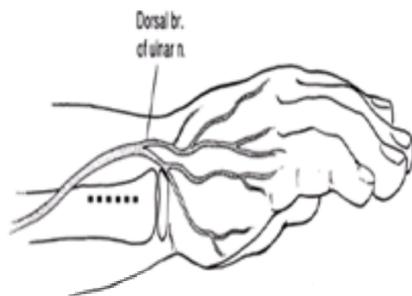


**FIGURE 10-30.** Once the pin enters the proximal fragment, it is passed to the level of the proximal radius. It is bent to 180 degrees and secured just outside the cortex, next to the metaphyseal cortex (*inset*).

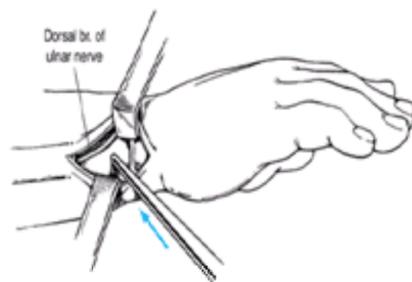
The ulnar rod is inserted through the posterior aspect of the olecranon using a small stab incision ( [Fig. 10-31](#)). The rod can be left percutaneously and removed in the office at 3 to 4 weeks after insertion at which time the ulnar fracture is sufficiently healed to avoid displacement with continued cast immobilization, particularly if the radius has also been stabilized. Verstreken et al. ( [135](#)) recommended inserting the ulnar pin from the distal metaphysis, citing advantages of ease of passage and avoidance of soft tissue problems associated with subcutaneous or percutaneous pin location about the olecranon ( [Fig. 10-32](#), [Fig. 10-33](#), [Fig. 10-34](#) and [Fig. 10-35](#)). However, antegrade insertion of the intramedullary rod through the olecranon takes advantage of the straight anatomy of the ulna, which, in my opinion, facilitates passage of the implant. The arm must be abducted and either internally or externally rotated with the elbow flexed to start the nail and follow its path past the fracture site with the image intensifier.



**FIGURE 10-31. A:** Injury films of a 13-year-old with midshaft fractures of the radius and ulna. **B:** After closed reduction, fixation was achieved with 0.062<sup>2</sup> Kirschner wires. The original Marote technique of inserting the ulnar rod into the olecranon was used in this case.



**FIGURE 10-32.** Location of the skin incision for insertion of the pin in the distal ulna.



**FIGURE 10-33.** The ulnar metaphysis is also drilled cephalad with a 2.5-mm drill bit, retracting the skin and nerves.



**FIGURE 10-34.** In the same manner, the rod is passed proximally up the ulnar shaft to cross the fracture site into the olecranon.



**FIGURE 10-35.** Intramedullary fixation using the distal ulna as the entrance site. Note that the bent ends of the rod are juxtaposed against their respective metaphyses (arrows).

The incisions are then closed. Delayed primary closure at the fracture site is done for open fractures. When closing the distal insertion incisions, care is taken to ensure that the protruding portions of the wire are not impinging on the superficial radial or ulnar nerves.

*Postoperative Management.* The usual postoperative wound care that is necessary for open fractures is followed. I immobilize the forearm with a sugar-tong splint for 3 to 4 weeks, after which I remove the ulnar rod in the office and then use a short arm cast for an additional 3 weeks. At 6 weeks, if there is adequate callus, elbow and forearm motion are initiated. Patients are instructed to avoid strenuous activities for approximately 3 months after injury.

I usually leave the radial pin in place for a total of 6 to 9 months. The criteria for removal are a solid callus across the fracture site and obliteration of the fracture lines. Pin removal does require a second surgical procedure under regional or general anesthesia.

*Plate Fixation.* My primary indication for plate fixation is a refracture in which the medullary canal has a high risk of being obstructed by fracture callus ( Fig. 10-21). This is not to say that I use plates in all refractures. Each fracture is evaluated to determine if an intramedullary device can be passed with ease. In addition, some severely comminuted fractures or those with segmental blood loss may require plate fixation because they may be unstable with intramedullary fixation. This is especially true when rotational alignment is an issue. But with the level of stable fixation achieved with the current intramedullary devices in most fractures, rarely do I find an indication for plate fixation in a skeletally immature patient.



## ASSOCIATED INJURIES AND COMPLICATION

### Refracture

Refracture of the forearm occurs in approximately 5% of patients, despite the appearance of solid union at the original fracture site ( 43,53,96,136). Refracture is more likely to occur after greenstick than complete fractures. Gruber ( 43) postulated that the intact cortex of a greenstick fracture heals by primary bone healing and maintains length as a resorption zone develops on the side of the broken cortex. Refractures also are common after plate removal ( 6,49,60,106,120). Complications after routine plate removal have caused some surgeons to question the value of this practice ( 6,58,74,114).

Refractures with displacement can be difficult to reduce. Poor results are common after closed management ( 4,51). Open reduction and internal fixation are justified unless excellent alignment can be maintained by closed means ( Fig. 10-21).

### Malunion

Despite every precaution, some fractures heal with deformity. If the angulation develops up to 3 to 4 weeks after fracture, often the forearm can be remanipulated into a satisfactory position and placed in a cast for another 4 to 6 weeks ( 140,141) ( Fig. 10-36). After 8 weeks, unless the deformity is severe, it is best to wait 4 to 6 months before considering corrective osteotomy.



**FIGURE 10-36.** Remanipulation. **A:** Radiographic appearance of a 6-year-old 6 weeks after fracture with approximately 20 degrees of angulation at the fracture sites. This was unacceptable functionally and cosmetically. **B:** The fracture was remanipulated under general anesthesia into a satisfactory position, correcting the angulation. **C:** Three months after remanipulation, there was full forearm rotation.

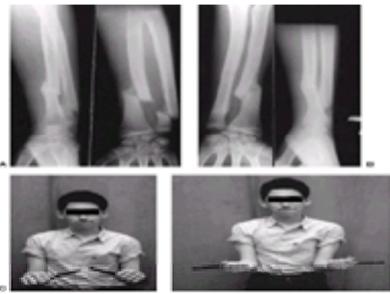
Remodeling is unpredictable and may be surprising. Also, muscular atrophy and joint stiffness after immobilization initially exaggerate the appearance of deformity. Function may return to normal despite malunion ( Fig. 10-37, Fig. 10-38 and Fig. 10-39). Numerous clinicians (24,50,51,84,95) have noted that there is poor correlation between residual deformity and limitation of forearm rotation.



**FIGURE 10-37.** Proximal remodeling potential. **A:** Fracture of the proximal third of the radius with bayonet apposition of the radius and the undisplaced lower third of the ulna. **B:** Position and remodeling at 6 weeks. **C:** Status at 4 months. **D:** At 2.5 years, note almost complete remodeling.



**FIGURE 10-38.** Midshaft remodeling. **A:** A 7-year-old patient with almost 20 degrees of angulation of both the radial and ulnar shafts. There was limitation of forearm motion. **B:** Two years later, there was complete remodeling of the radius and only slight angulation of the ulna. The patient regained essentially 75% normal forearm rotation. The parents and patient were satisfied with the cosmetic and functional outcome.



**FIGURE 10-39.** **A:** A 13-year-old sustained a fracture of the distal radius and ulna that healed with 1 cm of shortening and distal bayonet apposition. **B:** Ten weeks after injury, there is early remodeling. **C:** Six months after injury, the patient had essentially equal forearm rotation and did not appreciate any clinical shortening.

Linscheid and Trousdale (67) reported 19 patients who had corrective osteotomy of the forearm to improve rotation after malunion. Patients treated within 12 months of the initial injury regained an average of 81 degrees of rotation; those treated later gained an average of 42.5 degrees. Drill osteoclasts to correct deformity in children was reported by Blount (9). Blackburn et al. (8) used this method in 15 children with disability and cosmetic deformity from malunited midshaft forearm fractures. After osteotomy, 10 patients were immobilized solely in plaster casts. Results were satisfactory in 10 of 12 patients with adequate follow-up.

## AUTHORS' PREFERRED METHOD OF TREATMENT

When the malunion is recognized at time of cast removal, I prefer to allow 6 to 8 weeks for mobilization of the soft tissues before correction of deformity. An accurate preoperative analysis of the deformity is required. Plain radiographs may suffice, but three-dimensional computed tomographic scanning may aid in planning the proper location, direction, and magnitude of rotational and angular correction. I prefer adequate exposure to perform the osteotomies and fixation with a compression plate. Occasionally, single-plane deformity can be corrected by osteotomy through a limited exposure and stabilized with intramedullary fixation and cast immobilization (Fig. 10-40).



**FIGURE 10-40.** Corrective osteotomy. **A:** This girl, 5 years and 1 month of age, acquired severe angulation 5 weeks after closed reduction. **B:** Because of the abundant callus, osteotomies of the radius and the ulna were performed with intramedullary stabilization of the ulna. **C:** Five years after injury, alignment, range of motion, and function are normal.

## Synostosis

Synostosis is a rare complication of forearm fractures in children. It is more likely to follow high-energy trauma, surgical intervention, repeated manipulations, or fractures associated with head injury (137,138). Vince and Miller (138) reported 10 children with cross-union after forearm fracture. None was in the distal third, four were in the middle third, and six were in the proximal third. Surgical excision was performed in six patients. The synostosis recurred in three patients. The remaining three patients regained 25, 40, and 70 degrees, respectively, of forearm rotation. The authors observed that results after excision of cross-union were not as good in children as in adults (Fig. 10-41).



**FIGURE 10-41. A:** Type II cross-union (middle third). (Courtesy of K. G. Vince, MD.) **B:** Type I cross-union after open reduction and fixation with wires. Note protrusion of wires. **C:** Cross-union fully established at 3 months. No pronation or supination possible. (B and C from Vince KG, Miller JE. Cross-union complicating fracture of the forearm. Part II: children. *J Bone Joint Surg Am* 1987;69:655; with permission.) **D:** Type III cross-union (proximal third). (Courtesy of K. G. Vince, MD) **E:** Cross-union (type I). Initial treatment consisted of open reduction and fixation with Steinmann pin performed within 24 hours. Displacement recurred and unsuccessful closed reduction was performed at 7 days. Thirteen days after injury, open reduction and fixation of the radius and ulna with plate was done through two incisions. The radiograph was taken 11 months after injury, immediately after removal of plate and excision of cross-union. **F:** Refracture of ulna that occurred during excision of cross-union. **G:** Recurrence of cross-union 1 year after excision.

### Compartment Syndrome

There are several reports of compartment syndrome after forearm fractures in children ([47,71,108](#)). Most have resulted from severely displaced distal radial and ulnar fractures.

Casts should be split at the time of reduction if swelling is present or anticipated. As a rule, the child is much more comfortable after reduction and cast stabilization of the fracture, and the development of a compartment syndrome should be considered when severe pain or loss of active or passive finger motion persists after the reduction. Compartment pressure measurements should be obtained if signs and symptoms of compartment syndrome develop. The indications and techniques for forearm fasciotomy are the same in children as in adults.

### Nerve Injury

Injuries to the median, ulnar, and posterior interosseous nerves have been reported. These usually are transitory. Entrapment of the median nerve in a greenstick fracture has been reported ([40,87,150](#)). Geissler et al. ([39](#)) described anterior interosseous nerve palsy complicating a closed fracture of both bones of the forearm in an 8-year-old child. Surgical exploration revealed a bone spike from the fracture of the proximal radius perforating the median nerve. Isolated injuries to the ulnar nerve ([75,151](#)) and to the anterior interosseous branch of the median nerve ([143](#)) also have been reported. Gainor and Olson reported entrapment of both the median and anterior interosseous nerves ([36](#)). Rijnberg and MacNicol reported entrapment of a superficial radial nerve ([103](#)). Moore et al. ([80](#)) reported an 8.5% incidence of iatrogenic injury in fractures that were stabilized surgically.

In almost every reported instance of nerve entrapment after closed treatment, the initial reduction was difficult or incomplete. Surgical exploration was delayed from 5 days to 5 months after injury. Recovery was complete or almost complete in all but one patient after surgical repair.

A thorough prereduction neurologic examination is recommended, although this may be difficult with an anxious, uncooperative patient. Nerve entrapment should be suspected when the prereduction examination was negative, the reduction was incomplete, and the postreduction examination indicated a neurologic deficit. When neurologic deficit is noted after reduction, the appropriate length of observation before surgical exploration is unclear. I recommend 8 weeks when the reduction is anatomic and early exploration if entrapment is suspected.

### Other Complications

Muscle entrapment can prevent reduction of complete fractures and also has been reported with greenstick fractures ([45,62](#)). More serious complications, including hematogenous osteomyelitis ([14,134](#)) after closed treatment and gas gangrene ([32](#)) after open fractures, also have been reported. Nonunion is rare and usually heals with appropriate management.

## PLASTIC DEFORMATION OF THE RADIUS AND ULNA

Plastic deformation, or bending of bone, was described by Barton in 1821. This reference is quoted by Sir Astley Cooper ([19](#)):

Dr. Barton recognizes two varieties of it which, however, are scarcely more than different degrees of the same injury; first, a simple bending of the bone, and the second, what he regards as truly a partial or incomplete fracture.

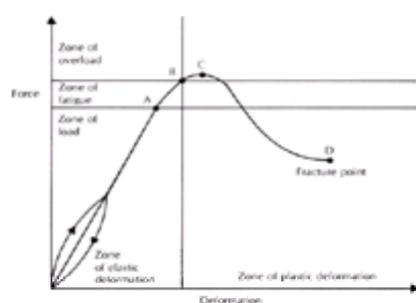
In 1975, Borden ([12](#)) reported traumatic bowing of the bones of the forearm in children. Since then, other reports emphasized the difficulty in diagnosis and treatment of these fractures ([22,26,63,73,77,85,110,112,125](#)).

### Surgical Anatomy

A child's bone is more flexible than an adult's because the haversian canals are larger and thus render the bone more porous. This allows a greater degree of flexibility—and hence more plastic deformation—to occur before fracture.

### Mechanism of Injury

Chamay ([16](#)) showed that low-level stresses applied to bone cause bending proportional to the stress applied, but the bone retains its normal configuration when stress is removed. This results in elastic deformation ([Fig. 10-42](#)).



**FIGURE 10-42.** Graphic relation of bony deformation (bowing) and force (longitudinal compression) showing that the limit of an elastic response is not a fracture but plastic deformation. If the force continues, a fracture results. (Redrawn from Borden S IV. Roentgen recognition of acute plastic bowing of the forearm in children. *AJR Am J Roentgenol* 1975;125:524; with permission.)

Stress that exceeds the elastic limit but is less than the fracture limit causes fixed bending that does not change when the stress is removed. This fixed bending is not associated with gross fracture and is called *plastic deformation*. Plastic bowing has been reported in young adults ([101,131](#)), but is more common in children because of the larger plateau of plastic response in children than in adults.

Longitudinal compression forces applied to a naturally curved bone have been shown to cause plastic deformation ([16,129](#)), and lateral bending or tangential forces caused by compression around a cylindrical object have been shown to produce traumatic bowing ([7,101,131](#)). Tensile stress is produced at the convex surface and

compression stress occurs at the concave surface. Bending occurs through oblique slip lines or microfractures along the concave border of the bone. These slip lines extend from the periosteum to the medullary canal at an angle of approximately 30 degrees to the long axis to the bone ( 16).

### Classification

Plastic deformation can occur in the radius, the ulna, or both. Other patterns show either the radius or ulna deformed in combination with a fracture of the diaphysis of the other bone.

Demos (26) reviewed 74 cases of plastic deformation and found that 58 (78%) occurred in the forearm: 26 (35%) in the ulna, 15 (20%) in the radius, 11 (15%) in both bones, and 6 (8%) unspecified.

### Signs and Symptoms

Plastic deformation appears clinically with deformity, tenderness, pain, and, if both bones are involved, a decrease in pronation and supination ( Fig. 10-43).



**FIGURE 10-43.** **A:** A 7-year-old boy with plastic deformation of the radius and ulna consisting only of a gradual curvature of the forearm. **B:** The curvature is more apparent when compared with the normal right forearm. **C:** Because of the plastic deformation, there is limitation of supination and pronation.

### Radiographic Findings

Characteristically, there is a broad fixed curvature of the entire bone ( Fig. 10-44). In doubtful cases, a radiograph of the unaffected forearm in the same position reveals subtle deformation. A bone scan can differentiate a fresh injury from an old injury when physical signs are inconsistent ( 77). The elbow and wrist must be included in the radiograph to rule out disruption at these levels.



**FIGURE 10-44.** **A:** A 4-year-old sustained acute injury to the left forearm. Both the radius and ulna demonstrate plastic deformation. The patient had supination only to neutral. **B:** Unaffected right forearm for comparison. **C:** After reduction, most of the angulation has been corrected, but there is still a slight residual. Full forearm rotation has been reestablished.

The most common pattern is plastic deformation of the ulna with a greenstick fracture of the radius ( Fig. 10-45). The plastic deformation of the ulna must be corrected before the radius can be fully reduced.



**FIGURE 10-45.** Plastic deformation of ulna. In this 3-year-old, there is an obvious greenstick fracture of the radius. However, to achieve a full reduction, the plastic deformation of the ulna must first be corrected.

In 4 to 6 weeks, gradual bony remodeling manifests itself by cortical thickening in the concavity of the deformity. However, there may be no periosteal callus even after several months (125).

### Treatment

#### Early Recognition Critical

Early recognition of plastic deformation is important. The deformity may hinder reduction of a true fracture in the adjacent bone of the forearm. If not recognized, the residual deformity may cause limitation of pronation and supination. Rydholm and Nilsson ( 110), along with Demos (26), believed that some remodeling can occur in patients younger than 10 years of age even without correction of the bowing. Plastic deformation in a child younger than 4 years of age probably does not need correction because the bone will remodel sufficiently to give full function ( 112). Parents should be cautioned that this remodeling will take 6 months to 1 year. Older

children may have residual loss of pronation and supination if the bowing is not reduced.

### Considerable but Gradual Force Needed

If the curvature is significant, correction should be attempted in the acute phase (63,73) under general anesthesia. Correction may be difficult, and considerable force is required. Borden (12), in his experiments with dogs, showed that a force of 100% to 150% of body weight was required to produce plastic deformation and concluded that a similar force was needed to effect reduction. This amount of force cannot be obtained by longitudinal traction, but must be effected by placing the apex of the curve over a fulcrum and applying the force at right angles to the deformity. Sanders and Heckman (112) have effectively used this method to correct the deformity by the use of a wooden cylinder wrapped in a towel (Fig. 10-46). They caution that a great deal of pressure must be applied gradually over several minutes to straighten the curvature slowly and that care should be taken not to apply this pressure over the physes.



**FIGURE 10-46.** The deformity is gradually corrected by twisting the deformity with the apex in a roll, wrapped in a towel (white arrow). The correcting force is applied proximally and distally (black arrows) on the concave side of the deformity.

If both bones are deformed, the one with the greatest deformity is reduced first (Fig. 10-44). If one bone is deformed and the other fractured, the plastically deformed bone must be reduced before the fracture (Fig. 10-45).

Immobilization in a long arm cast is necessary for 6 weeks to achieve adequate healing and to avoid recurrence of the deformity.

Blount (9) proposed drilling the apex of the deformed bone through a small incision to fracture the bone and effect the reduction. This seems no longer necessary in view of the effective means of reduction described by Sanders and Heckman (112).

### AUTHORS' PREFERRED METHOD OF TREATMENT

The gradual and sustained reduction method described by Sanders and Heckman (112) usually is effective (Fig. 10-46). The bone most deformed must be reduced first to achieve a reduction of the other, whether deformed or fractured. If the forearm cannot be fully supinated and pronated after manipulation, reduction has not been successful. It is advisable to caution the parents that it may be necessary to fracture one or both bones to effect a reduction.

A long arm cast is applied with the elbow flexed 90 degrees and the forearm in full supination. This cast is worn for 6 to 8 weeks, depending on the child's age.

### Prognosis

Full reduction may not be achieved, but the clinician should strive for 85% correction (Fig. 10-44C). There should be no recurrence of the deformity, but a check radiograph should be made at weekly intervals for the first 2 or 3 weeks. Healing should be expected in 6 to 8 weeks.

### Complications

With bowing of the proximal ulna, it may be difficult to apply adequate pressure to effect a reduction and the deformity may persist, but it usually is not sufficient to produce significant loss of pronation and supination. The major complication is failure to recognize or correct the deformity fully, with residual angulation and loss of motion. A more significant problem may be caused by failure to recognize subluxation of the radial head, which typically requires anatomic reduction of the ulnar deformity (Fig. 10-47).



**FIGURE 10-47.** Persistent deformation. **A:** Fracture of the distal third of both the radius and ulna. The ulna has plastic deformation. **B:** The fracture healed uneventfully; however, the plastic deformation of the ulna had not been corrected, which led to some residual bowing with loss of forearm rotation. The persistent deformation of the ulna influenced the development of an angular deformity of the radius as it healed.

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### CHAPTER REFERENCES

1. Alpar EK, Thompson K, Owen R, et al. Midshaft fractures of forearm bones in children. *Injury* 1981;13:153.
2. American Academy of Pediatrics Committee on Drugs. Guideline for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures. *Pediatrics* 1992;89:1110.
3. Amit Y, Salai M, Checkik A, et al. Closed intramedullary nailing for the treatment of diaphyseal forearm fractures in adolescence: a preliminary report. *J Pediatr Orthop* 1985;5:143.
4. Arunachalam VSP, GJC. Fracture recurrence in children. *Injury* 1975;7:37.
5. Barnes CL, BRD, Dodge DM. Intravenous regional anesthesia: a safe and cost-effective outpatient anesthetic for upper extremity fracture treatment in children. *J Pediatr Orthop* 1991;11:717.
6. Bedner DA, Grandwilewski W. Complications of forearm plate removal. *Can J Surg* 1992;35:428.
7. Benz G, Kallieris D, Daum R, et al. Does lateral bending lead to bowing fracture in an infantile lower arm? *Eur J Pediatr Surg* 1992;2:177.
8. Blackburn N, Ziv I, Rang M. Correction of malunited forearm fracture. *Clin Orthop* 1984;188:54.

9. Blount WP. Osteoclasia of the upper extremity in children. *Acta Orthop Scand* 1962;32:374.
10. Blount WP. Forearm fractures in children. *Clin Orthop* 1967;51:93.
11. Blount WP, SAA, Johnson JH. Fractures of the forearm in children. *JAMA* 1942;120:111.
12. Borden IVS. Roentgen recognition of acute plastic bowing of the forearm in children. *AJR Am J Roentgenol* 1975;125:524.
13. Boriani S, Lefevre C, Malingue E, et al. The Lefevre ulnar nail. *Chir Organi Mov* 1991;76:151.
14. Canale ST, Puhl J, Watson FM, et al. Acute osteomyelitis following closed fractures: report of three cases. *J Bone Joint Surg Br* 1975;57:415.
15. Carey PJ, Alburger PD, Betz RR, et al. Both-bone forearm fractures in children. *Orthopaedics* 1992;15:1015.
16. Chamay A. Mechanical and morphological aspects of experimental overload and fatigue in bone. *J Biomech* 1970;3:263.
17. Chapman KW, FVH. Rotation deformity of both bones following forearm fractures. *J Bone Joint Surg Br* 1973;55:666.
18. Clarke AC, SRF. Operative fixation of fractures in children. *S Afr Med J* 1991;79:206.
19. Cooper A. *Dislocations and fractures of the joints*. Boston: TR Marvin 1844;391–400.
20. Cramer KE, Glasson S, Mencio GA, et al. Reduction of forearm fractures in children using axillary block anesthesia. *J Orthop Trauma* 1995;9:407.
21. Creaseman C, Zaleske DJ, Ehrlich MG. Analyzing forearm fractures in children: the more subtle signs of impending problems. *Clin Orthop* 1984;188:40.
22. Crowe JE, Swischuk, LE. Acute bowing fractures of the forearm in children: a frequently missed injury. *AJR Am J Roentgenol* 1977;128:981.
23. Cullen MC, RDR, Giza E, et al. Complications of intramedullary fixation of pediatric forearm fractures. *J Pediatr Orthop* 1998;18:14.
24. Daruwalla JS. A study of radioulnar movements following fractures of the forearm in children. *Clin Orthop* 1979;139:114.
25. Davis DR, Green DP. Forearm fractures in children: pitfalls and complications. *Clin Orthop* 1976;120:172.
26. Demos T. Radiologic case study. *Orthopaedics* 1980;3:1108.
27. DePablos J, Franzreb M, Barrios C. Longitudinal growth pattern of the radius after forearm fractures conservatively treated in children. *J Pediatr Orthop* 1994;14:492.
28. Dickson JA. Intramedullary fixation of certain fractures of both bones of forearm: report of two cases. *Cleve Clin Q* 1944;11:62.
29. Evans EM. Rotational deformity in the treatment of fractures of both bones of the forearm. *J Bone Joint Surg* 1945;27:373.
30. Evans EM. Fractures of the radius and ulna. *J Bone Joint Surg Br* 1951;33:548.
31. Evans JK, Buckley SL, Alexander AH, et al. Analgesia for the reduction of fractures in children in children: a comparison of nitrous oxide with intramuscular sedation. *J Pediatr Orthop* 1995;15:73.
32. Fee NF, Dobranski A, Bisla RS. Gas gangrene complicating open forearm fractures: report of five cases. *J Bone Joint Surg Am* 1977;59:135.
33. Fernandez DL. *Conservative treatment of forearm fractures in children*. New York: Thieme-Stratton, 1981.
34. Flynn JM, Waters PM. Single-bone fixation of both-bone forearm fractures. *J Pediatr Orthop* 1996;16:655.
35. Fuller DJ, McCullough CJ. Malunited fractures of the forearm in children. *J Bone Joint Surg Br* 1982;64:364.
36. Gainor BJ, Olson S. Combined entrapment of the median and anterior interosseous nerves in a pediatric both bone forearm fracture: a case report. *J Orthop Trauma* 1990;4:197.
37. Gainor JW, Hardy IJ. Forearm fractures treated in extension. *J Trauma* 1969;9:167.
38. Gandhi RK, WP, Brown JM, et al. Spontaneous correction of deformity following fractures of the forearm in children. *Br J Surg* 1962;50:5.
39. Geissler WB, Fernandez DL, Grace R. Anterior interosseous nerve palsy complicating a forearm fracture in a child. *J Hand Surg [Am]* 1990;15:44.
40. Genelín F, Karlbauer AF, Gasperschitz F. Greenstick fracture of the forearm with median nerve entrapment. *J Emerg Med* 1988 Sep-Oct; 6:381–385.
41. Glatzner RL, PRD, Michaels G, et al. Fractures of both bones of the distal forearm in children. *Bull Hosp Joint Dis* 1967;28:14.
42. Grant, Weiss. Forearm fractures in children: a retrospective study. Meeting highlights. *J Pediatr Orthop* 1986;6:506.
43. Gruber R. The problem of the relapse fracture of the forearm in children. In: Chapchal G, ed. *Fractures in children*. Stuttgart: Georg Thieme Verlag, 1981.
44. Haasbeek JF, Cole WG. Open fractures of the arm in children. *J Bone Joint Surg Br* 1995;77:576.
45. Hendel D, Aner A. Entrapment of the flexor digitorum profundus of the ring finger at the site of an ulnar fracture: a case report. *Ital J Orthop Traumatol* 1992;18:417.
46. Hennrikus WL, Shin AY, Klingelberger CE. Self-administered nitrous oxide and a hematoma block for analgesia in the outpatient reduction of fractures in children. *J Bone Joint Surg Am* 1995;77:335.
47. Hernandez J, Peterson HA. Fracture of the distal radial physis complicated by compartment syndrome and premature physeal closure. *J Pediatr Orthop* 1986;6:626.
48. Hey Groves EW. *On modern methods of treating fractures*. Bristol: John Wright & Sons, 1916.
49. Hidaka S, Gustilo RB. Refracture of bones of the forearm after plate removal. *J Bone Joint Surg Am* 1984;66:1241.
50. Hogstrom H, Nilsson BE, Willner S. Correction with growth following diaphyseal forearm fracture. *Acta Chir Scand* 1976;47:229.
51. Holdsworth BJ, Sloan JP. Proximal forearm fractures in children: *Resid Disabil Injury* 1983;14:174.
52. Huber RI, Keller HW, Huber PM, et al. Flexible intramedullary nailing as fracture treatment in children. *J Pediatr Orthop* 1996;16:602.
53. Hughston JC. Fractures of the forearm in children. *J Bone Joint Surg Am* 1962;44:1667.
54. Johari AN, OD, Sinha M. Remodeling of forearm fractures in children. *J Pediatr Orthop B* 1999;8:84.
55. Johnson PQ, Noffsinger MA. Hematoma block of distal forearm fractures: is it safe? *Orthop Rev* 1991;20:977.
56. Jones K, Weiner DS. The management of forearm fractures in children: a plea for conservatism. *J Pediatr Orthop* 1999;19:811.
57. Juliano PJ, Mazur JM, Cummings RJ, et al. Low-dose lidocaine intravenous regional anesthesia for forearm fractures in children. *J Pediatr Orthop* 1992;12:633.
58. Kahle WK. The case against routine metal removal. *J Pediatr Orthop* 1994;14:229.
59. Kay S, SC, Oppenheim WL. Both-bone midshaft forearm fractures in children. *J Pediatr Orthop* 1986;6:306.
60. Kessler SB, DS, Schiffli-Dieler M, et al. Refractures: a consequence of impaired local bone viability. *Arch Orthop Trauma Surg* 1992;111:96.
61. Knight RA, PGD. Fractures of both bones of the forearm in adults. *J Bone Joint Surg Am* 1949;31:755.
62. Kolkman KA, van Niekerk JLM, Rieu PNMA, et al. A complicated forearm greenstick fracture: a case report. *J Trauma* 1992;32:116.
63. Komara JS, KL, Kottamasu SR. Acute plastic bowing fractures in children. *Ann Emerg Med* 1986;15:585.
64. Landin LA. Fracture patterns in children. *Acta Paediatr Scand Suppl* 1983;54:192.
65. Landin LA. Fracture patterns in children: analysis of 8628 fractures with special reference to incidence, etiology, and secular changes in a Swedish urban population, 1950–1979. *Acta Chir Scand Suppl* 1983;202:1-109.
66. Lascombes P, PJ, Ligier JN, et al. Elastic stable intramedullary nailing in forearm shaft fractures in children: 85 cases. *J Pediatr Orthop* 1990;10:161.
67. Linsheid RL, Trousdale RT. Surgical treatment of forearm malunions. *Orthop Trans* 1993;17:82.
68. London PS. *A practical guide to the care of the injured*. Edinburgh: E & S Livingstone, 1967.
69. Luhman SJ, Gordon JE, Schoenecker PL. Intramedullary fixation of unstable both bone forearm fractures in children. *J Pediatr Orthop* 1998;18:451.
70. Marek FM. Axial fixation of forearm fractures. *J Bone Joint Surg Am* 1961;43:1099.
71. Matsen FA III, Veith RG. Compartmental syndromes in children. *J Pediatr Orthop* 1981;1:33.
72. McCarty EM, Mencio GA, Green NE. Anesthesia and analgesia for management of fractures in children. *J Am Acad Orthop Surg* 1999 Mar-Apr; 7(2):81-91. Review.
73. Medich G, SCL. Acute plastic forearm deformation in children. *Ortho Consult* 1986;7–12.
74. Mih AD, Cooney WP, Idler RS, et al. Long term follow-up of forearm bone diaphyseal plating. *Clin Orthop* 1994;299:256.
75. Mikic Z. Galeazzi fracture-dislocations. *J Bone Joint Surg Am* 1975;57:1071.
76. Milch H. Roentgenographic diagnosis of torsional deformities in tubular bones. *Surgery* 1944;15:440.
77. Miller JH, OJA. Scintigraphy in acute plastic bowing of the arm. *Radiology* 1982;142:742.
78. Moed BR, Kellam JF, Foster RI, et al. Immediate internal fixation of open fractures of the diaphysis of the forearm. *J Bone Joint Surg Am* 1986;68:1008.
79. Moesner J, Ostergaard AH. Diafysefrakturer hos born. *Nordisk Medicin* 1966;31 III bd 75:355–357.
80. Moore TM, KJP, Patzakis MJ, et al. Results of compression plating of closed Galeazzi fractures. *J Bone Joint Surg Am* 1985;67:1015.
81. Muller ME, AM, Willenegger H. *Manual of internal fixation*, 2nd ed. Berlin: Springer-Verlag, 1979.
82. Naimark A, Kossoff J, Leach RE. The disparate diameter: a sign of rotational deformity in fractures. *J Assoc Can Radio* 1983;34:8.
83. Nielson AB, Simonsen O. Displaced forearm fractures in children treated with AO plates. *Injury* 1984;15:393.
84. Nilsson BE, Obrant K. The range of motion following fracture of the shaft of the forearm in children. *Acta Chir Scand* 1977;48:600.
85. Nimityongskul P, Anderson LD, Prasit Sri. Plastic deformation of the forearm: a review and case reports. *J Trauma* 1991 Dec;31:1678–1685.
86. Noonan KJ, Price CT. Forearm and distal radius fractures in children. *J Am Acad Orthop Surg* 1998;6(3):146.
87. Nunley JA, Urbaniak JR. Partial bony entrapment of the median nerve in a greenstick fracture of the ulna. *J Hand Surg* 1980;5:557.
88. Olney BW, Lugg PC, Turner PL, et al. Outpatient treatment of upper extremity injuries in children using intravenous regional anaesthesia. *J Pediatr Orthop* 1988;8:576.
89. Onne L, Sandblom PH. Late results in fractures of the forearm in children. *Acta Chir Scand* 1949;98:549.
90. Ono M, Bechtold JE, Merkoco MD, et al. Rotational stability of diaphyseal fractures of the radius and ulna fixed with Rush pins and /or fracture bracing. *Clin Orthop* 240:236.
91. Ortega R, Loder RT, Louis DS. Open reduction and internal fixation of forearm fractures in children. *J Pediatr Orthop* 1996;16:651.
92. Orthopaedic Trauma Association. Fracture and dislocation compendium. *J Orthop Trauma* 1996;10[Suppl 1]:v.
93. Parsch K. The Marote wiring in proximal and midshaft fractures of the child. *Oper Orthop Trauma* 1990;2:245.
94. Perez-Sicilia JE, et al. Osteosintesis percutanea en fracturas diafisarias de antebrazo en niños y adolescentes. *Rev Esp Cir Ost* 1977;12:321.
95. Price CT. Fractures of the midshaft radius and ulna. In: Letts RM, ed. *Management of pediatric fractures*. New York: Churchill Livingstone, 1994;323.
96. Price CT, Scott DS, Kurzner ME, et al. Malunited forearm fractures in children. *J Pediatr Orthop* 1990;10:705.
97. Putnam JD, Walsh TM. External fixation for open fractures of the upper extremity. *Hand Clin* 1993;4:613.
98. Rang M. *Children's fractures*. Philadelphia: JB Lippincott, 1983.
99. Reed FE Jr, Apple DF. Ipsilateral fractures of the elbow and forearm. *South Med J* 1976;69:149.

100. Reed MH. Fractures and dislocations of the extremities in children. *J Trauma* 1977;17:351.
101. Reisch RB. Traumatic plastic bowing deformity of the radius and ulna in a skeletally mature adult. *J Orthop Trauma* 1994;8:258.
102. Richter D, Ostermann PA, Ekkernkamp A, et al. Elastic intramedullary nailing: a minimally invasive concept in the treatment of unstable forearm fractures in children. *J Pediatr Orthop* 1998;18:457.
103. Rijnberg WJ, MacNicol MF. Superficial radial nerve entrapment within a radial fracture in a child. *Int J Care Injurec* 1993;24:426.
104. Roberts JA. Angulation of the radius in children's fractures. *J Bone Joint Surg Br* 1986;68:751.
105. Rockwood CA Jr, Green DP, eds. *Fractures*, 4th ed. Philadelphia: JB Lippincott, 1995.
106. Rosson JW, Shearer JR. Refracture after removal of plates from the forearm. *J Bone Joint Surg Br* 1991;73:415.
107. Roy DR, Crawford AH. Operative management of fractures of the shaft of the radius and ulna. *Orthop Clin North Am* 1990;21:245.
108. Royle SG. Compartment syndrome following forearm fracture in children. *Injury* 1990;21:73.
109. Rush LV. *Atlas of Rush pins techniques*, 2nd ed. Meridian, MS: Berivon, 1976.
110. Rydholm V, Nilsson JE. Traumatic bowing of the forearm. *Clin Orthop* 1979;139:121.
111. Sage FP. Medullary fixation of fractures of the forearm. *J Bone Joint Surg Am* 1959;41:1489.
112. Sanders WE, Heckman JD. Traumatic plastic deformation of the radius and ulna: a closed method of correction of deformity. *Clin Orthop* 1984;188:58.
113. Sarmiento A, Ebrahimzadeh E, Brys D, et al. Angular deformities and forearm function. *J Pediatr Orthop* 1992;10:121.
114. Schmalzried TP, Gorgan TJ, Neumeier PA, et al. Metal removal in a pediatric population: benign procedure or necessary evil? *J Pediatr Orthop* 1991;11:72.
115. Schone G. Behandlung von Vorderarfrakturen mit Bolzung. *Munch Med Wochenschr* 1913;60:2327.
116. Shoemaker SD, Comstock CP, Mubarak SJ, et al. Intramedullary Kirschner wire fixation of open or unstable forearm fractures in children. *J Pediatr Orthop* 1999;19:329.
117. Smith H, SFP. Medullary fixation of forearm fractures. *J Bone Joint Surg Am* 1957;39:91.
118. Spiegel P, Mast JW. Internal and external fixation of fractures in children. *Orthop Clin North Am* 1980;11:405.
119. Stanitski CL, Micheli LJ. Simultaneous ipsilateral fractures of the arm and forearm in children. *Clin Orthop* 1980;153:218.
120. Stern PJ, Drury WJ. Complications of plate fixation of forearm fractures. *Clin Orthop* 1983;175:25.
121. Street DM. Intramedullary forearm nailing. *Clin Orthop* 1986;212:219.
122. Tarr RR, Garfinkel AI, Sarmiento A. The effects of angular and rotational deformities of both bones of the forearm. *J Bone Joint Surg Am* 1984;66:65.
123. Thomas EMT, KWR, Browne PSH. Fractures of the radius and ulna in children. *Injury* 1975;7:120.
124. Thompson GH, WJH, Marcus RE. Internal fixation of fractures in children and adolescents. *Clin Orthop* 1984;188:10.
125. Thomson JL. Acute plastic bowing of bone. *J Bone Joint Surg Br* 1982;64:123.
126. Thorndike A Jr, Dimmler JCL. Fractures of the forearm and elbow in children: an analysis of 364 consecutive cases. *N Engl J Med* 1941;225:475.
127. Tolat AR, Sanderson PL, De Smet L, et al. The gymnast's wrist: acquired positive ulnar variance following chronic epiphyseal injury. *J Bone Joint Surg Br* 1992;17:678.
128. Tredwell SJ, Van Peteghem K, Clough M. Pattern of forearm fractures in children. *J Pediatr Orthop* 1984;4:604.
129. Tschantz P, RE. La surchsrsg mecanique de l'os vivant. *Ann Anat Pathol* 1967;12:223.
130. Vainionpaa S, BO, Patiala H, et al. Internal fixation of forearm fractures in children. *Acta Orthop Scand* 1987;58:121.
131. van den Wildenberg FAJM, Greve JW. Intramedullary stabilization of a bowing fracture of the forearm with Ender's nails: case report. *J Trauma* 1993;35:808.
132. Van der Reis WL, Otsuka NY, Moroz P, et al. Intramedullary nailing versus plate fixation for unstable forearm fractures in children. *J Pediatr Orthop* 1998;18:9.
133. Varela CD, Lorfing KC, Schmidt TL. Intravenous sedation for the closed reduction of fractures in children. *J Bone Joint Surg Am* 1995;77:340.
134. Veranis N, Laliotis N, Vlachos E. Acute osteomyelitis complicating a closed radial fracture in a child. *Acta Orthop Scand* 1992;63:341.
135. Verstrecken L, Delonge G, Lamoureux J. Shaft forearm fractures in children: intramedullary nailing with immediate motion. A preliminary report. *J Pediatr Orthop* 1988;8:450.
136. Victor J, Fabry G. Refracture of the radius and ulna in a female gymnast: a case report. *Am J Sports Med* 1993;21:753.
137. Vince KG, Miller JE. Cross-union complicating fracture of the forearm. *J Bone Joint Surg Am* 1987;69:640.
138. Vince KG, Miller JE. Cross-union complicating fracture of the forearm. Part II: children. *J Bone Joint Surg Am* 1987;69:654.
139. Vittas D, Larsen E, Torp-Pedersen S. Angular remodeling of midshaft forearm fractures in children. *Clin Orthop* 1991;265:261.
140. Voto SJ, Weiner DS, Leighley B. Redisplacement after closed reduction of forearm fractures in children. *J Pediatr Orthop* 1990;10:79.
141. Voto SJ, Weiner DS, Leighley B. Use of pins and plaster in the treatment of unstable pediatric forearm fractures. *J Pediatr Res* 1990;10:85.
142. Walker JL, Rang M. Forearm fractures in children: cast treatment with elbow extension. *J Bone Joint Surg Br* 1991;73:299.
143. Warren JD. Anterior interosseous nerve palsy as a complication of forearm fractures. *J Bone Joint Surg* 1963;45:511.
144. Watson-Jones R. *Fractures and joint injuries*, vol 2, 4th ed. Edinburgh: ES Livingstone, 1955.
145. Wattenmaker I, Kasser JR, McGravey A. Self-administered nitrous oxide for fracture reduction in children in an emergency room setting. *J Orthop Trauma* 1990;4:35.
146. Weber BG, Brunner C, Freuler F. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980.
147. Wedel DJ, Krohn JS, Hall JA. Brachial plexus anesthesia in pediatric patients. *Mayo Clin Proc* 1991;66:583.
148. Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994.
149. Williamson DM, Cole WG. Treatment of ipsilateral supracondylar and forearm fractures in children. *Injury* 1992;23:159.
150. Wolfe JS, Eyring EJ. Median-nerve entrapment within a greenstick fracture: a case report. *J Bone Joint Surg Am* 1974;56:1270.
151. Wong PC. Galeazzi fracture dislocations in Singapore 1960–1964: incidences and results of treatment. *Singapore Med J* 1967;8:186.
152. Wyrsh B, Mencio GA, Green NE. Open reduction and internal fixation of pediatric forearm fractures. *J Pediatr Orthop* 1996;16:644.
153. Younger AS, Tredwell SJ, MacKenzie WG, et al. Accurate prediction of outcome after pediatric forearm fracture. *J Pediatr Orthop* 1994;14:200.
154. Yung SH, Lam CY, Choi KY, et al. Percutaneous intramedullary Kirschner wiring for displaced forearm fractures in children. *J Bone Joint Surg Br* 1998;80:91.

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Fractures of the proximal radius in skeletally immature patients usually involve the metaphysis or physis. True isolated radial head fractures are rare. In the proximal ulna, the olecranon, which biomechanically is a metaphysis, often fails with a greenstick pattern. Fractures in this area also may involve the physis. Fractures of the olecranon associated with proximal radioulnar joint disruption are considered part of the Monteggia fracture–dislocation complex and are discussed in [Chapter 12](#).

## FRACTURES OF THE PROXIMAL RADIUS

### Incidence (Overall Percentage)

Fractures of the radial neck account for slightly more than 1% of all children's fractures ([47](#)). In skeletally immature children, the radial head or epiphysis is rarely fractured, probably because of the large amount of cartilage in the radial head. If the fracture involves the epiphysis, it usually is part of a Salter-Harris type IV fracture pattern. In 90% of proximal radial fractures, the fracture line involves either the physis or the neck ([39](#)). In six large series of elbow fractures, the incidence of fracture of the radial neck was remarkably consistent, varying only from 5% to 8.5% ([10,27,39,41,51,68](#)). Fractures of the radial neck and head in skeletally immature patients account for only 14% to 20% of the total injuries of the proximal radius occurring in all age groups ([31,41](#)).

In most series, the age of occurrence varies from 4 to 14 years of age, with the median age ranging from 9 to 10 years ([16,39,48,67,84,98,106,110](#)). There is very little difference in the occurrence rates between boys and girls ([16,39,67](#)). On the average, however, this injury seems to occur in girls approximately 2 years earlier than in boys ([98](#)).

### Anatomy

#### Ossification Process

In the embryonic development of the proximal radius, the neck and head become well defined by 9 weeks of gestation. At 4 years of age, the radial head and neck have assumed the same overall shape as in an adult ([68](#)). Ossification of the epiphysis begins at approximately 5 years of age as a small, flat nucleus ([Fig. 11-1](#)). Some variation in ossification pattern can occur. This ossific nucleus can originate as a small sphere or it can be bipartite, which may be misinterpreted as a fracture ([13,59,94](#)).



**FIGURE 11-1.** Ossification pattern. **A:** At 5 years, ossification begins as a small oval nucleus. **B:** As the head matures, the center widens but remains flat.

#### Angulation Normal

In the preossification stage, on the anteroposterior (AP) projection radiograph, the edge of the metaphysis of the proximal radius slopes distally on its lateral border, and may give the false impression that the radial neck is angulated.

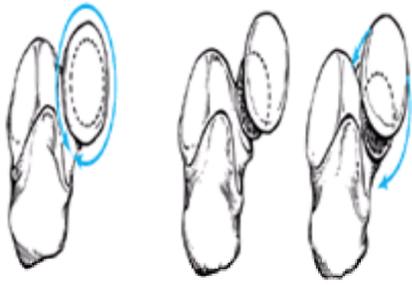
Some angulation of the radial head is normal compared with the radial shaft. In the AP view, the lateral angulation varies from 0 to 15 degrees, with the average being 12.5 degrees ([110](#)). In the lateral view, the angulation can vary from 10 degrees anterior to 5 degrees posterior, with the average being 3.5 degrees anterior ([110](#)).

#### Soft Tissue Attachments

No ligaments attach directly to the radial neck or head. The radial collateral ligaments attach to the orbicular ligament, which originates from the radial side of the ulna. The articular capsule takes its origin from the proximal third of the neck. From there, the capsule protrudes distally from under the orbicular ligament to form a pouch (recessus sacciformis). Thus, only a small portion of the neck lies within the articular capsule ([113](#)). Because much of the neck is extracapsular, fractures involving only the neck may not produce an intraarticular effusion. Therefore, the fat pad sign may be negative with some of these injuries ([13,94,40](#)).

#### The “Cam” Effect

The proximal radioulnar joint has an exacting congruence. The axis of rotation lies directly in the center of the radial neck. Any deviation of the center of the radial head from its alignment with the center of the radius of the neck changes the arc of rotation of the head. If the head is displaced on the neck, instead of rotating smoothly in a pure circle, the radial head rotates with a “cam” effect. This disrupts the congruity of the proximal radioulnar joint, with subsequent loss of supination and pronation ([115](#)) ([Fig. 11-2](#)).



**FIGURE 11-2. A:** Normal rotation of the forearm causes the radial head to circumscribe an exact circle within the proximal radioulnar joint. **B:** Any translocation of the radial head limits rotation because of the “cam” effect described by Wedge and Robertson ([115](#))

## Diagnosis

### Clinical Findings

The symptoms of proximal radial fractures vary with the magnitude of the injury. Because of minimal initial symptoms, the patient or patient's parents may not seek medical treatment until a few days after the initial injury. Much of the swelling and pain is caused by the distention of the elbow joint with blood, and a few hours may be required to develop enough pressure in the joint to cause significant symptoms.

Often the pain is localized over the radial head or neck. The pain usually is increased more with passive forearm supination and pronation than with elbow flexion and extension. In a young child, the primary complaint may be wrist pain ([2](#)), and pressure over the proximal radius may accentuate this referred wrist pain. The wrist pain may be secondary to radial shortening and subsequent distal radioulnar joint dysfunction.

### Radiographic Evaluation

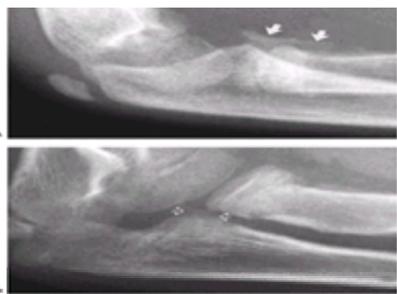
#### Supination–Pronation Views

On radiography, the fracture is usually easily seen on both AP and lateral views. Occasionally, the forearm may need to be both supinated and pronated to reveal the fracture line fully.

#### Perpendicular Views

If the elbow cannot be extended because of pain, two views and oblique views may be necessary to see the elbow in full AP profile. One view is taken with the beam perpendicular to the distal humerus, and the other with the beam perpendicular to the proximal radius. This allows the physal line of the radius to be seen in full profile.

In a minimally displaced or undisplaced fracture, the fracture line may be difficult to visualize because it is superimposed on the proximal ulna. In these cases, oblique views of the proximal radius may be helpful in bringing the fracture line into view ([13,113](#)). One oblique view that is especially helpful is the radiocapitellar view suggested by Greenspan et al. and Hall-Craggs et al. ([36,37](#) and [38](#)). This projects the radial head anterior to the coronoid process ([Fig. 11-3](#)). It is especially helpful if full supination and pronation views are difficult to obtain because of acute injury.



**FIGURE 11-3. The radiocapitellar view. A:** Radiographs of a 13-year-old girl who had sustained a radial neck fracture associated with an elbow dislocation. There is ectopic bone formation (*arrows*). In this view, it is difficult to tell the exact location of the ectopic bone. **B:** The radiocapitellar view separates the radial head from the coronoid process. This demonstrates that the ectopic bone is from the coronoid process (*arrows*) and not the radial neck.

If the epiphysis is ossified, the displacement of the head is usually obvious, but displacement may be difficult to determine before ossification ([85](#)). The key to diagnosis is a thorough clinical examination, including careful palpation of the radial neck. On radiography, the smoothness of the metaphyseal margin may be lost. Ultrasonography can be used to evaluate for hemarthrosis and displacement of the fracture. It also allows a dynamic range-of-motion evaluation ([49](#)).

The supinator fat pad is a small layer of fat that overlies the supinator muscle in the proximal forearm. In soft tissue views of the proximal forearm or elbow, this layer of fat is often well defined. Displacement of the supinator fat pad may indicate that the injury is localized to the proximal radius ([88](#)). The supinator fat pad and distal humeral fat pads are not always displaced with occult fractures of the radial neck or physis ([40,92,94](#)).

Arthrography or magnetic resonance imaging (MRI) can be used to determine any displacement of the unossified radial head.

## Classification

Jeffrey's classification ([41](#)) is the most widely used. It is based on the two major mechanisms of injury (valgus stress and those associated with a dislocation of the elbow).

## AUTHORS' CLASSIFICATION

We have classified these fractures, on the basis of the mechanism of injury and displacement of the radial head, into three major groups ([Table 11-1](#)):

- Group I: Primary displacement of the radial head
  - A. Valgus fractures
    1. Type A—Salter-Harris type I and II injuries of the proximal radial physis
    2. Type B—Salter-Harris type IV injuries of the proximal radial physis
    3. Type C—Fractures involving only the proximal radial metaphysis
  - B. Fractures associated with elbow dislocation
    1. Type D—Reduction injuries
    2. Type E—Dislocation injuries
- Group II: Primary displacement of the radial neck
  - A. Angular injuries (Monteggia type III variant)
  - B. Torsional injuries
- Group III: Stress injuries
  - A. Osteochondritis dissecans of the radial head
  - B. Physeal injuries with neck angulation

**TABLE 11-1. CLASSIFICATION OF FRACTURES INVOLVING THE PROXIMAL RADIUS**

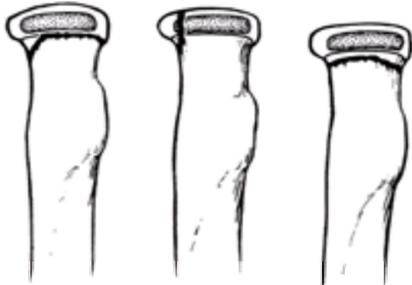
Group I: Those in which the radial head is primarily displaced (most proximal radial injuries are in this group)

Group II: Those in which there is primary displacement of the radial neck

Group III: Stress injuries

### Head-Displaced Fractures

For the head-displaced fractures (group I), we combined the classifications of Jeffrey (41) and Newman (67) to produce a new classification based primarily on the mechanism of injury. The two main subclasses of fractures in group I are valgus injuries and those associated with elbow dislocations. Valgus injuries are subdivided into three types (A, B, and C) based on the location of the fracture line (Fig. 11-4). Fractures associated with an elbow dislocation are subdivided into two types. The first, type D, is based on the original concept proposed by Jeffrey (41) that the fracture occurs during the period of spontaneous reduction (Fig. 11-5A). In this case, the radial head lies proximal to the posterior aspect of the joint. The second, type E, is based on Newman's concept (67) that the fracture and displacement occur during the process of dislocation of the elbow. In this type, the radial head lies distal to the anterior portion of the joint (Fig. 11-5B). There appears to be no need to separately classify fractures involving only the radial head. Most radial head fractures in children described in the literature have been Salter-Harris type IV injuries containing portions of both the epiphysis and metaphysis.



**FIGURE 11-4.** Types of valgus injuries. **Left:** Type A: Salter-Harris type I or II physeal injury. **Center:** Type B: Salter-Harris type IV injury. **Right:** Type C: Total metaphyseal fracture pattern.

### Neck-Displaced Fractures

For the neck-displaced fractures (group II), there are two subgroups, angular and torsional.

### Stress Injuries

The final group (group III), stress injuries, includes osteochondritis of the radial head and physeal injuries of the neck that produce angular deformities.

### Mechanisms of Injury

Table 11-2 lists the proposed mechanisms for fractures of the radial head and neck in children.

- I. Primary displacement of the head (incongruous)
  - A. Valgus injuries
  - B. Associated with dislocation of the elbow
    1. During reduction
    2. During dislocation
- II. Primary displacement of the neck
  - A. Angular forces
  - B. Rotational forces
- III. Chronic stress forces

**TABLE 11-2. FRACTURES OF THE RADIAL HEAD AND NECK: PROPOSED MECHANISMS IN CHILDREN**

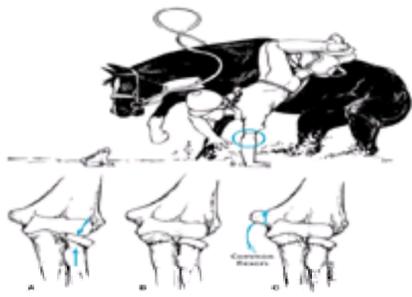
### Primary Displacement of the Radial Head (Group I)

In general, when these fractures occur, the major force is applied to the radial head and is secondarily transmitted to the radial neck, which fractures because it is weaker metaphyseal bone. As a result, the radial head usually is displaced by angulation, rotation, translocation, or complete separation of the radial head from the neck. This displacement of the radial head produces an incongruity of the proximal radioulnar joint, which is the major cause of dysfunction. In this type of injury, treatment emphasizes reduction of the head to its normal congruous position with the shaft and proximal radioulnar joint.

### Valgus Injuries

**Angular Force on the Neck.** Most of these injuries appear to be caused by a fall on the outstretched arm with the elbow in extension (32,39,41,67,68,113) and an

associated valgus thrust to the forearm (Fig. 11-6). This valgus strain puts a compression force across the radiocapitellar joint. Because much of the head is cartilage, the valgus force is transmitted to the weaker physis or metaphysis of the neck (113). These fractures characteristically appear as an angular deformity of the head with the neck (Fig. 11-6A). The direction of angulation depends on whether the forearm is in a supinated, neutral, or pronated position at the time of the fall. Vostal (113) has shown that in neutral, the pressure is concentrated on the lateral portion of the head and neck. In supination, the pressure is concentrated anteriorly, and in pronation it is concentrated posteriorly.



**FIGURE 11-6.** The most common mechanism of radial neck fractures involves a fall on the outstretched arm. This produces an angular deformity of the neck (A). Further valgus forces can produce a greenstick fracture of the olecranon (B) or an avulsion of the medial epicondylar apophysis (C). (Redrawn with permission from Jeffery CC. Fracture of the head of the radius in children. *J Bone Joint Surg Br* 1950;314-324.)

**Associated Injuries.** This valgus stress pattern causes associated injuries about the elbow (41,42,68,98,32) (Fig. 11-6B, Fig. 11-6C) such as greenstick fracture of the olecranon (Fig. 11-7), which Bado (4) considered an equivalent of a type I Monteggia lesion. An avulsion fracture of the medial epicondylar apophysis may also occur (15). In Fowles and Kassab's series of patients with radial neck fractures (27), more than 61% had one of these associated injuries.



**FIGURE 11-7.** Associated fractures of valgus stress. Anteroposterior view of a fracture of the radial neck associated with a greenstick fracture of the olecranon (arrows).

Children with an increased carrying angle may be predisposed to injury of the proximal radius. Henrikson (39) found that the degree of cubitus valgus in patients who sustained this injury was greater than in patients with other types of elbow fractures.

**Fracture Patterns.** With these valgus injuries, the fracture pattern can be one of three types (Fig. 11-4). In the first two types, the fracture line involves the physis. Type A represents either a Salter-Harris type I or II physeal injury. In a Salter-Harris type II injury, the metaphyseal fragment is triangular and lies on the compression side. In type B fractures, the fracture line courses vertically through the metaphysis, physis, and epiphysis to produce a Salter-Harris type IV physeal fracture pattern (Fig. 11-8). This is the only fracture type that involves the articular surface of the radial head. In type C fractures, the fracture line lies completely within the metaphysis (Fig. 11-9), and the fracture can be transverse or oblique. Type B fractures are rare. The incidences of types A and C fractures are approximately equal (98).



**FIGURE 11-8.** Valgus (type B) injury. **A:** Three weeks after the initial injury, there was evidence of distal migration of this Salter-Harris type IV fracture fragment. Periosteal new bone formation has already developed along the distal metaphyseal fragment (arrow). **B:** Six months after the initial injury, there is evidence of an osseous bridge formation between the metaphysis and the epiphysis. Subsequently, the patient had secondary degenerative arthritis with loss of elbow motion and forearm rotation.



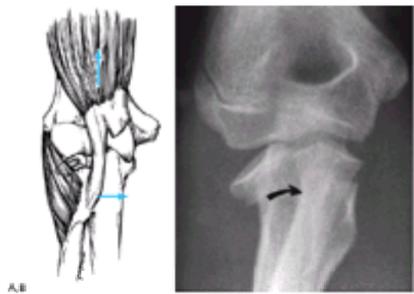
**FIGURE 11-9.** Valgus type C injury. The fracture line is totally metaphyseal and oblique (arrows)

**Displacement Patterns.** Regardless of the type of fracture pattern, displacement can vary from minimal angulation to complete separation of the radial head from the neck (Fig. 11-10). With minimal angulation alone, the congruity of the proximal radioulnar joint usually is retained. If the radial head is displaced in relation to the radial neck, the congruity of the proximal radioulnar joint is lost because of the cam effect. With complete displacement, there is very little contact between the fracture surfaces. Completely displaced fractures often are associated with more severe injuries.



**FIGURE 11-10.** Displacement patterns. The radial head can be angulated (A), translated (B), or completely displaced (C).

**Neck Migration.** Distally, there is also displacement of the neck and shaft fragment. Patterson (72) believed that once the stabilizing effect of the radial head is lost, the distal fragment can migrate proximally. This proximal migration of the distal fragment tends to be ulnarward because of the forces exerted in this direction by the supinator and biceps muscles (Fig. 11-11). Patterson attempted to counteract these forces in his manipulative technique (see section on Patterson's Manipulative Technique). When there is a strong valgus component, the proximal portion of the distal fragment of the radius can get locked medial to the coronoid process, making a closed reduction almost impossible (26,55).

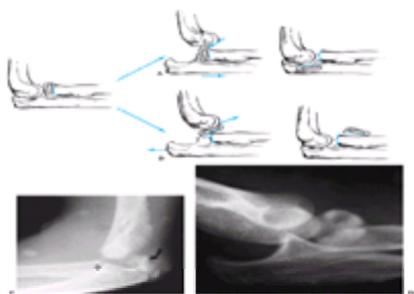


**FIGURE 11-11.** Forces producing displacement. **A:** Once the stabilizing effect of the radial head is lost, the distal fragment (radial neck and proximal shaft) is displaced ulnarward and proximally by the unopposed biceps and supinator muscles (arrows). (Redrawn with permission from Patterson RF. Treatment of displaced transverse fractures of the neck of the radius in children. *J Bone Joint Surg* 1934;16:695.) **B:** Radiograph demonstrating proximal and medial (ulnar) displacement of distal neck fragment (arrow). (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:55).

#### Associated with Elbow Dislocation

In two rare types of fractures of the radial neck, the head fragment is totally displaced from the neck (5,15,28,41,67,114). These types are associated with an elbow dislocation. The proposed mechanism is a fall on the hand with the elbow flexed, which causes a momentary partial dislocation of the elbow and forces the radial head posterior to the capitellum.

**During Reduction (Type D).** In the original description of this injury, Jeffrey (41) suggested that displacement and fracture occurred during spontaneous reduction of the transiently dislocated elbow. During this reduction process, the capitellum applies a proximal force to the distal lip of the radial head, causing it to separate as the forearm and distal radius are reduced distally (Fig. 11-5A). The radial neck and olecranon return to their anatomic locations while the radial head remains in the posterior aspect of the joint (114).



**FIGURE 11-5.** Dislocation fracture patterns. **A:** Type D: The radial neck is fractured during the process of reduction by the capitellum pressing against the distal lip of the radial head (41). **B:** Type E: The radial neck is fractured during the process of dislocation by the capitellum pressing against the proximal lip of the radial head (67). **C:** Radiographs of a radial head that fractured during the reduction of the dislocation (type D). The radial head (solid arrow) lies posterior to the distal humerus, and the distal portion of the neck (open arrow) is anterior. (Courtesy of Richard E. King, MD.) **D:** Radiograph of the dislocated elbow in which the fracture of the radial neck occurred during the process of dislocation (type E).

**During Dislocation (Type E).** Newman (67) described a type of radial head fracture in which the fracture occurs during the process of dislocation. In this case, the capitellum applies a distally directed force to the proximal lip of the radial head as the elbow is dislocated (Fig. 11-5B). The elbow may remain dislocated with the radial head lying anterior and often parallel to the long axis of the neck fragment. If the dislocation is reduced, either by manipulation or spontaneously, the radial head lies free in the anterior portion of the elbow joint (67,110,114).

#### Primary Displacement of the Radial Neck (Group II)

Rarely, angular or torsional forces cause a primary disruption or deformity of the neck, while the head remains congruous within the proximal radioulnar joint.

Treatment of these injuries is manipulation of the distal neck fragment to align it with the head.

### Angular Forces

Angular forces always produce type III Monteggia equivalents. A Monteggia type III fracture pattern is created when a varus force is applied across the extended elbow, resulting in a greenstick fracture of the olecranon or proximal ulna and a lateral dislocation of the radial head ( [120](#)). Occasionally, however, the failure occurs at the radial neck (Monteggia III equivalent), and it displaces laterally, leaving the radial head and proximal neck fragment reduced under the orbicular ligament ( [69](#)) ([Fig. 11-12](#)). The problem in the management of this injury is alignment of the distal neck fragment with the proximal radial and neck fragments.



**FIGURE 11-2.**Angular forces. This 8-year-old sustained a type III Monteggia equivalent in which the radial neck fractured (*arrow*), leaving the radial head reduced proximally. (Courtesy of Ruben D. Pechero, MD.)

### Rotational Forces

Rotational forces appear to occur in young children before ossification of the proximal radial epiphysis. Both reports of this injury are in the European literature ([32,39](#)), and in both, the initial rotational force was in supination. Reduction was achieved by pronation of the forearm. Diagnosis of these injuries is difficult and may require arthrography or an examination under general anesthesia. This injury should be differentiated from the more common subluxation of the radial head (pulled elbow syndrome), in which the forearm usually is held in pronation with resistance to supination. In addition, on radiography there usually are no signs of hemarthrosis, as may be seen in the torsional fractures.

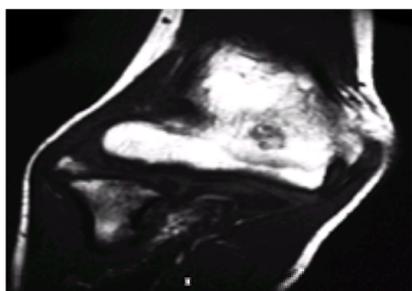
### Stress Injuries (Group III)

A final mechanism is chronic, repetitive stress, both longitudinal and rotational, on either the head or the proximal radial physis. These injuries usually are the result of high-performance athletic activity in which the upper extremity is required to perform repetitive motions. These repetitive stresses disrupt growth of either the neck or the head, with eventual deformity. These are actually forms of stress fractures.

In the United States, the popularity of organized sports has produced a number of unique injuries in pediatric athletes related to repetitive stress applied to growth centers. This is especially true in the immature elbow. Most injuries are related to throwing sports, primarily Little League baseball. Most of this "Little League pathology" involves tension injuries on the medial epicondyle. In some athletes, however, the lateral side may be involved as well because of the repetitive compressive forces applied to the capitellum and radial head and neck. In the radial head, lytic lesions similar to osteochondritis dissecans may occur ( [22,108,118](#)) ([Fig. 11-13](#) and [Fig. 11-14](#)). If the stress forces are transmitted to the radial neck, the anterior portion of the physis may be injured, producing an angular deformity of the radial neck ([23](#)) ([Fig. 11-15](#)).



**FIGURE 11-13.** Osteochondritis dissecans. Radiograph of this 11-year-old Little League pitcher's elbow shows fragmentation of the subchondral surfaces of the radial head. These changes and the accelerated bone age are evidence of overuse.



**FIGURE 11-14.**Baseball player with osteochondritis dissecans lesion and fragmentation after direct blow to elbow.

### Treatment

The methods of treatment for radial head injuries include:

1. Simple immobilization with no manipulation
2. Manipulative closed reduction
3. Percutaneous pin reduction
4. Intramedullary pin reduction
5. Open reduction with or without internal fixation

## 6. Excision of either the entire head or the small head fragment

Determination of the proper method of treatment depends on many factors, including the degree and type of displacement, the association of other injuries, the age of the patient, and the time elapsed since the injury.

### **Prognostic Factors**

#### **Severity of Injury**

Some factors may be more important than the type of treatment in determining the final result. It is still controversial whether age has a favorable or unfavorable effect on the outcome (98). A poor result can be expected if the fracture is associated with other injuries, such as an elbow dislocation, a fracture of the olecranon, or avulsion of the medial–epicondylar apophysis (27,92). The magnitude of injurious force to the elbow appears to be a major factor in determining the quality of the result (51,98).

#### **Quality of Reduction**

Another factor that appears to influence the result is the quality of the reduction. Residual tilt of the head is tolerated better than displacement of the head. Tibone and Stoltz (106) reported that the number of good results decreased if the initial angulation exceeded 30 degrees or the amount of displacement exceeded 3 mm. Newman (67,98) found that more than 4 mm of displacement increased the frequency of poor results and the risk of synostosis with the proximal ulna.

#### **Acceptable Angulation**

The amount of acceptable angulation varies. Some clinicians accept up to 30 degrees of residual angulation (51,59,67,110,80), whereas others believe that up to 45 degrees of residual angulation can yield a satisfactory result (9,16,68,112). At the other end of the spectrum, Pollen (78) believed that in older children, only 15 degrees of angulation should be accepted without attempting manipulation. The spontaneous correction that can be expected to occur with growth in younger children is approximately 10 degrees.

#### **Outcomes**

In theory, the cam effect should limit supination and pronation if there is significant displacement of the proximal fragment. However, adequate remodeling with a functional range of motion can occur with as much as 40% displacement (Fig. 11-16).



**FIGURE 11-16.** Translocation remodeling. **A:** Injury film of a 9-year-old who had 60 degrees of supination and pronation by clinical examination with local anesthesia into the elbow joint. Because range of motion was functional, the position was accepted. **B:** Two months postfracture, there was almost complete remodeling of the translocation. The patient's forearm rotation was 75 degrees in both directions. (Courtesy of Earl A. Stanley, Jr, MD.)

In general, fractures in which an adequate closed reduction can be obtained have better results than those requiring open reduction. This may be because injuries that can be managed by closed methods are the result of less severe trauma than those requiring open reduction. The poor results in those managed with open methods may be due just as much to the associated soft tissue injuries as to the surgical insult.

The overall incidence of poor results in large series varies from 15% to 33% (27,39,42,98,110). If only severely displaced fractures were considered, the incidence of poor results could be as high as 50% (98). Thus, at least one in five or six children can be expected to have a poor result despite adequate treatment. Predicting which children will have poor results before treatment is initiated can be valuable. Forewarning the parents of a possible poor result is much wiser than telling them after the treatment has been completed. A child of approximately 7 years of age with minimal angulation or displacement and no associated injuries can be expected to have a good result. In a 12-year-old with a fracture associated with a dislocation and with complete displacement of the radial head, motion of the elbow may be significantly limited.

After 6 months, very little improvement in motion can be expected. Steinberg et al. (98) found that function in their patients at 6 months was almost equal to that when the patients were examined years later.

### **Nonoperative Methods**

#### **Simple Immobilization (Up to 30 Degrees of Angulation)**

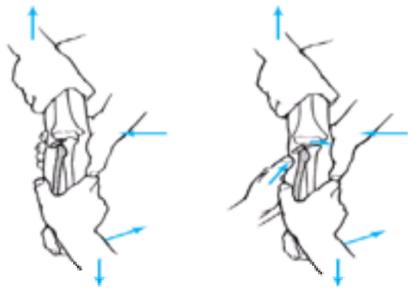
Fractures in younger children in which the angulation of the head is less than 20 to 30 degrees usually can be managed with some type of simple immobilization. A collar and cuff, a posterior splint, or a light long arm cast usually is sufficient to provide comfort and protection from further injury. Aspiration of the intraarticular hematoma may provide added comfort.

#### **Manipulative Closed Reduction (30 to 60 Degrees)**

In uncomplicated fractures with angulation of up to 60 degrees, a satisfactory result usually can be achieved with manipulative closed reduction (9,41,115). The chance of achieving a satisfactory reduction when the initial angulation exceeds 60 degrees is much less. Although angulation of up to 45 degrees can produce acceptable results, fractures with more than 30 degrees of angulation should probably be manipulated.

#### **Patterson's Manipulative Technique**

Many authors (20,59) have adopted a technique of manipulative reduction with the elbow in extension, as advocated by Patterson (72). Adequate relaxation is required and can be achieved only by a general or some type of regional anesthetic technique. The orbicular ligament should be intact to stabilize the proximal radial head fragment (59). In Patterson's technique, an assistant grasps the arm proximal to the elbow joint with one hand (Fig. 11-17, left) and places the other hand medially over the distal humerus to provide a medial fulcrum for the varus stress applied across the elbow. The surgeon applies distal traction with the forearm supinated to relax the supinators and biceps. A varus force is applied across the elbow to overcome the ulnar deviation of the distal fragment so that it can be aligned with the proximal fragment. This varus force also helps open up the lateral side of the joint, which facilitates manipulation of the head fragment.



**FIGURE 11-17.** Patterson's manipulative technique. **Left:** The assistant grabs the arm proximally with one hand placed medially against the distal humerus. Distal traction with the forearm supinated is applied by the surgeon. A varus force is also applied to the forearm. **Right:** Digital pressure is then applied directly over the tilted radial head to complete reduction. (Redrawn with permission from Patterson RF. Treatment of displaced transverse fractures of the neck of the radius in children. *J Bone Joint Surg* 1934;16:696-698.)

Although forearm supination may relax the supinator muscle, it may not be the best position for manipulation of the head fragment. Jeffrey (41) points out that the tilt of the radial head can be anterior or posterior depending on the position of the forearm at the time of injury. The forearm should be rotated until the maximal tilt of the proximal fragment is felt laterally. The direction of maximal tilt can be confirmed on radiography. If the x-ray beam is perpendicular to the head in maximal tilt, it casts an oblong or rectangular shadow. If not, the shadow is oval or almost circular (41). With the varus force applied across the extended elbow and the maximal tilt directed laterally, digital pressure is applied over the radial head to manipulate it back into position ( Fig. 11-17, right).

Kaufman et al. (44) proposed another technique in which the elbow is manipulated in the flexed position. The surgeon's thumb is pressed against the anterior surface of the radial head and the forearm is forced into pronation ( Fig. 11-18).



**FIGURE 11-18.** Flexion = npronation (Israeli) technique of reduction. **A:** The thumb is placed over the radial head fracture site. The forearm is grasped distally. **B:** The forearm is then gradually pronated (arrow) until full pronation is achieved. During this procedure, pressure is maintained proximally over the area of the radial head fracture.

After the quality of the reduction is confirmed radiographically, the arm usually is immobilized in pronation. Pronation is recommended because it is the forearm motion most often restricted after fracture (115). Some have recommended that the forearm be placed in supination because they believe it is easier for the patient to regain active pronation than active supination during rehabilitation. Whether pronation or supination is chosen, the elbow is placed in 90 degrees of flexion.

Although most fractures are stable after reduction, redisplacement can occur, especially if the initial tilt was more than 60 degrees ( 21,42). Fractures with more than 90 degrees of angulation, especially those in which the head fragment is lying free in the joint, are almost impossible to reduce by closed methods.

Since the late 1960s, four patients have been reported in whom the head fragment was lying proximal to the neck and parallel to its long axis ( 28,117,119). In each, manipulation was attempted and initially a satisfactory position appeared to be achieved. A closer inspection showed, however, that the head had been completely rotated 180 degrees so that the articular surface of the head was facing the fracture surface of the radial neck ( Fig. 11-19).



**FIGURE 11-19.** One hundred eighty-degree rotation. Radiograph of a radial head that was rotated 180 degrees in which the articular surface (arrows) is facing the fracture surface of the distal fragment. (From Fraser KE. Displaced fracture of the proximal end of the radius in a child: a case report of the deceptive appearance of a fragment that had rotated 180°. *J Bone Joint Surg Am* 1995;77:782-783; with permission.)

## Operative Techniques

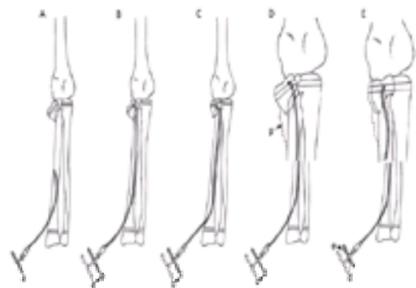
### Percutaneous Pin Reduction

The use of percutaneous pin reduction with an image intensifier has become a popular method of satisfactorily reducing moderately to severely displaced fractures without the trauma of a formal surgical incision ( 3,6,20,75,97). The radial head is manipulated into place under image control with a single-pointed awl, a Steinmann pin (86), a periosteal elevator (29), or a double-pointed "bident" (3). The latter in theory controls rotation better. Pesudo et al., in their series of 22 displaced radial neck fractures, found that the results after percutaneous pin reduction were superior to those after open reduction ( 75).

Biyani et al. (7) drive the pin that is used to reduce the radial head across the fracture site to stabilize it. The pin is left in for 3 weeks and then removed to allow motion.

### Intramedullary Pin Reduction

In 1980, Metaizeau et al. (61) proposed reducing severely tilted radial neck fractures with an intramedullary wire passed from the distal metaphysis. A report 13 years later (60) demonstrated the effectiveness of this technique. The wire is inserted into the medullary canal through an entrance hole in the distal metaphysis ( Fig. 11-20). Once the wire reaches the fracture site, the angulation at the tip enables it to engage the proximal fracture site at the neck. Once engaged, the wire is then twisted to reduce the head and neck fragment. This technique has produced results superior to open reduction with fewer complications ( 33,93).



**FIGURE 11-20.** Intramedullary pin reduction. **A:** The curved flexible pin is inserted distally in the metaphysis and passed up the shaft proximally. **B:** The curved end of the rod is engaged into the proximal fragment. **C:** The rod is used to distract and disimpact the head fragment. **D and E:** Once the head fragment is disimpacted, it is then rotated into position with the intramedullary rod. (From Metaizeau JP, Lascombes P, Lemelle JL, et al. Reduction and fixation of displaced radial neck fractures by closed intramedullary pinning. *J Pediatr Orthop* 1993;13:55-356; with permission.)

### Open Reduction

The primary consideration in treatment choice is whether open reduction produces better results than manipulative closed reduction. With valgus injuries, a residual tilt of 45 degrees probably produces as good a result as trying to achieve a perfect reduction surgically. A satisfactory closed reduction produces better results than an anatomically perfect open surgical reduction ( Table 11-3).

<45 degrees of angulation
No translation
50 to 60 degrees of pronation and supination on clinical examination

**TABLE 11-3. ACCEPTABLE REDUCTION**

Early reviews reported poor results with significant loss of range of motion in cases managed operatively ( 16,67,106,115). In a more recent review, Steinberg et al. (98) combined their results of open reduction of severely displaced fractures with those of five other series ( 41,42,75,106) and reported 49% good results compared with 25% in those treated nonoperatively. Percutaneous pin reduction was not used in any of these series. The results of moderately displaced fractures treated operatively were equal to the results of those treated nonoperatively.

If the head of the radius is completely displaced, results are usually better with surgical intervention. Several authors have shown that the completely separated head remains viable if surgically replaced as late as 48 hours after injury ( 31,45).

Some fractures may be irreducible by closed means because interposition of the capsule or annular ligament between the head and neck impedes reduction ( 113).

Strong et al. (100) described an unusual fracture pattern in which the radial head remained reduced by the orbicular ligament. They found that these fractures were irreducible by closed methods and required open reduction.

Surgery should be done as soon as possible after the injury. The later the surgical intervention, the poorer the results. McBride and Monnet ( 58) reported three patients in whom an osteotomy of the neck was done for residual angulation 3 to 5 weeks after injury. All had further loss of range of motion because of the development of a proximal cross-union. Blount (9) set a limit of 5 days, after which surgical intervention is more likely to produce a poorer result than if the fracture is left untreated.

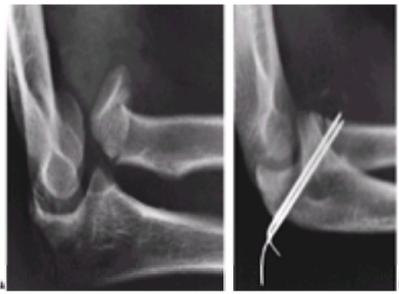
**Fixation Methods.** A small wire through the capitellum across the radial head and into the neck has been recommended for fixation of radial head fractures (59,85,86), but this technique has a high incidence of complications ( 27,67,92,115). Even in a long arm cast the elbow joint has slight motion, which can cause the pin to fatigue, with subsequent breakage ( Fig. 11-21). The retrieval of the remaining portion of the pin from the proximal radius is almost impossible without imposing considerable trauma. Even if the pin does not break, the slight motion of the pin has been reported to cause erosion of the joint surface and even fragmentation of the radial head (67,115).



**FIGURE 11-21.** Transcapitellar pin in a 4-year-old with a completely displaced fracture of the radial neck. Open reduction was performed and fixation achieved with a small transcapitellar pin. **A:** Three weeks after injury, when the pin was removed, it had fractured distally and a portion remained in the proximal radius. **B:** An arthrogram revealed that the pin was fractured at the joint surface of the radial head. The pin was left in place, with subsequent resumption of normal elbow motion.

Other suggested methods of fixing the displaced radial neck after open reduction include direct pin insertion through the head through an olecranon osteotomy ( 52) and intramedullary bone pegs with a forked plate ( 50).

The most common technique in current use involves placement of a pin obliquely across the fracture site into the head ( 20,27,42). The pin can be placed either proximal to distal (Fig. 11-22) or, preferably, distal to proximal. The pin is left protruding from the skin for easy removal.



**FIGURE 11-22.** Oblique pin. **A:** Displaced fracture of the radial neck in a 10-year-old. **B:** A closed reduction was performed, and to stabilize the head fragment two pins were placed percutaneously and obliquely across the fracture site from proximal to distal. If this is performed open, the preferred alignment is to go obliquely across the fracture site from distal to proximal. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994;57; with permission.)

Many surgeons believe that internal fixation is unnecessary after open reduction ( 9,67,115). Wedge and Robertson ( 115) found that patients without internal fixation had more good results than those in whom internal fixation was used. Use of internal fixation does not guarantee that the fragment will not slip. Newman ( 67) reported two patients in whom the head slipped postoperatively despite Kirschner wire fixation.

One complication of open reduction is injury to the posterior interosseous nerve. To avoid this complication, Jones and Esah ( 42) recommended that the nerve first be identified and isolated as it courses through the supinator muscle. Kaplan ( 43) and Strachan and Ellis ( 99) demonstrated that with the forearm in pronation, the posterior interosseous nerve is displaced ulnarward, out of the way of the surgical dissection. They also recommended that the patient be placed prone during exploration of the proximal radius to facilitate keeping the forearm pronated.

The orbicular ligament should be preserved if possible. If it is necessary to cut it to achieve an adequate reduction, careful repair is important ( 27).

*Type B Injuries.* Type B (Salter-Harris type IV) injuries can be successfully treated by removing the small marginal fragment. If the remaining large portion of the head is also displaced, it is simply reduced without internal fixation ( 115).

*Radial Head Fractures.* Pelto et al. ( 74) reported good to excellent results with the use of absorbable polyglycolide pins in patients older than 13 years of age. There was a significant "transient local abacterial tissue reaction" that did not lead to any long-term reactions. The effect on the physis was not mentioned.

In the rare Salter-Harris types III and IV fracture patterns in which there is a large head fragment, fixation can be achieved with a small minifragment plate placed in a transepiphyseal manner (Fig. 11-23).



**FIGURE 11-23.** Mini-screw fixation. **A and B:** Anteroposterior and lateral views of the elbow of a 6-year-old boy in whom the head fragment is lying posterior to the capitellum (arrows). **C:** An open reduction was performed, and it was discovered that this was a Salter-Harris type III fracture through the epiphysis and proximal physis. The fragment involved 60% of the head diameter. It still had soft tissue attachments. **D:** The fracture was reduced and fixation was achieved with a screw placed through the epiphysis. **E:** Six months after surgery, when the screw was removed, an arthrogram showed maintenance of the architectural structure of the medial head. The patient had 60 degrees of supination and pronation. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:58; with permission.)

### Radial Head Excision

Resection of the radial head fragment was popular in the 1920s and 1930s, but reported results have been uniformly poor ( 21,39,42). Cubitus valgus and radial deviation at the wrist are common sequelae.

### AUTHORS' PREFERRED METHOD OF TREATMENT

We tend to be as conservative as possible in treating these fractures ( Table 11-4). Although they may appear reasonably minor, both radiographically and clinically, we initially emphasize to the parents that some loss of motion may occur even though an anatomic reduction is achieved. The problem is usually a loss of range of motion in supination and pronation. Little pain or loss of upper extremity function occurs despite the residual loss of range of motion.

Fracture Status	Treatment
Minimally displaced (<30 degrees angulation, no translation)	Long arm cast or posterior splint (7-10 days) Early motion
Angulation >30 degrees	Closed reduction under general anesthesia using flexion-pronation (Israeli) technique Long arm cast (10-14 days)
Angulation >45 degrees	Elastic bandage wrap reduction Flexion-pronation (Israeli) reduction technique Percutaneous pin reduction Long arm cast (10-14 days)
Angulation fixed at >40 degrees, translation >3 mm displacement, with <60 degrees supination-pronation, radial head completely displaced	Intramedullary wire reduction Open reduction (+ internal fixation)

**TABLE 11-4. TREATMENT OF RADIAL HEAD FRACTURES**

### Aspiration Facilitates Evaluation

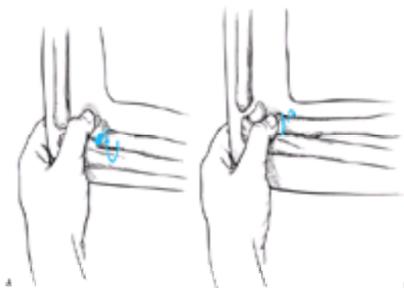
If there is a great amount of pain or resistance to pronation and supination, we aspirate the elbow and frequently inject 2 to 3 mL of 1% lidocaine after a sterile preparation (19).

### Nonoperative Methods

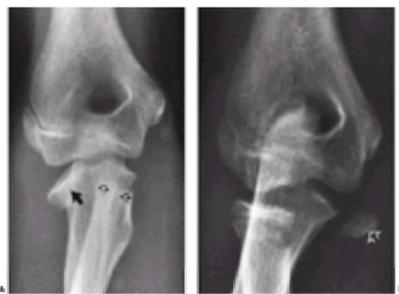
**Moderate Tilt (30 to 60 Degrees).** If the radial head is tilted more than 30 degrees, we usually attempt a manipulative closed reduction. This should be done with the use of a general anesthetic to achieve full relaxation. We prefer the Israeli technique ( Fig. 11-18) for simple, moderately displaced radial head fractures ( Fig. 11-24). The important aspect is to ensure that the manipulation is done with the elbow flexed at 90 degrees. Usually there is resistance to pronation ( Fig. 11-25). Once the pressure is applied to the radial head with the thumb, the opposite hand forces the forearm into full pronation ( Fig. 11-25). After reduction, the forearm is tested for range of motion to ensure there are at least 60 degrees of supination and pronation. Follow-up radiographs usually reveal a near-anatomic reduction ( Fig. 11-24B). If, however, there is an adequate range of supination and pronation, we accept the reduction regardless of the radiographic appearance. We have also used this as an initial attempt even in completely displaced radial neck fractures, with surprising results ( Fig. 11-26).



**FIGURE 11-24.** Flexion-pronation (Israeli) reduction technique. **A:** Radiograph of the best reduction obtained by the Patterson method. **B:** Position of the radial head after use of the flexion = npronation method. (Courtesy of Gerald R. Williams, MD.)

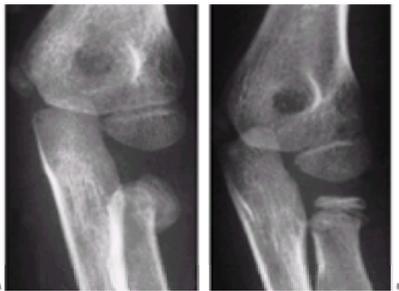


**FIGURE 11-25.** Flexion = npronation (Israeli) reduction technique. **A:** With the elbow in 90 degrees of flexion, the thumb stabilizes the radial head, which is displaced. Usually the distal radius is fixed in a position of supination. The forearm is then pronated to swing the shaft up into alignment with the neck (arrow). **B:** The final alignment is achieved by continuing to force the forearm into full pronation (arrow). (See also Fig. 11-18.)



**FIGURE 11-26.** Widely displaced fracture. **A:** A 9-year-old girl sustained this widely displaced fracture in which the neck fragment (open arrows) was medial and the head fragment (closed arrow) remained within the orbicular ligament. **B:** A satisfactory reduction was achieved using the flexion = npronation method. The small fragment medially (arrow) is from the metaphysis. The patient resumed full rotation of the forearm after reduction. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:55; with permission.)

**Elastic Bandage Wrap.** If we cannot get an adequate reduction using the Patterson or Israeli technique, we try some other techniques before surgical intervention. In a few patients we have found that, after wrapping the extremity tightly from distal to proximal with an elastic Esmarch, a satisfactory reduction was serendipitously achieved (Fig. 11-27).



**FIGURE 11-27.** Elastic bandage wrap reduction. **A:** The final position achieved after manipulation by the Patterson method. **B:** Position of the radial head after an elastic bandage had been applied to exsanguinate the extremity.

### Operative Techniques

**Percutaneous Pin Reduction.** For most moderately or severely displaced fractures, we prefer to attempt a semiclosed reduction using the percutaneous pin reduction technique (Fig. 11-28). A good image intensifier is essential. We have found that a single-point awl or the blunt end of a Steinmann pin is adequate. If the head is small or soft, a small Craig biopsy needle or the blunt end of the Steinmann pin can be used to prevent penetration of the radial head with a sharp Steinmann pin.



**FIGURE 11-28.** Percutaneous pin reduction. **A:** Under an image intensifier, the sharp-pointed object (awl) is inserted next to the olecranon and directed up proximally toward the radial head fragment. This is to avoid injury to the posterior interosseous nerve. **B:** Totally displaced valgus injury. **C:** Position of the Steinmann pin during reduction. **D:** Appearance 2 months after surgery. The patient has 60 degrees of supination and pronation with full elbow extension and flexion.

To avoid injury to the posterior interosseous nerve, we try to insert the pin as closely as possible to the lateral aspect of the olecranon ( Fig. 11-28A). Again, we accept 45 to 60 degrees of angulation as long as the displacement is corrected and the patient has at least 50 to 60 degrees of supination and pronation after reduction.

**Intramedullary Pin Reduction.** Before open reduction of fractures that are severely angulated but still have some apposition, we attempt to achieve reduction using an intramedullary pin passed from distal to proximal, as described by Metaizeau et al. ( 60) (Fig. 11-20). Although this method has achieved success where other manipulative techniques failed, in most patients simple percutaneous pin manipulation produces reductions equal to this technique. We try every manipulative technique before we resort to open reduction.

**Open Reduction.** If an adequate closed reduction cannot be obtained and loss of motion is still significant, we prefer an open reduction with as little dissection as possible. We approach the fracture with the patient prone and the forearm pronated. After making the skin incision, we dissect between the anconeus and extensor carpi ulnaris muscles to reach the orbicular ligament. The head is repositioned as gently as possible. Usually, we have found the head fragment to be stable after reduction. If it is not, fixation is achieved with a small pin placed obliquely through a separate stab incision from distal to proximal across the fracture site. Two oblique, crossed pins also can be inserted at the articular margin in very unstable fractures.

**Delayed Reduction.** If the fracture is more than 4 days old and there is no significant supination or pronation forearm motion, we often reduce the head surgically if it cannot be done closed. However, we warn the parents that myositis ossificans and proximal radioulnar synostosis can occur.

**Reduction After Immobilization.** After the reduction, we prefer to immobilize the upper extremity with the elbow in 90 degrees of flexion and the forearm in slight pronation. Active motion is started as soon as possible. If a closed reduction was done, we start gradual active motion at 10 to 14 days, depending on the initial displacement and the degree of stability achieved at the time of reduction. We prefer to use a long arm cast that can be bivalved and used as a splint during rehabilitation.



### ASSOCIATED INJURIES AND COMPLICATIONS

Complications of fractures of the radial neck and head, especially those associated with other fractures or a dislocation of the elbow, range from the most common problem of loss of range of motion to rare nerve injuries (Table 11-5). The most complete reviews of these complications can be found in reports by Steinberg et al. (98) and D'Souza et al. (16).

- Failure to obtain acceptable reduction
- Loss of motion (expected)
- Radial head overgrowth
- Notching of the radial neck
- Premature physal closure
- Angular deformities
- Nonunion
- Avascular necrosis
- Radioulnar synostosis
- Problems with initial diagnosis

**TABLE 11-5. COMPLICATIONS OF RADIAL HEAD AND NECK FRACTURES**

### **Loss of Motion**

Loss of motion is secondary to a combination of loss of joint congruity and fibrous adhesions. Loss of pronation appears to be more common than loss of supination. Flexion and extension are rarely significantly limited. Enlargement of the radial head, a common sequela, can contribute to the subsequent loss of elbow motion ( [31](#)).

### **Radial Head Overgrowth**

Next to loss of range of motion of the elbow and forearm, radial head overgrowth is probably the most common sequela (20%–40%) ( [16,110](#)). The increased vascularity from the injury probably stimulates epiphyseal growth. Radial head overgrowth usually does not compromise functional results ( [20,42](#)), but it may produce some crepitus or clicking with forearm rotation ( [16](#)).

### **Notching of the Radial Neck**

O'Brien ( [68](#)) suggested that notching of the radial head in several patients was caused by scar tissue forming around the neck from the orbicular ligament. It did not seem to result in any functional deficit.

### **Premature Physeal Closure**

Although premature physeal closure has been reported in many series ( [27,31,67,68,98,115](#)), this complication did not appear to affect the overall results significantly, except in one patient reported by Fowles and Kassab ( [27](#)), in whom it produced a severe cubitus valgus. Newman ( [67](#)) found that shortening of the radius was never any greater than 5 mm compared with the opposite uninjured side.

### **Nonunion of the Radial Neck**

Nonunion of the radial neck is rare; union eventually occurs ( [92](#)) if enough time elapses ( [Fig. 11-29](#)).



**FIGURE 11-29.** Nonunion. **A:** Eight months after radial neck fracture in an 8.5-year-old girl. Patient had mild aching pain, but no loss of motion. There was some suggestion of proximal subluxation of the distal radioulnar joint. **B:** Patient was immobilized in a long arm cast with external electromagnetic stimulation. Three months later, there was evidence of union of the fracture. (Courtesy of Charles T. Price, MD.)

### **Avascular Necrosis of the Radial Head**

The incidence of avascular necrosis is probably higher than recognized. D'Souza et al. ( [16](#)) reported the frequency to be 10% to 20% in their patients, 70% of whom had open reductions. In patients with open reduction, the overall incidence of avascular necrosis was 25%. Jones and Esah ( [42](#)) and Newman ( [67](#)) found that patients with avascular necrosis had poor functional results. It has been our experience, however, that revascularization can occur without any significant functional loss. Only in those in whom a residual functional deficit occurs is avascular necrosis considered a problem ( [Fig. 11-30](#)).



**FIGURE 11-30.** Avascular necrosis with nonunion in a radial head 1 year after open reduction. Both nonunion and avascular necrosis of the radial neck and head are demonstrated. Severe degenerative arthritis developed subsequently. (Courtesy of Richard E. King, MD.)

### **Changes in Carrying Angle (Cubitus Valgus)**

In patients who have fractures of the radial neck, the carrying angle often is 10 degrees greater (increased cubitus valgus) than on the uninjured side ( [16,42](#)). The increase in carrying angle appears to produce no functional deficit and usually is insignificant cosmetically.

### **Vascular Injuries**

No major vascular injuries have been reported as occurring with isolated fractures of the proximal radius.

### **Nerve Injuries**

Partial ulnar nerve injury ( [39](#)) and posterior interosseous nerve injury have been reported as a direct result of the fracture, but most injuries to the posterior interosseous nerves have occurred during surgical exploration ( [16](#)) or with percutaneous pin reduction ( [5](#)). These posterior interosseous nerve injuries usually are transient.

### **Compartment Syndrome**

Peters and Scott ( [76](#)) reported three patients with volar forearm compartment syndrome after minimally displaced or angulated fractures of the radial head. All

required volar fasciotomy.

### **Radioulnar Synostosis**

Proximal synostosis is the most serious complication that can occur after radial head fracture ( [Fig. 11-31](#)). It occurs most often after open reduction of severely displaced fractures ( [31,39,67,97](#)), but it can occur after closed reduction. Delayed treatment seems to increase the likelihood of this complication. All of the three patients reported by Gaston et al. ( [31](#)) were treated more than 5 days after injury.



**FIGURE 11-31.** Radioulnar synostosis. **A:** Surgical intervention with wire fixation was necessary to perform a satisfactory reduction in this patient who had a totally displaced radial neck fracture. **B:** Six weeks after surgery, there was evidence of a proximal radioulnar synostosis. **C:** Radiograph taken 6 months after reduction shows a solid synostosis with anterior displacement of the proximal radius. (Courtesy of R.E. King, M.D.)

### **Myositis Ossificans**

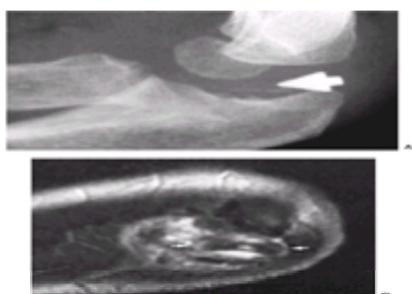
Myositis ossificans is relatively common, but usually does not impair function. Vahvanen ( [110](#)) noted that some myositis ossificans occurred in almost 32% of his patients. In most, it was limited mainly to the supinator muscle. If ossification was more extensive and was associated with a synostosis, the results were poor.

Some variants in the ossification process can resemble a fracture. Most of these involve the radial head, although a step-off can also develop in the normal growth process of the metaphysis. There may be a persistence of the secondary ossification centers of the epiphysis. If unusual ossification centers are seen after an acute elbow injury, comparison views should be obtained.

The diagnosis of a partially or completely displaced fracture of the radial neck may be difficult in the preosseous age group. The only clue may be a little irregularity in the smoothness of the proximal metaphyseal margin ( [Fig. 11-32](#)). Rokito et al. ( [89](#)) reported complete displacement of the radial head in a 5-year-old boy, in whom the only clue on radiography was a very small speck of ossification in the elbow joint ( [Fig. 11-33A](#)). The full extent of the injury was not appreciated until the radial head was outlined with MRI ( [Fig. 11-33B](#)).



**FIGURE 11-32.** Preosseous fracture. The only clue to the presence of a fracture of the radial neck with displacement of the radial head was loss of smoothness of the metaphyseal margin ( *arrow*).



**FIGURE 11-33.** Unappreciated diagnosis. **A:** Radiographs of a 5-year-old taken 4 weeks after injury. Despite immobilization in the cast, the patient continued to have swelling and very limited motion. There is a small flake of ossification between the capitellum and the olecranon ( *arrow*). The proximal aspect of the radial neck is also somewhat irregular. There is continued effusion, as manifested by displaced fat pads. **B:** Because of the persistent effusion and suspected intraarticular fragments, magnetic resonance imaging was performed and demonstrated that the unossified radial head was lying distal and somewhat posterior to the capitellum ( *arrows*). An open reduction was performed and the patient regained a satisfactory range of forearm motion and was pain free at 1-year follow-up. (From Rokito SE, Anticevic D, Strongwater AM, et al. Chronic fracture-separation of a radial head in a child. *J Orthop Trauma* 1995;9:260; with permission.)

### **Osteomyelitis**

A rare case of hematogenous osteomyelitis after a closed fracture of the radial neck was reported by Veranis et al. ( [111](#)). The initial diagnosis was not appreciated despite the fact that the child had fever and continuous pain after the fracture.

### **Malunion**

Failure to reduce a displaced and angulated proximal radius in a young child often results in an angulated radial neck with subsequent incongruity of both the proximal radioulnar joint and the radiocapitellar joint ( [Fig. 11-34](#)). Partial physeal arrest also can create this angulation ( [Fig. 11-15](#)).



**FIGURE 11-34.** Angulation. **A:** Injury film showing 30 degrees of angulation and 30% lateral translocation of a radial neck fracture in a 10-year-old. **B:** Radiographic appearance of the proximal radius taken about five months later, showing lateral angulation of the neck. **C:** Lateral view showing the anterior relationship of the radial neck with proximal migration. At this point the patient had full supination and pronation but a clicking sensation with forearm rotation in the area of the radial head. **D:** 3-D reconstruction showing the incongruity of the proximal radiocapitellar joint. (Courtesy of Vince Mosca, MD.)



**FIGURE 11-15.** Angular stress deformity: anterior angulation of the radial head and neck in a 12-year-old baseball pitcher. There is evidence of some disruption of the normal growth of the anterior portion of the physis (*black arrow*). The capitellum also demonstrates radiographic signs of osteochondritis dissecans (*white arrow*). (Courtesy of Kenneth P. Butters, MD.)

In our experience, this malunion, because of the incongruity of the radiocapitellar joint, often results in erosion of the articular surface of the capitellum, with subsequent degenerative joint disease. In the English literature, there is little information about using osteotomies of the radial neck to correct this deformity.

#### Summary

In summary, results after open reduction of radial head fractures are less favorable than those after closed reduction. However, fractures requiring open reduction usually are the result of a more severe injury. Closed reductions with angulations of up to 45 degrees produce clinical results as good as those in which a more anatomic reduction is achieved by operative methods. The surgeon should warn the parents of the likelihood of residual loss of motion after open reduction. Whenever possible, internal fixation should be avoided.

## FRACTURES OF THE PROXIMAL ULNA (OLECRANON)

### Fractures Involving the Proximal Apophysis

#### Incidence

“Separation of the olecranon epiphysis is the rarest form of epiphyseal detachment” (77). This quote from Poland's 1898 textbook on epiphyseal fractures is appropriate even today. Only 16 acute fractures in which apophyseal involvement is mentioned can be found in the recent English literature (34,77,91,96). In addition to these acute injuries in children, seven have been described in young adults in whom there has been persistence of the physal line (46,73,95,107). In the French literature, Bracq (12) described 10 patients in whom the fracture was just distal and parallel to the apophyseal line and then crossed it at the articular surface.

#### Anatomy

##### The Ossification Process

At birth, the metaphysis of the proximal ulna extends only to the mid-portion of the semilunar notch. At this age, the leading edge of the metaphysis usually is perpendicular to the long axis of the olecranon (Fig. 11-35A, Fig. 11-35B). As ossification progresses, the leading edge of the metaphysis becomes more oblique. The anterior margin migrates proximally and extends to almost three fourths of the semilunar notch by 6 years of age. At this point, some of the physis and cartilage still extends distally to include the coronoid process (Fig. 11-35C). A secondary center of ossification never develops in the coronoid process. Just before the development of the secondary center of ossification of the epiphysis of the olecranon, the leading edge of the metaphysis develops a well-defined sclerotic margin (91). Ossification of the olecranon develops in the area of the triceps insertion at approximately 9 years of age (91) (Fig. 11-35D). Once this secondary ossification begins, the tongue of the physal and epiphyseal cartilage that extends up to include the coronoid process disappears (77). All that remains of this tongue is a fine line of articular cartilage.



**FIGURE 11-35.** Olecranon ossification. **A:** Limits of the border of ossification at birth, 8 years, and 12 years. **B:** Lateral view of olecranon at 6 months of age. The proximal margin is perpendicular to the long axis of the ulna. **C:** Lateral view of the olecranon at 6 years of age. The proximal margin is oblique. **D:** Secondary ossification center developing in the olecranon in a 10-year-old. A sclerotic border has developed on the proximal metaphyseal margin. **E:** Bipartite secondary ossification center. The larger center is the traction center (*open arrow*). The smaller, more proximal center is the articular center (*white arrow*). **F:** Before complete fusion, a partial line remains (*arrow*),bordered by a sclerotic margin.

## Bipartite Centers

The secondary ossification center of the olecranon may be bipartite (79) (Fig. 11-35E). The major center lies within the tip of the olecranon and is enveloped by the triceps insertion. This was referred to by Porteous (79) as a *traction center*. The second and smaller center, an articular center, lies under the proximal fourth of the articular surface of the semilunar notch.

## Closure Process

Fusion of the epiphysis with the metaphysis, which progresses from anterior to posterior, occurs at approximately 14 years of age. Often a posterior aspect of the physal line can be mistaken for a fracture. This line is always surrounded by a well-defined sclerotic margin (91) (Fig. 11-35F). Rarely, the physal line persists into adulthood (46,73,107), usually in athletes who have used the extremity in high-performance throwing activities (17,83,95,109,116). The chronic tension forces applied across the apophysis are believed to prevent its normal closure.

## Patella Cubiti

Occasionally, a separate ossification center called a *patella cubiti* develops in the triceps tendon at its insertion on the tip of the olecranon (105). This ossicle is completely separate and can articulate with the trochlea. It usually is unilateral, unlike the persistent secondary ossification centers, which are more likely to be bilateral and familial. Zeitlin (121) believed that the patella cubiti was a traumatic ossicle rather than a developmental abnormality.

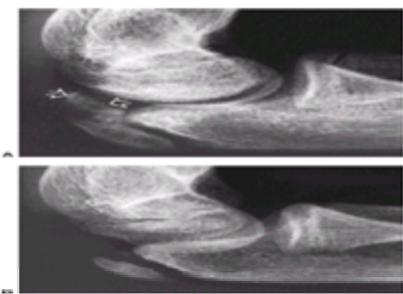
## Signs and Symptoms

### Clinical: Swelling Plus Defect

The primary clinical findings are tenderness over the olecranon and localized soft tissue swelling. If the fragment is completely displaced, there is a lack of active extension of the elbow. A palpable defect may be present between the apophysis and the proximal metaphysis. Poland (77) described the crepitus between the fragments as being muffled because cartilage covers the fractured surfaces.

### Radiography: Proximal Metaphyseal Displacement

The radiographic diagnosis may be difficult before ossification of the olecranon apophysis. The only clue may be a displacement of the small ossified metaphyseal fragment (Fig. 11-36). The diagnosis may have to be based only on the clinical signs of tenderness over the epiphyseal fragment. If there is any doubt about the degree of displacement, injection of radiopaque material into the joint may delineate the true nature of the fracture.



**FIGURE 11-36.** Apophysitis. **A:** Chronic stimulation with irregular ossification of the articular apophyseal center (arrows) in a basketball player who practiced dribbling 3 hours per day. **B:** Normal side for comparison.

## Mechanism of Injury

The fact that much of the triceps expansion inserts on the metaphysis distal to the physis probably accounts for the rarity of this injury. Only a few reports mention the mechanism of these physal injuries. In most of the fractures reported by Poland (77), three of which were confirmed by amputation specimens, the force of the injury was applied directly to the elbow. The force may be applied indirectly, producing an avulsion type of injury.

In our experience, this fracture usually is due to avulsion forces across the apophysis occurring with the elbow flexed, similar to the more common flexion metaphyseal injuries.

Children with osteogenesis imperfecta (usually the tarda form) seem especially predisposed to this injury (18,63).

Stress fractures of this apophysis are not uncommon in athletes (especially baseball players) who place considerable recurrent tension forces across the apophysis (71). Stress injuries also have been reported in elite gymnasts (54) and tennis players (83). If the recurring activity persists, a symptomatic malunion can develop (73,83,107,109,116).

## Classification

Injuries to the apophysis of the olecranon can be one of three types (Table 11-6).

Type I: Apophysitis

Type II: Incomplete stress fracture

Type III: Complete fractures

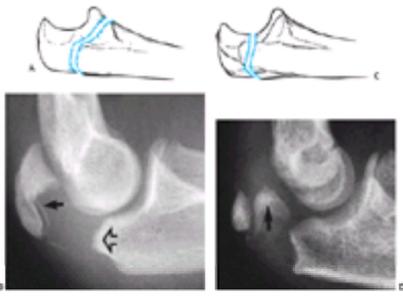
A. Pure apophyseal avulsions

B. Apophyseal-metaphyseal combinations

### TABLE 11-6. CLASSIFICATION OF APOPHYSEAL INJURIES OF THE OLECRANON

The first type is a simple apophysitis in which there is irregularity in the ossification process of the secondary ossification center (17,54) (Fig. 11-37A). In addition,

there may be some widening of the apophyseal line.



**FIGURE 11-37.** Apophyseal avulsions. Pure apophyseal avulsions. **A:** The fracture follows the contour of the apophyseal line. **B:** The distal fracture line is in the shape of the apophyseal line (*open arrow*) with a small metaphyseal flake attached to the apophysis (*solid arrow*). Apophyseal-metaphyseal combination. **C:** The fracture line follows the line of tension stress. **D:** A large portion of the metaphysis (*arrow*) is often with the proximal metaphyseal fragment.

The second type is more of an incomplete stress fracture that involves primarily the apophyseal line, with widening and irregularity ( [Fig. 11-37B](#)). There may be some small adjacent cyst formation, but usually the architecture of the secondary ossification center is normal. These injuries are seen primarily in sports requiring repetitive extension of the elbow, such as baseball pitching ( [71](#)), tennis ( [83](#)), or gymnastics ( [54](#)).

The third type involves a complete avulsion. There appear to be two separate patterns in which the apophysis is completely avulsed. Pure apophyseal avulsions occur in younger children as a pure fracture through the apophyseal plate ( [Fig. 11-37A](#), [Fig. 11-37B](#)). In some of his amputation specimens, Poland ( [77](#)) found that the proximal apophyseal fragment included the distal tongue, which extends up to the coronoid process. Apophyseal-metaphyseal combination fractures usually occur in older children. Large metaphyseal fragments may be attached to the apophysis ( [Fig. 11-37C](#), [Fig. 11-37D](#)). Grantham and Kiernan ( [34](#)) likened it to a Salter-Harris type II physeal injury, in some instances involving a small metaphyseal fragment. Proximal displacement of the fragment by the triceps may be the only radiographic clue to this type of injury.

### Treatment

Because of the few fractures described, there is no standard method of treatment. In fractures with significant displacement, treatment most often must be open reduction with internal fixation using a combination of axial pins and tension-band techniques ( [34,77,96](#)) ( [Fig. 11-38](#)).



**FIGURE 11-38.** Operative treatment of an apophyseal fracture. **A:** Postoperative radiograph of the patient seen in [Fig. 11-40D](#) who was stabilized with small Steinmann pins alone. **B:** Five months later, there is continued growth of the traction center and the articular center has become ossified (*arrow*). **C:** One year after injury, the apophysis is partially avulsed a second time. The two secondary ossification centers are now fused. **D:** Three months after the second fracture, the fracture gap has filled in, producing a normal olecranon.

There has been some concern about applying compressive forces across the apophysis because this may lead to growth arrest. In our experience, fusion of the apophysis to the metaphysis is accelerated, but in all apophyseal injuries we have treated there was already a well-developed ossification of the apophysis itself. In addition, the apophysis of the olecranon appears to be like that of the greater trochanter (i.e., most of the growth proximally is appositional rather than lengthwise across the apophyseal plate itself). As a result, we have not found any functional shortening of the olecranon because of the early fusion of the apophysis to the metaphysis ( [Fig. 11-38D](#)).

Young children who sustain injuries before the development of the secondary ossification center may have some deformity on radiographs, especially if the center primarily is affected ( [Fig. 11-39](#)). Although there may be shortening of the olecranon, it does not appear to produce functional problems. There are no reports of the effects of this injury in very young children or infants, in whom there is very little proximal migration of the metaphyseal margin.



**FIGURE 11-39.** Proximal olecranon fracture. **A:** Comminuted fracture of the proximal olecranon from a direct blow to the elbow in an 8-year-old boy. This fracture was treated nonoperatively. **B:** Radiograph 18 months later shows cessation of the proximal migration of the metaphyseal margin and a lack of development of a secondary ossification center. Despite this arrest of the apophysis, the patient had a full range of elbow motion.

Most stress injuries respond to simple rest from the offending activity. However, a chronic stress fracture can proceed to a symptomatic nonunion that requires operative intervention. Simple application of a compressive internal fixation device alone across the nonunion often is sufficient ( [54](#)). Supplemental bone grafting may be necessary to achieve union ( [46,73](#)).

## AUTHORS' PREFERRED METHOD OF TREATMENT

### Undisplaced Injuries

For apophysitis and undisplaced stress fractures, we have the patient cease the offending activity. However, during this period of rest, the patient, usually a high-performance athlete, should maintain upper extremity strength with a selective muscle exercise program. In addition, he or she must maintain cardiovascular condition with a program of appropriate aerobic exercises.

When a persistent nonunion of the apophyseal line in an older child (usually an adolescent) does not demonstrate healing in a reasonable period of time with simple rest, we place a cannulated compression screw across the apophysis to stimulate healing.

### Displaced Fractures

For displaced fractures, if there is minimal displacement and a satisfactory closed reduction can be achieved with the elbow extended, we usually use immobilization in a long arm cast with the elbow extended. However, we rarely find this to be possible. Occasionally, percutaneous pinning can be done, if there is anatomic reduction on the AP and lateral radiographs.

Completely displaced fractures are treated operatively using a tension-band technique. In young children, we use small Steinmann or Kirschner pins. The tension band is a strong absorbable suture of one of the polyglycolic acid substances. Alternatively, standard 16- to 18-gauge wire can be used in older adolescents. Patients with large ossification centers are treated the same as those with metaphyseal fractures.



## ASSOCIATED INJURIES AND COMPLICATIONS

### Spur Formation

Smith (96) noted that overgrowth of the epiphysis proximally sometimes occurred with spur formation. In some patients these proximal spurs became symptomatic and had to be removed.

### Nonunion

The cause and treatment of nonunion were discussed in the previous section.

### Apophyseal Arrest

Apophyseal arrest has not been reported. If it does occur, it appears to have no significant effect on elbow function ( Fig. 11-39).

### Metaphyseal Fractures of the Olecranon

#### Incidence

Isolated metaphyseal fractures of the olecranon are relatively rare ( Table 11-7). They often are associated with other fractures about the elbow. In the combined series of 4,684 elbow fractures reviewed, there were 230 olecranon fractures reported, for an incidence of 4.9%. This agrees with the incidence of 4% to 6% in the major series reported (24,57,70). Only 10% to 20% of the total fractures reported in these series were considered displaced significantly enough to require surgical intervention.

Age distribution: first decade, 25%; second decade, 25%; third decade 50%

Peak age: 5-10 yr

Extremity predominance: left (55%)

Sex predominance: male (65%)

Associated elbow injuries: 20%

Requiring surgical intervention: 19%

**TABLE 11-7. INCIDENCE OF METAPHYSEAL FRACTURES OF THE OLECRANON**

The rarity of this injury and the relative unimportance given to it are reflected by the paucity of reports in the literature. Reports dealing only with fractures of the olecranon in children did not appear in the English literature until 1975. Since then there have been six reports, totaling 302 patients ( 30,34,57,66). When fractures of the olecranon for all age groups are considered, 25% occur in the first decade and another 25% in the second decade ( 48). During the first decade, the peak age for this type of fracture is between age 5 and 10 years (35,66). Twenty percent had an associated fracture or dislocation of the elbow region, most involving the proximal radius. Only 10% to 20% were classified as being displaced enough to require surgical intervention.

#### Anatomic Considerations

Because the olecranon is a metaphyseal area, the cortex is relatively thin, allowing for the development of greenstick-type fracture deformities. The periosteum is immature and thick, which may prevent the degree of separation seen in adults. Likewise, the larger amount of epiphyseal cartilage in children may serve as a cushion to lessen the effects of a direct blow to the olecranon.

In the production of supracondylar fractures, ligamentous laxity in this age group tends to force the elbow into hyperextension when the child falls on the outstretched upper extremity. This puts a compressive force across the olecranon and locks it into the fossa in the distal humerus, where it is protected. An older person, whose elbow does not go into hyperextension, is more likely to fall with the elbow semiflexed. This unique biomechanical characteristic of the maturing olecranon predisposes it to different fracture patterns than those in adults.

#### Signs and Symptoms

Flexion injuries cause soft tissue swelling over the olecranon. The abrasion or contusion associated with a direct blow to the posterior aspect of the elbow may provide a clue as to the nature of the injury. If there is wide separation, a defect may be palpable between the fragments. In addition, there may be weakness or even lack of active extension of the elbow. This latter finding may be difficult to evaluate in an anxious young child with a swollen elbow.

On radiography, the fracture lines associated with flexion injuries usually are perpendicular to the long axis of the olecranon. This differentiates them from the residual

physeal line, which is oblique and directed proximal and anterior (91). In extension injuries, the longitudinally directed greenstick fracture lines may be difficult to appreciate. The radiograph should be carefully studied to look for associated injuries of the proximal radius or distal humerus.

### Classification

Papavasiliou et al. (70) defined two major groups of olecranon fractures in which the fracture line is either intraarticular or extraarticular. Subclassifications in each group are determined by the degree of displacement.

We prefer to classify these fractures based on the mechanism of injury (Table 11-8): those associated with flexion injuries, those associated with extension injuries, and shear injuries. Extension injuries are further divided into varus and valgus patterns.

- Group A: Flexion injuries
- Group B: Extension injuries
  - 1. Valgus pattern
  - 2. Varus pattern
- Group C: Shear injuries

TABLE 11-8. CLASSIFICATION OF METAPHYSEAL FRACTURES OF THE OLECRANON

### Mechanism of Injury

Three main mechanisms produce metaphyseal olecranon fractures, depending on whether the elbow is flexed or extended at the time of injury. First, in injuries occurring with the elbow flexed, posterior tension forces play an important role. Second, in injuries in which the fracture occurs with the elbow extended, a varus or valgus bending stress is placed across the olecranon, which is responsible for the typical fracture pattern. Third, a less common mechanism involves a direct blow to the elbow that produces an anterior bending or shear force across the olecranon. In this type, the tension forces are concentrated on the anterior portion of the olecranon.

### Flexion Injuries

A fall with the elbow semiflexed places considerable tension forces across the posterior aspect of the olecranon process. Proximally, the triceps applies a force to the tip of the olecranon process. Distally, there is some proximal pull by the insertion of the brachialis muscle. Thus, the posterior cortex is placed in tension. This tension force alone, if applied rapidly enough and with sufficient force, may be enough to cause the olecranon to fail at its mid-portion (Fig. 11-40). A direct blow applied to the posterior aspect of the stressed olecranon makes it more vulnerable to failure. With this type of mechanism, the fracture line usually is transverse and perpendicular to the long axis of the olecranon (Fig. 11-41). Because the fracture extends into the articular surface of the semilunar notch, it is classified as intraarticular.



FIGURE 11-40. Mechanism of flexion injuries. **Center:** In the flexed elbow, a tension force develops on the posterior aspect of the olecranon (*small double arrow*) because of the pull of the brachialis and triceps muscles (*large arrows*). **Right:** Failure occurs on the tension side, which is posterior as a result of the muscle pull or a direct blow to the prestressed posterior olecranon.



FIGURE 11-41. Radiograph of flexion injury showing greater displacement on the posterior surface.

The degree of separation of the fracture fragments depends on the magnitude of the forces applied versus the integrity of the soft tissues. The low incidence of displaced olecranon fractures indicates that the soft tissues are quite resistant to these avulsion forces. In flexion injuries, there are relatively few associated soft tissue injuries or fractures (66).

### Extension Injuries

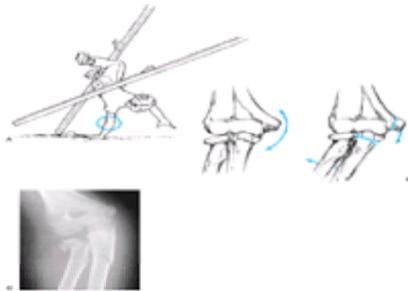
Because the ligaments are more flexible in children, the elbow tends to become hyperextended when a child breaks a fall with the outstretched upper extremity. In this situation, the olecranon may become locked into its fossa of the distal humerus. If the elbow is forced into extreme hyperextension, usually the supracondylar area fails. If, however, the major direction of the force across the elbow is abduction or adduction, a bending movement is created across the olecranon. Most of this movement is concentrated in the distal portion of the olecranon. Because the olecranon is composed of metaphyseal bone, fractures here are usually greenstick type with multiple longitudinal fracture lines (Fig. 11-42). Most of these fracture lines are linear and remain extraarticular. In addition, because the fulcrum of the bending force is more distal, many of the fracture lines may extend distal to the coronoid process into the proximal ulnar shaft regions. The major deformity of the olecranon

with this type of fracture pattern usually is an angulated greenstick type of fracture.



**FIGURE 11-42.** **A:** Anteroposterior view of a linear greenstick fracture line (*arrow*) in the medial aspect of the olecranon. **B:** Lateral view showing the posterior location of the fracture line (*arrow*)

Many of these fractures are associated with other injuries in the elbow region, which are determined by whether the bending force is directed toward varus or valgus. If a child falls with the forearm in supination, the carrying angle tends to place a valgus stress across the elbow. The result may be a greenstick fracture of the ulna with an associated fracture of the radial neck or avulsion of the medial epicondylar apophysis ( [Fig. 11-43](#)). If the fracture involves the radial neck, Bado ( [4](#)) classified it as an equivalent of the type I Monteggia lesion.



**FIGURE 11-43.** Valgus pattern of an extension fracture. **A:** A fall with the elbow extended places a valgus stress on the forearm. **B:** With increased valgus, there can be a greenstick fracture of the olecranon with or without a fracture of the radial neck or avulsion of the medial epicondylar apophysis. **C:** Radiograph of a valgus extension fracture of the olecranon with an associated fracture of the radial neck.

If the body falls against the inner aspect of the elbow or if the forearm is pronated, a varus force may be placed across the elbow ( [Fig. 11-44](#)). The major injury associated with this varus force is a partial or total lateral dislocation of the radial head. Bado ( [4](#)) classified this as a type III Monteggia lesion. In this type of fracture, the posterior interosseous nerve may be injured.

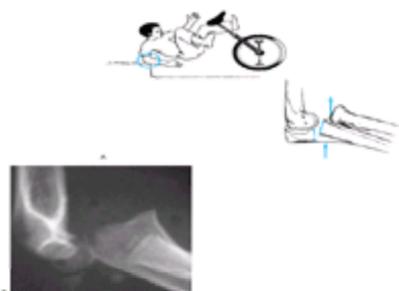


**FIGURE 11-44.** Varus pattern of an extension fracture. **A:** A fall against the extended elbow places a varus stress on the forearm. **B:** The result is a greenstick fracture of the olecranon with a lateral dislocation of the radial head (type III Monteggia lesion). **C:** Radiograph of a varus extension injury showing subluxation of the radial head and greenstick fracture of the ulna (*arrows*).

### Shear Injuries

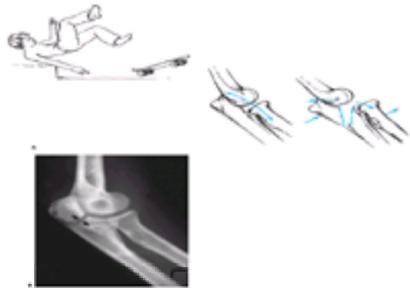
Anterior tension failure is a rare injury that can occur when a direct blow to the proximal ulna causes it to fail with an anterior tension force; the integrity of the proximal radioulnar joint is maintained.

The most common type of shear injury occurs when a shear force is applied directly to the posterior aspect of the olecranon and the distal fragment is displaced anteriorly ( [Fig. 11-45](#) and [Fig. 11-46](#)). The intact proximal radioulnar joint is displaced with the distal fragment. In this type of injury, the elbow may be either flexed or extended when the direct shear force is applied to the posterior aspect of the olecranon.



**FIGURE 11-45.** Flexion shear injuries. **A:** Fracture with the elbow flexed. The direct blow to the distal portion of the posterior olecranon causes the fracture to fail in tension of the anterior surface. The intact proximal radioulnar joint is displaced anteriorly. **B:** Radiograph of a flexion shear injury showing the distal fragments that are

displaced anteriorly as a unit.



**FIGURE 11-46.** Extension shear injuries. **A:** Fracture with the elbow extended. If the elbow is extended when the direct blow is applied to the posterior aspect of the elbow, the olecranon fails in tension but with an oblique or transverse fracture line (arrows). **B:** With the elbow extended, the initial failure is in the anterior articular surface (arrows).

These fractures can be considered as failing in tension, with the force concentrated along the anterior cortex. This is opposite to the tension failure occurring on the posterior aspect of the cortex in the more common flexion injuries. In these shear types, the fracture line may be transverse or oblique. The differentiating feature from the more common flexion injury is that the proximal olecranon fragment is often not displaced proximally. Instead, it is the distal fragment that becomes displaced anteriorly by the brachialis and biceps muscles. The thick posterior periosteum usually remains intact.

Newman (67) described one patient in whom a shear force was directed medially; the radial neck was fractured and the radial head remained with the proximal fragment.

### Associated Injuries

Associated injuries occur in 48% to 77% of patients with olecranon fractures (14,35,70,104), especially varus and valgus greenstick extension fractures, in which the radial head and neck are most commonly injured (Fig. 11-43). Other associated injuries include fractures of the ipsilateral radial shaft (101), Monteggia type I lesions with fractures of both the ulnar shaft and olecranon (69), and fractures of the lateral condyle (12) (Fig. 11-47).



**FIGURE 11-47.** **A:** Undisplaced fracture of the lateral condyle (arrow) associated with a varus greenstick fracture of the olecranon. **B:** Lateral view showing greenstick fractures in the olecranon (solid arrows) and a nondisplaced fracture of the lateral condyle (open arrows).

### Treatment

The mechanism of injury can serve as a useful guide in choosing the proper treatment method.

### Flexion Injuries

Flexion injuries are the most common olecranon fractures. Most are minimally displaced and require only simple immobilization with the elbow in no more than 75 to 80 degrees of flexion (Fig. 11-48). Even if the fracture is severely displaced, immobilization in full or partial extension usually allows the olecranon to heal satisfactorily (24,96,122).



**FIGURE 11-48.** Simple immobilization of a flexion injury. **A:** Injury film, lateral view, showing minimal displacement. **B:** Three weeks later, some displacement has developed. Periosteal new bone is seen along the posterior border of the olecranon (arrow). Healing was complete with a normal range of motion. (Courtesy of Jesse C. DeLee, MD.)

If the fracture is significantly displaced or comminuted, open reduction with some method of internal fixation usually is required. Recommended fixation devices vary from catgut or absorbable suture (56) to an axial screw (52), to tension-band wiring with axial pins (25,34,56,87,90,96,122) or a plate (103). This usually allows sufficient fixation to begin early motion. No significant growth disturbances from internal fixation have been reported.

Murphy et al. (64) compared the failure of various fixation devices to rapid loading: (a) figure-of-eight wire alone, (b) cancellous screw alone, (c) AO tension band, and (d) a cancellous screw with a figure-of-eight wire combination. The cancellous screw alone or figure-of-eight wire alone was by far the weakest. The greatest resistance to failure was provided by the combination of a screw plus figure-of-eight wire, followed closely by the AO tension-band fixation. In their clinical evaluation

of patients, comparing the AO tension band and combination screw and figure-of-eight wire, they found that more clinical problems were associated with the AO technique (65). The main problem with the AO technique is the subcutaneous prominence of the axial wires (53). To prevent proximal migration of these wires, Montgomery (62) devised a method of making eyelets in the proximal end of the wires through which he passes the figure-of-eight fixation wire.

### Extension Injuries

Treatment of extension injuries requires both adequate realignment of the angulation of the olecranon and treatment of the secondary injuries. Often in varus injuries, correction of the alignment of the olecranon also reduces the radial head. The olecranon angulation must be corrected with the elbow in extension. This locks the proximal olecranon into the olecranon fossa of the humerus so that the distal angulation can be corrected at the fracture site with a valgus force applied to the forearm. Occasionally, in extension fractures, complete separation of the fragments requires open reduction and internal fixation ( Fig. 11-49).



**FIGURE 11-49.** Open reduction of a valgus extension injury. **A:** Anteroposterior view of the injury film shows complete displacement of the radial head. **B:** Lateral view also shows the degree of displacement of the olecranon fracture. This patient required surgical intervention with internal fixation to achieve a satisfactory reduction.

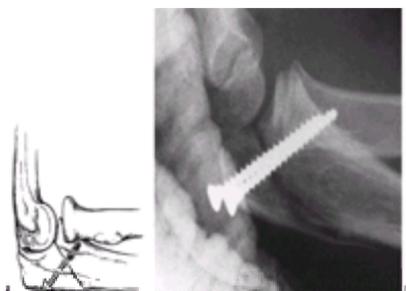
Zimmerman (122) reported that the original angulation tends to redevelop in some fractures. If it is a varus fracture, the proximal ulna or olecranon may drift back into varus, which can cause a painful subluxation of the radial head. A delayed osteotomy of the proximal ulna or olecranon may be necessary if the angulation becomes significant.

### Shear Injuries

In anterior shear fractures, the key to management is recognition that the distal fragment is displaced anteriorly and the posterior periosteum remains intact. The intact posterior periosteum can serve as an internal tension band to facilitate reduction.

Some of these fractures are best reduced in flexion so that the posterior periosteum serves as a compressive force to maintain the reduction. Smith (96) reported treatment of this fracture using an overhead sling placed to apply a posteriorly directed force against the proximal portion of the distal fragment. The weight of the arm and forearm helps supplement the tension-band effect of the posterior periosteum.

If the periosteum is torn or early motion is desirable, Zimmerman (122) advocated internal fixation of the two fragments with an oblique screw perpendicular to the fracture line (Fig. 11-50).



**FIGURE 11-50.** Operative treatment of extension shear fractures. **A:** If the periosteum is insufficient to hold the fragments apposed, an interfragmental screw may be used, as advocated by Zimmerman (122). **B:** An extension shear type of fracture secured with two oblique interfragmentary screws.

## AUTHORS' PREFERRED METHOD OF TREATMENT

We use a classification based on the mechanism of injury in deciding on the method of treatment ( Table 11-8).

- Type I: Avulsion of the tip of the coronoid process only
- Type II: A single or comminuted fragment involving  $\leq 50\%$  of the coronoid process
- Type III: A single or comminuted fragment involving  $>50\%$  of the coronoid process

**TABLE 11-9. CLASSIFICATION OF FRACTURES OF THE CORONOID PROCESS**

### Flexion Injuries

**Nonoperative.** We immobilize most nondisplaced flexion injuries with the elbow in 5 to 10 degrees of flexion for approximately 3 weeks. It is important to obtain radiographs of these fractures at approximately 5 to 7 days in the cast to ensure that there has not been any significant displacement of the fragment.

**Operative: Tension Band.** To determine which injuries need internal fixation, we usually palpate the fracture for a defect or flex the elbow to determine the integrity of the posterior periosteum. If the fragments separate with either of these procedures, they are considered unstable and are fixed internally so that active motion can be started as soon as possible.

We prefer a modification of the tension-band technique. Originally, we used the standard AO technique with axial Kirschner wires or Steinmann pins and figure-of-eight stainless steel as the tension band ( Fig. 11-51A). Because removal of the wire often required reopening the entire incision, we now use an absorbable suture for the figure-of-eight tension band. Number 1 polydioxanone (PDS) suture, which is slowly absorbed over a few months, is ideal ( Fig. 11-51B). When rigid internal fixation is applied, rapid healing at the fracture site produces internal stability before the integrity of the PDS is lost. We prefer Kirschner wires in patients who are very young and have very little ossification of the olecranon apophysis ( Fig. 11-38). If the axial wires become a problem, they can be removed with only a small incision. Most recently, we have used a combination of an oblique cortical screw with PDS as the tension band ( Fig. 11-51C, Fig. 11-51D) and have been pleased with the results. In the past we had to remove almost all the axial wires; very few of the screws have become symptomatic enough to require removal. Occasionally, the tension-band wire technique with 16- or 18-gauge wire must be used if compression is necessary.



**FIGURE 11-51.** Internal tension-band techniques. **A:** Standard AO technique with stainless steel wire (see Fig. 11-40D for original injury film). The wire can be prominent in the subcutaneous tissues. **B:** Axial wires plus polydioxanone (PDS) 6 weeks after surgery. **C:** A displaced flexion-type injury in an 11-year-old boy. There is complete separation of the fracture fragments. **D:** This fracture was secured with a cancellous lag screw plus PDS suture. The screw engages the anterior cortex of the coronoid process. The PDS is passed through the olecranon through a separate drill hole (open arrow), crossed in a figure-of-eight manner over the fracture site, and then tied over the neck of the screw.

### Extension Injuries

In extension injuries, adequate relaxation should be achieved to allow a forceful manipulation of the olecranon while it is locked in its fossa in extension. Because this is a greenstick fracture, we try to achieve a slight overcorrection to prevent the development of reangulation. These fractures should be followed closely and possibly remanipulated in 1 to 2 weeks if the original angulation recurs. The associated injuries are treated as if they were isolated fractures.

Most shear fractures can be treated nonoperatively. We usually immobilize them in enough hyperflexion to hold the fragments together, providing the posterior periosteum is intact (Fig. 11-52).



**FIGURE 11-52.** Shear injuries. **A:** Flexion pattern: radiograph of the patient seen in Fig. 11-45A after the elbow was flexed. The intact posterior periosteum acted as a tension band and held the fracture reduced. **B:** Radiograph taken 4 weeks after surgery shows new bone formation under the intact periosteum (arrows) on the dorsal surface of the olecranon. **C:** Extension pattern: radiograph of patient with an extension shear injury showing an increase in the fracture gap (arrows) (see also Fig. 11-45B). **D:** Because the dorsal periosteum and cortex were intact, the fracture gap (arrows) closed with flexion of the elbow.

If the periosteum is torn, an oblique screw is an excellent way to secure this type of fracture pattern ( Fig. 11-50). It also can be used if there has been considerable swelling, which prevents the elbow from being hyperflexed enough to use the posterior periosteum as a tension band.

### Postoperative Care

The elbow is placed at a 70- to 80-degree angle and a long arm cast is placed. The cast can be bivalved and the anterior portion removed if there is excessive swelling. This cast or posterior splint is used for 3 weeks, at which time active range of motion is begun.



## ASSOCIATED INJURIES AND COMPLICATIONS

Relatively few complications arise from the olecranon fracture itself.

### Irreducibility

An and Loder (1) reported inability to reduce the fracture in one of their patients because the proximal fragment had been entrapped in the joint.

### Nonunion

Nonunion is unusual. A true nonunion from a fracture should not be confused with a rare form of congenital pseudarthrosis of the ulna ( Fig. 11-53). In the latter condition, there is usually no known antecedent trauma.



**FIGURE 11-53.** Congenital pseudarthrosis of the olecranon in a 9-year-old girl who had limited elbow extension and no antecedent trauma. The edges of the bone were separated by thick fibrous tissue. (Courtesy of Michael J. Rogal, MD.)

### Delayed Union

Delayed radiographic union is usually asymptomatic (56). In Mathews' series (56), one fracture that was internally secured with catgut ultimately progressed to a nonunion. Despite this, the patient had only a 10-degree extension lag and grade 4 triceps strength. The presence of an accessory ossicle such as a patella cubiti should not be confused with nonunion.

### Compartment Syndrome

Mathews (56) reported one patient with Volkmann ischemic contracture after an undisplaced linear fracture in the olecranon.

### Nerve Injuries

Transient neurapraxia of the ulnar nerve has been reported. Zimmerman (122) reported ulnar nerve irritation from the development of a pseudarthrosis of the olecranon where inadequate fixation was used.

### Elongation

One complication recently observed has been elongation of the tip of the olecranon. This was seen in a young boy who had a partial avulsion of the tip of the apophysis (Fig. 11-54). Because of the delayed union, the apophysis became elongated to the point that it limited extension. This proximal overgrowth of the tip of the apophysis has also been reported in other simple olecranon fractures after routine open reduction and internal fixation (70).



**FIGURE 11-54.** **A:** Injury film showing partial avulsion of the tip of the olecranon apophysis (arrow). **B:** Radiograph taken 4 years later showed a marked elongation and irregular ossification of the apophysis. (Courtesy of Joel Goldman, MD.)

### Loss of Reduction

Apparently stable fractures treated with external immobilization must be watched closely for a delayed loss of reduction that can result in a significant loss of elbow function (Fig. 11-55).



**FIGURE 11-55.** Loss of reduction. **A:** Lateral radiograph of what appeared to be a simple undisplaced fracture (arrow) of the olecranon in a 13-year-old girl. **B:** On the anteroposterior radiograph, the fracture also appears undisplaced. The mild lateral subluxation of the radial head was not appreciated initially. **C:** Radiographs taken 5 months later showed further lateral subluxation with resultant incongruity of the elbow joint. (Courtesy of Richard W. Williamson, MD.)

### Fractures of the Coronoid Process

#### Anatomy

Up to age 6 years, the coronoid process is mostly epiphyseal and physeal cartilage at the distal end of a tongue extending from the apophysis of the olecranon. The coronoid process does not develop a secondary center of ossification, but instead is ossified by the advancing edge of the metaphysis (Fig. 11-35).

#### Incidence

The incidence in some major series of elbow fractures varies from less than 1% to 2% (57).

Because most fractures of the coronoid process occur with dislocations of the elbow, it seems logical that they would happen in older children. However, in a review of 23 coronoid fractures in children, Bracq (11) found that the injuries occurred in two peak age groups: one was between 8 and 9 years of age and the other between 12 and 14 years.

Although most coronoid fractures are associated with elbow dislocations, fractures of the olecranon, medial epicondyle, and lateral condyle also have been reported (11). The fracture of the coronoid may be part of a greenstick olecranon fracture (i.e., the extension-type metaphyseal fracture; Fig. 11-56). Isolated coronoid fractures are thought to be due to pure avulsion by the brachialis or secondary to an elbow dislocation that reduced spontaneously, which usually is indicated by hemarthrosis and a small avulsion of the tip of the olecranon process (Fig. 11-57).



**FIGURE 11-56.** Fracture of the coronoid (arrows) as part of an extension valgus olecranon fracture pattern. There was an associated fracture of the radial neck. Both the neck fracture and the distal portion of the coronoid process demonstrate periosteal new bone formation (open arrows).

### Diagnosis

The radiographic diagnosis of this fracture often is difficult because on the lateral view the radial head is superimposed over the coronoid process. Evaluation of a minimally displaced fracture may require oblique views (13) (Fig. 11-58). The radiocapitellar view (Fig. 11-3) provides good visualization of the profile of the coronoid process.

In young children, in whom the coronoid process contains considerable cartilage, an unusual flap injury has been described (8). In this rare injury, the elbow is dislocated and the articular surface is flipped back into the joint. The only clue to this fracture may be the presence of a small flake of bone in the anterior part of the joint on the lateral radiograph.

### Classification

Regan and Morrey (82) classified coronoid fractures into three types based on the amount of the coronoid process involved (Table 11-9). This classification was found to be useful in predicting the outcome and in determining the treatment. Type I fractures involve only the tip of the process (Fig. 11-57); type II fractures involve more than just the tip but less than 50% of the process (Fig. 11-58), and type III fractures involve more than 50% of the process.



**FIGURE 11-57.** Lateral radiograph of an 11-year-old boy who injured his left elbow. The presence of displaced anterior and posterior fat pads, plus a small fracture of the coronoid (arrow), indicates a probable partially dislocated elbow as the primary injury.



**FIGURE 11-58.** **A:** Based on this original lateral radiograph, a 12-year-old boy with a swollen elbow was thought to have a fracture of the radial neck (arrow). **B:** With an oblique view, it is now obvious that the fragment is from the coronoid process. **C:** Five months later, the protuberant healed coronoid process (arrow) is seen on this radiocapitellar view.

### Treatment

Treatment is based on the degree of displacement or the presence of elbow instability. The associated injuries are also a factor in treatment.

Regan and Morrey (81,82) determined that types I and II could be treated with early motion if there were no contradicting associated injuries.

In initially immobilizing these fractures, if they are associated with an elbow dislocation, the elbow should be placed in approximately 100 degrees of flexion, with the forearm in full supination (11). Occasionally, in isolated avulsion fractures in which the fragment is attached distally, reduction is better accomplished with the elbow

held in some extension. In these rare cases, it is believed that the brachialis muscle may be an aid in reducing the fragment in extension ( [102](#)).

Regan and Morrey ([81](#)) found that the elbow was often unstable in type III fractures. As a result, they thought that the fragment must be reduced and secured with internal fixation. In their review, satisfactory results could be expected with type I and II injuries, but in only 20% of type III injuries.

## AUTHORS' PREFERRED METHOD OF TREATMENT

We usually treat coronoid fractures with early motion, much as we do elbow dislocations. The presence of a coronoid fracture alerts us to be especially thorough in looking for other injuries. In children, it is rarely necessary to replace the fragments surgically. If there is a large fragment and marked displacement, open reduction and internal fixation can be done through a Henry anterior approach to the elbow. The fragment can be fixed with a mini-fragment screw or sewn in place through two drill holes to the posterior aspect of the ulna.

## ASSOCIATED INJURIES AND COMPLICATIONS

Complications are rare. In fractures with a large fragment (type III), the elbow may be unstable and prone to recurrent dislocations. Nonunion with the production of a free fragment in the joint occurs rarely in children ( [96](#)).

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## CHAPTER REFERENCES

1. An HS, Loder RT. Intra-articular entrapment of a displaced olecranon fracture. *Orthopedics* 1989 ; 12:289–291.
2. Anderson TE, Breed AL. A proximal radial metaphyseal fracture presenting as wrist pain. *Orthopedics* 1982;5:425–428.
3. Angelov AA. New method for treatment of the dislocated radial neck fracture in children. In: Chapchal G, ed. *Fractures in children*. New York: Georg Thieme, 1981:192–194.
4. Bado JL. The Monteggia lesion. *Clin Orthop* 1967;50:71–86.
5. Baehr FH. Reduction of separated upper epiphysis of the radius. *N Engl J Med* 1932;24:1263–1266.
6. Bernstein SM, McKeever P, Bernstein L. Percutaneous pinning for radial neck fractures. *J Pediatr Orthop* 1993;13:84–88.
7. Biyani A, Mehara A, Bhan S. Percutaneous pinning of radial neck fractures. *Injury* 1994;25:169–171.
8. Blasier RD. Intra-articular flap fracture of the olecranon in a child. *J Bone Joint Surg Am* 1989;71:945–947.
9. Blount WP. Fractures in children. *Instr Course Lect* 1950;7:194–202.
10. Boyd HB, Altenberg AR. Fractures about the elbow in children. *Arch Surg* 1944;49:213–224.
11. Bracq H. Fracture de l'apophyse coronoïde. *Rev Chir Orthop* 1987;73:472–473.
12. Bracq H. Fractures de l'olecrane. *Rev Chir Orthop* 1987;73:469–471.
13. Brodeur AE, Silberstein JJ, Graviss ER. *Radiology of the pediatric elbow*. Boston: GK Hall, 1981.
14. Burge P, Benson M. Bilateral congenital pseudoarthrosis of the olecranon. *J Bone Joint Surg Br* 1987;69:460–462.
15. Carl AL, Ain MC. Complex fracture of the radial neck in a child: an unusual case. *J Orthop Trauma* 1994;8:255–257.
16. D'Souza S, Vaishya R, Klenerman L. Management of radial neck fractures in children: a retrospective analysis of 100 patients. *J Pediatr Orthop* 1993;13:232–238.
17. Danielson LG, Hedlund ST, Henricson AS. Apophysitis of the olecranon: a report of four cases. *Acta Orthop Scand* 1983;54:777–778.
18. Di Cesare PE, Sew-Hoy A, Krom W. Bilateral isolated olecranon fractures in an infant as presentation of osteogenesis imperfecta. *Orthopedics* 1992;15:741–743.
19. Dooley JF, Angus PD. The importance of elbow aspiration when treating radial head fractures. *Arch Emerg Med* 1991;8:117–121.
20. Dormans JP, Rang M. Fractures of the olecranon and radial neck in children. *Orthop Clin North Am* 1990;21:257–268.
21. Dougall AJ. Severe fracture of the neck of the radius in children. *J R Coll Surg Edinb* 1969;14:220.
22. Ellman H. Osteochondrosis of the radial head. *J Bone Joint Surg Am* 1972;54:1560.
23. Ellman H. Anterior angulation deformity of the radial head. *J Bone Joint Surg Am* 1975;57:776–778.
24. Fahey JJ. Fractures of the elbow in children. *Instr Course Lect* 1980;17:13–46.
25. Fan G-F, Wu C-C, Shin C-H. Olecranon fractures treated with tension band wiring techniques. Comparisons among three different configurations. *Chang Keng I Hsueh* 1993;16:231–238.
26. Fogarty EE, Blake NS, Regan BF. Fracture of the radial neck with medial displacement of the shaft of the radius. *Br J Radiol* 1983;56:486–487.
27. Fowles JV, Kassab MT. Observations concerning radial neck fractures in children. *J Pediatr Orthop* 1986;6:51–57.
28. Fraser KE. Displaced fracture of the proximal end of the radius in a child. a case report of the deceptive appearance of a fragment that had rotated 180. *J Bone Joint Surg Am* 1995;77:782–783.
29. Futami T, Tsukamoto Y, Itoman M. Percutaneous reduction of displaced radial neck fractures. *J Shoulder Elbow Surg* 1995;4:162–167.
30. Gaddy BC, Strecker WB, Schoenecker PL. Surgical treatment of displaced olecranon fractures in children. *J Pediatr Orthop* 1997;17:321–324.
31. Gaston SR, Smith FM, Boab OD. Epiphyseal injuries of the radial head and neck. *Am J Surg* 1953;85:266–276.
32. Gille P, Mourot M, Aubert F, et al. Fracture par torsion du col du radius chez l'enfant. *Rev Chir Orthop* 1978;64:247–248.
33. Gonzalez-Herranz P, Alvarez-Romera A, Burgos J, et al. Displaced radial neck fractures in children treated by closed intramedullary pinning (Metaizeau technique). *J Pediatr Orthop* 1997;17:325–331.
34. Grantham SA, Kiernan HA. Displaced olecranon fractures in children. *J Trauma* 1975;15:197–204.
35. Graves SC, Canale ST. Fractures of the olecranon in children: long-term follow-up. *J Pediatr Orthop* 1993;13:239–241.
36. Greenspan A, Norman A. The radial head-capitellum view: useful technique in elbow trauma. *AJR Am J Roentgenol* 1982;138:1186–1188.
37. Greenspan A, Norman A, Rosen H. Radial head-capitellum view in elbow trauma: clinical application and radiographic-anatomic correlation. *AJR Am J Roentgenol* 1984;143:355–359.
38. Hall-Craggs MA, Shorvon PJ, Chapman M. Assessment of the radial head-capitellum view and the dorsal fat-pad sign in acute elbow trauma. *AJR Am J Roentgenol* 1985;145:607–609.
39. Henrikson B. Isolated fracture of the proximal end of the radius in children. *Acta Orthop Scand* 1969;40:246–260.
40. Irshad F, Shaw NJ, Gregory RJ. Reliability of fat-pad sign in radial head/neck fractures of the elbow. *Injury* 1997;28:433–435.
41. Jeffrey CC. Fracture of the head of the radius in children. *J Bone Joint Surg Br* 1950;32:314–324.
42. Jones ERW, Esah M. Displaced fracture of the neck of the radius in children. *J Bone Joint Surg Br* 1971;53:429–439.
43. Kaplan EB. Surgical approach to the proximal end of the radius and its use in fractures of the head and neck of the radius. *J Bone Joint Surg* 1941;23:86–92.
44. Kaufman B, Rinott MG, Tanzman M. Closed reduction of fractures of the proximal radius in children. *J Bone Joint Surg Br* 1989;71:66–67.
45. Key AJ. Survival of the head of the radius in a child after removal and replacement. *J Bone Joint Surg* 1946;28:148–149.
46. Kovach JI, Baker BE, Mosher JF. Fracture-separation of the olecranon ossification center in adults. *Am J Sports Med* 1985;13:105–111.
47. Landin LA. Fracture patterns in children. *Acta Paediatr Scand Suppl* 1983;54:192.
48. Landin LA, Danielsson LG. Elbow fractures in children: an epidemiological analysis of 589 cases. *Acta Orthop Scand* 1986;57:309.
49. Lazar RD, Waters PM, Jaramillo D. The use of ultrasonography in the diagnosis of occult fracture of the radial neck: a case report. *J Bone Joint Surg Am* 1998;80:1361–1364.
50. Leung KS, Tse PYT. A new method of fixing radial neck fractures: brief report. *J Bone Joint Surg Br* 1989;71:326–327.
51. Lindham S, Hugasson C. Significance of associated lesions including dislocation of fracture of the neck of the radius in children. *Acta Orthop Scand* 1979;50:79–83.
52. Mac Lennan A. Common fractures about the elbow joint in children. *Surg Gynecol Obstet* 1937;64:447–453.
53. Macko D, Azabo RM. Complications of tension band wiring of olecranon fractures. *J Bone Joint Surg Am* 1985;67:1396–1401.
54. Maffulli N, Chan D, Aldridge MJ. Overuse injuries of the olecranon in young gymnasts. *J Bone Joint Surg Br* 1992;74:305–308.
55. Manoli A II. Medial displacement of the shaft of the radius with a fracture of the radial neck. *J Bone Joint Surg Am* 1979;61:788–789.
56. Mathews JG. Fractures of the olecranon in children. *Injury* 1981;12:207–212.
57. Maylahn DJ, Fahey JJ. Fractures of the elbow in children. *JAMA* 1958;166:220–228.
58. McBride ED, Monnet JC. Epiphyseal fracture of the head of the radius in children. *Clin Orthop* 1960;16:264–271.
59. McCarthy SM, Ogden JA. Radiology of postnatal skeletal development. *Skeletal Radio*. 1982;9:17–26.
60. Metaizeau JP, Lascombes P, Lemelle JL, et al. Reduction and fixation of displaced radial neck fractures by closed intramedullary pinning. *J Pediatr Orthop* 1993;13:355–360.
61. Metaizeau JP, Prevot J, Schmitt M. Reduction et fixation des fractures et décollements epiphysaires de la tete radiale par broche centromedullaire. *Rev Chir Orthop* 1980;66:47–49.
62. Montgomery RJ. A secure method of olecranon fixation: a modification of tension band wiring technique. *J R Coll Surg Edinb* 1986;31:179–182.
63. Mudgal CS. Olecranon fractures in osteogenesis imperfecta: a case report. *Acta Orthop Belg* 1992;58:453–456.
64. Murphy DF, Greene WB, Dameron TB. Displaced olecranon fractures in adults. *Clin Orthop* 1987;224:215–223.
65. Murphy DF, Greene WB, Gilbert JA, et al. Displaced olecranon fractures in adults. biomedical analysis of fixation methods. *Clin Orthop* 1987;224:210–214.
66. Newell RLM. Olecranon fractures in children. *Injury* 1975;7:33–36.
67. Newman JH. Displaced radial neck fractures in children. *Injury* 1977;9:114–121.
68. O'Brien PI. Injuries involving the radial epiphysis. *Clin Orthop* 1965;41:51–58.

69. Olney BW, Menelaus MB. Monteggia and equivalent lesions in childhood. *J Pediatr Orthop* 1989;9:219–223.
70. Papavasiliou VA, Beslikas TA, Nenopoulos S. Isolated fractures of the olecranon in children. *Injury* 1987;18:100–102.
71. Pappas AM. Elbow problems associated with baseball during childhood. *Clin Orthop* 1982;164:30–41.
72. Patterson RF. Treatment of displaced transverse fractures of the neck of the radius in children. *J Bone Joint Surg* 1934;16:695–698.
73. Pavlov H, Torg JS, Jacobs B, et al. Nonunion of olecranon epiphysis: two cases in adolescent baseball pitchers. *AJR Am J Roentgenol* 1981;136:819–820.
74. Pelto K, Hirvensalo E, Bostman O, et al. Treatment of radial head fractures with absorbable polyglycolide pins: a study on the security of the fixation in 38 cases. *J Orthop Trauma* 1994;8:94–98.
75. Pesudo JV, Aracil J, Barcelo M. Leverage method in displaced fractures of the radial neck in children. *Clin Orthop* 1982;169:215–218.
76. Peters CL, Scott SM. Compartment syndrome in the forearm following fractures of the radial head or neck in children. *J Bone Joint Surg Am* 1995;77:1070–1074.
77. Poland J. *A practical treatise on traumatic separation of the epiphyses*. London: Smith, Elder & Co, 1898.
78. Pollen AG. *Fractures and dislocations in children*. Baltimore: Williams & Wilkins, 1973.
79. Porteous CJ. The olecranon epiphyses. *Proc J Anat* 1960;94:286.
80. Radomisli TE, Rosen AL. Controversies regarding radial neck fractures in children. *Clin Orthop* 1998;353:30–39.
81. Regan W, Morrey B. Fractures of the coronoid process of the ulna. *J Bone Joint Surg Am* 1989;71:1348–1354.
82. Regan W, Morrey BF. Classification and treatment of coronoid process fractures. *Orthopedics* 1992;15:845–848.
83. Retrum RK, Wepfer JF, Olen DW, et al. Case report 355: delayed closure of the right olecranon epiphysis in a right-handed tournament-class tennis player. *Skeletal Radio*. 1986;15:185–187.
84. Robert M, Moulies D, Longis B, et al. Les fractures de l'extremite superieure du radius chez l'enfant. *Chir Pediatr* 1986;27:318–321.
85. Rodriguez-Merchan EC. Percutaneous reduction of displaced radial neck fractures in children. *J Trauma* 1994;37:812–814.
86. Rodriguez-Merchan EC. Displaced fractures of the head and neck of the radius in children: open reduction and temporary transarticular internal fixation [Review article]. *Orthopedics* 1991;14:697–700.
87. Roe SC. Tension band wiring of olecranon fractures: a modification of the AO technique [Letter; comment]. *Clin Orthop* 1994;308:284–286.
88. Rogers SL, Mac Ewan DW. Changes due to trauma in the fat plane overlying the supinator muscle: a radiologic sign. *Radiology* 1969;92:954–958.
89. Rokito SE, Anticevic D, Strongwater AM, et al. Case report and review of the literature: chronic fracture-separation of the radial head in a child. *J Orthop Trauma* 1995;9:259–262.
90. Rowland SA, Burkhart SS. Tension band wiring of olecranon fractures. A modification of the AO technique. *Clin Orthop* 1992;277:238–242 [comment in *Clin Orthop* 1994;308:284–286].
91. Saberstein MJ, Brodeur AE, Graviss ER, et al. Some vagaries of the olecranon. *J Bone Joint Surg* 1981;63:722–725.
92. Scullion JE, Miller JH. Fracture of the neck of the radius in children: prognostic factors and recommendations for management. *J Bone Joint Surg Br* 1985;67:491.
93. Sessa S, Lascombes P, Prevot J, et al. Fractures of the radial head and associated elbow injuries in children. *J Pediatr Orthop B* 1996;5:200–209.
94. Silberstein MJ, Brodeur AE, Graviss ER. Some vagaries of the radial head and neck. *J Bone Joint Surg Am* 1982;64:1153–1157.
95. Skak SV. Fracture of the olecranon through a persistent physis in an adult: a case report. *J Bone Joint Surg Am* 1993;75:272–275.
96. Smith FM. *Surgery of the elbow*. Philadelphia: WB Saunders, 1972.
97. Steele JA, Graham HK. Angulated radial neck fractures in children: a prospective study of percutaneous reduction. *J Bone Joint Surg Br* 1992;74:760–764.
98. Steinberg EL, Golomb D, Salama R, et al. Radial head and neck fractures in children. *J Pediatr Orthop* 1988;8:35–40.
99. Strachan JCH, Ellis BW. Vulnerability of the posterior interosseous nerve during radial head resection. *J Bone Joint Surg Br* 1971;53:320–323.
100. Strong ML, Kropp M, Gillespie R. Fracture of the radial neck and proximal ulna with medial displacement of the radial shaft. Report of two cases. *Orthopedics* 1989;12:1577–1579.
101. Suprock MD, Lubahn JD. Olecranon fracture with unilateral closed radial shaft fracture in a child with open epiphysis. *Orthopedics* 1990;13:463–465.
102. Tanzman M, Kaufman B. Fracture of the coronoid process of the ulna requiring reduction in extension. *J Hand Surg [Am]* 1988;13:741–742.
103. Teasdall R, Savoie FH, Hughes JL. Comminuted fractures of the proximal radius and ulna. *Clin Orthop* 1993;292:37–47.
104. Theodorou SD, Ierodiconou MN, Roussis N. Fracture of the upper end of the ulna associated with dislocation of the head of the radius in children. *Clin Orthop* 1988;228:240–249.
105. Thijn CJP, van Ouwkerk WPL, Scheele PM, et al. Unilateral patella cubiti: a probable post-traumatic disorder. *Eur J Radio*. 1992;14:60–62.
106. Tibone JE, Stoltz M. Fracture of the radial head and neck in children. *J Bone Joint Surg* 1981;63:100–106.
107. Torg JS, Moyer R. Nonunion of a stress fracture through the olecranon epiphyseal plate observed in an adolescent baseball pitcher. *J Bone Joint Surg Am* 1977;59:264–265.
108. Tullos HS, King JW. Lesions of the pitching arm in adolescents. *JAMA* 1972;220:264–271.
109. Turtel AH, Andrews JR, Schob CJ, et al. Fractures of unfused olecranon physis: a re-evaluation of this injury in three athletes. *Orthopedics* 1995;18:390–394.
110. Vahvanen V. Fracture of the radial neck in children. *Acta Orthop Scand* 1978;49:32–38.
111. Veranis N, Laliotis N, Vlachos E. Acute osteomyelitis complicating a closed radial fracture in a child: a case report. *Acta Orthop Scand* 1992;63:341–342.
112. Vocke AK, Von Laer L. Displaced fractures of the radial neck in children: long-term results and prognosis of conservative treatment. *J Pediatr Orthop B* 1998;7:217–222.
113. Vostal O. Fracture of the neck of the radius in children. *Acta Chir Orthop Traumatol Cech* 1970;37:294–301.
114. Ward WT, Williams JJ. Radial neck fracture complicating closed reduction of a posterior elbow dislocation in a child: case report. *J Trauma* 1991;31:1686–1688.
115. Wedge JH, Robertson DE. Displaced fractures of the neck of the radius. *J Bone Joint Surg Br* 1982;64:256.
116. Wilkerson RD, Johns JC. Non-union of an olecranon stress fracture in an adolescent gymnast: a case report. *Am J Sports Med* 1990;18:432–434.
117. Wood SK. Reversal of the radial head during reduction of fractures of the neck of the radius in children. *J Bone Joint Surg Br* 1969;51:707–710.
118. Woods GW, Tullos HS, King JW. The throwing arm: elbow joint injuries. *J Sports Med* 1973;1:43–47.
119. Wray CC, Harper WM. The upside-down radial head: brief report. *Injury* 1989;20:241–242.
120. Wright PR. Greenstick fracture of the upper end of the ulna with dislocation of the radio-humeral joint or displacement of the superior radial epiphysis. *J Bone Joint Surg Br* 1963;45:727–731.
121. Zeitlin A. The traumatic origin of accessory bones at the elbow. *J Bone Joint Surg* 1935;17:933–938.
122. Zimmerman H. Fractures of the elbow. In: Weber BG, Brunner C, Freuler F, eds. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980.

## MONTEGGIA FRACTURE–DISLOCATION IN CHILDREN

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JOSE F. DE LA GARZA

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Monteggia fracture–dislocations are a complex of injuries that represent a link between injuries of the forearm and the elbow. The original description of an anterior dislocation of the proximal radius and fracture of the proximal ulna has been expanded to include dislocations of the radial head in multiple directions and combinations of injuries to the ulna and distal humerus. The mechanisms of injury and protocols for treatment are similar in the different types of Monteggia fracture–dislocations. Fortunately, if the injury is recognized, the fracture course follows closely that of the forearm fractures rather than the more complex injuries of the child's elbow. Treatment results are better in children than in adults.

## HISTORICAL BACKGROUND

### Original Description

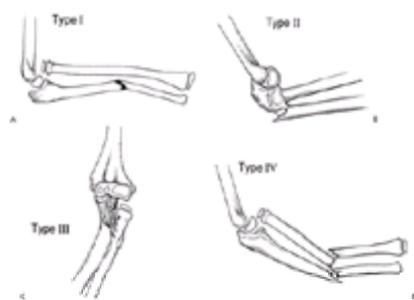
Giovanni Batista Monteggia, a surgical pathologist and public health official in Milan, first described the Monteggia fracture in 1814. He observed the original two injuries in cadavers and provided the description, "A traumatic lesion distinguished by a fracture of the proximal third of the ulna and an anterior dislocation of the proximal epiphysis of the radius." Monteggia also described the injury in a young girl who was treated late with external compression, which failed to reduce the radial head, leading to a loss of elbow function. The complexity of this fracture–dislocation was not fully appreciated until the classic monograph coining the term *Monteggia lesion* was written in English by Jose Luis Bado (7).

### Defining The Monteggia Lesion

Jose Luis Bado, while Chief Director of the Instituto de Ortopedia y Traumatologia in Montevideo, Uruguay, studied proximal radial dislocations in association with fractures of the ulna, writing extensively on this subject in the late 1950s. In this monograph, Bado defined the Monteggia lesion as an association of a radial head fracture or dislocation with a fracture of the middle or proximal ulna.

### Classification

Numerous classification schemes have been developed and are useful in various situations, but Bado's classification has stood the test of time. It is used widely when a series of injuries are analyzed and new variations of Monteggia fractures are discovered. Bado's classification divides Monteggia fractures into four types of true Monteggia lesions and equivalent lesions (Fig. 12-1).

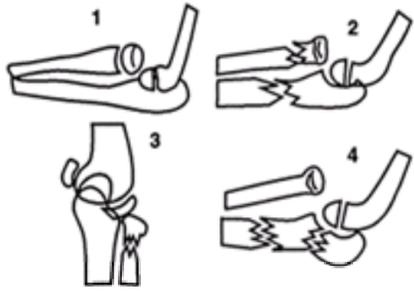


**FIGURE 12-1.** Bado's classification. **A:** Type I (anterior dislocation): the radial head is dislocated anteriorly; and the ulna has a short oblique or greenstick fracture in the diaphyseal or proximal metaphyseal area. **B:** Type II (posterior dislocation): the radial head is posteriorly and posterolaterally dislocated; the ulna is usually fractured in the metaphysis in children. **C:** Type III (lateral dislocation): there is lateral dislocation of the radial head with a greenstick metaphyseal fracture of the ulna. **D:** Type IV (anterior dislocation with radius shaft fracture): the pattern of injury is the same as with a type I injury, with the inclusion of a radius shaft fracture below the level of the ulnar fracture.

### True Lesions

#### Type I

A type I lesion (Fig. 12-2) is described as an anterior dislocation of the radial head with a fracture of the ulnar diaphysis. The ulnar fracture can be at any level, but usually is described as midshaft. Most Monteggia injuries in children are of this type (approximately 70% in most series).



**FIGURE 12-2.** Type I Monteggia equivalents. **1** Isolated anterior radial head dislocation. **2** Ulnar and proximal radius fractures, including fractures of the radial neck. **3** Isolated radial neck fractures. **4** Elbow (ulnohumeral) dislocation with or without fracture of the proximal radius.

### Type II

Posterior dislocation of the radial head with associated ulnar diaphyseal or metaphyseal fracture with posterior angulation is defined as a type II lesion. This lesion is uncommon in children, with most early series recognizing this as a fracture of adults only. Peiró et al. (82) reported one type II lesion in their series of 25 acute Monteggia lesions in children. In 1965, Pavel et al. (81), in their review of a series of posterior Monteggia fracture dislocations, reported only one type II lesion in a child. In both series, the children were older, approximately 13 years of age. Although reported as high as 6% by some authors, the overall prevalence of type II fractures in children is 3% when all studies are considered. In their series of 36 consecutive Monteggia fracture–dislocations in children, Ring and Waters found only 1 (3%) type II injury (94).

### Type III

A type III lesion consists of a lateral or anterolateral dislocation of the radial head associated with a fracture of the ulnar metaphysis. The ulnar metaphyseal fracture usually is a greenstick type in children. This is the second most common type of Monteggia fracture–dislocation in children (23%). This fracture pattern often is associated with radial nerve injuries and a coexisting intraarticular obstruction to radial head reduction, which requires surgical reduction of the radial head.

Extension of the ulnar fracture into the olecranon leads some investigators to classify this type of fracture–dislocation as a different lesion (11,18,46,116). If there is no concurrent radioulnar disassociation with the proximal ulnar fracture and radiocapitellar dislocation, the injury should be considered a fracture–dislocation of the elbow, and not strictly a Monteggia fracture. This lesion is included by others in series of Monteggia fractures, classifying them by the direction of the radial head dislocation, usually lateral and anterolateral (36,77,79,127).

### Type IV

Type IV Monteggia lesions, characterized by anterior dislocation of the radial head with fractures of the ulna and radius at the same level, or with the radial fracture distal to the ulnar fracture, are relatively rare in children, with an average incidence of 1% (Fig. 12-1D). Wiley and Galey (127), Olney and Menelaus (77), and Dormans and Rang (28) each reported an average frequency of approximately 1% for type IV lesions. Ring and Waters, however, reported 4 type IV fractures (11%) in 36 consecutive Monteggia fracture–dislocations in children; they recorded any otherwise unclassified fracture of both bones of the forearm with proximal radioulnar dislocation as a type IV lesion (94).

### Monteggia Equivalent Injuries

Bado classified certain injuries as equivalents to the classic or true Monteggia lesions because of their associated mechanism of injuries, radiographic pattern, or methods of treatment.

#### Type I Equivalents

Type I equivalents include an isolated anterior dislocation of the radial head; however, minimal plastic deterioration at the ulna accompanies the radial head dislocation, so “isolated” dislocation probably is a misnomer. Also included in this subclassification is the pulled elbow syndrome. If one considers the typical mechanism of longitudinal traction with pronation, possibly combined with hyperextension of the elbow joint, the relationship between the pulled elbow syndrome and the type I Monteggia fracture is evident. Other type I equivalent lesions include a fracture of the ulnar diaphysis with fracture of the radial neck. Fracture of the radius and ulnar shafts may be considered a type I equivalent if the radial fracture is above the junction of the middle and proximal thirds. Two additional type I equivalents are fracture of the ulnar diaphysis with either anterior dislocation of the radial head and an olecranon fracture, or posterior dislocation of the ulnohumeral joint with or without fracture of the proximal radius.

#### Type II Equivalents

In his original classification, Bado stated that there were no equivalents to type II Monteggia lesions other than epiphyseal fracture of the radial head or fracture of the radial neck. Considering the mechanism of this injury, as defined by Penrose (84), a posterior elbow dislocation could be included as a type II equivalent in children.

#### Types III and IV Equivalents

Bado stated that there were no equivalents for types III and IV lesions. More recent reviews have reported fracture patterns that, when the mechanism of injury is considered, might be classified as type III or IV equivalents. Ravessoud reported an oblique fracture of the ulna with varus alignment and a displaced lateral condyle fracture in a 13 year-old patient (90). This fracture pattern, which suggests hyperextension of the elbow combined with varus force, is produced by the same mechanism of injury as a type III lesion, as described by Wright (129).

De la Garza described fractures of the distal humerus, ulnar diaphysis, and radial neck, which he classified as equivalent to a type III Monteggia fracture–dislocation because of the mechanism of injury. Bhandari and Jindal reported a type IV variant that included a fracture of the mid-third radial shaft, an anterolaterally angulated fracture of the proximal ulna, and a posterior lateral dislocation of the radial head. A similar lesion was reported by Givon et al. (40).

#### Hybrid Lesion

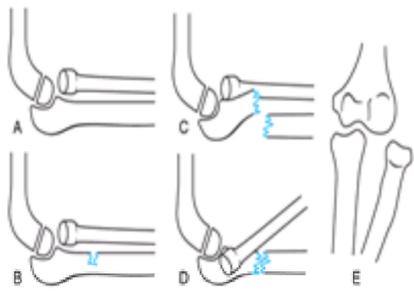
A hybrid lesion similar to a type III Monteggia lesion has been described in which anterior dislocation of the radial head was associated with a fracture of the metaphyseal region of the ulna that extended into the olecranon. Other studies noted the radial head to be dislocated in multiple directions. The treatment and outcome of each fracture are determined by the direction of the radial head dislocation combined with the pattern of the ulnar fracture.

#### Other Classifications

Dormans and Rang (28) extended Bado's classification by adding a type V, the intermittent and habitual dislocation of the radiocapitellar joint and proximal radioulnar joint. Others (76,127) have considered the Bado type IV lesion to be a variant of Bado's type I and have excluded it from their classifications.

Letts et al. (61) devised a classification of Monteggia fractures in children based on both the direction of the radial head dislocation and the type of ulnar fracture (Fig. 12-3). The Bado type I class was subdivided into three subtypes. Letts type A is anterior bowing of the ulna due to plastic deformation with anterior dislocation of the radial head. Type B includes a greenstick fracture of the ulna, and type C has a complete ulnar fracture. Letts types D and E correspond, respectively, to Bado types II

and III.



**FIGURE 12-3.** Pediatric Monteggia lesion classification by Letts. **A:** Anterior dislocation of the radial head with plastic deformation of the ulna. **B:** Anterior dislocation of the radial head with greenstick fracture of the ulna. **C:** Complete fracture of the ulna with anterior dislocation of the radial head. **D:** Posterior dislocation of the radial head with fracture of the ulnar metaphysis. **E:** Lateral dislocation of the radial head and metaphyseal greenstick fracture of the ulna.

## AUTHORS' PREFERRED CLASSIFICATION

We prefer to use Bado's classification in evaluating and treating the Monteggia lesion ( [Table 12-1](#)). Although recognizing the contributions of other researchers, we believe that treatment principles are better determined by the direction of the radial head dislocation than the pattern of the ulnar fracture. Further, it is important not to exclude a type IV lesion from any classification scheme because the recognition and proper management of the radial fracture is important to the success of its treatment.

Type	Dislocation	Fracture
<b>True lesions</b>		
I	Anterior	Metaphysis-diaphysis
II	Posterior	Metaphysis-diaphysis
III	Lateral	Metaphysis
IV	Anterior	Radial diaphysis, ulnar diaphysis
<b>Hybrid Lesion</b>		
	Anterior, posterior, or lateral	Metaphysis or olecranon
Type	Description	
<b>Equivalent lesions</b>		
I	Isolated dislocation of radial head	
	Radial neck fracture (isolated)	
	Radial neck fracture in combination with a fracture of the ulnar shaft	
	Radial and ulnar fractures with the radial fracture above the junction of the middle and proximal thirds	
	Fracture of ulnar diaphysis with anterior dislocation of radial head and an olecranon fracture	
II	Posterior dislocation of the elbow	
III	Ulnar fracture with displaced fracture of the lateral condyle	
IV	None described	

**TABLE 12-1.** AUTHORS' CLASSIFICATION OF MONTEGGIA FRACTURE - DISLOCATIONS

We also recognize a hybrid lesion consisting of a radial head dislocation and metaphyseal ulnar fracture extending into the olecranon. In this fracture–dislocation pattern, there may or may not be disassociation of the proximal radioulnar complex. The technique of treatment of the hybrid lesion is the same as for the comparable true Monteggia lesion. This necessitates including these hybrid lesions when considering the recognition and management of Monteggia fracture–dislocations in general.

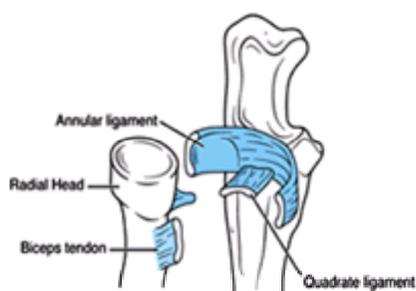
## ANATOMY AND BIOMECHANICS

Certain anatomic relations between the proximal radius and ulna and their surrounding structures must be taken into account in the management of the Monteggia fracture. They are divided into four anatomic categories: ligaments, bones, muscles, and nerves.

### Ligaments

#### *Annular Ligament*

Stability of the proximal radioulnar joint is provided by the surrounding ligaments, primarily the annular ligament. Failure of this structure usually leads to failure of the others. It maintains the position of the radial head within the radial notch of the ulna through the entire range of rotation, becoming tighter in supination because of the shape of the radial head. The substance of the annular ligament is reinforced by the radial collateral ligament at the elbow joint ( [Fig. 12-4](#)).



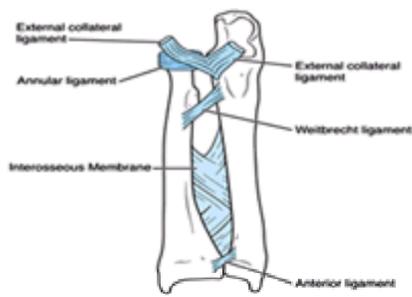
**FIGURE 12-4.** Ligamentous anatomy of the proximal radioulnar joint.

#### *Quadrate Ligament*

The quadrate ligament (52), or ligament of Denucé (27), is located between the radius and ulna distal to the annular ligament ( [Fig. 12-4](#)). It consists of a dense anterior border and a thinner posterior border with an even thinner central portion. The two borders limit rotation and stabilize the radial head at limits of supination and pronation. The anterior border becomes tight in supination, adding stability to the proximal radioulnar joint. Excessive pronation causes the posterior border to fail, producing instability of the radial head.

#### *Oblique Ligament*

The oblique ligament, or ligament of Weitbrecht (126), extends from the ulna proximally to the radius distally (Fig. 12-5). It begins below the radial notch of the ulna and ends just below the biceps tuberosity on the radius. The bow of the radius tightens the oblique ligament when the radius is in supination and provides additional stability to the proximal radioulnar joint.



**FIGURE 12-5.** Ligaments of the forearm. In supination, the annular ligament, quadrate ligament, Weitbrecht ligament, and interosseous membrane are taut, stabilizing the radial head.

### **Interosseous Membrane**

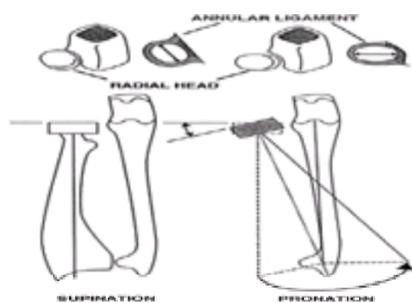
The interosseous membrane is distal and separate from the oblique ligament, with its fibers running in the opposite direction (Fig. 12-5). It acts in concert with the oblique ligament to approximate the radioulnar joint. The fibers of both the oblique and interosseous membranes tighten with supination of the radius.

### **Osseous Relations**

There is little inherent osseous stability to the proximal radioulnar joint. The shape of the radial head contributes to tightness of the ligament as the radius rotates.

### **Shape of the Radial Head**

The head of the radius is elliptic in its cross section (Fig. 12-6). In supination, the long axis of the ellipse is perpendicular to the ulna, causing the annular ligament and the anterior border of the quadrate ligament to stabilize the proximal radioulnar joint further by tightening.



**FIGURE 12-6.** The radial head is an elliptic structure secured by the annular ligament, which allows movement and gives stability. Because of the shape of the radial head, the stability provided by the annular ligament is maximized in supination.

The contact area between the radius and the radial notch is greater in supination because of the broadened surface area of the circumference of the radial head in that position. The portion of the radial head circumference adjacent to the radial notch, when supinated, is less curved and is longer, proximal to distal, than other portions of the circumference. This provides a minimal increase in stability as the ligaments tighten.

### **Bow of the Radius**

In the anatomic position, the apex of the radial bow is lateral (Fig. 12-5). This curvature allows increased range of pronation as the radius rotates along the axis between the proximal and distal radioulnar joints. When the radius is in supination, the bow tightens the oblique and interosseous ligaments and increases proximal radioulnar stability.

### **Muscles**

Whereas the ligaments help stabilize the humeral–radioulnar complex, the muscles play an active role in the mechanism of injury that produces the Monteggia fracture–dislocation.

### **Biceps Brachii**

The biceps, inserting into the biceps tuberosity on the proximal radius, is a major deforming force. It is active in the pathomechanics of the Monteggia fracture–dislocation, violently pulling the proximal radius away from the capitellum as the elbow goes into extension. During treatment, the effect of the biceps must be regulated by elbow flexion, preventing recurrence of the radial head dislocation.

### **Anconeus and Forearm Flexors**

The anconeus and forearm flexors act together to create a radially angulated bow in the ulna in a Monteggia fracture–dislocation. The function of the anconeus in the uninjured person is enigmatic. Electromyographic studies show that it helps stabilize the elbow as it goes into extension, creating a valgus moment at the joint, and during forearm pronation counteracts the varus moment produced by the pronator teres against an intact ulna (9,121).

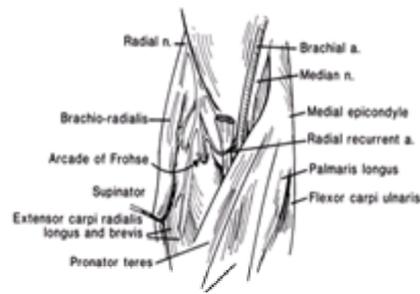
The forearm flexors create a bowstring effect, producing a tendency for the fractured ulna to shorten and angulate radially after reduction (121).

### **Nerves**

#### **Posterior Interosseous Nerve**

There is a close relation between the posterior interosseous nerve and the proximal radius, with the nerve coursing anterior and anterolateral to the radial head and neck on its path through the supinator muscle. With anterolateral dislocations of the radial head, this relation becomes particularly intimate, causing a paresis of the

nerve. In adults, there is a more discrete organization of the proximal border of the superficial head of the supinator, forming the arcade of Frohse ( [103](#)) ([Fig. 12-7](#)). This band is not as well defined in children, which may partially account for the lower incidence of permanent injury to the posterior interosseous nerve in children, as well as the high incidence of resolution once reduction of the radius is obtained ( [104](#)).



**FIGURE 12-7.** Dissection of the forearm at the level of the elbow. (Redrawn from Spinner M. *Injuries to major branches of peripheral nerves of forearm*, 2nd ed. Philadelphia: WB Saunders, 1978; with permission.)

### **Ulnar Nerve**

The ulnar nerve, relatively tethered by the cubital tunnel, can be at risk in Monteggia fractures involving the proximal end of the ulna, particularly type II injuries ( [106](#)).

## **CHARACTERISTICS AND MANAGEMENT OF MONTEGGIA INJURIES**

### **Type I Lesions**

#### **Clinical Findings**

Bado, in his original description, provided an accurate clinical picture of the Monteggia fracture–dislocation. In general, there is fusiform swelling about the elbow. The child has significant pain and cannot move the elbow in either flexion and extension or pronation and supination. There is usually an angular change to the forearm itself, with the apex shifted anteriorly. There may be tenting of the skin or an area of ecchymosis on the volar aspect of the forearm. Later, as the swelling subsides, there still may be an anterior fullness in the cubital fossa. If this lesion is seen late, as in a Monteggia fracture with only plastic deformation of the ulna but complete dislocation of the radial head, there is a persistent anterior prominence at the elbow and loss of full flexion at the elbow. On further observation, it may be noted that the patient cannot extend the digits at the metacarpophalangeal joint or at the interphalangeal joint in the thumb because of a paresis of the posterior interosseous nerve.

#### **Radiographic Evaluation**

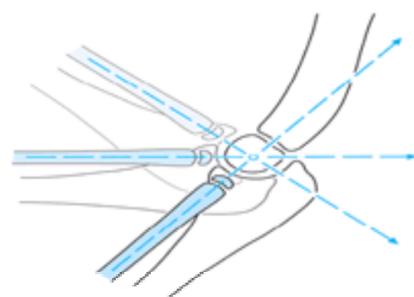
The standard evaluation of a type I Monteggia fracture includes anteroposterior (AP) and lateral radiographs of the forearm. Any disruption of the ulna, especially an apparently innocuous bowing, should alert the physician to look for a disruption of the joints at either end of the forearm, in this case proximally ( [26,53,63](#)).

#### **Radiocapitellar Relation**

The radiocapitellar relation is particularly important and is best defined by a true lateral view of the elbow. This relation in a type I Monteggia fracture may appear normal on an AP radiograph despite obvious disruption on the lateral view ( [Fig. 12-8](#)). If there is doubt regarding the radiocapitellar relation, a true lateral view of the opposite elbow should be obtained for comparison. Smith ( [100](#)) and, later, Storen ( [109](#)) noted that a line drawn through the center of the radial neck and head should extend directly through the center of the capitellum. This alignment should remain intact regardless of the degree of flexion or extension of the elbow ( [Fig. 12-9](#)). In some instances, there is disruption of the radiocapitellar line in a normal elbow. Miles and Finlay ( [69](#)) pointed out that the radiocapitellar line passes through the center of the capitellum only on a true lateral projection. They reported five patients in whom the elbow was clinically normal but the radiocapitellar line appeared disrupted. In analyzing the radiographs, they found that the radiographic projection of the elbow usually was an oblique view or that the forearm was pronated in the radiograph. If this disruption appears on radiographs in a child with an acute injury, however, it is the treating surgeon's responsibility to ensure that it is an insignificant finding.



**FIGURE 12-8.** Anteroposterior view (A) demonstrates an apparently located radial head, but the lateral view (B) shows an anterior dislocation of the radial head. Note the disruption of the radiocapitellar line.



**FIGURE 12-9.** Composite drawing with the elbow in various degrees of flexion. A line drawn down the long axis of the radius bisects the capitulum of the humerus regardless of the degree of flexion or extension of the elbow. (Redrawn from Smith FM. *Children's elbow with injuries: fractures and dislocations*. *Clin Orthop* 1967;50:25-26; with permission.)

### Traumatic Versus Congenital Dislocation

When the radiocapitellar relation is disrupted, radiographic evaluation of the shape of the radial head and neck helps determine the cause of the disruption, especially if there is no history of trauma or the significance of the trauma is questioned. Bucknill ( 20) reviewed the difference between congenital and old traumatic dislocations of the radial head. He believed that McFarland's ( 66) classic description of a atypical deformed radial head with a dysplastic capitellum and concavity of the posterior border of the proximal ulna and the association of paraarticular ossifications probably represented old traumatic dislocations. Lloyd-Roberts and Bucknill ( 64) believed that the congenital nature of some unilateral anterior dislocations remained unproved and that their existence as old traumatic dislocations should be suspected. Lloyd-Roberts acknowledged Caravias ( 22) as recognizing that the existence of a congenital anterior dislocation as a separate entity was doubtful and that true congenital dislocation of the head was indeed rare. When true congenital dislocation occurs, it is usually bilateral and posterior and is often associated with various syndromes such as Ehlers-Danlos, nail-patella, and Silver's syndrome ( 2,64) (Fig. 12-10). Therefore, all isolated anterior and anterolateral dislocations of the radial head, regardless of symptoms, should be considered as having a traumatic origin. Their management should be the same as for known, previously undetected, posttraumatic dislocations of the radial head.



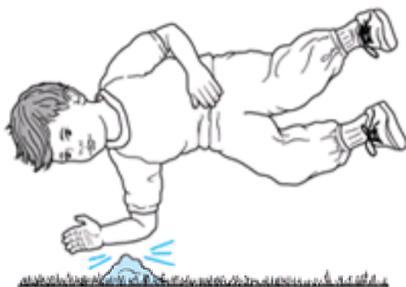
**FIGURE 12-10.** Congenital versus traumatic dislocation. **A:** Anteroposterior (AP) view of the elbow of a 7-year-old who presented with limited forearm rotation. **B:** Lateral radiograph of the same child. Note dysplastic radial head and an attempt at synostosis with the ulna. There is also anterior bowing of the ulna. Note also the hypoplastic capitellum. Is this congenital or old trauma? **C:** AP radiograph of congenital synostosis. **D:** Lateral radiograph of congenital synostosis and posterior dislocation. Note posterior bow of the ulna and hypoplasia of the capitellum. This is congenital. (Interestingly, these are opposite elbows of the same child.)

### Mechanism of Injury

The mechanisms of injury of type I lesions have been studied thoroughly ( 8,19,33,73,75,89,91,102,121). Three separate mechanisms have been developed to describe the production of this injury. Each mechanism probably can occur under specific conditions ( 19,91).

#### Direct Blow Theory

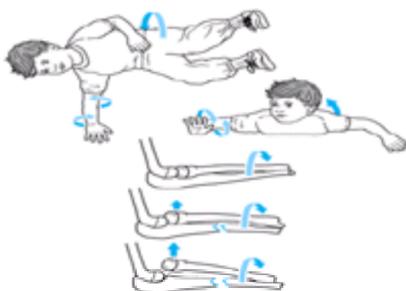
The first theory proposed in the English literature was the direct blow mechanism by Speed and Boyd ( 102) (Fig. 12-11). This was later confirmed by Smith ( 100), but actually this theory was proposed by Monteggia himself ( 70). He noted that the fracture occurs when a direct blow on the posterior aspect of the forearm first produces a fracture through the ulna. Then, either by continued deformation or direct pressure, the radial head is forced anteriorly with respect to the capitellum, causing the radial head to dislocate. Monteggia explained that these injuries sometimes resulted from a blow by a staff or cudgel on the forearm raised to protect the head ( 83). Another eponym for the Monteggia fracture–dislocation, the *parry fracture*, has been mentioned in the literature. During the American Civil War, there was a high incidence of Monteggia fractures owing to direct blows on the forearm received while attempting to parry the butt of a rifle during hand-to-hand combat. The major argument presented against this theory as being the sole mechanism is that in the usual clinical situation there rarely is evidence of a direct blow to the posterior aspect of the forearm, such as a contusion or laceration ( 33,121).



**FIGURE 12-11.** The fracture-dislocation is sustained by direct contact on the posterior aspect of the forearm, either by falling onto an object or by the object striking the forearm. The continued motion of the object forward dislocates the radial head after fracturing the ulna.

#### Hyperpronation Theory

In 1949, Evans ( 33) published his observations regarding the anterior Monteggia fracture. Previous investigators had based their direct blow theory purely on clinical observation, but Evans used cadaveric experiments to support his hypothesis. He demonstrated that hyperpronation of the forearm produced a fracture of the ulna with a subsequent dislocation of the radial head. It was postulated that during a fall, the outstretched hand, initially in pronation, is forced into further pronation as the body twists above the planted hand and forearm (Fig. 12-12). This hyperpronation causes the radius to be crossed over the mid-ulna, resulting in anterior dislocation of the radial head or fracture of the proximal third of the radius and fracture of the ulna. In the patients reported in Evans' article, the ulnar fractures demonstrated a pattern consistent with anterior tension and shear or longitudinal compression. His cadaveric studies, however, showed the ulna fracture pattern to be consistent with a spiral or torsional force. This theory also was supported by Bado ( 7).

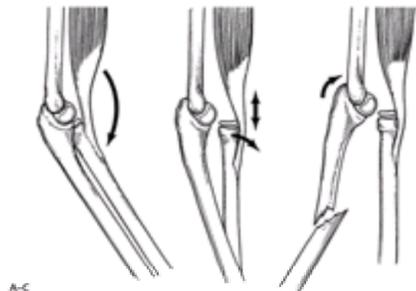


**FIGURE 12-12.** Hyperpronation theory (Evans). Rotation of the patient's body externally forces the forearm into pronation. The ulnar shaft fractures with further rotation, forcibly dislocating the radial head

Two arguments have been used to dispute Evans' mechanism (121). First, the ulnar fracture rarely presents clinically in a spiral pattern; it is usually oblique, indicating an initial force in tension with propagation in shear rather than rotation. Second, Evans' experiments, which were done on totally dissected forearms, did not take into consideration the dynamic muscle forces at play during a fall on an outstretched hand *in vivo*.

### Hyperextension Theory

In 1971, Tompkins (121) analyzed both theories and presented good clinical evidence that the type I Monteggia fracture was caused by a combination of dynamic and static forces. His study postulated three steps in the fracture mechanism (Fig. 12-13): hyperextension, radial head dislocation, and ulnar fracture.



**FIGURE 12-13.** Hyperextension theory. **A:** Hyperextension: forward momentum caused by a fall on an outstretched hand forces the elbow into extension. **B:** Radial head dislocation: the biceps contracts, forcibly dislocating the radial head. **C:** Ulnar fracture: forward momentum causes the ulna to fracture because of tension on the anterior surface.

**Hyperextension.** The patient falls on an outstretched arm with forward momentum, forcing the elbow joint into hyperextension (Fig. 12-13A).

**Radial Head Dislocation.** The radius is first dislocated anteriorly by the violent reflexive contracture of the biceps, forcing the radius away from the capitellum (Fig. 12-13).

**Ulnar Fracture.** Once the radius is dislocated, the weight of the body is transferred to the ulna. Because the radius is usually the main weight-transmitting bone in the forearm, the ulna cannot handle the longitudinal force and subsequently fails in tension (Fig. 12-13C). This tension force produces an oblique fracture line, often with a butterfly fragment, or a greenstick fracture in the ulnar diaphysis or diaphyseal–metaphyseal junction. In addition to the momentum of the injury, the anterior angulation of the ulna results from the pull of the intact interosseous membrane on the distal fragment, causing it to follow the radius. The brachialis muscle causes the proximal ulnar fragment to flex at the elbow.

### Summary of Mechanisms of Injury

The Monteggia lesion can probably occur by any of the three proposed mechanisms in a given situation. We believe that the most common mechanism is a fall on an outstretched hand that forces the elbow into complete extension, locking the olecranon into the humerus. The forearm is in a rotational position of neutral to mid-pronation. As the ulna locks into the humerus, the bending force stresses the radioulnar joint. Because of the relatively pronated position of the joint, the ligamentous restraints are lax, providing only tenuous stability for the radial head. The anterior bending force, combined with a reflexive contraction of the biceps, violently dislocates the radial head anteriorly.

The radioulnar joint and its ligamentous complex are at risk because of the ligamentous laxity and decreased contact area between the proximal radius and ulna created by the rotational attitude of the forearm. At mid-rotation, the short axis of the elliptical radial head is perpendicular to the ulna, causing the annular ligament and the dense anterior portion of the quadrate ligament to be relaxed. The contact area of the proximal radioulnar joint, owing to the shape of the radial head, is also decreased, further reducing the stability of the joint. The ulna, now the main weight-bearing member of the forearm, is loaded by a continued bending moment, causing tension on the anterior cortex and producing failure. The force at the site of failure is propagated in shear at approximately 45 degrees to the long axis of the ulna. This mechanism may produce plastic deformation with an anterior bow, a greenstick fracture, or an oblique fracture pattern, all of which are seen clinically. As the anterior bending movement continues, the vector of the biceps changes, acting as a tether and resisting any further advance of the proximal radius. The distal fragment of the ulna continues to advance, acting as a fulcrum against the radial shaft. The anteriorly directed force of the distal ulnar fragment, combined with the retrograde resistance of the biceps, may create a fracture of the radius, or a type IV Monteggia lesion.

### Treatment

Although most treatment recommendations are based on the Bado classification, Ring and Waters based their treatment choices on the type of ulnar fracture rather than on the Bado type. *Plastic deformation* of the ulna is treated with reduction (with general anesthesia) of the ulnar bow to obtain stable reduction of the radioulnar joint. *Incomplete (greenstick or buckle) fractures* are treated with closed reduction and casting. They suggested that almost all Monteggia fractures in children (types I and III) are most stable when immobilized in 100 to 110 degrees of flexion and full supination. For a *nearly complete* greenstick fracture or one associated with a radial fracture (type IV), intramedullary Kirschner wire is considered for more stability. For *complete transverse* or *short oblique fractures*, which often are in bayonet apposition with malalignment or shortening, Kirschner wires are used to manipulate the proximal fragment into a reduced position and to hold the reduction. Long oblique or comminuted fractures, which may develop shortening and malalignment even with intramedullary fixation, are stabilized with a short plate and screw. Using this treatment protocol, Ring and Waters reported excellent results in all 28 patients treated within 24 hours of injury (Table 12-2). Two poor results occurred in patients who referred late with persistent radial head dislocations.

Type of Ulnar Injury	Treatment
Plastic deformation	Closed reduction of the ulnar bow and cast immobilization
Incomplete (greenstick or buckle) fracture	Closed reduction and cast immobilization
Complete transverse or short oblique fracture	Closed reduction and intramedullary Kirschner wire fixation
Long oblique or comminuted fracture	Open reduction and internal fixation with plate and screws

From Ring D, Jupiter JB, Waters PM. Monteggia fractures in children and adults. *J Am Acad Orthop Surg* 1998;6:215–224; with permission.

**TABLE 12-2. TREATMENT OF MONTEGGIA FRACTURE - DISLOCATIONS IN CHILDREN ACCORDING TO ULNAR INJURY**

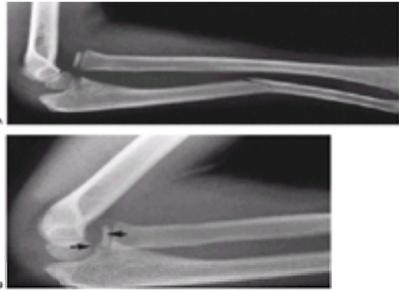
## Nonoperative Treatment

In all series ([4,17,19,28,36,61,77,82,91,92,100,127](#)), anterior Monteggia lesions in children have uniformly good results when treated by manipulative closed reduction.

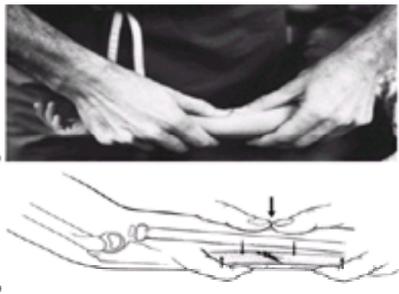
### AUTHORS' PREFERRED METHOD OF TREATMENT—NONOPERATIVE

Nonoperative treatment should generally involve three steps: correcting the ulnar deformity, providing a stable reduction of the radial head, and relieving the deforming muscle forces to prevent recurrent radial head dislocation and ulnar angulation.

**Reduction of the Ulnar Fracture.** The first step is to reestablish the length of the ulna by longitudinal traction and manual correction of any angular deformities present. The forearm is held in relaxed supination as longitudinal traction is applied, with manual pressure directed over the apex until the angular deformity is corrected clinically and radiographically ([Fig. 12-14](#)). With greenstick fractures, the plastic deformity must be corrected or the fracture completed to prevent recurrence of the angular deformity and, possibly, dislocation of the radial head ([Fig. 12-15](#)). Up to 10 degrees of angulation is acceptable in a complete fracture, providing a concentric radial head reduction is maintained ([88](#)). Angulation up to 25 degrees was noted in one series ([77](#)) to be compatible with full elbow and forearm motion. This degree of deformity usually remodels with time ([89](#)) ([Fig. 12-16](#)).



**FIGURE 12-14.** Closed reduction, type I lesion. **A:** Injury film of a typical type I lesion in a 7-year-old. **B:** The deformity of the ulna is corrected first, and then the elbow is hyperflexed. However, the radial head is still anteriorly subluxed (arrows), and the ulna still has some anterior plastic deformation

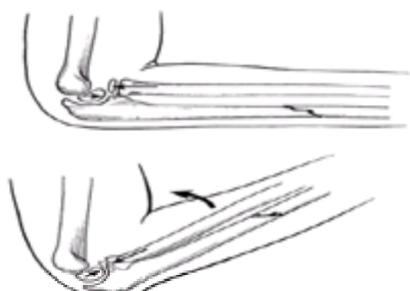


**FIGURE 12-15. A and B:** Correction of plastic deformation. Plastic deformation of the ulna must be corrected to prevent recurrence of the angular deformity. This allows reduction of the radial head and prevents its late subluxation. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. American Academy of Orthopaedic Surgeons Monograph Series. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:46; with permission.)

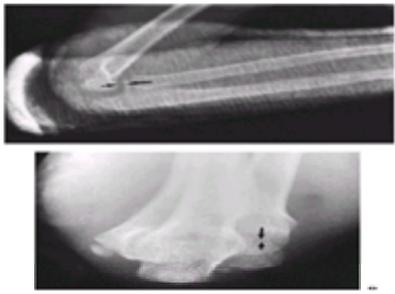


**FIGURE 12-16. A:** Type I Monteggia fracture-dislocation with posterior angulation of the ulna at 3 weeks postinjury. **B:** Lateral radiograph at 3 months postinjury shows remodeling. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. American Academy of Orthopaedic Surgeons Monograph Series. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:48; with permission.)

**Reduction of the Radial Head.** Once ulnar length and alignment have been reestablished, the radial head can be relocated. This often is accomplished simply by flexing the elbow to 90 degrees or more, thus producing spontaneous reduction ([Fig. 12-17](#)). Occasionally, posteriorly directed pressure over the anterior aspect of the radial head is necessary to facilitate reduction of the radial head. Flexion of the elbow to 110 to 120 degrees stabilizes the reduction of the radial head. Once the radial head position is thought to be established, it should be scrutinized radiographically in numerous views to ensure a concentric reduction. With a type I fracture, the optimal radiographic view is a true lateral of the elbow with the forearm held in supination. The longitudinal axis of the radius should pass directly through the center of the capitellum ([Fig. 12-18](#)).



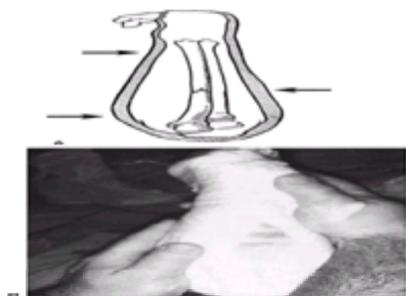
**FIGURE 12-17.** Reduction of the radial head. Flexing the elbow spontaneously reduces the radial head. Occasionally, manual pressure is required in combination with flexion.



**FIGURE 12-18.** Once the reduction is complete, radiographs should be analyzed for reestablishment of the radiocapitellar line (arrows) and ulnar alignment on both the lateral (A) and Jones (B) views.

**Alleviation of Deforming Forces.** Once the concentric reduction of the radial head is confirmed, the elbow should be placed at approximately 110 to 120 degrees of flexion to alleviate the force of the biceps, which could redislocate the radial head (see [Fig. 12-17](#)). The forearm is placed in a position of mid-supination to neutral rotation to alleviate the forces of the supinator muscle and the anconeus, as well as the forearm flexors, which tend to produce radial angulation of the ulna.

**Immobilization.** Once the fracture is reduced and the neutralization position is established, a molded long arm splint or cast is applied ([Fig. 12-19](#)) to hold the elbow joint in the appropriate amount of flexion, usually 110 to 120 degrees. Once the cast is completed, careful radiographic assessment should establish the concentric reduction of the radial head with respect to the capitellum, as well as satisfactory alignment of the ulna.



**FIGURE 12-19.** Postreduction immobilization. **A:** After reduction, the forearm is immobilized with three-point molding (arrows) to maintain ulnar alignment. **B:** The forearm component is incorporated into a long arm cast with the elbow flexed to 110 to 120 degrees of flexion.

**After Care.** The patient is followed at 7- to 10-day intervals to confirm continued satisfactory reduction by radiography. At 4 to 6 weeks after the initial reduction, if there is radiographic evidence of consolidation of the ulnar fracture and stability of the radial head, the long arm cast can be removed, with progressive guarded return to full activity.

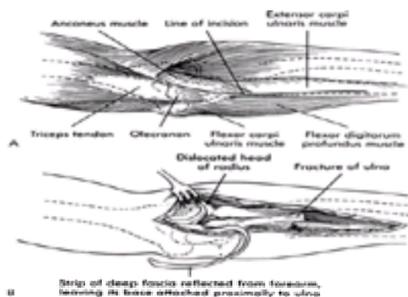
### Operative Treatment

**Indications.** Although uncommon, there are two indications for operative treatment of type I fracture–dislocations: failure of ulnar reduction and failure of radial head reduction.

**Failure of Ulnar Reduction.** If the ulnar fracture cannot be reduced or held in satisfactory alignment by closed treatment, operative intervention is indicated. The quality of the ulnar reduction affects the ability to reduce the radial head, which is of primary importance. Watson and Singer ([125](#)) reported median nerve entrapment that prevented ulnar reduction in a greenstick fracture in a 6-year-old with a type I Monteggia lesion. If the ulnar fracture can be reduced but not maintained because of the obliquity of the fracture, internal fixation combined with open or closed reduction may be necessary ([36,77](#)). Intramedullary fixation, rather than fixation with a plate, is standard in most series of Monteggia fracture–dislocations in children ([8,28,36,58,61,77,82,119,127](#)). This method of fixation may be accomplished percutaneously, using image intensification and flexible nails or Kirschner wires; this avoids the potential complications of open reduction and plate fixation ([60,80,85,118,122](#)).

**Failure of Radial Head Reduction.** The second indication is failure to reduce the radial head satisfactorily by closed means. This is more common in type III Monteggia lesions, but it can also occur in type I lesions. It results from the interposition of material in the joint. The pathologic description of the impeding structures usually includes interposition of torn fragments of the ruptured orbicular ligament and capsule or interposition of an intact orbicular ligament that has been pulled over the radial head ([121,128](#)). Interposed cartilaginous or osteochondral fractures may also be found in the radiocapitellar joint or radial notch in the ulna, preventing complete reduction of the radial head ([76,121](#)). Morris ([71](#)) described a patient in whom reduction of the radial head was obstructed by radial nerve entrapment between the radial head and ulna.

**Surgical Approach.** The most direct approach to the radiocapitellar joint is from the posterolateral aspect of the elbow. The interval between the anconeus and the extensor carpi ulnaris, using the distal portion of a Kocher “J” incision, provides sufficient visualization of the radial head and the interposed structures ([41,110](#)). This approach protects the posterior interosseous nerve, providing the forearm is positioned in pronation. A more extensile approach was described by Boyd ([16](#)). This exposure is begun by making an incision following the lateral border of the triceps posteriorly to the lateral condyle and extending it along the radial side of the ulna ([Fig. 12-20](#)). The incision is carried under the anconeus and extensor carpi ulnaris in an extraperiosteal manner, elevating the fibers of the supinator from the ulna. This carries the approach down to the interosseous membrane, allowing exposure of the radiocapitellar joint, excellent visualization of the orbicular ligament, access to the proximal fourth of the entire radius, and approach to the ulnar fracture, all through the same incision ([16,17,102](#)).



**FIGURE 12-20.** Surgical Approach. **A:** The incision is carried under the anconeus and extensor carpi ulnaris to expose the radial head and orbicular ligament. **B:** The incision can be extended distally to allow exposure of the ulnar fracture and proximally to facilitate harvesting of the tendinous strip for orbicular ligament reconstruction, if necessary. (From Canale ST, Beaty JH, eds. *Operative pediatric orthopedics*, 2nd ed.. St. Louis: Mosby-Year Book, 1994:1069; with permission.)

## AUTHORS' PREFERRED METHOD OF TREATMENT—OPERATIVE

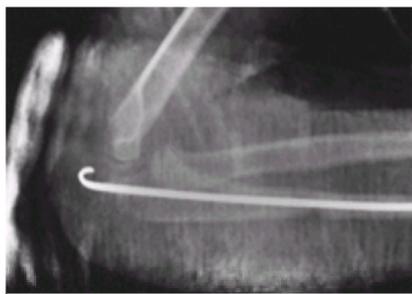
The importance of radial head reduction should be emphasized because failure of reduction affects the long-term outcome. Ulnar reduction directly affects the surgeon's ability to reduce the radial head. In children, the preferred treatment is by closed manipulative reduction. If, however, reduction of the radial head or ulnar fracture cannot be obtained or if the instability of the ulnar fracture threatens the radial reduction, operative treatment should be performed.

**Surgical Approach.** We prefer to use the Boyd approach because of its extensile nature ( [Fig. 12-20](#)).

**Treatment of the Annular Ligament.** Once the reduction is obtained through this approach, we repair the annular ligament, providing it can be done simply. If the ligament has been pulled over the radial head, it should be incised, replaced in its normal location, and repaired. Immediate reconstruction of the annular ligament is usually unnecessary because the radial head usually maintains its stability once the ulnar fracture is reduced to a satisfactory position.

If the reduction is unstable, we repair or reconstruct the annular ligament, combining it with the use of a transcapitellar Steinmann pin if necessary. The details of annular ligament reconstruction are discussed later in the section on old, undetected Monteggia lesions.

**Treatment of the Ulnar Fracture.** The ulnar fracture usually is treated closed, provided a satisfactory alignment can be obtained. If the fracture pattern seems to be unstable on the initial films or at the initial reduction, or if the child is younger than 12 years of age, internal fixation using an intramedullary pinning technique is done ([60,80,85,122](#)). This often can be accomplished by using either a single pin of sufficient size or multiple small pins, nesting them within the medullary canal to provide stability ([Fig. 12-21](#)). This technique is minimally invasive and provides adequate stability in most fractures. It also allows easy retrieval of the instrumentation and has an overall low complication rate ([118](#)). In our experience, more aggressive fixation has been unnecessary in children, but compression plating could be used if more rigid fixation were indicated or in an adolescent with a comminuted fracture.



**FIGURE 12-21.** Stabilization of the ulna. Unstable fractures or those in children 12 years of age or older can be immobilized using a percutaneous pinning technique. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. American Academy of Orthopaedic Surgeons Monograph Series. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:49; with permission.)

**After Care (Same as Closed Reduction).** Once reduction is obtained, a long arm cast is applied with the elbow flexed 90 to 100 degrees and the forearm in neutral position.

### Type II Lesions

#### Incidence

Posterior Monteggia fracture–dislocations are uncommon, accounting for 6% in most series of Monteggia lesions in children ([59](#)). When it occurs during childhood, it usually is in older patients (approximately 13 years) ([81](#)) who have sustained significant trauma ([32,91,92](#)). Type II Monteggia lesions are more common than reported. They often go unrecognized, being classified as type III lesions with a posterolateral radial head dislocation.

#### Clinical Findings

As with type I Monteggia lesions, the elbow region is swollen but exhibits posterior angulation of the proximal forearm and a marked prominence in the area posterolateral to the normal location of the radial head. Examination of the upper extremities should continue in areas remote from the elbow because of the high incidence of associated fractures ([57,81](#)).

#### Radiographic Evaluation

Standard radiographic views of the forearm demonstrate the pertinent features for classifying this fracture. The typical finding is a proximal metaphyseal fracture of the ulna with possible extension into the olecranon ([32,77,116](#)) ([Fig. 12-22](#)). Midshaft fractures also occur, presenting with an oblique fracture pattern ([6,32,77](#)). The radial head is dislocated posteriorly or posterolaterally ([8](#)) and should be carefully examined for other associated injuries. Accompanying fractures of the anterior margin of the radial head have been noted in adults ([32,81](#)) but not in children.

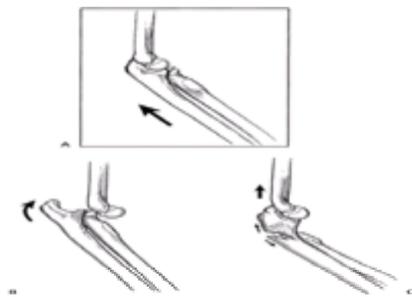


**FIGURE 12-22.** Type II fracture-dislocation. The typical radiographic findings include **(A)** a posterior dislocation of the radial head (*arrows*) and **(B)** a proximal metaphyseal fracture, which may extend into the olecranon (*arrows*). In addition to being posterior, the head may be lateral as well (*arrows*) **(C)**. (Courtesy of Dr. Gail S. Chorney, MD, Hospital for Joint Diseases, New York, New York.)

### Mechanism of Injury

The cause of the type II Monteggia lesion is subject to debate. Bado thought the lesion was caused by direct force and sudden rotation and supination (8). Penrose analyzed a series of seven fractures, all in adults (84), and noted that a proximal ulnar fracture was the typical pattern. He postulated that the injury occurred by longitudinal loading rather than direct trauma (102). Olney and Menelaus (77) reported four type II lesions in their series of children's Monteggia fractures. Three of these patients demonstrated a proximal ulnar fracture and one an oblique midshaft fracture pattern, suggesting possibly two different mechanisms of injury.

The mechanism proposed and experimentally demonstrated by Penrose (84) shows the type II lesion occurring when the forearm is suddenly loaded in a longitudinal direction with the elbow bent to 60 degrees of flexion. He showed that the type II lesion occurred consistently if the anterior cortex of the ulna was weakened; otherwise, a posterior elbow dislocation was produced (Fig. 12-23). The difference in bony integrity of the ulna suggested a reason for the high incidence of type II Monteggia fractures in older adults and its rarity in children. Penrose further noted that the rotational position of the forearm did not seem to affect the type of fracture produced.



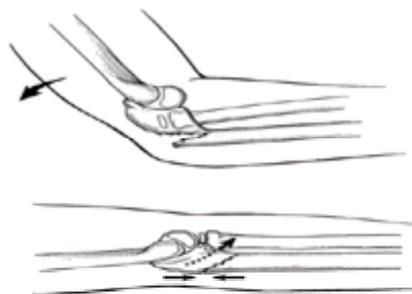
**FIGURE 12-23.** Mechanism of injury for type II fracture-dislocation. **A:** The elbow is flexed approximately 60 degrees; a force is applied longitudinally, parallel to the long axis of the forearm. **B:** A posterior elbow dislocation may occur. **C:** If the integrity of the anterior cortex of the ulna is compromised, a type II fracture-dislocation occurs.

Haddad et al. (42) described type II Monteggia injuries caused by low-velocity injuries in six adults, five of whom were on long-term corticosteroid therapy. They suggested that this supports the theory that the type II (posterior) Monteggia injury is a variant of posterior elbow dislocation, in that it occurs when the ulna is weaker than the ligaments surrounding the elbow joint, resulting in an ulnar fracture before the ligament disruption associated with dislocation occurs.

### Treatment

#### Nonoperative

As with type I injuries, type II lesions usually have a satisfactory result after closed reduction (61,77,82,89,127). The ulnar fracture is reduced by longitudinal traction in line with the long axis of the forearm while the elbow is held at 60 degrees of flexion. The radial head may reduce spontaneously or may require gentle, anteriorly directed pressure applied to its posterior aspect. The elbow is extended once the radial head is reduced and is immobilized in that position to stabilize the radial head and allow molding posteriorly to maintain the ulnar reduction (28,56,82,123) (Fig. 12-24).



**FIGURE 12-24.** Reduction of type II lesion. The elbow is held at 60 degrees of flexion; traction is applied in line with the forearm and the elbow is extended. It may be necessary to apply pressure over the radial head to complete the reduction.

#### Operative

Because of the rarity of the lesion, there are no established indications for operative treatment. Treatment goals are stable concentric reduction of the radial head and alignment of the ulnar fracture. The radial head should be reduced by open technique if there is interposed tissue or if it is accompanied by a fractured capitellum or radial head. The ulnar fracture is exposed along its subcutaneous border if necessary and fixed internally. Fixation can be accomplished by plating or intramedullary fixation. Because of children's ability to heal rapidly, intramedullary fixation is adequate and sometimes can be done percutaneously after a closed reduction of the ulna (80,85).

## AUTHORS' PREFERRED METHOD OF TREATMENT

The goals of treatment in the type II Monteggia lesion are to obtain a stable concentric reduction of the radial head, with adequate alignment of the ulna.

First, ulnar length is reestablished by applying longitudinal traction and straightening the angular deformity. The radial head may reduce spontaneously or with gentle, anteriorly directed force directly over the radial head. Once reduced, the position of the head can be stabilized by holding the elbow in extension. The alignment of the ulnar fracture can be maintained by applying a cast with the elbow in extension or by using percutaneous intramedullary fixation. If intramedullary pinning is used, the elbow may be flexed to 80 degrees and a cast applied. Radiographs should be taken after cast application and approximately every 7 to 10 days to confirm continued reduction of the radial head.

The Boyd approach can be used to obtain reduction of the radial head if it cannot be obtained through closed manipulation. Management of the annular ligament is the same as described previously in type I Monteggia lesions.

The cast, either in full extension or flexed to 80 degrees at the elbow, is maintained for 3 to 4 weeks, then changed to a removable splint. This allows early intermittent range of motion in a protected manner for 3 to 4 more weeks. At 6 to 8 weeks after injury, the patient may be released to full activities. This protocol may avoid the necessity for formal physical therapy (82).

### Type III Lesions

#### Clinical Findings

Lateral swelling and varus deformity of the elbow and significant limitation of motion, especially supination, are the hallmarks of lateral Monteggia fracture–dislocations, or type III Monteggia lesions.

#### Incidence

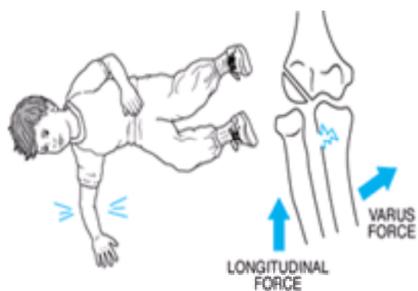
Type III lesions are common in children, occurring second in frequency to anterior Monteggia fracture–dislocations (8,72,77,82,127). Injuries to the radial nerve, particularly the posterior interosseous branch, occur frequently with this lesion (11,105). Open reduction of the radial head often is necessary because of interposition of soft tissue between it and the ulna or capitellum (11,46,114,128,129).

#### Radiographic Evaluation

The radial head may be displaced laterally or anterolaterally (80,82). The ulnar fracture often is in the metaphyseal region (8,11,46,82,128,129), but it can occur more distally (77,127,128). Radial angulation at the fracture site is common to all lesions, regardless of the level. Radiographs of the entire forearm should be obtained because of the association of distal radial and ulnar fractures with this elbow injury complex (115).

#### Mechanism of Injury

Wright (129) studied fractures of the proximal ulna with lateral and anterolateral dislocations of the radial head and concluded that the mechanism of injury was varus stress at the level of the elbow, in combination with an outstretched hand planted firmly against a fixed surface (Fig. 12-25). This usually produces a greenstick ulnar fracture with tension failure radially and compression medially. The radial head dislocates laterally, rupturing the annular ligament. Hume (46) suggested that the injury may be the result of hyperextension of the elbow combined with pronation of the forearm. Other authors confirmed the mechanism of varus force at the elbow as the cause of these injuries (8,28,72,82,115). The direction of the radial head dislocation is probably determined by the rotation and angulation force applied simultaneously with the varus moment at the elbow (72).



**FIGURE 12-25.** Mechanism of injury for type III lesions. A forced varus stress causes a greenstick fracture of the proximal ulna and a true lateral or anterolateral radial head dislocation.

### Treatment

#### Nonoperative

Nonoperative treatment is effective in nearly all patients (8,11,28,46,61,72,77,82,115,127,129), although the rate of operative treatment has been reported to be as high as 12% (77).

**Focus on Ulnar Reduction.** Reduction is carried out by reversing the mechanism of injury (28,72,82,116). The elbow is held in extension with longitudinal traction. Valgus stress is placed on the ulna at the site of the fracture, producing clinical realignment. The radial head may spontaneously reduce or need assistance with gentle pressure applied laterally (Fig. 12-26). Reduction is sometimes heralded by a palpable click (116).



**FIGURE 12-26.** Reduction of type III lesion. Valgus stress is placed on the ulna at the fracture site (arrow), producing clinical realignment. The radial head may

spontaneously reduce or need an assistive push.

**Confirm Radial Head Reduction.** Radiographs are obtained in the standard views (69). The position of the radial head is important. Any malalignment in either view implies the possibility of interposed tissue or persistent malalignment of the ulna. The angular alignment of the ulna must be as straight as possible to allow and maintain reduction of the radial head (36).

**Maintenance of Reduction.** Degree of Flexion. Reduction is maintained by a long arm cast with the elbow in flexion. The degree of flexion varies depending on the direction of the radial head dislocation. When the radius is in a straight lateral or anterolateral position, flexion to 110 to 120 degrees improves stability (28,72,89,127). If there is a posterolateral component to the dislocation, a position of only 70 to 80 degrees of flexion has been recommended (116).

**Forearm Rotation.** Forearm rotation usually is in supination, which tightens the interosseous membrane and further stabilizes the reduction (8,28,72,127). Over the years, one researcher has changed the suggested position of immobilization from pronation (115) to semisupination (116). Ramsey and Pedersen (89) recommended neutral as the best position of rotation to avoid loss of motion; their patients demonstrated no loss of reduction using that position.

## Operative

Surgical intervention has two goals: reduction and stabilization of the radial head and the ulnar fracture.

**Radial Head Reduction.** The radial head is reduced through a Boyd approach (16,128). This allows removal of the interposed tissues (76,121,128) and repair or reconstruction of the annular ligament if necessary (12,20,36,38,102,114) (Fig. 12-27). The surgical technique is essentially the same as previously described for the type I Monteggia fracture–dislocation.



**FIGURE 12-27.** Irreducible type III lesion. **A:** Injury films showing typical greenstick olecranon fracture and lateral dislocation of a type III Monteggia lesion. **B:** After manipulation and correction of the ulnar deformity, the radial head still was not reduced. **C:** Open reduction was performed to extract the interposed torn orbicular ligament. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. American Academy of Orthopaedic Surgeons Monograph Series. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:50; with permission.)

**Ulnar Stabilization.** Stabilization of the ulna is necessary to prevent recurrent lateral dislocation of the radial head. Varus alignment or radial bow, particularly with oblique fractures, may lead to subluxation of the radius (36,77) (Fig. 12-28). Fixation with plates (36) and Kirschner wires (6) has been performed with satisfactory results. Up to 25 degrees of varus angulation is consistent with normal alignment and function (77), and any residual deformity decreases with time (89).



**FIGURE 12-28.** Radial bow of the ulna. **A:** Anteroposterior views of both elbows showing residual radial bow of the proximal ulna after an incompletely reduced Monteggia type III lesion. **B:** This bow has produced a symptomatic lateral subluxation of the radial head. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. American Academy of Orthopaedic Surgeons Monograph Series. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:51; with permission.)

## AUTHORS' PREFERRED METHOD OF TREATMENT

We concur with Cunningham (25), who believed that reduction of the dislocated radial head is an important part of the management of the Monteggia fracture–dislocation. Our method of treatment is aimed primarily at obtaining and maintaining reduction of the radial head, either by open or closed technique. The type of treatment of the ulnar fracture we choose is determined by its effect on the primary goal, radial head reduction.

### Nonoperative

The results of nonoperative reduction of a type III lesion are shown in Fig. 12-29.



**FIGURE 12-29.** Type III lesion, nonoperative treatment. **A:** Injury film showing lateral dislocation of the radial head. **B:** Radiograph taken after closed reduction showing reduction of the radial head and healing of the ulna by nonoperative methods.

**Reduction Technique.** We hold the elbow in extension to control the proximal fragment by locking the olecranon into the humerus. We then can apply longitudinal traction in line with the forearm with valgus stress applied at the ulnar fracture site ( [Fig. 12-26](#)). This usually allows the radial head to become reapposed to the capitellum, but pressure laterally over the prominence of the radial head may be necessary.

**Radiographic Evaluation.** Radiographs are taken in the AP and lateral planes to confirm the reduction of the radial head and assess the ulnar alignment. Up to 10 degrees of ulnar angulation is acceptable in younger children, provided the radial head reduction is concentric and stable.

**Immobilization.** A long arm cast is applied in two portions. First, a Muenster type of short arm cast is applied with the forearm in neutral rotation. This allows careful molding of the cast to maintain the alignment of the ulnar fracture. The elbow is then flexed to approximately 110 degrees in lateral or anterolateral dislocations, and the cast is completed to the axilla. In younger children, where the cast may be hard to mold, a loop sling is incorporated into the cast to prevent it from migrating distally. Although we have no experience with the method, treatment of a posterolateral dislocation with an extended elbow cast with valgus molding seems logical.

**After Care.** Radiographs should be taken in the completed cast to confirm continued reduction of the radial head and satisfactory ulnar alignment. The cast is left in place for 3 to 4 weeks, depending on the age of the child. It is replaced by a removable splint for an additional 3 to 4 weeks while early ranging of the elbow has begun. Restoration of rotation is particularly important at this time. Before application of the splint 3 to 4 weeks postinjury and at discontinuation at 6 to 8 weeks postfracture, radiographs are taken to assess the reduction. At 6 to 8 weeks after injury, sufficient healing has occurred to allow gradual return to full activities.

### Operative

Similar to our goals for closed reduction, our operative indications are failure to obtain and maintain reduction of the radial head and alignment of the ulnar fracture.

**Reducing the Radial Head.** Failed closed reduction of the radial head implies interposition of soft tissue, which we remove through a Boyd approach ( [Fig. 12-20](#)). If possible, the annular ligament is repaired, but we find that formal reconstruction usually is unnecessary if the radiocapitellar relation is restored and stable. If stability is in question, we reconstruct the annular ligament as described for the type I Monteggia lesion.

**Ulnar Realignment.** Before a formal annular ligament reconstruction, ulnar alignment must be evaluated critically. If inadequate, the ulnar alignment may prevent relocation of the radial head. The ulnar fracture can usually be reduced closed, but open realignment may be necessary due to interposed tissue. Once reduced, the ulna can be maintained by the insertion of intramedullary pins, either in a retrograde fashion or percutaneously. The ends of the pins can be buried subcutaneously and removed later with minimal surgery.

**After Care.** Postoperative care is the same as for a fracture treated nonoperatively, with emphasis on early motion.

### Type IV Lesions

#### Clinical Findings

The appearance of the patient and limb with a type IV lesion is similar to that of the type I lesion. More swelling and pain would be expected because of the magnitude of force required to create this complex injury. Particular attention should be given to the neurovascular status of the limb, anticipating the possible increased risk for a compartment syndrome. Although this injury is uncommon in general and rare in children, the radiocapitellar joint should be examined in all midshaft forearm fractures to avoid missing this injury complex ( [Fig. 12-30](#)). Failure to recognize the radial head dislocation is the major complication of this fracture ( [10](#)).



**FIGURE 12-30.** Type IV Monteggia lesion. **A** Anterior dislocation of the head of the radius with fracture of the upper third of the radial shaft with the ulna angulated anteriorly. The dislocation of the radial head was not recognized. **B** Five years later, the radial head was still dislocated, misshapen, and prominent. A full range of motion was present, with the exception of a loss of 10 degrees of full supination. The patient had no pain, but generalized weakness was noted in this extremity, especially in throwing motions. (Courtesy of Fred C. Tucker, MD.)

#### Radiographic Evaluation

The pattern of radial head dislocation is similar to that in a type I Monteggia lesion—that is, it is directed anteriorly ( [Fig. 12-31](#)). The radial and ulnar fractures usually are seen at any level in the middle third ( [30](#)), with the radial fracture usually distal to the ulnar injury. They may be complete or greenstick.



**FIGURE 12-31.** Type IV lesion. There is an anterior dislocation of the radial head. The radius and ulna fractures are usually in the middle third of the shaft, with the radial fracture distal to the ulnar fracture.

### **Mechanism of Injury**

Bado proposed that the type IV lesion is caused by hyperpronation (6). Two case reports discuss the mechanism of injury. One implies that the injury is due to the hyperpronation mechanism of Evans (39), and the other demonstrates a type IV lesion caused by a direct blow (96). Olney and Menelaus (77) report a single type IV lesion in their series but do not discuss the mechanism. Our preferred mechanism for this type of lesion was described in the section on type I lesions.

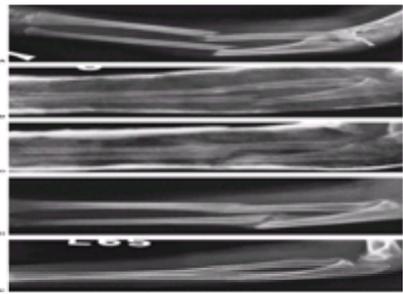
### **Treatment**

This complex lesion has been treated by both closed (77) and open (8) techniques. Two clinicians (39,96) treated the radial and ulnar fractures with percutaneous intramedullary fixation using flexible pins. This allowed closed reduction of the radial head. One of the authors of this chapter has reported on four type IV Monteggia lesions in children, treated by both operative and nonoperative techniques. He recommends plating of the radius in older children.

### **AUTHORS' PREFERRED METHOD OF TREATMENT**

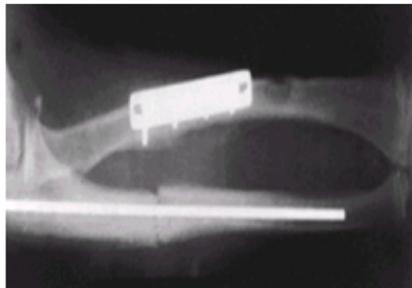
The goals of treatment for a type IV Monteggia lesion are similar to those of the other Bado types. The presence of the free-floating proximal radial fragment hampers the ability to reduce the radial head. Stabilization of the radial fracture converts the type IV lesion to a type I lesion, greatly facilitating treatment. The goals for treatment of a type IV lesion are, therefore, stabilization of the radial fracture, restoration of the ulnar alignment, and concentric reduction of the radial head.

**Nonoperative.** Closed reduction should be attempted initially, especially if the radial and ulnar fractures have greenstick patterns. Use of the image intensifier facilitates treatment by allowing immediate confirmation of reduction of the elements of the injury complex, especially the radial head. Closed treatment of unstable lesions may be attempted, but it is our experience that multiple remanipulations are required to obtain a satisfactory outcome ( Fig. 12-32).



**FIGURE 12-32.** Type IV closed reduction. **A:** Injury film in a 7-year-old with fractures of the shafts of the radius and ulna and associated dislocation of the radial head. **B:** Initial closed reduction with some residual posterior angulation. **C:** At 3 weeks postinjury, the forearm was remanipulated to correct the posterior angulation. **D:** Appearance 8 weeks postreduction, still with slight posterior angulation. **E:** Six months later, there has been considerable remodeling. (From Wilkins KE, ed. *Operative management of upper extremity fractures in children*. American Academy of Orthopaedic Surgeons Monograph Series. Rosemont, IL: American Academy of Orthopaedic Surgeons, 1994:51-52; with permission.)

**Operative.** If the fractures are unstable, we reduce the fractures closed and fix them percutaneously with intramedullary pins. In children 12 years of age or older, plating the radius through Henry's extensile approach (43) provides more rigid stabilization ( Fig. 12-33). Once stability is achieved, a closed reduction of the radial head is attempted. This is usually successful, but any intraarticular obstruction can be removed using the Boyd approach, which is more direct than extending Henry's approach.



**FIGURE 12-33.** Operative treatment, type IV lesion. The initial goal is to stabilize the radius. In older children, a plate may be indicated. Intramedullary pinning usually is adequate. (See Fig. 12-31 for injury film.)

**After Care.** The elbow is immobilized for 4 weeks in 110 to 120 degrees of flexion with the forearm in neutral rotation. The cast is reduced to a short arm cast for an additional 4 weeks while beginning early range of motion at the elbow and forearm. Forearm rotation, within the limits of cast restrictions, is specifically encouraged to avoid permanent loss of rotation. At 8 weeks postinjury, there is gradual return to full activities.

### **Bado's Equivalent Lesions**

#### **Clinical Findings**

Clinical findings are similar to those for the corresponding Bado lesion, with the common triad of pain, swelling, and deformity.

#### **Radiographic Evaluation**

As with the Bado types, careful radiographic study should be made with at least two orthogonal views. Special views such as obliques should be obtained to delineate the associated injuries (e.g., radial head or neck fractures, lateral humeral condyle fractures) clearly to allow adequate pretreatment planning.

#### **Mechanism of Injury**

The mechanism of injury by which the fracture occurs helps define its equivalent type and is discussed in the sections on the relevant Bado type.

#### **Treatment**

As with the other Bado types, treatment addresses two general components of the lesion: ulnar fracture and associated injury.

### Ulnar Fracture

This fracture is treated as for the other Bado types. The method is dictated by the fracture pattern and location and its stability after reduction.

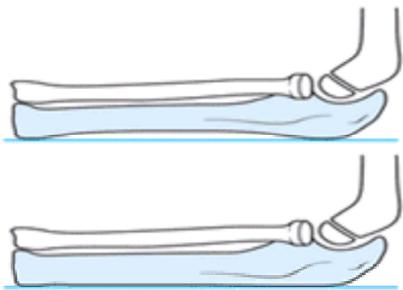
### Associated Injury

These associated fractures and dislocations are evaluated and treated using principles based on the particular injury. They are discussed thoroughly in other sections of this chapter.

### Complications (All Lesions)

#### Old Undetected Fracture–Dislocations

An undetected dislocation of the radial head is uncommon ([28,36,100,113](#)). Isolated radial head dislocations with no apparent lesion of the ulna caused by remote trauma have been mistaken for radial head dislocations of congenital etiology ([29,66](#)). Subsequent authors have refuted the existence of a unilateral congenital dislocation of the radial head ([64](#)). Observations of the shape of the ulna in patients with an isolated dislocation of the radial head have indicated the existence of persistent plastic deformation of the ulna associated with this radial lesion ([63](#)) ([Fig. 12-34](#) and [Fig. 12-35](#)). The diagnosis of a dislocated radial head has been aided by the acceptance of the veracity of the radiocapitellar line ([69,100,109](#)) ([Fig. 12-9](#)).



**FIGURE 12-34.** The ulnar bow line. This line, drawn between the distal ulna and the olecranon, defines the ulna bow. The “ulnar bow sign” is defined as deviation of the ulnar border from the reference line of more than 1 mm. (From Lincoln TL, Mubarak SJ. “Isolated” traumatic radial head dislocation. *J Pediatr Orthop* 1994;14:455; with permission.)



**FIGURE 12-35.** Ulnar bow line. **A:** The injury film of an 8-year-old girl who fell, spraining her arm. Note anterior bow of the ulna (*black arrows*) and loss of the radiocapitellar relation (*open arrow*). **B:** Film at time of diagnosis. Note persistent ulnar bow and overgrowth of radius. (Courtesy of David Mann, MD, VW Hospital and Clinics, Madison, Wisconsin.)

### Natural History

The ability to diagnose the dislocated radial head at the time of injury has helped prevent the difficult problem of persistent radial head dislocation. When a previously undetected dislocation is encountered, the natural history of the untreated lesion should be considered in determining the method of treatment. The belief that many children with persistent dislocation of the radial head do well has been supported by several investigators ([73,87,98,107](#)). Fahey ([34](#)) believed that although in the short term persistent dislocations do well, they present problems later. At that time, the problem can be treated by resection of the radial head.

Other reports, however, suggested that the natural history of persistent dislocation is not benign and is associated with restricted motion, deformity, functional impairment (weakness, instability), pain, degenerative arthritis, and late neuropathy ([5,13,14,22,38,40,46,47,50,52,63](#)). Kalamchi ([50](#)) reported pain, instability, and restricted motion, especially loss of pronation and supination. He also noted that children have a valgus deformity and a prominence on the anterior aspect of the elbow. Tardy nerve palsies have been reported as developing subsequent to an unrecognized Monteggia lesion of long-standing duration ([1,5,45,62](#)).

### Indications for Treatment

As experience has developed with reconstructive relocation of the radial head in Monteggia lesions, the indications have become clear. Blount ([15](#)), in his classic monograph, and Fowles et al. ([36](#)), in their analysis, suggested that reconstruction provides the best results in patients who have had a dislocation for 3 or 6 months or less, respectively. Fowles et al., however, reported successful relocations up to 3 years after injury, and Freedman et al. ([38](#)) up to 6 years after injury. Throughout the literature, the appropriate age seems to be younger than 10 years ([108](#)). Hirayama et al. ([44](#)) suggested that the procedure not be performed if there is significant deformity of the radial head, flattening of the capitellum, or valgus deformity of the neck of the radius.

In a more recent report, however, Seel and Peterson ([99](#)) suggested that the age of the patient and the duration of the dislocation are unimportant. Their criteria for surgical repair were (a) normal concave radial head articular surface and (b) normal shape and contour of the ulna and radius (deformity of either was corrected by osteotomy). They treated seven patients (all girls) ranging in age from 5 to 13 years for chronic dislocations that had been present from 3 months to 7 years. All seven were fully active with no elbow pain or instability at an average of 4 years after surgery.

Although they recommended surgical treatment of chronic Monteggia lesions in children because of the long-term sequelae, Rodgers et al. ([97](#)) cautioned that the results of reconstructive procedures are unpredictable and associated with a number of serious complications. Fourteen complications occurred in their seven patients (11 months to 12 years of age) treated from 5 weeks to 39 months after injury, including malunion of the ulnar shaft, residual radiocapitellar subluxation, radiocapitellar dislocation, transient ulnar nerve palsy, partial laceration of the radial nerve, loss of fixation, nonunion of the ulnar osteotomy, compartment syndrome, conversion reaction, and possible fibrous synostosis.

## Historical Development of Surgical Repair

The technique for delayed reduction of the radial head in a Monteggia fracture is attributed to Bell-Tawse ( 12). He used the surgical approach described by Boyd ( 16). Other surgical approaches have been developed subsequent to Boyd, but are not as adaptable to annular ligament reconstruction ( 41,110).

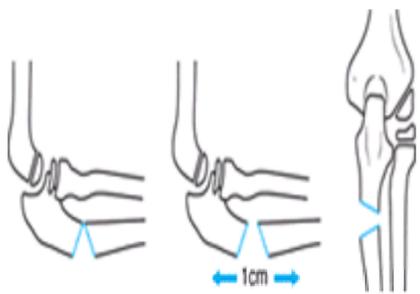
## Annular Ligament Reconstruction

Once the capsule has been debrided from the joint, consideration is given as to whether the annular ligament needs to be reconstructed. Kalamchi ( 50) restored stability after open reduction and osteotomy by reefing the remnants of the annular ligament.

Various authors have considered it necessary to reconstruct the annular ligament, probably because of insufficiency of the natural structure. Although Bell-Tawse ( 12) used a strip of triceps tendon, other authors have tried other material. Corbett ( 24) reported stabilizing an open reduction of the radial head using the lacertus fibrosus through an anterior approach. Speed and Boyd ( 102) used a strip of the forearm fascia to establish stability of the radial neck. Watson-Jones ( 124) used a strip of palmaris longus tendon. May and Mauck ( 65) used chromic ligature. Bell-Tawse ( 12) used the central portion of the triceps tendon passed through a drill hole and around the radial neck to stabilize the reduction. This was aided by immobilizing the elbow in a long arm cast in extension. Bucknill ( 20) and Lloyd-Roberts ( 64) used the Bell-Tawse procedure but modified it by changing the site of procurement of the ligament to the lateral portion of the triceps tendon. They found that immobilizing the elbow in flexion and using a transcapitellar pin to establish stability worked well, avoiding the inconvenience of a long arm cast in extension. Hurst and Dubrow ( 47) used the central portion of the triceps tendon. Distally, their dissection of periosteum was carried along the ulna to the level of the radial neck, providing more stable fixation than the technique of stopping at the olecranon described by Bell-Tawse. Further, a periosteal tunnel was used rather than a drill hole to establish fixation for the tendinous strip of the ulna. Thompson and Lipscomb ( 117) have used a fascia lata graft passed through a drill hole in the ulna to stabilize an unstable radial head.

## Osteotomy

In a Monteggia lesion with a mildly displaced ulnar fracture but persistent dislocation of the radial head, an osteotomy of the ulna usually is not indicated ( 64). However, various types of osteotomies have been used to facilitate reduction of the radial head, as well as to prevent recurrent subluxation after annular ligament reconstruction (Fig. 12-36). Kalamchi ( 50) reported using a drill osteotomy and allowing it to float, thus facilitating reduction of the radial head. He stated that this afforded the advantages of minimal periosteal stripping, allowing the osteotomy to heal rapidly. Hirayama et al. ( 44) and Mehta ( 67,68) used ulnar osteotomies to facilitate and stabilize the reduction of the radial head. Hirayama did a 1-cm distraction osteotomy approximately 5 cm distal to the tip of the olecranon. Internal fixation in the form of plate and screws was used, but there were significant complications with loosening and plate breakage. Mehta used an osteotomy of the proximal ulna in which a bone graft was used to stabilize the osteotomy. In neither series was an annular ligament repair performed. Oner and Diepstraten ( 78) suggested avoiding radial osteotomies because of the restriction of motion thought to occur owing to adhesions, as well as ulnar osteotomies because of the high rate of complications. Although they did not perform routine ulnar osteotomy, they pointed out that in type III lesions in which osteotomies were not performed, there was recurrent subluxation because of the persistent ulnar angulation ( Fig. 12-28). Freedman et al. ( 38) reduced a delayed type I Monteggia lesion using open reduction of the head without annular ligament reconstruction, ulnar osteotomy combined with radial shortening, and deepening of the radial notch of the ulna.



**FIGURE 12-36.** Ulnar osteotomies. **Left** Floating open osteotomy without fixation or bone graft. **Center** Hirayama distraction osteotomy, grafted and fixed with a plate and screws. Mehta's osteotomy is similar but is held with a bone graft only. **Right** Valgus osteotomy for a type III lesion: floating osteotomy with bone graft. This osteotomy may be easily stabilized with an intramedullary pin.

Inoue and Shionoya ( 48) compared the results of simple corrective ulnar osteotomy in six patients with those of posterior angular (overcorrected) osteotomy in six others and found that better clinical outcomes were obtained with the angular osteotomy. Tajima and Yoshizu ( 112), in a series of 23 neglected Monteggia fractures, found that the best results were obtained by opening wedge osteotomy of the proximal ulna without ligament reconstruction.

## AUTHORS' PREFERRED METHOD OF TREATMENT

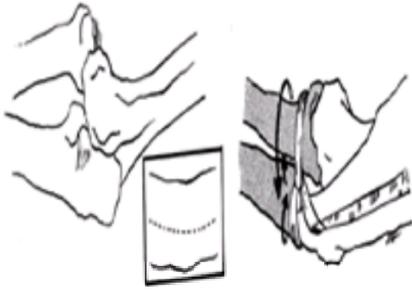
**Indications.** If a patient younger than 12 years of age presents with delayed diagnosis of a Monteggia lesion, the radial head should be replaced in its appropriate relation with the capitellum. The child does well in the short term, but problems develop later. As the child matures to adulthood, there is a significant chance of progressive instability, pain, weakness of the forearm, and restriction of motion. There is also a risk of tardy radial nerve or ulnar nerve palsies. There should be no radiocapitellar incongruity due to radial head deformity after reduction.

**Surgical Approach.** The skin incision begins approximately halfway between the central portion of the triceps and the lateral border, beginning proximal enough to obtain 6 to 7 cm of tendinous strip. The incision continues distally along the lateral border of the ulna just past the level of the radial neck. Dissection is carried deep, lifting the anconeus off the ulna sharply in an extraperiosteal manner. The capsule is entered and any interposed capsule, old ligament, or osteochondral fragment is removed from the joint. The radial notch of the ulna is also cleaned to allow the radial head to fall into place once reduced. Capsular adhesions are removed from the radial head to allow it to fall away from the anterior capsule.

**Radial Head Reduction.** Radial head reduction is then attempted. If it is accomplished, it is scrutinized for congruity between the radial head and the capitellum. If this is satisfactory, a ligamentous reconstruction can be performed. If the radius cannot be reduced, the ulna is osteotomized. The osteotomy is formed at the site of maximum deformity through a small incision, leaving the periosteum as undisturbed as possible. If reduction of the radial head still cannot be achieved, radial shortening can be considered, but we have no experience with this procedure.

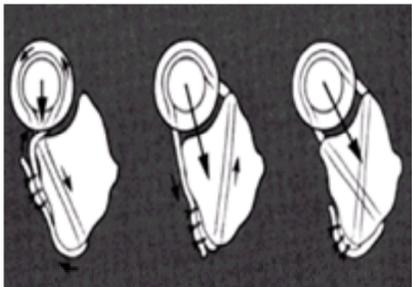
After ligament reconstruction, the ulnar osteotomy can be stabilized or left to float. Stabilization can be performed by several techniques, including plating, bone graft, or intramedullary fixation. Percutaneous intramedullary fixation with flexible Kirschner wires allows self-adjustment but adds stability to the entire reconstruction; it is our preferred treatment.

**Annular Ligament Reconstruction.** At the time of exposure, the triceps aponeurosis and tendon are examined for their thickest portion. A 1- to 6-cm strip is developed and carried distally, carefully elevating the periosteum from the proximal ulna down to the level of the radial neck. In the Bell-Hawse technique, a drill hole slightly larger than the strip of tendon is made in the ulna. The strip of tendon is then passed around the radial neck and through the drill hole in the ulna. The ligament is then brought back and sutured to itself ( Fig. 12-37).



**FIGURE 12-37.** The central slip of the triceps is used to reconstruct an annular ligament. (Redrawn from Bell-Tawse AJS. The treatment of malunited anterior Monteggia fractures in children. *J Bone Joint Surg Br* 1965;47:718; with permission.)

More recently, Seel and Peterson (99) described the use of two holes drilled in the proximal ulna. The holes are placed at the original attachments of the annular ligament and thereby allow repair of the annular ligament (frequently avulsed from one attachment and trapped within the joint) or reconstruction of the annular ligament with whatever tissue or material is desired (they suggested triceps tendon). It secures the radial head in its normal position from any dislocated positions and allows osteotomy for correction of any accompanying deformity of the ulna or radius. Seel and Peterson noted that the Bell-Tawse procedure tends to pull the radius posterolaterally (Fig. 12-38, left) and possibly constricts the neck of the radius, thereby potentially limiting the growth of the radial neck (“notching”) and reducing forearm rotation. They suggested that although this repair might be satisfactory for anteromedial dislocations, it may not be optimal for anterior, anterolateral, lateral, and posterolateral dislocations. Initially, Seel and Peterson placed a single drill hole obliquely across the ulna to exit medially at the site of the medial attachment of the annular ligament on the coronoid process of the ulna (Fig. 12-38, center). The tendon was routed through the tunnel, brought around the neck, and sutured to the lateral side of the ulna. With this construct, the direction of stability was posteromedial. The use of two drill holes to secure the annular ligament or other reconstructive tendon at both normal attachments of the annular ligament on the ulna achieved a more normal posteromedial holding force on the neck of the radius (Fig. 12-38, right).



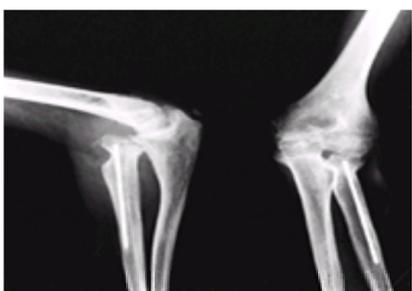
**FIGURE 12-38.** Drawings of transverse cuts of the proximal right radius and ulna (viewed distally) at the level of the radial head. **Left:** Route of the triceps tendon in Bell-Tawse reconstruction. The direction of stability is posterior (*large arrow*). **Center:** Drill hole placed obliquely to exit the ulna at the site of the medial annular ligament attachment. The direction of stability is posteromedial (*large arrow*). **Right:** Two drill holes exit the ulna at sites of medial and lateral annular ligament attachments. The direction of stability is anatomic (*arrow*). (From Seel MJ, Peterson HA. Management of chronic posttraumatic radial head dislocation in children. *J Pediatr Orthop* 1999;19:306-312; with permission.)

An alternative to the drill hole is to use a small bone staple or bone-anchoring device ( Fig. 12-39).



**FIGURE 12-39.** Fixation of radial head. After the ligament is reconstructed, the radial head is stabilized using a transcapitellar pin. Note the use of a bone anchor as an alternative to the drill hole.

**Stabilizing the Radial Head.** Once concentric reduction is achieved, the radiocapitellar relation is secured by passing a transcapitellar Steinmann pin through the posterior aspect of the capitulum into the radial head and neck with the elbow at 90 degrees and the forearm in supination ( Fig. 12-39). A pin of sufficient size is mandatory to avoid pin failure (55); a small pin will fatigue (Fig. 12-40). Alternative techniques to secure the reduction of the radius include transversely pinning the radius to the ulna (61) and immobilizing the elbow in extension (12).



**FIGURE 12-40.** Radiograph showing a broken transcapitellar wire with subsequent dislocation of the radial head. A pin of sufficient size is mandatory to avoid pin failure

**After Care.** After wound closure, a long arm cast is applied with the forearm in 60 to 90 degrees of supination. The position of the elbow is predetermined by the transcapitellar Steinmann pin. This position relaxes the biceps and maintains the ulna in neutral position. The cast is maintained for 6 weeks, after which the cast and pin are removed and active motion is begun, especially pronation and supination. Elbow flexion and extension return rapidly but rotary motion of the forearm may take 6 to 9 months to improve, with pronation possibly being limited permanently (96).

## Nerve Injuries

### Radial Nerve

The literature reflects a 10% to 20% incidence of radial nerve injury, making it the most common complication associated with Monteggia fractures (49). It is most commonly associated with type I and III injuries, with the latter being more frequent (11,73,100). The posterior interosseous nerve is most commonly injured because of its proximity to the radial head and its intimate relation to the arcade of Frohse (Fig. 12-7). The arcade may be thinner and therefore more pliable in children (103). This may account in part for the rapid resolution of the nerve injury in children.

Such a lesion in a child is treated expectantly. Nerve function usually returns by approximately 9 weeks after reduction, if not sooner (104,106). It has been recommended that the nerve be explored if there is no clinical or electromyographic return of neurologic function by 12 weeks (104,106). These recommendations are drawn from series of adult patients and may be premature and unnecessary in children. A review of a series of children's Monteggia lesions (77) recommends waiting 6 months before intervention in a posterior interosseous nerve injury. Most series report 100% resolution both in fractures treated promptly and in those treated remotely from the date of injury (1,5,62).

Spinner (103) reported three posterior interosseous nerve injuries in children. All function returned spontaneously, but he recommended that if no clinical or electromyographic return occurs in approximately 6 weeks, exploration and neurolysis of the nerve should be performed. There are two reports (71,101) of irreducible Monteggia fractures caused by interposition of the radial nerve posterior to the radial head. After the nerve was replaced to its normal anatomic position and the head reduced, function returned in approximately 4 months. Morris (71), in cadaveric studies, demonstrated that significant anterior dislocation of the radial head and varus angulation of the elbow allowed the radial nerve to slide posterior to the radial head and, with subsequent reduction of the radial head, become entrapped.

### Ulnar Nerve

Bryan (19) reported one adult with an ulnar nerve lesion associated with a type II Monteggia lesion, with spontaneous resolution. Stein and colleagues (106) reported three combined radial nerve and ulnar nerve injuries, two of which required exploration for functional return of the nerve.

### Median Nerve

The association of median nerve injuries with Monteggia fractures is low. Injury to the anterior interosseous nerve has been reported in two series (124,127). In one case, exploration was necessary to release the entrapped nerve from the ulna (124). Both cases had complete resolution of the neurologic deficit. Stein et al. (106), in their report specifically examining nerve injuries in Monteggia lesions, reported no median nerve deficits. Watson and Singer (125) reported entrapment of the main trunk of the median nerve in a greenstick ulnar fracture in a 6-year-old girl. Completion of the fracture was necessary for release of the nerve. At 6 months after surgery, there was full motor recovery but sensation was slightly reduced in the tips of the index finger and thumb.

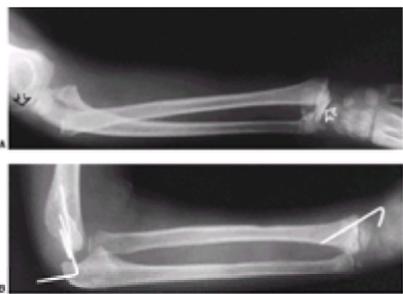
### Tardy Nerve Palsy

The literature highlights seven cases of tardy radial nerve injury associated with radial head dislocation (1,5,45,62,130). The type of treatment has varied. Usually, excision of the radial head is included with exploration and neurolysis of the nerve, with uniformly good results (1,5). Other surgeons have explored the nerve alone, with good but variable results (45,62). Yamamoto and associates (130) combined radial head resection and nerve exploration with tendon transfers, producing good results in two patients.

One patient, who had a combination of radial head dislocation and a malunited distal humerus fracture, presented with combined tardy radial and ulnar nerve palsies (1). The ulnar nerve lesion was treated with anterior transposition, which provided resolution of the paresis. The radial nerve was treated by resection of the radial head and exploration of the nerve.

## Associated Fractures and Unusual Lesions

Monteggia lesions have been associated with fractures of the wrist and the distal forearm (8). Distal radial and ulnar joint dislocation or sprain and fracture of the distal radius through the physis or distal metaphysis are most frequently reported (8,14,51,96) (Fig. 12-41). Galeazzi fractures may also occur concurrently with Monteggia lesions (8,75). Radial head and neck fractures are commonly associated with type II fractures (8,32), but may occur with others (35,37,51). With a type II lesion, the radial head may fracture, usually on its anterior rim (32). Strong et al. (111) reported two cases of a type I equivalent lesion consisting of a fractured radial neck and midshaft ulnar fracture. This equivalent was unique in having significant medial displacement of the distal radial fragment. They had significant difficulty in obtaining and maintaining reduction of the radius through closed technique in this fracture pattern.



**FIGURE 12-41.** Associated injuries. An 8-year-old child with a high-velocity injury to the entire forearm. **A:** Radiograph of a type III Monteggia fracture and associated lateral condyle (black arrow) and distal radial physeal (white arrow) fractures. **B:** Postoperative management with percutaneous pin fixation.

Fractures of the lateral condyle have been associated with Monteggia fractures (82). Ravessoud (90) reported an ipsilateral ulnar shaft lesion and a lateral condyle fracture without loss of the radiocapitellar relation, suggesting a Monteggia type II equivalent.

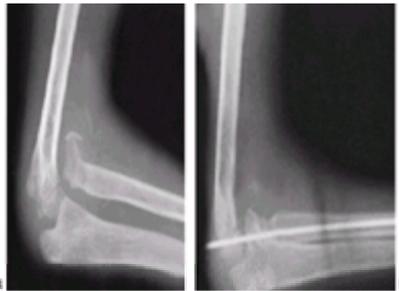
### Periarticular Ossification

Two patterns of ossification have been noted radiographically. Various investigators have commented on the formation of ossification around the radial head and neck (12,46,62,64,107,109). It appears as a thin ridge of bone in a caplike distribution and may be accompanied by other areas resembling sesamoid bones (Fig. 12-42); they resorb with treatment and time. Ossification may also occur in the area of the annular ligament (31). Although noting their appearance, none of the authors detailed elbow function after the formation of these lesions (12,46,64,107,109).



**FIGURE 12-42.** Periarticular ossification. Persistent dislocations of the radial head are frequently accompanied by a thin cap of bone and other areas resembling sesamoid bones

The other form of ossification is true myositis ossificans. This occurred in approximately 3% of elbow injuries and 7% of Monteggia lesions in a series of adults and children (120). Myositis ossificans has a good prognosis in patients younger than 15 years of age, appearing at 3 to 4 weeks postinjury and resolving in 6 to 8 months (Fig. 12-43). Its occurrence is related to the severity of the initial injury, association with a fractured radial head, the number of remanipulations during treatment, and passive motion of the elbow during the postoperative period (74,120).



**FIGURE 12-43.** Periarticular ossification. **A:** Lateral radiograph of an old anterior dislocated radial head. **B:** Two months after treatment, with resolution of ossification.

## SUMMARY

Adherence to several fundamental principles helps ensure good outcomes after Monteggia fractures in children:

- If, in a radiograph of the forearm, one bone overlaps or angulates, subluxation of either radioulnar joint should be considered.
- Evaluation of the radial head location requires an AP view of the proximal forearm and a true lateral view of the elbow.
- The radiocapitellar line must be intact in both views.
- Achievement of concentric, congruent radial head reduction is of primary importance, whether by open or closed means.
- In type IV injuries, the radial fracture must be stabilized before radial head reduction.
- Stability of the ulnar reduction is required to help maintain reduction of the radial head. Stability may be inherent to the fracture pattern or achieved by internal fixation.
- If additional stability of the radial head reduction is necessary, it can be achieved by reconstruction of the annular ligament.
- Immobilization should be in the position that best achieves stability of the radial head and maintains the position of the ulna.
- Early mobilization, especially rotation, is important to avoid stiffness.
- Frequent radiographic evaluation is necessary to monitor the position of the radial head and ulnar fracture.

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## CHAPTER REFERENCES

1. Adams JR, Rizzoli H. Tardy radial and ulnar nerve palsy: a case report. *J Neurosurg* 1959;16:342.
2. Almquist EE, Gordon LH, Blue AI. Congenital dislocation of the head of the radius. *J Bone Joint Surg Am* 1969;51:1118–1127.
3. Altner P. Monteggia fractures. *Orthop Rev* 1981;10:115.
4. Anderson HJ. Monteggia fractures. *Adv Orthop Surg* 1989;4:201–204.
5. Austin R. Tardy palsy of the radial nerve from a Monteggia fracture. *Injury* 1926;7:202–204.
6. Bado JL. La lesion de Monteggia. Inter-Medica Sarandi 328 S.R.L, 1958.
7. Bado JL. *The Monteggia lesion*. Springfield, IL: Charles C Thomas, 1962.
8. Bado JL. The Monteggia lesion. *Clin Orthop* 1967;50:71–86.
9. Basmajian JV, Griffen WR. Function of anconeus muscle. *J Bone Joint Surg Am* 1972;54:1712–1714.
10. Beaty JH. Fractures and dislocations about the elbow in children: section on Monteggia fractures. *Instr Course Lect* 1991;40:373–384.
11. Beddow FH, Corkery PH. Lateral dislocation of the radio-humeral joint with greenstick fracture of the upper end of the ulna. *J Bone Joint Surg Br* 1960;42:782–784.
12. Bell-Tawse AJS. The treatment of malunited anterior Monteggia fractures in children. *J Bone Joint Surg Br* 1965;47:718–723.
13. Best TN. Management of old unreduced Monteggia fracture dislocations of the elbow in children. *J Pediatr Orthop* 1994;14:193–199.
14. Blasler D, Trussell A. Ipsilateral radial head dislocation and distal fractures of both forearm bones in a child. *Am J Orthop* 1995;24:498–500.
15. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955.
16. Boyd HB. Surgical exposure of the ulna and proximal one-third of the radius through one incision. *Surg Gynecol Obstet* 1940;71:86–88.
17. Boyd HB, Boals JC. The Monteggia lesion: a review of 159 cases. *Clin Orthop* 1969;66:94.
18. Bruce HE, Harvey JP, Wilson JC. Monteggia fracture. *J Bone Joint Surg Am* 1974;56:1563–1576.
19. Bryan RS. Monteggia fracture of the forearm. *J Trauma* 1971;11:992–998.
20. Bucknill TM. The elbow joint. *Proc R Soc Med* 1977;70:620.
21. Campbell WC. *A textbook on orthopedic surgery*. Philadelphia: WB Saunders, 1930.
22. Caravias DE. Some observations on congenital dislocation of the head of the radius. *J Bone Joint Surg Br* 1957;39:86–90.
23. Cooper A. *Dislocations and fractures of the joints*. Boston: TR Marvin, 1844:391–400.
24. Corbett CH. Anterior dislocation of the radius and its recurrence. *Br J Surg* 1931;19:155.
25. Cunningham SR. Fracture of the ulna head with dislocation of the head of the radius. *J Bone Joint Surg* 1934;16:351–354.
26. Curry GJ. Monteggia fracture. *Am J Surg* 1947;123:613–617.
27. Denucé A. *Mémoire sur les luxations du coude*. Thèse de Paris, 1854.
28. Dormans JP, Rang M. The problem of Monteggia fracture–dislocations in children. *Orthop Clin North Am* 1990;21:251.
29. Duverney JG. *Traité des maladies de os*. Paris: De Bure l'Aire, 1751.
30. Eady JL. Acute Monteggia lesions in children. *J S C Med Assoc* 1975;71:107–112.
31. Earwaker J. Post-traumatic calcification of the annular ligament of the radius. *Skeletal Radio*. 1992;21:149–154.

32. Edwards EG. The posterior Monteggia fracture. *Am Surg* 1952;18:323–337.
33. Evans M. Pronation injuries of the forearm. *J Bone Joint Surg Br* 1949;31:578–588.
34. Fahey JJ. Fractures of the elbow in children: Monteggia's fracture–dislocation. *Instr Course Lect* 1960;17:39.
35. Fahmy NRM. Unusual Monteggia lesions in kids. *Injury* 1980;12:399–404.
36. Fowles JV, Sliman N, Kassah MT. The Monteggia lesion in children. *J Bone Joint Surg Am* 1983;65:1276–1283.
37. Frazier JL, Buschmann WR, Insler HP. Monteggia type I equivalent lesion: diaphyseal ulna and proximal radius fracture with a posterior elbow dislocation in a child. *J Orthop Trauma* 1991;5:373–375.
38. Freedman L, Luk K, Leong JC. Radial head reduction after a missed Monteggia fracture: brief report. *J Bone Joint Surg Br* 1988;70:846–847.
39. Gibson WK, Timperlake RW. Orthopedic treatment of 4 type IV Monteggia fracture–dislocations in a child. *J Bone Joint Surg Br* 1992;74:780–781.
40. Givon U, Pritsch M, Levy O, et al. Monteggia and equivalent lesions: a study of 41 cases. *Clin Orthop* 1997;337:208–215.
41. Gorden ML. Monteggia fracture: a combined surgical approach employing a single lateral incision. *Clin Orthop* 1967;50:87–93.
42. Haddad ES, Manktelow ARJ, Sarkar JS. The posterior Monteggia: a pathological lesion? *Injury* 1996;27:101–102.
43. Henry AK. *Extensile exposure*. Baltimore: Williams & Wilkins, 1970.
44. Hirayama T, Takemitsu Y, Yagihara K, et al. Operation for chronic dislocation of the radial head in children. *J Bone Joint Surg Br* 1987;69:639–642.
45. Holst-Nielson F, Jensen V. Tardy posterior interosseus nerve palsy. *J Hand Surg [Am]* 1984;9:572–575.
46. Hume AL. Anterior dislocation and fracture of olecranon. *J Bone Joint Surg Br* 1957;39:508–512.
47. Hurst LC, Dubrow EN. Surgical treatment of symptomatic chronic radial head dislocation: a neglected Monteggia fracture. *J Pediatr Orthop* 1983;3:227–230.
48. Inoue G, Shionoya K. Corrective ulnar osteotomy for malunited anterior Monteggia lesions in children: 12 patients followed for 1–12 years. *Acta Orthop Scand* 1998;69:73–76.
49. Jessing P. Monteggia lesions and their complicating nerve damage. *Acta Orthop Scand* 1975;46:601–609.
50. Kalamchi A. Monteggia fracture–dislocation in children. *J Bone Joint Surg Am* 1986;68:615–619.
51. Kamali M. Monteggia fracture. *J Bone Joint Surg Am* 1974;56:841–843.
52. Kaplan EB. The quadrate ligament of the radio-ulnar joint in the elbow. *Bull Hosp Joint Dis* 1964;25:126–130.
53. Karachalios T, Smith EJ, Pearse MF. Monteggia equivalent injury in a very young patient. *Injury* 1992;23:419–420.
54. Kay RM, Skaggs DL. The pediatric Monteggia fracture. *Am J Orthop* 1998;27:606–609.
55. King RE. Letter to the editors. *J Pediatr Orthop* 1983;3:623.
56. King RE. The Monteggia lesion. In: Rockwood CA Jr, Wilkins KE, King RE, eds. *Fractures in children*, 3rd ed.. Philadelphia: JB Lippincott, 1991:453–497.
57. Kristiansen B, Eriksen AF. Simultaneous type II Monteggia lesion and fracture separation of the lower radial epiphysis. *Injury* 1986;17:51–62.
58. Lambrinudi C. Intramedullary Kirschner wires in the treatment of fractures. *Proc R Soc Med* 1940;33:153.
59. Landin LA. Fracture patterns in children. *Acta Paediatr Scand Suppl* 1983;54:192.
60. Lascombes P, Prevot J, Ligen JN, et al. Elastic stable intramedullary nailing in forearm shaft fractures in children: 85 cases. *J Pediatr Orthop* 1990;10:167–171.
61. Letts M, Loch R, Wiens J. Monteggia fracture–dislocations in children. *J Bone Joint Surg Br* 1985;67:724–727.
62. Lichter R, Jackson T. Tardy palsy of posterior interosseous nerve with Monteggia fracture. *J Bone Joint Surg Am* 1975;57:124–125.
63. Lincoln TL, Mubarak SJ. "Isolated" traumatic radial head dislocation. *J Pediatr Orthop* 1994;14:454–457.
64. Lloyd-Roberts GC, Bucknill TM. Anterior dislocation of the radial head in children. *J Bone Joint Surg Br* 1977;59:402–406.
65. May Y, Mauck W. Dislocation of the radial head with associated fracture of the ulna. *South Med J* 1961;54:1255–1261.
66. McFarland B. Congenital dislocation of the head of the radius. *Br J Surg* 1936;24:41–49.
67. Mehta SD. Flexion osteotomy of ulna for untreated Monteggia fracture in children. *Indian J Surg* 1985;47:15–19.
68. Mehta SD. Missed Monteggia fracture. *J Bone Joint Surg Br* 1993;75:337.
69. Miles KA, Finlay DBL. Disruption of the radio-capitellar line in the normal elbow. *Injury* 1989;20:365–367.
70. Monteggia GB. *Instuzioni chirurgiche*. Milan: Maspero, 1814.
71. Morris A. Irreducible Monteggia lesion with radial nerve entrapment. *J Bone Joint Surg Am* 1974;56:1744–1746.
72. Mullick S. The lateral Monteggia fracture. *J Bone Joint Surg Am* 1977;57:543–545.
73. Naylor A. Monteggia fractures. *Br J Surg* 1942;29:323.
74. Neviasser RJ, LeFevre GW. Irreducible isolated dislocation of the radial head: a case report. *Clin Orthop* 1971;80:72–74.
75. Odena IC [Milch H, trans]. Bipolar fracture–dislocation of the forearm. *J Bone Joint Surg Am* 1952;34:968–976.
76. Ogden JA. *Skeletal injury in children*. Baltimore: Lea & Febiger, 1990:478.
77. Olney B, Menelaus M. Monteggia and equivalent lesions in childhood. *J Pediatr Orthop* 1989;9:219.
78. Oner FC, Diepstraten AFM. Treatment of chronic post-traumatic dislocation of the radial head in children. *J Bone Joint Surg Br* 1993;75:577–581.
79. Papavasiliou V, Nenopoulos S. Monteggia-type elbow fracture in childhood. *Clin Orthop* 1988;233:230–233.
80. Parsch K. Die Morote-Drahtung bei proximalen und mittleren Unterarm Schaft Frakturen des Kindes. *Oper Orthop Traumatol* 1990;2:245–255.
81. Pavel A, Pitman J, Lance E, et al. The posterior Monteggia fracture: a clinical study. *J Trauma* 1965;5:185.
82. Peiró A, Andres F, Fernandez-Esteve F. Acute Monteggia lesions in children. *J Bone Joint Surg Am* 1977;59:92–97.
83. Peltier LF. Eponymic fractures: Giovanni Battista Monteggia and Monteggia's fracture. *Surgery* 1957;42:585–591.
84. Penrose JH. The Monteggia fracture with posterior dislocation of the radial head. *J Bone Joint Surg Br* 1951;33:65–73.
85. Perez Sicialia JE, Morote Jurado JL, Corbach Girones JM, et al. Osteosintesis percuntabea eu fracturas diafisaris de ante brazo en ninos y adolescentes. *Rev Esp Cir Ost* 1977;12:321–334.
86. Perrin J. *Les fractures du cubitus accompagnées de luxation de l'extrémité supérieure du radius*. Thèse de Paris, Paris G. Steinheil, 1909.
87. Pollen AG. *Fractures and dislocations in children*. Edinburgh: Churchill Livingstone, 1973:67.
88. Price CT, Scott D, Kurener M, et al. Malunited forearm fracture in children. *J Pediatr Orthop* 1990;10:705–712.
89. Ramsey R, Pedersen H. The Monteggia fracture–dislocation in children. *JAMA* 1962;82:115.
90. Ravessoud F. Lateral condyle fracture and ipsilateral ulnar shaft fracture: Monteggia equivalent lesions. *J Pediatr Orthop* 1985;5:364–366.
91. Reckling F. Unstable Fracture–dislocations of the forearm: Monteggia and Galeazzi lesions. *J Bone Joint Surg Am* 1982;64:857–863.
92. Reckling FW, Cordell LD. Unstable fracture–dislocations of the forearm. *Arch Surg* 1968;96:999.
93. Ring D, Jupiter JB, Waters PM. Monteggia fractures in children and adults. *J Am Acad Orthop Surg* 1998;6:215–224.
94. Ring D, Waters PM. Operative fixation of Monteggia fractures in children. *J Bone Joint Surg Br* 1996;78:734–739.
95. Roach JW. Delayed discovery of traumatic radial head dislocation. *J Pediatr Orthop* 1996.
96. Rodgers WB, Smith BG. A type IV Monteggia injury with a distal diaphyseal radius fracture in a child: case report. *J Orthop Traumatol* 1993;7:84–86.
97. Rodgers WB, Waters PM, Hall JE. Chronic Monteggia lesions in children: complications and results of reconstruction. *J Bone Joint Surg Am* 1996;78:1322–1329.
98. Salter RB, Zaltz C. Anatomic investigations of the mechanism of injury and pathologic anatomy of "pulled elbow" in young children. *Clin Orthop* 1971;77:134.
99. Seel MJ, Peterson HA. Management of chronic posttraumatic radial head dislocation in children. *J Pediatr Orthop* 1999;19:306–312.
100. Smith FM. Monteggia fractures: an analysis of 25 consecutive fresh injuries. *Surg Gynecol Obstet* 1947;85:630–640.
101. Spar I. A neurologic complication following Monteggia fracture. *Clin Orthop* 1977;122:207–209.
102. Speed JS, Boyd HB. Treatment of fractures of ulna with dislocation of head of radius: Monteggia fracture. *JAMA* 1940;125:1699.
103. Spinner M. The arcade of Frohse and its relationship to P.I.N. paralysis. *J Bone Joint Surg Br* 1968;50:809.
104. Spinner M, Freundlich B, Teicher J. Posterior interosseous nerve palsy as a complication of Monteggia fracture in children. *Clin Orthop* 1968;58:141.
105. Spinner M, Kaplan E. The quadrate ligament of the elbow. *Acta Orthop Scand* 1970;41:632–647.
106. Stein F, Grabias S, Deffer P. Nerve injuries complicating Monteggia lesions. *J Bone Joint Surg Am* 1971;53:1432.
107. Stelling F, Cote R. Traumatic dislocation of head of radius in children. *JAMA* 1956;160:732–736.
108. Stoll TN, Baxter Nillis R, Paterson DC. Treatment of the missed Monteggia fracture in the child. *J Bone Joint Surg Br* 1992;74:436–440.
109. Storen G. Traumatic dislocation of radial head as an isolated lesion in children. *Acta Chir Scand* 1958–1959;116:144–147.
110. Strachen JCH, Ellis BW. Vulnerability of the posterior interosseous nerve during radial head reduction. *J Bone Joint Surg Br* 1971;53:320–323.
111. Strong M, Kopp M, Gillespie R. Fracture of the radial neck and proximal ulna with medial displacement of the radial shaft. *Orthopedics* 1989;12:1577.
112. Tajima T, Yoshizu T. Treatment of long-standing dislocation of the radial head in neglected Monteggia fractures. *J Hand Surg [Am]* 1995;20:S91–S94.
113. Tait G, Sulaiman SK. Isolated dislocation of the radial head: a report of two cases. *Injury* 1988;19:125.
114. Thakore HKD. Lateral Monteggia fracture in children. *Int J Orthop Trauma* 1983:55.
115. Theodorou SD. Dislocation of the head of the radius associated with fracture of the upper end of ulna in children. *J Bone Joint Surg Br* 1969;51:700–706.
116. Theodorou SD, Ierodiakonou MD, Rousis N. Fracture of the upper end of the ulna associated with dislocation of the head of the radius in children. *Clin Orthop* 1988;228:240–249.
117. Thompson D, Lipscomb B. Recurrent radial head subluxation treated with annular ligament reconstruction. *Clin Orthop* 1989;246:131–135.
118. Thompson GH, Wilber J, Marcus R. Internal fixation of fractures in children and adolescents. *Clin Orthop* 1984;188:10.
119. Thompson HA, Hamilton AT. Monteggia fracture: internal fixation of fractured ulna with I.M. Steinmann pin. *Am J Surg* 1950;79:579–584.
120. Thompson HC, Garcia R. Myositis ossificans: aftermath of elbow injuries. *Clin Orthop* 1967;50:129–134.
121. Tompkins DG. The anterior Monteggia fracture. *J Bone Joint Surg Am* 1971;53:1109–1114.
122. Verstrecken LG. Shaft forearm fractures in children. intramedullary nailing with immediate motion: a preliminary report. *J Pediatr Orthop* 1988;8:450–453.
123. Walker J, Rang M. Forearm fractures in children. *J Bone Joint Surg Br* 1991;73:299.
124. Watson-Jones R. *Fracture and joint injury*, 4th ed. Edinburgh: ES Livingstone, 1956.

125. Watson JAS, Singer GC. Irreducible Monteggia fracture: beware nerve entrapment. *Injury* 1994;25:325–327.
126. Weitbrecht J. *Syndesmologia sive historia ligamentorum corporis humani*. Petropoli Typographia Academiae Sclantiarum, Brecht, 1742.
127. Wiley JJ, Galey JP. Monteggia injuries in children. *J Bone Joint Surg Br* 1985;67:728–731.
128. Wise RA. Lateral dislocation of the head of radius with fracture of the ulna. *J Bone Joint Surg* 1941;23:379.
129. Wright PR. Greenstick fracture of the upper end of the ulna with dislocation of the radio-humeral joint or displacement of the superior radial epiphysis. *J Bone Joint Surg Br* 1963;45:727.
130. Yamamoto K, Yoshiaki Y, Tomihara M. Posterior interosseous nerve palsy as a complication of Monteggia fractures. *Arch Jpn Chin* 1977;46:46–56.

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We thank Kaye Wilkins for his contribution to this chapter and previous editions. Much of this chapter is his effort.

At the turn of the century, Sir Robert Jones ([30](#)) echoed the opinion of that era about elbow injuries: "The difficulties experienced by surgeons in making an accurate diagnosis; the facility with which serious blunders can be made in prognosis and treatment; and the fear shared by so many of the subsequent limitation of function, serve to render injuries in the neighborhood of the elbow less attractive than they might otherwise have proved." These concerns are applicable even today. The presentation of a child with a swollen, injured elbow still brings some anxiety to the treating orthopaedic surgeon. Fractures in other regions of the body can often be managed with minimal treatment to obtain uniformly good results. In the region of the elbow, however, there are often more indications for aggressive treatment, including operative management. Due to the high incidence of injuries and complications in children's elbow injuries, the orthopaedic surgeon must devote much time to learning the basic principles.

## INCIDENCE

### Vulnerability of Upper Extremities

Children tend to protect themselves with their outstretched arms when they fall, thus accounting for the vulnerability of the upper extremities to fractures. In the three major series reviewing large numbers of fractures sustained by children ([3,24,35](#)), the upper extremity consistently accounted for 65% to 75% of all the fractures sustained. The most common area of the upper extremity injured is the distal forearm ([3,36](#)). In these and other series, again the incidence of fractures about the elbow was consistent, ranging from 7% to 9% ([3,24,35,36,43](#)).

### Specific Percentages

Considerable data are available relating the specific percentages of the various fractures in the elbow region to the total number of elbow injuries. The distal humerus accounts for 86.4% of the fractures about the elbow region. In a combined series of 5,228 fractures ([3,4,7,8,15,16,21,27,34,36,37](#) and [38,45,49](#)) of the distal humerus, 79.8% involved the supracondylar area, 16.9% involved the lateral condyle, and 12.5% represented avulsion of the medial epicondyle. Both T-condylar fractures and fractures of the medial condyle had an incidence of less than 1%. In this large series, there was no mention of any fracture of the lateral epicondyle.

### Higher in 5- to 10-Year-Old Boys

Elbow injuries are much more common in the skeletally immature than in older people ([11,53](#)). The peak for fractures in the distal humerus is in the first decade of life, with most of the injuries occurring between ages 5 and 10 years ([27](#)). Elbow injuries are more common in boys. In the Northern Hemisphere, the seasonal peak is during the summer, the low point during the winter ([27,34](#)). Thus, the most likely candidate for an elbow injury is a boy 5 to 10 years of age playing during summer vacation.

The peak incidence of physeal (epiphyseal plate) injuries in most parts of the body is toward the termination of skeletal maturity. The Petersons ([42](#)) found that the incidence of physeal injuries in all parts of the body peaked between ages 10 and 13 years. However, the peak age for injuries to the distal humeral physes was 4 to 5 years in girls and 5 to 8 years in boys. In most physeal injuries, the increased incidence with advanced age is said to be due to weakening of the perichondrial ring as it matures (see [Chapter 5](#)). Thus, some different biomechanical forces and conditions must exist about the elbow to make the physis more vulnerable to injuries at an earlier age. (For more data on the relationship of fractures about the elbow to all types of fractures, see [Chapter 1](#)).

## ANATOMY

The elbow is a complex joint composed of three individual joints contained within a common articular cavity. This chapter emphasizes anatomic concepts unique to the growing elbow.

### The Ossification Process

#### Central-Distal Process

The process of differentiation and maturation begins at the center of the long bones and progresses distally. The ossification process begins in the diaphysis of the humerus, radius, and ulna at the same time. By term, ossification of the humerus has extended distally to the condyles. In the ulna, it extends to greater than half the distance between the coronoid process and the tip of the olecranon. The radius is ossified proximally to the level of the neck. The bicipital tuberosity remains largely unossified ([Table 13-1](#)) ([23](#)). Brodeur et al. compiled a complete atlas of ossification of the structures about the elbow ([9](#)), and their work is an excellent reference source for finer details of the ossification process about the elbow.

	Girls (yr)	Boys (yr)
Capitellum	1.0	1.0
Radial head	5.0	6.0
Medial epicondyle	5.0	7.5
Olecranon	8.7	10.5
Trochlea	9.0	10.7
Lateral epicondyle	10.0	12.0

Data from Cheng JC, Wing-Man K, Shen WY, et al. A new look at the sequential development of elbow-ossification centers in children. *J Pediatr Orthop* 1998;18:161-167.

**TABLE 13-1. SEQUENCE AND TIMING OF OSSIFICATION IN THE ELBOW**

### ***Distal Humerus***

#### **Sex Variation**

Ossification of the distal humerus proceeds at a predictable rate. In general, the rate of ossification in girls exceeds that of boys ( [20,22,25](#)). In some areas, such as the olecranon and lateral epicondyle, the difference between boys and girls in ossification age may be as great as 2 years ( [22](#)).

#### **Initial Symmetry**

During the first 6 months, the border of ossification of the distal humerus is symmetric ( [Fig. 13-1](#)).



**FIGURE 13-1.** During the first 6 months, the advancing ossifying border of the distal humerus is symmetric.

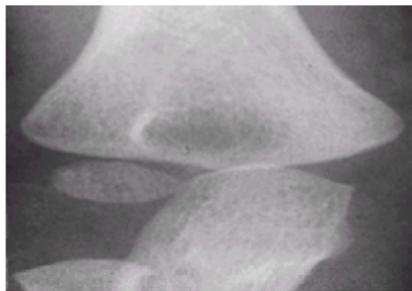
### ***Lateral Condyle***

The ossification center of the lateral condyle, on average, appears just before 1 year of age but may be delayed as late as 18 to 24 months ( [11](#)).

When the ossific nucleus of the lateral condyle first appears, the distal humeral metaphyseal border becomes asymmetric. The lateral border slants and becomes straight to conform with the ossification center of the lateral condyle ( [Fig. 13-2](#)). By the end of the second year, this border becomes well defined, possibly even slightly concave. This ossification center is usually spherical when it first appears. It becomes more hemispherical as the distal humerus matures ( [10](#)). This ossific nucleus is now extending into the lateral ridge of the trochlea ( [Fig. 13-3](#)). On the lateral view, the physis of the capitellum is wider posteriorly. This is a normal variation and should not be confused with a fracture ( [10](#)).



**FIGURE 13-2.** Ossification at 12 months. As the ossification center of the lateral condyle develops ( *arrow*), the lateral border of the metaphysis becomes straighter.



**FIGURE 13-3.** At 24 months, the oval-shaped secondary ossification center of the lateral condyle extends into the lateral crista of the trochlea. The lateral border of the neck (metaphysis) of the radius is normally angulated both anteriorly and laterally.

### ***Medial Epicondyle***

At about 5 to 6 years of age, a small concavity develops on the medial aspect of the metaphyseal ossification border. In this area, a medial epicondyle begins to ossify ([Fig. 13-4](#)).



**FIGURE 13-4.** At about 5 or 6 years of age, a secondary center develops in the medial epicondylar apophysis ( *white arrows*). At this same time, the ossification center of the radial head also develops ( *open arrow*). Note that the physis of the proximal radius is widened laterally ( *curved arrow*).

### **Trochlea**

At about 9 to 10 years of age, the trochlea begins to ossify. Initially, it may be irregular with multiple centers ( [Fig. 13-5](#)).



**FIGURE 13-5.** At about 9 years of age, the ossification of the medial crista of the trochlea may begin as two well-defined centers ( *arrows*). These multiple centers can give the trochlea a fragmented appearance.

### **Lateral Epicondyle**

The lateral epicondyle is last to ossify and is not always visible ( [Fig. 13-6](#)). At about 10 years of age, it may begin as a small, separate oblong center, rapidly fusing with the lateral condyle ( [10](#)).



**FIGURE 13-6.** The apophysis of the lateral epicondyle ossifies as either an oblong or a triangular center ( *arrows*). The wide separation of this center from the metaphyseal and epiphyseal borders of the lateral condyle is normal.

### **The Fusion Process**

Just before completion of growth, the capitellum, lateral epicondyle, and trochlea fuse to form one epiphyseal center. Metaphyseal bone separates the extraarticular medial epicondyle from this common humeral epiphyseal center ([Fig. 13-7](#)). The common epiphyseal center ultimately fuses with the distal humeral metaphysis. The medial epicondyle may not fuse with the metaphysis until the late teens.



**FIGURE 13-7.** The secondary ossification centers of the lateral condyle, trochlea, and lateral epicondylar apophysis fuse to form one center ( *white arrows*). This common center is separated from the medial epicondylar apophysis by advancing metaphyseal bone ( *black arrows*).

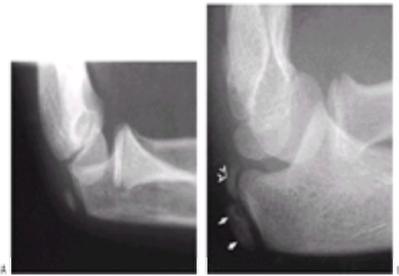
## Proximal Radius

The head of the radius begins to ossify at about the same time as the medial epicondyle ( [Fig. 13-4](#)). The ossification center is present in at least 50% of girls by 3.8 years of age but may not be present in the same proportion of boys until around 4.5 years ( [20](#)). Initially, the ossification center is elliptical and the physis is widened laterally because of the obliquity of the proximal metaphysis. The ossification center flattens as it matures. At about age 12, it develops a concavity opposite the capitellum ( [10](#)).

Ossification of the radial head may be bipartite or may produce an irregularity of the second center. These secondary or irregular ossification centers should not be interpreted as fracture fragments.

## Olecranon

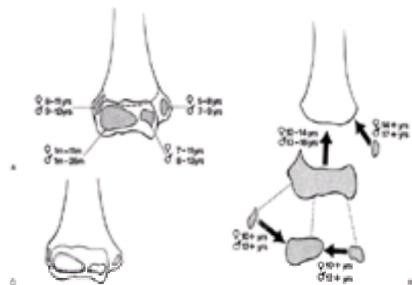
There is a gradual proximal progression of the proximal ulnar metaphysis. At birth, the ossification margin lies halfway between the coronoid process and the tip of the olecranon. By about 6 or 7 years of age, it appears to envelop about 66% to 75% of the capitellar surface. The final portion of the olecranon ossifies from a secondary ossification center that appears around 6.8 years of age in girls and 8.8 years in boys ( [Fig. 13-8A](#)). Porteous ( [43](#)) described two separate centers, one articular and the other a traction type ( [Fig. 13-8B](#)). This secondary ossification center of the olecranon may persist late into adult life ( [41](#)).



**FIGURE 13-8.** Ossification of the olecranon. **A:** Secondary ossification begins as an oblique oblong center at about 6 to 8 years of age. **B:** It may progress as two separate ossification centers: articular ( *open arrow*) and traction ( *closed arrows*).

## Fusion of the Ossification Centers

The epiphyseal ossification centers of the distal humerus fuse as one unit and then fuse later to the metaphysis. The medial epicondyle is the last to fuse to the metaphysis. The ranges of onset of ossification of the various centers and their fusion to other centers or the metaphysis are summarized in [Fig. 13-9](#). Each center contributes to the overall architecture of the distal humerus ( [Fig. 13-9C](#)).



**FIGURE 13-9.** Ossification and fusion of the secondary centers of the distal humerus. **A:** The average ages for the onset of ossification of the various ossification centers are shown for both boys and girls. **B:** The ages at which these centers fuse with each other are shown for both boys and girls. (Redrawn from Haraldsson S. On osteochondrosis deformans juvenilis apituli humeri including investigation of the intra-osseous vasculature in the distal humerus. *Acta Orthop Scand Suppl* 1959;38; with permission.) **C:** The contribution of each secondary center to the overall architecture of the distal humerus is represented by the stippled areas.

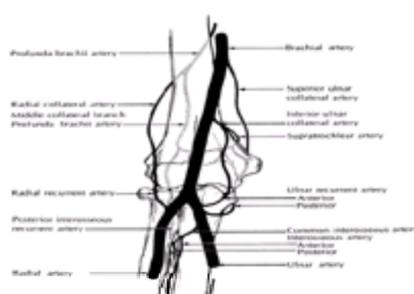
Fusion of the proximal radial and olecranon epiphyseal centers with their respective metaphyses occurs at around the same time that the common distal humeral epiphysis fuses with its metaphysis (i.e., between 14 and 16 years of age) ( [5,9,47](#)).

Noting that the pattern and sequence of ossification of the six secondary ossification centers around the elbow were mainly derived from studies conducted more than 30 years ago, Cheng et al. evaluated elbow radiographs of 1,577 Chinese children ( [14](#)). They found that the sequence of ossification was the same in boys and girls—capitellum, radial head, medial epicondyle, olecranon, trochlea, and lateral epicondyle—but ossification was delayed by about 2 years in boys in all ossification centers except the capitellum ( [Table 13-1](#)).

## Blood Supply

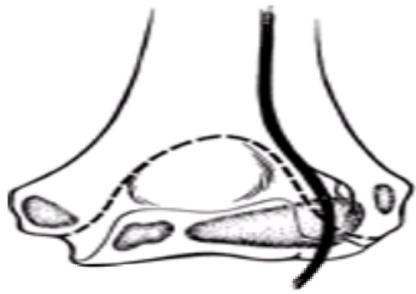
### Extrasosseous

There is a rich arterial network around the elbow ( [Fig. 13-10](#)) ( [55](#)). The major arterial trunk, the brachial artery, lies anteriorly in the antecubital fossa. Most of the intraosseous blood supply of the distal humerus comes from the anastomotic vessels that course posteriorly.



**FIGURE 13-10.** The major arteries about the anterior elbow.

Three structural components govern the location of the entrance of the vessels into the developing epiphysis. First, there is no communication between the intraosseous metaphyseal vasculature and the ossification centers. Second, vessels do not penetrate the articular surfaces. The lateral condyle is nonarticular only at the origin of the muscles and collateral ligaments. Third, the vessels do not penetrate the articular capsule except at the interface with the surface of the bone. Thus, only a small portion of the lateral condyle posteriorly is both nonarticular and extracapsular ( [Fig. 13-11](#)) (26).

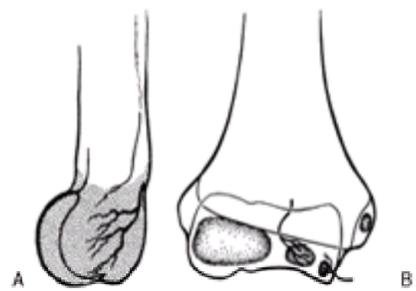


**FIGURE 13-11.** The vessels supplying the lateral condylar epiphysis enter the posterior aspect of the condyle, which is extraarticular. (Redrawn from Haraldsson S. On osteochondrosis deformans juvenilis capituli humeri including investigation of the intra-osseous vasculature in the distal humerus. *Acta Orthop Scand Suppl* 1959;38; with permission.)

### **Intraosseous**

#### **Posterior End Vessels**

The most extensive study of the intraosseous blood supply of the developing distal humerus was conducted by Haraldsson ( [Fig. 13-12](#)) (25,26). He demonstrated that two types of vessels exist in the developing lateral condyle. These vessels enter the posterior portion of the condyle just lateral to the origin of the capsule and proximal to the articular cartilage near the origin of the anconeus muscle. They penetrate the nonossified cartilage and traverse it to the developing ossific nucleus. In the young child, this is a relatively long course ( [Fig. 13-12A](#)). These vessels communicate with one another within the ossific nucleus but do not communicate with vessels in either the metaphysis or nonossified chondroepiphysis. Thus, for practical purposes, they are end vessels.



**FIGURE 13-12.** Intraosseous blood supply of the distal humerus. **A:** The vessels supplying the lateral condylar epiphysis enter on the posterior aspect and course for a considerable distance before reaching the ossific nucleus. **B:** Two definite vessels supply the ossification center of the medial crista of the trochlea. The lateral one enters by crossing the physis. The medial one enters by way of the nonarticular edge of the medial crista. (Redrawn from Haraldsson S. On osteochondrosis deformans juvenilis capituli humeri including investigation of the intra-osseous vasculature in the distal humerus. *Acta Orthop Scand Suppl* 1959;38; with permission.)

#### **Lateral Crista Is Part of Lateral Condyle**

The ossification center of the lateral condyle extends into the lateral portion of the trochlea. Thus, the lateral crista or ridge of the trochlea derives its blood supply from these condylar vessels. The medial ridge or crista remains unossified for a longer period of time.

#### **Trochlear Vessels Traverse Physis**

The trochlea is covered entirely by articular cartilage and lies totally within the confines of the articular capsule. The vessels that supply the nucleus of the ossific centers of the trochlea must therefore traverse the periphery of the physis to enter the epiphysis.

#### **Two Separate Trochlear Vessels**

Haraldsson's (26) studies have shown two sources of blood supply to the ossific nucleus of the medial portion of the trochlea ( [Fig. 13-12B](#)). The lateral vessel, on the posterior surface of the distal humeral metaphysis, penetrates the periphery of the physis and terminates in the trochlear nucleus. Because this vessel supplying the trochlea is an end vessel, it is especially vulnerable to injury by a fracture that courses through either the physis or the very distal portion of the humeral metaphysis. Injury to this vessel can markedly decrease the nourishment to the developing lateral ossific nucleus of the trochlea. The medial vessel penetrates the nonarticulating portion of the medial crista of the trochlea. This multiple vascular source may account for the development of multiple ossification centers in the maturing trochlea, giving it a fragmented appearance ( [Fig. 13-5](#)).

#### **Final Anastomosis**

When growth is complete, metaphyseal and epiphyseal vessels anastomose freely. The blood supply from the central nutrient vessel of the shaft reaches the epicondylar regions in the skeletally mature distal humerus ( 33).

### **Capsule**

#### **Intraarticular Structures**

The articular surface lies within the confines of the capsule, but nonarticulating areas involving the coronoid and radial fossae anteriorly and the olecranon fossa posteriorly are also within the confines of the articular cavity ( 54). The capsule attaches just distal to the coronoid and olecranon processes. Thus, these processes

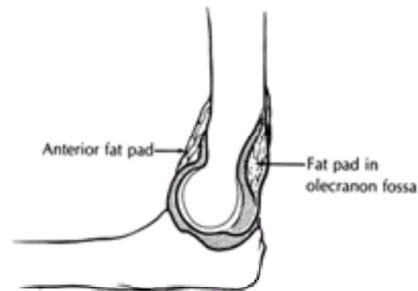
are intraarticular (29). The entire radial head is intraarticular, with a recess or diverticulum of the articular cavity of the elbow extending distally under the margin of the orbicular ligament. The medial and lateral epicondyles are extraarticular.

### **Anterior Portion Strongest**

The anterior capsule is thickened anteriorly. These longitudinally directed fibers are very strong and become taut with the elbow in extension. In hyperextension, the tight anterior bands of the capsule force the ulna firmly into contact with the humerus. Thus, the fulcrum of rotation becomes transmitted proximally into the tip of the olecranon in the supracondylar area. This is an important factor in the etiology of supracondylar fractures.

### **Fat Pads**

At the proximal portion of the capsule, between it and the synovial layer, are two large fat pads ( Fig. 13-13). The posterior fat pad lies totally within the depths of the olecranon fossa when the elbow is flexed. The anterior fat pad extends anteriorly out of the margins of the coronoid fossa. The significance of these fat pads in the interpretation of radiographs of the elbow is discussed later.



**FIGURE 13-13.** The elbow fat pads. Some of the coronoid fat pad lies anterior to the shallow coronoid fossa. The olecranon fat pad lies totally within the deeper olecranon fossa.

### **Ligaments**

The pertinent ligamentous anatomy involving the orbicular and collateral ligaments is discussed in the sections on the specific injuries involving the radial neck, medial epicondyle, and elbow dislocations.

## **RADIOGRAPHIC FINDINGS**

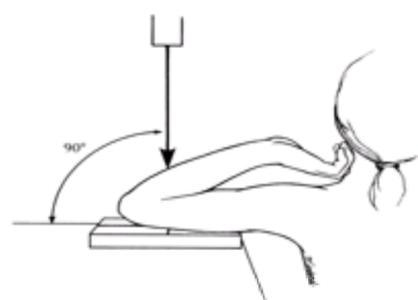
Because of the ever-changing ossification pattern, identification and delineation of fractures about the elbow in the immature skeleton may be subject to misinterpretation. The variables of ossification of the epiphyses should be well known to the orthopaedic surgeon who treats these injuries.

### **Standard View**

The standard radiographs of the elbow include an anteroposterior view with the elbow extended and a lateral view with the elbow flexed to 90 degrees and the forearm neutral.

### **Jones View**

In the injured elbow, it is often difficult to extend the elbow. In these cases, an axial view of the elbow, the Jones view, may be helpful ( Fig. 13-14). The distal humerus is often difficult to interpret because of the superimposed proximal radius and ulna. Often there is a high index of suspicion for a fracture, but none is visualized on the routine anteroposterior and lateral radiographs. In this case, internal and external oblique views may be helpful. This is especially true in visualizing fractures of the radial head and coronoid process.

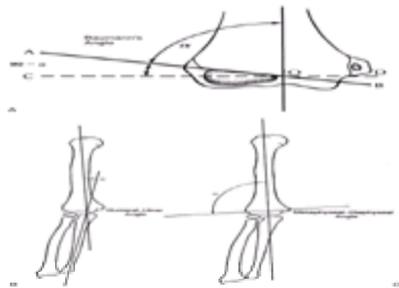


**FIGURE 13-14.** Jones axial radiographic view of the elbow.

### **Anteroposterior Landmarks**

#### **Baumann's Angle**

In the standard anteroposterior view, the major landmark is the angulation of the physal line between the lateral condyle and the distal humeral metaphysis. The ossification center of the lateral condyle extends into the radial or lateral crista of the trochlea ( Fig. 13-9C). This physal line forms an angle with the long axis of the humerus. The angle formed by this physal line and the long axis of the humerus is termed Baumann's angle ( Fig. 13-15A) (1). Baumann's angle is not equal to the carrying angle of the elbow in the older child ( 10). This is a consistent angle when both sides are compared and the x-ray beam is directed perpendicular to the long axis of the humerus.



**FIGURE 13-15.** Anteroposterior radiograph angles of the elbow. **A:** Baumann's angle (a). **B:** The humeral-ulnar angle. **C:** The metaphyseal-diaphyseal angle. (Reproduced from O'Brien WR, Eilert RE, Chang FM, et al. The metaphyseal-diaphyseal angle as a guide to treating supracondylar fractures of the humerus in children. Unpublished data; with permission.)

### Effects of Angulation

Caudad-cephalad angulation of the x-ray tube or right or left angulation of the tube by as much as 30 degrees changes Baumann's angle by less than 5 degrees. If, however, the tube becomes angulated in a cephalad-caudad direction by greater than 20 degrees, the angle is changed significantly and the measurement is inaccurate.

### Effects of Rotation

Camp et al. (12) found in their cadaver studies that rotation of the distal fragment or the entire reduced humerus also can alter the projection of Baumann's angle. They found that to be accurate, the humerus must be parallel to the x-ray plate, with the beam directed perpendicular to the film as well.

Thus, in the routine anteroposterior radiographs of the distal humerus including the Jones view, Baumann's angle is a good measurement of any deviation of the angulation of the distal humerus (16).

### Other Angles

Two other angles are commonly used in anteroposterior radiographs to determine the proper alignment of the distal humerus or carrying angle. The humeral-ulnar angle is determined by lines longitudinally bisecting the shaft of the humerus with the shaft of the ulna ( Fig. 13-15B) (2,28,41). The metaphyseal-diaphyseal angle is determined by a line that longitudinally bisects the shaft of the humerus with a line that connects the widest points of the metaphysis of the distal humerus (see Fig. 13-15C) (40). The humeral-ulnar angle is the most accurate in determining the true carrying angle of the elbow. Baumann's angle also has a good correlation with the clinical carrying angle. However, it may be difficult to measure in the adolescent when the ossification center of the lateral condyle is beginning to fuse with other centers. The metaphyseal-diaphyseal angle is the least accurate of the three (50).

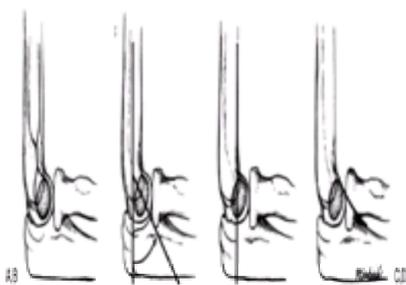
### Lateral Landmarks

#### Teardrop

The lateral projection of the distal humerus presents a teardrop-like shadow above the capitellum (47). The anterior dense line making up the teardrop represents the posterior margin of the coronoid fossa. The posterior dense line represents the anterior margin of the olecranon fossa. The inferior portion of the teardrop is the ossification center of the capitellum. On a true lateral projection, this teardrop should be well defined ( Fig. 13-16A).

#### Shaft-Condylar Angle

On the lateral radiograph, there is an angulation of 40 degrees between the long axis of the humerus and the long axis of the lateral condyle ( Fig. 13-16B). This also can be measured by the flexion angle of the distal humerus, which is calculated by measuring the angle of the lateral condylar physeal line with the long axis of the shaft of the humerus (18).



**FIGURE 13-16.** Lateral radiographic lines of the distal humerus. **A:** The teardrop of the distal humerus. **B:** The angulation of the lateral condyle with the shaft of the humerus. **C:** The anterior humeral line. **D:** The coronoid line.

### Anterior Humeral Line

If a line is drawn along the anterior border of the distal humeral shaft, it should pass through the middle third of the ossification center of the capitellum. This is referred to as the anterior humeral line (Fig. 13-16C). Passage of the anterior humeral line through the anterior portion of the lateral condylar ossification center or anterior to it indicates the presence of posterior angulation of the distal humerus. In a large study of minimally displaced supracondylar fractures, Rogers et al. (44) found that this anterior humeral line was the most reliable factor in detecting the presence or absence of occult fractures.

### Coronoid Line

A line directed proximally along the anterior border of the coronoid process should barely touch the anterior portion of the lateral condyle ( Fig. 13-16D). Posterior displacement of the lateral condyle projects the ossification center posterior to this coronoid line (47).

### Pseudofracture

Some vagaries of the ossification process about the elbow may be interpreted as a fracture (47). For example, the ossification of the trochlea may be irregular, producing a fragmented appearance (Fig. 13-5). This fragmentation can be misinterpreted, especially if the distal humerus is slightly oblique or tilted. These

secondary ossification centers may be mistaken for fracture fragments lying between the semilunar notch and lateral condyle ( [Fig. 13-17](#)).



**FIGURE 13-17.** Pseudofracture of the elbow. The trochlea with its multiple ossification centers may be misinterpreted as fracture fragments lying between the joint surfaces (*arrow*).

On the lateral projection, the physal line between the lateral condyle and the distal humeral metaphysis is wider posteriorly. This may give a misinterpretation that the lateral condyle is fractured and tilted ( [10](#)).

In the anteroposterior projection before the radial head ossifies, there is normally some lateral angulation to the radial border of the neck of the radius that may give the appearance of subluxation ( [Fig. 13-3](#)). The true position of the radial head can be confirmed by noting the relationship of the proximal radius to the ossification center of the lateral condyle on the lateral projection ( [46](#)).

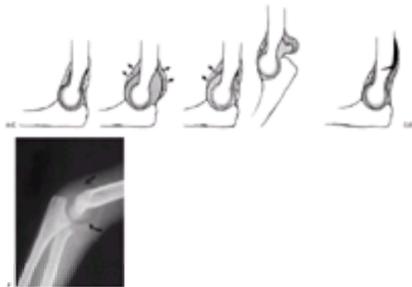
### **Fat Pad Signs of the Elbow**

#### **Three Fat Accumulations**

There are three areas in which fat pads overlie major structures of the elbow. Displacement of any of the fat pads can indicate the presence of an occult fracture. The first two are the fat pads that overlie the capsule in the coronoid fossa anteriorly and the olecranon fossa posteriorly. Displacement of either or both of these fat pads is usually referred to as the classic elbow fat pad sign. A third accumulation of fat overlies the supinator muscle as it wraps around the proximal radius.

#### **Olecranon (Posterior) Fat Pad**

Because the olecranon fossa is deep, the fat pad here is totally contained within the fossa. It is not visualized on a normal lateral x-ray of the elbow flexed to 90 degrees ( [Fig. 13-18A](#)). Distention of the capsule with an effusion, as occurs with an occult intraarticular fracture, a spontaneously reduced dislocation, or even an infection, can cause the dorsal or olecranon fat pad to be visualized ( [56](#)).



**FIGURE 13-18.** Radiographic variations of the elbow fat pads. **A:** Normal relationships of the two fat pads. **B:** Displacement of both fat pads (*arrows*) with an intraarticular effusion. **C:** In some cases, the effusion may displace only the anterior fat pad (*arrows*). **D:** In extension, the posterior fat pad is normally displaced by the olecranon. **E:** An extraarticular fracture may lift the distal periosteum and displace the proximal portion of the posterior fat pad. (Redrawn from Murphy WA, Siegel MJ. Elbow fat pads with new signs and extended differential diagnosis. *Radiology* 1977;124:656–659; with permission.) **F:** An x-ray showing displacement of both fat pads (*arrows*) from an intraarticular effusion.

#### **Coronoid (Anterior) Fat Pad**

Likewise, the ventral or coronoid fat pad may be displaced anteriorly ( [Fig. 13-18B](#)) ( [6](#)). Because the coronoid fossa is shallow, the fat pad in this area projects anterior to the bony margins and can be seen normally as a triangular radiolucency anterior to the distal humerus. Although displacement of the classic elbow fat pads is a reliable indication of an intraarticular effusion, there may be instances in which only one of the fat pads is displaced. Brodeur and colleagues ( [10](#)) and Kohn ( [32](#)) have shown that the coronoid fat pad is more sensitive to small effusions. It can be displaced without a coexistent displacement of the olecranon fat pad ( [Fig. 13-18C](#)).

#### **Supinator Fat Pad**

A layer of fat on the anterior aspect of the supinator muscle wraps around the proximal radius. This layer of fat or fat pad may normally bow anteriorly to some degree. Brodeur et al. ( [10](#)) stated displacement may indicate the presence of an occult fracture of the radial neck. Displacement of the fat line or pad is often difficult to interpret; in a review of fractures involving the proximal radius, Schunk et al. ( [47](#)) found it to be positive only 50% of the time.

#### **Fat Pad Variations**

For the fat pads to be displaced, the capsule must be intact. This can explain why there may be no displacement of the fat pads with an elbow dislocation that has spontaneously reduced because of rupture of the capsule. Murphy and Siegel ( [39](#)) have shown other variations of displacement of the classic fat pads. If the elbow is extended, the fat pad is normally displaced from the olecranon fossa by the olecranon ( [Fig. 13-18D](#)). Distal humeral fractures may cause subperiosteal bleeding and may lift the proximal portion of the olecranon fat pad without the presence of an effusion ( [Fig. 13-18E](#)). These false-negative and false-positive findings must be kept in mind when interpreting the presence or absence of a fat pad with an elbow injury.

#### **Posterior Fat Pad Is Most Reliable**

Corbett's ( [16](#)) review of elbow injuries indicated that if a displacement of the posterior fat pad existed, a fracture was almost always present. Displacement of the anterior fat pad alone, however, could occur without a fracture. Corbett also determined that the degree of displacement bore no relation to the size of the fracture. Skaggs and Mirzayan ( [48](#)) reported that 34 of 45 children (76%) with a history of elbow trauma and an elevated posterior fat pad had radiographic evidence of elbow fractures at an average of 3 weeks after injury, although anteroposterior, lateral, and oblique radiographs at the time of injury showed no other evidence of fracture. They recommended that a child with a history of elbow trauma and an elevated fat pad be treated as if a nondisplaced elbow fracture were present. Donnelly et al.

(19), however, found evidence of fracture in only 9 of 54 children (17%) who had a history of trauma and elbow joint effusion but no identifiable fracture on initial radiographs. They concluded that joint effusion without a visible fracture on initial radiographs does not correlate with the presence of occult fracture in most patients (83%). Persistent effusion did correlate with occult fracture: 78% of those with occult fractures had persistent effusions, compared with 16% of those without fractures.

## Comparison Radiographs

Often it is tempting to order comparison radiographs in a child with an injured elbow because of the difficulty evaluating the irregularity of the ossification process. However, the indications for ordering comparison radiographs are rare. Kissoon et al. (13,31) found that the use of routine comparison radiographs in children with injured elbows did not significantly increase the accuracy of diagnosis, regardless of the interpreter's training.

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## CHAPTER REFERENCES

1. Baumann E. Beitrage zur Kenntnis der Fracturen am Ellbogengelenk. *Bruns Beitr F Klin Chir* 1929;146:1-50.
2. Beals RK. The normal carrying angle of the elbow. *Clin Orthop* 1976;19:194-196.
3. Beekman F, Sullivan JE. Some observations on fractures of long bones in children. *Am J Surg* 1941;51:722-738.
4. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955.
5. Blount WP, Cassidy RH. Fractures of the Elbow in Children. *JAMA* 1951;146:699-704.
6. Bohrer SP. The fat pad sign following elbow trauma. its usefulness and reliability in suspecting "invisible" fractures. *Clin Radio* 1970;21:90-94.
7. Boyd HB, Altenberg AR. Fractures about the elbow in children. *Arch Surg* 1944;49:213-224.
8. Brewster AH, Karp M. Fractures about the elbow in children. An end-result study. *Surg Gynecol Obstet* 1940;71:643-649.
9. Brodeur AE, Silberstein JJ, Graviss ER. *Radiology of the Pediatric Elbow*. Boston: GK Hall, 1981.
10. Brodeur AE, Silberstein JJ, Graviss ER, et al. The basic tenets for appropriate evaluation of the elbow in pediatrics. In: *Current problems in diagnostic radiology*. Chicago: Year Book Medical, 1983.
11. Buhr AJ, Cooke AM. Fracture Patterns. *Lancet* 1959;1:531-536.
12. Camp J, Ishizue K, Gomez M, et al. Alteration of Baumann's angle by humeral position: implications for treatment of supracondylar humerus fractures. *J Pediatr Orthop* 1993;13:521-555.
13. Chacon D, Kissoon N, Brown T, et al. Use of comparison radiographs in the diagnosis of traumatic injuries of the elbow. *Ann Emerg Med* 1992;21:895-899.
14. Cheng JC, Wing-Man K, Shen WY, et al. A new look at the sequential development of elbow-ossification centers in children. *J Pediatr Orthop* 1998;18:161-167.
15. Conn J Jr, Wade PA. Injuries of the elbow: a ten year review. *J Trauma* 1961;1:248-268.
16. Corbett RH. Displaced fat pads in trauma to the elbow. *Injury* 1978;9:297-298.
17. Dai L. Radiographic evaluation of Baumann angle in Chinese children and its clinical relevance. *J Pediatr Orthop* 1999;8:197-199.
18. D'Arienzo M, Innocenti M, Pennisi M. The treatment of supracondylar fractures of the humerus in childhood (cases and results). *Arch Putti Chir Organi Mov* 1983;33:261-269.
19. Donnelly LF, Klostermeier TT, Klosterman LA. Traumatic elbow effusions in pediatric patients: are occult fractures the rule? *AJR* 1998;171:243-245.
20. Eigenmark O. The normal development of the ossific centers during infancy and childhood. *Acta Paediatr Scand Suppl* 1946;33.
21. Fahey JJ. Fractures of the elbow in children. *Instr Course Lect* 1960;17:13-46.
22. Francis CC. The appearance of centers of ossification from 6-15 years. *Am J Phys Anthropol* 1940;27:127-138.
23. Gray DJ, Gardner E. Prenatal development of the human elbow joint. *Am J Anat* 1951;88:429-469.
24. Hanlon CR, Estes WL. Fractures in childhood: a statistical analysis. *Am J Surg* 1954;87:312-323.
25. Haraldsson S. The intraosseous vasculature of the distal end of the humerus with special reference to capitulum. *Acta Orthop Scand* 1957;27:81-93.
26. Haraldsson S. On osteochondrosis deformans juvenilis capituli humeri including investigation of intra-osseous vasculature in distal humerus. *Acta Orthop Scand Suppl* 1959;38.
27. Henrikson B. Supracondylar fracture of the humerus in children. *Acta Chir Scand Suppl* 1966;369.
28. Ippolito E, Caterini R, Scola E. Supracondylar fractures of the humerus in children. Analysis at maturity of 53 patients treated conservatively. *J Bone Joint Surg [Am]* 1986;68:333-344.
29. Jenkins F. The functional anatomy and evolution of the mammalian humeroulnar articulation. *Am J Anat* 1973;137:281-298.
30. Johansson O. Capsular and ligament injuries of the elbow joint. *Acta Chir Scand Suppl* 1962;287.
31. Jones R. A note on the treatment of injuries about the elbow. *Provincial Med J* 1895;1:28-30.
32. Kissoon N, Galpin R, Gayle M, et al. Evaluation of the role of comparison radiographs in the diagnosis of traumatic elbow injuries. *J Pediatr Orthop* 1995;15:449-453.
33. Kohn AM. Soft tissue alterations in elbow trauma. *AJR* 1959;82:867-874.
34. Laing PG. The arterial supply of the adult humerus. *J Bone Joint Surg [Am]* 1956;38:1105-1116.
35. Landin LA. Fracture patterns in children. Analysis of 8682 fractures with special reference to incidence, etiology and secular changes in a Swedish urban population, 1950-1979. *Acta Orthop Scand Suppl* 1983;54.
36. Landin LA, Danielsson LG. Elbow fractures in children: an epidemiological analysis of 589 cases. *Acta Orthop Scand* 1986;57:309.
37. Lichtenberg RP. A study of 2532 fractures in children. *Am J Surg* 1954;87:330-338.
38. Marion J, Lagrange J, Faysse R, et al. Les fractures de l'extremite inferieure de l'humerus chez l'enfant. *Rev Chir Orthop* 1962;48:337-413.
39. Maylahn DJ, Fahey JJ. Fractures of the elbow in children. *JAMA* 1958;166:220-228.
40. Murphy WA, Siegel MJ. Elbow fat pad with new signs and extended differential diagnosis. *Radiology* 1977;124:659-665.
41. O'Brien WR, Eilert RE, Chang FM, et al. The metaphyseal diaphyseal angle as a guide to treating supracondylar fractures of the humerus in children. (Unpublished.)
42. O'Donoghue DH, Stanley L. Persistent olecranon epiphyses in adults. *J Bone Joint Surg* 1942;24:677-680.
43. Peterson CA, Peterson HA. Analysis of the incidence of injuries to the epiphyseal growth plate. *J Trauma* 1972;12:275-281.
44. Porteous CJ. The olecranon epiphyses. *J Anat* 1960;94:286.
45. Rogers LF, Malave S Jr, White H, et al. Plastic bowing, torus, and greenstick fractures of the humerus. *Radiology* 1978;128:145-150.
46. Sandegrad E. Fracture of the lower end of the humerus in children: treatment and end results. *Acta Chir Scand* 1944;89:1-16.
47. Schunk VK, Grossholz M, Schild H. Der Supinatorfettkorper bei Frakturen des Ellbogengelenkes. *ROFO* 1989;150:294-296.
48. Silberstein JJ, Brodeur AE, Graviss ER. Some vagaries of the capitellum. *J Bone Joint Surg [Am]* 1979;61:244-247.
49. Skaggs DL, Mirzayan R. The posterior fat pad sign in association with occult fracture of the elbow in children. *J Bone Joint Surg [Am]* 1999;81:1429-1433.
50. Smith L. Deformity following supracondylar fractures of the humerus. *J Bone Joint Surg [Am]* 1960;42:235-252.
51. Webb AJ, Sherman FC. Supracondylar fractures of the humerus in children. *J Pediatr Orthop* 1989;9:315-325.
52. Wilkins KE. Fractures and dislocations of the elbow region. In: Rockwood CA Jr, Wilkins KE, King RE, eds. *Fractures in children*, 3rd ed. Philadelphia: JB Lippincott, 1991:509-828.
53. Wilkins KE. Fractures and dislocations of the elbow region. In: Rockwood CA Jr, Wilkins KE, Beaty JH, eds. *Fractures in children*, 4th ed. Philadelphia: Lippincott-Raven, 1996:653-904.
54. William PL, Warwick R. *Gray's anatomy*. Philadelphia: WB Saunders, 1980.
55. Wilson PD. Fractures and dislocations in the region of the elbow. *Surg Gynecol Obstet* 1933;56:335-359.
56. Yang Z, Wang Y, Gilula LA, et al. Microcirculation of the distal humeral epiphyseal cartilage: implications for post-traumatic growth deformities. *J Hand Surg [Am]* 1998;23:165-172.
57. Zanella FE. Injuries of the elbow and forearm in children. *Rontgenblatter* 1984;37:111.

# 14 SUPRACONDYLAR FRACTURES OF THE DISTAL HUMERUS

JAMES R. KASSER  
JAMES H. BEATY

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[Extension-Type Supracondylar Fractures](#)  
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We thank Kaye Wilkins for his contribution to this chapter and previous editions. Much of this chapter is his effort.

Between the mid-20th century, when early reports of pinning of fractures of the distal humerus first appeared, and now, treatment of supracondylar fractures has evolved tremendously. Blount's caution against operative management (26) has given way to modern concepts of treatment involving skeletal stabilization and soft tissue management, which have greatly improved outcome (29,48,69,147,181).

Both cost and clinical result have been significantly affected over the past 25 years (176). Problems of vascular compromise, Volkmann ischemic contracture, and neurovascular entrapment in the fracture callus have been greatly decreased, but not eliminated.

## INCIDENCE AND GENERAL CONSIDERATIONS

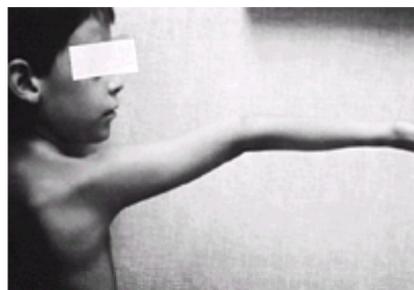
The peak age at which supracondylar fractures occur is between 5 and 7 years. The rate of occurrence increases steadily in the first 5 years of life, and traditionally boys have had a higher incidence of this fracture than girls. Combining 61 reports of supracondylar fractures totaling 7,212 displaced fractures of the distal humerus yields a consistent pattern, as shown in Table 14-1. Boys have outnumbered girls by about 3 to 2. The average age at fracture is 6.7 years. The left or nondominant side predominates in almost all studies. Two thirds of children hospitalized with elbow injuries have supracondylar fractures. Nerve injury occurs in at least 7%. The radial nerve has been the most frequently involved nerve in older studies; however, the median nerve is much more commonly injured, particularly the anterior interosseous nerve (AIN), in more recent studies (49,174). The ulnar nerve is most commonly injured iatrogenically during pinning or in a flexion-type of supracondylar fracture. It is possible that there has been a change in the pattern of displacement of these fractures, accounting for the change in frequency of median nerve injuries, but it is more likely that the subtle loss of thumb and finger flexion that indicates an AIN injury is more frequently recognized.

Incidence	Percentage of Total Number of Fractures (%)	Nerve Involved	Percentage of Total Nerve Injuries (%) <sup>a</sup>
Side involved		Radial	41.2
Right	35.2	Median	36.0
Left	63.8	Ulnar	22.8
Sex incidence			
Male	62.8		
Female	37.2		
Isolated fractures	1.0		
Open fractures	1.0		
Volkmann contracture	0.5		
Flexion type	2.0		
Fractures with nerve injuries	7.7		

Data were compiled from 7212 fractures occurring in 61 major series.  
Data from References: 1, 5, 11, 15, 17, 18, 22, 23, 27, 28, 34, 41, 45, 47, 51, 53, 55, 56, 60-63, 64, 68, 83, 84, 85, 86, 87, 88, 89, 92, 93, 94, 97, 100, 105, 114, 117, 118, 124, 130, 132, 134, 136, 137, 144, 145, 148, 152, 153, 156, 167, 168, 173, 180, 183, 184, 186, 187, 188, 195, 197.  
<sup>a</sup>Average age: mean 6.7 years.

**TABLE 14-1. EFFICIENCY OF SUPRACONDYLAR FRACTURES**

Increased ligamentous laxity (Fig. 14-1) has been correlated with the occurrence of supracondylar fractures (25,139) and with ulnar nerve subluxation (198). Supracondylar fractures are more likely in children with ulnar nerve subluxation, which makes the nerve vulnerable in medial pinning techniques.



**FIGURE 14-1. Hyperflexibility.** At the peak age for supracondylar fractures there is a naturally occurring hyperextension of the elbow, which predisposes the distal humerus to this type of fracture. (Reprinted from Wilkins KE. The operative management of supracondylar fractures. *Orthop Clin North Am* 1990;21:269-289; with permission.)

Almost all supracondylar fractures are caused by accidental trauma. A fall from a height accounts for 70% of all supracondylar fractures (64). In children under 3 years of age, the fracture usually occurs in a fall from a bed, from furniture, or down stairs. In children over 3 years of age, the fracture generally results from a fall from

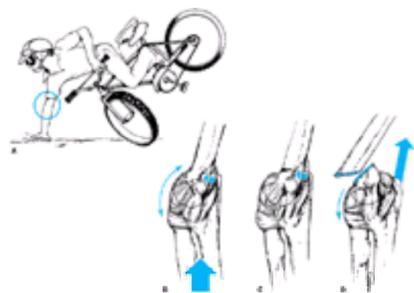
monkey bars, swings, or other playground equipment. In one study of 99 patients with supracondylar fractures seen over a 15-month period, only 1 was secondary to abuse (175). When a child under 15 months of age has a supracondylar fracture of the distal humerus, abuse should be considered; with a reasonable history, however, such a cause is unlikely.

The most commonly associated fractures are distal radial fractures, but fractures of the scaphoid and proximal humerus do occur. Pulse is absent at presentation in 12% to 15% of patients, but vascular insufficiency requiring operative intervention is relatively rare (32,36,44,162) (2%–4%). Volkmann ischemic contracture is rare, occurring in about 0.5% of patients. Most distal humeral supracondylar fractures are displaced in extension, but flexion-type injuries do occur.

## EXTENSION-TYPE SUPRACONDYLAR FRACTURES

### Mechanism of Injury

Supracondylar fractures generally occur as a result of a fall onto the outstretched hand with the elbow in full extension (40) (Fig. 14-2). The flexed elbow may be stabilized by the biceps, brachialis, and triceps muscles, but once extended beyond neutral, the elbow flexor muscles are at a poor mechanical advantage and there is little resistance to injury. The olecranon in its fossa in the distal humerus acts as a fulcrum, whereas the capsule transmits an extension force to the distal humerus just proximal to the physis as the elbow hyperextends. The capsular insertion, distal to the olecranon fossa and proximal to the physis, is critical in producing a consistent failure pattern of a supracondylar fracture in children. In full extension the elbow becomes tightly interlocked, concentrating bending forces in the distal humerus (133).



**FIGURE 14-2.** Hyperextension Forces. **A:** Most young children attempt to break their falls with the upper extremity extended. Because of the laxity of the ligaments, the elbow becomes locked into hyperextension. **B:** This converts the linear applied force (*large arrow*) to an anterior tension force. Posteriorly, the olecranon is forced into the depths of the olecranon fossa (*small arrow*). **C:** As the bending force continues, the distal humerus fails anteriorly in the supracondylar area. **D:** When the fracture is complete, the distal fragment becomes posteriorly displaced. The strong action of the triceps (*large arrow*) produces proximal displacement of the distal fragment.

Posterior displacement of the distal fragment occurs, with the proximal or metaphyseal fragment impaling the anterior soft tissue structures. The fracture in the sagittal plane was classically described by Kocher (106) as extending obliquely from anterior and distal to posterior and proximal. However, clinical studies by Holmberg (91) and Nand (137) have demonstrated that the fracture pattern is transverse on lateral radiographs in more than 80% of patients (Fig. 14-3). The fracture line in the anteroposterior (AP) view extends transversely across both the medial and lateral columns of the distal humerus at the level of the middle of the olecranon fossa.



**FIGURE 14-3.** Orientation of fracture lines. **A:** The typical transverse fracture line originates just above the epicondyles and courses through the supracondylar area (*arrows*). **B:** In the lateral projection, the fracture line is usually also transverse.

### Role of the Periosteum

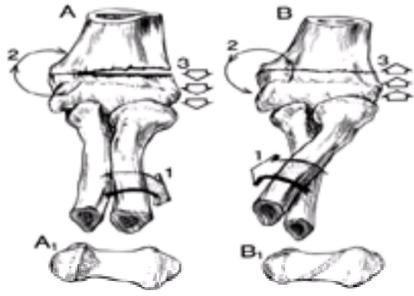
As the supracondylar fracture displaces posteriorly, the anterior periosteum fails and tears away from the displaced distal fragment. The anterior loss of periosteal integrity leads to frequent failure of anterior callus formation in early fracture healing, which is of little significance clinically (Fig. 14-4). Further fracture displacement is accompanied by corresponding increased periosteal disruption with decreased fracture stability.



**FIGURE 14-4.** Periosteal changes. Radiographs taken 2 months after fracture. Lateral view demonstrates a persistent gap (*white arrow*) in the anterior cortical margin where interposed periosteum prevented anatomic reduction. Note the absence of periosteal new bone anteriorly on the proximal fragment, although there is abundant periosteal new bone (*black arrows*) on the posterior surface.

Intact medial or lateral periosteum, the periosteal hinge, has been said to provide stability after fracture reduction (81,103,161). Pronation of the forearm (13,104) after reduction of a posteromedially displaced supracondylar fracture is said to stabilize reduction by closing the fracture gap laterally, tensioning the medial periosteal hinge, and tightening the lateral ligaments of the elbow (Fig. 14-5). Conversely, a laterally displaced supracondylar fracture is more stable in supination. Experimental

data by Abraham (2) questioned this generally accepted concept, suggesting that little stability results from an intact periosteal hinge. If the periosteum is intact medially and laterally in a supracondylar fracture with pure posterior displacement, it may yield a very stable reduction.

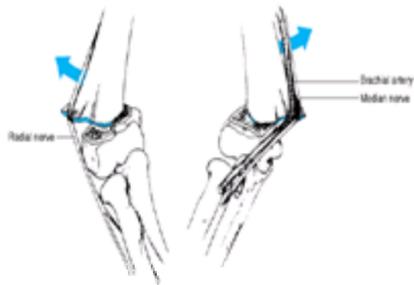


**FIGURE 14-5.** Intraarticular stabilizing forces. **A:** Supination of the forearm ( 1) creates a downward lateral tilt of the distal fragment ( 3). This produces compressive forces between the articulating surface of the ulna and the medial border of the trochlea ( A1, stippled area), which in turn generates clockwise forces ( 2) about the medial side of the fracture. **B:** Pronation of the forearm ( 1) creates an upward tilt of the distal fragment ( 3) because of the compressive forces between the articulating surface of the ulna and the lateral border of the trochlea ( B1, stippled area), which in turn generates counterclockwise forces ( 2) about the medial side of the fracture. (Reprinted from Abraham E, Powers T, Witt P, et al. Experimental hyperextension supracondylar fractures in monkeys. *Clin Orthop* 1982;171:309-318; with permission.)

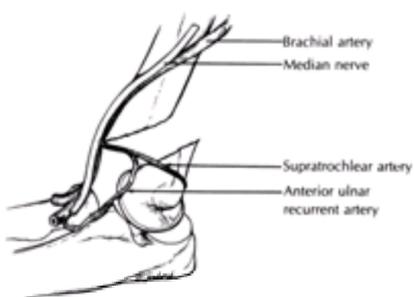
### Posteromedial versus Posterolateral Displacement of Extension-Type Supracondylar-Type Fractures

Generally, medial displacement of the distal fragment is more common than lateral displacement, occurring in approximately 75% of patients in most series ( 191). However, in one series with a high preponderance of median nerve and brachial artery injuries, 47% of patients had posterolateral displacement of the distal humeral fragment (36). The biceps tendon insertion and axis of muscle pull lies medial to the shaft of the humerus, and Holmberg ( 91) suggested that this anatomic location of muscle pull created a force that tended to displace the distal humeral fragment medially.

The position of the hand and forearm at the time of injury plays a role in the direction of displacement of the distal humeral fragment. In a patient who falls onto an outstretched supinated arm, the forces applied tend to disrupt the posteromedial periosteum first and displace the fragment posterolaterally. Conversely, if a patient falls with the arm pronated, the distal fragment tends to become displaced posteromedially. Whether the displacement is medial or lateral is important because it determines which soft tissue structures are at risk from the penetrating injury of the proximal metaphyseal fragment. Medial displacement of the distal fragment places the radial nerve at risk, and lateral displacement of the distal fragment places the median nerve and brachial artery at risk ( Fig. 14-6). The brachial artery and median nerve may become entrapped in the fracture site with lateral displacement, but they are highly unlikely to become entrapped with the distal fragment displaced medially. The brachial artery is placed further at risk by the ulnar-sided tether of the supratrochlear artery ( Fig. 14-7).



**FIGURE 14-6.** Neurovascular relations. **Left:** If the distal spike penetrates the brachialis muscle laterally (posteromedial fractures), the radial nerve may be tethered. **Right:** If the spike penetrates medially (posterolateral fractures), both the median nerve and brachial artery can be tethered.



**FIGURE 14-7.** Arterial pathology. The supratrochlear branch that arises from the anterior ulnar recurrent artery may bind the main trunk of the brachial artery against the sharp end of the proximal fragment. (Redrawn from Rowell PJW. Arterial occlusion in juvenile humeral supracondylar fracture. *Injury* 1974;6:254-256; with permission.)

The Gartland classification of supracondylar fractures is currently the most commonly used system based on its prevalence in the literature and use in pediatric fracture texts. It is easy to use and facilitates treatment decisions and communication. The Gartland ( 74) classification ( Table 14-2) is based on the radiographic appearance of fracture displacement. A type I or nondisplaced fracture ( Fig. 14-8) has an anterior humeral line that intersects the capitellum, an intact olecranon fossa, no medial or lateral displacement, no medial column collapse, and a normal

Type I	Undisplaced
Type II	Displaced (with intact posterior cortex)
Type III	Displaced (no cortical contact)
	Posteromedial
	Posterolateral

**TABLE 14-2. CLASSIFICATION OF SUPRACONDYLAR FRACTURES**

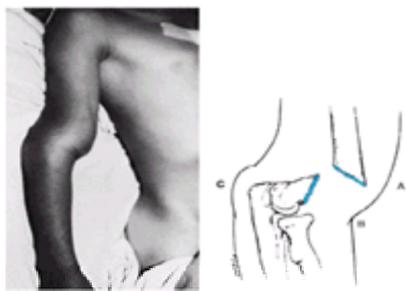


**FIGURE 14-8.** Types of supracondylar fractures. **A:** Type I fracture, where the anterior humeral line (*long arrows*) crosses through the ossification center of the capitellum. There is also posterior displacement of the olecranon fat pad (*large arrows*). **B:** Three weeks postinjury, there is evidence of new periosteal bone formation from both the anterior and posterior cortices (*arrows*). Because a definite fracture line was not seen in the original radiographs, this new bone formation confirms the original suspicion of a fracture. **C:** Type II. Lateral view of a displaced supracondylar fracture with the posterior cortex intact. There is both rotation and angulation of the distal fragment. **D:** Type III. Totally displaced fracture. There is no contact between the fragments.

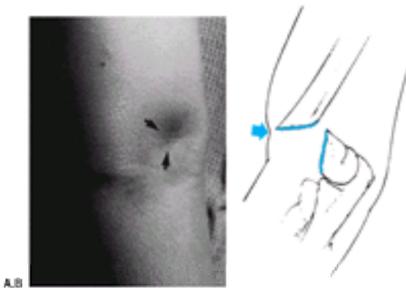
Baumann angle. A type II fracture is extended but not translated with an intact posterior cortex. The anterior humeral line does not intersect the capitellum. Some rotational displacement and tilt into varus, as determined by the Baumann angle, may be present. A type III fracture has a circumferential break in the cortex with displacement of the fracture fragments. In the common extension type supracondylar fracture, the distal fragment is displaced posteriorly with the metaphyseal fragment impaled into the brachialis muscle and anterior soft tissues. The radiographic appearance of the distal fragment may be highly variable depending on (a) the degree of ossification of the distal humeral epiphysis, (b) size of the ossified metaphyseal fragment, and (c) position of flexion and rotation of the distal humeral fragment.

### Signs and Symptoms

An elbow or forearm fracture should be suspected in a child with elbow pain or failure to use the upper extremity after a fall. Initial radiographs should include the entire extremity, because multiple fractures may be present even with what seems like minor trauma. In children with elbow pain and failure to use the upper extremity, the differential diagnosis should include occult fracture, nursemaid's elbow, and infection. With a clear history of a "pulling type" of injury, manipulation for a nursemaid's elbow may be performed before a radiograph is obtained. In general, if the history is not clear or there is any question of a fall onto an outstretched hand as the mechanism of injury, a radiograph should always be obtained before elbow manipulation. With a type I supracondylar fracture, there may be distal humeral tenderness, lack of the anconeus soft spot (elbow effusion), restriction of motion, and evidence of bruising. In type III fractures, gross displacement (Fig. 14-9) of the elbow is evident. An anterior pucker sign (191) may be present if the proximal fragment has penetrated the brachialis and the anterior fascia of the elbow (Fig. 14-10). When the proximal fragment is disengaged from its pucker in the skin, there is sometimes bleeding with signs of a grade I open fracture. Careful motor, sensory, and vascular examinations should be performed in all patients. This may be quite difficult in a young child but should be attempted. Sensation should be tested in discrete sensory areas of the radial nerve (dorsal first web space), medial nerve (palmar index finger), and ulnar nerve (palmar little finger). Motor examination should include finger, wrist, and thumb extension (radial nerve), index distal interphalangeal and thumb interphalangeal flexion (AIN), and thenar (median) and interosseus (ulnar nerve) muscle function. The vascular examination should include determining the presence of pulse, as well as warmth, capillary refill, and color of the hand. Tenseness of the volar compartment should be evaluated, and the amount of swelling about the elbow should be noted. Passive finger extension and flexion should be tested and findings accurately recorded. In the initial examination of a child with a severe supracondylar fracture with high parental and patient anxiety, it is easy to overlook vital information. However, because further decision making is dependent on an accurate initial examination, care should always be taken to obtain all of the above information as accurately as possible. When the elbow injury is obvious, its examination should be delayed until the proximal humerus and distal radius have been fully examined for associated or occult fractures.



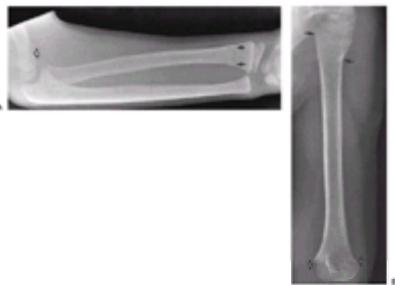
**FIGURE 14-9.** Clinical appearance. The S-shaped configuration is created by the prominence of the spike of the proximal fragment (A), flexion of the distal fragment (B), and the posterior prominence of the olecranon (C).



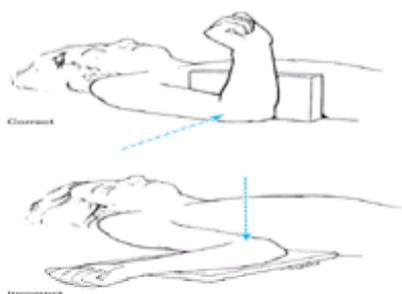
**FIGURE 14-10.** The pucker sign. This patient had penetration of the spike of the proximal fragment into the subcutaneous tissue. In the anteroposterior view (A), there is a large puckering or defect in the skin where the distal fragment has pulled the skin inward. Laterally (B), there is puckering of the skin (*arrow*) in the area where the spike has penetrated into the subcutaneous tissue.

### Radiologic Evaluation

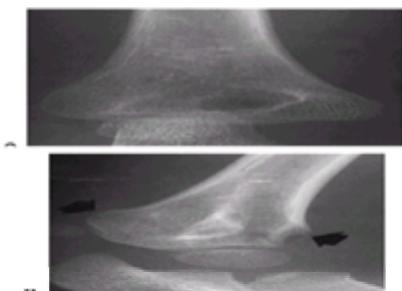
All patients with a history of a fall onto an outstretched hand and pain and inability to use the extremity should undergo a thorough radiologic evaluation. This may include obtaining AP views of the entire upper extremity ( [Fig. 14-11](#) ). Comparison views rarely are required by an experienced physician, but occasionally may be needed in the evaluation of an ossifying epiphysis. The AP radiograph should always be taken as an AP of the distal humerus rather than an AP of the elbow in which the elbow is held in flexion. This allows more accurate evaluation of the distal humerus and decreases the error in determining angular malalignment in the distal humerus. It also allows better evaluation of the olecranon fossa as an indication of injury in the distal humerus. The lateral film should be taken as a true lateral with the humerus held in the anatomic position and not externally rotated ( [Fig. 14-12](#) ). Oblique views of the distal humerus ( [Fig. 14-13](#) ) occasionally may be helpful when a supracondylar fracture or occult lateral condylar fracture is suspected but not seen on standard AP and lateral views, but should not be routinely ordered in the evaluation of an elbow injury.



**FIGURE 14-11.** Occult ipsilateral fractures. **A:** Type II supracondylar fracture ( *open arrow* ) with an occult distal radial fracture ( *solid arrows* ). **B:** Type II supracondylar fracture ( *open arrows* ) with an occult proximal humeral fracture ( *solid arrows* ).



**FIGURE 14-12.** Radiographic positioning. The correct method of taking a lateral view is with the upper extremity directed anteriorly rather than externally rotated.



**FIGURE 14-13.** Oblique views. Often, the fracture line is not visualized on any of the lateral or anteroposterior views ( **A** ). An oblique view ( **B** ) of the distal humerus may demonstrate the extent of the fracture line ( *arrows* )

If the AP and lateral views show a displaced type II or III supracondylar fracture but do not show full detail of the distal humeral fragment, we usually obtain further radiologic evaluation in the operating room with the patient anesthetized. Repeat trips for radiologic evaluation in the emergency setting generally result in increased pain without significant improvement in radiograph quality. Detailed radiographs need to be obtained at some point, however, to define the fracture anatomy with particular emphasis on (a) impaction of the medial column, (b) supracondylar comminution, and (c) vertical split of the epiphyseal fragment. T-condylar fractures ( [Fig. 14-14](#) ) can initially appear to be supracondylar fractures, but these generally occur in children 8 to 10 years of age, in whom supracondylar fractures are less likely, although T-condylar fractures have been reported in young children.



**FIGURE 14-14.** Occult T-condylar. **A:** Original radiographs appear to show a type III posteromedial supracondylar fracture. **B:** After manipulation, the vertical intercondylar fracture line ( *arrows* ) was visualized.

In a young child, an epiphyseal separation ( [55,56,200](#) ) can mimic an elbow dislocation. In an epiphyseal separation, the fracture propagates through the physis without a metaphyseal fragment. This fracture occurs in very young children with primarily chondral epiphyses. On physical examination, the patient appears to have a supracondylar fracture with gross swelling about the elbow and marked discomfort. The key to making the diagnosis and differentiating this injury from an elbow dislocation is seeing that the capitellum moves with the radial head. Sometimes a supracondylar fracture with a small metaphyseal fragment can mimic a lateral condylar fracture ( [Fig. 14-15](#) ). In such cases, more data are required to initiate treatment. An arthrogram may be helpful to determine the extent of the elbow injury. In

selected patients, magnetic resonance imaging or ultrasonography ([200](#)) also may aid in evaluation of injury to the unossified epiphysis.



**FIGURE 14-15.** This 1.2-year-old girl sustained a fracture that on the anteroposterior view **(A)** appears like an elbow dislocation and on the lateral view **(B)** has the appearance of a lateral condyle fracture. **(C)** Arthrography showed the outline of the entire cartilaginous epiphysis. This is an epiphyseal separation with a metaphyseal fragment (Salter II).

## Treatment

### General Principles

Initial management of all patients suspected of having an elbow injury is splinting in a comfortable position, generally 20 to 30 degrees of elbow flexion, pending careful physical examination and radiologic evaluation. The initial responder to a child with an elbow injury and polytrauma should make an assessment of neurovascular status and other injuries, including but not limited to head injury and general cardiorespiratory status. Excessive flexion or forced extension should be avoided, because both may lead to vascular compromise.

### Type I (Nondisplaced)

The distal humeral injury may not be the primary problem: neurologic compromise has been reported with nondisplaced supracondylar fractures ([160](#)). Radiographs of the entire extremity should be reviewed to be sure that there is no injury in other parts of the humerus or in the forearm bones. On radiography, a type I supracondylar fracture has a visible fracture line traversing the medial and lateral columns at the level of the olecranon fossa without displacement. The anterior humeral line transects the capitellum. In general, in a type I fracture, the periosteum is intact with significant inherent stability of the fracture.

Simple immobilization with a posterior splint applied in 90 degrees with side supports or simple collar and cuff is all that is necessary ([40,193](#)). This allows swelling to occur and does not put the brachial artery at risk of compression. Mapes and Hennrikus ([121](#)), using Doppler examination of the brachial artery after supracondylar fractures, found that flow was decreased in the brachial artery in positions of pronation and increased flexion. A simple splint with the forearm in neutral position and the elbow flexed no more than 90 degrees should be used initially. Before the splint is applied, it should be confirmed that the pulse is intact and that there is good capillary refill. A circumferential cast may be applied if additional fractures require treatment, but careful observation is necessary to avoid any risk of compartment syndrome.

Radiographs are obtained 3 to 7 days after fracture to document lack of displacement, and a long arm cast can be applied with a ring over the distal portion of the cast and a sling around the neck to support the weight of the cast ([Fig. 14-16](#)). Without support, the weight of the cast applies an extension torque about the distal humerus, leading to fracture displacement. If there is any evidence of extension of the distal fragment, as judged by lack of intersection of the anterior humeral line with the capitellum, the fracture should be reduced with hyperflexion of the elbow to 120 degrees with pinning. The most common cause of cubitus varus deformity is inadequate treatment of types I and II fractures, rather than the deformity complicating type III fractures. An acceptable position is determined by the anterior humeral line transecting the capitellum, a Baumann angle of 70 to 78 degrees or equal to the other side, and an intact olecranon fossa. Medial column collapse can lead to varus deformity in an otherwise minimally displaced and normally healing distal humeral fracture. The duration of immobilization for supracondylar fractures is about 3 weeks, whether type I, II or III. In general, no physical therapy is required after this injury. Patients are seen at 2 to 4 weeks after immobilization is removed to be sure that range of motion and strength are returning normally.



**FIGURE 14-16.** Because the weight of the cast can produce an extension movement about the distal humerus, a sling to support the weight of the cast is necessary.

### Type II Fracture (Displaced with an Intact Posterior Cortex)

This fracture category encompasses a broad array of distal humeral hyperextension injuries, depending on the degree of rotation. Even greater than the variation in fracture stability is the spectrum of soft tissue injuries with type II supracondylar fractures. Careful assessment of the soft tissue injury is critical in treatment decision making. Radiologic definition of this injury demonstrates an incomplete osseous separation with some posterior cortical contacts. Therefore, good stability should be obtained with closed reduction. Medial column collapse ([Fig. 14-17](#)) or buckling must be identified because a varus deformity may result from a simple closed reduction without stabilization when these are present. For closed reduction, traction is applied first, followed by correction of rotational deformity. The extension deformity is corrected with pressure by the surgeon's thumb over the olecranon and posterior humeral condyles. The alignment of the distal humeral fragment is verified in AP and lateral views. With the elbow held in hyperflexion, Jones' views, rotating the arm slightly to expose the medial and lateral column of the distal humerus, are valuable to document reduction.

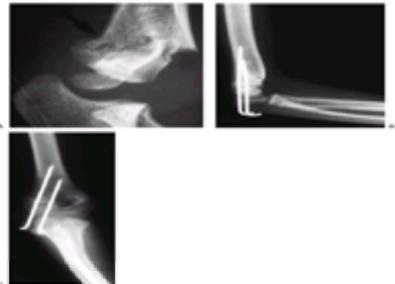


**FIGURE 14-17.** Greenstick medial collapse. **A:** An anteroposterior view of a type II fracture showing greater collapse of the weaker medial column ( *arrows*). **B:** Lateral view showing the posterior cortex is still partially intact. (Reprinted from Wilkins KE. The management of severely displaced supracondylar fractures of the humerus. *Techniques Orthop* 1989;4:12-24; with permission.)

Millis (132), as well as others (13,26,86,94,147,161), have shown the necessity of elbow hyperflexion to more than 120 degrees to maintain reduction of an extension-type supracondylar fracture without pinning ( Fig. 14-18). Therefore, the decision must be made to either place Kirschner wires in the distal humerus to stabilize the fracture, allowing immobilization in a comfortable position of less than 90 degrees of elbow flexion, or to place the child in a cast with the elbow in 120 degrees of flexion. The presence of significant swelling, obliteration of pulse with flexion, neurovascular injuries, and other injuries in the same extremity are indications for pin stabilization of a type II fracture. If pinning is chosen, two lateral pins ( 42,181) through the distal humeral fragment, engaging the opposite cortex of the proximal fragment, are generally sufficient to maintain fracture alignment ( Fig. 14-19). Some degree of inherent stability is provided by the posterior cortex and the intact periosteum. Cross pinning of this fracture rarely is needed. The techniques for crossed and lateral pinning are described later on in this chapter. If pin stabilization is used, the pins are left protruding through the skin and are removed at 3 to 4 weeks after fixation, generally without the need for sedation or anesthesia.



**FIGURE 14-18.** Necessity for hyperflexion. **A:** Injury film. **B:** Hyperflexion of the elbow adequately reduced the fracture. **C:** When the elbow is extended to only 90 degrees, the reduction is lost. (Reprinted from Wilkins KE. The management of severely displaced supracondylar fractures of the humerus. *Techniques Orthop* 1989;4:12-24; with permission.)



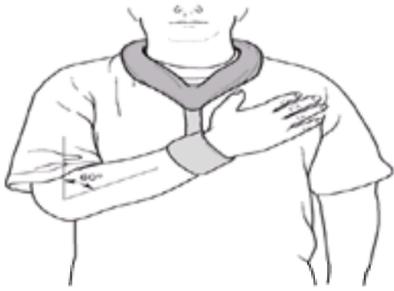
**FIGURE 14-19.** This type II supracondylar fracture (**A**) is stable after closed reduction and is ideal for pinning with two lateral pins (**B and C**) placed parallel and engaging the opposite cortex.

If cast immobilization is chosen, the cast should be a figure-of-eight ( Fig. 14-20) cast sparing the antecubital fossa. We generally make this with fiberglass to allow better radiographic evaluation with the cast in place. Hadlow ( 83) reported that closed reduction and casting were satisfactory without pinning in 37 of his 48 patients.



**FIGURE 14-20.** Figure-of-eight wrap. In the figure-of-eight cast, both the padding and the plaster are wrapped in a figure-of-eight manner ( *arrows*). (Reprinted from Wilkins KE. The management of severely displaced supracondylar fractures of the humerus. *Techniques Orthop* 1989;4:5-24; with permission.)

An alternative method of closed management is the use of a collar and cuff ( 40) ( Fig. 14-21). The advantages of this treatment are a lack of circumferential rigid casting and the avoidance of the weight of the cast, which may produce an extension moment. In this technique, the elbow is held in hyperflexion by the collar and cuff, but not in a rigid manner, which decreases the risk of vascular compromise. After 3 weeks of immobilization, the patient is allowed to begin active motion out of the collar and cuff.



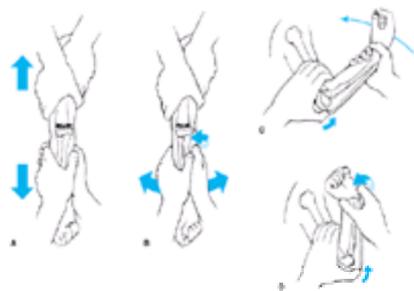
**FIGURE 14-21.** A collar and cuff is a safe treatment that can be used to maintain reduction in hyperflexion.

### **Type III Extension Supracondylar Fracture**

In this fracture the periosteum is torn, there is no cortical contact between the fragments, and soft tissue injury may accompany the fracture. Careful preoperative evaluation is mandatory. If circulatory compromise is indicated by absent pulse and a pale hand, or if compartment syndrome is suspected, immediate reduction and skeletal stabilization are mandatory. Alternative methods of management, including traction and closed reduction with collar and cuff or casting can be used in the absence of acute vascular insufficiency. There are two viable alternatives for achieving fracture alignment: acute manipulative reduction and traction.

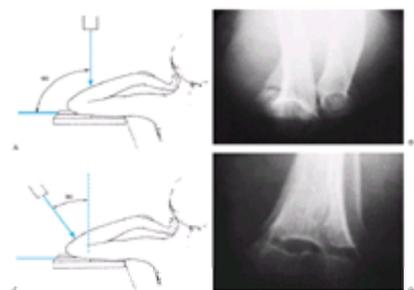
### **Technique of Reduction**

Whether a fracture is held in place with pins or a cast, realignment of the humerus is necessary and the technique of manipulative reduction is the same ( Fig. 14-22). The patient is anesthetized and the affected arm is extended over the screen of the C-arm fluoroscopic unit. Longitudinal traction is applied first to dislodge the proximal fragment, which may be entrapped in the brachialis muscle, the antecubital fascia, or the superficial fat and skin. If traction does not restore length and alignment, a “milking maneuver” has been described by Archibeck ( 11) and Peters (145) to disengage the proximal fragment from the soft tissue. This is done by manipulating the soft tissue over the fracture to pull the soft tissue away from the proximal fragment rather than simply applying traction on the bones, which may not allow reduction of a buttonholed proximal fragment. Once length is restored, medial or lateral translation is corrected. The image intensifier is helpful for this because the medial and lateral columns should be realigned on the AP image. Rotation is corrected simultaneously, but in general, malrotation resolves as traction is applied and the medial and lateral alignment corrected, because of the effect of the surrounding soft tissue. A flexion reduction maneuver is then performed with pressure of the thumb over the olecranon and to a variable degree over the distal condyles of the humerus. Generally the fracture reduction can be felt, and the elbow is then held in hyperflexion and pronation to achieve a stable reduction.



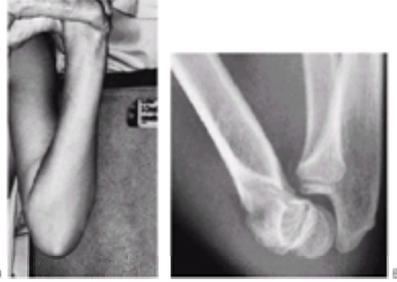
**FIGURE 14-22.** Manipulative closed reduction. **A:** Traction is applied with the elbow in extension and the forearm in supination. The assistant stabilizes the proximal fragment. After traction has been applied and the length regained, the fracture is hyperextended to obtain apposition of the fragments. **B:** With traction being maintained, the varus or valgus angulation along with the rotation of the distal fragment is corrected. **C:** Once the length and alignment have been corrected, the elbow is flexed. Pressure is applied over the posterior aspect of the olecranon to facilitate reduction of the distal fragment. **D:** The distal fragment is finally secured to the proximal fragment by pronating the forearm.

Anteroposterior and lateral views are obtained using the image intensifier. With the elbow flexed, a pure AP view is nearly impossible to interpret, so the actual AP view is taken by rotating the arm slightly medially and laterally to view the columns of the distal humerus and the reduction of the fracture ( Fig. 14-23). The arm is then externally rotated to obtain a lateral view of the distal humerus. The surgeon rotates the entire arm by placing one hand on the proximal humerus and the other holding the wrist pronated with the elbow hyperflexed. This is generally a stable position that will allow rotation of a reduced supracondylar fracture. The lateral image is then evaluated for restoration of the cortical contour of the distal humerus, reduction of the fracture gap, and presence of the anterior humeral line intersecting the capitellum.



**FIGURE 14-23.** Jones view. **A:** The proper elbow position and tube direction for the Jones view. **B:** radiograph of an elbow taken with the tube 90 degrees to the film. **C:** Cephalad angulation of the tube distorts the image of the distal humerus. **D:** Radiograph of the same elbow with the tube angulated cephalad.

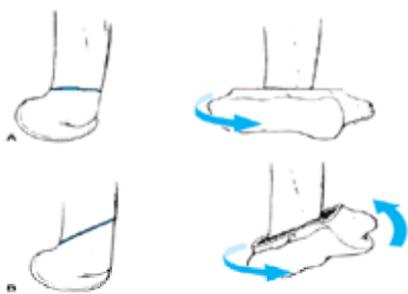
Rotating the arm with the fracture reduced and held in a stable position is possible in nearly all posteromedially displaced supracondylar fractures ( Fig. 14-24). In posterolaterally displaced supracondylar fractures, however, the reduction is frequently unstable in hyperflexion and may be less stable in pronation. If the fracture reduction is unstable, the C-arm should be rotated to obtain AP and lateral views rather than attempting to rotate the arm and losing a tenuous reduction. An anatomic or nearly anatomic reduction is a prerequisite for skeletal stabilization. If this cannot be achieved by closed reduction, traction or open reduction can be used.



**FIGURE 14-24.** Stability in external rotation. **A:** The stability of type II fractures is tested by taking a lateral radiograph with the extremity fully externally rotated. **B:** If there is sufficient intrinsic stability, the fracture will remain reduced on the radiograph.

### Cast Immobilization Technique

Because type III fractures are intrinsically unstable, the elbow must be held in extreme flexion to prevent the distal fragment from rotating; these fractures tend to rotate with flexion of less than 120 degrees (132). Usually, however, severe swelling prevents the elbow from being kept in hyperflexion. When the swelling subsides, the distal fragment tends to rotate horizontally, making it susceptible to varus angulation, particularly if the fracture line is angulated ( Fig. 14-25). In most series (48,110,147,186) the results of type III fractures treated with closed reduction and cast immobilization are not as good as those treated with pinning. However, Hadlow et al. (83) suggested that selective use of casting is beneficial, reporting that in their series, 61% of type III and 77% of type II fractures were successfully treated without pinning.



**FIGURE 14-25.** Effects of obliquity of the fracture line. If the fracture line is transverse (**A**), pure rotation does not produce angulation. If the fracture line is oblique (**B**), rotation of the distal fragment produces a secondary distal angulation.

A figure-of-eight cast is used to maintain flexion of at least 120 degrees. If the arm can be flexed to 120 degrees with an intact pulse, casting can be used as primary treatment. Alburger et al. (4) reported that using skin traction initially until swelling decreases allowed successful use of casting without pinning. Although a number of historic series used casting as primary treatment, most recent reports favor pinning of this fracture. When a cast is used as primary treatment, it should be worn for 3 to 4 weeks. After cast removal, motion is encouraged and the patient is placed in a sling until comfortable, which may vary from hours to days. Physical therapy usually is not required, because simple active motion is encouraged and usually is sufficient.

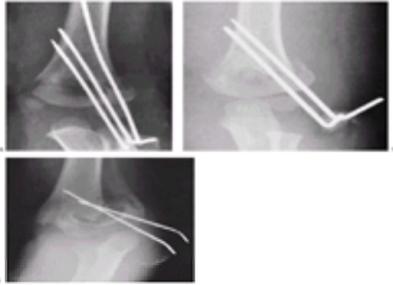
### Technique of Pin Fixation

Pin fixation of supracondylar fracture of the humerus has been performed for over 50 years (131). Jones (99) and Swenson (178) were early advocates of this technique. Before the development of the fluoroscopic unit, blind pinning (91) was performed; Jones noted that this could be done without complications by even the "average surgeon." Modern imaging techniques and improved power equipment have made percutaneous pinning the standard treatment for this difficult fracture. Flynn (66,67), and later Wilkins (190,191), among others (29,127,128,147,148) popularized modern pinning techniques and documented their value. Present controversy includes (a) crossed pins ( Fig. 14-26) or two lateral pins; (b) burying pins or leaving them out through the skin; (c) timing of pinning, that is, emergent, urgent, or elective; and (d) the role of open reduction. Absorbable polyglycolide pins have not been found suitable for fixation of supracondylar fractures (28).



**FIGURE 14-26.** Crossed pinning is a more stable configuration than two lateral pins. The lateral pin is placed at the border of the ossified lateral condyle. The medial pin enters the medial epicondyle and is generally more transverse than the lateral pin.

Zionts (199) evaluated the torsional strength of pin configurations in an adult humeral cadaver model. The maximal strength was gained by using two crossed pins with one placed through the medial condyle and one through the lateral condyle, extending up the corresponding columns and engaging the opposite cortex. The torque required to produce 10 degrees of rotation averaged 37% less with two lateral pins parallel and 80% less with two lateral pins crossed than with medial and lateral crossed pins. Thus, lateral pinning required significantly less force to produce 10 degrees of displacement than did crossed pinning ( Fig. 14-27). The question, however, is whether the resistance to torque provided by lateral pinning is sufficient to allow fracture healing without displacement, if supplemented by cast immobilization in the clinical setting. In addition, the experimental models have no intact periosteum, which will provide further fracture stability, nor do they have pediatric cancellous bone, which may be quite different in terms of pin fixation mechanics.



**FIGURE 14-27.** Lateral pin constructs. **A:** Three lateral pins. **B:** Two parallel pins. **C:** Late displacement in a patient with two small lateral pins that crossed at the fracture site. (Reprinted from Wilkins KE. The management of severely displaced supracondylar fractures of the humerus. *Techniques Orthop* 1989;4:212-224; with permission.)

Onwuanyi (141), in a clinical series, also concluded that there was greater stability or ability to maintain reduction with the crossed pinning technique. Cheng (42) reported 180 type III supracondylar fractures treated with lateral pinning as primary treatment in nearly all. He reported very low incidences of cubitus varus deformity and loss of reduction and concluded that in actual practice there was no difference between two lateral pins and crossed pins. He recommended lateral pins as the treatment of choice because there is less risk of ulnar nerve injury with this technique.

The primary risk with crossed pinning is injury to the ulnar nerve by a medial pin. The frequency of ulnar nerve injury in reported series ranges from 0 to 5% (33,93,153,157). Because the ulnar nerve may sublux anteriorly when the elbow is hyperflexed in as many as 30% of patients (198), it is at significant risk in the crossed pinning technique. Locating the ulnar nerve with a nerve stimulator has been suggested (129). Making a small 1.5-cm incision over the medial epicondyle for placement of the medial pin also has been proposed as a way to locate and protect the ulnar nerve. This technique works well and may avert the problem of iatrogenic ulnar nerve injury when crossed pinning is required for very unstable fractures. Also, placing the lateral side pin first with the elbow hyperflexed and then placing the medial pin with the elbow in only 90 degrees of flexion lessens this risk.

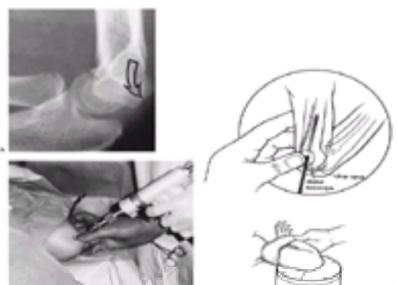
#### **Technique of Pinning: Crossed Pins**

Steel pins 0.062 or 0.075 mm (5/64th inches) in diameter are used. (Fig. 14-28). After reduction of the fracture and with the C-arm screen used as an operating table, the arm is held with the elbow hyperflexed and the forearm pronated to maintain the reduction. Anatomic reduction is confirmed with the image intensifier before pinning. The lateral pin is inserted first. The insertion site is made so that the pin will traverse the lateral portion of the ossified capitellum, cross the physis, proceed up the lateral column, and engage the opposite medial cortex proximally. Using a Kirschner wire or radiodense object, the position for insertion of the pin is documented. A small incision is made in the skin. The pin is placed using a power drill and sharp Kirschner wire or Steinmann pin. Provisional stability is achieved with the first pin. Then the elbow is gently extended to about 45 degrees of flexion to allow a perfect AP radiograph of the distal humerus to ensure anatomic alignment. The elbow is then flexed to 120 degrees and externally rotated and a lateral image is obtained with fluoroscopy. A second pin is then placed medially. A small 1.5-cm incision is made over the medial epicondyle and dissection is performed down to the level of the medial epicondyle. If the ulnar nerve has subluxed over the medial epicondyle, sometimes blunt dissection stimulates the nerve and the fingers will move slightly. If the ulnar nerve is found, it should be retracted posteriorly and a pin placed through the medial epicondyle under direct vision. The position of the Kirschner wire from the medial side is confirmed with fluoroscopy. The pin should enter the medial epicondyle and not the ulnar groove. The medial pin is generally more horizontal than the lateral pin and should traverse the medial column and engage the opposite lateral cortex.



**FIGURE 14-28.** Lateral pin placement. **A:** The lateral pin is placed first using the C-arm screen as an operating room table. The elbow is held in hyperflexion for the placement of the lateral pin. **B:** The lateral view is obtained by externally rotating the arm, holding the elbow in hyperflexion for stability.

Variations in the crossed pinning technique include: (a) no medial incision (Fig. 14-29); (b) maintenance of reduction by strapping the arm in a position of hyperflexion before pinning (Wilkins; Fig. 14-30) (191); (c) using a bar as a fulcrum to facilitate reduction, as described by Flynn (66); and (d) pinning the medial side first with the elbow in hyperflexion. If no medial incision is used, the ulnar nerve is palpated and pushed posteriorly as the pin is inserted. Pinning medially with the elbow in hyperflexion, as described by Wilkins, has the benefit of holding the fracture in better position while pinning, but does place the ulnar nerve at higher risk.



**FIGURE 14-29.** Medial pin placement. **A:** The medial epicondyle is posteromedial (arrow). **B:** The medial pin is placed directly through the medial epicondyle, using the opposite thumb to pull posterior the soft tissues, thus protecting the ulnar nerve. **C:** The relationship of vital structures during medial pin placement. The pin is directed from posteromedial to anterolateral directly through the center of the medial epicondyle under control of the image intensifier, with the upper extremity fully externally rotated.



**FIGURE 14-30.** Temporary stabilization. Once reduced, the elbow is taped into hyperflexion **(A)**. This allows the elbow to be manipulated into full external **(B)** and internal **(C)** rotation, providing direct lateral visualization of the respective medial and lateral condyles. (Reprinted from Wilkins KE. The operative management of supracondylar fractures. *Orthop Clin North Am* 1990;21:269-289; with permission.)

### **Two Lateral Pins**

Two lateral pins ([12,42,141](#)) (0.062 or 0.75 mm) provide less stability than crossed pins, but there is no risk to the ulnar nerve, which is a significant benefit of this technique. The operating room set up for two lateral pins is exactly the same as that for crossed pins. After achieving closed reduction and confirming the position on AP and lateral views, the first pin site should be chosen with the second pin in mind. The goal is to have two pins that are parallel on the AP and lateral views. If the pins are placed so that they cross well proximal to the fracture, this may be satisfactory, but is not as secure as parallel pins. Two pins crossing at the fracture is unsatisfactory because torque will not be satisfactorily resisted and a rotational deformity may result.

In choosing a site for the first pin, we generally place it up through the center of the ossified capitellum. It engages the edge of the olecranon fossa, giving it greater stability, and then further penetrates the opposite cortex. A second pin is placed through the distal humeral epiphysis lateral to the capitellum but clearly within the epiphysis. It proceeds parallel to the first pin up the lateral column and engages the opposite cortex. If the first pin is placed centrally in the lateral column, the second pin will nearly always cross the first just above the fracture, and this will be a less satisfactory lateral pinning. Maximal pin separation and parallel alignment increase the stability with this technique.

The “shake test” can be used to test the stability of two lateral pins. If the arm can be grasped by the proximal humerus and shaken mildly without displacement of the fracture, the fixation is satisfactory. If displacement occurs, it is generally rotational with loss of reduction of the medial column. We simply reduce the fracture to an anatomic position and place a medial pin as described in the technique for crossed pinning. A third lateral pin will increase stability slightly, but is rarely necessary.

After pinning by any technique, the patient is placed in a splint or bivalved long arm cast with the forearm in neutral and the elbow flexed slightly less than 90 degrees. Although this position of flexion is chosen as a general rule, it is imperative that the elbow be placed in a position that is most favorable for neurovascular structures. If the pulse becomes weak or obliterated at 80 degrees of flexion, the arm should be put in a position where the pulse is strongest. The study by Mapes and Hennrikus ([121](#)), using Doppler evaluation of the brachial artery, showed that with progressive flexion and pronation, the pulse pressure and therefore flow was incrementally diminished. Fracture stability is gained by skeletal stabilization, and the splint or cast is simply placed to protect the patient. If a circular cast is used, it should be bivalved and spread to prevent constriction. In no case should a child be in a circular cast with the elbow hyperflexed following this procedure.

Iyengar et al. compared early and delayed pinning to determine whether fractures treated more than 8 hours after trauma had higher incidences of open reductions and complications ([96](#)). They concluded that there was no difference. However, this was a retrospective review and it is possible that severe fractures were treated emergently, which would bias the conclusion. In general, these fractures are treated emergently. With smooth pins left out through the skin, there is rarely a need for anesthesia for pin removal. A very low infection rate accompanies pinning (less than 1%) because they are in place only 3 to 4 weeks.

### **Intramedullary Pin Fixation**

Prevot et al. ([149](#)) reported their experience with intramedullary fixation of supracondylar fractures in children. After closed reduction, the pins were inserted proximally at the junction of the middle and proximal third and passed distally in the shaft, diverging in each of the supracondylar columns. Their laboratory experiments comparing this type of fixation with medial-lateral and lateral pins demonstrated that proximally placed pins diverging in the distal fragment were more stable than other methods of percutaneous pin fixation.

## **AUTHORS' PREFERRED METHOD OF TREATMENT**

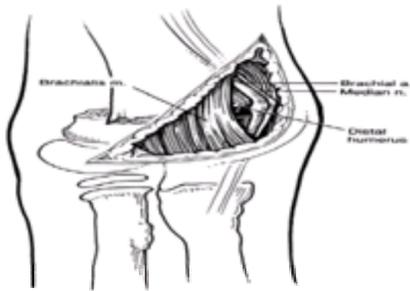
When possible, we prefer closed reduction and pinning of type 2 and type 3 supracondylar fractures. Two lateral pins are chosen for a “stable” fracture, and crossed pins are used for very unstable fractures. We always make a small incision over the medial epicondyle to protect the ulnar nerve when using crossed pins.

### **Open Reduction**

Closed reduction may not be possible because of interposed soft tissue, especially in posterolaterally displaced supracondylar fractures. Open reduction should be considered to obtain alignment if there is vascular compromise or neural entrapment, as judged by soft tissue interposition. An open reduction is also indicated if there is an open fracture that requires irrigation and debridement or simply if a satisfactory closed reduction cannot be performed.

During the early part of this century there was a reluctance to recommend open reduction of supracondylar fractures. With improvement in operative techniques and more experience with surgical intervention, open reduction of acute supracondylar fractures has now become widely accepted.

Fleuriau-Chateau et al. ([65](#)) reported excellent results with open reductions of irreducible fractures. The range of motion was satisfactory in 94% of patients, and none had myositis or cubitus varus deformity. Fifteen of the 25 children with irreducible fractures had entrapment of neurovascular structures preventing reduction. The high incidence of entrapment of neurovascular structures in individuals skilled in closed reduction supports the use of open reduction in irreducible fractures. The approach for open reduction has been somewhat controversial. Anterior, medial, lateral, and posterior approaches have all been recommended. Those who advocate medial and lateral approaches ([189](#)) operate through the side in which the periosteal hinge is torn; that is, a lateral approach is used for posteromedial displacement or a medial approach is used for posterolateral displacement. The posterior approach ([166](#)) to the elbow through the triceps muscle and tendon has been used with excellent results. Our preference is the direct anterior approach ([52,77](#)) through a transverse anterior incision in the antecubital fossa, extending proximally, medially as needed. The brachialis muscle is generally transected by the proximal humeral fragment. The structures remaining intact anteriorly are the biceps tendon and neurovascular bundle ([Fig. 14-31](#)). The anteromedial approach gives easy access to an entrapped neurovascular bundle and allows anatomic reduction of the humeral fracture under direct vision by simply placing a retractor on the remaining portion of the brachialis muscle.



**FIGURE 14-31.** Tenting of neurovascular bundle. With posterolateral fractures, the neurovascular structures often are tented over the medial spike of the proximal fragment. This fragment can be "buttonholed" through the brachialis muscle.

Pins are then placed either medially and laterally or two lateral pins are used, as described above, to provide stable fixation. After open reduction, pins may be left in place slightly longer than after closed reduction, but there are no firm data on this. Good callus should be observed at the fracture before pin removal, generally 3 to 4 weeks after injury. Prolonged pin fixation is not necessary.

#### ***Incidence of Complications Lessened***

One of the reasons for an increase in open reductions has been the recognition that complications are relatively few. The fears of infection, myositis ossificans, and neurovascular injury ([74,163,170,187](#)) have been dispelled by surgical experience ([10,14,39,48,65,71,76](#)). In a combined series of 470 fractures treated by open reduction, the incidence of infection was 2.5%, all of which resolved ([5,21,37,52,75,79,82,85,103,108,138,147,151,165,186,189](#)). The incidence of neurovascular complications from the procedure itself was essentially zero. Four patients with myositis ossificans (1.4%) were reported, all in a single series ([79](#)).

The most frequent complication of surgical management appears to be a loss of range of motion. One of the reasons given in the past for loss of motion was the use of a posterior approach. It has been stated that approaching the fracture through the relatively uninvolved posterior tissues, induces added scar leading to stiffness. In earlier reported series using a posterior approach, loss of range of motion was significant. However, in the past decade, use of a posterior approach with no significant increase in postoperative elbow stiffness has been reported. Residual cubitus varus occurred in as many as 33% of patients in some of the earlier series ([7,51,79,189](#)), most due to inadequate surgical reduction. When good reduction was accomplished, the incidence of cubitus varus was low. Surgical intervention does not guarantee an anatomic reduction; it is the quality of the reduction achieved at the time of surgery that is important.

Lal and Bahn ([112](#)) reported that delayed open reduction, 11 to 17 days after injury, did not increase the frequency of myositis ossificans. If a supracondylar fracture is unreduced or poorly reduced, delayed open reduction and pin fixation appear to be justified.

Open supracondylar fractures generally have an anterior puncture wound where the metaphyseal spike penetrates the skin. Even if the open wound is only a small puncture in the center of an anterior pucker, open irrigation and debridement are indicated. The anterior approach, using a transverse incision with medial or lateral extension as needed, is recommended. The neurovascular bundle is directly under the skin and tented over the metaphyseal fragment, so care should be taken in approaching this fracture surgically. Whereas the skin incision can be extended medially proximally and laterally distally, often only the transverse portion of the incision is required, which gives a better cosmetic result. The brachialis muscle usually is transected, because it is a muscle belly to its insertion on the coronoid attachment and is highly vulnerable to trauma from the proximal metaphyseal fragment. The fracture surfaces are examined and washed, and a curet is used to remove any dirt or entrapped soft tissue. Once the debridement and washing are complete, the fracture is stabilized with Kirschner wires. All patients with open fractures are also treated with antibiotics: cephalothin for Gustilo types 1, 2, and 3A injuries, with the addition of an aminoglycoside for types 3B and C fractures.

#### ***Traction Management of Type III Supracondylar Fractures***

Traction can be used in two ways in the management of type III supracondylar fractures: to allow swelling to decrease and to facilitate closed reduction, as described by Alburger et al. ([4](#)) and others ([171,197](#)). In this technique, patients are placed in sidearm or overhead skin traction for 3 to 5 days until elbow hyperflexion can be tolerated for closed reduction. Alburger et al. reported 92% good or excellent results in 39 patients treated in this manner.

Definitive treatment of the fracture with 14 days of traction or until healing has occurred historically has led to a very low incidence of cubitus varus deformity, as documented by Smith ([171,172](#)). D'Ambrosia ([51](#)) in a series of supracondylar fractures treated with overhead skeletal traction, found no cubitus varus deformities; with sidearm or skin traction for definitive treatment, the rate of varus deformity was 33%. Piggot ([146](#)) documented the efficacy of straight lateral skin traction with a report of only 4% cubitus varus deformities and no vascular complications. A comparison by Prietto ([150](#)) of pinning and Dunlop's traction demonstrated the relative benefits of pinning in decreased costs and shorter hospital stays in addition to a lower cubitus varus rate. He reported a 5% rate of cubitus varus deformity in the pinning group and 33% in the Dunlop's traction group. Skeletal traction overhead with use of an olecranon wing nut ([Fig. 14-32](#)) has been advocated by Palmer ([144](#)), Worlock ([197](#)), and Badhe ([16](#)). Overhead traction was superior to sidearm traction and had a low incidence of cubitus varus (2%–9%) in these series. The olecranon wing nut is a simple device that can be placed in the proximal ulna without risk of injury to the ulnar nerve, as might occur in placing a transverse traction pin. The wing nut offers the advantage of applying a torque about the distal humeral fragment by changing the position of the traction rope into the holes in the wing nuts.



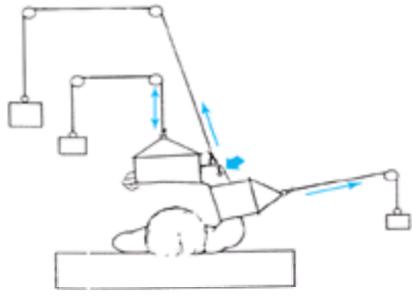
**FIGURE 14-32.** The olecranon wing nut is a simple skeletal traction device. Varying the insertion point of the rope alters torque about the distal fragment.

The indications for traction as the definitive management of a supracondylar fracture are (a) an unstable fracture reduction or pattern that cannot be pinned, and (b) supracondylar comminution or medial column comminution that is not suitable for pinning and would certainly collapse with simple casting after reduction. The duration of traction usually is about 14 days before cast immobilization in this rapidly healing fracture. Prolonged traction may be necessary if poor reduction is obtained in traction, because significant callus must be present for fracture stability before casting.

#### ***Technique of Traction Wing Nut Insertion***

With the arm prepped in a sterile manner, a hole is drilled in the cortex opposite the coronoid process of the ulna. A 3.2-mm drill bit is used and a hole is made through both cortices just distal to the coronoid process. A wing nut is then placed through the small incision. The wing nut engages the opposite cortex but does not penetrate it. A sterile dressing is placed around the interface between the wing nut and skin. A traction rope and weights are applied ([Fig. 14-33](#)). A sling is used to support the hand and forearm. Initially, traction of about 5 pounds is applied, depending on the size of the patient. The shoulder should be lifted just off the bed ([144](#)).

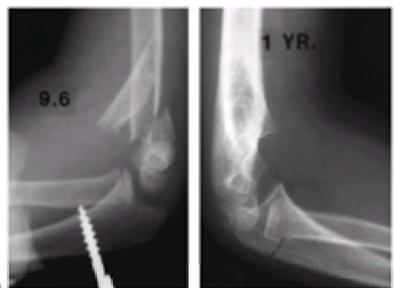
AP and lateral radiographs should be taken in traction to judge the adequacy of reduction. After there is good callus formation (generally after 10 to 15 days), the patient is removed from traction and placed in a long arm cast, which is worn for about 2 weeks.



**FIGURE 14-33.** Overhead olecranon pin traction. The arm is suspended by a pin through the olecranon ( *short arrow*). The forces maintaining the reduction ( *long arrows*) are exerted upward through the pin and sideways through a counter-sling against the arm. The forearm is supported with a small sling ( *double arrow*).

## AUTHORS' PREFERRED METHOD OF TREATMENT

For supracondylar comminution ([Fig. 14-34](#)) where stable pinning is impossible to achieve, we have uniformly chosen traction. Although these fractures are relatively rare, they do occur, and orthopaedic surgeons should be familiar with the use of traction as a technique of management of this fracture. We use a traction wing nut rather than a transverse traction pin, but this is a matter of choice. Anatomic reduction of the fracture is not critical with traction, but maintenance of alignment is. Callus formation is rapid in the area of the fracture, and the distal fragment should be confirmed to be neither hyperextended nor rotated into varus or valgus malalignment, based on radiographic evaluation while in traction. After good callus formation (generally after about 14 days), the patient can be removed from traction and placed in a long arm cast.



**FIGURE 14-34.** **A:** Because of supracondylar comminution, stable pinning could not be achieved and traction was chosen. **B:** Remodeling occurred over 1 year, and the patient regained full motion with normal alignment.

### Vascular Injury

Type III supracondylar fractures have significant incidences of brachial artery injury, vascular insufficiency, and compartment syndrome, each of which should be evaluated separately in a patient with a severe elbow injury.

#### **Brachial Artery Injuries and Vascular Insufficiency**

About 10% to 20% of patients with type III supracondylar fractures present with an absent pulse ([44,58,164](#)). In the emergency management of a patient with a type III supracondylar fracture, the arm should be splinted with the elbow in about 30 degrees of flexion. This will facilitate transport of the patient to the appropriate facility for care and radiographic evaluation. The presence of a pulse and perfusion of the hand should be documented. Perfusion is estimated by color, warmth, and capillary refill. The question sometimes raised in the emergency room evaluation of a patient with supracondylar fracture and poor blood flow to the hand is whether an arteriogram should be performed as part of the preoperative evaluation. The answer is an emphatic “no” ([164](#)). The initial approach to management of a patient with vascular compromise secondary to a supracondylar fracture should be immediate closed reduction and stabilization with Kirschner wires. If an anatomic reduction cannot be obtained closed, open reduction through an anterior approach ([14,65](#)) with medial extension allows evaluation of the brachial artery and removal of the neurovascular bundle entrapped within the fracture site.

After closed reduction and stabilization, the pulse and perfusion of the hand should be evaluated. Most extension type supracondylar fractures are reduced and pinned with the elbow in hyperflexion. With more than 120 degrees of elbow flexion, the radial pulse generally is lost, even in patients with an initially intact pulse. After pinning when the arm is extended, the pulse frequently does not return immediately. This is presumably secondary to arterial spasm, aggravated by swelling about the artery and decreased peripheral perfusion in the anesthetized, somewhat cool intraoperative patient.

Because of this phenomenon, 10 to 15 minutes should be allowed for recovery of perfusion in the operating room before any decision is made about the need for exploration of the brachial artery and restoration of flow to the distal portion of the extremity. Because most patients without a palpable pulse maintain adequate distal perfusion, the absence of a palpable pulse alone is not an indication for exploration of a brachial artery. Gillingham and Rang ([78](#)) recommended observation of patients with absent pulse, because most pulses returned within 10 days. The decision to explore a brachial artery needs to be based on objective criteria, if possible.

Traditionally, the decision has been based only on whether the hand was warm and pink. The following case indicates the difficulty with this. A 2-year-old girl was injured in a fall from a couch ([Fig. 14-35](#)). A type III supracondylar fracture and a pale, cool hand were documented on presentation to a local emergency department. Two hours later, the patient was brought to the operating room, where a cool, pale hand with poor capillary refill and an absent pulse was noted. Immediate closed reduction and pinning was performed with nearly anatomic reduction. The hand felt warmer and capillary refill was present. The pulse was not palpable, but because of the improved state of the hand, no exploration of the brachial artery was performed. During the next 4 hours the patient was observed closely with increasing fussiness, a nonpalpable pulse, and slow capillary refill. At this point an arteriogram was performed, showing brachial artery obstruction. Compartment pressures were measured, and increased pressure was noted in the volar compartment. A decision was made to return to the operating room for exploration and repair of the brachial artery and forearm fasciotomy. Whereas the outcome in this case was satisfactory with no long-term sequelae, other than scarring, the question is whether the low perfusion with subsequent ischemia and compartment syndrome could have been identified at the time of the closed reduction and immediate repair done.



**FIGURE 14-35.** **A:** This 2-year-old patient sustained a type III supracondylar fracture with vascular compromise. **B:** Pinning was performed in a nearly anatomic position. **C and D:** Six hours postoperatively, increasing pain, a pale hand, and evolving compartment syndrome prompted arteriography, showing brachial artery occlusion.

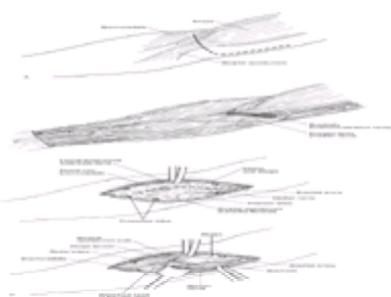
What would happen if no repair had been performed in the above case? Had a repair been performed immediately, could the development of a compartment syndrome have been averted? What is the relationship between compartment syndrome and perfusion? Is there a way to determine when flow is insufficient in a more objective way?

Several investigators have attempted to provide criteria for repair in addition to warmth and color in the patient with absence of a palpable pulse. The use of simple Doppler ultrasonography in the operating room has been studied by two groups ([162,164](#)). In the absence of a palpable pulse, a Doppler device can be used to measure lower flow states with small pulse amplitude. Shaw ([164](#)) found no false-positive explorations when patients with pulses that could not be palpated but were identified on Doppler evaluation were observed and exploration was performed in those in whom no pulse was found with either palpation or Doppler. The brachial artery was either transected or entrapped in all patients with surgical exploration, and none with “spasm” had surgery. Schoenecker et al. ([162](#)), using the same criteria, identified six patients for exploration. Three had a damaged or transected brachial artery with no flow, and three had an artery kinked or trapped in the fracture. At follow-up, all patients with vascular repair had a radial pulse. One patient with more than 24 hours of vascular insufficiency had an unsatisfactory outcome. Copley et al. ([44](#)) reported that of 17 patients with type III supracondylar fractures and no palpable pulse at presentation, 14 recovered pulse after reduction. The three explorations identified significant vascular injury, and the brachial artery was repaired. In addition to Doppler evaluation, pulse oximetry ([104,154](#)) has been recommended. Our experience with the use of a Nelkor system for this technique is that when used in the operating room, it often underestimates perfusion. We believe this is secondary to its use in an anesthetized person with low blood pressure and peripheral vasoconstriction. As a patient is waking up and pulse pressure and peripheral circulation are increased, pulse oximetry becomes a valid way to determine perfusion. We have found it useful for evaluating patients after vascular repair or pinning but not for intraoperative decision making. Copley and Dormans, ([44](#)) reported that some patients who initially had good perfusion and an intact pulse lost both in the postoperative period. Two of 14 patients showed signs of increased vascular insufficiency over a 48-hour period and required subsequent reconstruction. Data such as this supports in-hospital observation of patients with supracondylar fractures, especially those with evidence of vascular compromise.

Obliteration of the radial pulse after closed reduction and pinning is a strong indication for brachial artery exploration. After 10 to 15 minutes is allowed for resolution of arterial spasm as a cause for loss of pulse, the brachial artery should be explored. Either direct arterial entrapment at the fracture or arterial compression by a fascial band pulling across the artery may cause loss of pulse after fracture reduction. As described above, the other indication for brachial artery exploration is persistent vascular insufficiency after reduction and pinning.

#### **Exploration of the Brachial Artery**

The orthopaedic surgeon and vascular surgeon need to work together in the management of this problem. Often release of a fascial band or adventitial tether resolves the problem of obstructed flow. This is a simple procedure performed at the time of exploration of the antecubital fossa and identification of the brachial artery. In some patients, however, a formal vascular repair and vein graft are required. The brachial artery should be approached through a transverse incision across the antecubital fossa, with a medial extension turning proximally at about the level of the medial epicondyle ([Fig. 14-36](#)). Care must be taken because the neurovascular bundle may be difficult to identify when it is surrounded by hematoma, but it may lie in a very superficial position. At the level of the fracture, the artery may seem to disappear into the fracture site, covered with shredded brachialis muscle. This occurs when the artery is tethered by a fascial band or arterial adventitia attached to the proximal metaphyseal spike pulling the artery in the fracture site. Dissection should occur proximally to distally, along the brachial artery, identifying both the artery and the median nerve. Arterial injury generally is at the level of the supratrochlear artery ([Fig. 14-7](#)), which provides a tether, making the artery vulnerable at this location. Arterial transection or direct arterial injury can be identified at this level. Entrapment of the neurovascular bundle in the fracture is best identified by proximal to distal dissection.



**FIGURE 14-36.** Brachial artery exploration through anteromedial approach.

The decision to repair a damaged artery or use a vein graft is generally made by the vascular surgeon. If spasm is the cause of arterial insufficiency, several techniques have been recommended. If arterial spasm is the cause of inadequate flow, and collateral flow is not sufficient to maintain the hand, attempts to relieve the spasm may be tried. Both stellate ganglion block and application of Paverin or local anesthetic to the artery have been found to be beneficial in this situation. If spasm is not relieved by these techniques and collateral flow is insufficient, the injured portion of the vessel is excised and a vein graft is inserted. When flow is restored, the wound is closed and the patient is placed in a splint with the elbow flexed less than 90 degrees and the forearm supinated. Postoperative monitoring should include temperature, pulse oximetry, and frequent examinations for signs of compartment syndrome or ischemia. Injection of urokinase has been suggested to increase flow ([35](#)); we have had no experience with this technique.

Sabharwal et al. ([159](#)) documented that 3.2% of patients with type III supracondylar fractures have an absent pulse at presentation, for which they recommended noninvasive monitoring. Magnetic resonance angiography and color flow duplex Doppler were helpful in deciding whether or not to explore the brachial artery. Although repair is technically feasible, Sabharwal et al. cautioned that high rates of reocclusion and residual stenosis argued against early revascularization if not absolutely necessary. Early reocclusion, however, was not reported by Schoenecker et al. ([162](#)) or Shaw et al. ([164](#)).

The necessity of early treatment of vascular compromise was emphasized by Ottolenghi ([143](#)), who found no Volkmann ischemic contractures ([135](#)) in patients in whom vascular compromise was treated within 12 hours. The frequency of this complication increased steadily with repair between 12 and 24 hours; after 24 hours of delay in treatment, outcomes were uniformly poor. This series presents convincing evidence that prompt exploration of arterial insufficiency markedly decreases the incidence of Volkmann ischemic contracture. However, it should be understood that brachial artery obstruction and compartment syndrome, although related, are not

equivalent, and both are fortunately rare problems. Ischemia will lead to a compartment syndrome, but the presence of a radial pulse does not preclude it.

### Compartment Syndrome

In acute compartment syndrome (90,135), increased pressure in a closed fascial space causes muscle ischemia. With untreated ischemia, muscle edema increases, further increasing pressure, decreasing flow, and leading to muscle necrosis, fibrosis, and death of involved muscles. Forearm compartment syndrome occurs after 1% or fewer supracondylar fractures. A compartment syndrome of the forearm may occur with or without brachial artery injury and in the presence or absence of a radial pulse. The diagnosis of a compartment syndrome is based on resistance to passive movement of the fingers and dramatically increasing pain after fracture. The classic five "P's" for the diagnosis of compartment syndrome—pain, pallor, pulselessness, paresthesias, and paralysis—are poor indicators of a compartment syndrome.

Mubarak and Carroll (135) recommended forearm fasciotomy if clinical signs of compartment syndrome are present or if intracompartmental pressure is greater than 30 mm Hg. Heppenstal et al. (88) suggested that a difference of 30 mm Hg between diastolic blood pressure and compartment pressure should be the threshold for release. If pain is increasing and finger extension is decreasing, fasciotomy is clearly indicated. Measuring compartment pressures in a terrified, crying child is difficult, and if clinical signs of compartment syndrome are present, a trip to the operating room for evaluation and possible fasciotomy is often a better course of action than pressure measurement and observation.

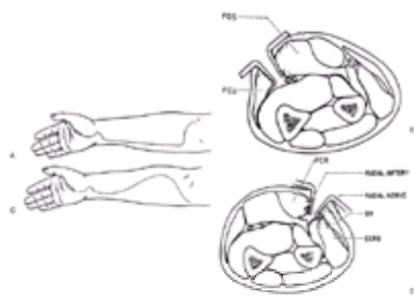
Clinical conditions that contribute to the development of compartment syndrome are direct muscle trauma at the time of injury, swelling with intracompartmental fractures (associated forearm fracture), decreased arterial inflow, and restricted venous outflow. The mechanism of injury of the supracondylar fracture is critical. An associated forearm fracture or forearm crush injury significantly increases the likelihood of compartment syndrome. An arterial injury in association with multiple injuries or crush injury further diminishes blood flow to the forearm musculature and increases the probability of a compartment syndrome.

Even if distal pulse is found by palpation or Doppler examination, an evolving compartment syndrome may be present. Increased swelling over the compartment, increased pain, and decreased finger mobility are cardinal signs of an evolving compartment syndrome. Evaluation of possible compartment syndrome cannot be based on the presence or absence of a radial pulse alone. If a compartment syndrome does appear to be evolving, initial management includes removal of all circumferential dressings. The volar compartment should be palpated and the elbow should be extended. We believe that the fracture should be immediately stabilized with Kirschner wires to allow proper management of the soft tissues.

Another factor that contributes to the development of compartment syndrome is warm ischemic time after injury. When blood flow is compromised and the hand is pale with no arterial flow, muscle ischemia is possible, depending on the time of oxygen deprivation. After fracture reduction and restoration of flow, the warm ischemic time should be noted. If this time is more than 6 hours, compartment syndrome secondary to ischemic muscle injury is likely. Prophylactic volar compartment fasciotomy can be performed at the time of arterial reconstruction. The exact indication for prophylactic fasciotomy in the absence of an operative revascularization is uncertain. Even when the diagnosis is delayed or the compartment syndrome is chronic, fasciotomy has been shown to be of some value.

### Technique for Volar Fasciotomy

The volar compartment of the forearm can be approached through the classic Henry approach or the ulnar approach ( Fig. 14-37). If the compartment syndrome is associated with a brachial artery and median nerve injuries, we generally use the Henry approach as an extension of the vascular repair. The advantage of the ulnar approach, as described by Willis and Rorabeck ( 194) is that it produces a more cosmetically pleasing scar. A volar fasciotomy involves opening the volar compartment from the carpal tunnel distally to the lacertus fibrosa and antecubital fascia proximally. The fascia over the deep flexors is opened, as is the superficial fascia, to decompress the deep volar compartment of the forearm. Failure to release the deep volar fascia may cause contracture of the deep finger flexors. Generally only the volar compartment is released, with an associated decrease in pressure in the dorsal or extensor compartment. If the volar Henry approach is used, the interval between the brachioradialis and flexor carpi radialis and radial artery are retracted ulnarward. The deep volar compartment is exposed. The flexor digitorum profundus and flexor pollicis longus are exposed along with the pronator teres proximally and the pronator quadratus distally.



**FIGURE 14-37.** Surgical approach for forearm fasciotomy. **A:** Ulnar approach, skin incision. **B:** Ulnar approach, intermuscular interval (*FDS*, flexor digitorum sublimis; *FCU*, flexor carpi ulnaris). **C:** Henry approach, skin incision. **D:** Henry approach, *FCR*, flexor carpi radialis interval (*BR*, brachioradialis; *ECRB*, extensor carpi radialis brevis). (Reprinted from Willis RB, Rorabeck CH. Treatment of compartment syndrome in children. *Orthop Clin North Am* 1990;21:407-408; with permission.)

If the ulnar approach is used, as described by Willis and Rorabeck ( 194), the release is performed from the carpal canal to the antecubital fossa, as with the Henry approach. The skin incision begins above the elbow crease, medial to the biceps tendon ( Fig. 14-37); it crosses the elbow crease and extends distally along the ulnar border to the volar wrist, where it courses radially across the carpal canal. The fascia over the flexor carpi ulnaris is incised, and the interval between the flexor carpi ulnaris and the flexor digitorum sublimis is identified. The ulnar nerve and artery are retracted, exposing the deep flexor compartment of the forearm. The deep flexor fascia is incised. The ulnar nerve and artery, as well as the carpal tunnel, are decompressed distally.

After fasciotomy the wound generally is left open. An effective way to manage the wound is with a criss-crossed rubber band technique, securing the rubber bands in place with skin staples. An alternative is to simply place a sterile dressing over the open wound, but this makes wound closure difficult and probably increases the need for skin grafting. Definitive closure or skin grafting generally is performed within 5 to 7 days. Skeletal stabilization of the supracondylar and forearm fractures is necessary for proper management of compartment syndrome.

### Neurologic Deficit

In most modern series, the incidence of neurologic deficit with supracondylar fractures is 10% to 20% ( 127,160), with an incidence as high as 49% in one series ( 36). Reports differ as to whether the radial or median nerve is the most frequently injured. In modern series, the AIN appears to be the most commonly injured ( 49,58,126), with loss of motor power to the flexor pollicis longus and the deep flexor to the index finger as first described by Spinner in 1969 ( 174). The direction of the displacement of the fracture determines the nerve most likely to be injured. If the distal fragment is displaced posteromedially, the radial nerve is more likely to be injured. Conversely, if the displacement of the distal fragment is posterolateral, the neurovascular bundle is stretched over the proximal fragment, injuring the median nerve or AIN or both. In a flexion type of supracondylar fracture, which is rare, the ulnar nerve is the most likely nerve to be injured.

In general, if the nerve deficit is present when the patient arrives in the emergency department and if the fracture is reducible, open reduction of the fracture and exploration of the injured nerve are not indicated. In most series, nerve recovery, whether radial, median, or ulnar, generally occurs at an average of 2 to 2 1/2 months. Culp et al. (50) reported identification of eight injured nerves in 5 patients in which spontaneous recovery did not occur by 5 months following injury. Neurolysis was successful in restoring nerve function in all but one. Nerve grafting may be indicated for nerves not in continuity at the time of exploration. Neurolysis for perineural fibrosis is generally successful in restoring nerve function. There is no indication for early electromyographic analysis or treatment other than observation for nerve deficit until 5 to 6 months after fracture.

In their series of radial nerve injuries with humeral fractures, Amillo et al. (8) reported that of 12 injuries that did not spontaneously recover within 6 months of injury, only 1 was associated with a supracondylar fracture. Perineural fibrosis was present in 4 patients, 3 nerves were entrapped in callus, and 5 were either partially or totally transected.

In the supracondylar area, perineural fibrosis appears to be the most common cause of prolonged nerve deficit. Although nerve injury is related to fracture displacement, a neural deficit can exist with even minimally displaced fractures. Sairyo et al. (160) reported one patient in whom radial nerve palsy occurred with a slightly angulated fracture that appeared to be a purely extension type fracture on initial radiographs. Even in patients with mild injuries, a complete neurologic examination should be performed before treatment. An irreducible fracture with nerve deficit is an indication for open reduction of the fracture to be sure that there is no nerve entrapment. Chronic nerve entrapment in healed callus can give the appearance of a hole in the bone, Metev's sign. Nerve transection is rare, and almost always involves the radial nerve (19,30,50,123,126).

Iatrogenic injury to the ulnar nerve has been reported to occur in 1% to 5% of patients with supracondylar fractures (33,93,153,157). In a large series of type III supracondylar fractures, the rate of iatrogenic injury to the radial nerve was less than 1%. The course of the ulnar nerve through the cubital tunnel, between the medial epicondyle and the olecranon, makes it vulnerable when a medial pin is placed. The ulnar nerve generally is injured by direct trauma from the medial pin, with or without actual penetration of the nerve. If the pin is placed in the ulnar groove rather than in the medial epicondyle, injury is likely. The ulnar nerve may sublux over the medial epicondyle in as many as 30% of patients; this usually is bilateral and associated with ligamentous laxity. Ulnar nerve subluxation occurs most commonly with hyperflexion of the elbow and injury to the nerve is most likely when a medial pin is placed with the elbow in hyperflexion.

If an ulnar neuropathy is documented postoperatively when one was not present preoperatively and a medial pin has been placed, what is appropriate treatment? Brown and Zinar (33) reported four ulnar nerve injuries associated with pinning of supracondylar fractures, all of which resolved spontaneously 2 to 4 months after pinning. Lyons et al. (118) documented ulnar nerve injuries associated with pinning in 19 of 375 patients who had cross-pinning of supracondylar fractures. In only 4 was the medial pin removed. Two had explorations, which found no nerve transection. These researchers recommended leaving the medial pin in place until the fracture heals. Rasool (153) reported six patients with ulnar nerve injuries in whom early exploration was performed. In two the nerve was penetrated, and in three it was constricted by a retinaculum over the carpal tunnel, aggravated by the pin. In one patient, the nerve was subluxed and was fixed anterior to the cubital tunnel by the pin. Full recovery occurred in three patients, partial recovery in two, and no recovery in two. Royce et al. (157) reported spontaneous recovery of ulnar nerve function in three patients. One nerve that was explored had direct penetration of the nerve, and the pin was replaced in proper position. Two patients had late-onset ulnar nerve palsies, discovered during healing, and the medial pin was removed.

If a postoperative neural injury is documented, we prefer to explore the ulnar nerve and replace the pin in proper position. If an ulnar nerve lesion is documented late in the course of fracture healing, we remove the pin but do not explore the nerve.

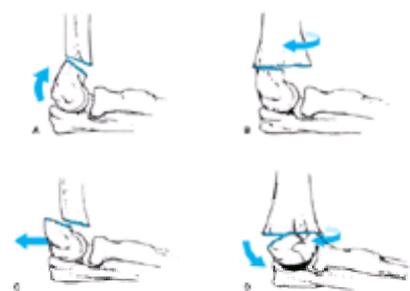
Prevention of ulnar nerve injury is obviously more desirable than treatment of ulnar neuropathy. Because of the frequency of ulnar nerve injury with crossed pinning, most surgeons prefer to use two lateral pins if possible and no medial pin. Nevertheless, it should be recognized that two lateral pins are about 30% less resistant to torque than crossed pins. However, successful maintenance of alignment of type III supracondylar fractures with two lateral pins has been reported (42). If cross-pinning is to be used, nerve penetration and indirect trauma to the nerve can be prevented by making a 1.5-cm incision over the medial epicondyle and being certain that the ulnar nerve is protected when the medial pin is placed. Another alternative for protection of the ulnar nerve was described by Michael and Stanislas, who attached a nerve stimulator to a needle, which was used for localization of the ulnar nerve (129). Once the ulnar nerve was identified, a standard pinning technique was used, placing the medial pin 0.5 to 0.75 mm anterior to the nerve. We have no experience with this technique. The contralateral elbow should always be examined for ulnar nerve subluxation in flexion because it usually is bilateral and associated with ligamentous laxity. Some surgeons palpate the ulnar nerve and push it posteriorly (Fig. 14-29).

Radial nerve injuries are rare complications of pinning of supracondylar fractures. The most common cause is probably a direct piercing injury to the radial nerve, as the medial pin exits the anterolateral cortex of the humerus. This injury can be best prevented by ensuring that pin penetration in the opposite cortex is limited to 1 to 2 mm. The medial pin should be backed out slightly if it protrudes significantly beyond the cortex. Spontaneous recovery of radial nerve function generally occurs.

### Elbow Stiffness

Loss of motion after extension-type supracondylar fractures is rare in children. Two series analyzed this complication in detail (47,87) and found that fractures treated closed had an average loss of motion of 4 degrees and a residual flexion contracture of 4 degrees. In those treated with open reduction, the loss of flexion was 6.5 degrees and the flexion contracture was 5 degrees or 1.2 degrees of hyperextension. Loss of motion has been reported with the posterior triceps splitting incision for open reduction (79,82,122).

Although loss of motion usually is minimal, significant loss of flexion can occur. This is generally caused by either posterior angulation of the distal fragment, posterior translation of the distal fragment with anterior impingement, or medial rotation of the distal fragment with a protruding medial metaphyseal spike proximally (Fig. 14-38). In young children with significant growth potential, there may be significant remodeling of anterior impingement, and any corrective surgery should be delayed at least 1 year. Although anterior impingement can significantly remodel, there is little remodeling to persistent posterior angulation or hyperextension.



**FIGURE 14-38.** Distal fragment rotation. **A:** Posterior angulation only of the distal fragment. **B:** Pure horizontal rotation without angulation. **C:** Pure posterior translation without rotation or angulation. **D:** Horizontal rotation with coronal tilting, producing a cubitus varus deformity. There is a positive crescent sign. (Redrawn from Marion J, LaGrange J, Faysse R, et al. Les fractures de l'extremite inferieure de l'humerus chez l'enfant. *Rev Chir Orthop* 1962;48:337-413; with permission.)

### Myositis Ossificans

Myositis ossificans is often mentioned as a possible complication (79,169) of supracondylar fractures, but it is remarkably rare (Fig. 14-39). This complication has been described after open reduction, but vigorous postoperative manipulation or physical therapy is believed to be the most commonly associated factor (6,107,147,167).



**FIGURE 14-39.** Myositis ossificans. Ossification of the brachialis muscle developed in this 8-year-old who had undergone multiple attempts at reduction. (Courtesy of John Schaeffer, M.D.)

In a report of two patients with myositis ossificans after closed reduction of supracondylar fractures, Aitken (3) noted that limitation of motion and calcification disappeared after 2 years. Postoperative myositis ossificans can be observed with the expectation of spontaneous resolution of both restricted motion and the myositis ossificans. There is no indication for early excision.

### Nonunion

The distal humeral metaphysis is a well-vascularized area with remarkably rapid healing, and nonunion of a supracondylar fracture is rare, with only a single case described by Wilkins and Beaty (191). We have not seen nonunion of this fracture. With infection, devascularization, and soft tissue loss, the risk of nonunion increases.

### Avascular Necrosis

Avascular necrosis of the trochlea after supracondylar fracture has been reported. The blood supply of the ossification center of the trochlea is fragile, with two separate sources. One small artery is lateral and courses directly through the physis of the medial condyle. It provides blood to the medial crista of the trochlea. If the fracture line is very distal, this artery can be injured, producing avascular necrosis of the ossification center and resulting in a classic fishtail deformity (Fig. 14-40). Symptoms of avascular necrosis of the trochlea do not occur for months or years. Healing is normal and motion is regained, but mild pain and occasional locking develop with characteristic radiologic findings. Routine follow-up radiographs of supracondylar fractures are not necessary because this complication is highly unusual and the fishtail deformity of the distal humerus does not compromise function.



**FIGURE 14-40.** Avascular necrosis of the trochlea. **A:** Anteroposterior injury film of an 8-year-old with a distal type III supracondylar fracture. **B:** The distal extension of the fracture (arrow) is best appreciated on the lateral reduction film. Postfracture, the patient was asymptomatic until 2 years later, when elbow stiffness developed. **C:** Repeat radiographs at that time showed atrophy of the trochlea (arrows). **D:** Three-dimensional computed tomographic reconstruction demonstrates atrophy of the trochlea.

### Angular Deformity

Angular deformities of the distal humerus after supracondylar fractures are much less common since the development of modern techniques of skeletal stabilization. In the past, the incidence of cubitus varus deformity after supracondylar fractures ranged from 9% to 58% (60,92,122). Pirone et al. (147) reported cubitus varus deformities in 14% of patients treated with cast immobilization compared with 3% in patients with percutaneous pin fixation. A decrease in frequency of cubitus varus deformity after the use of percutaneous pin fixation has been reflected in other recent series (28,29,66,67,101,128,147,148). The usual etiology of cubitus varus deformity is malunion of the distal humeral fragment rather than growth arrest. Distal humeral varus is generally believed to be a result of residual coronal angulation aggravated by malrotation and hyperextension.

Malunion of the distal humerus usually creates a static deformity (Fig. 14-41), which becomes evident as the patient gains full motion. A flexion deformity obscures the true varus deformity, which is generally associated with hyperextension. Although some investigators have proposed that unequal growth in the distal humerus causes varus deformity (17,31,91,92,144,167), this is unlikely to cause a significant varus deformity in the first few months after fracture. The growth of the distal humerus is 20% of that of the overall length of the humerus. In a 5-year-old, therefore, the amount of distal humeral growth in 1 year is approximately 2 mm, making it unlikely that growth asymmetry is a significant cause of varus deformity that occurs within the first 6 to 12 months after fracture. However, avascular necrosis of the trochlea or medial portion of the distal humeral fragment can result in progressive varus deformity. In a series of 36 varus deformities reported by Voss et al. (185), four patients had medial growth disturbance and distal humeral avascular necrosis as a cause of progressive varus deformity.



**FIGURE 14-41.** Cosmetic effects. A combination of varus coronal rotation and medial horizontal rotation of the distal fragment causes the lateral condyle to be unduly prominent (arrow). This accentuates the unpleasant appearance of the cubitus varus deformity. (Reprinted from Wilkins KE. Residuals of elbow trauma in children. *Orthop Clin North Am* 1990;21:291-314; with permission.)

Horizontal rotation (59,191) in a medial direction or internal rotation of the distal fragment is believed to predispose to distal fragment varus angulation ( Fig. 14-38). Eccentric position of the biceps has been suggested as a cause of varus tilting by the distal fragment. Chess et al. ( 43) evaluated the clinical appearance of cubitus varus in an anatomic study of 256 combinations of varus angulation, internal rotation, and posterior angulation. They found that the major component of cubitus varus deformity was true varus angulation in the coronal plane. Internal rotation did appear to worsen the deformity. In a clinical study ( 119,120), the true distal humeral rotation was measured on wedges removed from osteotomies of the distal humerus; the horizontal rotation of the distal humerus did not correlate with the severity of cubitus varus (185). Horizontal rotation may accentuate the clinical varus deformity, but it is not the primary cause. Coronal plane deformity and hyperextension are the factors in cubitus varus deformity and are important in planning surgical correction.

Some researchers have suggested that cubitus varus deformity may produce weakness, but most believe that the effects are primarily cosmetic. An increased risk of fracture (54), especially of the lateral condyle, has been linked with cubitus varus deformity ( Fig. 14-42). Varus angulation may complicate throwing. However, the functional effects of cubitus varus deformity are minimal and, other than decreasing fracture risk, the main indication for correction of a varus deformity is to improve the appearance of the elbow rather than to improve function.



**FIGURE 14-42.** Predisposition to lateral condyle fracture. This 5-year-old sustained a supracondylar fracture of this extremity 1 year before this acute stage II fracture of the lateral condyle (arrows). It was thought that the cubitus varus deformity predisposed the patient to this second fracture. (Courtesy of Kenneth P. Butters, M.D.)

The radiologic appearance of cubitus varus deformity is distinctive. On the AP view, the angle of the physis of the lateral condyle (Baumann angle) is more horizontal than normal. On the lateral view, a crescent sign is produced by the superimposition of the capitellum on the olecranon ( Fig. 14-43).



**FIGURE 14-43.** A patient with cubitus varus shows overlapping of the distal humerus, with the olecranon (arrow) producing the typical crescent sign.

Cubitus varus deformity also is associated with a significant increase in late ulnar nerve palsies, as reported in the Japanese literature ( 1,24,182). With a cubitus varus deformity, the olecranon fossa moves to the ulnar side of the distal humerus ( 140), and the triceps shifts a bit ulnarward. Investigators theorized that this ulnar shift may compress the ulnar nerve against the medial epicondyle, narrowing the cubital tunnel and resulting in chronic neuropathy. In a recent report ( 1), a fibrous band running between the heads of the flexor carpi ulnaris was thought to cause ulnar nerve compression.

#### **Treatment of Cubitus Varus Deformity**

As for the treatment of any posttraumatic malalignment, options include (a) observation with expected remodeling, (b) hemiepiphysiodesis and growth alteration, and (c) corrective osteotomy. Bracing of this deformity is not effective for improving alignment or preventing increasing varus. Observation generally is not appropriate because although hyperextension may remodel to some degree in a young child ( Fig. 14-44), in an older child, little remodeling occurs even in the plane of motion of the joint.



**FIGURE 14-44.** A hyperextension deformity in the distal humerus may remodel somewhat, whereas varus and valgus deformity do not. Hyperextension deformity in the distal humerus following fracture (A). Four years later (B), a more normal distal humeral anatomy is seen with remodeling of the hyperextension deformity; 2 years later, (C), a normal distal humeral anatomy is reconstituted.

Hemiepiphysiodesis of the distal humerus may occasionally be of value, particularly to prevent cubitus varus deformity from developing in a patient with clear medial growth arrest or trochlear avascular necrosis. If untreated, medial growth disturbance will lead to lateral overgrowth and progressive deformity. Lateral epiphysiodesis will not correct the deformity, but will prevent it from increasing. Voss et al. ( 185) used hemiepiphysiodesis with osteotomy in two patients with growth arrest and varus deformity. The humerus varies in length by a few centimeters from one individual to another, but in general is about 30 cm long at skeletal maturity. Approximately 65% of the length of the humerus is achieved by age 6 years. A 6-year-old child has approximately 10 cm of growth left in the entire humerus, with only approximately 2 cm provided by the distal physis. Growth arrest, in the absence of avascular necrosis or collapse, will be a very slowly evolving phenomenon, and epiphysiodesis in a child older than 6 years will have little effect on longitudinal growth. In general, prevention of increasing deformity from medial growth arrest is the only role for lateral epiphysiodesis. Because of the slow rate of growth in the distal humerus, we do not believe there is any role for lateral epiphysiodesis in correction of a varus

deformity in a child with otherwise normal physis.

### Osteotomy

Osteotomy is the only way to correct a cubitus varus deformity with a high probability of success. A variety of corrective osteotomies have been described, almost all with significant complications (Table 14-3). Stiffness, nerve injury, and recurrent deformities are the most commonly reported complications. An overall complication rate approaching 25% in many series has led to some controversy about the value of a distal humeral corrective osteotomy for cubitus varus deformity.

Study (Year)	Type of Osteotomy	Complications
Oppenheim et al. (1971)	Supracondylar	Stiffness, nerve injury, recurrent deformity
Wilkens and Beaty (1991)	Supracondylar	Stiffness, nerve injury, recurrent deformity
DeRosa and Graziano (1977)	Step cut	Stiffness, nerve injury, recurrent deformity
Clader et al. (1984)	Supracondylar	Stiffness, nerve injury, recurrent deformity
Voss (1985)	Supracondylar	Stiffness, nerve injury, recurrent deformity
Oppenheim et al. (1971)	Supracondylar	Stiffness, nerve injury, recurrent deformity
Wilkens and Beaty (1991)	Supracondylar	Stiffness, nerve injury, recurrent deformity
DeRosa and Graziano (1977)	Step cut	Stiffness, nerve injury, recurrent deformity
Clader et al. (1984)	Supracondylar	Stiffness, nerve injury, recurrent deformity
Voss (1985)	Supracondylar	Stiffness, nerve injury, recurrent deformity
Oppenheim et al. (1971)	Supracondylar	Stiffness, nerve injury, recurrent deformity
Wilkens and Beaty (1991)	Supracondylar	Stiffness, nerve injury, recurrent deformity
DeRosa and Graziano (1977)	Step cut	Stiffness, nerve injury, recurrent deformity
Clader et al. (1984)	Supracondylar	Stiffness, nerve injury, recurrent deformity
Voss (1985)	Supracondylar	Stiffness, nerve injury, recurrent deformity

TABLE 14-3. RESULTS OF SEVERAL STUDIES USING VARIOUS OSTEOTOMIES

To choose an appropriate osteotomy, the exact location of the deformity must be determined. Because malunion is the cause of most cubitus varus deformities, the angular deformity usually occurs at the level of the fracture. If the deformity is caused by growth arrest, the actual deformity will be centered at the site of the growth arrest. Rotation and hyperextension contribute to the deformity, but varus is the most significant factor (43). Hyperextension can produce a severe deformity in some patients. After determining the location and cause of the deformity, the appropriate osteotomy can be chosen. In general, a lateral closing wedge osteotomy with a medial hinge will correct the varus deformity, with some minor correction of hyperextension (9,20,46,70,72,80,98,100,111,177,185,196).

An oblique configuration (Fig. 14-45) places the center of rotation of the corrective osteotomy as close to the actual level of the deformity as possible. Oppenheim et al. recommended osteotomy at a higher level, emphasizing that the length of the cuts should be equal (Fig. 14-46) (142). The higher the osteotomy, the more translation is produced in correcting the angular deformity. Proper preoperative planning places the apex of the osteotomy close to the level of the deformity and obtains the best anatomic result. On an AP radiograph of the humerus with the forearm in full supination, the size of the wedge and the angular correction needed are determined. An "incomplete" lateral closing wedge osteotomy is performed, leaving a small medial hinge of bone intact. The osteotomy usually is fixed with two Kirschner wires placed laterally. In the absence of an intact medial hinge, two lateral wires probably are not sufficient to secure this osteotomy (185).

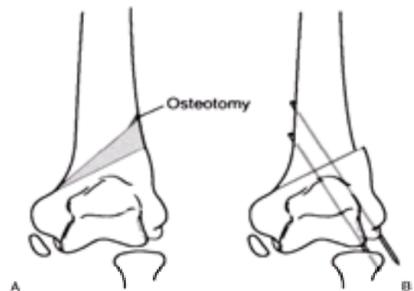


FIGURE 14-45. A: By moving the apex of the closing wedge distally, the center of rotation of the osteotomy is moved closer to the deformity. B: Upon closing a distally based wedge osteotomy, there is less translational effect than in a more proximally based osteotomy.

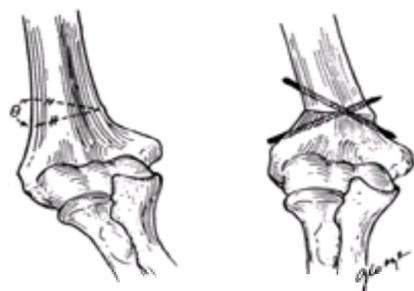
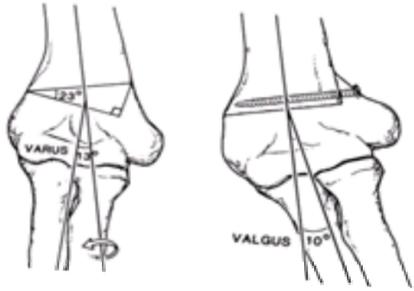


FIGURE 14-46. Technique of the lateral closing wedge osteotomy. The limbs should be of equal length, and the medial cortex should remain intact. (Reprinted from Oppenheim WL, Clader TJ, Smith C, et al. Supracondylar humeral osteotomy for traumatic childhood cubitus varus deformity. *Clin Orthop* 1984;188:36; with permission.)

Wilkens and Beaty (191) recommended crossed wires in this situation. With this incomplete osteotomy, rotation cannot be corrected, but rotational deformity was not found to be a significant problem in studies by Voss (185) and Oppenheim (142).

A dome osteotomy has been described by Japanese surgeons (89) in which a curved osteotomy is made in the supracondylar area. Proponents of this osteotomy suggest that multiplane correction is possible without inducing translation in the distal fragment and that rotation can be corrected. This osteotomy is often performed through a posterior approach.

DeRosa and Graziano (57) described a step cut osteotomy (Fig. 14-47) in which the distal fragment is slotted into the proximal fragment and the osteotomy is secured with a single screw. Multiple osteotomies with varying degrees of complexity have been recommended. In general, increasing complexity means increasing complications. No series of osteotomies has been reported without some significant complications.



**FIGURE 14-47.** The step-cut osteotomy. The distal fragment can be rotated in the horizontal plane ( *circular arrow*) to correct that part of the deformity. (Reprinted from De Rosa GP, Graziano CP. A new osteotomy for cubitus varus. *Clin Orthop* 1988;236:160-165; with permission.)

### Results of Osteotomy

Functional outcomes are generally good, but the preoperative functional deficit is nearly always minor in patients with cubitus varus deformities. Complications of humeral osteotomy include stiffness, nerve injury, and persistent deformity ( [Table 14-3](#)); however, with a properly performed osteotomy, complications are relatively few. Ippolito et al. ( [95](#)) reported long-term follow-up of patients with supracondylar osteotomies, 50% of whom had poor results. Increasing deficit has been reported after osteotomy in young children, but this did not occur in the series reported by Voss et al. ( [185](#)), in which four patients had growth arrest and avascular necrosis; lateral epiphysiodesis was performed to prevent recurrent deformity in two of these patients.

Hyperextension deformity may remodel over time ( [Fig. 14-44](#)), but correction is slow and inconsistent. In the series from Children's Hospital ( [185](#)), hyperextension deformities remodeled as much as 30 degrees in young children, but in children facing maturity, there was no significant remodeling in the flexion/extension plane. If hyperextension appears to be a major problem, osteotomy also should be directed at this deformity rather than simple correction of the varus deformity; this requires a multiplane osteotomy.



### AUTHORS' PREFERRED METHOD OF TREATMENT

I prefer an incomplete lateral closing wedge osteotomy for correction of most cubitus varus deformities. It is a simple procedure and in our hands has had a very low complication rate. A simple lateral approach is used rather than a posterior approach, and it is a muscle-preserving osteotomy that allows rapid rehabilitation after healing. Stiffness and nerve injury are rare complications of this procedure. Performing the procedure with the patient supine allows easy observation of the correction achieved. Kirschner wire fixation can be used in the juvenile age group and screw fixation in adolescents.

There is no reason to mobilize the ulnar nerve, but care is taken not to penetrate the medial cortex with a saw. The position of the radial nerve limits proximal lateral dissection. Symeonides et al. ( [179](#)) reported finding a radial nerve entrapped within fracture callus in the lateral aspect of the metaphysis. With severe deformity or neurologic abnormality, anatomy of neurovascular structures is unlikely to be normal.

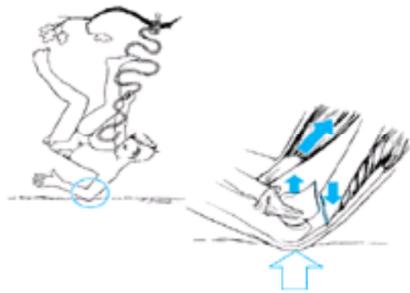
Wilkins recommended combining dome and lateral closing wedge osteotomies through a posterior approach for multiplanar correction. This procedure is performed with the patient prone. For patients with significant hyperextension deformity, we would choose this procedure for multiplanar correction.

### FLEXION-TYPE SUPRACONDYLAR FRACTURE

Flexion-type supracondylar humeral fractures account for about 2% of humeral fractures. A flexion pattern of injury may not be recognized until reduction is attempted because initial radiographs are inadequate. A key to the recognition of a flexion-type supracondylar fracture is that it is unstable in flexion, whereas extension-type fractures generally are stable in hyperflexion. A laterally displaced supracondylar fracture may actually be a flexion-type injury.

#### Etiology and Pathology

The mechanism of injury is generally believed to be a fall directly onto the elbow rather than a fall onto the outstretched hand with hyperextension of the elbow ( [Fig. 14-48](#)). The distal fragment is displaced anteriorly and may migrate proximally in a totally displaced fracture. The ulnar nerve is vulnerable in this fracture pattern ( [3,68,84,158](#)), and it may be entrapped in the fracture or in the healing callus ( [113](#)).



**FIGURE 14-48.** Flexion mechanism. Flexion-type fractures usually result from a blow to the posterior aspect of the elbow. The obliquity of the fracture line may be opposite that of an extension type. The large black arrows demonstrate the usual direction of displacement of the fragments.

#### Radiographic Findings

The radiographic appearance of the distal fragment varies from mild angular deformity to complete anterior displacement. Anterior displacement often is accompanied by medial or lateral translation ( [Fig. 14-49](#)). Associated fractures of the proximal humerus and radius mandate full radiographic evaluation of the upper extremity. Fracture classification is the same as for extension-type supracondylar fractures (Gaetland): type I, nondisplaced fracture; type II, minimally angulated greenstick fracture; and type III, totally unstable displaced distal fracture fragment.



**FIGURE 14-49.** Flexion valgus deformity. Lateral (A) and anteroposterior views (B) of a flexion-type supracondylar fracture. The distal fragment (arrow) is laterally displaced in the coronal plane as well. Despite aggressive treatment, there was mild residual cubitus valgus plus a flexion contracture when the fracture healed (C). (Reprinted from Wilkins KE. Residuals of elbow trauma in children. *Orthop Clin North Am* 1990;21:291-314; with permission.)

### Treatment

In general, type I flexion-type supracondylar fractures are stable nondisplaced fractures that can simply be protected in a long arm cast (62,137,152). If mild angulation, as in a type II fracture, requires some reduction in extension, the arm can be immobilized with the elbow fully extended. Radiologic evaluation with the elbow extended is easily obtained and accurate in determining the adequacy of reduction. Reduction is assessed by evaluating the olecranon fossa, Baumann angle, and the anterior humeral line intersecting the lateral condyle. If reduction cannot be obtained, as is often the case, or if rotation persists, soft tissue interposition, possibly the ulnar nerve, should be suspected.

A problem with type III flexion supracondylar fractures is that reduction is difficult to achieve and when achieved the elbow usually is in extension, making stabilization of the distal fragment with pins quite difficult.

Type I and II fractures (Fig. 14-50 and Fig. 14-51) generally are reduced if any angular displacement is seen on fluoroscopic intraoperative evaluation. Type II fractures can be immobilized in an extension cast with the elbow fully extended (Fig. 14-51). A ring is applied so that the weight of the cast can be supported by a sling around the neck. The cast is removed at 3 weeks. If closed reduction is performed without skeletal stabilization, follow-up radiographs usually are taken at 1 week and then when the cast is removed at 3 weeks.



**FIGURE 14-50.** Type I flexion injury. A type I flexion supracondylar fracture pattern (arrows) in a 6-year-old below-the-elbow amputee. There is only about a 10 degree increase in the shaft condylar angle. He was treated with a simple posterior splint.



**FIGURE 14-51.** Closed reduction, extension cast. A: A 5-year-old girl sustained a type II flexion pattern. B: She was manipulated into extension and found to be stable, and thus was maintained in a long-arm cast in extension.

For unstable type II and III flexion supracondylar fractures, pinning is generally required. The pinning technique described for extension-type supracondylar fractures is not appropriate for this fracture, because its instability in flexion precludes pinning with the elbow hyperflexed. Pinning should be performed after closed reduction with the elbow in mild flexion or full extension.

In a flexion-type supracondylar fracture, if reduction can be obtained in extension, it is rarely if ever stable in flexion. In general, a slightly less than anatomic reduction can be accepted as long as there is no soft tissue interposition of tissue, the Baumann angle is close to the other side, and neither flexion nor extension is seen on the lateral view. Although rotation of the arm is often possible for a lateral view of extension supracondylar fracture, the C-arm must be moved to get satisfactory radiographs when pinning a flexion-type supracondylar fracture, because they usually are rotationally unstable even when reduced.

Pinning is generally performed with the elbow in about 30 degrees of flexion, holding the elbow in a reduced position (Fig. 14-52). If closed reduction can be obtained, pinning can be accomplished in this position. Reduction may be facilitated by placing a traction type of olecranon wing nut through the proximal ulna to give a better grip on the distal fragment.



**FIGURE 14-52.** Closed reduction, pin fixation. **A:** Injury film of a 9-year-old with a type II flexion injury. **B:** A satisfactory reduction was achieved by fully extending the elbow. **C:** The elbow was then gradually flexed to full flexion, maintaining pressure proximally through the forearm (arrows) to keep the distal humeral fragment extended. **D:** The distal fragment was then secured with three pins placed laterally.

The lateral pin is generally placed first through the lateral condyle, extending through the proximal fragment and engaging the opposite cortex. The medial pin is then placed through the medial epicondyle. We make a small incision over the medial epicondyle to ensure that the ulnar nerve is not entrapped in the fracture.

After pinning of a flexion-type supracondylar fracture, the arm should be placed in a cast. If the fracture is held in anatomic position with pins, a flexed-arm cast can be used to provide better patient comfort, but a cast with the elbow in maximal extension is acceptable.

Open reduction frequently is required for flexion-type supracondylar fractures. Open reduction is best performed through an anteromedial approach, rather than an anterior approach, as is used for extension-type supracondylar fractures. With flexion-type fractures, brachialis remains intact and must be retracted in order to expose the fracture, necessitating a medial extension to the anterior approach. To be sure that the ulnar nerve is not entrapped in the fracture site, exploration of the ulnar nerve or at least identification is probably advisable with this fracture. Fracture reduction should be obtained under direct vision of the fracture, and pins should be used to stabilize the fracture fragments.

#### **Anteromedial Open Reduction of Flexion-Type Supracondylar Fractures**

A transverse incision is made across the antecubital fossa, curving proximally posterior to the neuromuscular bundle. Dissection is carried down to the level of the superficial fascia of the forearm and antecubital fossa. The neurovascular bundle is identified and retracted medially. The brachialis and biceps tendons are retracted laterally to expose the fracture site and facilitate reduction. If there is medial soft tissue impingement or a question of ulnar nerve entrapment within the fracture, the dissection should be carried around posterior to the medial epicondyle, so the ulnar nerve and fracture can be identified.

Wound closure is performed with interrupted stitches, either absorbable or nylon. Postoperative immobilization is maintained for 3 or 4 weeks until good callus formation is present. Pins generally are left out through the skin and removed in the office without need for anesthetic. No immediate rehabilitation is given, but the patient is encouraged to begin gentle activities with the arm and to begin regaining motion without a stressful exercise program.

#### **Traction**

Traction can be used for this fracture, but the elbow generally is unstable in increased flexion, which is a position of comfort for the patient in traction. Although overhead traction works best for extension-type supracondylar fractures, we think that side arm traction might be better for this fracture; however, there is no data on this issue that we are aware of. Radiographic evaluation in traction is mandatory to confirm the adequacy of reduction. After 14 days or so in traction, with a stable distal humeral fracture and good callus formation, the arm is immobilized in a cast with the elbow in extension, and radiographs are obtained. We prefer wing nut–type traction for this to apply skeletal traction with the elbow flexed 30 to 40 degrees for comfort. This may require some sling suspension, as well as a skeletal traction pin or wing nut. If an ulnar neuropathy develops in traction, the nerve is probably trapped in the fracture and open reduction and ulnar nerve exploration are indicated.

### **AUTHORS' PREFERRED METHOD OF TREATMENT**

In general, we treat type I flexion supracondylar fractures with a splint or cast with the elbow flexed for comfort. Minimally displaced type II fractures that reduce in extension are treated in an extension cast. Unstable types II and III fractures are pinned. We make a small incision over the medial epicondyle if a medial pin is placed. Open reduction is used if an anatomic closed reduction cannot be obtained.

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### **CHAPTER REFERENCES**

1. Abe M, Ishizu T, Shirai H, et al. Tardy ulnar nerve palsy caused by cubitus varus deformity. *J Hand Surg [Am]* 1995;20:5–9.
2. Abraham E, Powers T, Witt P, et al. Experimental hyperextension supracondylar fractures in monkeys. *Clin Orthop* 1982;171:309–318.
3. Aitken AP, Smith L, Blackette CW. Supracondylar fractures in children. *Am J Surg* 1943;59:161–171.
4. Alburger PD, Weidner PL, Randal RB. Supracondylar fractures of the humerus in children. *J Pediatr Orthop* 1992;12:16–19.
5. Alcott WH, Bowden BW, Miller PR. Displaced supracondylar fractures of the humerus in children: long-term follow-up of 69 patients. *J Am Osteopath Assoc* 1977;76:910–915.
6. Allison N. Fractures about the elbow. *JAMA* 1927;89:1568–1572.
7. Alonso-Llames M. Bilateral approach to the elbow. *Acta Orthop Scand* 1972;43:479–490.
8. Amillo S, Barrios R, Martinez-Peric R, et al. Surgical treatment of the radial nerve lesions associated with fractures of the humerus. *J Orthop Trauma* 1993;7:211–215.
9. Amspacher J, Merissenbaugh J. Supracondylar osteotomy of the humerus for correction of rotational and angular deformities of the elbow. *South Med J* 1964;57:846–850.
10. Archibald DAA, Roberts JA, Smith MGH. Transarticular fixation for severely displaced supracondylar fractures in children. *J Bone Joint Surg [Br]* 1991;73:147–149.
11. Archibeck M, Scott S, Peters C. Brachialis muscle entrapment in displaced supracondylar humerus fractures: a technique of closed reduction and report of initial results. *J Pediatr Orthop* 1997;17:298–302.
12. Arino VL, Lluch EE, Ramirez AM, et al. Percutaneous fixation of supracondylar fractures of the humerus in children. *J Bone Joint Surg [Am]* 1977;59:914–916.
13. Arnold JA, Nasca RJ, Nelson CL. Supracondylar fractures of the humerus. *J Bone Joint Surg [Am]* 1977;59:589–595.
14. Aronson DC, Van Vollenhoven E, Meeuwis JD. K-wire fixation of supracondylar humeral fractures in children: results of open reduction via a ventral approach in comparison with closed treatment. *Injury* 1993;24:179–181.
15. Aronson DD, Prager PI. Supracondylar fractures of the humerus in children: a modified technique for closed pinning. *Clin Orthop* 1987;219:174–184.
16. Badhe N, Howard P. Olecranon screw traction for displaced supracondylar fractures of the humerus in children. *Injury* 1998;29:457–460.
17. Bailey GG Jr. Nerve injuries in supracondylar fractures of the humerus in children. *N Engl J Med* 1939;221:260–263.
18. Bakalim G, Wilppula E. Supracondylar humeral fractures in children. *Acta Orthop Scand* 1972;43:366–374.
19. Banskota A, Volz RG. Traumatic laceration of the radial nerve following supracondylar fracture of the elbow. *Clin Orthop* 1984;184:150–156.
20. Bellemore M, Barret I, Middleton RW, et al. Supracondylar osteotomy of the humerus for correction of cubitus varus. *J Bone Joint Surg [Br]* 1984;66:566–572.
21. Bender J. Cubitus varus after supracondylar fracture of the humerus in children: can this deformity be prevented? *Reconstr Surg Traumatol* 1979;17:100–106.
22. Berghausen T, Leslie BM, Ruby LK, et al. The severely displaced pediatric supracondylar fracture of humerus. *Orthop Rev* 1986;15:510–515.
23. Bialik B, Weiner A, Fishman J. Scoring system for assessing the treatment of supracondylar fractures of the humerus. *Isr J Med Sci* 1983;19:173–175.
24. Bindra RR. Brachial artery aneurysm following supracondylar fracture of the humerus. 1990 (unpublished data).
25. Blount WP. Volkmann's ischemic contracture. *Surg Gynecol Obstet* 1950;90:244–246.
26. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955:26–42.
27. Bosanquet JS, Middleton RW. The reduction of supracondylar fractures of the humerus in children treated by traction-in-extension: a review of 18 cases. *Injury* 1983;14:373.
28. Bostman O, Makela E, Sodergard J, et al. Absorbable polyglycolide pins in internal fixation of fractures in children. *J Pediatr Orthop* 1993;13:242–245.

29. Boyd DW, Aronson DD. Supracondylar fractures of the humerus: a prospective study of percutaneous pinning. *J Pediatr Orthop* 1992;12:789–794.
30. Boyd HB, Altenberg AR. Fractures about the elbow in children. *Arch Surg* 1944;49:213–224.
31. Brewster AH, Karp M. Fractures in the region of the elbow in children: an end-result study. *Surg Gynecol Obstet* 1940;71:643–649.
32. Bristow WR. Myositis ossificans and Volkmann's paralysis: notes on two cases, illustrating the rarer complications of supracondylar fracture of the humerus. *Br J Surg* 1923;10:475–481.
33. Brown I, Zinar D. Traumatic and iatrogenic neurological complications after supracondylar humerus fractures in children. *J Pediatr Orthop* 1995;15:440–443.
34. Buhl O, Hellberg S. Displaced supracondylar fractures of the humerus in children. *Acta Orthop Scand* 1982;53:67–71.
35. Cairns RA, Mackenzie WG, Culham JAG. Urokinase treatment of forearm ischemia complicating supracondylar fracture of the humerus in three children. *Pediatr Radio* 1993;23:391–394.
36. Campbell CC, Waters PM, Emans JB, et al. Neurovascular injury and displacement in type III supracondylar humerus fractures. *J Pediatr Orthop* 1995;15:47–52.
37. Carcassone M, Bergoin M, Hornung H. Result of operative treatment of severe supracondylar fractures of the elbow in children. *J Pediatr Surg* 1972;7:676–679.
38. Carlson CS, Rosman MA. Cubitus varus: a new and simple technique for correction. *J Pediatr Orthop* 1982;2:199–201.
39. Celiker O, Pestilci FI, Tuzuner M. Supracondylar fractures of the humerus in children: analysis of the results in 142 patients. *J Orthop Trauma* 1990;4:265–269.
40. Charnley J. *Closed treatment of common fractures*, 3rd ed. Edinburgh: Churchill Livingstone, 1961:105–115.
41. Chattoadhyay A. Suggested method of fixation in supracondylar fracture. *J Ind Med Assoc* 1984;82:204–205.
42. Cheng J, Lam T, Shen W. Closed reduction and percutaneous pinning for type III displaced supracondylar fractures of the humerus in children. *J Orthop Trauma* 1995;9:511–515.
43. Chess DG, Leahey JL, Hyndman JC. Cubitus varus: significant factors. *J Pediatr Orthop* 1994;14:190–192.
44. Copley L, Dormans J, Davidson R. Vascular injuries and their sequelae in pediatric supracondylar humeral fractures: toward a goal of prevention. *J Pediatr Orthop* 1996;16:99–103.
45. Corkery PH. The management of supracondylar fractures in the humerus in children. *Br J Clin Pract* 1964;18:583–591.
46. Cotton FJ. Elbow fractures in children. *Ann Surg* 1902;35:252–269.
47. Coventry MB, Henderson CC. Supracondylar fractures of the humerus: 49 cases in children. *Rocky Mount Med J* 1956;53:458–465.
48. Cramer KE, DeVito DP, Green NE. Comparison of closed reduction and percutaneous pinning versus open reduction and percutaneous pinning in displaced supracondylar fractures of the humerus in children. *J Orthop Trauma* 1992;6:407–412.
49. Cramer KE, Green NE, DeVito DP. Incidence of anterior interosseous nerve palsy in supracondylar humerus fractures in children. *J Pediatr Orthop* 1993;13:502–505.
50. Culp RW, Osterman AL, Davidson RS, et al. Neurological complications associated with supracondylar fractures of the humerus in children. *J Bone Joint Surg [Am]* 1990;72:1211–1214.
51. D'Ambrosia RD. Supracondylar fractures of humerus: prevention of cubitus varus. *J Bone Joint Surg [Am]* 1972;54:60–66.
52. Danielsson L, Petterson H. Open reduction and pin fixation of severely displaced supracondylar fractures of the humerus in children. *Acta Orthop Scand* 1980;51:249–255.
53. Danielsson LG, Hussein S, El-Haddad I, et al. Staple fixation of osteotomy for cubitus varus: a simple technique used in 11 children. *Acta Orthop Scand* 1991;62:55–57.
54. Davids JR, Maguire MF, Mubarak SJ, et al. Lateral condylar fracture of the humerus following posttraumatic cubitus varus. *J Pediatr Orthop* 1994;14:466–470.
55. de Jager L, Hoffman E. Fracture–separation of the distal humeral epiphysis [Review]. *J Bone Joint Surg [Br]* 1991;73:143–146.
56. DeLee J, Wilkins K, Rogers L, et al. Fracture separation of the distal humeral epiphysis. *J Bone Joint Surg [Am]* 1980;62:46–51.
57. De Rosa GP, Graziano GP. A new osteotomy for cubitus varus. *Clin Orthop* 1988;236:160–165.
58. Dormans JP, Squillante R, Sharf H. Acute neurovascular complications with supracondylar humerus fractures in children. *J Hand Surg [Am]* 1995;20:1–4.
59. Dowd GSE, Hopcroft PW. Varus deformity in supracondylar fractures of the humerus in children. *Injury* 1978;10:297–303.
60. Edman P, Lohr G. Supracondylar fractures of the humerus treated with olecranon traction. *Acta Chir Scand* 1963;126:505–516.
61. Eid AM. Reduction of displaced supracondylar fracture of the humerus in children by manipulation in flexion. *Acta Orthop Scand* 1978;49:39–45.
62. El-Ahwany MD. Supracondylar fractures of the humerus in children with a note on the surgical correction of late cubitus varus. *Injury* 1974;6:45–46.
63. El-Sharkawi H, Fattah HA. Treatment of displaced supracondylar fractures of the humerus in children in full extension and supination. *J Bone Joint Surg [Br]* 1965;47:273–279.
64. Farnsworth C, Silva P, Mubarak S. Etiology of supracondylar humerus fractures. *J Pediatr Orthop* 1998;18:38–42.
65. Fleuriau-Chateau P, McIntyre W, Letts M. An analysis of open reduction of irreducible supracondylar fractures of the humerus in children. *Can J Surg* 1998;41:112–118.
66. Flynn JC, Matthews JG, Benoit RL. Blind pinning of displaced supracondylar fractures of the humerus in children. *J Bone Joint Surg [Am]* 1974;56:263–273.
67. Flynn JC, Zink WP. Fractures and dislocations of the elbow. In: MacEwen GD, Kasser JR, Heinrich SD, eds. *Pediatric fractures. A practical approach to assessment and treatment*. Baltimore: Williams & Wilkins, 1993:133–164.
68. Fowles JV, Kassab MT. Displaced supracondylar fractures of the elbow in children. *J Bone Joint Surg [Br]* 1974;56:490–500.
69. France J, Strong M. Deformity and function in supracondylar fractures of the humerus in children variously treated by closed reduction and splinting, traction, and percutaneous pinning. *J Pediatr Orthop* 1992;12:494–498.
70. French PR. Varus deformity of elbow following supracondylar fractures of the humerus in children. *Lancet* 1959;2:439–441.
71. Furrer M, Mark G, Ruedi T. Management of displaced supracondylar fractures of the humerus in children. *Injury* 1991;22:259–262.
72. Gaddy BC, Manske PR, Pruitt DL, et al. Distal humeral osteotomy for correction of posttraumatic cubitus varus. *J Pediatr Orthop* 1994;14:214–219.
73. Gao G. A simple technique for correction of cubitus varus. *Chin Med J [Engl]* 1986;99:853–854.
74. Gartland JJ. Management of supracondylar fractures of the humerus in children. *Surg Gynecol Obstet* 1959;109:145–154.
75. Gates DJ. Supracondylar fracture of humerus: problem in children managed with open reduction. *Orthop Rev* 1982;11:91–98.
76. Gehling H, Gotzen L, Giannadakis K, et al. Treatment and outcome of supracondylar humeral fractures in childhood. *Unfallchirurgie* 1995;98:93–97.
77. Gennari J, Merrot T, Piclet B, et al. Anterior approach versus posterior approach to surgical treatment of children's supracondylar fractures: comparative study of thirty cases in each series. *J Pediatr Orthop* 1998;7:307–313.
78. Gillingham BL, Rang M. Advances in children's elbow fractures [Editorial]. *J Pediatr Orthop* 1995;15:419–421.
79. Godley DR, Leong JCY, Yau A. Open reduction and internal fixation of supracondylar fractures of the humerus in children in Hong Kong: long-term results. *Abbot Proc* 1978;9:30–34.
80. Graham B, Tredwell SJ, Beauchamp RD, et al. Supracondylar osteotomy of the humerus for correction of cubitus varus. *J Pediatr Orthop* 1990;10:228–231.
81. Griffin PP. Supracondylar fractures of the humerus. *Pediatr Clin North Am* 1975;2:477–486.
82. Gruber MA, Hudson OC. Supracondylar fracture of the humerus in childhood. *J Bone Joint Surg [Am]* 1964;46:1245.
83. Hadlow A, Devane P, Nicol R. A selective treatment approach to supracondylar fracture of the humerus in children. *J Pediatr Orthop* 1996;16:104–106.
84. Hagen R. Skin traction treatment of supracondylar fractures of the humerus in children. *Acta Orthop Scand* 1964;35:138–148.
85. Hart GM, Wilson DW, Arden GP. The operative management of the difficult supracondylar fracture of the humerus in the child. *Injury* 1977;9:30–34.
86. Hart VL. Reduction of supracondylar fractures in children. *Surgery* 1942;11:33–37.
87. Henrikson B. Supracondylar fracture of the humerus in children. *Acta Chir Scand [Suppl]* 1966;369:1–72.
88. Heppenstall RB, Spega AA, Scott R, et al. The compartment syndrome. An experimental and clinical study of muscular energy metabolism. *Clin Orthop Rel Res* 1988;226:138–155.
89. Higaki T, Ikuta Y. The new operation method of the domed osteotomy for 4 children with varus deformity of the elbow joint. *J Jpn Orthop* 1982;31:300–335.
90. Holden CEA. The pathology and prevention of Volkmann's ischaemic contracture. *J Bone Joint Surg [Br]* 1979;61:296–299.
91. Holmberg L. Fractures in the distal end of the humerus in children. *Acta Chir Scand [Suppl]* 1945:103.
92. Hoyer A. Treatment of supracondylar fracture of the humerus by skeletal traction in an abduction splint. *J Bone Joint Surg [Am]* 1952;34:623–637.
93. Ikram M. Ulnar nerve palsy: a complication following percutaneous fixation of supracondylar fractures of the humerus in children. *Injury* 1996;27:303–305.
94. Ippolito E, Caterini R, Scola E. Supracondylar fractures of the humerus in children. *J Bone Joint Surg [Am]* 1986;68:333–344.
95. Ippolito E, Moneta MR, D'Arrigo C. Post-traumatic cubitus varus. Long-term follow-up of corrective supracondylar humeral osteotomy in children. *J Bone Joint Surg [Am]* 1990;72:757–765.
96. Iyengar S, Hoffinger S, Townsend D. Early versus delayed reduction and pinning of type III displaced supracondylar fractures of the humerus in children: a comparative study. *J Orthop Trauma* 1999;13:51–55.
97. Jefferiss CD. Straight lateral traction in selected supracondylar fractures of the humerus in children. *Injury* 1977;8:213–220.
98. Johnson E, Oppenheim WL. The problem: cubitus varus after elbow fracture. *Orthop Consultation* 1985:8–12.
99. Jones KG. Percutaneous pin fixation of fracture of the lower end of the humerus. *Clin Orthop* 1967;50:53–69.
100. Kagan N, Herold HZ. Correction of axial deviations after supracondylar fractures of the humerus in children. *Int Surg* 1973;58:735–737.
101. Kallio PE, Foster BK, Paterson DC. Difficult supracondylar elbow fractures in children: analysis of percutaneous pinning technique. *J Pediatr Orthop* 1992;12:11–15.
102. Kanaujia RR, Ikuta Y, Muneshige H, et al. Dome osteotomy for cubitus varus in children. *Acta Orthop Scand* 1988;59:314–317.
103. Kekomaki M, Luoma R, Rikalainen H, et al. Operative reduction and fixation of a difficult supracondylar extension fracture of the humerus. *J Pediatr Orthop* 1984;4:13–15.
104. Khare GN, Gautam VK, Kochhar VL, et al. Prevention of cubitus varus deformity in supracondylar fractures of the humerus. *Injury* 1991;22:202–206.
105. King D, Secor C. Bow elbow (cubitus varus). *J Bone Joint Surg [Am]* 1951;33:572–576.
106. Kocher T. *Beitrage zur Kenntniss Einiger Praktisch Wichtiger Fracturformen*. Basel, Switzerland: Carl Sallmann, 1895.
107. Kramhott M, Keller IL, Solgaard S. Displaced supracondylar fractures of the humerus in children. *Clin Orthop* 1987;221:215–220.
108. Krebs B. Operative treatment of supracondylar fractures of the humerus in children. A follow-up investigation of 23 children treated with open reduction and osteosynthesis with Kirschner wire. *Ugeskr Laeger* 1980;142:871–872.
109. Kristensen JL, Vivild O. Supracondylar fractures of the humerus in children. *Acta Orthop Scand* 1976;47:375–380.
110. Kurer MHJ, Regan MW. Completely displaced supracondylar fractures of the humerus in children. A review of 1708 cases. *Clin Orthop* 1990;256:205–214.
111. LaBelle H, Bunnell WP, Duhaime M, et al. Cubitus varus deformity following supracondylar fractures of the humerus in children. *J Pediatr Orthop* 1982;2:539–546.
112. Lal GM, Bhan S. Delayed open reduction for supracondylar fractures of the humerus. *Int Orthop* 1991;15:189–191.
113. Lalanandham T, Laurence WN. Entrapment of the ulnar nerve in the callus of a supracondylar fracture of the humerus. *Injury* 1984;16:129–130.
114. Langenskiold A, Kivilaakso R. Varus and valgus deformity of the elbow following supracondylar fracture of the humerus. *Acta Orthop Scand* 1967;38:313–320.
115. Laupattarakasem W, Mahaisavariya B, Kowsuwon W, et al. Pentalateral osteotomy for cubitus varus: clinical experiences of a new technique. *J Bone Joint Surg [Br]* 1989;71:667–670.
116. Laurence W. Supracondylar fractures of the humerus in children. A review of 100 cases. *Br J Surg* 1957;44:143–147.

117. Lipscomb PR, Burleson RJ. Vascular and neural complications in supracondylar fractures of the humerus in children. *J Bone Joint Surg [Am]* 1955;37:487-492.
118. Lyons J, Ashley E, Hoffer M. Ulnar nerve palsies after percutaneous cross-pinning of supracondylar fractures in children's elbows. *J Pediatr Orthop* 1998;18:43-45.
119. Mahaisavariya B, Laupattarakasem W. Supracondylar fracture of the humerus: malrotation versus cubitus varus deformity. *Injury* 1993;24:416-418.
120. Mahaisavariya B, Laupattarakasem W. Rotational deformity of the distal humerus in cubitus varus. *J Med Assoc Thai* 1994;77:19-24.
121. Mapes R, Hennrikus W. The effect of elbow position on the radial pulse measured by Doppler ultrasonography after surgical treatment of supracondylar elbow fractures in children. *J Pediatr Orthop* 1998;18:441-444.
122. Marion J, LaGrange J, Faysse R, et al. Les fractures de l'extremite inferieure de l'humerus chez l'enfant. *Rev Chir Orthop* 1962;48:337-413.
123. Martin DF, Tolo VT, Sellers DS, et al. Radial nerve laceration and retraction associated with a supracondylar fracture of the humerus. *J Hand Surg [Am]* 1989;14:542-545.
124. Maylahn DJ, Fahey JJ. Fractures of the elbow in children. *JAMA* 1958;166:220-228.
125. McCoy GF, Piggot J. Supracondylar osteotomy for cubitus varus: the value of the straight arm position. *J Bone Joint Surg [Br]* 1988;70:283-286.
126. McGraw JJ, Akbarnia BA, Hanel DP, et al. Neurological complications resulting from supracondylar fractures of the humerus in children. *J Pediatr Orthop* 1986;6:647-650.
127. Mehlman C, Crawford A, McMillion T, et al. Operative treatment of supracondylar fractures of the humerus in children: the Cincinnati experience [Review]. *Acta Orthop Belg* 1996;62(suppl):41-50.
128. Meherle WL, Meehan PL. Treatment of the displaced supracondylar fracture of the humerus (type III) with closed reduction and percutaneous cross-pin fixation. *J Pediatr Orthop* 1991;11:705-711.
129. Michael S, Stanislas M. Localization of the ulnar nerve during percutaneous wiring of supracondylar fractures in children. *Injury* 1996;27:301-302.
130. Miller HG, Wilkins KE. The supracondylar fracture of the humerus in children. An analysis of complications. (Unpublished data, University of Texas Medical School, San Antonio, Texas, 1979.)
131. Miller OL. Blind nailing of the T fracture of the lower end of the humerus which includes the joint. *J Bone Joint Surg* 1939;21:933-938.
132. Millis MB, Singer IJ, Hall JE. Supracondylar fracture of the humerus in children: further experience with a study in orthopaedic decision making. *Clin Orthop* 1984;188:90-97.
133. Minkowitz B, Busch MT. Supracondylar humerus fractures. Current trends and controversies. *Orthop Clin North Am* 1994;25:581-594.
134. Mitchell WJ, Adams JP. Fractures and dislocations of the elbow in children. *Curr Pract Orthop Surg* 1964;2:102-124.
135. Mubarak SJ, Carroll NC. Volkmann's contracture in children: aetiology and prevention. *J Bone Joint Surg [Br]* 1979;61:285-293.
136. Nacht J, Ecker M, Chung S, et al. Supracondylar fractures of the humerus in children treated by closed reduction and percutaneous pinning. *Clin Orthop* 1983;177:203-209.
137. Nand S. Management of supracondylar fractures of the humerus in children. *Int Surg* 1972;57:893-898.
138. Nassar A, Chater E. Open reduction and Kirschner wire fixation for supracondylar fracture of the humerus. *J Bone Joint Surg [Br]* 1976;58:135-136.
139. Nassar A, Chater E. Open reduction and Kirschner wire fixation for supracondylar fracture of the humerus [Scientific Exhibit]. Presented at the American Orthopaedic Association Annual Meeting, Toronto, 1992.
140. Ogino T, Minami A, Fukuda K. Tardy ulnar nerve palsy caused by cubitus varus deformity. *J Hand Surg [Br]* 1986;11:352-356.
141. Onwuanyi O, Nwobi D. Evaluation of the stability of pin configuration in K-wire fixation of displaced supracondylar fractures in children. *Int Surg* 1998;83:271-274.
142. Oppenheim WL, Clader TJ, Smith C, et al. Supracondylar humeral osteotomy for traumatic childhood cubitus varus deformity. *Clin Orthop* 1984;188:34-39.
143. Ottolenghi CE. Acute ischemic syndrome: its treatment; prophylaxis of Volkmann's syndrome. *Am J Orthop* 1960;2:312-316.
144. Palmer EE, Niemann KM, Vesely D, et al. Supracondylar fracture of the humerus in children. *J Bone Joint Surg [Am]* 1978;60:653-656.
145. Peters C, Scott S, Stevens P. Closed reduction and percutaneous pinning of displaced supracondylar humerus fractures in children: description of a new closed reduction technique for fractures with brachialis muscle entrapment. *J Orthop Trauma* 1995;9:430-434.
146. Piggot J, Graham HK, McCoy GF. Supracondylar fractures of the humerus in children. Treatment by straight lateral traction. *J Bone Joint Surg [Br]* 1986;68:577-583.
147. Pirone AM, Graham HK, Krajbich JI. Management of displaced extension-type supracondylar fractures of the humerus in children. *J Bone Joint Surg [Am]* 1988;70:641-650.
148. Pirone AM, Krajbich JI, Graham HK. Management of displaced supracondylar fractures of the humerus in children [Letter]. *J Bone Joint Surg [Am]* 1989;71:313.
149. Prevot J, Lascombes P, Metaizeau JP, et al. Supracondylar fractures of the humerus in children. Treatment by downward pinning. *Fr J Orthop Surg* 1990;76:171-177.
150. Prietto CA. Supracondylar fractures of the humerus. *J Bone Joint Surg [Am]* 1979;61:425-428.
151. Ramsey RH, Griz J. Immediate open reduction and internal fixation of severely displaced supracondylar fractures of the humerus in children. *Clin Orthop* 1973;90:130-132.
152. Rang M. *Children's fractures*. Philadelphia: JB Lippincott, 1974.
153. Rasool MN. Ulnar nerve injury after K-wire fixation of supracondylar humerus fractures in children. *J Pediatr Orthop* 1998;18:686-690.
154. Ray SA, Ivory JP, Beavis JP. Use of pulse oximetry during manipulation of supracondylar fractures of the humerus. *Injury* 1991;22:103-104.
155. Reed FE, Apple DF. Ipsilateral fractures of the elbow and forearm. *South Med J* 1976;69:149-151.
156. Reinaerts HHM, Cheriex EC. Assessment of dislocation in the supracondylar fracture of the humerus, treated by overhead traction. *Reconstr Surg Traumatol* 1979;17:92-99.
157. Royce RO, Dutkowsky JP, Kasser JR, et al. Neurological complications after K-wire fixation of supracondylar humerus fractures in children. *J Pediatr Orthop* 1991;11:191-194.
158. Royle SG, Burke D. Ulnar neuropathy after elbow injury in children. *J Pediatr Orthop* 1990;10:495-496.
159. Sabharwal S, Tredwell S, Beauchamp R, et al. Management of pulseless pink hand in pediatric supracondylar fractures of humerus. *J Pediatr Orthop* 1997;17:303-310.
160. Sairyo K, Henmi T, Kanematsu Y, et al. Radial nerve palsy associated with slightly angulated pediatric supracondylar humerus fracture. *J Orthop Trauma* 1997;11:227-229.
161. Salter RB. *Textbook of disorders and injuries of the musculoskeletal system*. Baltimore: Williams & Wilkins, 1970.
162. Schoenecker P, Delgado E, Rotman M, et al. Pulseless arm in association with totally displaced supracondylar fracture. *J Orthop Trauma* 1996;10:410-415.
163. Sharrard WJW. *Paediatric orthopaedics and fractures*. Oxford: Blackwell Scientific, 1971.
164. Shaw BA, Kasser JR, Emans JB, et al. Management of Vascular Injuries in Displaced Supracondylar Humerus Fractures Without Arteriography. *J Orthop Trauma* 1990;4:25-29.
165. Shifrin PG, Gehling HW, Iglesias LJ. Open reduction and internal fixation of displaced supracondylar fractures of the humerus in children. *Orthop Clin North Am* 1976;7:573-581.
166. Sibly TF, Briggs PJ, Gibson MJ. Supracondylar fractures of the humerus in childhood: range of movement following the posterior approach to open reduction. *Injury* 1991;22:456-458.
167. Siris IE. Supracondylar fracture of the humerus. *Surg Gynecol Obstet* 1939;68:201-220.
168. Skolnick MD, Hall JE, Micheli LJ. Supracondylar fractures of the humerus in children. *Orthopedics* 1980;3:395-406.
169. Smith FM. Kirschner wire traction in elbow and upper arm injuries. *Am J Surg* 1947;74:700-787.
170. Smith FM. *Surgery of the elbow*. Philadelphia: WB Saunders, 1972.
171. Smith L. Deformity following supracondylar fractures of the humerus. *J Bone Joint Surg [Am]* 1960;42:235-252.
172. Smith L. Supracondylar fractures of the humerus treated by direct observation. *Clin Orthop* 1967;50:37-42.
173. Sorrel E, Sorrel-Dejerine. Les lesions nerveuses dans les fractures fermies recentes de l'extremite inferieure de l'humerus. *Rev Orthop* 1938;25:609-647.
174. Spinner M, Schreiber SN. Anterior interosseous nerve paralysis as a complication of supracondylar fractures of the humerus in children. *J Bone Joint Surg [Am]* 1969;51:1584-1590.
175. Strait R, Siegel R, Shapiro R. Humeral fractures without obvious etiologies in children less than 3 years of age: when is it abuse? *Pediatrics* 1995;96(part 1):667-671.
176. Sutton WR, Greene WB, Georgopoulos G, et al. Displaced supracondylar humerus fractures in children. A comparison of results and costs in patients treated by skeletal traction versus percutaneous pinning. *Clin Orthop* 1992;278:81-87.
177. Sweeney JG. Osteotomy of the humerus for malunion of supracondylar fractures. *J Bone Joint Surg [Br]* 1975;57:117.
178. Swenson AL. The treatment of supracondylar fractures of the humerus by Kirschner wire transfixion. *J Bone Joint Surg [Am]* 1948;30:993-997.
179. Symeonides PO, Paschaloglou C, Pagelides T. Radial nerve enclosed in the callus of a supracondylar fracture. *J Bone Joint Surg [Br]* 1975;57:523-524.
180. Te Slaa RL, Faber FWM, Nollen AJG, et al. Supracondylar fractures of the humerus in children: a long-term follow-up study. *Neth J Surg* 1988;40:100-103.
181. Topping RE, Blanco JS, David TJ. Clinical evaluation of crossed-pin versus later-pin fixation in displaced supracondylar humerus fractures. *J Pediatr Orthop* 1995;15:435-439.
182. Uchida Y, Sugioka Y. Ulnar nerve palsy after supracondylar humerus fracture. *Acta Orthop Scand* 1990;61:118-119.
183. Vahvanen V, Aalto K. Supracondylar fractures of the humerus. *Acta Orthop Scand* 1978;49:225-233.
184. Van Egmond DB, Tavenier D, Meeuwis JD. Anatomical and functional results after treatment of dislocated supracondylar fractures of the humerus in children. *Neth J Surg* 1985;37:45-49.
185. Voss FR, Kasser JR, Trepman E, et al. Uniplanar supracondylar humeral osteotomy with preset Kirschner wires for posttraumatic cubitus varus. *J Pediatr Orthop* 1994;14:471-478.
186. Walloe W, Egund N, Eikelund L. Supracondylar fracture of the humerus in children: review of closed and open reduction leading to a proposal for treatment. *Injury* 1985;16:296-299.
187. Watson-Jones R. *Fractures and joint injuries*. Edinburgh: ES Livingstone, 1956.
188. Webb AJ, Sherman FC. Supracondylar fractures of the humerus in children. *J Pediatr Orthop* 1989;9:315-325.
189. Weiland AJ, Meyer S, Tolo VT, et al. Surgical treatment of displaced supracondylar fractures of the humerus in children. *J Bone Joint Surg* 1978;56:657-661.
190. Wilkins K. Supracondylar fractures: what's new? [Review]. *J Pediatr Orthop* 1997;6:110-116.
191. Wilkins K, Beaty J. *Fractures in children*, 4th ed. Vol. 3. Philadelphia: Lippincott-Raven, 1996.
192. Wilkins KE. Fractures and dislocations of the elbow region. In: Rockwood CA Jr, Wilkins KE, King RE, eds. *Fractures in children*, 3rd ed. Vol. 3. Philadelphia: JB Lippincott, 1991:509-828.
193. Williamson DM, Cole WG. Treatment of selected extension supracondylar fractures of the humerus by manipulation and strapping in flexion. *Injury* 1993;24:249-252.
194. Willis RB, Rorabeck CH. Treatment of compartment syndrome in children. *Orthop Clin North Am* 1990;21:401-412.
195. Wilson PD. Fractures and dislocations in the region of the elbow. *Surg Gynecol Obstet* 1933;56:335-359.
196. Wong HK, Balasubramaniam P. Humeral torsional deformity after supracondylar osteotomy for cubitus varus: its influence on the postosteotomy carrying angle. *J Pediatr Orthop* 1992;12:490-493.
197. Worlock PH, Colton C. Severely displaced supracondylar fractures of the humerus in children: a simple method of treatment. *J Pediatr Orthop* 1987;7:49-53.
198. Zaltz I, Waters P, Kasser J. Ulnar nerve instability in children. *J Pediatr Orthop* 1996;16:567-569.
199. Zions LE, McKellop HA, Hathaway R. Torsional strength of pin configurations used to fix supracondylar fractures of the humerus in children. *J Bone Joint Surg [Am]* 1994;76:253-256.
200. Ziv N, Litwin A, Katz K, et al. Definitive diagnosis of fracture-separation of the distal humeral epiphysis in neonates by ultrasonography. *Pediatr Radio* 1996;26:493-496.

## THE ELBOW: PHYSEAL FRACTURES, APOPHYSEAL INJURIES OF THE DISTAL HUMERUS, AVASCULAR NECROSIS OF THE TROCHLEA, AND T-CONDYLAR FRACTURES

JAMES H. BEATY  
JAMES R. KASSER

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- Fractures Involving the Lateral Condylar Physis
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- Percutaneous Pins
- Open Reduction
- Fractures of the Capitellum
- Fractures Involving the Medial Condylar Physis
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- Fractures Involving the Entire Distal Humeral Physis
- Apophyseal Injuries of the Distal Humerus
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### PHYSEAL FRACTURES

All the physes of the distal humerus are vulnerable to injury, each with a distinct fracture pattern. This vulnerability of the various physes to injury is altered by two major factors: age and mechanism of injury. Next to those of the distal radius, injuries to the distal humeral physes are the most common physeal injuries. The peak age incidences of these physeal injuries vary considerably. In general, the physes of the major long bones are most vulnerable to fracture just before puberty, when the perichondral ring is weakest (1,11). Fractures involving the medial epicondylar apophysis are most common in preadolescents, with the peak ages 11 to 15 years. This is probably because many avulsions of this apophysis are associated with posterolateral dislocations, which also are common in this age group. Fractures involving the lateral condylar physis occur early, with the average age around 6 years (2,4,7,9,10,11 and 12). Fractures involving the medial condylar physis are rare and occur most often in children 8 to 12 years of age (3,6,8). Fractures involving the total distal humeral physis may occur in neonates or within the first 2 to 3 years of life (5).

The specific fracture patterns, incidence, and mechanism of injury are discussed in detail in the following sections dealing with these specific fractures.

#### Fractures Involving the Lateral Condylar Physis

##### *Incidence and Outcome*

Fractures involving the lateral condylar region in the immature skeleton either cross the physis or follow it for a short distance into the trochlea. Fractures of the lateral condylar physis constitute 16.9% of fractures of the distal humerus.

Fractures of the lateral condylar physis are only occasionally associated with injuries outside the elbow region. Within the elbow region, the associated injuries that can occur with this fracture include dislocation of the elbow (which may be a result of the injury to the lateral condylar physis rather than a separate injury), fractures of the radial head, and fractures of the olecranon, which are often greenstick in nature. True acute fractures involving only the anatomic capitellum are rare in the immature skeleton.

The diagnosis of lateral condylar physeal injuries may be less obvious both clinically and radiographically than that of supracondylar fractures, especially if the fracture is minimally displaced. The incidence of a functional loss of range of motion in the elbow is much greater with fractures of the lateral condylar physis because the fracture line often extends into the articular surface. A difficult supracondylar fracture with cubitus varus, barring the immediate neurovascular complications, is likely to result in a surgically correctable cosmetic deformity with an essentially normal range of motion in the elbow. A poorly treated lateral condylar physeal injury, however, is likely to result in a significant loss of range of motion that is not as responsive to surgical correction. The complications of supracondylar fractures are usually evident in the immediate postinjury period. The poor outcome of a lateral condylar physeal fracture may not be obvious until months or even years later. Ippolito et al. (54) evaluated 49 individuals with humeral condylar fractures 18 to 45 years after the injury. Twenty fractures with displacement of 2 to 10 mm with no tilting of the osteochondral fragment had been treated without reduction and 16 fractures with marked displacement and tilting of the fragment had been treated surgically; all 36 had good results. All 13 patients treated operatively or nonoperatively for old, displaced fractures had poor results. Nonunion developed in 4 and osteonecrosis in 6. Arthrosis of the elbow was found in fractures complicated by osteonecrosis and nonunion, and in old fractures when the humeral condyle was resected, but it was not observed in uncomplicated fractures.

##### *Pathology*

## Early Descriptions

Milch (66,67) defined the fracture that exited through the trochleocapitellar groove as a type I (Milch type I). The type II (Milch type II) exits through the trochlea. Cotton (29) around the same time described more of the details of the various subluxations of both the fragment and elbow joint that occurred with this type of fracture. He noted that because the fragment usually was still attached to the proximal radius, both the radius and ulna were subluxed. The most common displacement was "outward and backward"; "inward and forward" displacement was rare. Cotton also noted that the main pathology was associated with the rotation of the condylar fragment. He observed that this fracture often resulted in limited extension, had some local lateral outgrowth at the fracture site, and rarely resulted in axial deviation of the elbow unless there was a resultant nonunion. Little has been added to his description of the pathology of this lesion by more recent investigators.

## Fracture Anatomy and Classification

Classification of lateral condylar physeal fractures can be described by either the anatomic location of the fracture line or the stage of displacement. An understanding of both of these classifications is essential in the management of these fractures.

**Anatomic Location.** Salter and Harris classified lateral condylar physeal injuries as type IV injuries in their classification of physeal fractures (83). A true Salter-Harris type IV injury through the ossific nucleus of the lateral condyle is rare. Although lateral condylar fractures are similar to Salter-Harris type II and IV fractures, treatment guidelines follow those of a type IV injury: open reduction and internal fixation of displaced intraarticular fractures, with the potential for mild growth disturbance of the distal humeral physis. There is no contact between the ossification center of the epiphysis and the exposed bone in the metaphyseal fragment.

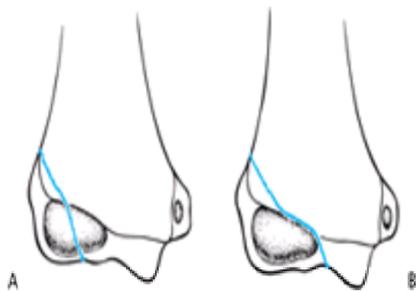
The Milch classification is based on the location of the fracture line through the epiphysis of the distal humerus.

**Milch Type I.** In this type, the fracture line originates in the metaphysis, crosses the physis more or less obliquely, and finally traverses the ossification center of the lateral condylar epiphysis to exit in the area of the capitulotrochlear groove (Fig. 15-1). There can be contact between the bony ossification center of the epiphysis and the bony metaphysis, leading to growth arrest due to an osseous bridge, especially in very young children. Fortunately, less than 20% of the growth of the humerus occurs through the distal humeral physis.



**FIGURE 15-1. A:** Injury film of a 7-year-old with an undisplaced fracture of the lateral condyle (small arrows). Attention was drawn to the location of the fracture because of extensive soft tissue swelling on the lateral aspect (white arrows). **B:** Because of the extensive soft tissue injury, there was little intrinsic stability, allowing the fracture to become displaced at 7 days (arrows).

**Milch Type II.** This more common fracture line originates in the posterolateral metaphysis, where there is a fragment of variable size (Fig. 15-2). The fracture then usually courses within the physis down to the depths of the trochlea. The fracture line does not traverse the lateral condylar epiphysis or ossification center. The ossification center of the lateral condyle extends to the lateral crista of the trochlea. Thus, the terminal portion of the fracture line courses through the physeal cartilage that lies between the ossification centers of the lateral condyle and the medial condyle. In the age group in which most of these fractures occur, there is little ossification of the medial crista of the trochlea.



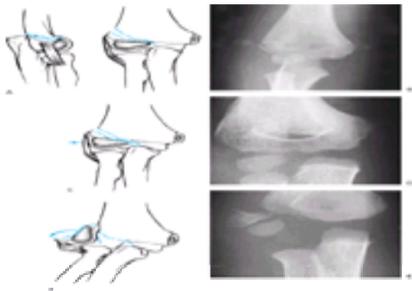
**FIGURE 15-2.** Physeal fractures of the lateral condyle. **A:** Physeal injury (Milch type II) through the nonossified trochlea. **B:** Physeal injury (Milch type I) through the ossific nucleus of the lateral condyle. (Redrawn from Milch HE. Fractures and fracture = ndislocations of the humeral condyles. *J Trauma* 1964;4:59-607; with permission.)

Because the fracture line starts in the metaphysis and then courses along the physeal cartilage, it has some of the characteristics of both type II and IV injuries according to the Salter-Harris classification. This fracture classification is debatable, because the fracture exits the joint in the not-yet-ossified cartilage of the trochlea.

Mirsky, Karas, and Weiner (71) compared intraoperative findings to preoperative radiographic classification in 25 displaced fractures of the lateral condyle and found that in 13 (52%) the Milch classification did not correlate with intraoperative findings. Eight of 17 fractures (47%) classified preoperatively as Milch type I fractures were unstable, and 5 of 8 fractures classified preoperatively as Milch type II fractures were extraarticular, extending across the distal humeral physis medially. Mirsky et al. identified three distinct fracture patterns: 9 fractures exited the distal humeral epiphysis just medial to the capitellum, 11 exited through the trochlear epiphysis, and 5 extended across the physis medially. No fracture appeared to traverse the ossified portion of the capitellum (Milch type I).

## Stage of Displacement

Displacement has been described as occurring in three stages (Fig. 15-3) (55,61,99). In the first stage, the fracture is relatively undisplaced and the articular surface is intact (Fig. 15-3A and Fig. 15-3B). Because the trochlea is intact, there is no lateral shift of the olecranon.



**FIGURE 15-3.** Stages of displacement. **A and B:** Stage I displacement—articular surface intact. **C and D:** Stage II displacement—articular surface disrupted. **E and F:** Stage III displacement—fragment rotated. (A, C, and E: Reprinted from Jakob R, Fowles JV, Rang M, et al. Observations concerning fractures of the lateral humeral condyle in children. *J Bone Joint Surg [Br]* 1975;57:430-436; with permission.)

In the second stage, the fracture extends completely through the articular surface ([Fig. 15-3C](#) and [Fig. 15-3D](#)). This allows the proximal fragment to become more displaced and can allow lateral displacement of the olecranon. In the third stage, the condylar fragment is rotated and totally displaced laterally and proximally, which allows translocation of both the olecranon and the radial head ([Fig. 15-3E](#) and [Fig. 15-3F](#)).

Badelon et al. ([14](#)) modified the description of stage I displacement to include fractures with less than 2 mm of displacement seen on the anteroposterior or lateral radiograph only or seen on both views.

### Soft Tissue Injuries

The fracture line usually begins in the posterolateral metaphysis, with a soft tissue tear in the area between the origins of the extensor carpi radialis longus and the brachioradialis muscle. The extensor carpi radialis longus and brevis muscles remain attached to the free distal fragment, along with the lateral collateral ligaments of the elbow. If there is much displacement, both the anterior and posterior aspects of the elbow capsule usually are torn.

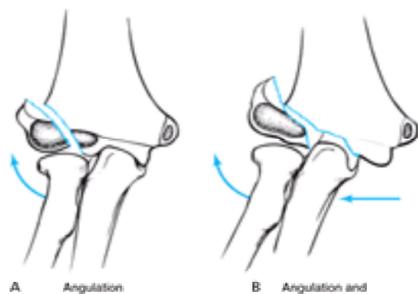
This soft tissue injury, however, is usually localized to the lateral side and may help identify a minimally displaced fracture. More extensive soft tissue swelling at the fracture site may indicate more severe soft tissue injury, which may be an indication that the fracture is prone to late displacement.

### Displacement of the Fracture and Elbow Joint

The degree of displacement varies according to the magnitude of the force applied and whether or not the cartilaginous hinge of the articular surface remains intact. If the articular surface is intact, the resultant displacement of the condylar fragment is simply a lateral tilt hinging on the intact medial articular surface. If the fracture is complete, the fragment can be rotated and displaced varying degrees; in the most severe fractures almost the full 180° so that the lateral condylar articular surface opposes the denuded metaphyseal fracture surface. Wilson ([101](#)) showed that in addition to this coronal rotation of the distal fragment, rotation also can occur in the horizontal plane. The lateral margin is carried posteriorly, and the medial portion of the distal fragment rotates anteriorly.

### Pattern Determines Stability

Because the usual fracture line disrupts the lateral crista of the trochlea (Milch type II), the elbow joint is unstable, creating the possibility of posterolateral subluxation of the proximal radius and ulna. Thus, the forearm rotates along the coronal plane into valgus, and there also may be lateral translocation of the lateral condyle with the radius and ulna ([Fig. 15-4](#)). This concept of lateral translocation is important in the late reconstruction of untreated fractures.



**FIGURE 15-4.** Angular deformities. **A:** Milch type I fractures tend only to angulate. **B:** Milch type II fractures are unstable with lateral translocation in addition to angulation. (Redrawn from Milch HE. Fractures and fracture-dislocations of the humeral condyles. *J Trauma* 1964;4:592-607; with permission.)

In Milch type I physeal fractures, where the fracture line traverses the lateral condylar epiphysis, the elbow remains reasonably stable because the trochlea remains intact. Total coronal rotation of the condylar fragment can occur with this injury. The axial deformity that results is pure valgus without translocation ([Fig. 15-4](#)).

This posterolateral elbow instability with the lateral condylar physeal injury has led to the mistaken concept that this injury is associated with a primary dislocation of the elbow ([26](#)). Such rarely is the case. The posterolateral instability of the elbow usually is a result of the injury, not a cause of it ([80](#)).

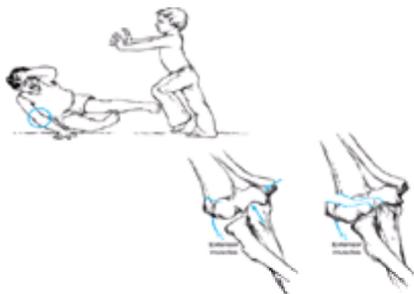
### Mechanism of Injury

As Heyl ([49](#)) has stated, the local biomechanics of the distal humerus must be different in children because of the rarity of this injury in adults. Two mechanisms have been suggested: “push off” or “pull off.”

The pull off or avulsion theory has more advocates ([13,15,55,79,95](#)). In early studies ([13,95](#)) this injury was consistently produced in young cadavers by adducting the forearm with the elbow extended and the forearm supinated. The results of these studies were confirmed by the work of Jakob and Fowles ([55](#)).

Some of Stimson's ([95](#)) work strengthens the push off theory. In his cadaver studies, he produced the injury by applying a sharp blow to the palm when the elbow was flexed ([15,29,34,42](#)). Others who support the push off theory have speculated that because the forearm goes into valgus when extended, the radial head can thus push off the lateral condyle. Some researchers have even proposed that it can be a result of a direct blow to the olecranon ([24,34](#)).

It is likely that both mechanisms can produce this injury. The more common type of fracture, which extends to the apex of the trochlea, is probably a result of avulsion forces on the condyle, with the sharp articular surface of the olecranon serving to direct the force along the physeal line into the trochlea ([Fig. 15-5](#)). When a child falls forward on the palm with the elbow flexed, the radial head is forced against the capitellum and may cause the less common Milch type I physeal fracture that courses through the capitulotrochlear notch.



**FIGURE 15-5.** The most common mechanism of injury is believed to occur when the elbow is forced into varus (*bottom left*), which, along with the extensor muscles and lateral collateral ligaments, applies an avulsion force to the lateral condyle. When the fracture line extends to the trochlear notch (*bottom right*), the elbow becomes unstable.

### Signs and Symptoms

Compared with the marked distortion of the elbow that occurs with displaced supracondylar fractures, little distortion of the elbow, other than that produced by the fracture hematoma, may be present with lateral condylar fractures. The key to the clinical evaluation of this fracture is the location of soft tissue swelling concentrated over the lateral aspect of the distal humerus. Stage I displacement may produce only local tenderness at the condylar fracture site, which may be increased by forcibly flexing the wrist (52). Stage II or III displacement may result in some local crepitus with motion of the lateral condylar fragment. The benign appearance of the elbow with some stage I and II displacements may account for the delay of parents seeking treatment for a child with a minimally displaced fracture.

### Radiographic Findings

The radiographic appearance varies according to the anatomic location of the fracture line and the stage of displacement. In the anteroposterior view, the metaphyseal flake may be small and seemingly minimally displaced. The degree of displacement can often be better appreciated on the true lateral view. In determining whether the articular hinge is intact (i.e., stage I vs. stage II), the relationship of the proximal ulna to the distal humerus is evaluated for the presence of lateral translocation. Oblique views are especially helpful in patients in whom a stage I displacement is suspected.

In a prospective study of 112 children with nondisplaced and minimally displaced fractures of the lateral condyle, Finnbogason et al. (36) identified three groups of fractures: stable fractures, fractures with an undefinable risk, and fractures with a high risk of later displacement. Stable fractures had no gap or a small gap and did not extend all the way to the epiphyseal cartilage; most of these 65 fractures were in younger children and none had later displacement. Fractures with undefinable risk of displacement were of the same type as stable fractures but the fracture could be clearly observed extending all the way to the epiphyseal cartilage; displacement occurred in 6 (17%) of these 35 fractures. High-risk fractures had a gap that was as wide or almost as wide laterally as medially; displacement occurred in 5 of 12 (42%) of these fractures.

Kamegaya et al. (59) reported that magnetic resonance imaging (MRI) evaluation of 12 minimally displaced (<2 mm on radiography) lateral condylar fractures identified 5 fractures that crossed the physis into the joint space and were unstable fractures. One of 5 fractures with 1-mm displacement was unstable, and 4 of 7 with 2-mm displacement were unstable. They suggested that MRI evaluation might prevent late displacement or delayed union by identifying those minimally displaced fractures that required percutaneous pin fixation rather than cast immobilization.

A major diagnostic difficulty lies in differentiating this fracture from a fracture of the entire distal humeral physis. In a young child in whom the condyle is unossified, an arthrogram or MRI may be helpful (Fig. 15-6 and Fig. 15-7). Potter (76) recommended MRI with thin (1.5–2 mm) sections and appropriate pulse sequencing to provide differential contrast between subchondral bone, cartilage, and joint fluid.



**FIGURE 15-6.** Unossified lateral condyle. **A:** Anteroposterior view. A small ossific nucleus can barely be seen (*arrow*) in the swollen lateral soft tissues. **B:** An arthrogram shows the defect left by the displaced lateral condyle (*open arrow*). The displaced condyle is outlined in the soft tissues (*solid arrow*).



**FIGURE 15-7.** Arthrogram of stage I fracture of the lateral condyle (*large arrows*). Articular surface is intact with no displacement (*small arrows*).

In fractures of the entire distal humeral physis, the proximal radius and ulna usually are displaced posteromedially (Fig. 15-8A). The relationship of the lateral condylar ossification center to the proximal radius remains intact. In true fractures involving only the lateral condylar physis, the relationship of the condylar ossification center to the proximal radius is lost (Fig. 15-8B). In addition, any displacement of the proximal radius and ulna is more likely to be lateral because of the loss of stability provided by the lateral crista of the distal humerus.



**FIGURE 15-8. A:** Total distal humeral physal fracture in a 2-year-old. The lateral condyle (*closed arrow*) has remained in line with the proximal radius. The proximal radius, ulna, and lateral condyle have all shifted medially (*open arrow*). **B:** Displaced fracture of the lateral condyle in a 2-year-old. The relationship of the lateral condyle (*closed arrow*) to the proximal radius is lost. Both the proximal radius and ulna (*open arrow*) have shifted slightly laterally.

### Methods of Treatment

Fractures involving the lateral condylar physis can be treated with simple immobilization alone, closed reduction and percutaneous pinning, or open surgical reduction.

### Fractures Requiring Immobilization Only

Minimally displaced fractures are stable and have considerable intrinsic soft tissue attachments that prevent displacement of the distal fragment. About 40% of lateral condylar physal fractures are sufficiently undisplaced that they can be treated by simple immobilization without surgical intervention ([55,63,84](#)). If the fracture line is barely perceptible on the original radiograph (stage I displacement), the degree of displacement usually is minimal and the chance for subsequent displacement is low ([Fig. 15-9](#)).



**FIGURE 15-9.** Stage I fracture of the lateral condyle. **A:** Anteroposterior radiograph shows 1-mm fracture line. **B:** Lateral view shows 2 mm of displacement with posterolateral Thurston-Holland fragment. **C and D:** Anteroposterior and lateral radiographs 4 weeks later show healed fracture with callus formation.

Badelon et al. ([14](#)) determined in their long-term study of fractures treated nonoperatively that only fractures with type I displacement (i.e., the fracture line is seen on only one radiographic view) can be safely treated nonoperatively. In their experience, any fracture with displacement, even of less than 2 mm, can displace later in the cast or splint. Beatty and Wood ([18](#)), in a review of 57 fractures of the lateral condyle, found that 2 of 24 fractures with stage I displacement displaced late. Bast, Hoffer, and Aval ([16](#)) reported a union rate of 98% after nonoperative treatment of 95 nondisplaced or minimally displaced fractures of the lateral humeral condyle. Their criteria for nonoperative treatment were acute fracture (less than 24 hours at initial evaluation) and displacement of less than 2 mm in three radiographic planes (anteroposterior, lateral, and internal oblique). Two fractures that displaced 6 and 9 days after closed reduction required open reduction and internal fixation before they united without complications.

### Late Displacement

Careful clinical examination is important in predicting which fractures will displace later. The potential to displace often depends more on the degree of associated soft tissue injury and whether the articular cartilage of the trochlea is intact, rather than on the amount of initial displacement. Considerable soft tissue swelling on the lateral aspect of the distal humerus, which can be appreciated both clinically and on radiographs, should alert the physician to the fact that the fracture may be unstable and has the potential to displace. If crepitus between the fragments is detected with motion of the forearm or elbow, significant loss of soft tissue attachments and a potentially unstable fracture should be suspected ([17](#)).

### Critical Value

Undisplaced fractures usually can be treated with simple immobilization with good results. Speed and Macey ([93](#)) reported uniformly excellent results both anatomically and functionally in patients with undisplaced fractures, none of whom had any abnormalities of growth or premature physal fusion. Simple immobilization of nondisplaced or minimally displaced (<2 mm) fractures in a sling, collar and cuff, or posterior splint appears adequate ([93,99,103](#)). Close follow-up and repeat radiographs to detect any late displacement are mandatory if this method is used.

### Closed Reduction and Percutaneous Pinning

Several techniques have been described for initial reduction, with the recommended elbow position ranging from hyperflexion to full extension; however, it appears from clinical experience ([425](#)) and experimental studies that reduction is best achieved with the forearm supinated and the elbow extended. Placing a varus stress on the extended elbow allows further room for manipulation of the fragment. Unfortunately, it is difficult to maintain reduction of a displaced lateral condylar fracture with closed techniques, and closed reduction is not recommended for treatment of type III displaced lateral condylar fractures. It has become popular to reduce minimally displaced fractures initially by closed manipulation, and then stabilize them by percutaneously placing pins across the fracture. In some fractures of the lateral condylar physis, simple immobilization is all that is necessary. In those with moderate displacement, confirmation of fracture stability by stress testing and arthrography may precede percutaneous pin fixation. Mintzer et al. ([69](#)) reported good results after percutaneous pin fixation of 12 lateral condylar fractures with displacement of more than 2 mm. They believed this method is appropriate for selected fractures with more than 2 mm of displacement and an arthrographically demonstrated congruent joint surface. If a satisfactory reduction cannot be obtained, then reduction should be achieved and maintained by open reduction and internal fixation.

### Fractures Requiring Open Reduction

Because of the high incidence of poor functional and cosmetic results with closed reduction methods, open reduction has become the most widely advocated method for unstable fractures with stage II displacement and fractures with stage III displacement ([15,19,20,21,22,25,26,30,33,34,47,53,55,56,63,64,77,79,86,87,89,90,96,99,100,102,103](#)). About 60% of all fractures involving the lateral condylar physis require reduction and internal fixation ([55,63,84](#)).

There is almost uniform agreement about the need for open reduction of displaced fractures of the lateral condylar physis. Most investigators recommend fixation with smooth Kirschner wires in children or screws in adolescents nearing skeletal maturity.

### **Pin and Screw Fixation**

Smooth pins are the most frequently used method of fixation of the fragment ([18,23,34,40,42,53,55,63,79,84,90,93,99,102](#) and [103](#)). Blount et al. ([19](#)) believed that at least two pins were necessary to prevent rotation. The passage of a smooth wire through the physis does not result in any growth disturbance ([84,93](#)). This is of note because only 20% of the growth of the humerus occurs through the distal humeral physis. It also appears that the wires can be placed either parallel or crossed in the distal fragments.

The ideal place for the pins is in the metaphyseal fragment. They should cross at the lateral aspect of the metaphysis and diverge as much as possible to enhance the stability of fixation. If there is only a small metaphyseal fragment, the pins can be placed across the physis without concern.

When adequate reduction and internal fixation are performed early (i.e., within the first few days of the injury), the results are uniformly good. The key, however, is to be sure that the reduction is adequate. Hardacre et al. ([47](#)) found that poor results with open reduction occurred when the reduction was incomplete. Surgery alone does not ensure a good result unless the reduction is nearly anatomic and the fixation is secure.

Early surgical intervention is essential, because organization of the clot with early fibrin development makes it difficult to achieve a reduction without extensive soft tissue dissection in fractures treated late. The pins can be buried or left protruding through the skin with no increase in the incidence of infection. Leaving pins buried requires a second operative procedure, even though it usually can be accomplished with a local anesthetic. The fracture is usually sufficiently stable to allow pin removal by 3 to 4 weeks and to allow the patient to begin protected active range of motion of the elbow at 2 to 3 weeks.

Screw fixation has been used less frequently in children, although it was recommended by Jeffrey in 1958. Connor and Smith ([56](#)) used fully threaded Glasgow screws in 35 patients and found limitations of 5 to 25 degrees of extension in 9; in 5 patients the screw backed out and was found loose. Sharma et al. ([85](#)) reported painless, full range of elbow motion in 36 of 37 children who had displaced (> 2 mm in any direction) lateral condylar fractures fixed with partially threaded 4-mm AO cancellous screws. One patient had delayed union, with loss of 10 degrees of elbow motion.

## **AUTHORS' PREFERRED METHOD OF TREATMENT**

### **Immobilization**

If the fracture is minimally displaced on the radiograph (i.e., the metaphyseal fragment is <2 mm from the proximal fragment on anteroposterior and lateral views) and the clinical signs also indicate there is reasonable soft tissue integrity, we simply immobilize the elbow in a posterior splint with the forearm in neutral rotation and the elbow flexed 90 degrees ([Fig. 15-9](#)). Radiographs are taken within the first 3 to 5 days after the fracture with the splint removed and the elbow comfortably extended. If there is no displacement, the radiographs are repeated in another 3 to 5 days. If the radiographs again show no displacement, then a long arm cast is applied and is worn for about 3 weeks, or until fracture union is apparent.

In some fractures with more than the allowable 2 mm of displacement (type II injury), the fracture pattern is such that the articular cartilage appears intact. If there is any question about the stability at the time of the fracture, the extremity should be examined with the patient under general anesthesia. Gentle varus stress views with the forearm supinated and the elbow extended should be taken to determine if the fracture displaces significantly. Arthrography may be performed to determine the stability of the nonossified articular cartilage of the trochlea.

### **Percutaneous Pins**

For fractures with stage II displacement (2–4 mm), varus stress views should be obtained and arthrography performed with the patient under anesthesia. If the fracture is stable, percutaneous pinning is indicated ([Fig. 15-10](#)).



**FIGURE 15-10.** Stage II fracture of the lateral condyle. **A:** Anteroposterior radiograph shows 4 mm of displacement of the metaphyseal segment; however, the fracture was stable by stress examination and arthrography. **B:** Four weeks after percutaneous pinning, the fracture is healed.

### **Open Reduction**

If the fracture is grossly unstable, open reduction and internal fixation should be performed. We prefer open reduction and internal fixation of all fractures with stage III displacement. It is important to perform the open reduction as soon as possible after the injury. The standard lateral Kocher approach provides sufficient exposure of the fragment. Often a tear in the aponeurosis of the brachioradialis muscle laterally leads directly to the fracture site. Extreme care must be taken to avoid dissecting near the posterior portion of the fragment, because this is the entrance of the only blood vessel supplying the lateral condylar epiphysis.

A posterolateral approach was recommended by Mohan, Hunter, and Colton ([71](#)) because of the excellent exposure it provides with minimal dissection. Another suggested advantage is the improved cosmetic results by more posterior placement of the surgical scar. Mohan et al. reported no complications in 20 patients in whom this approach was used.

The quality of the reduction is determined by evaluating the fracture line along the anterior aspect of the articular surface. This can usually be determined either by direct vision or by digital palpation. We prefer to use smooth Kirschner wires that cross just medial to the condylar fragment to maintain the reduction. The wires penetrate the skin through a separate stab incision posterior to the main incision. A long arm cast is applied with the elbow flexed 90 degrees and the forearm in neutral or slight pronation. The cast and pins are removed in 3 weeks if there is adequate healing on radiographs. Early active motion is started at that time. If necessary, pin removal can be delayed 1 to 2 weeks to allow further healing in older children.

### **Technique of Open Reduction and Internal Fixation of Fractures of the Lateral Humeral Condyle**

The elbow is exposed through a 5- to 6-cm lateral approach, placing two thirds of the incision above the joint and one third distal ([Fig. 15-11](#)). In the interval between the brachioradialis and the triceps, the dissection is carried down to the lateral humeral condyle. The anterior surfaces of the joint are exposed by separating the fibers of the common extensor muscle mass. Soft tissue detachment is limited to only that necessary to expose the fragment, the fracture, and the joint; the posterior soft tissues are left intact. The anterior joint surface is exposed by retracting the antecubital structures. The trochlea and the more medial point of entry of the condylar

fracture are inspected. The displacement and the size of the fragment are always greater than is apparent on the radiographs because much of the fragment is cartilaginous. The fragment usually is rotated as well as displaced. The joint is irrigated to remove blood clots and debris, the articular surface and the metaphyseal fragment are reduced accurately, and the reduction is confirmed by observing the articular surface, particularly at the trochlear ridge. The position is held with a small tenaculum, bone holder, or towel clip. When a large metaphyseal fragment is present, two smooth Kirschner wires are inserted across it into the medial portion of the metaphysis. When the epiphyseal portion is small, as is more common, two smooth Kirschner wires are inserted through the condyle, across the physis, and into the humeral metaphysis, penetrating the medial cortex of the humerus. The wires are directed 45 to 60 degrees; the reduction and the position of the internal fixation are checked by anteroposterior and lateral radiographs before closing the wound. The ends of the wires are cut off beneath the skin but are left long enough to allow easy removal. The arm is placed in a posterior plaster splint with the elbow flexed 70 to 90 degrees.



**FIGURE 15-11.** Lateral approach for open reduction and internal fixation of a lateral humeral condylar fracture of the left elbow. The approach is made through the brachioradialis-triceps interval, an anterior retractor is used to expose the joint surfaces, and the fracture is reduced and pinned percutaneously posterior to the incision.

The splint is worn for 2 to 3 weeks after surgery. The pins can be removed at 3 weeks if union is progressing. Gentle active motion of the elbow is then usually resumed and continued until full range of motion returns.

#### **Delayed Union and Nonunion**

If sophisticated surgical treatment is unavailable, these fractures may go untreated or unrecognized for a prolonged period. Even in modern medical settings, elbow injuries may be treated as “sprains,” and the diagnosis of a displaced lateral condylar fracture is not made. Thus, patients often present months or even years later with a nonunited or malunited fracture fragment.

#### **Delayed Union**

Delayed union, in contrast to nonunion or malunion, occurs in a fracture in which the fracture fragments are in satisfactory position but union of the lateral condylar fragment to the metaphysis is delayed. Various reasons have been suggested for delayed union of lateral condylar fractures. Flynn and Richards ( 37) speculated that it was caused by poor circulation to the metaphyseal fragment. Hardacre and colleagues ( 47) believed that bathing of the fracture site by articular fluid inhibited fibrin formation and subsequent callus formation. Probably a combination of these two factors, in addition to the constant tension forces exerted by the muscle arising from the condylar fragment, is responsible for delayed union.

This complication is most common in patients treated nonoperatively. The aggressiveness of treatment is determined by the symptoms and clinical examination. The fragment usually is stable during clinical examination, the elbow is nontender, and the range of elbow motion increases progressively. On radiography, the position of the fragment remains unchanged. With time, these fractures usually heal ( Fig. 15-12). Lateral spur formation or cubitus varus is relatively common with these fractures. The need for further treatment depends on the presence of significant symptoms or further displacement that may disrupt the joint surface and cause functional impairment. If neither of these conditions is present, the radiographic persistence of the fracture line requires only follow-up observation. If there is any question as to the integrity of the joint surface, an arthrogram may help determine any loss of continuity and the need for surgical treatment as a nonunion rather than a simple delayed union.



**FIGURE 15-12.** Delayed union and cubitus varus. **A:** Stage III lateral condylar fracture in a 7-year-old boy was treated in a cast. **B:** Seven months later, delayed union with malunion of the fracture and cubitus varus deformity were present.

Flynn (37,38 and 39) recommended long-term immobilization for minimally displaced fractures with delayed union. He found that 70% of minimally displaced fractures had united by 12 weeks. Jeffrey (56) recommended screw fixation with bone grafting. Hardacre et al. (47), however, found that minimally displaced fractures with delayed union ultimately united if there was no significant displacement of the condylar fragment.

Controversy exists as to whether elbow function can be improved by a late open reduction and internal fixation of the fracture fragment. Delayed open reduction has been complicated by osteonecrosis and further loss of elbow motion. Speed and Macey ( 93) were among the first investigators to question whether patients treated with late surgery did better than those not treated. In patients with malunion who were treated late, they found a high incidence of poor results due to “epiphyseal changes” that probably represented osteonecrosis. There have been many subsequent reports of osteonecrosis occurring after late open reduction. The high incidence of osteonecrosis of the fragment is believed to be due to the extensive soft tissue dissection necessary to replace the fragment. Böhler ( 21), on the other hand, had good results in his patients with delayed treatment. He avoided extensive soft tissue dissection by approaching the fragment transarticularly after performing an osteotomy of the olecranon.

The key to preventing osteonecrosis is to recognize the course of the blood supply to the lateral condyle. Only a small portion of the condyle is extraarticular. In his studies, Haraldsson (46) found that the vessels that supply the lateral condylar epiphysis penetrate the condyle in a small posterior nonarticular area ( Fig. 15-13).



**FIGURE 15-13.** Asymptomatic nonunion of a lateral condyle in a 19-year-old military recruit. Because the patient had a completely normal and asymptomatic range of motion in his nondominant extremity, operative stabilization was not thought to be necessary.

Jakob and Fowles (55) reported that patients treated later than 3 weeks after the fracture did no better than those who received no treatment at all. They found that early callus and fibrous tissue made it extremely difficult to obtain a satisfactory open reduction without performing considerable soft tissue dissection. All their patients treated after 3 weeks lost range of motion, the average being at least 34 degrees, and osteonecrosis, premature physal closure, and valgus deformity were common. In patients who received no treatment, valgus deformity due to nonunion and malunion was frequent, but no osteonecrosis of the lateral condylar epiphysis occurred. They recommended that no open reduction be performed for fractures older than 3 weeks, but that early ulnar nerve transposition be performed to eliminate the possibility of late ulnar nerve symptoms that occur with cubitus valgus deformity after nonunion. Dhillon et al. (32) and Zionts and Stolz (104) also reported that osteonecrosis was frequent after late open reduction and recommended no treatment for these fractures.

### Nonunion

In 1932, Cooper (28) drew attention to the development of nonunion after lateral humeral condylar fractures when he described his findings in two cadaver specimens. He pointed out that there was absolutely no bony continuity between the distal humerus and the condylar fragment. True nonunion with significant deformity is rare because it is usually the result of nontreatment of a displaced fracture of the lateral condylar physis (19,64,84).

True nonunion occurs in patients with progressive displacement of the fragment. The mobile fragment can be palpated, or the patient has weakness or pain in the elbow. According to the criteria of Flynn et al. (39), if the fracture has not united by 12 weeks, it is classified as a nonunion.

Nonunion can occur with or without angular deformity. Many patients with nonunions and minimal displacement of the fragment have no angulation and remain relatively asymptomatic for normal activities (Fig. 15-13). Others have weakness or symptoms when the arm is used for high-performance activities. Because they are not significantly displaced, these fractures can often be stabilized with minimal extraarticular dissection using a combination of screw fixation and a laterally placed bone graft.

Nonunion with subsequent displacement of the fragment is more common after unstable fractures with stage II and III displacement. If the fragment becomes free, it tends to migrate proximally with a subsequent valgus deformity of the elbow. Nonunion can lead to a cubitus valgus deformity, which in turn is associated with the development of a tardy ulnar nerve palsy.

Nonunion seems to occur when the distal fragment is displaced enough to allow the cartilaginous articular surface of the condylar fragment to oppose the bony surface of the humeral metaphysis. In such a situation, union is impossible.

Patients with established nonunion lose some range of motion but still function well. Smith (91) reported an 85-year follow-up of a patient with a nonunion, cubitus valgus, and mild ulnar neuropathy. Despite these complications, the patient was a musician, playing the French horn for over 35 years. Kalenak (58) reported a similar 50-year follow-up of nonunion of the lateral condyle in a 74-year-old laborer who had minimal symptoms. Other than the secondary effects of a tardy ulnar nerve palsy, the functional difficulties caused by an established nonunion are not severe (55,72,79). This has led many investigators to recommend no treatment at all or no treatment until the patient has achieved full skeletal growth for an established nonunion elbow deformity (18,20,47,55). Flynn and Richards (37), however, reported successful treatment of nonunion 9 months to 3 years after fracture and strongly advised early surgery for established nonunion when the condylar fragment is in "good position" in a child with open physes.

The most common sequela of nonunion with displacement is the development of a progressive cubitus valgus deformity. The fragment migrates both proximally and laterally, giving not only an angular deformity but also lateral translocation of the proximal radius and ulna (Fig. 15-14). Milch (67) noted that lateral translocation is not as likely to develop in the more lateral type of these fractures (Milch type I) because the lateral crista of the trochlea is intact (Fig. 15-15).

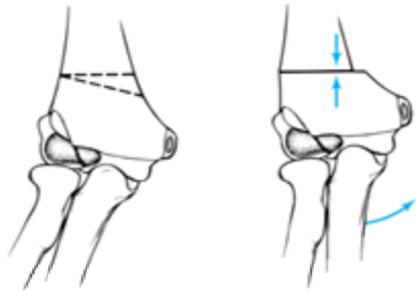


**FIGURE 15-14.** A: A 10-year-old boy with cubitus valgus resulting from a fracture of the lateral condylar physis with nonunion. B: Nonunion with cubitus valgus. Radiograph showing both angulation and translocation secondary to nonunion of the condylar fragment.



**FIGURE 15-15.** Nonunion without translocation. Milch type I fracture pattern. Despite nonunion, elbow stability was maintained because the lateral crista of the trochlea had remained intact (arrow). Valgus angulation also developed.

Surgical treatment of the nonunion deformity of the lateral condylar fragment is difficult and requires correction of two problems. First, articular cartilage may be opposing the distal humeral metaphysis, and union seldom can be obtained without mobilization of the fragments and application of an internal compressive device. The second problem is correction of the angular deformity. In a Milch type I fracture pattern, correction of the angulation may require only a simple closing wedge osteotomy (Fig. 15-16). Correction of the angular deformity in a Milch type II fracture pattern may involve not only correction of the angulation, but also translocation of the distal fragment back to its original linear alignment. Achieving solid fixation with this type of osteotomy is difficult without using appropriate internal fixation. Often the deformity is corrected, but because of the extensive soft tissue dissection required, the patient loses range of elbow motion postoperatively. Jakob and Fowles (55) pointed out that even with correction of the deformity, tardy ulnar nerve paralysis can still develop because it is trapped in the cubital tunnel. They recommended simply transposing the ulnar nerve and not treating the deformity.



**FIGURE 15-16.** In the Milch type I fracture pattern, there is only an angular deformity that can be easily corrected with a closing wedge osteotomy. (Redrawn from Milch HE. Fractures and fracture-dislocations of the humeral condyles. *J Trauma* 1964;4:592-607; with permission.)

Shimada et al. (88) reported excellent or good results in 15 of 16 patients at an average follow-up of 11 years after osteosynthesis for nonunion of fractures of the lateral humeral condyle. The one patient with a poor result had evidence of osteonecrosis of the fragment. The average interval between injury and osteosynthesis was 5 years (range 5 months to 10 years). Presenting symptoms were elbow pain ( $n = 7$ ), apprehension ( $n = 9$ ), cubitus valgus deformity ( $n = 6$ ), limitation of motion ( $n = 3$ ), and dysfunction of the ulnar nerve ( $n = 4$ ). Osseous union was obtained after the initial operation in 13 of the 16 patients. Of the 3 patients with persistent nonunions, 2 had union after a second osteosynthesis with bone-grafting and the other, who was asymptomatic, refused additional treatment. To prevent progression of cubitus valgus deformity and subsequent ulnar nerve dysfunction, Shimada et al. (88) recommended osteosynthesis for nonunion of lateral humeral condylar fractures in children because union is easily achieved, the range of motion is maintained, the function of the ulnar nerve usually returns, and remodeling of the articular surfaces can be expected. They noted that bone grafting is essential to bridge the defect, to obtain congruity of the joint, and to promote union; damage to the blood supply should be avoided to prevent osteonecrosis.

## AUTHORS' PREFERRED METHOD OF TREATMENT

We distinguish between fractures seen late (>7–14 days after injury) and established nonunions (usually from 3 months to several years after injury). If we believe that we can obtain fracture union without loss of elbow motion and avoid osteonecrosis of the lateral condyle, then we recommend surgery for selected patients.

Treatment of an established nonunion of a lateral humeral condylar fracture poses a difficult dilemma. If no treatment is rendered, a progressive cubitus valgus deformity occurs with growth. Patients usually are asymptomatic except for those with high-demand athletic or labor activities. A mild flexion contracture of the elbow is present, but the cubitus valgus deformity is more cosmetic than functional. The danger in this approach is failure to recognize and treat early a tardy ulnar nerve palsy. If surgery is performed for an established nonunion, the potential risks of osteonecrosis and loss of elbow motion must be carefully considered.

We believe the criteria outlined by Flynn et al. (37,39) are helpful in determining if surgical treatment is appropriate for an established nonunion:

1. A large metaphyseal fragment
2. Displacement of less than 1 cm from the joint surface
3. An open, viable lateral condylar physis

It also is helpful to distinguish between three distinct clinical situations. First, for an established nonunion with a large metaphyseal fragment, minimal migration, and an open lateral condylar physis, we recommend modified open reduction, screw fixation, and a lateral extraarticular iliac crest bone graft. This technique is markedly different from the surgical treatment of an acute lateral condylar fracture. The metaphyseal fragment of the lateral condyle and the distal humeral metaphysis are exposed, but no attempt is made to realign the articular surface. Intraarticular dissection should be avoided to help prevent any further loss of elbow motion. The metaphyseal fragments are debrided by gentle removal of any interposed fibrous tissue. The lateral condylar fragment usually can be moved distally a small distance. The metaphyseal fragments are firmly opposed, and a cancellous or cortical screw is used to fix the fragments with interfragmentary compression. Iliac crest bone graft may be placed between the metaphyseal fragments and laterally. The elbow is immobilized in 80 to 90 degrees of flexion for 3 to 4 weeks (Fig. 15-17).



**FIGURE 15-17.** A: Established nonunion with a large metaphyseal fragment. B and C: After fixation with a cancellous screw and bone grafting of the metaphyseal fragment.

Second, in patients with a nonunion who have cosmetic concerns but no functional complaints, treatment is similar to that for cubitus varus deformity after a supracondylar humeral fracture. If the patient and family desire, a supracondylar osteotomy may be performed (62). Rigid internal fixation should be used to allow early motion. Rarely in an adolescent or young adult with high functional demands and symptoms of instability, late osteosynthesis of the lateral condyle may be performed.

Third, patients with asymptomatic nonunion, cubitus valgus deformity, and symptomatic tardy ulnar nerve palsy should be treated with transposition of the ulnar nerve.

## Complications

If an adequate reduction is obtained promptly and maintained with solid fixation, results are uniformly good. In supracondylar fractures, an incomplete reduction may result in a cosmetic deformity, but functional results usually are good. In displaced fractures of the lateral condylar physis, a marginal reduction can result in both cosmetic deformities and functional loss of motion.

The complications that affect the outcome can be classified as either biologic or technical. Biologic problems occur as a result of the healing process, even if a perfect reduction is obtained. These problems include spur formation with pseudo-cubitus varus or a true cubitus varus. The technical problems arise usually from errors in management and result in nonunion or malunion with or without valgus angulation, and osteonecrosis. Other technical problems can arise from the injury itself, including neurologic injuries and myositis ossificans.

### Lateral Spur Formation

Lateral condylar spur formation is one of the most common deformities after a fracture involving the lateral condylar physis. Cotton ( 29) believed that it is caused by coronal rotation of the distal fragment, which tends to displace laterally the flap of periosteum associated with the distal fragment. This periosteum then produces new bone formation in the form of a spur. He believed that this spur formation produces no functional deformity. Maylahn and Fahey ( 63) found that this occurred in at least 28% of their patients. Wadsworth ( 99) agreed with Cotton that it was of no functional importance, and he also believed that the cosmetic effect is insignificant.

The spur occurs after both nonoperative and operative treatment. After nonoperative treatment, it results from the minimal displacement of the metaphyseal fragment and usually has a smooth outline. In patients with no real change in carrying angle, the lateral prominence of the spur may produce an appearance of mild cubitus varus (pseudovarus). In patients in whom a true cubitus varus develops, the presence of the lateral spur accentuates the varus alignment ( Fig. 15-18A and Fig. 15-18B). The spur that occurs after operative treatment has a more irregular outline and usually is the result of hypertrophic bone formation from extensive dissection at the time of open reduction and internal fixation ( Fig. 15-18C and Fig. 15-18D). When performing an open reduction, care should be taken to limit the aggressiveness of the dissection and to carefully replace the lateral periosteal flap of the metaphyseal fragment.

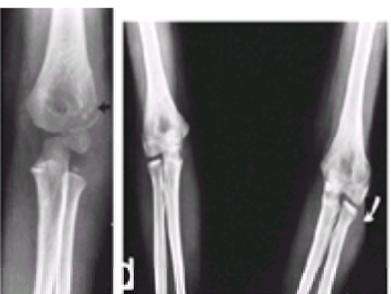


**FIGURE 15-18.** Spur formation. **A:** Follow-up radiograph of a boy whose lateral condyle was treated nonoperatively. The periosteal flap produced a spur on the lateral aspect of the metaphysis (arrow). This fracture healed with a mild varus angulation as well. **B:** Clinically, the spur accentuated the lateral prominence (arrow) of the elbow, which in turn accentuated the mild valgus angulation. (Reprinted from Wilkins KE. Residuals of elbow fractures. *Orthop Clin North Am* 1990;21:289-312; with permission.) **C:** Considerable soft tissue dissection was performed in the process of open reduction of this lateral condylar fracture. **D:** At 2 months postsurgery, there is a large irregular spur formation secondary to periosteal new bone formation from the extensive dissection.

Before treatment, the parents should be warned that either lateral overgrowth with mild cubitus varus or lateral spur may develop, regardless of the method of treatment. They should be told that this mild deformity is usually not of cosmetic or functional significance. If these problems are explained to the parents beforehand, they are less likely to be anxious and critical of the result should it be less than perfect.

### Cubitus Varus

Reviews of lateral condylar fractures demonstrate that a surprising number heal with some residual cubitus varus angulation ( 34,41,48,50,60,64,73,81,82,92,97). In some series, the incidence of cubitus varus is as high as 40% ( 41,92), and the deformity seems to be as frequent after operative treatment as after nonoperative treatment ( 50,81,92). The exact cause is not completely understood. In some instances, it is probably a combination of both an inadequate reduction and stimulation of growth of the lateral condylar physis from the fracture insult ( Fig. 15-19) ( 82,92).



**FIGURE 15-19.** True varus. **A:** The injury film with a minimally displaced fracture (arrow). This 5-year-old child was treated with simple immobilization until the fracture was healed. **B:** Five years later, the patient had a persistent cubitus varus (arrow) that remains clinically apparent. The carrying angle of the uninjured right elbow measures 5 degrees of valgus; the injured elbow has 10 degrees of varus. (Reprinted from Wilkins KE. Residuals of elbow trauma in children. *Orthop Clin North Am* 1990;21:289-312; with permission.)

Rarely is the cubitus varus deformity severe enough to cause concern or require further treatment. This is probably because it is a pure coronal varus angulation and does not have the horizontal anterior rotation of the lateral condyle along with the sagittal extension that makes the cubitus varus that occurs after supracondylar fractures such an unacceptable deformity. Some investigators have noted that children with cubitus varus deformities have pain, decreased range of motion, epicondylitis, and problems with sports such as sidearm pitching, swimming, judo, and pushups. Davids and colleagues ( 31) reported lateral condylar fractures in six children with preexisting cubitus varus deformities from previous elbow fractures, usually supracondylar humeral fractures. They concluded that posttraumatic cubitus varus deformity may predispose a child to subsequent lateral condylar fracture and should be viewed as more than just a cosmetic deformity. They recommended a two-stage correction of the deformity: anatomic reduction and internal fixation of the lateral condyle, followed by valgus supracondylar osteotomy of the distal humerus.

### Cubitus Valgus

Cubitus valgus is much less common after united lateral condylar fractures than cubitus varus. It rarely has been reported to result from premature epiphysiodesis of the lateral condylar physis ( 50,98). As with cubitus varus, it is usually minimal and rarely of clinical or functional significance. The more difficult type of cubitus valgus associated with nonunions was discussed in the preceding section on nonunions.

## Technical Problems

### Nonunion/Delayed Union

The most common technical problem is nonunion, usually due to inadequate treatment. Management of this problem was discussed in the previous section dealing with treatment of nonunions. Delayed union was also discussed in that section.

### Fishtail Deformity

Two types of fishtail deformity of the distal humerus may occur. The first, a sharp-angled wedge, commonly occurs after fractures of the lateral condyle ( [Fig. 15-20](#)). This type is believed to be caused by persistence of a gap between the lateral condylar physis ossification center and the medial ossification of the trochlea ( [98,102](#)). Because of this gap, the lateral crista of the trochlea may be underdeveloped, and this may represent a small “bony bar” in the distal humeral physis ( [48](#)). Rutherford ( [81](#)) found that this type of deformity occurred only in fractures that were inadequately reduced. Despite some reports of loss of elbow motion with this type of fishtail deformity ( [99](#)), most investigators ( [14,18,32,41](#)) have not found this type of radiographic deformity to produce any functional deficiency.



**FIGURE 15-20.** An angular fishtail deformity has persisted in this 14-year-old boy who sustained a lateral condylar fracture that was treated operatively 6 years previously.

The second type of fishtail deformity is a gentler smooth curve. It is usually believed to be associated with osteonecrosis of the lateral part of the medial crista of the trochlea ( [74](#)). The mechanisms of the development of this type of deformity are discussed in the section on osteonecrosis of the trochlea.

### Neurologic Complications

The neurologic complications can be divided into two categories: acute nerve problems at the time of the injury and delayed neuropathy involving the ulnar nerve (the so-called tardy ulnar nerve palsy).

#### Acute Nerve Injuries

Reports of acute nerve injuries associated with this injury are rare. Smith and Joyce ( [89](#)) reported two patients with posterior interosseous nerve injury after open reductions of the lateral condylar fragment, both of whom recovered spontaneously. McDonnell and Wilson ( [64](#)) reported a transient radial nerve paralysis after an acute injury.

Friedman and Smith ( [43](#)) reported a delayed radial nerve laceration from the tip of the screw that was used to stabilize a lateral condylar fracture 26 years earlier. This occurred when the patient sustained a hyperextension injury to the elbow.

#### Tardy Ulnar Nerve Palsy

Tardy ulnar nerve palsy as a late complication of fractures of the lateral condylar physis is well known. One of the earliest and most complete studies of this problem in the United States was conducted by Miller ( [68](#)) in 1924. Forty-seven percent of his patients with tardy ulnar nerve palsy had fractures of the lateral condylar physis as a child. The onset of the symptoms varied from 30 to 40 years. Subsequently, reports by numerous other investigators confirmed the frequency of this complication after the development of cubitus valgus from malunion or nonunion of fractures of the lateral condylar physis ( [27,35,45,57,65](#)). The symptoms are usually gradual in onset. Motor loss occurs first, with sensory changes developing somewhat later ( [27,45](#)). In Gay and Love's series ( [45](#)) of 100 patients, the average interval of onset was 22 years.

Various methods of treatment have been advocated, ranging from anterior transposition of the ulnar nerve (originally the most commonly used procedure) to simple relief of the cubital tunnel. In 1972, Wadsworth ( [99](#)) listed nine operative procedures described between 1898 and 1957 for treatment of tardy ulnar palsy. We prefer simple subcutaneous anterior transposition of the nerve.

#### Physeal Arrest

Physeal arrest may be manifest by no more than premature fusion of the various secondary ossification centers to each other, with little or no deformity. Such a situation occurs much later than the original fracture. This phenomenon probably occurs because the fracture stimulates the ossification centers to grow more rapidly and thus they reach maturity sooner, or, rarely, it is caused by inadvertent dissection in the lateral condylar physis. Because only 20% of humeral growth occurs in the distal physis, physeal arrest rarely causes any significant angular or length deformities.

#### Malunion

Rarely, the fragment unites in an undesirable position. Cubitus valgus has been reported to occur as a result of malunion of the fracture fragments ( [94,99](#)). Malunion of a Milch type I fracture pattern can result in the development of a bifid lateral condyle ( [Fig. 15-21](#)). No reliable operative treatment has been described to reestablish the congruity of the articular surfaces in condylar malunions, and they probably are best left untreated. We have seen several patients with malunions in which the lateral condyle rotates in the coronal plane, with subsequent cubitus varus deformity.



**FIGURE 15-21. A:** Injury film of a 7-year-old who sustained a Milch type I lateral condylar fracture. This patient was treated with cast immobilization alone. **B:** Film taken 2 years later showed complete fusion of the condylar epiphysis to the metaphysis, with the development of a “bifid” condyle.

### **Osteonecrosis**

Osteonecrosis of the condylar fragment may be iatrogenic in origin and is most commonly associated with the extensive dissection necessary to effect a late reduction (H. Brindley, personal communication) (47,55,64). Wilson (102), however, described partial osteonecrosis in an essentially nondisplaced fracture of the lateral condylar physis that had a radiographic appearance and clinical course similar to those of osteochondritis dissecans. Osteonecrosis is rare in fractures of the lateral condylar physis that receive little or no initial treatment and result in nonunion (55,101).

Overly vigorous dissection of fresh fractures can result in osteonecrosis of either the lateral condylar ossification center (41,75) or, rarely, the metaphyseal portion of the fragment, leading to nonunion (Fig. 15-22). If the fracture unites, osteonecrosis of the lateral condyle reossifies over many years, much like Legg-Calvé-Perthes disease in the hip. Any residual deformity usually is related to loss of motion.



**FIGURE 15-22.** Avascular necrosis developed in this child because of extensive dissection and difficulty in obtaining a primary open reduction. **A:** Injury film. **B:** Two years later, there was extensive loss of bone in the metaphysis and a nonunion of the condyle.

### **Myositis Ossificans**

The only report of myositis ossificans after lateral condylar fracture is that of Sandegard (84). In his patient, the fracture was associated with a dislocation. The myositis resolved with no functional residua. We have seen one patient with myositis ossificans after a delayed open reduction 1 week after injury. This patient had some residual loss of elbow extension.

### **Ipsilateral Injuries**

Fractures of the lateral condyle have been associated with elbow dislocations (14), fracture of the ulnar shaft (78), and fracture of the medial epicondyle (44). Often an elbow dislocation is misdiagnosed in a patient with a lateral condylar fracture. Loss of the lateral crista can make the elbow unstable and allow the proximal radius or ulna to translocate laterally. This is a part of a normal pathologic condition associated with completely displaced lateral condylar fractures. In a true elbow dislocation, the proximal radius and ulna are displaced, not only medially or laterally but also proximally (Fig. 15-23).



**FIGURE 15-23.** Ipsilateral injury. **A:** Anteroposterior lateral radiographs of an 8-year-old boy with a true posteromedial elbow dislocation (open arrow) and a Milch type I lateral condylar fracture. **B:** A small fracture of the coronoid process of the ulna (closed arrow) confirms the primary nature of the elbow dislocation.

### **Fractures of the Capitellum**

Fractures of the capitellum involve only the true articular surface of the lateral condyle. This includes, in some instances, the articular surface of the lateral crista of the trochlea. Usually this fragment comes from the anterior portion of the distal articular surface. There is some variation in the terminology used for this intraarticular fracture. It was originally described as Mouchet's fracture (118). Most articles in the English literature prefer the term *capitellum*, but occasionally the Latin term *capitulum humeri* is used. In the European literature, these fractures may be described as involving the eminentia capitata.

### **Incidence**

In adults these fractures are not uncommon, but they are rare in children. Marion and Faysse (117), in their review of 2,000 elbow fractures in children, found only one fracture of the capitellum. Since then, this fracture has been frequently reported in older adolescents (110,113,117,121). Marion and Faysse (117) pointed out that verified fractures of the capitellum have not been described in children under 12 years of age.

However, there have been two reports (105,108) of so-called anterior sleeve fractures of the lateral condyles, both in 8-year-olds (Fig. 15-24). These involved a good portion of the anterior articular surface. However, technically they could not be classified as pure capitellar fractures because they contained nonarticular epicondylar and metaphyseal portions in the fragment. This fracture often is difficult to diagnose because there is little ossified tissue. It is composed mainly of pure articular surface from the capitellum and essentially nonossified cartilage from the secondary ossification center of the lateral condyle.



**FIGURE 15-24.** Fracture of the capitellum. **A:** Osteochondral fracture of the capitellum in an 8-year-old girl. Note small fleck of bone ( *arrow*), which indicates possible osteochondral fragment. **B:** Single-spot lateral radiograph after double-contrast arthrography outlines the large osteoarticular fragment in the anterior recess of the elbow. **C:** Intraoperative photograph shows the size of the fracture fragment and its origin from the lateral humeral epiphysis. **D:** Intraoperative photograph after reduction of the fragment into the bed of the capitellum. **E:** Healed fracture with articular congruity, restoration of cartilage space, and no avascular necrosis. (Reprinted from Drvaric DM, Rooks MD. Case report. Anterior sleeve fracture of the capitellum. *J Orthop Trauma* 1990;4:188; with permission.)

### Classification

Two fracture patterns have been described. The first is the more common Hahn-Steinthal type ( [112,120](#)), which usually contains a rather large portion of cancellous bone of the lateral condyle. Often the lateral crista of the trochlea is also included ( [Fig. 15-25](#)). The second, or Kocher-Lorenz, type ( [114,116](#)) is more of a pure articular fracture with little if any subchondral bone attached, and may represent a piece of articular cartilage from an underlying osteochondritis dissecans. This type of fracture is rare in children ( [105](#)).



**FIGURE 15-25.** Fracture of the capitellum in a 13-year-old girl. **A:** Injury film, lateral view, shows the large capitellar fragment lying anterior and proximal to the distal humerus. Both the radiocapitellar ( *solid arrow*) and trochlear grooves ( *open arrow*) are seen in the fragment. **B:** In the anteroposterior view, only a faint outline of the fragment is seen ( *arrows*).

### Mechanism of Injury

The most commonly accepted mechanism is that the anterior articular surface of the lateral condyle is sheared off by the radial head ( [106,110](#)). The presence of cubitus recurvatum or cubitus valgus seems to predispose the elbow to this fracture pattern.

### Diagnosis

Often, swelling is minimal. Flexion is restricted by the presence of the fragment. If the fragment is large, it may be readily apparent on the lateral radiographs ( [Fig. 15-26](#)). On the anteroposterior radiographs, however, the fragment may be obliterated by the overlying distal metaphysis ( [Fig. 15-25](#)). If the fragment is small, oblique views may be necessary to show the fragment ( [106](#)). In younger children, arthrography or MRI may be necessary to diagnose this rare fracture. Letts et al. ( [115](#)) recommended computed tomography (CT) to help delineate the fracture type.



**FIGURE 15-26.** Fracture of the capitellum. **A:** Injury film. Lateral view of a 10-year-old with an isolated fracture of the capitellum. The capitellar fragment can be seen lying anteriorly ( *arrow*). **B:** On the anteroposterior view, the distal outline of the lateral condyle appears intact. The capitellar fragment is seen lying anterior to the trochlea ( *arrows*). **C:** Fixation of the fragment was achieved by retrograde placement of pins. **D:** Seven months after injury, the fragment has undergone resorption, leaving a defect in the lateral condyle ( *arrows*). **E:** Three years after injury, at the end of skeletal growth, the defect has enlarged. The radial head has migrated proximally into the defect. Elbow motion is greatly restricted. (Courtesy of Jack Earle, M.D.)

### Associated Injuries

Because the mechanism is postulated to be a pushing off of the capitellum by the radial head, it stands to reason that there may be an associated radial head or neck fracture ( [113](#)). In Palmer's ( [119](#)) large series, including both children and adults, 31% had associated injuries of the proximal radius.

### Treatment

Excision of the fragment and open reduction and reattachment are the two most common forms of treatment. Closed reduction is not likely to be successful.

### Excision of the Fragment

Through an open arthrotomy, the fragment can be either excised or reattached. Most investigators ([106,110,117](#)) favor excision because of its simplicity. In addition, motion and rehabilitation can be initiated early. In those series in which excision and replacement were compared ([110,116](#)), excision appeared to provide better results. In addition, the earlier the fragment was excised, the better were the results. Even when large fragments were excised, joint instability did not appear to be a problem ([111](#)). In patients in whom treatment is delayed, although the results are not as good as when treatment is provided immediately after injury, improvement in function can be expected, even with late excision ([110](#)).

### Reattachment of the Fragment

If the fragments are reattached, stability of the fracture is provided by wires or screws inserted through the posterior surface of the lateral condyle. The investigators who have advocated this procedure (489,491,494) have almost equally good results with reattachment. The major problems with reattachment are associated with osteonecrosis of the reattached fragment. Letts et al. ([115](#)) reported satisfactory results with fixation of five capitellar fractures with Kirschner wires (three fractures), Herbert screws (one fracture), and cannulated screw (one fracture). An advantage of screw fixation is that it does not require later removal.



### AUTHORS' PREFERRED METHOD OF TREATMENT

If the fragment is large, the fracture is acute, and an anatomic reduction can be achieved with a minimum of open manipulation or dissection, then we prefer to reattach it with two small compression or Herbert screws inserted from posterior to anterior through a lateral approach. Enough bone must be present in the capitellar fragment to engage the screw threads and if possible countersink the heads of the cancellous or Herbert screws ([Fig. 15-27](#)). However, if it is an old fracture, if there is any comminution of the fragment, or if there is little bone in which to engage the screw threads, we believe the appropriate treatment is simply to excise the fragment and start early motion.



**FIGURE 15-27.** Postoperative anteroposterior (A) and lateral (B) radiographs of the patient seen in [Fig. 15-26](#). There was enough bone in the fragment for it to be secured by the screw threads.

### Complications

The major complication is osteonecrosis of the fragment ([Fig. 15-26](#)). This, of course, occurs only in fractures in which the capitellar fragment is retained. Posttraumatic degenerative arthritis can occur whether the fragments are excised or retained. Many patients who are treated either operatively or nonoperatively can expect to lose some range of motion, but this is not always of functional or cosmetic significance. It is important to emphasize to the parents before the onset of treatment that complications can occur regardless of the method of treatment.

### Fractures Involving the Medial Condylar Physis

Fractures involving the medial condylar physis have two components. The intraarticular component involves, in some manner, the trochlear articular surface. The extraarticular portion includes the medial metaphysis and medial epicondyle. Because the fracture line extends into the articular surface of the trochlea, these are often called trochlear fractures. For purposes of description in this chapter, fractures of the trochlea are those that include only the articular surface.

### Incidence

Fractures involving the medial condylar physis are rare in skeletally immature children. In the combined series of 5,226 fractures involving the distal humerus mentioned previously, only 37 fractures involved the medial condylar physis. These were all grouped into three series ([163](#)).

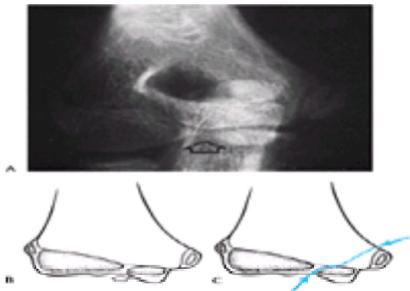
Many of the large series of elbow fracture in the literature ([128,130,133,140,143](#)) and early fracture texts (510,541) do not mention these fractures as a separate entity. Blount ([124](#)) described only one such fracture in his classic text. In Faysse and Marion's ([135](#)) review of more than 2,000 fractures of the distal humerus in children, only 10 fractures involved the medial condylar physis. It can be safely said that the occurrence rate is less than 1% of all elbow fractures in children.

Although it has been reported in a child as young as 2 years of age ([122](#)), this fracture pattern is generally considered to occur during later childhood.

Most series ([135,145](#)) show medial condylar fractures occurring somewhat later than lateral condylar fractures. A review of 38 patients in nine series ([122,127,129,132,134,135](#) and [136,147,150](#)) in which the specific ages were given showed that 37 patients were in the age range of 8 to 14 years. Thus, this fracture seems to occur after the ossification centers of the medial condylar epiphysis begin to appear. However, this fracture can occur as early as 6 months of age, before any ossification of the distal humerus has appeared ([123,131](#)), making the diagnosis extremely difficult.

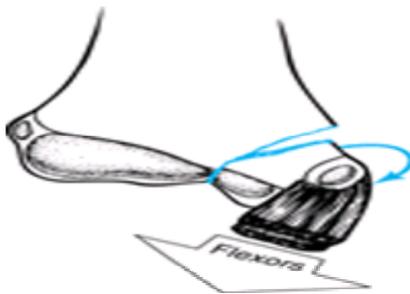
### Surgical Anatomy and Pathology

Fractures of the medial condylar physis involve both intra- and extraarticular components. They behave as Salter-Harris type IV physeal injuries, but not enough fractures have been described to show whether the fracture line courses through the secondary ossification center of the medial condylar epiphysis or whether it enters the common physeal line separating the lateral condylar ossification center from the medial condylar ossification center. This common physeal line terminates in the notch of the trochlea. The lateral crista of the trochlea is ossified from the lateral condylar epiphysis. Only the medial crista is ossified by the secondary ossification centers of the medial condylar epiphysis. We believe that this fracture is a mirror image of the lateral condylar physeal injury and thus has characteristics of Salter-Harris type IV physeal injuries ([Fig. 15-28](#)). The deformity that develops if the fracture is untreated is nonunion, similar to that after lateral condylar physeal fracture, rather than physeal fusion, as occurs after a typical Salter-Harris type IV injury. The resultant deformity is cubitus varus instead of the cubitus valgus deformity that occurs with nonunion of the lateral condyle.



**FIGURE 15-28. A:** Anteroposterior radiograph of a 9-year-old boy demonstrating the location of the ossification centers. A common physal line ( *arrow*) separates the medial and lateral condylar physes. **B:** Relationship of the ossification centers to the articular surface. The common physis terminates in the trochlear notch ( *arrow*). **C:** Location of the usual fracture line involving the medial condylar physis ( *arrow*).

Characteristically, the metaphyseal fragment includes the intact medial epicondyle along with the common flexor origin of the muscles of the forearm. These flexor muscles cause the loosened fragment to rotate so that the fracture surface is facing anteriorly and medially and the articular surface is facing posteriorly and laterally (Fig. 15-29) (122,127). Rotation of the fragment is especially accentuated when the elbow is extended. Chacha (127) also noted that often the lateral aspect of the common flexor origin and the anterior capsule of the joint were torn and the fracture surface could usually be reached through this anterior opening into the joint.



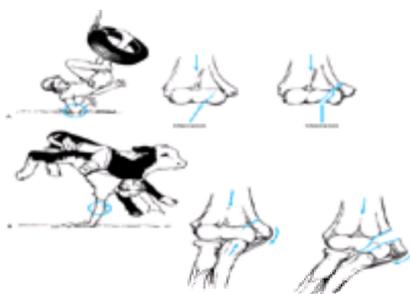
**FIGURE 15-29.** Displacement of the medial condyle. The pull of the forearm flexor muscles rotates the fragment so that the fracture surface is facing anteromedially and the articular surface is posterolateral. (Redrawn from Chacha PB. Fractures of the medial condyle of the humerus with rotational displacement. *J Bone Joint Surg [Am]* 1970;52:1453-1458; with permission.)

The blood supply to the medial epicondyle and medial metaphysis courses extraarticularly along with the medial flexor muscle groups. The blood supply to the lateral ossification center of the medial crista of the trochlea, however, must traverse the surface of the medial condylar physis. Disruption of these small intraarticular vessels by the fracture line can result in disruption and subsequent loss of circulation to the lateral portion of the medial crista, leading to the development of a fishtail deformity.

Because the fracture line enters the groove of the trochlea, the elbow joint becomes unstable, allowing a varus deformity to develop.

### **Mechanism of Injury**

Two separate mechanisms can produce physal fractures of the medial condyle. Ashhurst's (122) patients described falling directly on the point of the flexed elbow. This mechanism also was implicated in other reports (123,127,138,147). In this mechanism, it is speculated that the sharp edge of the semilunar notch of the olecranon splits the trochlea directly (Fig. 15-30A). In three more recent series (123,132,136), many patients sustained this injury when they fell on their outstretched arms. The theory is that this is an avulsion injury caused by a valgus strain at the elbow (Fig. 15-30B). Fowles and Kassab (136) reported a patient with a concomitant valgus greenstick fracture of the olecranon associated with a fracture of the medial condylar physis. They believed this provided further evidence that this was a valgus avulsion type of injury.



**FIGURE 15-30.** Medial condyle fracture mechanisms of injury. **A:** A direct force applied to the posterior aspect of the elbow causes the sharp articular margin of the olecranon to wedge the medial condyle from the distal humerus. **B:** Falling on the outstretched arm with the elbow extended and the wrist dorsiflexed causes the medial condyle to be avulsed by both ligamentous and muscular forces.

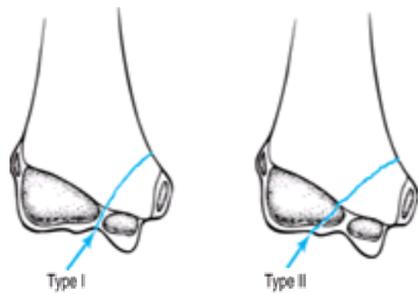
Once the fragment becomes disassociated from the distal humerus, the forearm flexor muscles produce a sagittal anterior rotation of the fragment.

### **Classification**

Classification, as with fractures of the lateral condylar physis, is based on the location of the fracture line and the degree of displacement of the fracture.

### **Location of the Fracture Line**

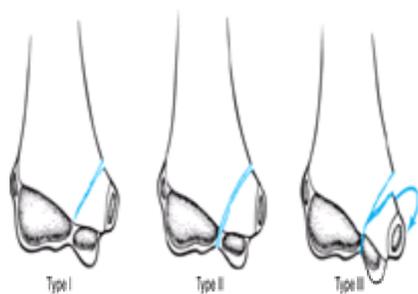
Milch (144) classified fractures of the medial condylar physis in adults into two types. In type I fractures, the fracture line traverses the apex of the trochlea. In type II fractures, it traverses the capitulotrochlear groove. He believed that the origin of the fracture line depended on whether the radial head, as in type II, or the semilunar notch of the olecranon, as in type I, served as the impinging force for the abduction injury. Both fracture patterns occur in children (Fig. 15-31), but type I fractures seem to be more common because the common physal line, which serves as a point of weakness, ends in the apex of the trochlea.



**FIGURE 15-31.** Medial condyle fracture patterns. Left: In the Milch type I injury, the fracture line terminates in the trochlear notch ( *arrow*). **Right:** In the Milch type II injury, the fracture line terminates more laterally in the capitulotrochlear groove ( *arrow*). (Redrawn from Milch H. Fractures and fracture-dislocations of the humeral condyles. *J Trauma* 1964;4:592-607; with permission.)

### Displacement of the Fracture

Kilfoyle (142) described three fracture displacement patterns that can be helpful in determining appropriate treatment ( Fig. 15-32). In type I, the fracture line in the medial condylar metaphysis extends down to the physis. He noted that some of these may represent incomplete supracondylar fractures. Unless there is a greenstick crushing of the medial supracondylar column, these fractures are usually of no clinical significance. In type II, the fracture line extends into the medial condylar physis. The intraarticular portion, as it is in preosseous cartilage, is often not recognized. In this second type, the medial condylar fragment usually remains undisplaced. In type III, the condylar fragment is both rotated and displaced.



**FIGURE 15-32.** Kifoye classification of displacement patterns. (Redrawn from Kifoye RM. Fractures of the medial condyle and epicondyle of the elbow in children. *Clin Orthop* 1965;41:43-50; with permission.)

Bensahel (123) and Papavasiliou (145) and their co-workers found that type III displacement fractures, which accounted for only 25%, were more likely to occur in older adolescents, and type I fractures were more common in younger children. These studies also confirmed the correlation between the type of displacement and the method of treatment.

Medial condylar physeal fractures have been reported in association with greenstick fractures of the olecranon (130) and with true posterolateral elbow dislocations (Fig. 15-33) (123,149). Some investigators (123,131) found that child abuse was more common in their younger patients with these fractures than with other elbow fractures.



**FIGURE 15-33. A:** Injury film of a 10-year-old girl who sustained a type III displaced fracture of the medial condyle associated with a posterolateral elbow dislocation. **B:** Open reduction and Kirschner wire fixation was performed using an anteromedial approach. (Courtesy of Elizabeth A. Szalay, M.D.)

### Diagnosis

Clinically and on radiographs, a fracture of the medial condylar physis is most often confused with a fracture of the medial epicondyle. In both types of intra- and extraarticular fractures, swelling is concentrated medially, and there may be valgus instability of the elbow joint. In a true intraarticular fracture, however, there is varus instability as well. Such is usually not the case with an isolated extraarticular fracture of the medial epicondyle. Ulnar nerve paresthesia may be present with both types of fractures.

In older children with a large metaphyseal fragment, involvement of the entire condyle usually is obvious on radiographs; in younger children, in whom only the epicondyle is ossified, fracture of the medial condylar physis may be erroneously diagnosed as an isolated fracture of the medial epicondyle ( Fig. 15-34) (129,134,136).



**FIGURE 15-34.** Missed medial condyle fracture. **A:** Initial film of a 6-year-old who was originally diagnosed as having a displaced fracture of the medial epicondyle (*arrow*). **B:** Normal side for comparison. **C:** Three months later, the patient continued to have a painful elbow, and there was ossification of the metaphysis (*arrow*) adjacent to the epicondyle.

In differentiating these two fractures, it is helpful to remember that medial epicondylar fractures often are associated with elbow dislocations, usually posterolateral, and that elbow dislocations are rare before ossification of the medial condylar epiphysis begins. With medial condylar physeal fractures, the elbow tends to subluxate posteromedially ([129](#)) because of the loss of trochlear stability.

Any metaphyseal ossification with the epicondylar fragment suggests the presence of an intraarticular fracture of the medial condyle and is an indication for further evaluation. Often the medial condyle and the medial epicondyle are markedly displaced as a unit ([139](#)). A positive fat pad sign indicates that the injury has entered the elbow joint and a fracture of the medial condyle is likely ([138](#)). Isolated fractures of the medial epicondyle are extraarticular and usually do not have positive fat pad signs.

If the true location of the fracture line is questionable in a younger child, arthrography or MRI of the elbow should be performed.

### **Treatment**

For displaced fractures, open reduction with internal fixation seems to be the most popular treatment method ([123,126,132,136,141,142,145,146,147](#) and [148](#)). The fracture fragment can be approached by a posteromedial incision that allows good exposure of both the fracture site and the ulnar nerve. Rigid fixation is imperative and is easily achieved with smooth Kirschner wires ([Fig. 15-33](#)) or with screws in older adolescents. Two wires are necessary because of the sagittal rotation forces exerted on the fracture fragment by the common flexor muscles. El Ghawabi ([132](#)) reported frequent delayed union and nonunion in fractures that were not rigidly stabilized.

In Kilfoyle's displacement type I and II fracture patterns, usually enough residual internal stability is present to allow the fracture to be simply immobilized in a cast or posterior splint ([123,132,135,142,145](#)). As with fractures of the lateral condylar physis, union may be slow. In fractures treated promptly, results have been satisfactory ([127,134,136](#)). Because there is usually more displacement in older children, the results in this age group are not as satisfactory as those in younger ones, who tend to have relatively nondisplaced fractures ([123](#)).

The real problem lies in fractures that are discovered or seen many weeks after the original injury. Fowles and Kassab ([136](#)) reported poor results when the fracture was treated surgically 3 to 4 weeks after the original injury. As with lateral condylar fractures, this was believed to be due to the extensive dissection necessary to achieve an adequate reduction. For this reason, they concluded that fractures in which the time span was greater than 3 weeks after injury should be left alone.

### **AUTHORS' PREFERRED METHOD OF TREATMENT**

We usually treat type I nondisplaced fractures with simple observation and a posterior splint. Follow-up radiographs at weekly intervals are taken to ensure there is no late displacement. When there is good callus at the metaphyseal portion of the fracture line, the splint is removed and early active motion initiated. We continue to follow the patient until there is a full range of motion and obliteration of the fracture line.

Type II and III displaced fractures must be reduced and stabilized. Usually this is difficult to do by closed methods because the swelling associated with this injury makes it difficult to identify the landmarks accurately for pin placement. Unless these can be defined without question, we usually proceed with an open reduction through a medial approach. The posterior surface of the condylar fragment and the medial aspect of the medial crista of the trochlea should be avoided in the dissection because these are the sources of blood supply to the ossific nuclei of the trochlea. Fixation with two parallel pins should be in the metaphyseal segment if possible ([Fig. 15-35](#)). Cancellous screw fixation can be used in adolescents near skeletal maturity.



**FIGURE 15-35.** Stage II fracture of the medial condyle in a 10-year-old girl. Anteroposterior (**A**) and lateral (**B**) radiographic views.

Osteonecrosis of the trochlea can occur after both operative and nonoperative treatment (see the following section on [Complications](#)). Thus, the parents should be warned of this possible complication before treatment.

### **Complications**

The major complication is failure to make the proper diagnosis. This is especially true in younger children, in whom a medial condylar fracture can be confused with a displaced fracture of the medial epicondyle ([Fig. 15-34](#)). When the diagnosis is questionable, especially in a child with no ossification of the trochlea, examination with anesthesia, arthrography, or MRI may be helpful.

Untreated displaced fractures usually result in nonunion with cubitus varus deformity ([Fig. 15-36](#)) ([136,150](#)). We have seen one nonunion after a fracture of the medial condyle. When the condylar fragment finally ossified, the lateral edge of the fragment appeared to extend to the capitulotrochlear groove.



**FIGURE 15-36.** Nonunion in addition to cubitus varus deformity. **A:** Original film of a 5-year-old girl who sustained an injury 1 year previously. The metaphyseal fragment (*arrow*) is attached to the medial epicondyle. **B:** Film taken 2 years later. Some ossification has developed in the medial condylar epiphysis (*arrow*). (Courtesy of Roy N. Davis, M.D.)

Delayed union has been reported in patients treated with insecure fixation or simply placed in a cast ( [132,142](#)).

Some disturbance of the vascular supply to the medial condylar fragment appears to occur during open reduction and internal fixation. Several investigators have reported subsequent avascular changes in the medial crista of the trochlea ( [132,136,142,145](#)). Hanspal ( [137](#)) reviewed Cothay's original patient ( [129](#)) 18 years after delayed open reduction and found that despite some minimal loss of motion, the patient was asymptomatic. Radiography, however, demonstrated changes compatible with osteonecrosis of the medial condyle.

Both cubitus varus and valgus deformities have been reported in patients whose fractures united uneventfully. The valgus deformity appears to be due to secondary stimulation or overgrowth of the medial condylar fragment. Some simple stimulation of the prominence of the medial epicondyle also may produce the false appearance of a cubitus valgus deformity. Cubitus varus appears to result from decreased growth of the trochlea, possibly caused by a vascular insult. Principles for treating nonunion of lateral condylar fractures generally are applicable to nonunions of the medial condyle.

El Ghawabi ( [132](#)) described one partial ulnar neuropathy occurring after this type of injury. The neuropathy almost completely recovered after anterior transposition of the ulnar nerve.

### Fractures of the Trochlea

Osteochondral fractures involving only the articular portion of the trochlea are extremely rare in skeletally immature children: only one such fracture has been reported in the English-language literature. Grant and Miller ( [152](#)) reported on a 13-year-old boy who had a posterolateral dislocation of the elbow with marked valgus instability and fractures of the medial epicondyle and radial neck. When the elbow was explored to secure the epicondyle, a large osteochondral fragment from the medial crista of the trochlea was found lying between the two articular surfaces. The fragment was replaced and fixed with Smillie nails, and a satisfactory result was obtained. However, the presence of the fragment was not detected preoperatively.

In an older child who sustains an elbow dislocation and in whom there is some widening of the joint after reduction, an intraarticular fracture of the trochlea or capitellum should be suspected. Arthrography or MRI, or occasionally CT-arthrography, should be used for confirmation.

### Fractures Involving the Entire Distal Humeral Physis

#### Incidence

After the first period of interest in this injury during the early 1900s, there was almost a complete absence of any discussion of this injury in the English literature until the early 1950s. In fact, two researchers, Siris ( [186](#)) and Wilson ( [193](#)), doubted whether this existed as a separate fracture type.

Smith's ( [187](#)) description in his 1954 textbook, *Surgery of the Elbow*, sparked a renewed interest in this fracture. He focused attention on the maintenance of the relationship between the radius and capitellum as a distinguishing feature in this type of injury.

From 1960 to 1978, many individual patients were reported ( [159,166,172,175,179,184,185,189](#)). Once the presence of this injury became recognized, larger series appeared. Seven separate series reported a total of 45 fractures ( [154,162,163,170,176,177,182](#)), and Abe et al. ( [153](#)) reported on a series of 21 fractures.

Originally thought to be a rare injury, it appears that fractures involving the entire distal humeral physis occur frequently in children. The major problem is the initial recognition of this injury.

#### Surgical Anatomy

The distal humeral epiphysis extends across to include the secondary ossification of the medial epicondyle until about 6 to 7 years of age in girls and 8 to 9 years in boys. Thus, fractures involving this total physeal line include the medial epicondyle up to this age. In older children, only the lateral and medial condylar physeal lines are included.

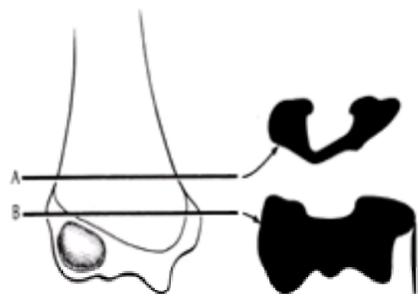
Most fractures involving the total distal humeral physis occur before the age of 6 or 7. The younger the child, the greater the volume of the distal humerus that is occupied by the distal epiphysis. As the humerus matures, the physeal line progresses more distally, with a central **V** forming between the medial and lateral condylar physes ( [Fig. 15-37](#)). Ashhurst ( [155](#)) believed that this **V**-shaped configuration of the physeal line helps protect the more mature distal humerus from physeal fractures.



**FIGURE 15-37.** **A:** At 5 months of age, the metaphysis has advanced only to the supracondylar ridges. **B:** By 4 years of age, the edge of the metaphysis has advanced well into the area of the epicondyles.

Because fractures coursing along the distal humeral physis traverse the anatomic centers of the condyles, they are the pediatric counterpart of the adult bicondylar fracture. Because the fracture is distal, the fracture surfaces are broader than those proximally through the supracondylar fractures ( [Fig. 15-38](#)). This broader surface

area of the fracture line may help prevent tilting of the distal fragment. Because the articular surface is not involved by the fracture lines, development of joint incongruity with resultant loss of elbow motion is unlikely if malunion occurs.



**FIGURE 15-38.** The cross-sectional surface of the supracondylar area (A) is much thinner than the cross-sectional area through the distal physis (B).

Finally, part of the blood supply to the medial crista of the trochlea courses directly through the physis. The blood supply to this area is vulnerable to injury, which may cause avascular changes in this part of the trochlea.

Because the physal line is more proximal in young infants, it is nearer the center of the olecranon fossa ( Fig. 15-37). A hyperextension injury in this age group is more likely to result in a physal separation than a bony supracondylar fracture ( 160,161).

### **Mechanism of Injury**

The exact mechanism of this injury is unknown and probably varies with the age group involved. A few consistent factors are evident.

First, many fractures of the entire distal humeral physis have occurred as birth injuries associated with difficult deliveries ( 154,156,157,165,167,175,185,190). Siffert (185) noted that the clinical appearance of these injured elbows at the time of delivery was not especially impressive. There was only moderate swelling and some crepitus.

Second, DeLee et al. (163) noted a high incidence of confirmed or suspected child abuse in their very young patients. Other reports ( 154,162,178,180,192) have confirmed the frequency of child abuse in infants and young children with these fractures.

Bright (158) has shown that a physis is more likely to fail with rotary shear forces than with pure bending or tension forces. Young infants have some residual flexion contractures of the elbow from intrauterine positioning; this prevents the hyperextension injury that results in supracondylar fractures of the elbow in older children. Rotary forces on the elbow, which can be caused by child abuse or birth trauma in young infants, are probably more responsible for this injury than hyperextension or varus or valgus forces, which produce other fracture patterns in older children.

Abe et al. (153) reported on 21 children, ranging in age from 1 to 11 years (average 5 years), with fracture-separations of the distal humeral epiphysis, all of which were sustained in falls.

### **Classification**

DeLee et al. (163) classified fractures of the entire distal humeral physis into three groups based on the degree of ossification of the lateral condylar epiphysis ( Fig. 15-39). Group A fractures occur in infants up to 12 months of age, before the secondary ossification center of the lateral condylar epiphysis appears ( Fig. 15-39A and Fig. 15-39B). They usually are Salter-Harris type I physal injuries. This injury often is not diagnosed because of the lack of an ossification center in the lateral condylar epiphysis. Group B fractures occur most often in children 12 months to 3 years of age in whom there is definite ossification of the lateral condylar epiphysis (Fig. 15-39C). Although there may be a small flake of metaphyseal bone, this is also essentially a type I Salter-Harris physal injury. Group C fractures occur in older children, from 3 to 7 years of age, and result in a large metaphyseal fragment that is most commonly lateral but can be medial or posterior (see Fig. 15-39D and Fig. 15-39E).



**FIGURE 15-39.** A: Group A—anteroposterior view of a small infant who had a swollen left elbow after a difficult delivery. The displacement medially of the proximal radius and ulna (arrow) helps to make the diagnosis of a displaced total distal humeral physis. B: Normal elbow for comparison. C: Group B—anteroposterior view showing the posteromedial displacement of the distal fragment (arrow). The relationship between the ossification center of the lateral condyle and the proximal radius has been maintained. D: Group C—anteroposterior view with marked medial displacement of the distal fragment. E: Group C—lateral view of the same patient showing posterior displacement of the distal fragment. There is also a large metaphyseal fragment associated with the distal fragment (arrow).

These fractures are almost always extension-type injuries with the distal epiphyseal fragment posterior to the metaphysis. A rare flexion type of injury can occur in which the epiphyseal fragment is displaced anteriorly ( 157). Stricker and colleagues (188) reported a coronal plane transcondylar (Salter-Harris type IV) fracture in a 3-year-old child that was initially misdiagnosed as a fracture of the lateral humeral condyle. Three years after open reduction and pin fixation, no growth disturbance was evident.

### **Clinical Signs and Symptoms**

In an infant less than 18 months of age, whose elbow is swollen secondary to trauma or suspected trauma, a fracture involving the entire distal humeral physis should be considered. In a young infant or newborn, swelling may be minimal with little crepitus. Poland ( 183) described the crepitus as “muffled” crepitus because the fracture ends are covered with softer cartilage than the firm osseous tissue in other fractures about the elbow. The relationship between the epicondyles and the olecranon is maintained. Because of the large, wide fracture surfaces, there is less tendency for tilting with rotation of the distal fragment, and the angular deformity is less severe than that with supracondylar fractures. Often in older children, the elbow is so swollen that a clinical assessment of the bony landmarks is impossible, and radiographic evaluation must provide confirmation of the diagnosis ( Fig. 15-39A and Fig. 15-39B).

## Radiographic Findings

Radiographic diagnosis can be difficult, especially if the ossification center of the lateral condyle is not visible in an infant. The only relationship that can be determined is that of the primary ossification centers of the distal humerus to the proximal radius and ulna. The proximal radius and ulna maintain an anatomic relationship to each other but are displaced posteriorly and medially in relation to the distal humerus. This posteromedial relationship is diagnostic. Although theoretically the distal fragment can be displaced in any direction, with rare exceptions (157) most fractures reported have been displaced posteromedially. Valdiserri and Kelescian (190) were the first to emphasize the consistency of this posteromedial displacement. Comparison views of the opposite uninjured elbow may be helpful to determine the presence of posteromedial displacement (Fig. 15-39A and Fig. 15-39B).

Once the lateral condylar epiphysis becomes ossified, displacement of the entire distal epiphysis is much more obvious. The anatomic relationship of the lateral condylar epiphysis with the radial head is maintained, even though the distal humeral epiphysis is displaced posterior and medial in relation to the metaphysis of the humerus (Fig. 15-39C and Fig. 15-39D).

Because they have a large metaphyseal fragment, type C fractures may be confused with either a low supracondylar fracture or a fracture of the lateral condylar physis. The key diagnostic point is the smooth outline of the distal metaphysis in fractures involving the total distal physis. With supracondylar fractures, the distal portion of the distal fragment has a more irregular border.

Differentiation from a fracture of the lateral condylar physis and the rare elbow dislocation in an infant can be made on radiography. With a displaced fracture of the lateral condylar physis, the relationship between the lateral condylar epiphysis and the proximal radius is usually disrupted (Fig. 15-8B). If the lateral crista of the trochlea is involved, the proximal radius and ulna may be displaced posterolaterally.

Elbow dislocations are rare in the peak age group for fractures of the entire distal humeral physis. With elbow dislocations, the displacement of the proximal radius and ulna is almost always posterolateral, and the relationship between the proximal radius and lateral condylar epiphysis is disrupted.

If differentiation of this injury from an intraarticular fracture is uncertain, arthrography or MRI may be helpful (Fig. 15-6C) (154,156,159,168,171,177).

In neonates and infants in whom ossification has not begun, ultrasonography can be used to outline the epiphysis of the humerus (164,191). Comparison with the opposite uninjured humeral epiphysis may help determine the presence of a separation.

If the diagnosis is delayed, new periosteal bone forms around the distal humerus, and the whole epiphysis may remain displaced posteriorly and medially (Fig. 15-40).



**FIGURE 15-40.** The true nature of this injury as involving the entire distal humeral physis was not appreciated until periosteal new bone became visible 3 weeks after injury.

## Treatment

Treatment is first directed toward prompt recognition of the injury. Because this injury may be associated with child abuse, the parents may delay seeking treatment.

De Jager and Hoffman (162) reported 12 fracture-separations of the distal humeral epiphysis, three of which were initially misdiagnosed as fractures of the lateral condyle and one as an elbow dislocation. Because of the frequency of cubitus varus after this injury in young children, they recommend closed reduction and percutaneous pinning in children under 2 years of age so that the carrying angle can be evaluated immediately after reduction and corrected if necessary.

Open reduction has been reported by several investigators, usually performed because of misdiagnosis as a displaced fracture of the lateral humeral condyle (154,170,179,184,192). Mizuno (177), however, recommended primary open reduction because of his poor results with closed reduction. He approached the fracture posteriorly by removing the triceps insertion from the olecranon with a small piece of cartilage.

If the fracture is old (>5–6 days) and the epiphysis is no longer mobile, manipulation should not be attempted and the elbow should be splinted for comfort. Many essentially untreated fractures remodel completely without any residual deformity if the distal fragment is only medially translocated and not tilted (Fig. 15-41).



**FIGURE 15-41.** Remodeling of untreated fractures. **A:** Anteroposterior view of a 2-year-old who had an unrecognized and untreated fracture of the distal humeral physis. The medial translocation is apparent. There was no varus or valgus tilting. **B:** Four years later, there had been almost complete remodeling of the distal humerus. A small supracondylar prominence (arrow) remains as a scar from the original injury. **C:** Clinical appearance 4 years after injury shows no difference in the alignment of the elbows.

## AUTHORS' PREFERRED METHOD OF TREATMENT

We usually first attempt a manipulative closed reduction of fresh fractures. The elbow is first manipulated into extension to correct the medial displacement, then the fragment is stabilized by flexing the elbow and pronating the forearm. The distal epiphysis is more securely held with the elbow flexed and the forearm pronated (Fig. 15-41).

15-42). When the forearm is supinated with the elbow flexed, the distal fragment tends to displace medially. This is usually a pure medial horizontal translocation without mediolateral coronal tilting.



**FIGURE 15-42.** Supination versus pronation. **A:** In this fracture involving the entire distal humeral physis in a 9-month-old, the elbow was initially flexed with the forearm in supination. The Jones view shows that the proximal radius and ulna remain translocated medially in relation to the distal humerus. **B:** Comparison Jones view of the uninjured elbow shows the true relationship of the proximal radius and ulna to the distal humerus. **C:** When the injured elbow was flexed with the forearm pronated, the normal relationship was reestablished.

In neonates and very small infants in whom general anesthesia or percutaneous pin fixation may be difficult, we usually simply immobilize the extremity in 110 to 120 degrees of hyperflexion with the forearm pronated. The extremity is then externally stabilized with a figure-of-eight splint.

In most older infants and young children, external immobilization usually is not dependable in maintaining the reduction. In these patients, we usually perform the manipulation with the patient under general anesthesia and secure the fragment with two lateral pins (Fig. 15-43). Because of the swelling and immaturity of the distal humerus, the medial epicondyle is difficult to define as a distinct landmark, making it risky to attempt the percutaneous placement of a medial pin. If a medial pin is necessary for stable fracture fixation, a small medial incision can be made to allow direct observation of the medial epicondyle. In small infants and young children with minimal ossification of the epiphyseal fragment, an intraoperative arthrogram is usually obtained to help determine the quality of the reduction.



**FIGURE 15-43.** **A:** Injury film of a 20-month-old showing medial displacement of the distal fragment. **B:** The posterior displacement of the condylar fragment (*arrow*) is better defined after an arthrogram. **C:** Manipulation with the forearm in pronation shows the fragment is anatomically reduced. The arthrogram outlines the articular margin of the condyles (*arrow*). **D:** Fixation is achieved by two lateral pins placed percutaneously.

The cast or splint and pins are removed in 3 weeks to allow resumption of active elbow motion. The patient is then followed until full motion is regained and there is radiographic evidence of normal physeal and epiphyseal growth. Usually, 3 weeks of immobilization is sufficient.

If treatment is delayed more than 3 to 5 days and the epiphysis is not freely movable, the elbow is simply immobilized in a splint or cast. Any resulting deformity is probably better treated later with a supracondylar osteotomy rather than risk the complication of physeal injury or devascularization of the epiphysis by performing a delayed open reduction. Only occasionally does an untreated patient have a deformity severe enough to require surgical correction at a later date. Because the articular surface is intact, complete functional recovery can usually be expected.

### Complications

#### Child Abuse

Child abuse should always be considered in children with this injury, especially a type A fracture pattern, unless it occurs at birth. A young infant is unlikely to incur this type of injury spontaneously from the usual falls that occur during the first year of life. Of the 16 fractures reported by DeLee et al. (163), 6 resulted from documented or highly suspected child abuse, all in children under 2 years of age (Fig. 15-44).



**FIGURE 15-44.** **A:** A 6-month-old victim of child abuse. The fracture involving the distal humeral physis was old, as indicated by the proliferative new periosteal bone formation (*open arrow*). Acutely there was a separation of the proximal humeral physis (*black arrow*). **B:** Another child, 9 months old, who had an acute fracture through the distal humeral physis, as manifest by posteromedial displacement of the proximal radius and ulna (*closed arrow*). An old healing fracture is also seen in the ipsilateral radius (*open arrow*).

Neurovascular injuries, either transient or permanent, are rare with this fracture, probably because the fracture fragments are covered with physeal cartilage and do not have sharp edges as do other fractures in this area. In addition, the fracture fragments usually are not markedly displaced. Hersch and Sanders (169) reported neurologic injury in one child who was treated for an "elbow dislocation." About 16 hours after reduction and splinting with the elbow acutely flexed, the child could not move her hand, and no radial, median, or ulnar nerve function was detectable. At last follow-up after open reduction and internal fixation with Kirschner wires, the

neurologic deficit was slowly resolving.

### Nonunion

Only one nonunion after this fracture has been reported; it occurred in a patient seen 3 months after the initial injury ( [177](#)). Because of the extreme vascularity and propensity for osteogenesis in this area, union is rapid even in patients who receive essentially no treatment.

### Malunion

Significant cubitus varus deformity is not uncommon after this injury ( [Fig. 15-45](#)). Marmor and Bechto ( [174](#)) reported an essentially untreated fracture that resulted in significant cubitus varus. Five of seven fractures in Holda and colleagues' series ( [170](#)) resulted in angles of cubitus varus of 10 to 15 degrees, even when open reduction was performed. Abe et al. ( [153](#)) reported cubitus varus deformities in 15 of 21 children, 7 of whom were treated with closed reduction and casting, 2 with closed reduction and percutaneous pinning, 4 with open reduction and internal fixation, and 2 with skin traction. Review of radiographs after reduction revealed that the deformity was caused by incorrect reduction (12 fractures), redisplacement during immobilization (2 fractures), or physal damage (1 fracture).



**FIGURE 15-45.** An anteroposterior view of a residual cubitus varus in a 2-year-old 6 months after the original injury. This patient was treated initially with only simple immobilization.

Because the fracture surfaces are wider with this injury than with supracondylar fractures, the distal fragment tends to tilt less, which seems to account for the lower incidence of cubitus varus after this injury than after untreated supracondylar fractures ( [Fig. 15-45](#)).

### Osteonecrosis

Osteonecrosis of the epiphysis of the lateral condyle or the trochlear epiphysis has rarely been reported after fractures of the entire distal humeral physis. Yoo et al. ( [194](#)) reported on eight patients with osteonecrosis of the trochlea after fracture–separations of the distal end of the humerus. Six of the eight fractures were misdiagnosed initially as medial condylar fractures, lateral condylar fractures, or traumatic elbow dislocation. All eight patients had rapid dissolution of the trochlea within 3 to 6 weeks after injury, followed by the development of a medial or central condylar fishtail defect. We have noted osteonecrosis of the trochlea after three fractures of the entire humeral physis, two of which were inadequately reduced and one of which was anatomically reduced by closed methods ( [Fig. 15-46](#)). All three had marked displacement of the distal epiphyseal fragment. In one, the osteonecrosis of the trochlea produced a secondary cubitus varus deformity that continued to progress with growth and a significant loss of elbow motion. The etiology of this complication was discussed in the section on osteonecrosis of the trochlea.



**FIGURE 15-46.** Avascular necrosis. **A:** Injury film in a 5-year-old with marked displacement of the distal epiphysis. Closed reduction followed by lateral pin fixation was performed. **B:** Nevertheless, radiographs taken 14 months later showed early evidence of avascular necrosis of the medial condyle. (Courtesy of Salvador J. Mendez, M.D.)

## APOPHYSEAL INJURIES OF THE DISTAL HUMERUS

### Fractures Involving the Medial Epicondylar Apophysis

#### History Review

#### Recognition of Entrapment

In the early 1900s, it was recognized that this fracture was often associated with dislocation of the elbow, and the apophyseal fragment could become entrapped within the joint ( [215,236,247](#)). Much of the discussion of that era centered on the manipulative techniques used to extract the fragment from the joint ( [215,236](#)).

#### Operative/Nonoperative Controversy

In 1950, Smith ( [242](#)) stated that many of the complications previously attributed to this injury were actually misconceptions. Since then, the proponents of uniform nonoperative management ( [205,224,233,252](#)) have far outnumbered the proponents of uniform operative management ( [222](#)). In the interval since the third edition of this volume, little has been written about the treatment of this injury; most of the focus has been on the increased recognition of this injury occurring during arm wrestling ( [227,228,232](#)).

#### Incidence

In the combined series of 5,226 fractures of the distal humerus (see [Chapter 13](#)), the percentage of fractures involving the medial epicondylar apophysis was just behind that of fractures involving the lateral condylar physis. Fractures involving the medial epicondylar apophysis constituted 14.1% of fractures involving the distal humerus and 11.5% of all fractures in the elbow region.

## Age

Fractures involving the epicondylar apophysis have a later peak age, much like that of fractures involving the medial condylar physis. The youngest reported patient with this injury was 3.9 years (206). In the large series of fractures of the medial epicondylar apophysis reported, most occurred between ages 9 and 14, with the peak age incidence being 11 to 12 years (199,217,221,222,225,233,242,251,252,279).

## Sex

Fractures of the epicondylar apophysis affect boys by a ratio of almost 4 to 1. In the six largest series in the literature on this injury, boys constituted 79% of the patients (217,229,234,237,247,252).

## Association with Dislocation of the Elbow

The reported incidence of association with dislocation of the elbow has varied from as low as 30% to as high as 55% in many of the reported series (199,208,213,231,252). Two bilateral injuries associated with bilateral elbow dislocations have been reported (200,246). Both patients sustained their injuries while participating in gymnastics.

In summary, the peak age for fractures of the epicondylar apophysis is 9 to 12 years. The injury occurs in boys four times more often than in girls. About 50% of such injuries are associated with elbow dislocations. In at least 15% to 18%, the fragment is incarcerated in the joint (Table 15-1).

Overall incidence: fractures of the distal humerus, 14.1%  
Overall incidence: fractures of the elbow region, 11.5%  
Age: peak, 11-12 years  
Sex: males, 79% (4:1, male:female)  
Association with elbow dislocation: approximately 50%  
(15%-18% of these involve incarceration of the epicondylar apophysis)

TABLE 15.1. FRACTURES OF THE MEDIAL EPICONDYLAR APOPHYSIS: INCIDENCE

## Surgical Anatomy

### Ossification

**Apophysis.** The medial epicondyle is a traction apophysis, so the term *apophysis* rather than *physis* is used throughout the description of this injury. The forces across its physeal plate are tension rather than the compressive forces present across the condylar physeal plates of the distal humerus. Because it is an apophysis, it does not contribute to the overall length of the distal humerus.

In the early ossification process, the medial epicondylar apophysis is part of the total distal humeral epiphysis. With growth and maturity, it becomes separated from the total distal humeral epiphysis by intervening metaphyseal bone. In younger children, when there is a separation of the total distal humeral epiphysis, the medial epicondylar apophysis is included as part of the distal fragment.

**Posteromedial Location.** The medial epicondylar apophysis actually arises from the posterior surface of the medial distal humeral metaphysis. As was mentioned in Chapter 14, this posterior location is important when percutaneous pin fixation is performed. Likewise, this posterior position affects the image of the apophysis on radiographs (Fig. 15-47).



FIGURE 15-47. Ossification of the medial epicondyle. **A:** The concentric oval nucleus of ossification of the medial epicondylar apophysis (arrow). **B:** As ossification progresses, parallel smooth sclerotic margins develop in each side of the physis. **C:** Because it is somewhat posterior, on a slightly oblique anteroposterior view the apophysis may be hidden behind the distal metaphysis. **D:** The posterior location of the apophysis (arrow) is appreciated on this slightly oblique lateral view. **E:** On the anteroposterior view, the line created by the overlapping of the metaphysis (arrow) can be misinterpreted as a fracture line (pseudofracture).

**Ossification Sequence.** Brodeur and Silberstein (203,240) have described many of the unique aspects of the ossification process of the medial epicondylar apophysis. The following discussion is paraphrased from their work.

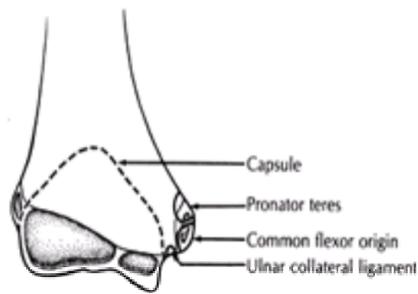
Ossification begins from 4 to 6 years of age, with fusion occurring at about 15 years of age. It is the last secondary ossification center to fuse with the distal humeral metaphysis. The ossification center starts as a small eccentric oval nucleus (Fig. 15-47A). As it matures, parallel sclerotic margins develop along both sides of the physis (Fig. 15-47B). There may be some irregularity of the ossification process, which gives the ossific nucleus a fragmented appearance. This fragmentation may be falsely interpreted as a fracture.

Because the apophysis is posteromedial, the ossification center may be difficult to visualize on the anteroposterior radiograph, especially if the elbow is slightly oblique (Fig. 15-47C). The posterior position of the apophysis is best appreciated on the lateral radiograph. If the elbow is slightly oblique, the outline of the epicondyle may be better appreciated (Fig. 15-47D). Because of this posterior location on the anteroposterior radiograph, the distal medial metaphyseal border may overlap the ossific nucleus of the apophysis. This overlapping may appear as a lucent line that can be misinterpreted as a fracture (Fig. 15-47E).

### Soft Tissue Attachments

**Flexor Mass.** The flexor mass, which includes the origin of the flexor carpi radialis, flexor carpi ulnaris, flexor digitorum superficialis, and palmaris longus and part of

the pronator teres, originates from the anterior aspect of the apophysis ( [Fig. 15-48](#)) (240,250). Part of the flexor carpi ulnaris also originates on the posterior aspect of the epicondyle.

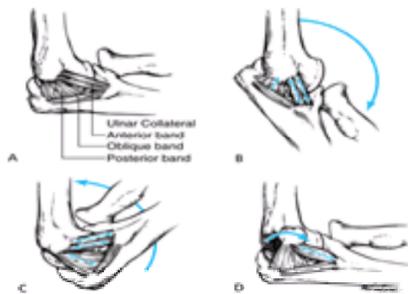


**FIGURE 15-48.** Soft tissue attachments. Anteroposterior view of the distal humerus demonstrating the relationship of the apophysis to the origins of the medial forearm muscles. The origin of the ulnar collateral ligament lies outside the elbow capsule. The margin of the capsule is outlined by the dotted line.

**Capsule.** In the younger child, some of the origin of the capsule extends up to the physal line of the epicondyle. In the older child and adolescent, as the epicondyle migrates more proximally, the capsule is attached only to the medial crista of the trochlea ( [198,212](#)). Thus, in the younger child, the fracture line involving the medial epicondylar apophysis can enter the joint because part of the capsule is attached to the epicondylar fragment. In the older child, however, if there is a pure avulsion force on the epicondyle, the capsule may remain attached to the outer border of the trochlea. In this age group, the fracture may be totally extraarticular.

### Ligamentous Structures

The two major medial collateral ligaments originate from this apophysis. The ulnar collateral ligament is composed of three separate bands ( [Fig. 15-49](#)) (223,254). Woods and Tullos (254) pointed out that the major stabilizing ligamentous structure in the elbow is the anterior band of the ulnar collateral ligament. The anterior portion of the band is taut in extension, and the posterior fibers are taut in flexion. The fibers of the posterior band of the ulnar collateral ligament are relaxed in extension and are taut in flexion ( [Fig. 15-49](#)). Thus, this posterior band provides stability only in flexion. Because the radial collateral ligaments do not attach directly to the ulna or radius, but instead attach to the orbicular ligament ( [239](#)) they provide only minimal stability to the elbow joint.



**FIGURE 15-49.** Ligamentous structures. **A:** The ulnar collateral ligament is divided into anterior, posterior, and oblique bands. **B:** On extension, the anterior fibers of the anterior band are taut. The posterior fibers of the anterior band and the entire posterior band are loose in this position. **C:** In flexion, the posterior fibers of the anterior bands and posterior band become taut. The anterior fibers of the anterior band become loose. **D:** When the epicondyle is rotated anteriorly, the entire anterior band can become loose. (Reprinted from Woods WG, Tullos HS. Elbow instability and medial epicondyle fractures. *Am J Sports Med* 1977;5:23-30; with permission.)

### Mechanism of Injury

#### Acute Injuries

Injuries to the medial epicondylar apophysis most commonly occur as acute injuries in which a distinct event produces a partial or a complete separation of the apophyseal fragment. Three theories have been proposed about the mechanism of acute medial epicondylar apophyseal injuries: a direct blow, avulsion mechanisms, and association with elbow dislocation ( [Table 15-2](#)).

#### Acute injuries

##### Direct blow

##### Avulsion mechanisms

##### Avulsion in elbow extension (valgus stress)

##### Avulsion with elbow flexed (pure muscle forces)

##### Associated with elbow dislocation

##### Chronic tension stress injuries

**TABLE 15.2. FRACTURES OF MEDIAL CONDYLAR APOPHYSIS: MECHANISM OF INJURY**

#### Direct Blow

Stimson (244) speculated that this type of injury could occur as a result of a direct blow on the posterior aspect of the epicondyle. However, among more recent investigators, only Watson-Jones (248) described this injury as being associated with a direct blow to the posterior medial aspect of the elbow.

In those rare patients in whom the fragment is produced by a direct blow to the medial aspect of the joint, the medial epicondylar fragment is often fragmented ( [Fig. 15-50](#)). In these injuries, there also may be more superficial ecchymosis in the skin.



**FIGURE 15-50.** Direct fragmentation. The fragmented appearance of the medial epicondyle ( *arrow*) in a 13-year-old who sustained a direct blow to the medial aspect of the elbow. (Reprinted from Wilkins KE. Fractures of the medial epicondyle in children. *Instr Course Lect* 1991;40:1-8; with permission.)

### Avulsion Mechanisms

Many investigators ascribe to the theory that some of these injuries are due to a pure avulsion of the epicondyle by the flexor muscles of the forearm ([225,235,238,253](#)). This muscle avulsion force can occur in combination with a valgus stress in which the elbow is locked in extension, or as a pure musculature contraction that may occur with the elbow partially flexed.

### Avulsion and Extension (Valgus Stress)

Smith ([242](#)) believes that when the child falls on the outstretched upper extremity with the elbow in extension, the wrist and fingers are often hyperextended as well, placing an added tension force on the epicondyle by the forearm flexor muscles ( [Fig. 15-51](#)). The normal valgus carrying angle tends to accentuate these avulsion forces when the elbow is in extension. Many of the proponents of this theory point to the other associated elbow fractures that have been seen with this injury as evidence to confirm that a valgus force is applied across the elbow at the time of the injury. These associated injuries include fractures of the radial neck with valgus angulation and greenstick valgus fractures of the olecranon ([225,243,253](#)).



**FIGURE 15-51.** Hyperextension forces. When a person falls on the outstretched upper extremity, the wrist and fingers are forced into hyperextension ( *solid arrow*), which places tension on the forearm flexor muscles. This sudden tension along with the normal valgus carrying angle tends to place a strong avulsion force on the medial epicondyle ( *open arrow*).

### Isolated Muscle Avulsions

Isolated avulsion also can occur in adolescents with the simple act of throwing a baseball. In this instance, the sudden contraction of the forearm flexor muscles may be sufficient to cause the epicondyle to fail ( [Fig. 15-52](#)). The literature has reflected a high incidence of medial epicondylar apophyseal avulsions occurring with arm wrestling in patients near skeletal maturity ([226,228](#)). The largest series, reported by Nyska et al. ([232](#)) from Israel, involved eight boys 13 to 15 years of age, all of whom were treated conservatively with good results.



**FIGURE 15-52.** Muscle avulsion. Isolated avulsion of the medial epicondyle occurred suddenly in this 14-year-old Little League pitcher after throwing a curve ball. (Reprinted from Wilkins KE. Fracture of the medial epicondyle in children. *Instr Course Lect* 1991;40:1-8; with permission.)

### Associated with Elbow Dislocations

The final mechanism proposed is that this injury is associated with a dislocation of the elbow in which the ulnar collateral ligament provides the avulsion force. If the elbow is dislocated when the patient is initially seen, there is no doubt that this is a major factor in the production of this fracture. However, the question of this being caused by an occult or partial elbow dislocation that has reduced spontaneously is often raised. Some investigators ([199,200,217](#)) have noticed the development of calcification in the lateral collateral ligaments and adjacent lateral periosteum in elbows postfracture. They believed this was evidence that this ligament had been stretched during the process of elbow dislocation. Marion and Faysse ([229](#)) found that most elbow dislocations associated with this injury were posterolateral, but some pure lateral, posterior, and posteromedial dislocations were also observed.

The question arises as to whether incarceration of the epicondylar fragment into the joint can occur without a dislocation. Patrick ([235](#)) believed that when an extreme valgus stress was applied to the joint, a vacuum was created within the joint that "sucked in" the avulsed epicondylar fragment. This theory has not been universally accepted ([238,241](#)).

Acutely, it appears that any of these mechanisms can produce this injury. The direct blow mechanism appears to occur only rarely. Most of these injuries probably are associated with an elbow dislocation that may or may not have reduced spontaneously.

## Pathology

### Acute Fractures

Usually the epicondyle is displaced distally. Cotton ( 210) described two patients in whom this fragment was displaced proximally. Avulsion usually involves only the apophysis. However, a small flake of metaphyseal bone is occasionally found attached to the apophyseal fragment. Rarely, the fracture line passes through the apophysis, with only part of the apophysis being avulsed ( 240). These partial avulsions may appear as only minor injuries. However, the partial fragment can be incarcerated within the joint just as easily as the full apophysis ( Fig. 15-53).



**FIGURE 15-53.** Partial avulsion. The epicondylar fragment was only partially avulsed. This partial fragment also had an associated portion of the metaphysis ( arrow), both of which were incarcerated into the elbow joint. (Courtesy of Randal R. Betz, M.D.)

### Chronic Incarceration

When the fragment becomes incarcerated into the joint, the raw bone surface may become adherent to the coronoid process of the ulna ( 237). In late cases, this union of the fragment to the coronoid process may make extraction difficult. A universal finding when the fragment is incarcerated within the joint is a thick fascial band that binds the ulnar nerve to the underlying muscle ( 197,210,235). The constriction by this band is believed to be responsible for either the immediate or late dysfunction of the ulnar nerve.

### Associated Injuries

Other elbow fractures that can be associated with this injury include fracture of the radial neck, olecranon, or coronoid process. If the epicondyle fragment is only rotated on its axis, laxity of the anterior band of the ulnar collateral ligament can occur. This can produce some medial elbow instability during extension ( 254).

### Classification

The various classifications proposed for this injury ( 199,229,243,249) are pretty much the same. We have combined them to form a comprehensive classification (Table 15-3) that can be useful in determining the proper method of treatment. In the discussion of this classification, the clinical and radiographic findings are delineated for each type of fracture pattern. Initially, we have separated these injuries into two primary categories based on whether the injuries are acute or chronic.

#### Acute injuries

Undisplaced or minimally displaced

Displaced fractures

Incarcerated fractures (without elbow dislocation)

Incarcerated fractures (with elbow dislocation)

Chronic tension (stress) injuries

**TABLE 15.3. FRACTURES OF THE MEDIAL EPICONDYLE APOPHYSIS: CLASSIFICATION**

### Acute Injuries

**Undisplaced or Minimally Displaced Fractures.** In undisplaced fractures, the physal line remains intact. The clinical manifestations usually consist only of swelling and local tenderness over the medial epicondyle. Crepitus and motion of the epicondyle usually are not present. On radiography, the smoothness of the edge of the physal line remains intact. Although there may be some loss of soft tissue planes medially on the radiograph, displacement of the elbow fat pads may not be present because the pathology is extraarticular ( 220).

Minimally displaced fractures usually result from a stronger avulsion force, so there is often more soft tissue swelling. Palpation of the fragment may elicit crepitus because the increased displacement allows motion of the fragment. On radiography, there is a loss of parallelism of the smooth sclerotic margins of the physis ( Fig. 15-54) (240). The radiolucency in the area of the apophyseal line is usually increased in width.



**FIGURE 15-54.** Minimally displaced. **A:** Anteroposterior view of a minimally displaced fracture. The smooth sclerotic margins of the physis are disrupted. **B:** Uninjured elbow for comparison.

**Significantly Displaced Fractures.** In significantly displaced fractures, there is no question as to whether the fragment is displaced: it may be palpable and freely movable. Because it is displaced a considerable distance from the distal humerus, crepitus between the fragments may not be present. There may have been an elbow dislocation that reduced spontaneously or by manipulation. On the other hand, there may have been no documentation of the original dislocation. On radiography, the long axis of the epicondylar epiphysis is rotated medially ( [Fig. 15-55](#)). The displacement usually exceeds 5 mm, but the fragment remains proximal to the true joint surface. This fragment may contain a metaphyseal fragment.



**FIGURE 15-55.** Significantly displaced. Anteroposterior view of an elbow in which the epicondyle ( *arrow*) is significantly displaced both distally and medially. In addition, the fragment is rotated medially.

**Entrapment of the Epicondylar Fragment into the Joint. Without Elbow Dislocation.** In many instances the elbow appears reduced. The key clinical finding is a block to motion, especially extension. The epicondylar fragment is usually between the joint surfaces of the trochlea and the semilunar notch of the olecranon. On radiography, any time the fragment appears at the level of the joint, it must be considered to be totally or partially within the elbow joint until proven otherwise ( [Fig. 15-56](#)) (235). If the radiograph is examined carefully, the elbow is usually still found to be incompletely reduced. Because of an impingement of the fragment within the joint, a good anteroposterior view may be difficult to obtain due to the inability to extend the elbow fully. If the fracture is old and the fragment is fused to the coronoid process, widening of the medial joint space may be the only clue that the fragment is lying in the joint ( 197). The epicondylar ossification center may become fragmented and mistaken for the fragmented appearance of the medial crista of the trochlea ( 206,237,241). Absence of the apophyseal center on radiographs may be further confirmatory evidence that the fragment is within the joint. Comparison radiographs of the opposite elbow may be necessary to delineate the true pathology.



**FIGURE 15-56.** Incarceration within the joint. **A:** The clues to entrapment are complete absence of the epicondyle from its usual medial location ( *open arrow*) and its subsequent location at the level of the joint ( *closed arrow*). **B:** On the lateral view the outline of the epicondyle is hidden by the overlying olecranon ( *closed arrow*). The fragment also contains a small portion of the metaphysis ( *open arrow*).

**With Elbow Dislocation.** Even if the elbow is dislocated, the fragment can still lie within the joint and prevent reduction. Recognition of this fragment as being within the joint before a manipulation should alert the physician of the possible need for open reduction. There should be adequate relaxation during the manipulative process. An initial manipulation to extract the fragment from the elbow joint may need to be accomplished first before a satisfactory closed reduction of the elbow joint can be obtained ( [Fig. 15-57](#)).



**FIGURE 15-57.** Dislocation with incarceration. **A:** Anteroposterior view showing a posterolateral dislocation of the elbow. The presence of the medial epicondyle within the elbow joint ( *arrow*) prevented a closed reduction. **B:** Lateral view of the same elbow demonstrating the fragment ( *arrow*) between the humerus and olecranon.

**Fractures Through the Epicondylar Apophysis.** Fractures through the body of the epicondyle can result from either a direct blow or avulsion of only part of the apophysis. In either case, the fragments may or may not be displaced. The normal lucent line formed by the overlying metaphyseal border should not be confused with this injury. Although described by Silberstein and colleagues ( 240), this intrafragment fracture is a rare presentation.

### Diagnosis

#### Clinical Findings

**Valgus Stress Test.** Because many of the clinical diagnostic points have been discussed in the previous section on the classification of this injury, much of the emphasis in this section is on the determination of elbow instability. Because the anterior oblique band of the ulnar collateral ligament may be attached to the medial epicondylar apophysis, the elbow may exhibit some instability postinjury. To evaluate the medial stability of the elbow, a simple valgus stress has been advocated by Woods and Tullos (254) and Schwab et al. (239). This test is performed with the patient supine and the arm abducted 90 degrees. The shoulder and arm are externally rotated 90 degrees. The elbow must be flexed at least 15 degrees to eliminate the stabilizing force of the olecranon. If the elbow is unstable, simple gravity forces will open the medial side. A small additional weight or sedation may be necessary to get an accurate assessment of the medial stability with this test. The

practicality of this valgus stress test is discussed in more detail in the forthcoming section on the AUTHORS' Preferred Method of Treatment.

**Evaluate Ulnar Nerve.** The function of the ulnar nerve must be carefully assessed. It is especially wise to document the presence or absence of an ulnar nerve injury before instituting therapy.

### X-Ray Findings

**Beware Absent Apophysis.** Widening or irregularity of the apophyseal line may be the only clue in fractures that are only slightly displaced or nondisplaced. If the fragment is significantly displaced, the radiographic diagnosis is usually obvious. However, if the fragment is totally incarcerated in the joint, it may be hidden by the overlying ulnar or distal humerus. The clue here is the total absence of the epicondyle from its normal position just medial to the medial metaphysis.

Potter (76) suggested that properly performed MRI may disclose acute or chronic injury to the medial epicondylar apophysis. Recommended pulse sequences for evaluation of the apophysis include fat-suppressed gradient echo imaging. On MRI, increased signal intensity and abnormal widening of the medial epicondylar physis are seen, typically with surrounding soft tissue edema.

**Fat Pad Signs Unreliable.** Fractures of the medial epicondyle, even if displaced, may not produce positive fat pad signs (220,240). If the fracture is only minimally displaced and the result of an avulsion injury, there may be no effusion because all the injured tissues remain extraarticular. In those associated with elbow dislocation, there is rupture of the capsule, so its ability to confine the hemarthrosis is lost. In minimally displaced fractures of the medial epicondyle with significant hemarthrosis, one must be especially thorough in the evaluation to ensure that an unrecognized fracture involving the medial condylar physis is not present.

### Differential Diagnosis

The major injury to differentiate is one involving the medial condylar physis. This is especially true if the secondary ossification centers are not present (see earlier section on [Fractures Involving the Medial Condylar Physis](#)). If there is a significant hemarthrosis or a significant piece of metaphyseal bone accompanying the medial epicondylar fragment, then arthrography may be indicated to determine if there is an intraarticular component to the fracture ( [Fig. 15-58](#)).



**FIGURE 15-58.** Intraarticular extension. **A:** Injury film in this 7-year-old girl who was initially suspected of having only a fracture of the medial epicondyle. In addition to moderate displacement, there was a significant metaphyseal fragment (*arrow*). **B:** An arthrogram reveals intraarticular components (*arrow*), which define this instead as a fracture involving the medial condylar physis. (Courtesy of Carl McGarey, M.D.)

### Treatment

#### Areas of General Agreement

There seems to be universal agreement as to the proper method of treating fractures that are undisplaced or only minimally displaced. These first two types are easily treated with simple immobilization for comfort. Many investigators have recommended initiation of motion early to prevent stiffness, the most common complication of this injury (200,242). Likewise, if the fragment is incarcerated in the joint, the accepted treatment is to extract the fragment from the joint by manipulation or surgical intervention.

The controversy seems to be in determining the proper method of treatment for patients with significant displacement (the fragment is displaced >5 mm).

#### Nonoperative Management

##### Operative Versus Nonoperative Controversy

**Operative Results No Better.** In reports in which only one method (i.e., operative or nonoperative) of treatment of all displaced fractures was used, one can find results to support either method. The best argument for surgical management comes from Hines et al. (222) in Oklahoma, where the practice was to surgically repair all fractures displaced more than 2 mm. They found that 96% of their patients had good to excellent results. Bad results were attributed mainly to technical errors. Equal results were obtained by Joseffson and Danielsson (224) from Sweden, where these injuries were all treated nonoperatively. Although more than 60% of their patients demonstrated radiographic nonunion, they had an equal number of good results at a mean follow-up of 11 years. Many more reports in the literature (211,233,252) also demonstrate the overall good results with nonoperative management.

**Comparison Studies.** The best comparison of the results of operative versus nonoperative treatment comes from reports in which both methods were practiced in the same institution. Bede et al. (199) had superior results in patients treated nonoperatively compared with those treated operatively. This same superiority of nonoperative management has been found in subsequent reports as well (200,217,252).

**Results Poorer with Dislocation.** The results of fractures associated with a documented elbow dislocation are poorer (199,217) for patients treated operatively and nonoperatively. Fowles and colleagues (217) wrote that surgical intervention only added to the original trauma produced by the dislocation, increasing the residual loss of motion.

**Indications for Operative Intervention.** The indications for operative intervention in acute injuries are divided into two categories: absolute and relative. The single absolute indication is incarceration of the epicondylar fragment within the joint. The relative indications include ulnar nerve dysfunction and a need for elbow stability.

**Incarceration in the Joint—Absolute.** If the fragment is found in the joint acutely, it must be removed. However, there are proponents of both nonoperative and operative techniques for extracting the fragment.

**Nonoperative Extraction.** Various methods of extracting the fragment by nonoperative methods have been proposed. The success rate of extracting the fragment successfully from the joint by manipulation alone is only about 40% (235). All the nonoperative methods require either heavy sedation or light general anesthesia.

**Roberts' Manipulative Technique.** The manipulative technique most commonly used is the one popularized by Roberts (236) which has gained wide acceptance by many other investigators (202,214,215,221,236,238,249). It involves placing a valgus stress on the elbow while supinating the forearm and simultaneously dorsiflexing the wrist and fingers to place the forearm muscles on stretch; theoretically, this should extract the fragment from the joint. To be effective, this procedure must be performed within the first 24 hours after injury (202,238)

*Faradic Stimulation.* Patrick (235) recommended stimulating the forearm muscles with faradic electric current to supplement the manual manipulative process.

*Joint Distention.* Fowles et al. (217) and Masse (230) found that if manipulation fails to dislodge the fragment from the joint, distending it with air or fluid might facilitate the extraction process.

**Operative Extraction.** Failure to extract the fragment by manipulative techniques is an indication to proceed with open surgical extraction. Once open extraction and reduction have been performed, many methods have been advocated to stabilize the fragment. The simplest appears to be the use of sutures (221). Excision also has been advocated, especially if the fragment is comminuted (197,202,245).

#### **Incarceration Discovered Late.**

*Original Opinions—No Treatment.* If the fragment has been lodged in the joint for more than 4 weeks, the results with late removal have been poor. Patrick (235) found that by 4 weeks, the fragment had become fused to the articular surface of the coronoid process. Forceful removal of the fragment after this period of time produced a raw defect in the articular surface of the coronoid process. This defect subsequently scarified the opposing trochlear articular surface. In addition, the long-term presence of the fragment in the joint subluxates the joint and stretches the capsule and ligaments. If the fragment is removed after these ligaments and capsules have undergone secondary changes, elbow stability may be lost. Blount (201) recommended that these fractures with the fragment incarcerated in the joint be left untreated if more than 6 weeks has passed since the original injury.

*More Recent Views—Removal Recommended.* The opinion that surgery is detrimental in patients with late incarceration has been challenged by the more recent studies of Fowles et al. from Tunisia (216). In their patients in whom the fragment was surgically extracted an average of 14 weeks after injury, 80% more elbow motion was regained. In addition, the patients' preoperative pain was relieved and the ulnar dysfunction resolved. On a long-term basis, intraarticular retention of the fragment may not be all that disabling. Rosendahl (237) reported an 8-year follow-up of a fragment retained within the joint. The epicondyle had fused to the semilunar surface of the ulna, producing a large bony prominence clinically. There was only minor loss of elbow motion, with little functional disability.

#### **Ulnar Nerve Dysfunction—Relative**

##### **Acute Management**

Ulnar nerve dysfunction is a relative indication for operative intervention. If there are mild to moderate ulnar nerve symptoms at the time of the injury, usually there is no need to explore the nerve, because most of these mild symptoms resolve spontaneously (200,211). If the dysfunction is complete, then the ulnar nerve is probably wrapped around the fragment and must be explored surgically. One of the original fears was that the raw surface of the fracture fragment would create scar tissue around or adjacent to the nerve and cause continued dysfunction. Thus, originally it was recommended that the ulnar nerve be transposed at the time of open reduction (221,243,248). Subsequent reports have not found this to be necessary (207,246).

##### **Delayed Dysfunction Nonexistent**

There is some question as to whether delayed ulnar nerve symptoms can even occur after fractures of the epicondyle that are not associated with dislocation of the elbow. Patrick (235) could not find any instance in which a delayed ulnar neuritis developed in a review of over 100 patients with uncomplicated fractures involving the medial epicondylar apophysis. He found some patients with late ulnar neuritis in fractures that were associated with dislocation of the elbow. Bernstein et al. (200,211) found that their patients with initial ulnar nerve symptoms all did well when treated nonoperatively. Thus, the original fear of delayed ulnar nerve dysfunction has been dispelled.

##### **Joint Stability—Relative**

Woods and Tullos (254) believe that athletes can exhibit a significant disability if they have even minor forms of valgus instability after elbow injuries involving the medial epicondylar apophysis. This is especially true in athletes who must have a stable upper extremity, such as baseball pitchers, gymnasts, or wrestlers. In the younger person (<14 years of age), the anterior band of the ulnar collateral ligament often displaces with the apophyseal fragment. In older individuals (15 years or older), large fragments may be avulsed without a ligamentous injury. Rather than depending on arbitrary measurements of fracture displacement, Woods and Tullos (254) recommended the use of the gravity valgus stress test to determine the presence or absence of valgus instability. They believed that demonstration of a significant valgus instability, using this simple gravity test, was an indication for surgical intervention in patients who require a stable elbow for their athletic activities. Pimpalnerkar et al. (146) also suggested that clinical evidence of instability, as shown by gravity valgus stress testing, is an indication for operative fixation.

## **AUTHORS' PREFERRED METHOD OF TREATMENT**

We use the classification scheme (Table 15-3) as a rough guide regarding our treatment options. We also strongly consider the expected activity of the involved extremity in deciding on nonoperative versus operative treatment (Table 15-4).

- Nonoperative treatment
  - Nondisplaced, minimally displaced
  - Significantly displaced in patients with low-demand upper extremity function
- Operative treatment
  - Absolute: irreducible incarcerated fragment in the elbow joint
  - Relative: ulnar nerve dysfunction
  - Relative: patient with high-demand upper extremity function

**TABLE 15.4. AUTHOR'S RECOMMENDED TREATMENT**

#### **Basically a Nonoperative Fracture**

For most uncomplicated fractures, regardless of the number of millimeters of displacement, we prefer nonoperative management (Fig. 15-59). This includes fractures associated with elbow dislocations. The parents are warned that regardless of the type of treatment, some loss of elbow extension may occur. They should be reassured, however, that this loss of motion, if it does occur, is usually not of any functional or cosmetic significance. The elbow is immobilized initially with a removable posterior splint, used mainly for comfort and some support.



**FIGURE 15-59.** Nonoperative management. **A:** Postreduction film of a 13-year-old girl who sustained a displaced medial epicondyle following an acute elbow dislocation in her nondominant extremity. She was treated nonoperatively. **B:** One year later the fragment has remained distally displaced with an apparent fibrous union. The patient, however, had a full painless range of elbow motion. (Reprinted from Wilkins KE. Fractures of the medial epicondyle in children. *Instr Course Lect* 1991;40:1-8; with permission.)

### Early Motion Essential

Because stiffness is the most common complication of this injury, we encourage early active motion. The patient is encouraged to remove the splint and start active motion as soon as 3 to 4 days postinjury. The splint is exchanged for a sling as soon as the patient feels he or she no longer needs it for support. The same goes for the sling: it also is discarded when it is no longer needed. This regimen of early motion also is used in fractures associated with a documented dislocation. Redislocation after elbow dislocation is rare, but elbow stiffness is common, so it is more important to initiate motion as soon as possible after reduction of the elbow. Because of the greater amount of soft tissue injury associated with an elbow dislocation, the patient may not feel comfortable initiating early motion until about 5 to 7 days postreduction.

### Avoid Early Aggressive Physical Therapy

Physical therapy should be used only if voluntary active motion is difficult to obtain. The therapist should emphasize modalities designed to decrease swelling and pain and reestablish strength. Range of motion should be achieved only by active means, not by passive stretching.

### Operative Indications

Our indications for operative intervention are basically twofold. First and foremost are fractures in which the fragments cannot be extracted by manipulative means from within the elbow joint. Secondly, we stabilize the epicondyles operatively in patients whose expected high-level physical activity requires a stable elbow. We realize, however, that it is difficult to predict the athletic or work potential of a young child.

### Acute Incarceration in the Joint

If the elbow is reduced and the ulnar nerve is intact, we use the manipulative technique advocated by Roberts (638) to attempt to extract the fragment before reduction. If this technique fails to remove the fragment, or if there is any ulnar nerve dysfunction, we proceed directly with an open procedure. The fragment is then extracted under direct vision just before reducing the elbow dislocation. If the elbow is reduced and the fragment is incarcerated, we avoid the initial manipulation and proceed directly with an open extraction. We usually stabilize these fractures with a single threaded screw rather than wires or pins ( [Fig. 15-60](#)). This allows almost immediate motion.



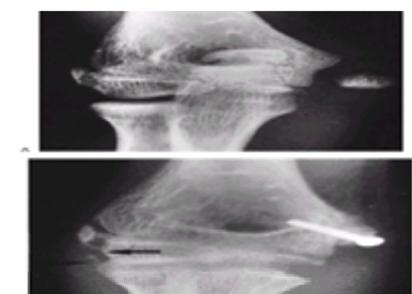
**FIGURE 15-60.** Pin fixation. Stabilization with pins, although adequate for stability, is unsatisfactory because it prevents early motion, which is essential for full recovery.

Follow-up is essentially the same as with closed treatment. Active motion is initiated 5 to 7 days postoperatively.

### Prevention of Valgus Instability

#### Primarily for High-Demand Athletes

Probably our most common indication for operative intervention is to ensure a stable elbow in patients participating in high-demand activities with their upper extremity ([Fig. 15-61](#)). This usually involves the dominant extremity of baseball pitchers, tennis players, or football quarterbacks. In wrestlers and gymnasts, the stability of the nondominant extremity also must be considered. This is best achieved with operative fixation.



**FIGURE 15-61.** Operative stabilization. **A:** Injury film in a 12-year-old gymnast. Even though this was a nondominant extremity, it was thought that both elbows needed stability. **B:** Radiographs taken 4 weeks postoperatively showing stabilization of the fragment with a single screw. There was also calcification of the lateral ligaments (*arrow*), confirming that the elbow was probably originally dislocated as well. (Reprinted from Wilkins KE. Fractures of the medial epicondyle in children.)

### **Valgus Stress Test Impractical**

We have not found the valgus stress test to be helpful in deciding on the need for operative stabilization of an athlete's medial epicondyle. Almost all of these patients with any significant displacement have a positive valgus stress test. Our decision is based primarily on the patient's need to have a very stable elbow for his or her athletic or work activity.

### **Stabilize for Early Motion**

Fixation must be stable enough to allow early motion. Pins provide stability but do not allow early motion. Fortunately, most patients are mature enough so that the fragment can be secured with a threaded screw. If a fully threaded screw is used, it is overdrilled in the epicondylar fragment to allow compression.

### **Operative Technique**

Our operative technique involves a direct medial approach to the fracture site. We make a longitudinal incision just anterior to the medial epicondyle. The fragment is usually displaced distally and anteriorly. The periosteum is removed from the fracture site and the clot is extracted by irrigation. It is important to identify and protect the ulnar nerve, but a complete dissection of the nerve usually is unnecessary. A small towel clip is used to reduce the fracture while the elbow is flexed and the forearm is pronated. Again, the medial epicondyle is normally situated posteriorly. The fragment is reduced and stabilized temporarily with one or two small Kirschner wires. The final fixation is achieved using a screw, either partially threaded and overdrilled in the epicondylar fragment to compress it against the metaphysis or a cannulated 4.0-mm screw. After removal of the Kirschner wires, the elbow is checked to ensure valgus stability and reestablishment of a full range of motion. After closure of the surgical incision, the extremity is placed in a long arm cast, which is bivalved at 7 to 10 days, and active motion is initiated.

### **Fragmented Apophysis**

If the epicondyle is fragmented and there is a need to achieve elbow stability, an ASIF spike washer can be used to secure the multiple pieces to the metaphysis. This may entail a second procedure to remove the spike washer once the epicondyle is securely united to the metaphysis. If this is impossible, we simply excise the fragments and reattach the ligament to the bone and periosteum at the base of the epicondylar defect.

### **Complications**

Although much has been written about fractures involving the medial epicondylar apophysis, few complications are attributed to the fracture itself. The major complications that result in loss of function are failure to recognize incarceration in the joint and ulnar or medial nerve dysfunction. Most of the other complications are minor and result in only minimal functional or cosmetic sequelae ([Table 15-5](#)).

#### Major

- Failure to recognize incarceration in the elbow
- Ulnar nerve dysfunction

#### Minor

- Loss of elbow extension
- Myositis ossificans
- Calcification of the collateral ligaments
- Loss of motion
- Cosmetic effects
- Nonunion in the high-performance athlete

**TABLE 15.5. FRACTURES OF THE MEDIAL EPICONDYLAR APOPHYSIS: COMPLICATIONS**

### **Major Complications**

**Failure to Recognize Incarceration.** Failure to recognize incarceration of the epicondylar fragment into the joint can result in significant loss of elbow motion, especially if it remains incarcerated for any length of time ([Fig. 15-62](#)). The management for late incarceration was detailed in the previous section on treatment.



**FIGURE 15-62.** Late incarceration. **A:** Anteroposterior radiograph of a 12-year-old who had unrecognized incarceration of the medial epicondyle. There was also some ulnar nerve dysfunction. **B:** Lateral view shows only a faint overlay (*open arrows*) of the epicondylar fragment. The fragment was extracted late. Normal motion was never regained.

**Ulnar Nerve Dysfunction.** The other major complication associated with this injury is the development of ulnar nerve dysfunction. The incidence of ulnar nerve dysfunction varies from 10% to 16% ([199](#), [299](#)). If the fragment is entrapped in the joint, the incidence of ulnar nerve dysfunction may be as high as 50% ([197](#)).

The incidence of delayed ulnar nerve neuritis is low. More profound ulnar nerve injury has been reported after manipulative procedures ([235](#)). Thus, in patients with incarcerated fragments in the joint, manipulation may not be the procedure of choice if a primary ulnar nerve dysfunction is present. Patients in whom the fragment was left incarcerated in the joint for a significant time have experienced poor recovery of the primary ulnar nerve injury ([229](#)).

**Dysfunction.** Although the ulnar nerve is the major nerve injured, the median nerve may be encased between a bony fragment and the distal humerus ([181](#)). It is speculated that the nerve can be entrapped between the apophyseal fragment and the distal humerus at the time of the original injury. This type of injury is described in greater detail in the section on complications of elbow dislocations.

**Minor Complications.** Other complications are minimal in nature. Nonunion of the fragment with the distal metaphysis occurs in up to 50% of fractures with significant displacement (199). This appears to be more of a radiographic problem than a functional problem.

Another common problem is loss of the final degrees of elbow extension. A loss of 5% to 10% can be expected to develop in about 20% of these fractures (243). Little functional deficit is attributed to this loss of elbow dysfunction. Prolonged immobilization seems to be the key factor in loss of elbow extension. Again, it is important to emphasize before treatment is begun that loss of motion is common after this injury, regardless of the method of treatment used.

Myositis ossificans has been described as a rare occurrence following vigorous and repeated manipulation to extract the fragment from the joint (212). As with many other elbow injuries, myositis may be a result of the treatment rather than the injury itself.

Myositis ossificans must be differentiated from ectopic calcification of the collateral ligaments, which involves only the ligamentous structures. This may occur after repeated injuries to the epicondyle and ligamentous structures (Fig. 15-63). Often this calcified ligament is asymptomatic and does not seem to create functional disability.



**FIGURE 15-63.** Heterotopic calcification. **A:** Injury to an 11-year-old who had moderate displacement of the medial epicondyle (arrow). **B:** One year later she had considerable calcification of the ulnar collateral ligament (arrow). Other than mild instability with valgus stress, she had full range of motion and was asymptomatic. (Courtesy of Mark R. Christofersen, M.D.)

The cosmetic effects are minimal. In some patients, an accentuation of the medial prominence of the epicondyle creates a false appearance of an increased carrying angle of the elbow (251). Smith (242), in his extensive review, recognized only a slight decrease in the carrying angle in two patients.

Nonunion in the high-performance athlete may be difficult to treat. One of the authors had as a patient a high-performance adolescent baseball pitcher who had to stop pitching after nonoperative management of a medial epicondyle fracture. The patient had developed a fibrous nonunion (Fig. 15-64). Attempts to establish union surgically were unsuccessful. The patient continued playing baseball but had to change to a position in the outfield.



**FIGURE 15-64.** Nonunion in an athlete. This 15-year-old baseball pitcher had an untreated medial epicondyle fracture 1 year before this radiograph. He developed a fibrous union, but the epicondyle was shifted distally (arrow). His elbow was unstable enough to prevent him from pitching.

## Fractures Involving the Lateral Epicondylar Apophysis

### Incidence

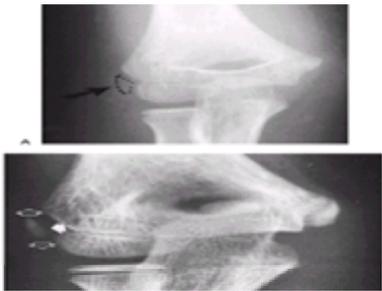
Fracture of the lateral epicondylar apophysis is a rare injury. In the review of 14 reports (see Chapter 13) discussing 5,226 fractures of the distal humerus, only fracture of the lateral epicondylar apophysis was mentioned (663). Five other isolated injuries have been described in textbooks addressing fractures in children (666,668,669).

### Anatomic Considerations

Because the presence of this apophysis is often misinterpreted as a small chip fracture, a thorough understanding of the anatomy and ossification process is essential for evaluating injuries in this area.

### Late Ossification

The lateral epicondylar apophysis is present for a considerable period but does not become ossified until the second decade. The best discussion of the anatomy of the ossification process is in a report by Silberstein et al. (261), and much of the following discussion is paraphrased from their work. Just before ossification of the apophysis, the ossification margin of the lateral supracondylar ridge of the distal metaphysis curves abruptly medially toward the lateral condylar physis (Fig. 15-65). This causes the osseous borders on the lateral aspect of the distal humerus to take the shape of the number 3. The central wedge of this defect contains the cartilaginous lateral epicondylar apophysis, which begins to ossify around 10 to 11 years of age. Ossification begins at the level of the lateral condylar physal line and proceeds proximally and distally to form a triangle, with the apex directed toward the physal line. The shape of the epicondylar apophyseal ossification center also may be in the form of a long sliver of bone with an irregular pattern of ossification.



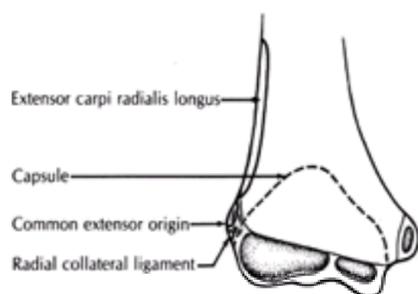
**FIGURE 15-65.** Lateral epicondylar apophysis. **A:** The cartilaginous apophysis occupies the wedge-shaped defect at the margin of the lateral condyle and metaphysis (*arrow*). The dotted line shows the margin of the cartilaginous apophysis. **B:** Ossification of the apophysis begins at the central portion of the wedge defect (*solid arrow*) and progresses both proximally and distally (*open arrows*) to form a triangular center.

### Part of Lateral Condyle

Silberstein et al. (261) noted that the fracture line involving the lateral condylar physis often involves the proximal physeal line of the lateral epicondylar apophysis. Thus, this apophysis is almost always included with the lateral condylar fragment.

### Mechanism of Injury

In adults, the most common etiology is that of a direct blow to the lateral side of the elbow. In children, because the forearm extensor muscles originate from this area, it is believed that avulsion forces from these muscles can be responsible for some of these injuries (260). Hasner and Husby (256) believe that the location of the fracture line in relation to the origins of the various extensor muscles determines the degree of displacement that can occur (Fig. 15-66). If the proximal part of the fracture line lies between the origin of the common extensors and the extensor carpi radialis longus, usually there is little displacement. If the fracture lines enter the area of origin of the extensor carpi radialis longus, then considerable displacement can occur.



**FIGURE 15-66.** Soft tissue attachments. The origins of the forearm and wrist extensor muscles, radial collateral ligament, and outline of the capsule are demonstrated in relation to the lateral epicondylar apophysis. (Reprinted from Hasner E, Husby J. Fracture of the epicondyle and condyle of humerus. *Acta Chir Scand* 1951;101:195-202; with permission.)

### X-Ray Findings

Because the ossification process starts on the external surface of the apophysis and proceeds centrally, the ossification center often appears separated from the lateral metaphysis and lateral condylar epiphysis. This natural separation can be confused with an avulsion fracture. The key to determining true separation is looking beyond the osseous tissues for the presence of associated soft tissue swelling (Fig. 15-67). If the ossification center lies distal to the osteochondral border of the lateral condylar epiphysis, it should be considered displaced (Fig. 15-68).



**FIGURE 15-67.** Lateral swelling. **A:** Soft tissue swelling in the area of the lateral epicondylar apophysis (*arrow*) should make one suspect that this is an undisplaced fracture involving the apophysis. The fragmentation of the apophysis is due to irregular ossification. **B:** A small avulsion of the lateral epicondyle (*open arrow*) in an adolescent who is almost skeletally mature. There was considerable soft tissue swelling in this area (*solid arrows*).



**FIGURE 15-68.** Avulsion injury. **A:** Avulsion of a portion of the lateral epicondyle in an adolescent (*arrow*). The fragment is at the level of the joint. Most of the epicondyle has fused to the condyle. **B:** Appearance 9 months later showing fragmentation and partial union of the fragment. (Courtesy of R. Chandrasekharan, M.D.).

## Treatment

Unless the fragment is incarcerated within the joint (257), treatment usually consists of simple immobilization for comfort. Nonunion of the fragment has been reported (255, 258). Even with this radiographic finding, the resultant elbow function has been described as being quite good.

## Complications

Only one rare major complication has been described with fractures involving the lateral epicondylar apophysis: entrapment of the fragment, either within the elbow joint (257) or between the capitellum and the radial head (264).

## Chronic Tension Stress Injuries (Little League Elbow)

This chronic injury is related to overuse in skeletally immature baseball pitchers. The original radiographic findings were described by Brogdon and Crow in 1960 (204). Later, Adams (195) demonstrated that the radiographic changes were due to excessive throwing and emphasized the need for preventive programs. This injury is thought to be due to excessive tension on the medial epicondyle with secondary tendinitis. There also can be a repeated compression on the lateral condyle, producing an osteochondritis.

Studies have shown that as long as the rules outlined by the Little League are followed (i.e., two innings per game, six innings per week), the incidence of these chronic tension stress injuries is fairly low (218,219,226). Most of the problems arise when overzealous parents and coaches require excessive pitching preseason and at home between practices. Albright (196) found a greater incidence in pitchers who had improper pitching techniques. The spectrum of these chronic injuries is outlined in Table 15-6.

Stress fracture of the epicondylar physis  
Calcification of the ulnar collateral ligaments  
Hypertrophy of the medial epicondyle  
Acceleration of growth maturity with generalized synovitis and stiffness  
Osteochondritis of the lateral condyle

**TABLE 15.6. SPECTRUM OF CHRONIC TENSION STRESS INJURIES OF MEDIAL EPICONDYLAR APOPHYSIS**

In chronic tension stress injuries (Little League elbow syndrome), the history is usually quite characteristic. It is found in young baseball pitchers who are throwing an excessive number of pitches or who are just starting to throw curve pitches (204,234). Clinically, this syndrome is manifested by a decrease in elbow extension. Medial epicondylar pain is accentuated by a valgus stress to the elbow in extension. There is usually significant local tenderness and swelling over the medial epicondyle.

On radiographs, the density of the bone of the distal humerus is increased due to the chronicity of the stress. The physal line is irregular and widened (Fig. 15-69). If the stress has been going on for a prolonged period, there may be hypertrophy of the distal humerus with acceleration of bone growth. The bone age of the elbow is greater than the patient's chronologic age (Fig. 15-70).



**FIGURE 15-69.** Acute tension stress. **A:** Anteroposterior view of the pitching arm of a 10-year-old with medial epicondylar pain. The apophyseal line is widened and slightly irregular. **B:** Same view of the opposite elbow for comparison.



**FIGURE 15-70.** Chronic tension stress. **A:** Anteroposterior view of a 13-year-old pitcher with chronic pain and significant loss of elbow motion. The bone age is around 15 years. **B:** Same view of the opposite elbow with a bone age of 13 years.

We use a multifaceted approach that involves education of the parents, coaches, and player. Once symptoms develop, all pitching activity must cease until the epicondyle and adjacent flexor muscle origins become nontender. In addition, local and systemic measures to decrease the inflammatory response are used. Once the initial pain and inflammation have decreased, a program of forearm and arm muscle strengthening is initiated. The pitching technique is also examined to see if any corrections need to be made. Once strength has been reestablished in the muscles in the upper extremity and motion has been fully reestablished, the patient is gradually returned to pitching. This is done on a gradual basis, carefully monitoring the number of innings and pitches within a specified period of time.

## AVASCULAR NECROSIS OF THE TROCHLEA

### Definition

This term is used to define three different pathologies characterized by a disturbance of the growth process of the centers of ossification of the trochlea. These are classified as idiopathic, congenital, and posttraumatic.

### *Idiopathic*

Hagemann's disease (aseptic osteonecrosis of the humeral trochlea, or osteochondritis of the humeral trochlea, or osteochondrosis of the humeral trochlea) is avascular necrosis of the trochlea that appears with no prior history of trauma ( [268](#)).

### *Congenital*

There are two reports of seemingly congenital forms of trochlear hypoplasia. In a report of Japanese patients ( [279](#)), the hypoplasia was bilateral, with symptoms present since early childhood. Another report ( [276](#)) involved an African-American family in which the mother and three offspring had severe bilateral forms of aplasia of the trochlea.

### *Posttraumatic*

This most common form follows some type of elbow trauma. In some cases, the trauma is occult or poorly defined. This form results in a spectrum from simply a small defect of the trochlea (fishtail deformity) to complete destruction of the medial aspect of the distal humerus with a progressive axial deformity, decreased range of motion, and associated disabling instability of the elbow.

### Historical Perspective

#### *Idiopathic Cases (Hagemann's Disease)*

Osteonecrosis of the trochlea was first described in 1933 by Uhrmacher ( [284](#)) in a 7-year-old patient. Almost 20 years after this initial description, Hagemann ( [268](#)) published his classic article on the idiopathic nature of this type of avascular necrosis. Thus, the idiopathic form has become known, at least in the European literature, as Hagemann's disease ( [263](#)). A close investigation of many of the cases of so-called Hagemann's disease, however, reveals that there was usually some antecedent elbow trauma that may or may not have contributed to the development of the avascular necrosis ( [263](#)).

#### *Trauma-Related Cases*

The first description of avascular necrosis of the trochlea following elbow trauma was in 1948 by McDonnell and Wilson ( [274](#)). In their series, the problem occurred as a complication of both lateral condylar and supracondylar fractures. Seven years later, Wilson ( [288](#)) described the typical fishtail deformity occurring after some fractures of the lateral condylar physis. There was little mention as to the consequence or etiology of this complication in these articles. Such was also the case in other articles in which avascular necrosis of the trochlea can be seen in the clinical photographs in many of the cases illustrated ( [264](#), [265](#), [269](#), [271](#), [272](#) and [273](#), [285](#), [286](#), [287](#) and [288](#)). Usually there was little or no mention of the significance of this finding in the captions.

#### *Vascular Etiology*

Not until 1984, when Morrissey and Wilkins ( [278](#)) reported five cases of avascular necrosis of the trochlea, was its relationship to the unique blood supply of this area suggested. They pointed out that according to the classic articles of Haraldsson ( [266](#), [267](#)) on the blood supply of the distal humerus, the medial crista of the trochlea has two separate blood supplies that can be injured with trauma. This relationship of the unique blood supply to the various patterns of avascular necrosis is discussed in detail in the following sections relating to etiology and the patterns of necrosis.

A more recent article by Yoo and co-workers ( [290](#)) also relates avascular necrosis to a vascular etiology with their observation that it occurred with a higher incidence in their cases of total distal humeral physal fractures.

### Incidence

#### *Seldom Reported*

Because it is seldom reported in the literature, the exact incidence is unknown. As mentioned previously, many cases are illustrated in various reports with little or no mention of its occurrence or significance. We recently reported on a series of 30 cases collected over the past 20 years from various sources ( [283](#)). It is also our experience that it is probably one of the most unrecognized sequela of injuries to the distal humerus.

#### *Trauma to Distal Humerus Most Common Cause*

It has been described as a complication of almost any fracture type to the distal humerus, except for isolated epicondylar injuries. Avascular necrosis of the trochlea has been reported as a consequence of the following injuries.

#### *Distal Fractures of the Lateral Condylar Physis*

The term *fishtail deformity* was coined in the classic article on lateral condylar fractures by Wilson published in 1955 ( [288](#)). This term, which usually represents a localized avascular necrosis of the trochlea, has subsequently been used by others in their reports on fractures of the lateral condyle ( [275](#), [278](#), [286](#), [288](#), [297](#)).

#### *Fractures of the Medial Condylar Physis*

This type of fracture is especially vulnerable to developing avascular necrosis of the trochlea because of the close proximity of the fracture line to the blood supply of the trochlea ( [264](#), [270](#), [273](#), [280](#), [285](#)). It can occur in cases treated either operatively or nonoperatively. Operative management of this fracture can increase the incidence or extent of the avascular necrosis by iatrogenic injury to the most medial blood supply to the trochlea, which is extraarticular.

#### *Supracondylar Fractures*

Avascular necrosis of the trochlea also has been described following very distal supracondylar fractures ( [263](#), [265](#), [274](#), [278](#)). This can occur in both displaced and nondisplaced fractures. The index case for Morrissey and Wilkins' article ( [278](#)) was actually a very distal nondisplaced supracondylar fracture that developed a typical fishtail deformity.

#### *Separation of the Total Distal Humeral Physis*

Again, the very distal location of the fracture line allows it to transect the transphyseal vessels at the lateral portion of the medial crista during the acute fracture ( [269](#), [278](#), [289](#), [290](#)). Many of these recover, and the avascular necrosis is never evident because this often occurs in the preosseous stage of the development of the trochlea.

### **T-Condylar Fracture**

If comminution occurs, some of the T-condylar fractures can injure the delicate blood vessels that supply the trochlea. Fortunately, most of these occur in the older age group, in which the vulnerability of the distal humerus to avascular necrosis is lessened.

### **Sprain of the Elbow**

This entity also has been described as “following a sprain” of the elbow ( [265](#)). However, this was probably an unrecognized and undisplaced very distal supracondylar or distal physeal injury.

### **Iatrogenic**

Sometimes an extensive surgical approach and dissection of the soft tissue is necessary to obtain a good open reduction of a fracture about the elbow, especially when seen and treated on a delayed basis. This can injure the vessels that supply the trochlea, with subsequent avascular necrosis ( [270](#)). When surgically approaching the medial condylar articular surface from a medial approach, the vessels supplying the medial aspect of the medial crista are on the extraarticular surface of the crista of the trochlea and must be carefully avoided.

### **Etiology**

Three theories have been proposed to account for the posttraumatic changes that occur in the distal humerus after fractures in the vicinity of the trochlea: malunion, partial growth arrest, and vascular injury.

### **Malunion**

Wilson ( [288](#)) thought this was the result of a malunion of a lateral condylar fracture with an intervening gap. This type of deformity is static, often asymptomatic, and nonprogressive.

### **Partial Growth Arrest**

Wadsworth ( [287](#)) thought this represented a premature fusion of the lateral condylar epiphysis to the metaphysis. Jakob and co-workers ( [271](#)) thought this was actually due to a primary disturbance of the growth cells adjacent to the fracture line of the lateral condyle. These theories can support the development of hypoplasia of the central or lateral portion of the trochlea. They do not, however, explain the total destruction of the trochlea that can occur. Growth arrest often takes months or years to produce deforming effects, and many of these changes occur rapidly after the initial injury.

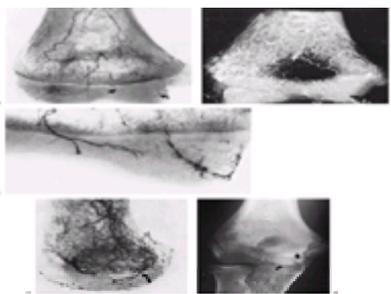
### **Vascular Injury**

#### **Two Separate Sources**

In Haraldsson's classic studies ( [266,267](#)) of the blood supply of the distal humerus, he demonstrated that the medial crista of the trochlea had two separate sources of blood supply. Neither has anastomoses with each other or with the other metaphyseal vessels. In the young infant, the vessels are small and lie on the surface of the perichondrium ( [Fig. 15-71A, Fig. 15-71B](#) and [Fig. 15-71C](#)). Immediately before and during the deposition of bony salts in the ossification centers of the trochlea, two distinct lateral and medial nonanastomotic sets of nutrient vessels are present.

#### **Lateral Vessels**

The lateral vessels supply the apex of the trochlea and the lateral aspect of the medial crista. These vessels cross the growth plate to enter the posterior aspect of the lateral trochlear ossification center. Their terminal branches lie just under the articular surface ( [Fig. 15-71D](#)). Thus, they are particularly vulnerable to injury when the fracture line occurs through this area, as is typical in fractures of the medial condylar physis or lateral condyle or a T-condylar fracture. By the same token, a fracture in the supracondylar area in which the fracture line is very distal, or a total distal humeral physeal displacement, also can disrupt the lateral trochlear epiphyseal vessels as they course along the surface of the metaphysis or at their entrance into the physeal plate.



**FIGURE 15-71.** Blood supply of the trochlea. **A:** Intraosseous vasculature in a 3-year-old boy. Only two small vessels supply the medial crista of the trochlea ( *arrow*). The central portion of the crista appears avascular. **B:** Injection studies showing the well-defined medial and lateral vessels ( *arrow*) supplying the medial crista of the trochlea. **C:** Close-up view showing the extent of the vascular supply of the trochlea. Note that no anastomoses are seen between these medial and lateral vessels. **D:** Lateral view through the medial crista of the trochlea. Note that the vessels penetrate the physis posteriorly ( *arrow*) to enter the epiphyseal cartilage. (A-D: Reprinted from Haraldsson S. On osteochondritis deformans juvenilis capituli humeri. *Acta Orthop Scand Suppl* 1959;38: with permission.) **E:** Radiograph of a 12-year-old boy. The persistence of the two separate ossification centers ( *arrow*) of the medial crista is seen. The area supplied by the lateral vessel is larger than that supplied by the medial vessel.

#### **Medial Vessels**

Another set of vessels enters medially through the nonarticulating surface of the trochlea ( [Fig. 15-71B](#) and [Fig. 15-71C](#)). This set of vessels supplies the most medial aspect of the medial crista or the medial portion of the trochlear epiphysis. As shown in Haraldsson's ( [266,267](#)) studies, there appear to be no anastomoses between these two sets of vessels supplying the trochlear epiphysis.

#### **Age Factors**

Most of the reported cases of avascular necrosis of the trochlea occur in children who are at least 6 or 7 years of age. However, it has been recently reported in younger patients after fracture-separation of the entire distal physis of the humerus ( [290](#)).

Ossification centers need blood supply for their appearance and development. Before these centers appear, the vessels are more superficial and less well defined. It is speculated that a lesion in these immature vessels in children leads only to a delay in the appearance of the centers. In older children, where there is already a well-defined ossification center, disruption produces a true bony avascular necrosis of one or both of the ossification centers of the trochlea. This can result in a

partial or total absence of further epiphyseal ossification, leading to hypoplasia of the central or whole medial aspect of the trochlea.

### **Fracture Location Critical**

The common factor of all these fracture pathologies producing avascular necrosis of the trochlea seems to be the presence of a fracture line or other injury in proximity to the lateral aspect of the medial condylar physis or the whole medial crista of the trochlea. These fracture lines injure the vessels as they enter their respective ossification centers of the trochlea.

The direct relationship between trauma and avascular necrosis of the trochlea also has been suggested by its development after surgical intervention of the original fracture, especially after open reduction from a medial approach.

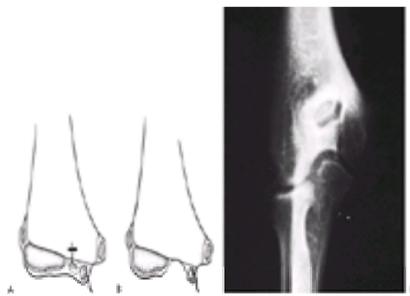
To support the vascular theory, there is also the observation that the necrosis of the trochlea follows the trauma rapidly. When recognized early, the period between the fracture and the development of changes consistent with avascular necrosis is months or weeks ( [278,290](#)).

### **Patterns of Necrosis**

Avascular necrosis of the trochlea can appear as either a central defect (type A) or total hypoplasia manifest by complete absence of the trochlea (type B), depending on the extent of the vascular injury.

#### **Type A—Fishtail Deformity**

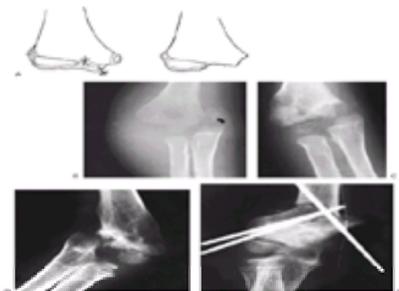
In the type A deformity, only the lateral portion of the medial crista or apex of the trochlea becomes involved in the necrotic process. This produces the typical fishtail deformity ( [Fig. 15-72](#)). This more common pattern of necrosis seems to occur with very distal supracondylar fractures or with fractures involving the lateral condylar physis.



**FIGURE 15-72.** Fishtail deformity. **A and B:** Type A deformity. Avascular necrosis only of the lateral ossification center creates a defect in the apex of the trochlear groove. **C:** Radiograph of a 14-year-old boy who sustained an undisplaced distal supracondylar fracture 5 years previously. The typical fishtail deformity is seen.

#### **Type B—Malignant Varus Deformity**

The type B deformity involves avascular necrosis of the entire trochlea and sometimes part of the metaphysis ( [Fig. 15-73](#)). This type of necrosis has occurred as a sequela of fractures involving the entire distal humeral physis or fractures of the medial condylar physis ( [285](#)). This can lead to a cubitus varus deformity in which the angulation progresses as the child matures.



**FIGURE 15-73.** Avascular necrosis of the entire trochlea. **A:** Type B deformity. Loss of blood supply from both the medial and lateral vessels results in avascular necrosis of the entire medial crista along with a portion of the metaphysis. **B:** Radiograph of a 4-year-old boy who sustained a type II physeal fracture involving the entire distal humeral physis. In this injury film there is a large metaphyseal fragment on the medial side ( *arrow*). **C:** Appearance 5 months later. A mild varus deformity present is due to an incomplete reduction. The ossification in the medial metaphyseal fragment has disappeared. **D:** Radiograph taken 4 years after injury. The entire medial crista and adjacent medial metaphysis have remained avascular. The varus deformity has progressed. **E:** Correction was achieved by performing a combination lateral closing wedge and medial translocation osteotomy.

### **Clinical Signs and Symptoms**

There is no particular clinical sign during the evolution of the necrosis. In most cases, the symptoms develop after healing of the fracture. In fact, in the typical scenario, in which avascular necrosis of the trochlea occurs after a supracondylar fracture, the patient initially has full recovery with healing of the fracture. However, as the necrotic process develops, the patient develops the late onset of symptoms of degenerative joint disease such as stiffness, loss of motion, and pain following physical activity.

### **Sequelae**

The clinical signs and symptoms differ considerably between the two patterns of necrosis.

#### **Type A—Symptoms**

Patients who have type A or fishtail deformity usually do not develop any angular deformities. Early degenerative joint disease with a loss of range of motion is the most common sequela. The severity of the fishtail deformity is related to the degree of necrosis and seems to dictate the severity of the symptoms. Wilson ( [288](#)) reported a 47-year follow-up in a patient whose major complaints related primarily to arthritis.

#### **Type B—Progressive Varus**

In children who have a pattern of total avascular necrosis of the trochlea, including part of the nonarticular surface, usually a progressive varus deformity develops. Because the total medial trochlear surface is disrupted, significant loss of range of motion also develops. These deformities usually worsen cosmetically and functionally as the child matures ([Fig. 15-73C](#) and [Fig. 15-73D](#)).

### ***Ulnar Neuropathy***

A late-onset ulnar neuropathy can develop in these patients ([277,282](#)). The hypoplasia of the medial condyle and its associated epicondyle produces a shallow ulnar groove. This allows the ulnar nerve to slip anteriorly. In addition, the medial head of the triceps muscle also slips anteriorly. There is no consensus on the etiology of the ulnar neuropathy. It is thought to be due to a multiplicity of factors, including malalignment of the joint, abnormal position of the ulnar nerve and triceps tendon, loss of protection by a deep ulnar groove, and the acute angle of entrance of the two heads of the flexor carpi ulnaris. Each of these factors has a variable bearing on the etiology of the neuropathy. In some of those cases reported with late-onset ulnar neuropathy, there were extensive bony changes with the presence of ganglions on the medial aspect of the joint ([282](#)). However, late-onset ulnar neuropathy can occur in children after other types of injuries without radiographic findings of trochlear hypoplasia ([281](#)).

### **Treatment**

Because the avascular necrosis of the trochlea is a direct consequence of trauma to the vessels occurring at the time of injury, there is no effective prevention or treatment of the primary necrosis. Treatment entails addressing only the sequelae of the avascular necrosis of the trochlea.

### ***Loss of Range of Motion***

If the loss of range of motion is due to a significant disruption of the articular surface itself, there does not appear to be any good operative or nonoperative method that significantly improves elbow function. A good program of muscle strengthening may lessen the symptoms to some degree.

In the one case treated surgically by Morrissey ([278](#)), he noticed that the olecranon had migrated proximally because of the central necrotic defect of the distal humerus. He felt this created an impingement between the olecranon and the distal humerus during full extension. In that case, a partial resection of the proximal olecranon improved extension of the elbow.

### ***Varus Deformity***

If the avascular necrosis of the trochlea has resulted in a varus deformity of the elbow, this deformity can be corrected by a supracondylar osteotomy ([Fig. 15-73E](#)). The correction of the carrying angle is mostly cosmetic, with little functional improvement. It does not seem to improve the range of motion. Surgical treatment carries the risk of increased stiffness to the already limited elbow. It can be argued that if this osteotomy is performed relatively early to correct the varus deformity, this could decrease the incidence of late-onset ulnar neuropathy due to the anterior migration of the ulnar nerve and medial head of the triceps.

### ***Ulnar Neuropathy***

In the cases of ulnar neuropathy occurring after hypoplasia of the trochlea, relief of ulnar nerve symptoms was achieved by transposing the ulnar nerve anteriorly in addition to releasing it from the adhesions around the hypoplastic medial epicondyle ([277,282](#)).

## **T-CONDYLAR FRACTURES**

In T-condylar fractures, the fracture line originates in the central groove of the trochlea and courses proximal to the olecranon and the coronoid fossae, where it divides and separates the medial and lateral bony columns of the distal humerus. If the proximal fracture lines are oblique, the fracture may be termed Y-condylar. This injury is rare in the skeletally immature child.

### **Incidence**

#### ***Primarily in Adolescents***

The early modern literature reflects only reports by Blount ([292](#)) and Zimmerman ([319](#)), who each described a case in an 11-year-old patient. The average age of patients reported in the three major recent series ([302,303,310](#)) was 12.6 years. Our clinical experience with 11 cases over a 20-year span has confirmed these data, with the average age being 11 years. Thus, Maylahn ([306](#)), who reported on six patients near skeletal maturity, was accurate when he said, "the fractures [T-condylar] take on the characteristics of an adult fracture and should be treated as such."

#### ***Occurrence in Young***

In 1902, Cotton ([296](#)) echoed the view of the surgical community at that time when he stated, "the fracture [T-condylar] is not only very uncommon, but very rare [in young children]." The actual incidence in younger children is certainly rare, but it may be underdiagnosed. This is because it is often confused with other fractures, such as those involving the lateral condylar physis or total distal physis. Special imaging studies such as arthrograms were necessary to demonstrate the intracondylar aspect in a recent report involving two patients under 3 years of age ([291](#)). The combination of an increased awareness of the possibility of this injury and a more aggressive diagnostic approach may result in more cases being uncovered in this younger age group.

The rare occurrence of the injury is reflected in the importance given in recent textbooks on children's fractures. A few ([298,309,312,319](#)) contain only a brief discussion of this fracture pattern; others ([294,295,300,311,317](#)) do not mention it at all.

### **Mechanism of Injury**

#### ***Wedge Effect of the Olecranon (Semilunar Notch)***

The primary mechanism of this injury is the direct wedge effect of the articular surface of the olecranon on the distal end of the humerus. The sharp edge of the semilunar notch or coronoid process acts as a wedge to break the trochlea and split the condyles, which in turn separates the two columns of the distal humerus ([297](#)). In our review of 11 skeletally immature patients with this injury, the vertical fracture line always extended down to the apex of the trochlea, tending to confirm this mechanism.

In a biomechanical study performed on adult cadaver specimens, Mehne and Matta ([307](#)) examined the force of the olecranon on the trochlea with varying degrees of flexion of the elbow. The situation that most consistently produced T-condylar fractures occurred when the force was applied directly to the olecranon with the elbow in more than 90 degrees of flexion.

The mechanism of these fractures has evolved from both biomechanical and clinical studies. The biomechanical studies involve the wedge mechanism of the olecranon against the distal humerus. The clinical studies are the results of indirect observations as to whether injuring force was applied to the distal humerus in either a flexion or extension position.

#### ***Flexion Injuries***

The most common mechanism producing this fracture pattern is said to be a direct blow to the posterior aspect of the elbow ([313](#)), usually when the child falls directly on the flexed elbow. This flexion mechanism in young children contributes to its rarity because most upper extremity injuries in children have a component of elbow

hyperextension. Injuries to the elbow caused by a flexion mechanism are rare. In these flexion injuries, the wedge effect is produced at the apex of the trochlea by the central portion of the trochlear notch. In these flexion injuries, the condylar fragments usually lie anterior to the shaft ( [Fig. 15-74A](#) and [Fig. 15-74B](#)).



**FIGURE 15-74.** Mechanism patterns. **A and B:** The more common flexion pattern in which the condylar fragments are situated anterior to the distal shaft. **C and D:** An extensor pattern in which the condylar fragments are situated posterior to the distal shaft. The muscle origins on the respective condyles cause them to diverge in the coronal plane (*arrow*) and flex in the sagittal plane.

### **Extension Injuries**

In a few cases, the T-condylar fracture may be caused by a fall on the outstretched arm with the elbow in only slight flexion. This extension mechanism has been suggested by patients in their description of the dynamics of the fall, and indirectly by the position of the distal fragments in relation to the diaphyses of the humerus—in other words, lying posterior ( [Fig. 15-74C](#) and [Fig. 15-74D](#) ). In the extension type of injury, the coronoid portion of the semilunar notch produces the wedge effect.

### **Role of Muscles**

It has been suggested that contraction of the elbow flexor and extensor muscles may play a role in the displacement pattern of this fracture. Because of their origins on the epicondyles, they accentuate both the separation in the coronal plane and the forward displacement in the sagittal plane. This displacement pattern often is evident on the injury films ( [Fig. 15-74C](#) and [Fig. 15-74D](#) ).

### **Pathology**

#### **The More Common Adolescent Pattern**

The fracture pattern in the adolescent is similar to that of the adult. The condylar fragments are often separated, with the articular surface being completely disrupted. In addition to separation of the condylar fragments by the force of the original injury, the muscles that originate on these condylar fragments rotate them in both the coronal and sagittal planes ( [Fig. 15-74C](#) and [Fig. 15-74D](#) ) (297). In the sagittal plane, the position of the condylar fragments in relation to the humeral shaft and metaphysis can either be anterior (flexor mechanism; see [Fig. 15-74B](#)) or posterior (extension mechanism; see [Fig. 15-74D](#)).

#### **The Rare Patterns in the Young Child**

##### **Articular Surface Remains Intact**

In the skeletally immature patient, the central portions of the condylar fragments are usually separated, but the articular surface may remain intact because of its large cartilage component ( [Fig. 15-75](#) ) (310). Thus, the disruption and displacement are primarily in the osseous supracondylar area. The elasticity of the cartilage of the distal end of the humerus often protects the articular surface from being completely disrupted.



**FIGURE 15-75.** Intact articular surface. A T-condylar fracture in a 7-year-old boy. The thick articular cartilage remains essentially intact, preventing separation of the condylar fragments. This fracture was secured with simple percutaneous pins.

### **Variable Resistance to Failure**

Cotton (296) reasoned that in the distal humerus, the condyles present variable resistance to failure. The external (lateral) condyle has a weakened resistance. The medial condyle exhibits a stronger resistance to separation. It was his opinion that this variable resistance explained the infrequency of both T-condylar and medial (internal) condyle fractures and the higher incidence of lateral condyle fractures.

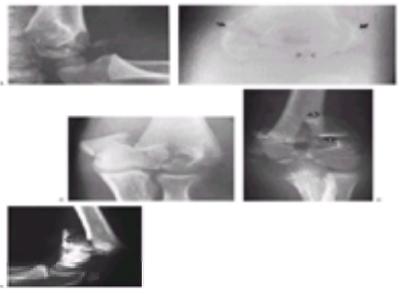
### **Classification**

Various classifications (302,308,314) for adult T-condylar fractures have been proposed, but there are problems with applying these classifications to children's injuries. For example, the number of children with this fracture is so small that no clinician can include all types of fracture patterns in his or her own experience. In addition, there is no useful classification for the younger patient, in whom the unossified intact articular cartilage is not visible on radiography. For example, the Jarvis type III (302) fracture-ndislocation or Chutro-Posados fracture has never been observed in our clinical experience, nor has it been reported in other series. The C-3 fracture in the AO classification (308,319), which has articular surface comminution, also has not been reported in the literature in children.

### **Authors' Suggested Classification**

We have tried to simplify the classification so that it will be useful in determining treatment options. We feel there are three major types based on the degree of displacement and comminution of the fracture fragments. Type I fractures are minimally displaced ( [Fig. 15-76A](#) and [Fig. 15-76B](#) ). Type II fractures are displaced but do not have comminution of the metaphyseal fragments ( [Fig. 15-76C](#) ). Type III fractures are displaced fractures with comminution of the metaphyseal fragments ( [Fig.](#)

15-76D and Fig. 15-76E).



**FIGURE 15-76.** Types of T-condylar fractures. **A:** Type I—Lateral view of undisplaced T-condylar fracture in a 6-year-old. **B:** The T-condylar fracture line (*open arrows*) was not appreciated until it healed. There are both medial and lateral Thurston-Holland fragments (*solid arrows*). (Courtesy of Ruben D. Pechero, M.D.) **C:** Type II—a displaced T-condylar fracture with very little metaphyseal comminution. **D and E:** Type III—two views of a markedly comminuted T-condylar fracture with multiple displaced fragments (*arrow*) in a 12-year-old.

In a child, the integrity of the articular surface may be difficult to determine without using arthrography or MRI. Because the initial integrity of the articular surface may not be that important to the prognosis, we feel that this factor does not significantly contribute to a general classification scheme. Adolescent T-condylar fractures may be classified as in adults.

### Diagnosis

#### Clinical Signs and Symptoms

Clinically, these fractures are most often confused with extension-type supracondylar fractures. The extended position of the elbow, along with the massive swelling, is almost identical to that of the displaced extension type of supracondylar fracture.

#### Imaging Studies

##### Plain X-Rays

**Diagnosis Not Always Obvious** Plain x-ray films are the cornerstone to the diagnosis. In the older child, the differentiation must be made from that of a comminuted supracondylar fracture. Sometimes the diagnosis is not obvious until the fragments have been partially reduced, which allows the vertical fracture lines splitting the trochlea to become more evident. In the younger child, the diagnosis is much more difficult because the articular surface is not visible. In addition, because of its rarity, the surgeon may not consider the possibility of a T-condylar fracture in this age group.

##### Differential Diagnosis

The diagnosis must exclude the more common fracture patterns of either the isolated lateral or medial condyles, or the complete separation of the distal humeral physis. In these latter fractures, an important sign is the presence of a medial or lateral Thurston-Holland fragment in the metaphysis ([291](#)). The key differential for the T-condylar fracture is the presence of a vertical fracture line extending down to the apex of the trochlea.

##### Computed Tomography and Magnetic Resonance Imaging

In the acute injury, the use of these imaging modalities does not appear to have much practical value. In younger patients, this often requires a separate anesthesia or heavy sedation outside the operating room.

##### Dynamic Studies Under Anesthesia

The diagnosis may be suspected after a careful evaluation of the static x-ray films. However, to settle the issue before definitive treatment, the surgeon may need to obtain either varus or valgus stress films under general anesthesia ([291](#)). In many cases, the use of contrast medium in the form of an arthrogram is also quite helpful.

### Treatment

#### Basic Principles

Because of the rarity of this injury, no one can recommend a plan of management based on multiple case experience. Most of the experience in the past has been based on isolated cases or small series ([291,302,303,305,310](#)). Regardless of the method of treatment, certain basic principles must be considered in treating these fractures. A treatment plan must be individualized for the specific fracture and the surgeon's level of expertise and experience. The following principles must be considered in planning a method of treatment:

1. Elbow articular mobility depends on articular congruity, correct alignment of the axis of motion, and debris- and bone-free fossae.
2. The stability depends on the integrity of the lateral and medial supracondylar columns.
3. The T-condylar fracture is an articular fracture, so the first goal is to restore and stabilize the joint surface.
4. Closed methods alone usually cannot produce an acceptable result because of the muscle forces applied to the fragment.
5. Most patients are adolescents with minimal potential for bone remodeling.
6. Although surgical reduction may produce an acceptable radiograph, it may add to the already extensive damage to soft tissues; this in turn can contribute to postoperative stiffness.

#### Current Trends Toward Surgical Stabilization

The current literature reflects good results with surgical management. Zimmerman ([319](#)) advocated establishing an anatomic reduction with internal fixation so that early motion could facilitate a more rapid rehabilitation. In the two cases in young children described by Beghin and colleagues ([291](#)), operative intervention was necessary to achieve a satisfactory reduction. A review of the three most recent series ([302,303,310](#)) indicates that surgical management has been established as the present trend by virtue of the fact that 29 of the 31 elbows in these combined series were treated operatively. The investigators in these series maintained that open reduction and internal fixation was the best way to restore the integrity of the articular surface and stabilize the fracture sufficiently to allow early mobilization. All but one of the patients in this combined series who were treated surgically demonstrated good or very good results at follow-up.

#### Popular Surgical Approaches

The surgical approach most widely accepted is the posterior longitudinal splitting of the triceps without an osteotomy of the olecranon. This approach gives adequate exposure of the fracture and the articular surface and does not seem to produce any loss of strength from splitting the triceps ([303](#)). Although one case demonstrated radiographic evidence of avascular necrosis of the trochlea ([310](#)), another reported a nonunion ([303](#)), and many had some loss of range of motion, none of these

surgically treated cases demonstrated any significant loss of elbow function or discomfort.

Another surgical approach that has become popular is the one advocated by Bryan and Morrey ( 293). This is a triceps-sparing approach in which the extensor mechanism is reflected laterally, exposing the whole distal humerus.

## AUTHORS' PREFERRED METHOD OF TREATMENT

Because of the rarity of this fracture in children, there is no standard recommended treatment. Therefore, we are outlining our recommendations based on a combination of our clinical experience and the experience of others in a few series ( 302,303,310). Our first consideration in these fractures is to reestablish the integrity of the articular surface to maintain the congruity of the joint. Usually this cannot be achieved adequately by closed methods, so we proceed with an open surgical technique. We have found our simple classification to the three types based on the degree of displacement or comminution to be helpful in guiding the aggressiveness of our treatment.

### Type I (Undisplaced or Minimally Displaced)

In type I injuries, there is little displacement of the bony supracondylar columns. In children, the periosteum is often intact and can provide some intrinsic stability. In addition, the thicker articular and epiphyseal cartilage in the skeletally immature child may still be intact, even if the bony epiphysis appears severed by a vertical fracture line. Because of this, we have found two methods to be successful for these types of fractures.

**Closed Reduction—Percutaneous Pin Fixation.** These fractures require minimal manipulation under general anesthesia and radiographic control to reestablish the supracondylar columns. If there is anterior or posterior rotation in the sagittal plane of the metaphyseal portion of the column, a pin placed into that column can be used as a “joy stick” to manipulate the fragment into a satisfactory position. Once a satisfactory reduction is achieved, the pin can then be advanced across the fracture site for fixation. These fractures usually require multiple pins placed percutaneously, such as those used in comminuted supracondylar fractures ( Fig. 15-77). Because of the rapid healing, the pins can be removed at 3 weeks to allow early active motion.



**FIGURE 15-77.** Closed reduction and pin fixation. **A and B:** Two views of a type II T-condylar fracture in a 15-year-old. **C and D:** Because an anatomic reduction was achieved by manipulative closed reduction, it was secured with simple multiple pin fixation placed percutaneously. The articular surface was minimally displaced. The pins were removed at 3 weeks. At this age, healing was rapid enough to pull the pins at 3 weeks to allow active motion. Ultimately he was deficient only 10 degrees from achieving full extension.

**Traction.** If the articular cartilage is intact, it may close as a hinge with traction ( Fig. 15-78). The rotational displacement of the condyles created by the origins of the forearm muscles can be neutralized with olecranon traction, in which the elbow is suspended at 90 degrees of flexion. There is usually adequate stability from the callus around the fracture site at 2 to 3 weeks to discontinue the traction. The elbow is then immobilized in a hinged cast brace for an additional 2 to 3 weeks. This allows the initiation of protected active motion. However, with the present emphasis on short hospitalization, we find that skeletal traction is less acceptable for both social and financial reasons. Skeletal traction may be the only acceptable method of treatment in patients seen on a delayed basis with extensive skin abrasions, severe soft tissue injury, or gross comminution, in which cast application or other operative interventions might carry a high risk of infection.

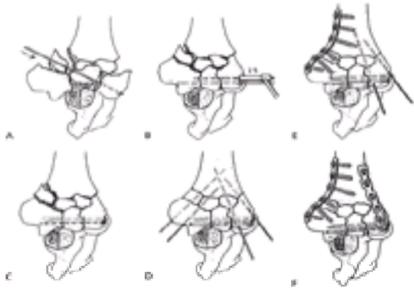


**FIGURE 15-78.** Treatment with traction. Treatment of the 6-year-old child with a T-condylar fracture shown in Fig. 15-76A and Fig. 15-76B. **A:** This patient was treated with traction. Once the fracture was reduced, the T-condylar nature of the fracture line was fully appreciated. The articular cartilage appeared intact and served as a hinge to maintain reduction. **B:** Lateral view in traction. Although the distal fragment remained extended, it was believed to be in an acceptable position. **C and D:** Anterior and lateral views 2 months after injury. The architecture of the condylar articular surface has been well maintained. (Courtesy of Marvin Mumme, M.D.)

### Type II (Displaced Without Comminution)

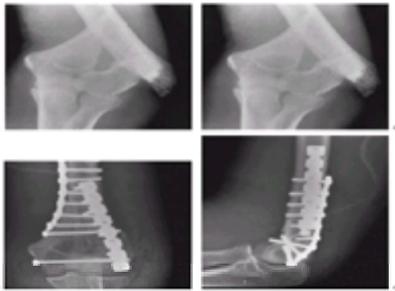
**Open Reduction and Internal Fixation.** If there is wide separation of the condylar fragments with marked disruption of the articular surface, stability and articular congruity can be established only with an open surgical procedure. We prefer the Bryan-Morrey posterior triceps-sparing approach ( 293). The patient is placed prone on the operating table with the arm supported on a pillow and the forearm hanging down off the edge of the operating table. This provides the best approach for direct visualization of the posterior surface of the distal humerus. Olecranon osteotomy is reserved for those fractures in adolescents with severe articular comminution.

**First, Reconstruct Articular Surface.** Our first priority is to reestablish the integrity of the articular fragments—in other words, to convert it to a supracondylar fracture (Fig. 15-79A, Fig. 15-79B and Fig. 15-79C). It is also critical that the olecranon and coronoid fossae be cleared of bony fragments or debris to eliminate the chance of bony impingement with their respective processes. The best way to stabilize the condyles is with a screw passed transversely through the center of the axis of rotation in such a manner as to apply transverse compression. This may require a small temporary secondary transverse pin proximal to the screw to prevent rotation of the fragments as the guide hole is drilled or the compression screw is being applied. This pin can be moved after the fragments are secured.



**FIGURE 15-79.** Sequence of reconstruction of the distal humerus. **A-C:** First, the articular portions are reassembled with provisional Kirschner wire fixation, followed by screw fixation. **D:** Kirschner wires can then also be used to provide temporary fixation of the distal humerus. **E:** A one-third tubular plate is attached to the medial side. **F:** A 3.5 pelvic reconstruction plate was attached to the posterolateral border. (Reprinted from Heim U, Pfeiffer KM. *Internal fixation of small fractures*, 3rd ed. Berlin: Springer-Verlag, 1988; with permission.)

*Secondly, Stabilize Supracondylar Columns.* Once the condylar and articular integrity has been reestablished, the distal fragments must be secured to the proximal fragment by stabilizing the supracondylar fragment columns. The decision here is how important it is to initiate early motion. If it is a younger child with rapid bony healing, pin fixation is often satisfactory so that they can be removed in 3 weeks to start protected motion, similar to treatment of a supracondylar humeral fracture. If it is an older adolescent nearer to skeletal maturity, we prefer fixation—usually plates or screws—that allows early motion ( [Fig. 15-79E](#) and [Fig. 15-79F](#) and [Fig. 15-80](#)). Before application of the plates, the supracondylar columns can be stabilized temporarily with pin fixation ( [Fig. 15-80D](#)).



**FIGURE 15-80.** Plate and screw fixation. **A and B:** Injury films of a type II flexion pattern in a 16-year-old boy. **C and D:** Articular integrity was first restored with a transcondylar compression screw. The condyles were secured to the metaphysis and distal shaft using pelvic reconstruction plates placed at 90 degrees to each other.

### Principles of Plate Fixation

The plates must be strong. The thin semitubular plates are inadequate and may break ( [Fig. 15-81](#)) (318). The reinforced malleable reconstructive type of plates used for fixation of pelvic fractures provide very secure fixation. Recently, a J type of plate has been designed that also can provide rigid fixation when used to stabilize the lateral column (316). It is best to place the plates at 90 degrees to each other, which makes for a more stable construct (301,315).



**FIGURE 15-81.** Inadequate fixation. **A:** Immediate postreduction film of a 14-year-old secured with short semitubular plates and a small transcondylar screw. **B:** Six weeks later the small compression screw in the condyle had lost fixation and the lateral plate had fractured. The patient was resecured with large pelvic reconstruction plates and a large transcondylar compression screw.

In most cases in the adolescent, this is essentially an adult type of fracture pattern. The reader is therefore referred to *Fractures in Adults* for a more detailed description of the various other techniques used in treating adults with this type of fracture.

### Postoperative Care

If plate fixation is used, we place the extremity in a supporting posterior splint for 5 to 7 days to allow the soft tissue swelling to decrease and the incisions to heal. At this time, active flexion and extension are initiated and the arm is protected with a removable cast brace. If there is considerable stiffness after 6 to 8 weeks, then we use the turnbuckle brace as advocated by Green and McCoy (299) to regain extension with increasing active range of motion.

### Type III (Displaced with Comminution)

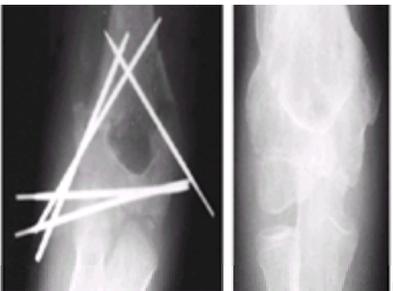
**Limited Open Reduction Followed by Traction.** Sometimes the supracondylar columns are too fragmented to produce adequate fixation. In such cases, we have found that in children, the best initial method of treatment involves reestablishing the articular surface and joint congruity with a limited open reduction. The separated condyles are secured with a transverse screw providing compression through the axis of rotation. This can usually be done with minimal soft tissue dissection. Once this is stabilized, the supracondylar columns are then reestablished by placing the extremity in olecranon traction and allowing them to reconstitute with callus formation. Traction must be maintained until there appears to be good osseous tissue formed in the supracondylar areas ( [Fig. 15-82](#)). While in traction, motion can be initiated. This technique also can be used in patients seen late with contaminated soft tissue abrasions or severe soft tissue problems.



**FIGURE 15-82.** Transcondylar fixation and traction. The anteroposterior and lateral radiographs of a 12-year-old boy who sustained a markedly comminuted T-condylar fracture are seen in [Fig. 15-76D](#) and [Fig. 15-76E](#). A major portion of the lateral supracondylar column was a totally avascular free fragment. **A:** Because it was totally avascular, it had to be removed, but the periosteum was preserved. The articular surface was reduced and stabilized with a transcondylar screw. The patient was then placed in overhead olecranon traction. **B:** Radiograph of the elbow 8 months after injury shows that the lateral supracondylar column is completely reconstituted. Even though the articular surface was maintained, there was about 30 degrees of loss of elbow motion ultimately.

### Complications

It is important to emphasize to the parents initially that this is a serious fracture. Because of the considerable soft tissue injury and the involvement of the articular surface of the distal humerus, stiffness and loss of motion of the elbow can be expected regardless of the mode of treatment ([292,302,306,310](#)). In the adolescent patient, failure to provide solid internal fixation that facilitates early motion (i.e., using only pin fixation) can result in a satisfactory radiographic appearance but considerable dysfunction due to residual loss of elbow motion ([Fig. 15-83](#)).



**FIGURE 15-83.** Stiff residua. **A:** Immediate postoperative film of an 11-year-old boy who sustained a comminuted T-condylar fracture from a direct blow to the flexed elbow. Fixation was achieved by way of a posterior approach, using multiple smooth pins. This prevented the initiation of early motion. **B:** The elbow 5 months postoperatively. Although the bony architecture had been restored, there was considerable restriction of motion from the soft tissue scarring. A better functional result may have resulted had more rigid internal fixation been applied that would not have allowed early motion.

Although neurovascular complications have not been mentioned in the few cases reported in the literature, it is expected that the incidence is about equal to that of supracondylar fractures. Because these fractures occur late in the growth process, partial or total growth arrest due to internal fixation is not thought to be a major complication. By the same token, because these are older children one cannot expect much in the way of remodeling. Nonunion ([303](#)), avascular necrosis of the trochlea ([310](#)), and failure of internal fixation ([Fig. 15-81](#)) also have been reported as complications.

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## CHAPTER REFERENCES

### Physeal Fractures

1. Bright RW. Physeal fractures. In: Rockwood CA, King RE, Wilkins KE, eds. *Fractures in children*. Vol. 3. Philadelphia: JB Lippincott, 1991.
2. Canale G, Beccario L, Orestano U. Detachment of external condyle of humerus in children. *Minerva Orthop* 1968;19:480-484.
3. Chacha PB. Fracture of medial condyle of humerus with rotational displacement. *J Bone Joint Surg* 1970;52:1453-1458.
4. Crabbe WA. Treatment of fracture separation of the capitular epiphysis. *J Bone Joint Surg* 1963;45:722-726.
5. DeLee JC, Wilkins KE, Rogers LF, et al. Fracture separation of the distal humeral epiphysis. *J Bone Joint Surg* 1980;67:46-51.
6. Fowles JV, Kassab MT. Displaced fracture of medial humeral condyle in children. *J Bone Joint Surg* 1980;62:1159-1163.
7. Hardacre JA, Nahigian SH, Froimson AI, et al. Fracture of the lateral condyle of humerus in children. *J Bone Joint Surg* 1971;53:1983-2095.
8. Holmberg L. Fracture of the distal end of the humerus in children. *Acta Chir Scand Suppl* 1945;92:1-69.
9. Marion J, LaGrange J, Faysse R, et al. Les fractures de l'extremite inferieure de l'humerus chez l'enfant. *Rev Clin Orthop* 1962;48:337-413.
10. Maylahn DJ, Fahey JJ. Fracture of the elbow in children. *JAMA* 1958;166:220-226.
11. Peterson CA, Peterson HA. Analysis of the incidence of injuries to the epiphyseal growth plate. *J Trauma* 1972;12:273-279.
12. Smith FM, Joyce JJ. Fracture of lateral condyle of the humerus in children. *Am J Surg* 1954;87:324-329.

### Fractures of the Lateral Condyle

13. Ashhurst APC. *An anatomical and surgical study of fractures of the lower end of the humerus*. Philadelphia: Lea & Febiger, 1910.
14. Badelon O, Bensahel H, Mazda K, et al. Lateral humeral condylar fractures in children: a report of 47 cases. *J Pediatr Orthop* 1988;8:31-34.
15. Badger FG. Fractures of the lateral condyle of the humerus. *J Bone Joint Surg* 1954;36:147-148.
16. Bast SC, Hoffer MM, Aval S. Nonoperative treatment for minimally and nondisplaced lateral humeral condyle fractures in children. *J Pediatr Orthop* 1998;18:448-450.
17. Beaty JH. Fractures and dislocations about the elbow in children. *Instr Course Lect* 1992;41:373-384.
18. Beaty JH, Wood AB. Fractures of the lateral humeral condyle in children. Presented at the Annual Meeting of the American Academy of Orthopedic Surgeons, Las Vegas, 1985.
19. Blount WP, Schalz I, Cassidy RH. Fractures of the elbow in children. *JAMA* 1951;146:699-704.
20. Blount WP. Unusual fractures in children. *Instr Course Lect* 1954;7:57-71.
21. Böhler L. *The treatment of fractures*. Vol. I. New York: Grune & Stratton, 1956.
22. Boyd HB. Fractures about the elbow in children. *Surg Gynecol Obstet* 1949;89:775.
23. Brewster AH, Karp M. Fractures in the region of the elbow in children. An end-result study. *Surg Gynecol Obstet* 1940;71:643-649.
24. Canale G, Becarig L, Orestano U. Detachment of external condyle of humerus in children. *Minerva Orthop* 1968;19:480-484.
25. Conn JJ, Wade PA. Injuries of the elbow: ten-year review. *J Trauma* 1961;1:246-266.
26. Conner A, Smith MGH. Displaced fracture of lateral humeral condyle in children. *J Bone Joint Surg* 1970;52:460-464.
27. Conway FM. Traumatic ulnar neuritis. *Ann Surg* 1933;97:425-433.
28. Cooper AP. *A treatise on dislocations and fractures of the joints*. Boston: Lilly, Wait, Carter & Hendee, 1932.
29. Cotton FJ. Elbow fractures in children. *Ann Surg* 1902;35:75-104.

30. Crabbe WA. Treatment of fracture—separation of the capitular epiphysis. *J Bone Joint Surg* 1963;45:772–776.
31. Davids JR, Maguire MF, Mubarak SJ, et al. Lateral condylar fracture of the humerus following posttraumatic cubitus varus. *J Pediatr Orthop* 1994;14:466–470.
32. Dhillon KS, Sengupta S, Singh BJ. Delayed management of fracture of the lateral humeral condyle in children. *Acta Orthop Scand* 1988;59:419–424.
33. Editorial. Fractures of the lateral condyle of the humerus in children. *Injury* 1985;16:363.
34. Fahey JJ. Fractures of the elbow in children. *Instr Course Lect* 1960;17:13–46.
35. Feindel W, Stratford J. Cubital tunnel compression in tardy ulnar palsy. *Can Med Assoc J* 1958;78:351–353.
36. Finnbogason T, Karlsson G, Lindberg L, et al. Nondisplaced and minimally displaced fractures of the lateral humeral condyle in children: a prospective radiographic investigation of fracture stability. *J Pediatr Orthop* 1995;15:422–425.
37. Flynn JC, Richards JF. Non-union of minimally displaced fractures of the lateral condyle of humerus in children. *J Bone Joint Surg [Am]* 1971;53:1096–1101.
38. Flynn JC, Richards JF, Saltzman R. Non-union of minimally displaced fractures of the lateral condyle of humerus in children. Scientific Exhibit at the Annual Meeting of the American Academy of Orthopedic Surgeons, Dallas, 1974.
39. Flynn JC, Richards JF, Saltzman RI. Prevention and treatment of non-union of slightly displaced fractures of the lateral humeral condyle in children. *J Bone Joint Surg [Am]* 1975;57:1087–1092.
40. Fontanetta P, Mackenzie DA, Rosman M. Missed, maluniting, and malunited fractures of the lateral humeral condyle in children. *J Trauma* 1978;18:329–335.
41. Foster DE, Sullivan JA, Gross RH. Lateral humeral condylar fractures in children. *J Pediatr Orthop* 1985;5:16–22.
42. Freeman RH. Fractures of lateral humeral condyle. *J Bone Joint Surg [Br]* 1959;41:631.
43. Friedman RJ, Smith RJ. Radial-nerve laceration twenty-six years after screw fixation of a humeral fracture. *J Bone Joint Surg [Am]* 1984;66:959–960.
44. Gaur SC, Vishwakarma DP, Varma B. An unusual injury of the lower humeral epiphysis in a child—a case report. *Injury* 1985;16:625–627.
45. Gay JR, Love JG. Diagnosis and treatment of tardy paralysis of the ulnar nerve. *J Bone Joint Surg* 1947;29:1087–1097.
46. Haraldsson S. On osteochondrosis deformans juvenilis capituli humeri including investigation of intra-osseous vasculature in distal humerus. *Acta Orthop Scand Suppl* 1959;38.
47. Hardacre JA, Nahigian SH, Froimson AI, et al. Fractures of the lateral condyle of the humerus in children. *J Bone Joint Surg* 1971;53:1083–1095.
48. Herring JA. Lateral condylar fracture of the elbow. *J Pediatr Orthop* 1986;6:724–727.
49. Heyl JH. Fractures of the external condyle of the humerus in children. *Ann Surg* 1935;101:1069–1077.
50. Holst-Nielsen F, Ottsen P. Fractures of the lateral condyle of the humerus in children. *Acta Orthop Scand* 1974;45:518–528.
51. Hopkins FS. Fractures of the capitellum. *N Engl J Med* 1932;206:259–261.
52. Huurman WW. Lateral humeral condylar fracture. *Nebr Med J* 1983;68:298–300.
53. Ingersoll R. Fractures of the humeral condyles in children. *Clin Orthop* 1965;41:32–42.
54. Ippolito E, Tudisco C, Farsetti P, et al. Fracture of the humeral condyles in children: 49 cases evaluated after 18-45 years. *Acta Orthop Scand* 1996;67:173–178.
55. Jakob R, Fowles JV. Observations concerning fractures of the lateral humeral condyles in children. *J Bone Joint Surg* 1975;40:430–436.
56. Jeffrey CC. Nonunion of epiphysis of the lateral condyle of the humerus. *J Bone Joint Surg* 1958;40:396–405.
57. Jones KG. Percutaneous pin fixation of fractures of the lower end of humerus. *Clin Orthop* 1967;50:53–69.
58. Kalenak A. Ununited fracture of the lateral condyle of the humerus. *Clin Orthop* 1977;124:181–183.
59. Kamegaya M, Shinohara Y, Kurokawa M, et al. Assessment of stability in children's minimally displaced lateral humeral condyle fracture by magnetic resonance imaging. *J Pediatr Orthop* 1999;19:570–572.
60. Kini M. Fractures of the lateral condyle of the lower end of the humerus with complication. *J Bone Joint Surg* 1942;24:268–278.
61. Marion J, LaGrange J, Faysse R, et al. Les fractures de l'extremite inferieure de l'humerus chez l'enfant. *Rev Chir Orthop* 1962;48:337–413.
62. Masada K, Kawai H, Kawabata H, et al. Osteosynthesis for old, established non-union of the lateral condyle of the humerus. *J Bone Joint Surg [Am]* 1990;72:32–40.
63. Maylahn DJ, Fahey JJ. Fractures of the elbow in children. *JAMA* 1958;166:220–226.
64. McDonnell DP, Wilson JC. Fracture of the lower end of humerus in children. *J Bone Joint Surg* 1948;30:347–358.
65. McGowan AJ. The results of transposition of the ulnar nerve for traumatic ulnar neuritis. *J Bone Joint Surg [Br]* 1950;32:291–299.
66. Milch H. Fracture of external humeral condyle. *JAMA* 1956;160:641–646.
67. Milch H. Fractures and fracture dislocations of humeral condyles. *J Trauma* 1964;4:592–607.
68. Miller EM. Late ulnar nerve palsy. *Surg Gynecol Obstet* 1924;38:37–46.
69. Mintzer CM, Water PM, Brown DJ, et al. Percutaneous pinning in the treatment of displaced lateral condyle fractures. *J Pediatr Orthop* 1994;14:462–465.
70. Mirsky EC, Karas EH, Weiner LS. Lateral condyle fractures in children: evaluation of classification and treatment. *J Orthop Trauma* 1997;11:117–120.
71. Mohan N, Hunter JB, Colton CL. The posterolateral approach to the distal humerus for open reduction and internal fixation of fractures of the lateral condyle in children. *J Bone Joint Surg [Br]* 2000;82:643–645.
72. Moorhead EL. Old untreated fracture of external condyle humerus. *Surg Clin Chicago* 1919;3:987–989.
73. Morin B, Fassier F, Poitras B, et al. Resultats du traitement chirurgical precoce des fractures du condyle humeral externe chez l'enfant. *Rev Chir Orthop* 1988;74:129–131.
74. Morrissy RT, Wilkins KE. Deformity following distal humeral fracture in childhood. *J Bone Joint Surg [Am]* 1984;66:557–562.
75. Papavasiliou VA, Beslikas TA. Fractures of the lateral humeral condyle in children: an analysis of 39 cases. *Injury* 1985;16:364–366.
76. Potter HG. Imaging of posttraumatic and soft tissue dysfunction of the elbow. *Clin Orthop* 2000;370:9–18.
77. Rang M. *Children's fractures*, 2nd ed. Philadelphia: JB Lippincott, 1983.
78. Ravessoud FA. Lateral condylar fracture and ipsilateral ulnar shaft fracture: monteggia equivalent lesions? *J Pediatr Orthop* 1985;5:364–366.
79. Rohl L. On fractures through the radial condyle of the humerus in children. *Acta Chir Scana* 1953;104:74–80.
80. Rovinsky D, Ferguson C, Younis A, et al. Pediatric elbow dislocations associated with a Milch type I lateral condyle fracture of the humerus. *J Orthop Trauma* 1999;13:458–460.
81. Rutherford AJ. Fractures of the lateral humeral condyle in children. *J Bone Joint Surg [Am]* 1985;67:851–856.
82. Sakakida K. Clinical observations on epiphyseal separation of long bones. *Clin Orthop* 1963;34:119–141.
83. Salter RB, Harris WR. Injuries involving the epiphyseal plate. *J Bone Joint Surg* 1963;45:587–632.
84. Sandegard E. Fractures of the lower end of humerus in childhood: treatment and end results. *Acta Chir Scana* 1944;89:1–16.
85. Sharma JC, Arora A, Mathur NC, et al. Lateral condylar fractures of the humerus in children: fixation with partially threaded 4.0-mm AO cancellous screws. *J Trauma* 1995;39:1129–1133.
86. Sharp IK. Fractures of the lateral humeral condyle in children. *Acta Orthop Belg* 1965;31:811–816.
87. Sharrard WJW. *Pediatric orthopaedics and fractures*. Oxford: Blackwell Scientific, 1971.
88. Shimada K, Masada K, Tada K, et al. Osteosynthesis for the treatment of non-union of the lateral humeral condyle in children. *J Bone Joint Surg [Am]* 1997;79:232–238.
89. Smith FM, Joyce JJ. Fracture of lateral condyle of humerus in children. *Am J Surg* 1954;87:324–329.
90. Smith FM. *Surgery of the elbow*, 2nd ed. Philadelphia: WB Saunders, 1972.
91. Smith FM. An 84-year followup on a patient with ununited fracture of the lateral condyle of humerus. *J Bone Joint Surg* 1973;55:378–380.
92. So YC, Fang D, Orth MC, et al. Varus deformity following lateral humeral condylar fracture in children. *J Pediatr Orthop* 1985;5:569–572.
93. Speed JS, Macey HB. Fracture of humeral condyles in children. *J Bone Joint Surg* 1933;15:903–919.
94. Stimson LA. *A treatise on fractures*. Philadelphia: Henry C. Lea, Son & Co., 1883.
95. Stimson LA. *A practical treatise on fractures and dislocations*. Philadelphia: Lea Brothers & Co., 1900.
96. Tachdjian MO. *Pediatric orthopaedics*. Philadelphia: WB Saunders, 1972.
97. Van Vugt AB, Severijnen RVSM, Festern C. Fractures of the lateral humeral condyle in children: late results. *Arch Orthop Trauma Surg* 1988;107:206–209.
98. Wadsworth TG. Premature epiphyseal fusion after injury of capitulum. *J Bone Joint Surg* 1964;46:46–49.
99. Wadsworth TG. Injuries of capitular epiphysis. *Clin Orthop* 1972;85:127–142.
100. Weber BG, Brunner C, Freuler F. *Treatment of fractures in children and adolescents*. Berlin: Springer-Verlag, 1980.
101. Wilson PD. Fracture of lateral condyle of humerus in children. *J Bone Joint Surg* 1936;18:299–316.
102. Wilson JN. Fracture of external condyle of humerus in children. *Br J Surg* 1955;43:88–94.
103. Zeir FG. Lateral condylar fracture and its many complications. *Orthop Rev* 1981;10:49–55.
104. Zions LE, Stolz MR. Late fracture of the lateral condyle of the humerus. *Orthopedics* 1984;7:541–545.

#### Fractures of the Capitellum.

105. Agins HJ, Marcus NW. Articular cartilage sleeve fracture of the lateral humeral condyle capitellum: a previously undescribed entity. *J Pediatr Orthop* 1984;4:620–622.
106. Alvarez E, Patel MR, Nimberg G, et al. Fracture of the capitulum humeri. *J Bone Joint Surg [Am]* 1975;57:1093.
107. Collert S. Surgical management of fracture of the capitellum humeri. *Acta Orthop Scand* 1977;48:603.
108. Drvaric DM, Rooks MD. Anterior sleeve fracture of the capitellum. *J Orthop Trauma* 1990;4:188–192.
109. Fioretta G, Rotolo F, Zanasi L. The treatment of fractures of the capitulum of the humerus. *Ital J Orthop Traumatol* 1984;10:81–84.
110. Fowles JV, Kassab MT. Fracture of the capitulum humeri, treatment by excision. *J Bone Joint Surg [Am]* 1974;56:794.
111. Gejrot W. On intra-articular fractures of the capitellum and trochlea of humerus with special reference to treatment. *Acta Chir Scana* 1932;71:231–268.
112. Hahn NF. Fall von eine Besonders Variet der Frakturen des Ellenbogens. *Zietschr F Wunder Geburt* 1953;6:185–189.
113. Johansson J, Rosman M. Fracture of the capitulum humeri in children: a rare injury, often misdiagnosed. *Clin Orthop* 1980;146:157–160.
114. Kocher T. *Beitrage zur Kenntniss Einiger Praktisch Wichtiger Fracturformen*. Basel: Carl Sallman, 1896:585–591.
115. Letts M, Rumball K, Bauermeister S, et al. Fractures of the capitellum in adolescents. *J Pediatr Orthop* 1997;17:313–318.
116. Lorenz H. Zur Kenntniss der Fractura Capituli Humeri (Eminentiae Capitatae). *Dtsch Z F Chir* 1905;78:531–545.

117. Marion J, Faysse R. Fracture du capitellum. *Rev Chir Orthop* 1962;48:484–490.
118. Mouchet A. *Fractures de l'extremite inferieure de l'humerus*. Paris: G. Steinheil, 1898:280–293.
119. Palmer I. Open treatment of transcondylar T-fracture of the humerus. *Acta Chir Scand* 1961;121:486–490.
120. Steintal D. Die Isolierte Fraktur der Eminencia Capitata im Ellenbogengelenk. *Zentralbl F Chir* 1898;15:17–20.
121. Yuan-Zhang M, Chun-Bo Z, Tai-Len Z, et al. Percutaneous probe reduction of frontal fractures of the humeral capitellum. *Clin Orthop* 1984;183:17–21.

#### Fractures of the Medial Condylar Physis

122. Ashhurst APC. *An anatomical and surgical study of fractures of the lower end of the humerus*. Philadelphia: Lea & Febiger, 1910.
123. Bensahel H, Csukonyi Z, Badelon O, et al. Fractures of the medial condyle of the humerus in children. *J Pediatr Orthop* 1986;6:430–433.
124. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955.
125. Bohler L. *The treatment of fractures*. New York: Grune & Stratton, 1956.
126. Case SL, Hennrikus WL. Surgical treatment of displaced medial epicondyle fractures in adolescent athletes. *Am J Sports Med* 1997;25:682–686.
127. Chacha PB. Fractures of the medial condyle of the humerus with rotational displacement. *J Bone Joint Surg* 1970;52:1453–1458.
128. Conn J Jr, Wade PA. Injuries of the elbow: a ten-year review. *J Trauma* 1961;1:246–266.
129. Cothay PM. Injury to the lower medial epiphysis of the humerus before development of the Ossific Centre. Report of a case. *J Bone Joint Surg* 1967;49:766–767.
130. Cotton FJ. Elbow fractures in children. *Ann Surg* 1902;35:75–104.
131. De Boeck H, Casteleyn PP, Opdecam P. Fracture of the medial humeral condyle. *J Bone Joint Surg [Am]* 1987;69:1442–1444.
132. El Ghawabi MH. Fracture of the medial condyle of the humerus. *J Bone Joint Surg* 1975;57:677–680.
133. Fahey JJ. Fractures of the elbow in children. *Instr Course Lect* 1960;17:13–46.
134. Fahey JJ, O'Brien E. Fracture—Separation of the medial humeral condyle in a child confused with fracture of the medial epicondyle. *J Bone Joint Surg* 1971;53:1102–1104.
135. Faysse R, Marion J. Fractures du condyle interne. *Rev Chir Orthop* 1962;48:473–477.
136. Fowles JV, Kassab MT. Displaced fractures of the medial humeral condyle in children. *J Bone Joint Surg* 1980;62:1159–1163.
137. Hanspal RS. Injury to the medial humeral condyle in a child reviewed after 18 years. Report of a case. *J Bone Joint Surg [Br]* 1985;67:638–639.
138. Harrison RB, Keats TE, Frankel CJ, et al. Radiographic clues to fractures of the unossified medial humeral condyle in young children. *Skel Radio*, 1984;11:209–212.
139. Hasner E, Husby J. Fracture of epicondyle and condyle of humerus. *Acta Chir Scand* 1951;101:195–203.
140. Holmberg L. Fractures in the distal end of the humerus in children. *Acta Chir Scand Supp*. 1945;103.
141. Ingersoll R. Fractures of the humeral condyles in children. *Clin Orthop* 1965;41:32–42.
142. Kilfoyle RM. Fractures of the medial condyle and epicondyle of the elbow in children. *Clin Orthop* 1965;41:43–50.
143. McDonnell DP, Wilson JC. Fractures of the lower end of the humerus in children. *J Bone Joint Surg* 1948;30:347–358.
144. Milch H. Fractures and fracture—dislocations of humeral condyles. *J Trauma* 1964;4:592–607.
145. Papavasiliou V, Nenopoulos S, Venturis T. Fractures of the medial condyle of the humerus in childhood. *J Pediatr Orthop* 1987;7:421–423.
146. Pimpalnerkar AL, Balasubramaniam G, Young SK, et al. Type four fractures of the medial epicondyle: a true indication for surgical intervention. *Injury* 1998;29:751–756.
147. Potter CMC. Fracture dislocation of the trochlea. *J Bone Joint Surg* 1954;36:248–251.
148. Sandegard E. Fracture of the lower end of the humerus in children. Treatment and end results. *Acta Chir Scand* 1944;89:1–16.
149. Saraf SK, Tuli SM. Concomitant medial condyle fracture of the humerus in a childhood posterolateral dislocation of the elbow. *J Orthop Trauma* 1989;3:352–354.
150. Varma BP, Srivastava TP. Fractures of the medial condyle of the humerus in children: a report of 4 cases including the late sequelae. *Injury* 1972;4:171–174.
151. Watson-Jones R. *Fractures and joint injuries*, 4th ed. Edinburgh: ES Livingstone, 1956.

#### Fractures of the Trochlea

152. Grant IR, Miller JH. Osteochondral fracture of the trochlea associated with fracture—dislocation of the elbow. *Injury* 1975;6:255–258.

#### Fractures of the Entire Distal Humeral Physis

153. Abe M, Ishizu T, Nagaoka T, et al. Epiphyseal separation of the distal end of the humeral epiphysis: a follow-up note. *J Pediatr Orthop* 1995;15:426–434.
154. Akbarnia BA, Silberstein MJ, Rende RJ, et al. Arthrography in the diagnosis of fractures of the distal end of the humerus in infants. *J Bone Joint Surg [Am]* 1986;68:599–602.
155. Ashhurst APC. *An anatomical and surgical study of fractures of the lower end of the humerus*. Philadelphia: Lea & Febiger, 1910.
156. Barrett WP, Almquist EA, Staheli LT. Fracture separation of the distal humeral physis in the newborn. *J Pediatr Orthop* 1984;4:617–619.
157. Berman JM, Weiner DS. Neonatal fracture separation of the distal humeral chondroepiphysis: a case report. *Orthopedics* 1980;3:875–879.
158. Bright RW. Epiphyseal-plate cartilage. A biomechanical and histological analysis of failure modes. *J Bone Joint Surg* 1974;56:688–703.
159. Chand K. Epiphyseal separation of distal humeral epiphysis in an infant. *J Trauma* 1974;14:521–526.
160. Cotton FJ. Elbow fractures in children. *Ann Surg* 1902;35:75–104.
161. Dameron TB. Transverse fractures of distal humerus in children. *Instr Course Lect* 1981;30:222–233.
162. de Jager LT, Hoffman EB. Fracture—separation of the distal humeral epiphysis. *J Bone Joint Surg [Br]* 1991;73B:143–146.
163. DeLee JC, Wilkins KE, Rogers LF, et al. Fracture—separation of the distal humerus epiphysis. *J Bone Joint Surg* 1980;62:46–51.
164. Dias JJ, Lamont AC, Jones JM. Ultrasonic diagnosis of neonatal separation of the distal humeral epiphysis. *J Bone Joint Surg [Br]* 1988;70:825–828.
165. Downs DM, Wirth CR. Fracture of the distal humeral chondroepiphysis in the neonate. A case report. *Clin Orthop* 1982;169:155–158.
166. Ekengran K, Bergdahl S, Ekstrom G. Birth injuries to the epiphyseal cartilage. *Acta Radiol Diagn [Stockh]* 1978;19:197–204.
167. Faysse R, Marion J. Decollement en masse de l'epiphyse inferieure de l'humerus. *Rev Chir Orthop* 1962;48:478–483.
168. Hansen PE, Barnes DA, Tullos HS. Case report—arthrographic diagnosis of an injury pattern in the distal humerus of an infant. *J Pediatr Orthop* 1982;2:569–572.
169. Hersh CK, Sanders JO. Case report of improper treatment of distal humeral epiphyseal separation resulting in neurologic injury (unpublished paper).
170. Holda ME, Manoli A, LaMont RL. Epiphyseal separation of the distal end of the humerus with medial displacement. *J Bone Joint Surg* 1980;62:52–57.
171. Houben JJ, Van Elegem P, Godart S, et al. Les glissements epiphysaires du coude chez le jeune enfant. Aspects diagnostiques. *Acta Orthop Belg* 1983;49:592–600.
172. Kaplan SS, Reckling FW. Fracture separation of lower humeral epiphysis with medial displacement. *J Bone Joint Surg* 1971;53:1105–1108.
173. Macafee AL. Infantile supracondylar fracture. *J Bone Joint Surg* 1967;49:768–770.
174. Marmor L, Bechto CO. Fracture separation of lower humeral epiphysis. *J Bone Joint Surg* 1960;42:333–336.
175. Mauer I, Kolovos D, Loscos R. Epiphysiolysis of distal humerus in a newborn. *Bull Hosp Joint Dis Orthop Insr* 1967;28:109–118.
176. McIntyre WM, Wiley JJ, Charette RJ. Fracture—separation of the distal humeral epiphysis. *Clin Orthop* 1984;188:98–102.
177. Mizuno K, Hirohata K, Kashiwagi D. Fracture—separation of distal humeral epiphysis in young children. *J Bone Joint Surg* 1979;61:570–573.
178. Odgen JA. *Skeletal injury in the child*, 2nd ed. Philadelphia: WB Saunders, 1990:386–391.
179. Omer GE, Simmons JW. Fracture of distal humeral metaphyseal growth plate. *South Med J* 1968;61:651–652.
180. Paige ML, Port RB. Separation of the distal humeral epiphysis in the neonate. A combined clinical and roentgenographic diagnosis. *Am J Dis Child* 1985;139:1203–1205.
181. Papandrea R, Waters PM. Posttraumatic reconstruction of the elbow in the pediatric patient. *Clin Orthop* 2000;370:115–126.
182. Peiro A, Mut T, Aracil J, et al. Fracture—separation of the lower humeral epiphysis in young children. *Acta Orthop Scand* 1981;52:293–296.
183. Poland J. *A practical treatise on traumatic separation of the epiphyses*. London: Smith, Elder & Co., 1898.
184. Rogers LF, Rockwood CA. Separation of entire distal humeral epiphysis. *Radiology* 1973;106:393–399.
185. Siffert RS. Displacement of distal humeral epiphysis in newborn infant. *J Bone Joint Surg* 1963;45:165–169.
186. Siris IE. Supracondylar fracture of the humerus. *Surg Gynecol Obstet* 1939;68:201–220.
187. Smith FM. *Surgery of the elbow*. Philadelphia: WB Saunders, 1954.
188. Stricker SJ, Thomson JD, Kelly RA. Coronal-plane transcondylar fracture of the humerus in a child. *Clin Orthop* 1993;292:306–309.
189. Sutherland DH. Displacement of the entire distal humeral epiphysis. *J Bone Joint Surg* 1974;56:206.
190. Valdiserri L, Kelescian G. Su un caso de distacco epifisario ostetrico dell'estremita distale dell'omero. *Osped Ital Chii* 1965;13:407–414.
191. Van Den Broek JAC, Vegter J. Diagnose van Epifysiolyse van het Proximale Deel van de Humerus bij een Pasgeborene met Echografie. *Ned Tijdschr Geneeska* 1988;132:1015–1017.
192. Willems B, Stuyck J, Hoogmartens M, et al. Fracture—separation of the distal humeral epiphysis. *Acta Orthop Belg* 1987;53:109–111.
193. Wilson PD. Fracture of the lateral condyle of humerus in childhood. *J Bone Joint Surg* 1936;18:299–316.
194. Yoo CI, Suh JT, Suh KT, et al. Avascular necrosis after fracture—separation of the distal end of the humerus in children. *Orthopedics* 1992;15:959–963.

#### Apophyseal Injuries of the Distal Humerus

##### Fractures Involving the Medial Epicondylar Apophysis.

195. Adams JE. Injury to the throwing arm. *Calif Med* 1965;102:127–132.

196. Albright JA. Clinical studies of baseball players: correlation of injury to throwing arm with method of delivery. *Am J Sports Med* 1978;6:15.
197. Aitken AP, Childress HM. Intra-articular displacement of the internal epicondyle following dislocation. *J Bone Joint Surg* 1938;20:161–166.
198. Ashurst APC. *An anatomical and surgical study of fractures of the lower end of the humerus*. Philadelphia: Lea & Febiger, 1910.
199. Bede WB, Lefebure AR, Rosmon MA. Fractures of the medial humeral epicondyle in children. *Can J Surg* 1975;18:137–142.
200. Bernstein SM, King JD, Sanderson RA. Fractures of the medial epicondyle of the humerus. *Contemp Orthop* 1981;637–641.
201. Blount WP. Unusual fractures in children. *Instr Course Lect* 1954;7:57–71.
202. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955.
203. Brodeur AE, Silberstein MJ, Graviss ER. *Radiology of the pediatric elbow*. Boston: GK Hall, 1981.
204. Brogdon BJ, Crow NE. Little leaguer's elbow. *AJR* 1960;83:671–675.
205. Cataliotti F, Giglio AL, Salomone G. L'osteosintesi percutanea nella frattura-distacco della epitroclea e del blocco condilo-epicondiloideo nell'infanzia. *Minerva Med* 1972;63:4254–4258.
206. Chessare JW, Rogers LF, White H, et al. Injuries of the medial epicondylar ossification center of the humerus. *AJR* 1977;129:49–55.
207. Collins R, Lavine SA. Fracture of the medial epicondyle. *Clin Proc Child Hosp* 1964;20:272–275.
208. Conn JJ, Wade PA. Injuries of the elbow: a 10-year elbow. *J Trauma* 1961;1:246–266.
209. Cotton FJ. Elbow fractures in children. *Ann Surg* 1902;35:75–104.
210. Cotton FJ. *Dislocations and joint fractures*. Philadelphia: WB Saunders, 1924.
211. Dias JJ, Johnson GV, Hoskinson J, et al. Management of severely displaced medial epicondyle fractures. *J Orthop Trauma* 1987;1:59–62.
212. Dunlop J. Traumatic separation of medial epicondyle of humerus in adolescence. *J Bone Joint Surg* 1935;17:577–587.
213. Fahey JJ. Fractures of the elbow in children [Abstract]. *Instr Course Lect* 1960;17:13–46.
214. Fairbank HAT, Buxton JD. Displacement of the internal epicondyle into the elbow joint. *Lancet* 1934;2:218.
215. Fevre M, Roudaitis. La reduction non saillante des fractures de l'epitrochlee avec interposition de ce fragment dans l'interligne articulaire du coude. *Rev Chir Orthop* 1933;20:298–312.
216. Fowles JV, Kassab MT, Moula T. Untreated intra-articular entrapment of the medial humeral epicondyle. *J Bone Joint Surg [Br]* 1984;60:562–565.
217. Fowles JV, Slimane N, Kassab MT. Elbow dislocation with avulsion of the medial humeral epicondyle. *J Bone Joint Surg [Br]* 1990;72B:102–104.
218. Frances R, Bunch T, Chandler B. Little league elbow: a decade later. *Phys Sports Med* 1978;88–94.
219. Gugenheim JJ, et al. Little league survey: the Houston study. *Am J Sports Med* 1976;4:189–200.
220. Harrison RB, Keats TE, Frankel CJ, et al. Radiographic clues to fractures of the unossified medial humeral condyle in young children. *Skel Radio*, 1984;11:209–212.
221. Higgs SL. Fractures of the internal epicondyle of the humerus. *BMJ* 1936;2:666–667.
222. Hines RF, Herndon WA, Evans JP. Operative treatment of medial epicondyle fractures in children. *Clin Orthop* 1987;221:170–174.
223. Johansson O. Capsular and ligament injuries of the elbow joint. *Acta Chir Scand Suppl* 1962;285.
224. Josefsson PO, Danielsson LG. Epicondylar elbow fracture in children: 35-year follow-up of 56 unreduced cases. *Acta Orthop Scand* 1986;57:311–313.
225. Kilfoyle RM. Fracture of the medial condyle and epicondyle of the elbow in children. *Clin Orthop* 1965;41:43–50.
226. Larsen RL, et al. Little league survey: the Eugene Study. *Am J Sports Med* 1976;4:209.
227. Lokiec F, Velkes S, Engel J. Avulsion of the medial epicondyle of the humerus in arm wrestlers: a report of five cases and a review of the literature. *Injury* 1991;22:69–70.
228. Low BY, Lim J. Fracture of humerus during arm wrestling: report of 5 cases. *Singapore Med J* 1991;32:47–9.
229. Marion J, Faysse R. Fractures de l'epitrochlea. *Rev Chir Orthop* 1962;48:447–469.
230. Masse P. Technique de reduction des luxations du coude avec fracture ou interposition de l'epitrochlea. *Rev Prat* 1955;5:1038.
231. Maylahn DJ, Fahey JJ. Fractures of the elbow in children. Review of three hundred consecutive cases. *JAMA* 1958;18:220–226.
232. Nyska M, Peiser J, Lukiec F, et al. Avulsion fracture of the medial epicondyle caused by arm wrestling. *Am J Sports Med* 1992;20:347–350.
233. Papavasiliou VA. Fracture–separation of the medial epicondylar epiphysis of the elbow joint. *Clin Orthop* 1982;171:172–174.
234. Pappas AM. Elbow problems associated with baseball during childhood and adolescence. *Clin Orthop* 1982;164:30–41.
235. Patrick J. Fracture of the medial epicondyle with displacement into the elbow joint. *J Bone Joint Surg* 1946;28:143–147.
236. Roberts NW. Displacement of the internal epicondyle into the joint. *Lancet* 1934;2:78–79.
237. Rosendahl B. Displacement of the medial epicondyle into the elbow joint: the final result in a case where the fragment has not been removed. *Acta Orthop Scand* 1959;28:212–219.
238. Schmier AA. Internal epicondylar epiphysis and elbow injuries. *Surg Gynecol Obstet* 1945;80:416–421.
239. Schwab GH, Bennett JB, Woods GW, et al. Biomechanics of elbow instability: the role of the medial collateral ligament. *Clin Orthop* 1980;146:42–52.
240. Silberstein JJ, Brodeur AE, Graviss ER, et al. Some vagaries of the medial epicondyle. *J Bone Joint Surg* 1981;63:524–528.
241. Smith FM. Displacement of the medial epicondyle of the humerus into the elbow joint. *Ann Surg* 1946;124:410–425.
242. Smith FM. Medial epicondyle injuries. *JAMA* 1950;142:396–402.
243. Smith FM. *Surgery of the elbow*. Philadelphia: WB Saunders, 1972.
244. Stimson LA. *A treatise on fractures*. Philadelphia: Lea & Sons, 1883.
245. Tachdjian MO. *Paediatric orthopaedics*. Philadelphia: WB Saunders, 1972.
246. Tayob AA, Shively RA. Bilateral elbow dislocations with intra-articular displacement of medial epicondyles. *J Trauma* 1980;20:332–335.
247. Walker HB. A case of dislocation of the elbow with separation of the internal epicondyle and displacement of the latter into the joint. *Br J Surg* 1928;15:667–679.
248. Watson-Jones R. Primary nerve lesions in injuries of the elbow and wrist. *J Bone Joint Surg* 1930;12:121–140.
249. Watson-Jones R. *Fractures and joint injuries*. Baltimore: Williams & Wilkins, 1946.
250. Williams PL, Warwick R. *Gray's anatomy*. Philadelphia: WB Saunders, 1980.
251. Wilson JN. Treatment of fractures of the medial epicondyle of the humerus. *J Bone Joint Surg* 1960;43:778–781.
252. Wilson NIL, Ingran R, Rymaszewski L, et al. Treatment of fractures of the medial epicondyle of the humerus. *Injury* 1988;19:342–344.
253. Wilson PD. Fractures and dislocations in the region of the elbow. *Surg Gynecol Obstet* 1933;56:335–359.
254. Woods GM, Tullos HG. Elbow instability and medial epicondyle fracture. *Am J Sports Med* 1977;5:23–30.

#### Fractures Involving the Lateral Epicondylar Apophysis.

255. Faysse R, Marion J. Fractures de l'epicondyle. *Rev Chir Orthop* 1962;48:471–472.
256. Hasner E, Husby J. Fracture of the epicondyle and condyle of the humerus. *Acta Chir Scand* 1951;101:195.
257. McLeod GG, Gray AJ, Turner MD. Elbow dislocation with intra-articular entrapment of the lateral epicondyle. *J R Coll Surg Edinb* 1993;38:112–113.
258. Ogden JA. *Skeletal injury in the child*, 2nd ed. Philadelphia: WB Saunders, 1990.
259. Rang M. *Children's fractures*, 2nd ed. Philadelphia: JB Lippincott, 1983.
260. Sharrard WJW. *Paediatric orthopaedics and fractures*. Edinburgh: Blackwell Scientific, 1971.
261. Silberstein JJ, Brodeur AE, Graviss ER. Some vagaries of the lateral epicondyle. *J Bone Joint Surg [Am]* 1982;64A:444–448.
262. Watson-Jones R. *Fractures and joint injuries*, 4th ed. Edinburgh: ES Livingstone, 1956.

#### Vascular Necrosis of the Trochlea

263. Beyer WF, Heppt P, Gluckert K, et al. Aseptic osteonecrosis of the humeral trochlea (Hegemann's disease). *Arch Orthop Trauma Surg* 1990;110:45–48.
264. Fowles JV, Kassab MT. Displaced fracture of the medial humeral condyle in children. *J Bone Joint Surg* 1980;62:1159–1163.
265. Graham HA. Supracondylar fractures of the elbow in children (Part 2). *Clin Orthop* 1967;54:93–102.
266. Haraldsson S. The interosseous vasculature of the distal end of the humerus with special reference to capitulum. *Acta Orthop Scand* 1957;27:81–93.
267. Haraldsson S. Osteochondrosis deformans juvenilis capituli humeri including investigation of intraosseous vasculature in distal humerus. *Acta Orthop Scand Suppl* 1959;38.
268. Hegmann G. Die "Spontanen," Aseptischen Knochen-nekrosen des Ellbogengelenkes. *Fortschr Rontgenstr* 1951;75:89–92.
269. Holda ME, Manoli A, LaMont RL. Epiphyseal separation of the distal end of the humerus with medial displacement. *J Bone Joint Surg* 1980;62:52–57.
270. Hurrman WH. Dislocation of the elbow and fractures of the medial humeral epicondyle and condyle. In: Letts RM, ed. *Management of pediatric fractures*. New York: Churchill Livingstone, 1994:225–226.
271. Jakob R, Fowles JV, Rang M, et al. Observations concerning fractures of the lateral humeral condyle in children. *J Bone Joint Surg* 1975;57:430–436.
272. Jakob RP, Fernandez DL. Late complications of fracture of the lateral humeral condyle in children. In: Chapchal G, ed. *Fractures in children. Ninth International Symposium on Topical Problems in Orthopedic Surgery, Lucerne (Switzerland)*. New York: Thieme-Stratton, 1981:170.
273. Kilfoyle RM. Fractures of the medial condyle and epicondyle of the elbow in children. *Clin Orthop* 1965;41:43–50.
274. McDonnell DP, Wilson JC. Fractures of the lower end of the humerus in children. *J Bone Joint Surg* 1948;30:347–358.
275. McIntyre W. Lateral condylar fractures of the humerus. In: Letts RM, ed. *Management of pediatric fractures*. New York: Churchill Livingstone, 1994:254.
276. Mead CA, Martin M. Aplasia of the trochlea—an original mutation. *J Bone Joint Surg [Am]* 1963;45:379–383.
277. Minami A, Sugawara J. Humeral trochlear hypoplasia secondary to epiphyseal injuries as a cause of ulnar nerve palsy. *Clin Orthop* 1988;221:225–230.
278. Morrissey TT, Wilkins KE. Deformity following distal humeral fracture in childhood. *J Bone Joint Surg [Am]* 1984;66A:557–562.
279. Murakami Y, Komiya Y. Hypoplasia of the trochlea and the medial epicondyle of the humerus associated with ulnar neuropathy—report of two cases. *J Bone Joint Surg [Br]* 1978;60:223–225.
280. Potter CMC. Fracture dislocation of the trochlea. *J Bone Joint Surg* 1954;36:248–251.

281. Royle SG, Burke D. Ulna neuropathy after elbow injury in children. *J Pediatr Orthop* 1990;10:495–496.
282. Tanabu S, Yamauchi T, Fukushima M. Hypoplasia of the trochlea of the humerus as a cause of ulnar-nerve palsy. Report of two cases. *J Bone Joint Surg [Am]* 1985;67:151–154.
283. Toniolo RM, Wilkins KE. Avascular necrosis of the trochlea. Scientific Presentation at 15th Annual Meeting of European Orthopaedic Society, April 13, 1996, Prague, Czech Republic.
284. Uhrmacher F. Über Osteochondritis Deformans Juvenilis des Ellenbogengelenkes. *Z Orthop Chir* 1933;59:398–411.
285. Varma BP, Srivastava TP. Fractures of the medial condyle of the humerus in children: a report of 4 cases including the late sequelae. *Injury* 1972;4:171–174.
286. Wadsworth TG. Premature epiphyseal fusion after injury of the capitulum. *J Bone Joint Surg* 1964;46:46–49.
287. Wadsworth TG. *The elbow*. Edinburgh: Churchill Livingstone, 1982:163–171.
288. Wilson JN. Fractures of the external condyle of the humerus in children. *Br J Surg* 1955;43:88–94.
289. Yngve DA. Distal humeral epiphyseal separation. *Orthopaedics* 1985;8:102.
290. Yoo CI, Kim YJ, Suh JT, et al. Orthopedic surgery in Korea. Avascular necrosis after fracture separation of the distal end of the humerus in children. *Orthopaedics* 1992;15:959–963.

#### T-Condylar Fractures

291. Beghin JL, Bucholz RW, Wenger DR. Intercondylar fractures of the humerus in young children. *J Bone Joint Surg [Am]* 1982;64A:1083–1086.
292. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955:56.
293. Bryan RS, Morrey BF. Extensive posterior exposure of the elbow. A triceps-sparing approach. *Clin Orthop* 1982;166:188–192.
294. Burgos J, Gonzalez Herranz R, Amaya S, eds. *Lesiones traumáticas del niño*. Madrid: Editorial Medica Panamericana, 1995.
295. Chapchal G, ed. *Fractures in children*. New York: Thieme-Stratton, 1981.
296. Cotton FJ. Elbow fractures in children. *Ann Surg* 1902;35:75–104.
297. Epright RH, Wilkins KE. Fractures and dislocations of the elbow. In: Rockwood CA, Green DP, eds. *Fractures in adults*. Philadelphia: JB Lippincott, 1975:487–563.
298. Flynn JC, Zink WP. Fractures and dislocations of the elbow. In: MacEwen GD, Kasser JR, Heinrich SD, eds. *Pediatric fractures: a practical approach to assessment and treatment*. Baltimore: Williams & Wilkins, 1993:161.
299. Green DP, McCoy H. Turnbuckle orthotic correction of elbow-flexion contractures after acute injuries. *J Bone Joint Surg [Am]* 1979;61:1092–1095.
300. Green NE, Swiontkowski MF. *Skeletal trauma in children*. Philadelphia: WB Saunders, 1994:241–254.
301. Helfet DL, Hotchkiss RN. Internal fixation of the humerus: a biomechanical comparison of methods. *J Orthop Trauma* 1990;4:258–262.
302. Jarvis JG, D'Astous JL. The pediatric T-supracondylar fracture. *J Pediatr Orthop* 1984;4:697–699.
303. Kasser JR, Richards K, Millis M. The triceps-dividing approach to open reduction of complex distal humerus fractures in adolescents: a cybex evaluation of triceps function and motion. *J Pediatr Orthop* 1990;10:93–96.
304. Kirk P, Goulet JA, Freiberg A, et al. A biomechanical evaluation of fixation methods for fractures of the distal humerus. *Orthop Trans* 1990;14:674.
305. Kocher T. Die Fracturen am Unteren Humerusende. In: *Praktisch Wichtiger Fracturformen*. Basel: Carl Sallmann, 1896.
306. Maylahn DJ, Fahey JJ. Fracture of the elbow in children. *JAMA* 1958;166:220–226.
307. Mehne DK, Mata J. Bicolumn fractures of the adult humerus. Presented at the 53rd Annual Meeting of the American Academy of Orthopedic Surgeons, New Orleans, 1986.
308. Muller ME, Allgöner M, Schneider R, et al. *Manual of internal fixation. Technique recommended by the AO Group*, 2nd ed. New York: Springer, 1979.
309. Ogden JA. *Skeletal injury in the child*, 2nd ed. Philadelphia: WB Saunders, 1990:386.
310. Papvasiliou VA, Beslikas TA. T-condylar fractures of the distal humeral condyles during childhood: an analysis of six cases. *J Pediatr Orthop* 1986;6:300–303.
311. Pollen AG. Fractures and dislocation in children. Baltimore: Williams & Wilkins, 1971.
312. Rang M. *Children's fractures*, 2nd ed. Philadelphia: WB Saunders, 1983:166.
313. Reich RS. Treatment of intercondylar fractures of the elbow by means of traction. *J Bone Joint Surg [Am]* 1936;18:997–1004.
314. Riseborough EJ, Radin EL. Intercondylar T fracture of the humerus in the adult. A comparison of operative and nonoperative treatment in 29 cases. *J Bone Joint Surg [Am]* 1969;51A:130–141.
315. Sanders RA, Raney EM, Pipkin S. Operative treatment of bicondylar intraarticular fractures of the distal humerus, original research. *Orthopaedics* 1992;15:159–163.
316. Schemitsch EH, Tencer AF, Henley MB. Biomechanical evaluation of methods of internal fixation of the distal humerus. *J Orthop Trauma* 1994;8:468–475.
317. Tachdjian MO. *Paediatric orthopaedics*, 2nd ed. Philadelphia: WB Saunders, 1990.
318. Wildburger R, Mahring M, Hofer HP. Supraintercondylar fractures of the distal humerus: results of internal fixation. Review of two consecutive series. *J Orthop Trauma* 1991;5:299–305.
319. Zimmerman H. Fractures of the elbow. In: Weber BG, Brunner C, Freuler F, eds. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980.

# 16 DISLOCATIONS OF THE ELBOW

GEORGE H. THOMPSON

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We thank Kaye Wilkins for his contribution to this chapter and previous editions. Much of this chapter is his effort.

Disruptions of the articular surfaces of the elbow represent a spectrum of injuries ranging from dislocation to subluxation. As such, collectively they are common injuries, although the incidence of each subtype can vary widely. An important anatomic feature of the elbow is its three separate articulations: radiocapitellar, ulnotrochlear, and proximal radioulnar joints. The latter articulation usually functions as a unit but can be disrupted.

## CLASSIFICATION

A modification of the classification of elbow dislocations from the previous edition ( 103) is presented in [Table 16-1](#). Elbow dislocations are described with respect to the position of the proximal radioulnar joint in relation to the distal humerus: posterior, anterior, medial, or lateral. The common posterior dislocation is subdivided into posterolateral and posteromedial dislocations.

Proximal radioulnar joint intact  
Posterior  
  Recurrent posterior  
  Unreduced posterior  
Anterior  
  Medial and lateral  
Proximal radioulnar joint displaced  
Divergent  
Translocation  
Radial head dislocation  
Subluxation of the radial head

Adapted from Wilkins KE, Beatty JH, Chambers HG et al. Fractures and dislocation of the elbow region. In: Rockwood CA, Wilkins KE, Beatty JH eds. *Fractures in Children*, 4th ed. Philadelphia: Lippincott-Raven, 1996.

**TABLE 16-1. CLASSIFICATION ELBOW DISLOCATIONS**

Occasionally, the proximal radioulnar joint is disrupted. Most commonly, the radius and ulna diverge from each other in a mediolateral direction. Rarely, the radius and ulna translocate, with the radius medial and the ulna lateral.

Isolated dislocations of the radial head are uncommon and must be differentiated from congenital dislocations. Isolated dislocations of the proximal ulna are exceedingly rare and have not been reported in children.

Included in this classification is the very common subluxation of the radial head or “pulled” elbow. This is not a true subluxation but rather a partial entrapment of the annular ligament in the radiocapitellar joint.

## POSTERIOR ELBOW DISLOCATIONS

Dislocations of the elbow joint are uncommon in children. Henrikson ( 36) studied 1,579 injuries about the elbow in skeletally immature patients in Gothenburg, Sweden, in 1966, and found only 45 dislocations, for an overall incidence of 3%. He found that the peak incidence of supracondylar fractures was in the first decade of life, whereas that of elbow dislocation was in the second decade, usually between 13 and 14 years of age, when the physes begin to close. The same second-decade peak incidence was reported by Josefsson and Nilsson ( 46) in 1986. In their series, most elbow dislocations occurred in conjunction with sports activities.

Within the past four decades, four series ( 55,71,83,85) have reported elbow dislocations in both adults and children, but some overall statistics regarding the incidence in children can be obtained by combining all four series. In the 317 dislocations reported, including 147 in children, all were posterior (posterior, posterolateral, or posteromedial) except for 10 lateral dislocations, 5 anterior dislocations, and 16 unclassified. Male gender and the left elbow were predominantly involved, and there was a high incidence of associated elbow fractures.

Carloz and Abols ( 11) reported 58 elbow dislocations in children and adolescents, of which all were posterior. The mean age at dislocation was 12 years, with a

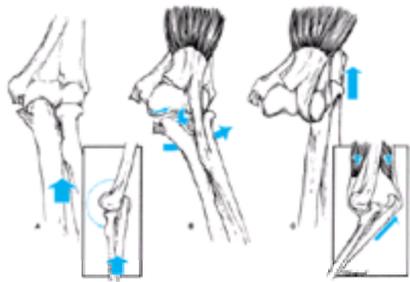
range from 6 to 15 years. There were 41 boys (71%) and 35 left elbows (60%). Associated elbow fractures occurred in 37 children (64%).

## Diagnosis

### Mechanism of Injury

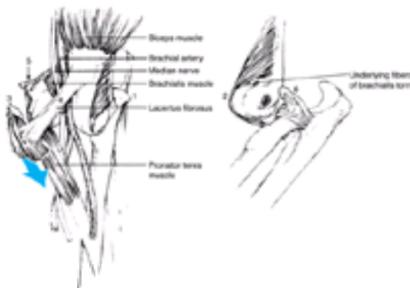
In posterior or posterolateral dislocations, first the medial or ulnar collateral ligaments are disrupted, allowing valgus instability. This can result in an avulsion fracture of the medial epicondyle and its associated flexor muscle origin. The proximal radius and ulna become displaced laterally with the intact biceps tendon acting as a center of rotation for the displaced forearm.

The most commonly accepted mechanism of posterior elbow dislocation involves the application of both abduction and extension forces. However, there is controversy as to whether the elbow is initially hyperextended. Osborne and Cotterill (74) believed that initially the elbow was in slight flexion and that the lateral sloping surface of the medial crista of the trochlea served as a cam mechanism to convert the vertical thrust on the forearm into one of lateral rotation and valgus strain. Other investigators believe that there is first hyperextension of the elbow with rupture of the ulnar collateral ligaments (44,87). Once this stabilizing force is disrupted, the elbow is forced into further valgus (Fig. 16-1). Experimentally, in cadavers, posterior dislocations have been most commonly produced by initial hyperextension (87,96). However, Sojbjerg et al. (90), in a study of 10 cadaver elbows, found that a posterior dislocation is the result of a valgus and external rotation force acting on a semiflexed elbow. Dislocation could not be produced with a varus and internal rotation force. O'Driscoll et al. (73) found similar results in their study on 13 fresh autopsy specimens. They also found that the anterior portion of the medial or ulnar collateral ligament remained intact and attributed to this to the frequent valgus stability following closed reduction.



**FIGURE 16-1.** Mechanism of injury. **A:** Initially the elbow is forced into extension with rupture of the medial collateral ligaments. The normal cubitus valgus accentuates a valgus force at the elbow. **B:** The lateral slope of the medial crista of the trochlea forces the proximal ulna posterolaterally ( *small arrow*). The biceps tendon serves as a fulcrum for rotation and valgus hinging of the forearm. **C:** The proximal ulna and radius are then impacted posteriorly and held against the distal articular surface by the contraction of the biceps and triceps

With posterior dislocations there is considerable soft tissue injury: and an associated risk of neurovascular injuries and associated fractures ( Fig. 16-2).



**FIGURE 16-2. Pathology.** 1: The radial head and olecranon are dislodged posterolaterally. 2: The brachialis muscle is stretched across the articular surface of the distal humerus. 3: The origins of the medial forearm flexion muscles are either torn or avulsed with the medial epicondyle from the medial condyle. 4: The median nerve and brachial artery are stretched across the medial condyle and held firmly by the lacertus fibrosus. 5: The medial condyle lies in the subcutaneous tissue between the brachialis anteriorly and the pronator teres posteriorly. 6: The lateral (radial) collateral ligaments often avulse a piece of cartilage or bone from the lateral condyle

### Soft Tissue Injury

The anterior capsule is ruptured with a tension force that allows the joint cavity to be exposed. Posteriorly, the radial head can strip the capsule from the posterolateral aspect of the lateral condyle, along with the adjacent periosteum. Because of the large amount of cartilage on the posterolateral aspect of the lateral condyle, the posterior capsule may not reattach firmly. This lack of a strong reattachment is believed to be a major factor in recurrent elbow dislocations (74).

Medially, the ulnar collateral ligament system is disrupted either by a direct tear of the ligament or avulsion of the medial epicondyle (87,90). Cromack (14) found that with medial epicondylar fractures, the origins of the ulnar collateral ligaments and medial forearm flexor muscles remain as a unit, along with most of the pronator teres, which is stripped from its humeral origin proximal to the epicondyle. These structures are then displaced posterior to the medial aspect of the distal humerus. If the epicondyle remains attached to the humerus, then the ulnar collateral ligaments and muscular origins of the common flexor muscles must be torn. With posterolateral displacement of the forearm, the medial aspect of the distal humerus dissects the intermuscular space between the pronator teres posteriorly and the brachialis anteriorly. The brachialis, because it has little distal tendon, is easily ruptured. The rent in the anterior capsule usually is in this same area.

On the lateral side, the structure most commonly torn is the annular ligament (90). On rare occasions, the entire lateral collateral ligament either avulses a small osteochondral fragment from the lateral epicondyle or tears completely within its substance.

### Neurovascular Injuries

When the elbow is dislocated, the medial aspect of the distal humerus lies subcutaneously between the pronator teres posteriorly and the brachialis anteriorly. The median nerve and brachial artery lie directly over the humerus in the subcutaneous tissues. In a cadaver and clinical study by Louis et al. (57), there was a consistent pattern of disruption of the collateral arterial system about the elbow. In almost all extremities studied, there was a complete disruption of the anastomosis between the inferior ulnar collateral artery and the anterior ulnar recurrent artery. If the main brachial arterial trunk is injured, the loss of this collateral system can result in severe compromise of circulation to the forearm and hand.

The ulnar nerve also is at risk because of its anatomic site posterior to the medial epicondyle.

## Associated Fractures

Concomitant fractures occur in over one half of posterior elbow dislocations ([55,71,83,85](#)). Fractures involving the medial epicondyle, radial head and neck, and coronoid process are most common. Fractures involving the lateral epicondyle, lateral condyle, olecranon, capitellum, and trochlea occur less frequently. In the 58 dislocations reported by Carioz and Abols ([11](#)), there were 24 fractures involving the medial aspect of the elbow, 6 fractures from the lateral region, 2 with fractures from both regions, 2 coronoid process fractures, and 3 radial neck fractures.

## Signs and Symptoms

Clinically, the presence of a posterior elbow dislocation usually is obvious. The primary differential diagnosis is an extension-type supracondylar fracture of the distal humerus. With both injuries, the elbow is held semiflexed, and swelling may be considerable. Swelling usually is less with a dislocation than with a supracondylar fracture. However, there is still enough soft tissue disruption, such as tearing of the brachialis muscle and collateral vessels, to produce considerable hemorrhage and swelling. Crepitus usually is absent with a dislocation. With a posterior elbow dislocation, the forearm is shortened. The prominence produced by the distal humeral articular surface is more distal and is palpable as a blunt articular surface. The tip of the olecranon is displaced posteriorly and proximally so that its triangular relationship with the epicondyles is lost. Overlying the olecranon fossa, the skin may have a dimpled appearance. If the dislocation is posterolateral, the radial head also may be prominent and easily palpable in the subcutaneous tissues.

## Radiographic and Other Imaging

Routine radiographs usually are diagnostic of a posterior elbow dislocation. In the anteroposterior view, there is a greater superimposition of the distal humerus on the proximal radius and ulna. The radial head may be proximally and laterally displaced, or it may be totally behind the distal humerus, depending on whether it is posterolateral, posterior, or posteromedial ([Fig. 16-3](#)). In addition, the normal valgus angulation between the forearm and the arm usually is increased. On the lateral view, the coronoid process lies posterior to the condyles. The radiographs must be examined closely for associated fractures.



**FIGURE 16-3. Radiographic Findings. A:** Anteroposterior radiograph. The radial head is superimposed behind the distal humerus. There is increased cubitus valgus. The medial epicondyle has not been avulsed. **B:** Lateral radiograph demonstrating that the proximal radius and ulna are both displaced posteriorly to the distal humerus.

## Treatment Options

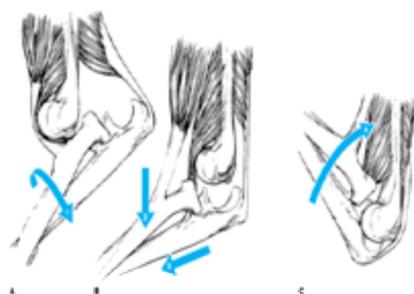
### Nonoperative

#### Closed Reduction

In the combined series ([55,71,83,85](#)) of 317 dislocations, only 2 cases in the series by Linscheid and Wheeler ([55](#)) could not be reduced by closed methods. In the Carioz and Abols series ([11](#)), 2 dislocations reduced spontaneously and closed reduction was successful in 50 cases, but failed in 6 cases (10%). Josefsson et al. ([45](#)) reported that all 25 dislocations without associated fractures were successfully reduced.

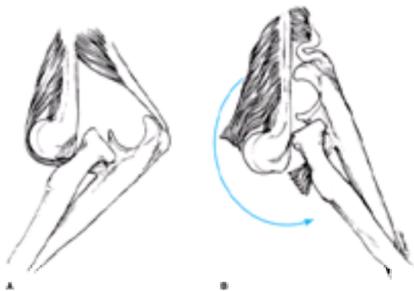
Because of the considerable soft tissue injury, it is essential to reduce the elbow promptly. Royle ([85](#)) found that those reduced soon after the injury had better outcomes than those in which reduction was delayed.

All methods of closed reduction are directed toward sufficiently overcoming the muscle forces so that the coronoid process and radial head can slip past the distal end of the humerus. Just before the reducing forces are applied, the forearm should be hypersupinated to dislodge the coronoid process and radial head from their position behind the distal humerus. The reducing forces must be directed in two major directions ([Fig. 16-4](#)). The first reducing force must be along the long axis of the humerus to overcome the contractions of the biceps and brachialis anteriorly and the triceps posteriorly. Once these forces are neutralized, the proximal ulna and radius must be passed from posterior to anterior. This requires a second force along the long axis of the forearm. This may require that the elbow be extended or even hyperextended to lever the coronoid process past the distal humerus.



**FIGURE 16-4. Reduction forces of posterior dislocations. A:** First the forearm is hypersupinated (*arrow*) to unlock the radial head. **B:** Simultaneous forces must be applied distally along the axis of the humerus (*arrow 2*) and distally along the axis of the forearm (*arrow 3*). **C:** Once the coronoid is manipulated distal to the humerus, the elbow is then flexed (*arrow 4*) to stabilize the reduction.

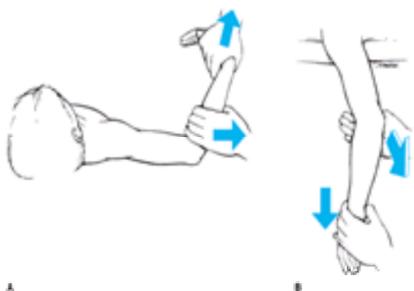
Some investigators ([56,101](#)), however, have strongly advised against initial hyperextension. Loomis ([56](#)) demonstrated that when the coronoid process is locked against the posterior aspect of the humerus and the elbow is extended, the force applied is magnified by as much as five times across the anterior muscles because of the increased leverage. This places a considerable strain on the already injured anterior capsule and brachialis muscle ([Fig. 16-5](#)). On the other hand, when the distal force is applied to the proximal forearm with the elbow flexed, the force exerted against the muscles across the elbow is equal to the distracting force.



**FIGURE 16-5. Hyperextension forces.** **A:** Normally, the brachialis is stretched across the distal humerus. **B:** Hyperextending the elbow before it is reduced greatly increases the arc of motion and leverage placed across the brachialis. This can result in rupture of large portions of the muscle. (Reprinted from Loomis LK. Reduction and after-treatment of posterior dislocation of the elbow. *Am J Surg* 1944;63:56-60; with permission.)

Osborne and Cotterill (74) suggested that the best way to dislodge the coronoid process and radial head from the distal humerus is initially to hypersupinate the forearm and then to apply traction along the forearm with the elbow flexed. Others (7,8) have warned that with posterolateral dislocations, the lateral displacement of the proximal radius and ulna must first be corrected to prevent the median nerve from being entrapped or injured during reduction. Pronating the forearm during reduction also may be a factor in entrapping the median nerve.

Although numerous reduction techniques have been advocated, they all have the common goal of applying the two major traction forces along the axes of the humerus and forearm. Each of these forces is resisted by its respective counterforce. There appears to be two main groups in the method of applying force to counteract the muscles of the arm: the “pullers” (6,15,75) (Fig. 16-6) and the “pushers” (54,66,68) (Fig. 16-7). There also are combined unassisted pusher-puller techniques (30,53).



**FIGURE 16-6. Reduction by “puller” techniques.** **A:** Supine position. With the elbow flexed to almost 90 degrees, a force is applied to the anterior portion of the forearm with one hand while the other hand pulls distally along the forearm. Appropriate counterforces must be applied to counteract these manipulating forces. **B:** Prone position. The same forces are applied to the proximal portion of the anterior forearm and distal forearm. In the prone position the table provides a counterforce against the anterior portion of the distal humerus. (Redrawn from Parvin RW. Closed reduction of common shoulder and elbow dislocations without anesthesia. *Arch Surg* 1957;75:972-975; with permission. Copyright 1957, American Medical Association.)



**FIGURE 16-7. Reduction by “pusher” techniques.** **A:** Lavine's method. The child is held by the parent while the elbow is draped over the edge of the chair. The olecranon is pushed distally past the humerus by the thumb of the physician while the other arm pulls distally along the axis of the forearm. **B:** Meyn's technique with patient lying prone on the table. (Reprinted from Meyn MA, Quigley TB. Reduction of posterior dislocation of the elbow by traction on the dangling arm. *Clin Orthop* 1974;103:106-107; with permission.)

### Postreduction Care

Some type of immobilization, usually a posterior splint, is advocated by most investigators after successful closed reduction. The standard period of immobilization recommended is usually 3 weeks (55,56,75,88), although some have advocated early motion (84,85,108). Ninety degrees of elbow flexion appears to be the standard position of immobilization. O'Driscoll et al. (73) believed that if the elbow was stable to valgus stress with the forearm pronated that the anterior portion of the medial collateral ligament was intact and the patient could begin early motion.

### Surgical Procedures

#### Open Reduction

Indications for primary open reduction are an inability to obtain a concentric closed reduction, an open dislocation, and a displaced osteochondral fracture.

**Inadequate Closed Reduction.** Carlioz and Abols (11) reported that 19 of 58 posterior dislocations required open reduction. In 13, the reduction was stable and the surgery was for the treatment of displaced fractures. In 6, open reduction of the joint was necessary: 2 each for an entrapped medial epicondyle, ulnar osteochondral fragment, and ligamentous fragment.

In adults with posterior elbow dislocations without concomitant fracture, those treated operatively for primary repair of ligaments had no better function or stability than those treated nonoperatively (47,48). The mean lack of elbow extension in both groups was 10 degrees. Similar results were reported by Josefsson et al. (45) in 28 children and adolescents with simple posterior dislocations treated nonoperatively. However, Durig et al. (18) recommended primary operative treatment of uncomplicated dislocations, because early surgery produced satisfactory functional results in all 10 of their patients. Cromak (14) reported full function in 14 children and adolescents treated by early operative repair.

**Open Posterior Dislocations.** Open dislocations usually have a high incidence of arterial injuries ( [34,50,55,57](#)). For this reason, operative intervention usually is necessary for debridement and evaluation of the brachial artery.

**Associated Fractures.** The presence of a displaced fracture, such as the medial epicondyle, is a common indication for surgical intervention ( [11,23,102](#)). Surgery for associated fractures produced slightly more excellent results than nonoperative treatment in the series of Carlioz and Abols ( [11](#)). Similar results were reported by Wheeler and Linscheid ( [102](#)). Fowles et al. ( [23](#)), however, had poorer results in 9 children in whom the medial epicondyles were stabilized surgically. They recommended surgery only for children in whom the medial epicondyle is entrapped or significantly displaced after closed reduction. Repair of an associated medial epicondylar fracture also may improve elbow stability in athletes ( [87,108](#)).

### Postoperative Care

Immobilization after surgery depends on the procedure performed. After open reduction, management is similar to that after satisfactory closed reduction. The length of immobilization for fractures usually is 4 to 6 weeks.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

The “push-off” technique of reduction of an elbow dislocation is preferred in younger children, usually 9 years of age or less. In this age group, the child often can be seated comfortably in the parent's lap ( [Fig. 16-7](#)). Hanging the arm over the back of the chair may provide some stabilization.

For the child 9 years of age or older and the young adolescent, the puller technique advocated by Parvin ( [75](#)) is used ( [Fig. 16-6](#)). The forearm must remain supinated during the process of reduction. Occasionally, it is necessary to hypersupinate the forearm to unlock the coronoid process and radial head before reduction. Closed reduction is done either with heavy sedation or general anesthesia. After reduction, radiographs are obtained to assess the adequacy of the reduction ( [Fig. 16-8](#)). The elbow is immobilized in a posterior splint with the elbow flexed 90 degrees and the forearm in mid-pronation. This forearm position is chosen simply to allow the patient to be more functional in the splint.



**FIGURE 16-8.** Closed reduction. **A:** Anteroposterior radiograph of a 9-year-old girl with a posterior dislocation of the right elbow. The proximal radius and ulna are superimposed on the distal humerus, and there is increased cubitus valgus. **B:** Lateral radiograph shows the proximal radius and ulna posterior to the distal humerus. **C:** Following closed reduction using a puller technique, there has been a concentric reduction. **D:** Lateral radiograph

Because the major complication of elbow dislocations is stiffness, the splint is removed in about 5 days and the patient begins active elbow motion. In 7 to 10 days, the patient can discard the splint and simply use a sling. The emphasis is on early active motion to prevent the stiffness that often occurs after this injury. Before reduction, it is important to emphasize to the parents that there may be some loss of motion, especially extension, regardless of the treatment. This is usually not of functional or cosmetic significance.

A careful neurologic examination must be done before and after the reduction with special attention to the median nerve. This same careful examination must be made at all follow-up evaluations.

### Complications

Complications of posterior elbow dislocations can be divided into those occurring early and those occurring later. Early complications include neurologic and vascular injuries. Late complications include loss of motion, myositis ossificans, recurrent dislocations, radioulnar synostosis, and cubitus recurvatum. The special problems of chronic, unreduced dislocations are not considered complications of treatment.

### Neurologic Injuries

In the combined series ( [55,71,83,85](#)) of 317 patients, 32 patients (10%) had nerve symptoms after reduction. There was no breakdown as to the ages of the patients with the nerve injuries. Twenty-one injuries involved the ulnar nerve, seven the median nerve; in four patients, both the median and ulnar nerves were involved. Most were transient paresthesias with rapid recovery. Only one patient failed to recover fully ( [55](#)). Carlioz and Abols ( [11](#)) reported no neurologic injuries with 58 dislocations in children and adolescents, although two children had transient ulnar nerve symptoms.

### Ulnar Nerve Lesions

There is an increased incidence of ulnar nerve injuries associated with elbow dislocations ( [11,13,24,55,101](#)). There were 21 ulnar nerve injuries plus 4 combined ulnar and median nerve injuries in the combined series ( [55,71,83,85](#)). Watson-Jones ( [101](#)) described 16 nerve lesions in 97 elbow dislocations, 12 of which involved the ulnar nerve; 9 of these there were associated with medial epicondylar fracture. Galbraith and McCullough ( [24](#)) found 6 ulnar nerve injuries in 187 elbow dislocations, with or without fractures, in their study of acute nerve injuries in closed elbow injuries. Four of these patients had medial epicondyle fractures and two had radial head fractures; all had posterior elbow dislocations. Linscheid and Wheeler ( [55](#)) reported neurologic complications in 24 of 110 elbow dislocations, including 16 ulnar nerve injuries alone and 4 injuries to both the ulnar and median nerves. They recommended ulnar nerve transposition if ulnar nerve symptoms were present in a patient undergoing open reduction and internal fixation of a displaced medial epicondylar fracture. Cotton ( [13](#)) reported 10 children with posterior dislocations associated with medial epicondylar fractures and ulnar nerve neuropathy. Except for the one patient described by Linscheid and Wheeler ( [55](#)), these reported ulnar nerve injuries were transient and resolved completely.

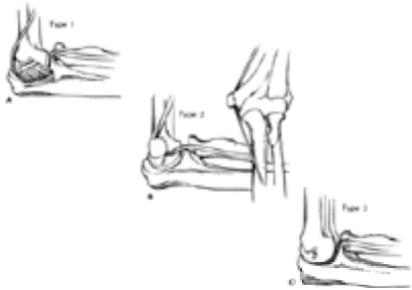
### Radial Nerve Lesions

Watson-Jones ( [101](#)) is the only investigator to report a radial nerve injury associated with an elbow dislocation. He described two patients in whom the symptoms rapidly resolved after reduction.

### Median Nerve Lesions

The most serious neurologic injury involves the median nerve, which can be damaged directly by the dislocation or can be entrapped within the joint. Median nerve injuries occur most commonly in children 5 to 12 years of age. There were seven median and four median-ulnar nerve injuries (3%) in the combined series ( [55,71,83,85](#)).

**Types of Median Nerve Entrapment.** Fourrier et al. (21) in 1977 delineated three types of medial nerve entrapment (Fig. 16-9):



**FIGURE 16-9.** Median nerve entrapment. **A:** Type 1. Entrapment within the elbow joint with the median nerve coursing posterior to the distal humerus. **B:** Type 2. Entrapment of the nerve between the fracture surfaces of the medial epicondyle and the medial condyle. **C:** Type 3. Simple kinking of the nerve into the anterior portion of the elbow joint. (Redrawn from Hallett J. Entrapment of the median nerve after dislocation of the elbow. *J Bone Joint Surg [Br]* 1981;63:408-412; with permission.)

**Type 1.** Either avulsion of the medial epicondyle along with the superficial head of the pronator teres muscle or simple rupture of the muscle origins and ulnar collateral ligaments (Fig. 16-9A) allows the median nerve, with or without the brachial artery, to become posteriorly displaced. If the lateral displacement of the proximal radius and ulna is not corrected before reduction, the nerve is especially prone to being entrapped between the trochlea and the olecranon during the process of reduction. Hallett (29) demonstrated in cadavers that pronation of the forearm while the elbow is hyperextended, forces the median nerve posteriorly during the process of reduction, making it also vulnerable to entrapment. This type of entrapment has been reported frequently (4,7,8,16,21,27,63,78,80,94). Delay in diagnosis is common. The medial epicondyle is commonly fractured. In a few patients, the nerve was so severely damaged after being entrapped that resection of the injured portion with reanastomosis was necessary (7,27,63). Good recovery of function was reported.

If the nerve has been entrapped for a considerable period, the Matev sign may be present on the radiographs (63). This represents a depression on the posterior surface of the medial epicondylar ridge where the nerve has been pressed against the bone (Fig. 16-10) (4,16,27,29,78,80,94). This groove is represented radiographically by two sclerotic lines parallel to the nerve. This sign disappears when the pressure from the nerve has been released.



**FIGURE 16-10.** The Matev sign impingement of the median nerve against the posterior surface of the medial condyle produces a depression with sclerotic margins. (Redrawn from Matev I. A radiographic sign of entrapment of the median nerve in the elbow joint after posterior dislocation. *J Bone Joint Surg [Br]* 1976;58:353-355; with permission.)

**Type 2.** The nerve is entrapped between the fracture surfaces of the medial epicondyle and the distal humerus (Fig. 16-9B). The fracture heals and the nerve is surrounded by bone, forming a neoforamen (78,82,94). This may or may not be visible radiographically. The medial epicondyle is osteomized to free the nerve.

**Type 3.** The nerve is kinked and entrapped between the distal humerus and the olecranon (Fig. 16-9C). Only three injuries of this type have been reported (4,77,79). Resection and reanastomosis resulted in return of good function over 6 to 24 months.

Al Qattan et al. (2) described a fourth type of median nerve entrapment. In a 14-year-old boy the median nerve was trapped in a healed medial epicondylar fracture (type 2) in an anterior to posterior direction and then passed through the elbow in a posterior to anterior direction (type 1) 18 months after a posterior dislocation with a medial epicondylar fracture. The nerve was so severely damaged that it was resected and repaired with sural nerve grafts.

The combination of an associated fracture of the medial epicondyle and significant median nerve dysfunction was cited by Rao and Crawford (80) as an absolute indication for surgical exploration of the nerve because of the frequency of median nerve entrapment with fractures of the medial epicondyle (types 1 and 2). Magnetic resonance imaging may be helpful in defining the course of the median nerve if entrapment is suspected. Once the entrapped nerve is removed from the joint, neurologic function typically improves even if the nerve appears seriously damaged. Resection and reanastomosis rarely are necessary.

### Arterial Injuries

Arterial injuries are common with posterior elbow dislocations in children and adolescents (28,34,35,38,50,57,61,76,86,105). Only eight vascular injuries (3%) were reported in the combined series (55,71,83,85). However, Carlioz and Abols (11) reported four patients with diminished radial pulses that resolved after reduction. Arterial injuries frequently are associated with open dislocations in which collateral circulation is disrupted (34,50,57,86), and they also have been reported in association with closed dislocations (28,35,38,105). Usually the brachial artery is ruptured (28,34,38,50,57,61), but it also can be thrombosed (105) as well as entrapped in the elbow joint (35,76,105). Pearce (76) reported an entrapped radial artery in which there was a high bifurcation of the brachial artery.

When there is a complete rupture, there usually is evidence of severe ischemia distally. Unfortunately, the presence of good capillary circulation to the hand or a Doppler pulse at the wrist does not always mean the artery is intact (28,38). Arteriograms usually are not necessary because the arterial injury is at the site of the dislocation. A new technique using intravenous digital angiography has been used to confirm complete obstruction of the brachial artery if there is clinical evidence of distal ischemia (38).

Treatment usually consists of relocation of displaced brachial arteries (35,105) and operative repair of those that are ruptured or severely damaged. Ligation of the ends has been done, especially if there was good capillary circulation distally (34,50), but this may predispose to late ischemic changes such as claudication and cold sensitivity. Most investigators recommend direct arterial repair or a vein graft (28,38,57,61,86). Louis et al. (57) recommended arterial repair because their cadaver studies demonstrated that a posterior elbow dislocation usually disrupted the collateral circulation necessary to maintain distal blood flow.

### Loss of Motion

Almost all patients with elbow dislocations lose some range of elbow motion (11,23,45,47,48). This loss is less in children than in adults (45) usually only 5 to 10 degrees of extension, and rarely is of functional or cosmetic significance. However, this potential for loss of motion must be explained to the parents before reduction

and may be an indication for a supervised rehabilitation program.

### **Myositis Ossificans versus Heterotopic Calcification**

True myositis ossificans should be differentiated from heterotopic calcification, which is a dystrophic process. Myositis ossificans involves ossification within the muscle sheath that can lead to a significant loss of range of motion of the elbow. Disruption of the brachialis muscle is believed to be a contributory factor ( 56). Fortunately, myositis ossificans is rare in children ( 45,97). Although heterotopic calcification in the ligaments and capsule of the elbow is common ( 45,83) it rarely results in loss of elbow function ( Fig. 16-11).



**FIGURE 16-11.** Heterotopic calcification. **A:** An elbow that had been dislocated 2 months previously. There is heterotopic calcification in the ulnar collateral ligaments (arrow). **B:** Lateral view of the same elbow. Some true ossification has occurred where the brachialis inserts into the coronoid process ( arrow)

Thompson and Garcia (97) found no myositis ossificans in 8 of 10 children 0 to 15 years of age with elbow injuries. The two children with myositis ossificans had supracondylar fractures. In Neviasser and Wickstrom's (71) series of 115 patients, 10 had radiographic evidence of myositis ossificans; all, however, were asymptomatic. Only Roberts (83) differentiated true myositis ossificans from heterotopic calcification. In his series of 60 elbow dislocations, only 3 patients had true myositis ossificans. Linscheid and Wheeler (55) noted that the incidence of some type of heterotopic calcification was 28%, which was most common around the condyles. Only in 5 patients was it anterior to the capsule (which probably represented true myositis ossificans in the brachialis muscle). Four of these patients had some decrease in elbow function. Josefsson et al. (45) reported that 61% of 28 children with posterior dislocations had periarticular calcification, but this did not appear to be functionally significant.

### **Recurrent Posterior Dislocations**

Recurrent posterior elbow dislocation is rare. It is discussed in detail in the next section. In the combined series of dislocations, only two (0.1%) recurrent dislocations were reported (55,71,83,85).

### **Radioulnar Synostosis**

In dislocations with an associated fractures of the radial neck, the incidence of a secondary proximal radioulnar synostosis is increased ( Fig. 16-12). This can occur regardless of whether the radial neck fracture is treated operatively or nonoperatively ( 8,11,73). Carlioz and Abols (11) reported a synostosis in one of three patients with a posterior elbow dislocation associated with radial neck fractures.



**FIGURE 16-12.** Radioulnar synostosis. **A:** Injury radiographs demonstrating a posterolateral dislocation associated with a type II proximal radius epiphyseal fracture (arrow). Open reduction of the proximal radial fracture was performed. **B:** Six months later, the patient developed a proximal radioulnar synostosis ( arrow). (Courtesy of Ruben Pachero, M.D.)

### **Cubitus Recurvatum**

Occasionally, a severe elbow dislocation results in significant tearing of the anterior capsule. As a result, after reduction, when all the stiffness created by the dislocation has subsided, the patient may have some hyperextension (cubitus recurvatum) of the elbow. This usually is minimally symptomatic.

### **Recurrent Posterior Elbow Dislocations**

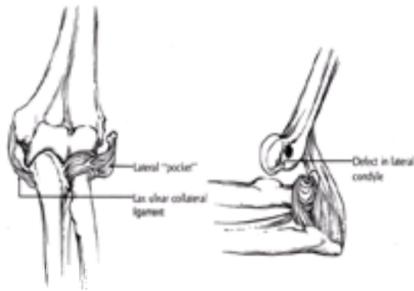
Although recurrent posterior elbow dislocations almost always involve adult patients ( 18,26,32,37,43,49,51,60,62,65,67,72,74,95,98,100,107,109) the initial dislocation has occurred before skeletal maturity. Approximately 80% of recurrent dislocations are in males. Three investigators have reported bilateral cases ( 49,67,81).

### **Diagnosis**

#### **Mechanism of Injury**

The pathology of recurrent dislocation involves the collateral ligaments, capsular laxity, and bone defects.

**Ligamentous and Capsular Laxity.** Osborne and Cotterill (74) suggested that articular changes are secondary and that the primary defect is a failure of the posterolateral ligamentous and capsular structures to become reattached after reduction ( Fig. 16-13). Posterior dislocations usually cause attenuation of the ulnar collateral ligaments, and this further contributes to the instability. With recurrent dislocations, the radial head impinges against the posterolateral margin of the capitellum, creating an osteochondral defect ( Fig. 16-14).



**FIGURE 16-13.** Pathology of recurrent dislocation. The three components that allow the elbow to dislocate: a lax ulnar collateral ligament, a pocket in the radial collateral ligament, and a defect in the lateral condyle. (Reprinted from Osborne G, Cotterill P. Recurrent dislocation of the elbow. *J Bone Joint Surg [Br]* 1966;48:340-346; with permission.)

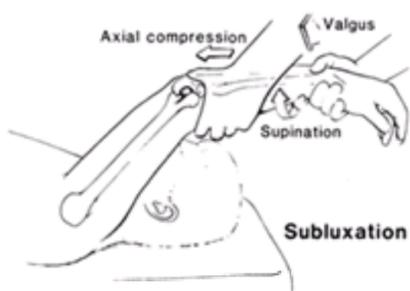


**FIGURE 16-14.** Pathologic changes of recurrent dislocation. **A:** Anteroposterior radiograph of a 13-year-old who had recurrent dislocations. An osteochondral fragment (*arrow*) is attached to the lateral ligament. **B:** An oblique radiograph shows the defect (*arrow*) in the posterolateral condylar surface. **C:** Radiographs of an 11-year-old after his first dislocation. **D:** One year later, after recurrent dislocation and subluxations, blunting of the radial head has developed (*arrow*). (Courtesy of Marvin E. Mumme, M.D.)

Osborne and Cotterill (74) attributed the failure of the capsule to reattach to the presence of a considerable amount of articular cartilage, which provides a poor surface for reattachment, and synovial fluid, which further inhibits healing. With recurrent dislocations, secondary changes tend to develop ( Fig. 16-14). In addition to the defect in the capitellar articular surface, a similar defect develops in the anterior articular margin of the radial head. When these two defects appose each other, recurrence of the dislocation is more likely. Subsequent studies have confirmed these findings in almost all recurrent dislocations, especially in children (18,32,72,95,98,107).

O'Driscoll et al. (88) described posterolateral instability in five patients, including two children, in whom laxity of the ulnar part of the radial collateral ligament allowed a transitory rotary subluxation of the ulnohumeral joint and a secondary dislocation of the radiohumeral joint. Patients with posterolateral instability often describe a history of recurrent temporary dislocation of the elbow, but when examined exhibit no unusual clinical findings.

The instability is diagnosed with a posterolateral rotary instability test. In some patients posterolateral rotary instability can be detected only with the patient completely relaxed under general anesthesia. This test is performed by holding the arm over the head while applying proximal axial compression plus a valgus and supination force to the forearm with the elbow flexed to only 20 to 30 degrees ( Fig. 16-15). O'Driscoll et al. reported that surgical repair of the lax ulnar portion of the radial collateral ligament eliminated the posterolateral rotary instability ( 72).



**FIGURE 16-15.** Posterolateral rotary instability. Posterolateral rotational instability is best demonstrated with the upper extremity over the radial head. The radial head can be subluxed or dislocated by applying a valgus and supination force to the forearm at the same time proximal axial compression is applied along the forearm. (Reprinted from O'Driscoll SW, Bell DF, Morrey BF. Posterolateral rotary instability of the elbow. *J Bone Joint Surg [Am]* 1991;73:441; with permission.)

Schwab et al. (87) described treatment of the laxity of the medial collateral ligament with advancement of the medial epicondyle proximally to tighten the medial collateral ligament.

**Bone Defects.** In addition to the previously described osteochondral defects in the capitellum and radial head, bone defects include a shallow semilunar notch resulting from a coronoid fossa process fracture or multiple recurrent dislocations. Osteochondral fragments in the elbow joint also can contribute to instability.

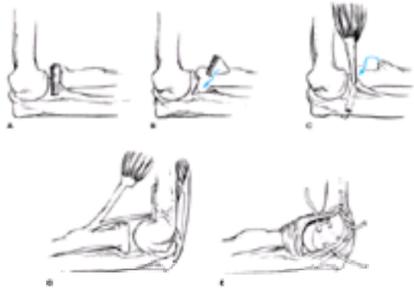
### Treatment Options

#### Nonoperative

There appears to be only one report of successful nonsurgical management of recurrent elbow dislocations. Herring ( 37) used an orthosis that blocked the last 15 degrees of extension. After his patient wore this orthosis constantly for 2 years and with vigorous activities for another 6 months, there were no further dislocations, but the follow-up period was only 1 year. Beatty and Donati ( 5) emphasized that physical therapy and the use of an orthosis should be tried before surgery is considered.

#### Surgical Procedures

The treatment of recurrent posterior elbow dislocations is predominately surgical. Various surgical procedures have been described to correct bone and soft tissue abnormalities ( Fig. 16-16).



**FIGURE 16-16.** Surgical procedures for recurrent dislocation. **A:** Bone block. **B:** Coronoid osteotomy. **C:** Biceps tendon transfer. **D:** Cruciate ligament reconstruction. **E:** Lateral capsular reattachment of Osborne and Cotterill

**Bone Procedures.** These are directed more toward correcting dysplasia of the semilunar notch of the olecranon. Milch (67) inserted a boomerang-shaped bone block. Others (26,65,100) found that a simple bone block was all that was necessary (Fig. 16-16A). Mantle (62) increased the slope of the semilunar notch in two patients with an opening wedge osteotomy of the coronoid process (Fig. 16-16B).

Schwab et al. (87), believing the ulnar collateral ligament to be the mainstay of elbow stability, advocated tightening the medial side by osteotomizing the medial epicondyle and reattaching it proximally, but reported no results.

**Soft Tissue Procedures.** Reichenheim (81) and King (51) transferred the biceps tendon just distal to the coronoid process to reinforce it (Fig. 16-16C). Kapel (49) developed a cruciate ligament reconstruction in which distally based strips of the biceps and triceps tendon were passed through the distal humerus and sutured to the olecranon and coronoid process respectively (Fig. 16-16D). Beaty and Donati (5) modified this technique by transferring a central slip of the triceps through the humerus posterior to anterior and attaching it to the proximal ulna.

The most widely accepted technique is that described by Osborne and Cotterill (74), in which the lateral capsule is reattached to the posterolateral aspect of the capitellum with sutures passing through holes drilled in the bone (Fig. 16-16E). The joint should be inspected at surgery because osteocartilagenous loose bodies may be present (32,60,95). Since Osborne and Cotterill's (74) initial report of eight patients, successful use of this technique has been reported in numerous others (18,32,60,72,95,98,107). Zeier (109) and O'Driscoll et al. (72) reinforced the lateral repair with strips of fascia lata, triceps fascia, or palmaris longus tendon. Malkawi (60) transferred the ulnar nerve and reinforced the medial repair in a patient with ulnar neuropathy.

#### Posttreatment Care

Postoperatively, especially after the repair described by Osborne and Cotterill (74), the arm is immobilized in a long arm cast with the elbow flexed 90 degrees for approximately 4 weeks. Active range of motion exercises are then begun.

#### Complications

The major complication after correction of recurrent dislocations is loose osteocartilagenous fragments and destruction of the articular surface of the joint (Fig. 16-17). Loose fragments must be removed at initial surgery and later should they occur.



**FIGURE 16-17.** Effects of recurrent dislocation. This girl began to have recurrent dislocations of her elbow at age 9. **A:** The ease at which the elbow redislocates is shown in this radiograph. **B and C:** Radiographs taken at the beginning of episodes of dislocation. Her dislocation continued. **D and E:** Four years later the elbow demonstrated marked changes in its architecture. (Courtesy of David J. Mallams, M.D.)

#### Unreduced Posterior Elbow Dislocations

Untreated or neglected posterior dislocations of the elbow in children are extremely rare in North America. Most reported series are from other countries (1,22,52,59,69,89). In 1925, Speed (93) from the United States reported six, including four in children.

#### Diagnosis

Children with untreated dislocations typically have pain and limited mid-range of motion. Pathologically, there is subperiosteal new bone formation that produces a radiohumeral horn, myositis ossificans of the brachialis muscle, capsular contractures, shortening of the triceps muscle, contractures of the medial and lateral collateral ligaments, and tightening of the ulnar nerve (21,22,89). These factors have to be considered when planning treatment.

#### Treatment Options

##### Nonoperative

Closed reduction of dislocations recognized within 3 weeks of injury may be possible (1,22). If this fails or if the dislocation is of longer duration, open reduction is necessary.

##### Surgical Procedures

Open reduction through a posterior approach, as described by Speed (93), involves lengthening of the triceps muscle and release or transposition of the ulnar nerve (22,69,89). Others have advocated a posterolateral or medial and lateral incisions for better exposure (1,52). Satisfactory results usually can be obtained if a stable concentric reduction is achieved within 3 months of the initial dislocation (1,52). Results decline thereafter but still may produce some improvement in function (22,69) (Fig. 16-18). Internal fixation of the elbow to maintain reduction is used for 2 to 4 weeks, followed by vigorous physical therapy (22,69). Two large Kirschner wires or

Steinmann pins may be used to stabilize the elbow and minimize the risk of intraarticular breakage.



**FIGURE 16-18.** Unreduced dislocation. **A:** Preoperative anteroposterior radiograph. The elbow sustained an injury 3 years before surgery. Elbow motion was extremely limited and painful. The lateral supracondylar ridge had been eroded by the radial head ( *arrow*). **B:** Lateral radiograph. The posterior position of the olecranon is apparent. **C:** Anteroposterior radiograph 3 months postoperatively. Total elbow motion was 30 degrees, but there was less pain and more stability

Mahaisavariya et al. (59) reported decreased flexion contracture and better functional results in 34 patients with unreduced elbow dislocations 1 to 3 months after injury in whom the triceps tendon was not lengthened compared with 38 patients who had the triceps lengthened at surgery.

### Congenital Elbow Dislocations

It must be remembered that a chronic elbow dislocation may be congenital. Because of the abnormal nature of these elbows, they are prone to injury. In addition, because of the relatively free range of motion of the elbow before injury, the child or parents may not have recognized that the elbow has been dislocated since birth.

The key to differentiating a congenital from an acute traumatic elbow dislocation is examination of the radiographic architecture of the articulating surfaces. In a congenitally dislocated elbow, there is marked atrophy of the humeral condyles and the semilunar notch of the olecranon ( Fig. 16-19). However, these same changes can result from chronic recurrent dislocation after trauma, making the differentiation between congenital and traumatic difficult. Certainly, if other congenital anomalies are present or the child has an underlying syndrome, such as Ehlers-Danlos or Larsen's syndrome, the dislocation is likely to be congenital.



**FIGURE 16-19.** Congenital elbow dislocation. Lateral (**A**) and anteroposterior (**B**) radiographs of an elbow with congenital dislocation. The patient had sustained a minor injury. The fact that similar findings were seen in the opposite uninjured elbow confirms the congenital origin. (Courtesy of Earl A. Staple, M.D.)

### Posteromedial Dislocation

Posteromedial elbow dislocations are rare. One patient described by Wilkins et al. ( 103) developed a recurrent dislocation. This 12-year-old boy had trochlear hypoplasia secondary to a very distal supracondylar fracture ( Fig. 16-20). It was believed that hypoplasia of the trochlea contributed to the instability of the medial aspect of his elbow joint, predisposing him to recurrent dislocations. As he grew, his medial ligaments tightened, and subsequently he became asymptomatic.



**FIGURE 16-20.** Posteromedial dislocation. **A:** At 6 years of age, this boy sustained what was thought to be a simple undisplaced but very distal supracondylar fracture. This radiograph taken 4 weeks after injury shows periosteal new bone formation along both supracondylar columns ( *arrow*). **B:** Four years later, he began sustaining recurrent posteromedial dislocations. **C:** Radiograph after reduction, at that time, shows delayed development of the medial ossification center of the crista ( *arrow*). It was thought that this hypoplastic trochlea contributed to his elbow instability. (Courtesy of Stephen A. Cord, M.D.)

## ANTERIOR ELBOW DISLOCATIONS

Anterior elbow dislocations are also rare. Of the 317 elbows in the combined series ( 55,71,83,85), only five were anterior, for an incidence of slightly over 1%. They are associated with an increased incidence of complications, such as brachial artery disruption and associated fractures, compared with posterior dislocations (12,40,42,92,104,106).

### Diagnosis

#### Mechanism of Injury

Anterior elbow dislocations usually are caused by a direct blow to the posterior aspect of the flexed elbow ( 40,106). However, hyperextension of the elbow also has

been implicated ([104](#)). Twisting of the forearm on the elbow commonly occurs.

### Signs and Symptoms

The elbow is in extension with fullness in the antecubital fossa. Swelling usually is marked because of the soft-tissue disruption associated with this type of dislocation. There is severe pain with attempted motion. A careful neurovascular examination is mandatory.

### Radiographic and Other Imaging Studies

Routine anteroposterior and lateral radiographs are diagnostic. In most cases, the proximal radius and ulna dislocate anteromedially rather than purely anteriorly ([Fig. 16-21](#)). Associated fractures are common. In children, the triceps insertion may be avulsed from the olecranon with a small piece of cortical bone ([106](#)). This fracture usually reattaches to the olecranon after reduction. Wilkerson ([104](#)) reported an anterior dislocation associated with a displaced olecranon fracture in a 7-year-old boy. Inoue and Horii ([40](#)) reported an 11-year-old girl with an anterior elbow dislocation with displaced fractures of the trochlea, capitellum, and lateral epicondyle. These were repaired with open reduction and internal fixation using Herbert bone screws.

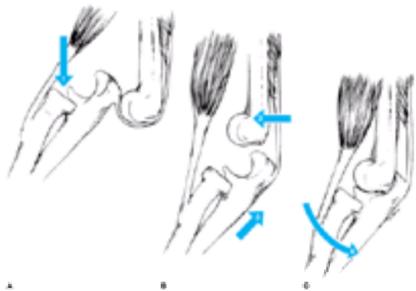


**FIGURE 16-21.** Anterior dislocation of the elbow. **A:** Initial lateral radiograph. The olecranon lies anterior to the distal humerus. **B:** Initial anteroposterior radiograph. The proximal ulna and radial head lie anteromedial, and the elbow carrying angle has drifted into varus. (Courtesy of Hilario Trevino, M.D.)

### Treatment Options

#### Nonoperative: Closed Reduction

Reduction usually is accomplished by flexing the elbow and pushing the forearm proximally and downward at the same time ([106](#)). As for the posterior dislocations, a force must first be applied longitudinally along the axis of the humerus with the elbow semiflexed to overcome the forces of the biceps and triceps. The longitudinal force along the axis of the forearm is directed toward the elbow ([Fig. 16-22](#)). To make reduction easier, the distal humerus can be forced in an anterior direction by pushing on the posterior aspect of the distal arm.



**FIGURE 16-22.** Reduction of anterior dislocation. **A:** With the elbow semiflexed, a longitudinal force is applied along the long axis of the humerus (*arrow 1*). Pulling distally on the forearm may be necessary to initially dislodge the olecranon. **B:** Once the olecranon is distal to the humerus, the distal humerus is pushed anteriorly (*arrow 2*) while a proximally directed force is applied along the long axis of the forearm (*arrow 3*). **C:** Finally, the elbow is immobilized in some extension (*arrow 4*).

### Surgical Procedures

Surgery usually is not required unless the dislocation is open, the brachial artery injury is present, or there is an associated fracture that does not realign satisfactorily after closed reduction. Open reduction and internal fixation of the fracture may then be necessary ([40,104](#)).

### Postreduction Care

Because most anterior dislocations occur in flexion, the elbow should be immobilized in some extension for 2 to 3 weeks, followed by active range of motion exercises. Early motion after open reduction and internal fixation of an associated olecranon fracture usually can be allowed ([40,104](#)).



### AUTHOR'S PREFERRED METHOD OF TREATMENT

Closed reduction ([Fig. 16-22](#)) is the initial procedure of choice. A distal force must be applied in line with and parallel to the long axis of the humerus first. Once the length has been reestablished, a posteriorly directed force along the axis of the forearm is applied until the elbow is reduced.

### Complications

There appears to be an increased incidence of brachial artery rupture or thrombosis associated with anterior elbow dislocations ([42,92](#)). When present, prompt arterial repair is necessary.

### MEDIAL AND LATERAL ELBOW DISLOCATIONS

These are rare dislocations. Lateral dislocations, either incomplete or complete, are more common than medial dislocations. There are no recent reports of medial dislocations in children.

### Diagnosis

## Signs and Symptoms

In an incomplete lateral dislocation, the semilunar notch articulates with the capitulotrochlear groove and the radial head appears more prominent laterally. There may be reasonably free extension and flexion, so lateral dislocation may be overlooked. In a complete lateral dislocation, the olecranon is totally lateral to the capitellum. This gives the elbow a markedly widened appearance.

## Radiographic and Other Imaging Studies

Anteroposterior and lateral radiographs of the elbow usually are diagnostic. On the lateral view the elbow may appear reduced.

## Treatment Options

These rare dislocations are treated by closed reduction. A longitudinal force is applied along the axis of the humerus to distract the elbow, and then direct medial or lateral pressure (opposite the direction of the dislocation) is applied over the proximal forearm ( [Fig. 16-23](#)).



**FIGURE 16-23.** Lateral elbow dislocation. **A:** Initial anteroposterior radiograph in this 6-year-old with a lateral dislocation and displaced medial epicondyle fracture. **B:** Lateral radiograph shows slight posterior dislocation. **C and D:** Postreduction radiographs demonstrate anatomic reduction of the dislocation. The medial epicondyle is satisfactorily aligned.

## DIVERGENT ELBOW DISLOCATION

Divergent dislocation represents a posterior elbow dislocation with disruption of the interosseous membrane between the proximal radius and ulna with the radial head displaced laterally and the proximal ulna medially ( [Fig. 16-24](#)). These dislocations are extremely rare ( [3,9,17,20,33,39,64,70,88,91,99](#)).



**FIGURE 16-24.** Medial-lateral divergent dislocation. **A:** Anteroposterior view demonstrating disruption of the proximal radioulnar joint with the radius lateral and the ulna medial. **B:** Lateral radiograph confirms that the radius and ulna are both posterior to the distal humerus. **C:** A radiograph taken 4 weeks after injury shows periosteal new bone formation (*arrows*), indicating where the soft tissues were extensively torn away from the proximal ulna.

Divergent dislocations usually are caused by high-energy trauma. Associated fractures of the radial neck, proximal ulna, and coronoid process are common ( [9,20,99](#)). It has been speculated that, in addition to the hyperextension of the elbow that produces the dislocation, a strong proximally directed force is applied parallel to the long axis of the forearm, disrupting the annular ligament and interosseous membrane and allowing the divergence of the proximal radius and ulna.

## Treatment Options

### Nonoperative: Closed Reduction

Divergent dislocations are typically easily reduced via the closed method with general anesthesia ( [Fig. 16-24A](#)). Reduction is achieved by applying longitudinal traction with the elbow semiextended and at the same time compressing the proximal radius and ulna together.

### Surgical Procedures: Open Reduction

This is rarely indicated. There have been only two divergent dislocations reported that required open reduction ( [20,64](#)). Closed reduction failed in one child ( [64](#)), and the other had a displaced fracture of the proximal ulna ( [20](#)).

### Postreduction Care

After successful closed reduction, the elbow is immobilized in 90 degrees of flexion and the forearm in neutral for approximately 2 to 3 weeks. Active range of motion exercises are then begun. Most patients typically regain full elbow motion, including forearm pronation and supination.

## PROXIMAL RADIOULNAR TRANSLOCATION

Translocation of the proximal radius and ulna is another extremely rare injury ( [9,10,19,25,31,41,58](#)). It is commonly missed on the anteroposterior radiograph unless the proximal radius and ulna are noted to be completely reversed in relation to the distal humerus.

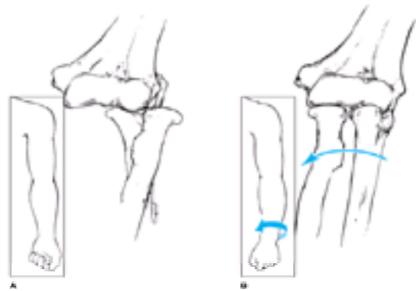
Translocations are believed to be caused by a fall onto the pronated hand with the elbow in full or nearly full extension, producing an axial force on the proximal radius. The anterior radial head dislocation occurs first, followed by the posterior dislocation. The radial head, depending on the degree of pronation, can be lodged in the coronoid fossa or dislocated posteriorly. More pronation allows the radial head to dislocate posteriorly beneath the trochlea. As a consequence, fractures of the radial head, radial neck, or coronoid process may occur ( [9,10,19,58](#)). Isbister ( [41](#)), however, believed the etiology may be iatrogenic: an inappropriate reduction

technique of the posterior dislocation.

## Treatment Options

### Nonoperative: Closed Reduction

Closed reduction with general anesthesia can be attempted but is usually unsuccessful ( [Fig. 16-25](#)). MacSween ([58](#)) reported a successful closed reduction with the elbow flexed 100 degrees and the forearm fully supinated.



**FIGURE 16-25.** Proximal radioulnar translocation. **A:** Position of the proximal radius and ulna with a proximal radioulnar translocation. **B:** If the forearm is forced into pronation during reduction, the ulna may become lodged in the capitulotrochlear groove and the radius forced anterior to the trochlea. (Redrawn from Harvey S, Tchelebi H. Proximal radio-ulnar translocation. *J Bone Joint Surg* 1979;61:447-449; with permission.)

### Surgical Procedures: Open Reduction

Most reported radioulnar translocations have required open reduction ( [9,10,19,25,31,41](#)). Internal fixation also may be necessary for an associated displaced fracture. Harvey and Tchelebi ([31](#)) used an osteotomy of the proximal ulna to expose and reduce the radius. This was complicated by a postoperative ulnar nerve paralysis that recovered completely over 2 months.

### Postreduction Care

After successful closed or open reduction, a long arm cast with the forearm in neutral position is worn approximately 4 weeks, followed by an active range of motion exercises. The presence of a fracture may prolong the immobilization. Mild residual loss of motion is a common sequela of proximal radioulnar translocation.

## RADIAL HEAD DISLOCATION

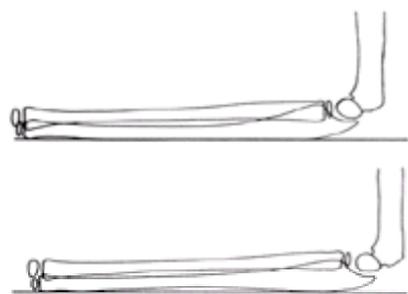
Isolated traumatic dislocation of the radial head is an uncommon injury. Most reports have single case reports ( [113,121,124,136,140,143,145](#)), but some small series have been reported ( [117,118,126,127,129,130,135,137,141,144,145](#) and [146,148,149](#)). The diagnosis usually is missed initially.

In reports in which ages were given, the mean was 7 years. Vesely ([146](#)) combined the experience of the members of the Association of Bone and Joint Surgeons to accumulate 17 dislocations; 13 were anterior, 3 were lateral, and 1 was posterior. Two recurred.

### Unusual Dislocation Patterns

Isolated radial head dislocations are variants of Monteggia lesions ( [118,123,126,127,129,133](#)). Often, if the patient and the radiographs are carefully examined, an occult fracture of the olecranon or proximal ulna is discovered ( [127](#)). The fracture may not be apparent until bowing of the proximal ulna or olecranon develops during the healing phase ( [118](#)).

Lincoln and Mubarak ([129](#)) reviewed isolated anterior radial head dislocations in which there was no overt evidence of a fracture of the ulna. In each, there was subtle anterior bowing of the shaft of the ulna. They described this as the ulnar bow sign. Normally the dorsal margin of the ulna is a straight line ( [Fig. 16-26](#)). Based on their findings, they suggested that the term *isolated radial head dislocation* was a misnomer and that these were actually variants of type I Monteggia injuries. Others have reported radial head dislocation in association with nondisplaced (greenstick or plastic deformation) fracture of the olecranon ( [122,127,130](#)).



**FIGURE 16-26.** Ulnar bow sign. **A:** The normal posterior border of the ulna is represented by a straight line. **B:** In minimal type I Monteggia lesions, there is loss of this ulnar straight line with anterior bowing of the posterior border of the ulna and complete dislocation or partial subluxation of the radial head anteriorly. (Reprinted from Lincoln TL, Mubarak SH. "Isolated" traumatic radial-head dislocation. *J Pediatr Orthop* 1944;14:455; with permission.)

Because most of these injuries are occult Monteggia lesions, there is often subtle bowing of the ulna. In type I Monteggia lesions, the ulna demonstrates the typical ulnar bow sign in which the shaft of the ulna is bowed anteriorly and the radial head is dislocated anteriorly ( [Fig. 16-26](#)). In type III Monteggia lesions, the proximal ulnar metaphysis or olecranon is bowed radially (laterally) and the radial head is dislocated anterolaterally. Late repair produces better results in anterior dislocations than in anterolateral dislocations ( [137](#)).

### Rare Causes

#### Cubitus Varus Deformity

Cubitus varus after a supracondylar fracture of the distal humerus has been shown by Abe et al. ( [110](#)) to predispose to radial head dislocation. They described four dislocations that were chronic, recurrent, and precipitated by further injury. The radial head dislocated when the forearm was supinated and reduced when it was pronated. In addition to ligamentous repair, a supracondylar osteotomy was performed to correct the distal humerus deformity.

## Osteochondritis Dissecans

Klekamp et al. (128) reported seven older children and adolescents with osteochondritis dissecans of the capitellum who developed instability of the radial head: five subluxations and two posterolateral dislocations. Treatment was based on the presence of loose bodies and the characteristics of the osteochondral defect.

## Birth Trauma

This is an extremely rare mechanism of injury. Danielsson and Theander (121) reported an anterior dislocation, Schubert (140) an isolated anterolateral radial head dislocation, and Bayne and Rang (113) an anteromedial dislocation. Interestingly, all three infants were breech deliveries.

## Diagnosis

### Mechanism of Injury

The mechanism creating an isolated dislocation of the radial head appears to be the same as in Monteggia lesions (123,126,127,129,137). The only difference is that the olecranon and proximal ulna are not visibly fractured. The mechanism of injury of the various Monteggia lesions are discussed in Chapter 12.

In the 12-year-old reported by Stanley (143), he found an extreme degree of hyperextension of the elbow due to ligamentous laxity. He believed this laxity contributed significantly to the etiology. This finding has not been mentioned by other investigators, but most of these dislocations occur at around 7 years of age, when ligamentous laxity is at its peak (Fig. 16-23).

Wiley et al. (148) found in cadaver studies that anterior dislocations of the radial head could be produced only with great force. Dislocation required complete division of the anterior capsule and annular ligament, pronation, and application of an anterior force to the posterior aspect of the radial head. Tearing of the interosseous membrane sometimes occurred before the dislocation could be achieved.

### Relationship to Congenital Dislocation of the Radial Head

It is important to be able to differentiate a congenital dislocation of the radial head that requires no treatment from an acute or chronic traumatically dislocated radial head that may require treatment. Congenital dislocations are commonly bilateral, posterior, and associated with other anomalies such as Ehlers-Danlos syndrome, but some are idiopathic (112,131,132,134,139). Because of the lack of significant disability, the parents often are unaware that their child has a congenital radial head dislocation. If the child sustains an injury to the elbow with subsequent pain and swelling, accurate radiographic interpretation may be difficult.

Isolated traumatic dislocations of the radial head in newborns have been documented (113,121,140). This gives credence to the theory that many congenital dislocations may actually have a traumatic etiology that was unrecognized.

Because most congenital dislocations are bilateral, it was initially thought that unilateral dislocations were probably traumatic in origin. However, Agnew and Davis (111) reported six patients with isolated unilateral dislocations in whom there had been no history of trauma and the presence of the dislocation had been recognized since early childhood. Thus, there is doubt whether all unilateral radial head dislocations are traumatic. Southmayd and Ehrlich (142) reported three children with symptomatic subluxations of the radial head, two of whom had unilateral involvement. It was uncertain whether there was a relationship with congenital dislocation of the radial head. Similarly, Bell et al. (114) reported congenital subluxations and dislocations of 34 elbows in 27 patients without other musculoskeletal anomalies; only 7 were bilateral.

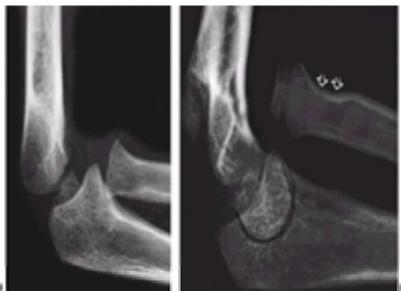
Mardam-Bey and Ger (131) argued that if there were other congenital musculoskeletal abnormalities, if there was a positive family history of dislocated radial heads, or if the patient had bilateral dislocations, these were probably congenital in origin. The absence of early trauma alone is not a reliable criterion, because trauma may occur as an occult incident at an early age.

## Signs and Symptoms

In an acute injury the elbow is swollen, motion is limited, and the radial head may or may not be palpable. In a chronic dislocation, the radial head usually is palpable and there is a lack of full flexion and extension. Pronation usually is full while supination is limited. In chronic dislocations there is typically some loss of pronation and flexion. The carrying angle may be increased and there may be valgus instability.

## Radiographic and Other Imaging Studies

Routine radiographs demonstrate an isolated radial head dislocation, but distinguishing a traumatic from a congenital dislocation can be difficult. In a review of 50 patients with congenital dislocations of the radial head, Mardam-Bey and Ger (131) found the following radiographic characteristics in all their patients: relatively short ulna or long radius, hypoplastic or absent capitellum, partially defective trochlea, prominent ulnar epicondyle, dome-shaped radial head with long neck, and grooving of the distal humerus. Similar findings have been reported by McFarland (132) and Miura (134). Unfortunately, these same changes can be found in children with long-standing traumatic dislocations (112,118,120) (Fig. 16-27). When the radiographic findings described by Mardam-Bey and Ger (131) are seen, all that can be said with certainty is that the dislocation is long-standing. The congenital nature is determined by the presence of other factors.



**FIGURE 16-27.** Progression of changes. **A:** Initial injury radiograph in a 5-year-old who sustained an acute minimally displaced anterior dislocation of the radial head. This dislocation was not appreciated initially. **B:** Three years later, the displacement had progressed and the radius had grown proximally. The radial neck also has become narrowed and elongated (arrows). These findings are similar to those described for a congenital dislocation of the radial head. (Courtesy of Charles T. Price, M.D.)

## Arthrography

Arthrograms were advocated by Mizuno et al. (135) in the differentiation of congenital and traumatic dislocations. Their differentiation was based on whether the radial head was intraarticular or extraarticular. If the radial head remained within the intact but distended capsule, the dislocation was considered congenital. In 15 traumatic dislocations, the capsule was torn and the radial head was extraarticular.

## Treatment Options

### **Nonoperative: Closed Reduction**

Most acute radial head dislocations can be satisfactorily treated by closed reduction and immobilization ( [126,144,145](#) and [146,148,149](#)), but nonoperative treatment is not appropriate for chronic radial head dislocations. Previously it was believed that dislocations older than 1 month should be left alone and possibly treated by radial head excision if the elbow became symptomatic ([144](#)). More recently, it has been recognized that chronic dislocations can be significantly improved by surgical reconstruction.

### **Surgical Procedures**

#### **Open Reduction**

Neviaser and LeFevre ([136](#)) described an acute dislocation that required open reduction because the radial head was buttonholed through the capsule. Danielsson and Theander ([121](#)) reported an infant with an irreducible anterior dislocation of the radial head in which complete displacement of the annular ligament required division and then repair. If treatment is delayed for more than a week, open reduction may be necessary if a closed reduction is unsuccessful. Open reduction is universally necessary 3 weeks following injury. As long as 3 years after injury, open reduction with annular ligament reconstruction can produce good results ([118,130,141](#)). Techniques used for reconstruction are modifications of the procedure described by Bell Tawse ([115](#)). A more anatomic and physiologic reconstruction technique of the annular ligament using two holes drilled in the proximal ulna has been described by Seel and Peterson ([141](#)).

#### **Ulnar Osteotomy**

In addition to open reduction and reconstruction of the annular ligament, osteotomy of the proximal ulna sometimes is necessary to achieve reduction of the radial head ([117,118,133,137](#)). Bouyala et al. ([117](#)) reported the use of this technique in 15 patients, including 9 with isolated dislocations of the radial head. There were no redislocations, and all had improved elbow motion. Their technique is essentially the same as that used for chronic Monteggia lesions ( [115,116,119,137,138](#) ). McGuire and Myers ([133](#)) reported seven patients treated with open reduction and ulnar osteotomy without annular ligament reconstruction. The ulnar plate was contoured until the radial head rotated against the capitellum during a full range of pronation and supination. All patients regained full elbow motion. McGuire and Myers believed that redirecting the radial head to the capitellum resulted in stability. This is essentially what occurs when a Monteggia fracture is reduced closed. The annular ligament is torn, but does not require reconstruction for stability.

Complications can occur with the reconstruction technique of open reduction, annular ligament reconstruction, and ulnar osteotomy. Rodgers et al. ( [138](#) ) reported 14 complications in 7 patients undergoing these procedures. Ultimately, there were 3 good, 2 fair, and 2 poor results.

#### **Radial Osteotomy**

If the radial head was anterior and could be reduced closed by manually supinating the forearm, Futami et al. ( [124](#) ) achieved stability with a rotation osteotomy of the radius at mid-shaft. With the radial head reduced in maximum supination, the distal shaft segment was then rotated and fixed in pronation. This was thought to decrease the tension on the biceps, theorized to be one of the major deforming forces.

#### **Radial Head Excision**

Excision of the radial head in long-standing dislocations relieves discomfort but usually produces minimal improvement in motion ( [149](#) ). However, Hresko et al. ([125](#)) demonstrated significant improvement in motion in 27 elbows in 25 patients under 18 years of age who had radial head excision for a variety of conditions that caused stiff, painful radiocapitellar joints.

#### **Postreduction Care**

After successful closed reduction, the arm is immobilized in a long arm cast with the elbow flexed 90 degrees and the forearm in full supination for 3 to 6 weeks ([126,144, 145](#) and [146](#)). Supination decreases the pull of the biceps muscle and tenses the interosseous membrane. Weisman et al. ( [147](#) ) reported two children with late dislocations in whom there appeared to have been an elbow dislocations with spontaneous resolution. Dislocation occurred with the arm immobilized in 90 degrees of elbow flexion but without supination.

A similar immobilization position is used after open reduction, with or without an osteotomy. This is continued for approximately 6 weeks or until the osteotomy is healed.

### **AUTHOR'S PREFERRED METHOD OF TREATMENT**

Most acute radial head dislocations can be treated by closed manipulation. The key to maintaining the reduction is to immobilize the elbow in hyperflexion (<sup>3</sup>120 degrees) ([Fig. 16-28](#)). The forearm is usually held in neutral rotation. The radius should always point toward the capitellum. Forearm supination can be increased to decrease the deforming force of the biceps brachii. To support the hyperextended elbow, a figure-of-eight cast, similar to the one used for supracondylar fractures, is used.



**FIGURE 16-28.** Closed reduction. **A:** Acute injury radiograph shows that the long axis of the radius ( *dotted line* ) passes proximal to the center of the capitellum, indicating a dislocation. **B:** After reduction, this relation has been reestablished and is maintained with the elbow immobilized in hyperflexion.

For long-standing post traumatic dislocations, I prefer open reduction and reconstruction of the annular ligament using a lateral strip of triceps tendon as a modification of the Bell Tawse ([115](#)) procedure popularized by Lloyd-Roberts and Bucknill ( [130](#) ) and others ([Fig. 16-29](#)) ([116,118,119,137,141](#)). I have no experience with the technique described by Seel and Peterson ([141](#)), although it appears to be an excellent alternative. The radiocapitellar joint can be stabilized with a small Steinmann pin rather than a Kirschner wire to decrease the risk of pin breakage.



**FIGURE 16-29.** Open reduction. **A:** Radiograph of a 5-year-old who had sustained an acute elbow injury 6 months previously. The radial head dislocation was not appreciated on the initial radiographs. **B:** After a Bell Tawse procedure, a Steinmann pin is used for fixation. The strap from the triceps fascia is passed through the olecranon (arrow). **C:** Six months after surgery, the reduction has been maintained.

## Complications

### **Annular Ligament Calcification**

Dystrophic calcification of the capsule and annular ligament after reduction of radial head dislocations is common ( [122,130,136,144](#)). It appears as a semilunar calcification surrounding the radial head and may become evident as early as 2 weeks after injury. In most patients, this calcification spontaneously resolves. This localized, well-defined calcification is not to be confused with the diffuse pattern of myositis ossificans after elbow injuries.

### **Loss of Motion**

Lloyd-Roberts and Bucknill suggested that untreated traumatic lesions result in a significant loss of elbow motion that can considerably decrease the power and dexterity of the extremity ( [130](#)). Their patient had no distal radial ulnar subluxation and could do heavy labor without difficulty. He was also essentially pain free. Most patients with untreated radial head dislocations develop secondary cubitus valgus deformity and valgus instability when performing upper extremity weight-bearing activities (i.e., as in gymnastics).

## ISOLATED DISLOCATIONS OF THE ULNA

Isolated dislocations of the ulna have been described in adults (see [Chapter 22](#) in *Fractures in Adults*). However, there have been no known reports of this injury occurring in children.

## SUBLUXATION OF THE RADIAL HEAD (PULLED ELBOW SYNDROME)

Subluxation of the head of the radius or pulled elbow is a common elbow injury in young children ( [150,152, 153](#) and [154, 156, 157](#) and [158,162,167,168, 172,173](#)). The term *nursemaid's elbow* and other eponyms and synonyms have been used to describe this condition ( [161,171](#)). The demographics associated with the subluxation of the radial head have been well described ( [150, 152, 153](#) and [154, 156, 157](#) and [158,162,167, 168, 172, 173](#)). The mean age at injury is usually 2 to 3 years, with the youngest reported patient 2 months of age. It rarely occurs after 7 years of age. Sixty percent to 65% of the children affected are girls, and the left elbow is involved in approximately 70%. It is difficult to determine the actual incidence because many subluxations are treated in primary care physician's offices or resolve spontaneously before being seen by a physician.

## Diagnosis

### **Mechanism of Injury**

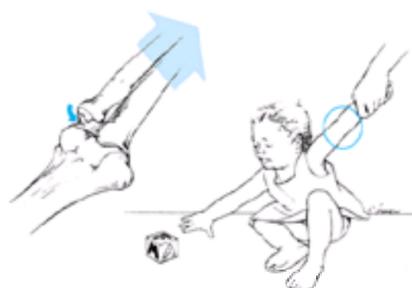
Longitudinal traction on the extended elbow is the usual mechanism of injury. Cadaver studies have shown that longitudinal traction on the extended elbow can produce a partial slippage of the annular ligament over the head of the radius and into the radiocapitellar joint, sometimes tearing the subannular membrane. Displacement of the annular ligament occurs most easily with the forearm in pronation. In this position the lateral edge of the radial head, which opposes the main portion of the annular ligament, is narrow and round at its margin ( [162,163](#)). In supination the lateral edge of the radial head is wider and more square at its margin, thereby restricting slippage. McRae ( [164](#)) demonstrated that forearm pronation maintains the displacement of the annular ligament.

Although the annular ligament slips proximally, it only partially covers the radial head. This anatomic finding has been confirmed in numerous cadaver experiments ( [163,164,171](#)). Salter and Zaltz ( [171](#)) found that if the annular ligament slipped over the equator of the radial head, the maximum anteroposterior diameter, the ligament could not be reduced to its original position. The two reports of surgical exploration of this injury in the acute stage confirmed this observation ( [171,176](#)). After 5 years of age, the distal attachment of the annular ligament to the neck of the radius has strengthened sufficiently to prevent its tearing and subsequent displacement ( [171](#)).

It was initially believed that the radial head diameter was less in children than in adults and this contributed to subluxation of the annular ligament. However, cadaver studies of infants, children, and adults have shown that the ratio of the head and neck diameters is essentially the same ( [169,171](#)). Griffin ( [156](#)) suggested that the lack of ossification of the proximal radial epiphysis in children less than 5 years of age made it more pliable, thereby facilitating slippage of the annular ligament.

Amire et al. ( [150](#)) performed a controlled study comparing 30 normal children with 100 who had sustained subluxation of the radial head. They found an increased incidence of hypermobility or ligamentous laxity among children with pulled elbows. Also, there was increased incidence of hypermobility in one or both parents of the involved children compared with normals. It was these investigators' contention that hypermobility could be a factor predisposing children to this condition.

Thus, the most widely accepted mechanism today is that the injury occurs when the forearm is pronated, the elbow extended, and longitudinal traction is applied to the patient's wrist or hand ( [Fig. 16-30](#)) ( [163,164,172](#)). Such an injury typically occurs when a young child is lifted or swung by the forearm or when the child suddenly steps down from a step or off a curb while one of the parents is holding the hand or wrist. The investigator also has seen a number of children who, while falling backward, grab an object for support, thereby applying longitudinal traction across an extended, pronated elbow.



**FIGURE 16-30.** The injury most commonly occurs when a longitudinal pull is applied to the upper extremity. Usually the forearm is pronated. There is a partial tear in

the orbicular ligament, allowing it to subluxate into the radiocapitellar joint.

### Unusual Mechanisms

Newman (166) reported that five of six infants under 6 months of age with a pulled elbow sustained the injury when rolling over in bed with the extended elbow trapped under the body. It was believed that this maneuver, especially if the infant was given a quick push to turn over by an older sibling or a parent, provided enough longitudinal traction to displace the annular ligament proximally.

### Signs and Symptoms

The history is critical in making the diagnosis. There usually is an episode of a sudden longitudinal pull on the elbow of the young child. The initial pain usually subsides rapidly and the child does not appear to be in distress except that he or she is reluctant to use the involved extremity. The upper extremity is typically held at the side with the forearm pronated. A limited painless arc of flexion and extension may be present. However, any attempt to supinate the forearm produces pain. Although there is no evidence of an elbow effusion, local tenderness may be present over the radial head and annular ligament. In some patients the pain may be referred proximally to the shoulder or distally to the wrist (150,157).

Unfortunately, the classic history is not always present (153,167,168,170,172). In some studies 33% to 49% of patients had no history of a sudden longitudinal pull (170,172). There are several reasons why the history may not be characteristic: the parents are reluctant to give the true mechanism for fear they may be accused of child abuse, the injury can be sustained in a fall (153,170), or the injury was not observed by a reliable adult witness. Often, the child is crying and thus impossible to examine adequately until he or she has calmed down. In these nonclassic patients, other causes, such as occult fractures or early septic arthritis, must be carefully ruled out.

### Radiographic and Other Imaging Studies

Results of anteroposterior and lateral radiograph usually are normal (152,153,156,158,163,168,171,172,174), but subtle abnormalities may be present. Normally, the line down the center of the proximal radial shaft should pass through the center of the ossification center of the capitellum (radiocapitellar line) (155,174). Frumkin (155) demonstrated that this did not occur in 3 infants with subluxation of the radial head. Careful review of the radiographs of children with true subluxation found that the line was more than 3 mm lateral to the center of the capitellum in almost 25% (174). To determine this subtle change requires a direct measurement on the radiograph. One problem is that these views often are taken with the forearm supinated and the subluxation is reduced when the technician forces the forearm into supination to position it for the radiograph. Bretland (151) found that if the best radiograph that can be obtained is an oblique with the forearm in pronation, radial head subluxation is the likely diagnosis.

Mehara and Bhan (165) reported a new radiologic sign of distal shift of the radius compared with the ulna. They found that the proximal radial length was altered in 21 of 25 patients (84%) with radial head subluxations. The normal relationships were restored after reduction.

Should radiographs be taken of every child before manipulation is attempted? If there is a classic history, the child is 5 years of age or younger, and the clinical findings strongly support the diagnosis, radiographs probably are not necessary (150,153,168,171,176). If, however, there is an atypical history or clinical examination, radiographs should be obtained before manipulation is attempted.

### Arthrography

Matles and Eliopoulos (163) reported the use of arthrograms in some patients. Interposition of the annular ligament produces a defect between the radial head and the capitellum that is visible on arthrography (Fig. 16-31).



**FIGURE 16-31.** Irreducible annular ligament. **A:** Arthrogram of the normal uninjured left side shows a well-defined margin to the capsule and annular ligament at the radial neck (arrows). **B:** On the affected side, this margin has lost its sharp definition and the lateral aspect has migrated somewhat proximally. **C:** On the lateral radiograph of the normal elbow the radial head articulates directly with the capitellum (arrows) when the elbow is fully flexed. **D:** On the injured side there is some limitation to full flexion. At surgery, the gap between the two articular surfaces (arrows) was filled with the interposed annular ligament. (Courtesy of Robert M. Campbell, Jr., M.D.)

### Ultrasonography

When the diagnosis is not evident, ultrasonography may be helpful (160,162). The diagnosis is made by demonstrating an increase in the echonegative area between the articular surfaces of the capitellum and the radial head (radiocapitellar distance). Kosuwon et al. (160) found that this distance is normally about 3.8 mm with forearm pronated. With a subluxated radial head, this measured 7.2 mm. A difference of 3 mm between the normal and affected sides, therefore, suggests radial head subluxation.

### Treatment Options

#### Nonoperative: Closed Reduction

Almost all subluxations are correctable by closed reduction. This is usually best done by forearm supination (153,156,157,161,162,167,168,172,173). Some have recommended that supination be performed with the elbow flexed, and others have found that supination alone with the elbow extended can effect a reduction. In all patients, a snapping sensation can be both heard and palpated when the annular ligament reduces. Macias et al. (161) reported that hyperpronation was more successful than supination in a randomized study. Reduction was successful in 40 of 41 patients (98%) in the hyperpronation group, compared with 38 of 44 patients (86%) in the supination group. They concluded that the hyperpronation technique was more successful, required fewer attempts, and was often successful when supination failed.

#### Surgical Procedures: Open Reduction

This is rarely indicated. Even if untreated, most radial head subluxations reduce spontaneously. The only indication for surgery is a chronic symptomatic irreducible

subluxation ([171,176](#)). In these, the annular ligament must be partially transected to achieve reduction ([Fig. 16-31](#)).

### Postreduction Care

After a successful closed reduction of a first time radial head subluxation, immobilization of the extremity is not necessary if the child is comfortable and using the arm normally. Salter and Zaltz ([171](#)) recommended the use of a sling, mainly to prevent the elbow from being pulled a second time. Kohlhaas and Roeder ([159](#)) recommended a T-shirt technique for flexed elbow stabilization in very young children. This provided adequate immobilization without the use of a sling by pinning the sleeve of the long sleeve T-shirt to the opposite chest.

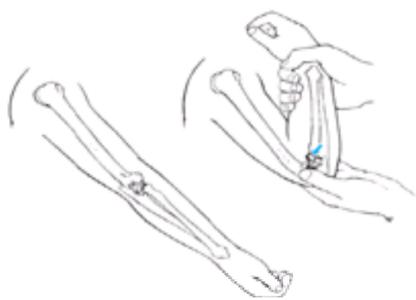
### Parent Education

After the reduction, it is important to explain to the parents the mechanism of injury and to emphasize the need to prevent longitudinal pulling on the upper extremities.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

First, it is important to try to elicit the classic history of the child having had a longitudinal force applied across the extended elbow. The entire extremity is then carefully examined. The characteristic focal tenderness should be pinpointed directly over the radial head. If there is clinical evidence of an elbow effusion, then radiographs of the upper extremity are obtained to assess for other injuries before manipulating the elbow.

Once the diagnosis of subluxation of the radial head is clearly established clinically, manipulation is performed. It is first explained to the parents there will be a brief episode of pain followed by complete or significant relief of the symptoms. The patient usually is seated on the parent's lap. The patient's forearm is grasped with the elbow semiflexed while the thumb of the surgeon's opposite hand is placed over the lateral aspect of the elbow. The forearm is first supinated. If this fails to produce the characteristic snap of reduction, then the elbow is flexed maximally until the snap occurs ([Fig. 16-32](#)). Just before reaching maximal flexion, there often is an increase in the resistance to flexion. At this point, a little extra pressure toward flexion must be applied, which usually produces the characteristic snap as the annular ligament suddenly returns to its normal position. If this fails, the hyperpronation technique of Macias et al. ([161](#)) is used.



**FIGURE 16-32.** Reduction technique, “nurse maid’s” elbow. **Left:** The forearm is first supinated. **Right:** The elbow is then hyperflexed. The surgeon’s thumb is placed laterally over the radial head to feel the characteristic snapping as the ligament is reduced.

What should be done if a definite snap or pop is not felt or if the patient fails to use the extremity after manipulation? If the subluxation has occurred more than 12 to 24 hours before the child is seen, there often is a mild secondary synovitis, and recovery may not be as dramatic. There also may be a small amount of blood in the elbow joint. The important fact to determine is whether the initial diagnosis was correct. This requires radiographs, if they were not taken before the manipulation, and a careful reexamination of the entire extremity. If the radiograph results are normal and the elbow can be fully flexed with free supination and pronation, the physician can be assured that the subluxated annular ligament has been reduced. In this circumstance, the patient's arm is placed in a splint or sling for a few days to one week and reexamined clinically first and radiographically if needed.

### Complications

#### Unreduced Subluxations

There are no reports of long-term sequelae from unrecognized and unreduced subluxations. Almost all subluxations reduce spontaneously. The only problem seems to be the discomfort to the patient until the annular ligament reduces.

#### Recurrent Subluxations

The reported incidence of recurrent subluxation has varied from 5% to 39% ([153,156,157,167,168,172,173,175](#)). Children 2 years of age or younger appear to be at greatest risk for recurrence ([172,176](#)). Recurrent subluxations usually respond to the same manipulative procedure as the initial injury. They eventually cease after 4 to 5 years when the annular ligament strengthens. These recurrences do not lead to any long-term sequelae.

Recurrent subluxations are relatively common. After 2 or 3 episodes, the elbow is immobilized in a long arm cast for 1 to 2 weeks. This treatment seems to be effective because it allows healing of the soft tissues and decreases recurrent episodes.

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## CHAPTER REFERENCES

### Elbow Dislocations

1. Allende G, Freytes M. Old Dislocation of the Elbow. *J Bone Joint Surg* 1944;26:692–706.
2. al-Qattan MM, Zuker RM, Weinberg MJ. Type 4 median nerve entrapment after elbow dislocation. *J Hand Surg [Br]* 1994;19:613–615.
3. Andersen K, Mortensen AC, Gron P. Transverse divergent dislocation of the elbow. A report of two cases. *Acta Orthop Scand* 1985;56:442–443.
4. Ayala H, De Pablos J, Gonzalez J, Martinez A. Entrapment of the median nerve after posterior dislocation of the elbow. *Microsurgery* 1983;4:251–220.
5. Beaty JH, Donati NL. Recurrent dislocation of the elbow in a child. A case report and review of the literature. *J Pediatr Orthop* 1991;11:392–396.
6. Bhan S, Mehara AK. A method of closed reduction of posterior dislocation of the elbow. *Int Orthop* 1994;18:271–272.
7. Boe S, Holst-Nielsen F. Intra-articular entrapment of the median nerve after dislocation of the elbow. *J Hand Surg [Br]* 1987;12:356–358.
8. Capo SR, Tito AV, Cuesta FJG, et al. Parálisis del nervio mediano consecutivas a desfracturas y luxaciones del condilo. A propos d'une serie de douze cas. *Ann Chir* 1984;38:270–273.
9. Carey RPL. Simultaneous dislocation of the elbow and the proximal radioulnar joint. *J Bone Joint Surg [Br]* 1984;66:254–256.
10. Carl A, Prada S, Teixeira K. Case report and review of the literature. Proximal radioulnar transposition in an elbow dislocation. *J Orthop Trauma* 1992;6:106–109.
11. Carliz H, Abols Y. Posterior dislocation of the elbow in children. *J Pediatr Orthop* 1984;4:8–12.
12. Cohn I. Forward dislocation of both bones of the forearm at the elbow. Review of recorded cases and the literature with report of a case. *Surg Gynecol Obstet* 1922;35:776–788.
13. Cotton FJ. Elbow dislocation and ulnar nerve injury. *J Bone Joint Surg* 1929;11:348–352.
14. Cromack PI. The mechanism and nature of the injury in dislocations of the elbow and a method of treatment. *Aust NZ J Surg* 1960;30:212–216.
15. Crosby EH. Dislocation of the elbow reduced by means of traction in four directions. *J Bone Joint Surg* 1936;18:1077.
16. Danielsson LG. Median nerve entrapment and elbow dislocation. A case report. *Acta Orthop Scand* 1986;57:450–452.

17. DeLee JC. Transverse divergent dislocation of the elbow in a child. Case report. *J Bone Joint Surg [Am]* 1981;63:322–323.
18. Durig M, Gauer EF, Muller W. Die Operative Behandlung der Rezidivierenden und Traumatischen Luxation des Ellenbogengelenkes nach Osborne und Cotterill. *Arch Orthop Unfall Chir* 1976;86:141–156.
19. Eklof O, Nybonde T, Karlsson G. Luxation of the elbow complicated by proximal radio-ulnar translocation. *Acta Radio* 1990;31:145–146.
20. El Bardouni A, Mahfoud M, Ouadghiri M, et al. Divergent dislocation of the elbow. A case report. *Rev Chir Orthop* 1994;80:150–152.
21. Fourrier P, Levai JP, Collin JPh. Incarceration du nerf median au cours d'une luxation du coude. *Rev Chir Orthop* 1977;63:13–16.
22. Fowles JV, Kassab MT, Douik M. Untreated posterior dislocation of the elbow in children. *J Bone Joint Surg [Am]* 1984;66:921–926.
23. Fowles JV, Slimane N, Kassab MT. Elbow dislocation with avulsion of the medial humeral epicondyle. *J Bone Joint Surg [Br]* 1990;72:102–104.
24. Galbraith KA, McCullough CJ. Acute nerve injury as a complication of closed fractures or dislocations of the elbow. *Injury* 1979;11:159–164.
25. Gillingham BL, Wright JG. Convergent dislocation of the elbow. *Clin Orthop* 1997;340:198–201.
26. Gosman JA. Recurrent dislocation of the ulna at the elbow. *J Bone Joint Surg* 1943;25:448–449.
27. Green NE. Case report. Entrapment of the median nerve following elbow dislocation. *J Pediatr Orthop* 1983;3:384–386.
28. Grimer RJ, Brooks S. Brachial artery damage accompanying closed posterior dislocation of the elbow. *J Bone Joint Surg [Br]* 1985;67:378–381.
29. Hallett H. Entrapment of the median nerve after dislocation of the elbow. A case report. *J Bone Joint Surg [Br]* 1981;63:408–412.
30. Hankin FM. Posterior dislocation of the elbow. A simplified method of closed reduction. *Clin Orthop* 1984;190:254–256.
31. Harvey S, Tchelebi H. Proximal radio-ulnar translocation. A case report. *J Bone Joint Surg [Am]* 1979;61:447–449.
32. Hassmann GC, Brunn F, Neer CS. Recurrent dislocation of the elbow. *J Bone Joint Surg [Am]* 1975;57:1080–1084.
33. Hemmadi SS, Trivedi JM. Divergent dislocation of the elbow in a child (a case report). *J Postgrad Med* 1991;37:221–222.
34. Henderson RS, Roberston IM. Open dislocation of the elbow with rupture of the brachial artery. *J Bone Joint Surg [Br]* 1952;34:636–637.
35. Hennig K, Franke D. Posterior displacement of brachial artery following closed elbow dislocation. *J Trauma* 1980;20:96–98.
36. Henrikson B. Supracondylar fractures of the humerus in children. *Acta Chir Scand* 1966;(suppl)369:1–72.
37. Herring JA. Instructional case. Recurrent dislocation of the elbow. *J Pediatr Orthop* 1989;9:483–484.
38. Hofmann KE III, Moneim MS, Omer GE, et al. Brachial artery disruption following closed posterior elbow dislocation in a child. A case report with review of the literature. *Clin Orthop* 1984;184:145–149.
39. Holbrook JL, Green NE. Divergent pediatric elbow dislocation. A case report. *Clin Orthop* 1988;234:72–74.
40. Inoue G, Horii E. Case report. Combined shear fractures of the trochlea and capitellum associated with the anterior fracture–dislocation of the elbow. *J Orthop Trauma* 1992;6:373–375.
41. Isbister ES. Proximal radioulnar translocation in association with posterior dislocation of the elbow. *Injury* 1991;22:479–482.
42. Jackson JA. Simple anterior dislocation of the elbow joint with rupture of the brachial artery. Case report. *Am J Surg* 1940;47:479–486.
43. Jacobs RL. Recurrent dislocation of the elbow joint. A case report and review of the literature. *Clin Orthop* 1971;74:151–154.
44. Johnsson O. Capsular and ligament injuries of the elbow joint. A clinical and arthrographic study. *Acta Chir Scand* 1962;(suppl)287:50–65.
45. Josefsson PO, Johnell O, Gentz CF. Long-term sequelae of simple dislocation of the elbow. *J Bone Joint Surg [Am]* 1984;66:927–930.
46. Josefsson PO, Nilsson BE. Incidence of elbow dislocation. *Acta Orthop Scand* 1986;57:537–538.
47. Josefsson P, Gentz C-F, Johnell O, et al. Surgical versus nonsurgical treatment of ligamentous injuries following dislocations of the elbow joint. *Clin Orthop* 1987;214:165–169.
48. Josefsson PO, Gentz C-F, Johnell O, et al. Surgical versus nonsurgical treatment of ligamentous injuries following dislocations of the elbow joint. A prospective randomized study. *J Bone Joint Surg [Am]* 1987;69:605–608.
49. Kapel O. Operation for habitual dislocation of the elbow. *J Bone Joint Surg [Am]* 1951;33:707–710.
50. Kilburn P, Sweeney JG, Silk FF. Three cases of compound posterior dislocation of the elbow with rupture of the brachial artery. *J Bone Joint Surg [Br]* 1962;44:119–121.
51. King T. Recurrent dislocation of the elbow. *J Bone Joint Surg [Br]* 1953;35:50–54.
52. Krishnamoorthy S, Bose K, Wong KP. Treatment of old unreduced dislocation of the elbow. *Injury* 1976;8:39–42.
53. Kumar A, Ahmed M. Technical tricks. Closed reduction of posterior dislocation of the elbow: a simple technique. *J Orthop Trauma* 1999;13:58–59.
54. Lavine LS. A simple method of reducing dislocations of the elbow joint. *J Bone Joint Surg [Am]* 1953;35:785–786.
55. Linscheid RL, Wheeler DK. Elbow dislocations. *JAMA* 1965;194:113–118.
56. Loomis LK. Reeducation and after-treatment of posterior dislocation of the elbow. With special attention to the brachialis muscle and myositis ossificans. *Am J Surg* 1944;63:56–60.
57. Louis DS, Ricciardi JE, Spengler DM. Arterial injury: a complication of posterior elbow dislocation. A clinical and anatomical study. *J Bone Joint Surg [Am]* 1974;56:1631–1636.
58. MacSween WA. Transposition of radius and ulna associated with dislocation of the elbow in a child. *Injury* 1978;10:314–316.
59. Mahaisavariya B, Laupattarakasem W, Supachutikul A, et al. Late reduction of dislocated elbow. Need triceps be lengthened? *J Bone Joint Surg [Br]* 1992;75:426–428.
60. Malkawi H. Recurrent dislocation of the elbow accompanied by ulnar neuropathy. A case report and review of the literature. *Clin Orthop* 1981;161:270–274.
61. Manouel M, Minkowitz B, Shimotsu G, et al. Brachial artery laceration with closed posterior elbow dislocation in an eight year old. *Clin Orthop* 1993;296:109–112.
62. Mantle JA. Recurrent posterior dislocation of the elbow. *J Bone Joint Surg [Br]* 1966;48:590.
63. Matev I. A radiological sign of entrapment of the median nerve in the elbow joint after posterior dislocation. A report of two cases. *J Bone Joint Surg [Br]* 1976;58:353–355.
64. McAuliffe TB, Williams D. Transverse divergent dislocation of the elbow. *Injury* 1988;19:279–280.
65. McKeller Hall R. Recurrent posterior dislocation of the elbow joint in a boy. Report of a case. *J Bone Joint Surg [Br]* 1953;35:56.
66. Meyn MA, Quigley TB. Reduction of posterior dislocation of the elbow by traction on the dangling arm. *Clin Orthop* 1974;103:106–108.
67. Milch H. Bilateral recurrent dislocation of the ulna at the elbow. *J Bone Joint Surg* 1936;18:777–780.
68. Minford EJ, Beattie TF. Hanging arm method for reduction of dislocated elbow. *J Emerg Med* 1993;11:161–162.
69. Naidoo KS. Unreduced posterior dislocations of the elbow. *J Bone Joint Surg [Br]* 1982;64:603–606.
70. Nakano A, Tanaka S, Hirofujii E, et al. Transverse divergent dislocation of the elbow in a six-year-old boy: case report. *J Trauma* 1992;32:118–119.
71. Neviasser JS, Wickstrom JK. Dislocation of the elbow: a retrospective study of 115 patients. *South Med J* 1977;70:172–173.
72. O'Driscoll SW, Bell DF, Morrey BF. Posterolateral rotatory instability of the elbow. *J Bone Joint Surg [Am]* 1991;73:440–446.
73. O'Driscoll SW, Morrey BF, Korinek S, et al. Elbow subluxation and dislocation. A spectrum of instability. *Clin Orthop* 1992;280:186–197.
74. Osborne G, Cotterill P. Recurrent dislocation of the elbow. *J Bone Joint Surg [Br]* 1966;48:340–346.
75. Parvin RW. Closed reduction of common shoulder and elbow dislocations without anesthesia. *Arch Surg* 1957;75:972–975.
76. Pearce MS. Radial artery entrapment. *Int Orthop* 1993;17:127–182.
77. Pritchard DJ, Linscheid RL, Svien HJ. Intra-articular median nerve entrapment with dislocation of the elbow. *Clin Orthop* 1973;90:100–103.
78. Pritchett JW. Case report. Entrapment of the median nerve after dislocation of the elbow. *J Pediatr Orthop* 1984;4:752–753.
79. Rana NA, Kenwright J, Taylor RG, et al. Complete lesion of the median nerve associated with dislocation of the elbow. *Acta Orthop Scand* 1974;45:365–369.
80. Rao SB, Crawford AH. Median nerve entrapment after dislocation of the elbow in children. A report of 2 cases and review of the literature. *Clin Orthop* 1995;312:232–237.
81. Reichenheim PP. Transplantation of the biceps tendon as a treatment for recurrent dislocation of the elbow. *Br J Surg* 1947;35:201–204.
82. Roaf R. Foramen in the humerus caused by the median nerve. *J Bone Joint Surg [Br]* 1957;39:748–749.
83. Roberts PH. Dislocation of the elbow. *Br J Surg* 1969;56:806–815.
84. Ross G, McDevitt ER, Chronister R, et al. Treatment of simple elbow dislocation using an immediate motion protocol. *Am J Sports Med* 1999;27:308–311.
85. Royle SG. Posterior dislocation of the elbow. *Clin Orthop* 1991;269:201–204.
86. Rubens MK, Auliciano PL. Open elbow dislocation with brachial artery disruption: case report and review of the literature. *Orthopaedics* 1986;9:539–542.
87. Schwab GH, Bennett JB, Woods GW, et al. A biomechanics of elbow instability: the role of the medial collateral ligament. *Clin Orthop* 1980;146:42–52.
88. Shankarappa YK, Tello E, Ferris BD. Transverse divergent dislocation of the elbow with ipsilateral distal radius epiphyseal injury in a seven year old. *Injury* 1998;29:798–802.
89. Silva JF. Old dislocations of the elbow. *Ann R Coll Surg Eng* 1958;22:363–381.
90. Sojbjerg JO, Helmgig P, Kjaersgaard-Andersen P. Dislocation of the elbow: an experimental study of the ligamentous injuries. *Orthopedics* 1987;12:461–463.
91. Sovio OM, Tredwell SJ. Case report. Divergent dislocation of the elbow in a child. *J Pediatr Orthop* 1986;6:96–97.
92. Spear HC, Jones JM. Rupture of the brachial artery accompanying dislocation of the elbow or supracondylar fracture. *J Bone Joint Surg [Am]* 1951;33:889–894.
93. Speed JS. An operation for unreduced posterior dislocation of the elbow. *South Med J* 1925;18:193–197.
94. Steiger RN, Larrick RB, Meyer TL. Median-nerve entrapment following elbow dislocation in children. A report of two cases. *J Bone Joint Surg [Am]* 1969;51:381–385.
95. Symeonides PO, Paschaloglou C, Stavrou Z, et al. Recurrent dislocation of the elbow. Report of three cases. *J Bone Joint Surg [Am]* 1975;57:1084–1086.
96. Thomas TT. A contribution of the mechanism of fractures and dislocations in the elbow region. *Ann Surg* 1929;89:108–121.
97. Thompson HC, Garcia A. Myositis ossificans after elbow injuries. *Clin Orthop* 1967;50:129–134.
98. Trias A, Comeau Y. Recurrent dislocation of the elbow in children. *Clin Orthop* 1974;100:74–77.
99. Vicente P, Orduna M. Transverse divergent dislocation of the elbow in a child. A case report. *Clin Orthop* 1993;294:312–313.
100. Wainwright D. Recurrent dislocation of the elbow joint. *Proc R Soc Med* 1947;40:885–886.
101. Watson-Jones R. Primary nerve lesions in injuries of the elbow and wrist. *J Bone Joint Surg* 1930;12:121–140.
102. Wheeler DK, Linscheid RL. Fracture–dislocations of the elbow. *Clin Orthop* 1967;50:95–106.
103. Wilkins KE, Beaty JH, Chambers HG, et al. Fractures and dislocations of the elbow region. In: Rockwood CA, Green DP, Bucholz RW, et al., eds. *Rockwood and Green's fractures in adults*, 4th ed. Philadelphia, Lippincott-Raven, 1996:653–904.
104. Wilkerson RD. Anterior elbow dislocation associated with olecranon fractures. Review of the literature and case report. *Iowa Orthop J* 1993;13:223–225.
105. Wilmshurst AD, Millner PA, Batchelor AG. Brachial artery entrapment in closed elbow dislocation. *Injury* 1989;20:240–241.
106. Winslow R. A case of complete anterior dislocation of both bones of the forearm at the elbow. *Surg Gynecol Obstet* 1913;16:570–571.

107. Witvoet J, Tayon B. La luxation recidivante du coude. A propos de 6 cas. *Rev Chir Orthop* 1974;60:485–495.
108. Woods GW, Tullos HS. Elbow instability and medial epicondyle fracture. *Am J Sports Med* 1977;5:23–30.
109. Zeier FG. Recurrent traumatic elbow dislocation. *Clin Orthop* 1982;169:211–214.

#### Radial Head Dislocation

110. Abe M, Ishizu T, Nagaoka T, et al. Recurrent posterior dislocation of the head of the radius in post-traumatic cubitus varus. *J Bone Joint Surg [Br]* 1995;77:582–585.
111. Agnew DK, Davis RJ. Congenital unilateral dislocation of the radial head. *J Pediatr Orthop* 1993;13:526–528.
112. Almqvist EE, Gordon LH, Blue AI. Congenital dislocation of the head of the radius. *J Bone Joint Surg [Am]* 1969;51:1118–1127.
113. Bayne O, Rang M. Case report. Medial dislocation of the radial head following breech delivery. *J Pediatr Orthop* 1984;4:485–487.
114. Bell SN, Morrey BF, Bianco AJ. Chronic posterior subluxation and dislocation of the radial head. *J Bone Joint Surg [Am]* 1991;73:392–396.
115. Bell Tawse AJS. The treatment of malunited anterior Monteggia fractures in children. *J Bone Joint Surg [Br]* 1965;47:718–723.
116. Best TN. Management of old unreduced Monteggia fracture dislocations of the elbow in children. *J Pediatr Orthop* 1994;14:193–199.
117. Bouyala JM, Bollini G, Jacquemier M, et al. Le traitement des luxations anciennes de la tete radial e chez l'enfant par l'osteotomie haute du cubitus. *Rev Chir Orthop* 1988;74:173–182.
118. Bucknill TM. Anterior dislocation of the radial head in children. *Proc R Soc Med* 1977;70:620–624.
119. Cappellino A, Wolfe SW, Marsh JS. Use of a modified Bell Tawse procedure for chronic acquired dislocation of the radial head. *J Pediatr Orthop* 1998;18:410–414.
120. Caravias DE. Some observations on congenital dislocation of the head of the radius. *J Bone Joint Surg [Br]* 1957;39:86–90.
121. Danielsson LG, Theander G. Traumatic dislocation of the radial head at birth. *Acta Radiol Diagn [Stockh]* 1981;22:279–382.
122. Earwaker J. Posttraumatic calcification of the annular ligament of the radius. *Skel Radio*. 1992;21:149–154.
123. Evans EM. Pronation injuries of the forearm with special reference to the anterior Monteggia fracture. *J Bone Joint Surg [Br]* 31:578–588.
124. Futami T, Tsukamoto Y, Fujita T. Rotation osteotomy for dislocation of the radial head. 6 Cases followed for 7 (3–10) years. *Acta Orthop Scand* 1992;63:455–456.
125. Hresko MT, Rosenberg BN, Pappas AM. Excision of the radial head in patients younger than 18 years. *J Pediatr Orthop* 1999;19:106–113.
126. Hudson DA, De Beer JDV. Isolated traumatic dislocation of the radial head in children. *J Bone Joint Surg [Br]* 1986;68:378–381.
127. Hume AC. Anterior dislocation of the head of the radius associated with undisplaced fracture of the olecranon in children. *J Bone Joint Surg [Br]* 1957;39:508–512.
128. Klekamp J, Green NE, Mencia GA. Osteochondritis dissecans as a cause of developmental dislocation of the radial head. *Clin Orthop* 1997;338:36–41.
129. Lincoln TL, Mubarak SJ. "Isolated" traumatic radial-head dislocation. *J Pediatr Orthop* 1994;14:454–457.
130. Lloyd-Roberts GC, Bucknill TM. Anterior dislocation of the radial head in children. Aetiology, natural history and management. *J Bone Joint Surg [Br]* 1977;59:402–407.
131. Mardam-Bey T, Ger E. Congenital radial head dislocation. *J Hand Surg* 1979;4:316–320.
132. McFarland B. Congenital dislocation of the head of the radius. *Br J Surg* 1936;24:41–49.
133. McGuire TP, Myers P. Ulnar osteotomy for missed Monteggia fractures. *J Bone Joint Surg [Br]* 1986;68:336.
134. Miura T. Congenital dislocation of the radial head. *J Hand Surg* 1990;15:477–481.
135. Mizuno K, Usui Y, Kohyama K, et al. Familial congenital unilateral anterior dislocation of the radial head: differentiation from traumatic dislocation by means of arthrography. A case report. *J Bone Joint Surg [Am]* 1991;73:1086–1089.
136. Neviasser RJ, LeFevre GW. Irreducible isolated dislocation of the radial head. *Clin Orthop* 1971;80:72–74.
137. Oner FC, Diepstraten AFM. Treatment of chronic post-traumatic dislocation of the radial head in children. *J Bone Joint Surg [Br]* 1993;75:577–581.
138. Rodgers WB, Waters PM, Hall JE. Chronic Monteggia lesions in children. Complications and results of reconstruction. *J Bone Joint Surg [Am]* 1996;78:1322–1329.
139. Sachar K, Mih AD. Congenital radial head dislocations. *Hand Clin* 1998;14:39–47.
140. Schubert JJ. Dislocation of the radial head in the newborn infant. Case report and review of the literature. *J Bone Joint Surg [Am]* 1965;47:1019–1023.
141. Seel MJ, Peterson HA. Management of chronic posttraumatic radial head dislocation in children. *J Pediatr Orthop* 1999;19:306–312.
142. Southmayd W, Ehrlich MG. Idiopathic subluxation of the radial head. *Clin Orthop* 1976;121:271–274.
143. Stanley D. Isolated traumatic anterior dislocation of the radial head—a mechanism of injury in children. *Injury* 1986;17:182–183.
144. Stelling FH, Cote RH. Traumatic dislocation of head of radius in children. *JAMA* 1956;160:732–736.
145. Tait GR, Sulaiman SK. Isolated dislocation of the radial head: a report of two cases. *Injury* 1988;19:125–126.
146. Vesely DG. Isolated traumatic dislocations of the radial head in children. *Clin Orthop* 1967;50:31–36.
147. Weisman DS, Rang M, Cole WG. Tardy displacement of traumatic radial head dislocation in childhood. *J Pediatr Orthop* 1999;19:523–526.
148. Wiley JJ, Pegington J, Horwich JP. Traumatic dislocation of the radius at the elbow. *J Bone Joint Surg [Br]* 1974;56:501–507.
149. Wiley JJ, Loeher J, McIntyre W. Isolated dislocation of the radial head. *Orthop Rev* 1991;20:973–976.

#### Subluxation of the Radial Head (Pulled Elbow Syndrome)

150. Amir D, Frankl U, Pogrund H. Pulled elbow and hypermobility of joints. *Clin Orthop* 1990;257:94–99.
151. Bretland PM. Pulled elbow in childhood. *Br J Radiol* 1994;67:1176–1185.
152. Broadhurst BW, Buhr AJ. The pulled elbow. *BMJ* 1959;1:1018–1019.
153. Choung W, Heinrich SD. Acute annular ligament interposition into the radiocapitellar joint in children (nursemaid's elbow). *J Pediatr Orthop* 1995;15:454–456.
154. Costigan PG. Subluxation of the annular ligament at the proximal radio-ulnar joint. *Alberta Med Bul*. 1952;17:7–9.
155. Frumkin K. Nursemaid's elbow. A radiographic demonstration. *Ann Emerg Med* 1985;14:690–693.
156. Griffin ME. Review article. Subluxation of the head of the radius in children. *Pediatrics* 1955;15:103–106.
157. Illingsworth CM. Pulled elbow: a study of 100 patients. *BMJ* 1975;2:672–674.
158. Jongschaap HCN, Youngson GG, Beattie TF. The epidemiology of radial head subluxation ("pulled elbow") in the Aberdeen City area. *Health Bull [Edinb]* 1990;48:58–61.
159. Kohlhaas AR, Roeder J. Tee shirt management of nursemaid's elbow. *Am J Orthop* 1995;24:74.
160. Kosuwon W, Mahaisavariya B, Saengnipanthkul S, et al. Ultrasonography of pulled elbow. *J Bone Joint Surg [Br]* 1993;75:421–422.
161. Macias CG, Bothner J, Wiebe R. A comparison of supination/flexion to hyperpronation in the reduction of radial head subluxations. *Pediatrics* 1998;102:10.
162. Magill HK, Aitken AP. Pulled elbow. *Surg Gynecol Obstet* 1954;98:753–756.
163. Matles A, Eliopoulos K. Internal derangement of the elbow in children. *Int Surg* 1967;48:259–263.
164. McRae R, Freeman PA. The lesion of pulled elbow. *J Bone Joint Surg [Br]* 1965;47:808.
165. Mehara AK, Bhan S. A radiological sign in pulled elbows. *Int Orthop* 1995;19:174–175.
166. Newman J. "Nursemaid's elbow" in infants 6 months and under. *J Emerg Med* 1985;2:403–404.
167. Piroth P, Gharib M. Die Traumatische Subluxation des Radiuskopfchens (Chassaignac). *Deutsch Med Wochenschr* 1976;101:1520–1523.
168. Quan L, Marcuse EK. The epidemiology and treatment of radial head subluxation. *Am J Dis Child* 1985;139:1194–1197.
169. Ryan JR. The relationship of the radial head to radial neck diameters in fetuses and adults with reference to radial-head subluxation in children. *J Bone Joint Surg [Am]* 1969;51:781–783.
170. Sacchetti A, Ramoska EE, Glasgow C. Nonclassic history in children with radial head subluxations. *J Emerg Med* 1990;8:151–153.
171. Salter RB, Zaltz C. Anatomic investigations of the mechanism of injury and pathologic anatomy of pulled elbow in young children. *Clin Orthop* 1971;77:134–143.
172. Schunk JE. Radial head subluxation: epidemiology and treatment of 87 episodes. *Ann Emerg Med* 1990;19:1019–1023.
173. Snellman O. Subluxation of the head of the radius in children. *Acta Orthop Scand* 1959;28:311–315.
174. Snyder HS. Radiographic changes with radial head subluxation in children. *J Emerg Med* 1990;8:265–269.
175. Teach SJ, Schutzman SA. Prospective study of recurrent radial head subluxation. *Arch Pediatr Adolesc Med* 1996;150:164–166.
176. Triantafyllou SJ, Wilson SC, Rychak J. Irreducible pulled elbow in a child. A case report. *Clin Orthop* 1992;284:153–155.

**Fractures of the Proximal Humerus**[Diagnosis](#)[Classification](#)[Surgical Anatomy](#)[Treatment Options](#)[Complications](#)**Fractures of the Scapula**[Diagnosis](#)[Classification](#)[Surgical Anatomy](#)[Biomechanics](#)[Treatment Options](#)[Complications](#)**Fractures of the Clavicle**[Diagnosis](#)[Classification](#)[Surgical Anatomy](#)[Biomechanics](#)[Treatment Options](#)[Complications](#)**Fractures of the Humeral Shaft****Embryology and Development**[Applied Anatomy](#)[Mechanisms of Injury](#)[Classification](#)[Incidence](#)[Signs and Symptoms](#)[Radiographic Findings](#)[Treatment](#)[Rehabilitation](#)[Prognosis](#)[Complications](#)**Distal Humeral Diaphyseal Fractures**[Etiology](#)[Classification](#)[Treatment](#)**Supracondylar Process Fractures**[Anatomy](#)[Etiology](#)[Classification](#)[Treatment](#)**Glenohumeral Subluxation and Dislocation**[Anatomy](#)[Mechanism of Injury](#)[Classification](#)[Signs and Symptoms](#)[Radiographic Findings](#)[Treatment](#)[Rehabilitation](#)[Complications](#)[Chapter References](#)**FRACTURES OF THE PROXIMAL HUMERUS**

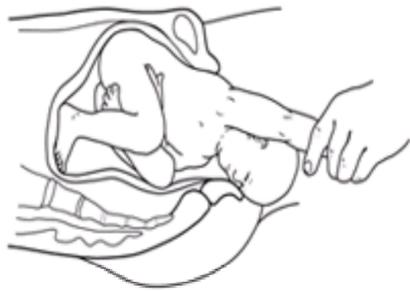
Fractures of the proximal humerus are relatively uncommon injuries of childhood, with an incidence of 1.2–4.4 per 1,000 per year ([7,57,125](#)), fewer than 5% of all pediatric fractures ([40,43,56,57,88](#)).

Fractures of the proximal humerus can be a common birth-related injury ([61,101](#)). As an infant is passing through the birth canal, the arm may be placed in a variety of awkward positions that will result in a separation through the physis of the proximal humerus ([22,34,36,37,61,72,101](#)). In older children, trauma, both direct and indirect, is the predominant cause of fractures in the proximal humerus. In this age group, these fractures can involve either the metaphysis, the physis, or both.

Because of the thick periosteum in this area and the proximity to the physis, fractures in this region have enormous potential to heal and remodel, perhaps more so than anywhere else in the body. Thus, proximal humeral fractures in children, in general, can be expected to heal without significant residual functional or cosmetic deficits.

**Diagnosis*****Mechanism of Injury*****Birth Fractures**

Fractures in the proximal humerus frequently occur during delivery ([61,101](#)). These fractures are generally believed to result from hyperextension or rotation of the arm during the passage through the birth canal ([Fig. 17-1](#)) ([22,34,36,37,61,72,101](#)). As might be expected, obstetric proximal humeral fractures occur most frequently during vaginal deliveries of infants with larger size or breech presentation ([14,29,47,101](#)). Prenatal size and presentation, however, have not been accurate predictive factors for these fractures, because proximal humeral fractures can and do occur during vaginal deliveries of infants of all sizes and weights ([14,29,47](#)).



**FIGURE 17-1.** Hyperextension or rotation of the ipsilateral arm may result in a proximal humeral or physeal injury during birth.

### Childhood/Adolescent Fractures

For older children, the mechanism of injury in most proximal humeral fractures is trauma. The trauma can be a direct blow to the shoulder area, especially to the posterior aspect (22,77,103) or indirect as in a fall onto an outstretched hand that transmits the force through the arm to the proximal humerus (2,9,47). Indirect trauma can result in forced or nonphysiologic positioning of the upper extremity, which in turn may cause a fracture of the proximal humerus. Specifically, six potential mechanisms of upper extremity positioning have been proposed to explain the resulting proximal humeral fractures: forced extension, forced flexion, forced extension with lateral or medial rotation, and forced flexion with lateral or medial rotation (123). Although trauma has been acknowledged as the most common mechanism of pediatric proximal humeral fractures, it is still controversial whether a fall or a direct blow is the more common etiology of the fracture.

Pediatric proximal humeral fractures often occur in children involved in motor vehicle accidents and sports (Fig. 17-2 and Fig. 17-3) (51). It has been estimated that approximately 50% of shoulder girdle fractures in children are related to sports and play activities (78). Athletic activities associated with proximal humeral fractures include contact sports, horseback riding (fall from horses), gymnastics (upper extremity impact and weight bearing), and baseball (repetitive throwing) (20,56,66,114,115).



**FIGURE 17-2.** Motor vehicle crashes may result in proximal humeral fracture due to blunt trauma to the shoulder region.



**FIGURE 17-3.** Blunt trauma from contact sports may result in fracture of the proximal humerus in children.

Less often, pediatric proximal humeral fractures result from other illnesses such as malignant or benign tumors and pituitary gigantism (1,54,87,94). They also can be a complication of radiation therapy to the shoulder region (24). In addition, shoulder joint neuropathy secondary to Arnold-Chiari malformation, myelomeningocele, or syringomyelia has been implicated as an etiologic factor in proximal humeral fractures (5,67). An unknown percentage of pediatric proximal humeral fractures are part of the injuries associated with child abuse (Fig. 17-4) (28). Because no clear fracture pattern in the proximal humerus is suggestive of abuse, an index of suspicion must remain high when evaluating infants or young children with humeral fractures (99).



**FIGURE 17-4.** Although the exact mechanism of injury may vary in child abuse, fracture of the proximal humerus may result from twisting at the elbow or forearm.

### Signs and Symptoms

Clinical features of proximal humeral fractures in newborns may be subtle and not readily identified. For example, the infant may be irritable when lifted by the arms or when the involved upper extremity is moved. The infant may refuse to move the arm, giving the appearance of paralysis (i.e., pseudoparalysis). In addition to proximal humeral fractures, the differential diagnosis for such paralysis includes brachial plexus injury, septic shoulder, and clavicular fractures. Infants exhibiting upper

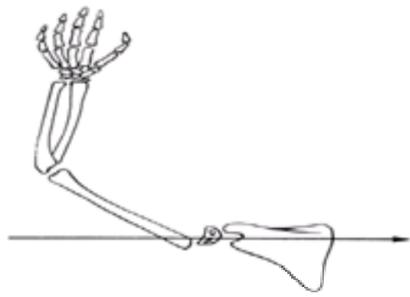
extremity paralysis also may be suffering from posterior humeral head dislocation ( [111](#)).

Older children typically report a history consistent with a proximal humeral fracture. They often present with an obvious deformity in the anterior shoulder region and refuse to use the involved arm. Pain, swelling, and ecchymosis, to some degree, are invariably present, and the overall contour of the shoulder is altered in comparison with the contralateral uninjured shoulder.

Most children with displaced proximal humeral fracture hold the involved upper extremity in internal rotation to reduce the pull of the pectoralis major muscle on the distal fragment. With posterior fracture dislocations, children demonstrate limited and extremely painful external rotation. Some children with fractures of the greater tuberosity have an unusual presentation of luxatio erecta where the involved shoulder is positioned in extreme abduction ( [27](#)). This position reduces the displacement across the fracture as the greater tuberosity is pulled superiorly by the supraspinatus muscle. The elbow is typically flexed in luxatio erecta, allowing the hand to be near or above the head ( [27,52](#)). Because fractures of the lesser tuberosity will affect the function of the inserting subscapularis muscle, internal rotation with the shoulder in adduction and abduction with the shoulder in external rotation will be limited and painful ( [49,89,119](#)).

### Radiographic Studies

The proximal humeral epiphysis is not visible on plain radiographs until about 6 months of age ( [53,81](#)), and plain radiographs are of limited value in evaluation of proximal humeral fractures in infants. On an anteroposterior (AP) radiograph, a change in the positional relationship between the proximal humeral metaphysis and the scapula and acromion often is visible. A comparison with the uninjured contralateral shoulder may reveal this alteration more clearly. A vanishing epiphysis sign also has been reported to describe posteriorly displaced transphyseal fractures of the proximal humerus ( [Fig. 17-5](#)) ( [50,95](#)). In comparison with the unaffected contralateral side, the epiphysis appears to vanish on an AP radiograph when it is displaced posteriorly. For complete evaluation of proximal humeral fractures in newborns and infants, ultrasonographic studies often are diagnostic and informative ( [11,41,116](#)). Computed tomography (CT) also may be useful, especially for complex fractures with posterior dislocations ( [112](#)).



**FIGURE 17-5.** Vanishing epiphysis sign.

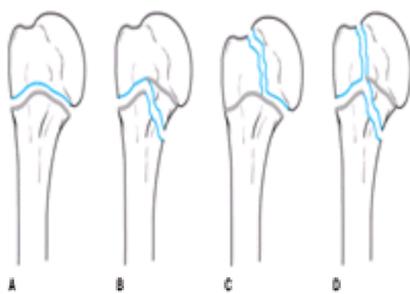
For evaluation of proximal humeral fractures in older children, two radiographs in perpendicular views can be diagnostic ( [108](#)). Ideally, a true AP view of the shoulder and an axillary lateral view provide the most information about the fracture. Because some lesser tuberosity fractures may only be visible on an axillary lateral view, this radiograph should be included whenever possible ( [42](#)). Often, however, an axillary lateral view is difficult to obtain in a child with an acutely fractured proximal humerus. In these instances, transthoracic axillary view or Y view radiographs can be obtained. An apical oblique view, an AP radiograph with the x-ray beam at 45 degrees of caudal tilt, also can provide significant information about the proximal humerus ( [102](#)). In fact, some authors report that most shoulder trauma can be evaluated with AP and apical oblique radiographs and that a lateral view (axillary lateral or Y view) can be obtained if a humeral fracture is suspected ( [10](#)).

When adequate radiographs cannot be obtained, CT is useful in evaluating proximal humeral fractures. CT may be especially useful in characterizing posterior fracture dislocations ( [33,119](#)). If the child continues to complain of shoulder pain despite negative radiographic and CT results, an occult fracture must be ruled out. For this purpose, magnetic resonance imaging (MRI) is diagnostic ( [7,90,110](#)). A bone scan also may be useful in such situations. However, due to the normally increased radionuclide uptake in the physis of the proximal humerus, a bone scan in this region may be difficult to interpret.

### Classification

Fractures of the proximal humerus in the pediatric population are broadly categorized by their anatomic location. They may involve the physis, the metaphysis, the lesser tuberosity, or the greater tuberosity. In addition, the degree of fracture displacement plays an important role in the overall treatment option. Other fracture characteristics that must be evaluated include the presence or absence of open fractures, concomitant glenohumeral dislocations, and fracture stability.

Fractures involving the physis are classified according to the Salter-Harris classification ( [92](#)) ( [Fig. 17-6](#)). Salter-Harris type I injuries with fractures through the physis occur mostly in patients under 5 years of age ( [22,83](#)). After 11 years of age, most fractures of the proximal humerus are Salter-Harris type II injuries, with the fracture line exiting through the metaphysis ( [12,22,83](#)). Some Salter-Harris type II injuries are associated with an additional anterolateral bony fragment ( [12](#)). Salter-Harris type III injuries with the fracture line exiting through the epiphysis rarely occur in the proximal humerus of children ( [22,83](#)). The few reported such injuries occurred with and without concomitant glenohumeral dislocation ( [16,33,109,120,124](#)). Salter-Harris type IV injuries involving both the metaphysis and the epiphysis of the proximal humerus have not been reported in children.



**FIGURE 17-6.** Physeal fractures of the proximal humerus.

Fractures of the metaphysis occur mostly in children 5 to 12 years of age ( [Fig. 17-7](#)) and are categorized by their anatomic location and degree of displacement ( [22](#)). The anatomic location is described in relation to the major deforming forces in the region, namely the insertions of the pectoralis major and the deltoid muscles. Presence or absence of other fractures in the ipsilateral upper extremity also must be documented, because segmental fractures may require alternative treatments ( [46,71,82](#)). Other isolated fractures of the proximal humerus may include the greater and the lesser tuberosities ( [27,49,52,89,121](#)).



**FIGURE 17-7.** Healing undisplaced fracture of the proximal humerus in a 5-year-old child.

The degree of fracture displacement in the proximal humerus is classified with respect to the shaft diameter of the humerus ( [77](#)). In grade I injuries, there is up to 5 mm of displacement. In grade II and III injuries, fractures are displaced by up to one third and two thirds of the humeral shaft diameter, respectively. Displacement of greater than two thirds of the shaft diameter, including total displacement, is classified as a grade IV injury. In addition to degree of displacement, fractures in this region may demonstrate angulation deformities. Although varus angulation is most common, fractures can be angulated in any direction.

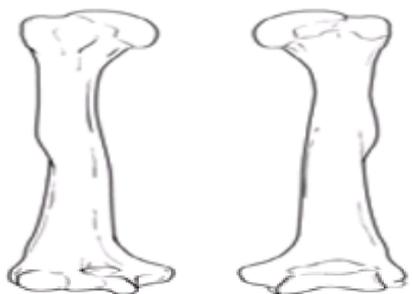
In high-energy trauma, fractures of the proximal humerus may be associated with concomitant dislocations of the glenohumeral joint. The direction of the dislocation may be anterior, posterior, or inferior ( [16,27,28,33,52,80,117](#)). A stress fracture of the metaphysis or a slipped epiphysis can be produced by chronic or repetitive trauma, such as repetitive throwing, gymnastics with humeral weight bearing, and localized radiation therapy ( [20,24,66,114,115](#)). Because of the tremendous healing and remodeling potential in the pediatric proximal humerus, these injuries can be successfully treated with conservative nonsurgical therapy.

### Surgical Anatomy

The proximal humeral ossification center cannot be seen on plain radiographs until about 6 months of age ( [53,81](#)). In addition to the proximal humerus, both the greater and lesser tuberosities contain their own separate ossification centers. The ossification center for the greater tuberosity appears at around 1 to 3 years of age, while the ossification center for the lesser tuberosity takes form at 4 to 5 years of age ( [81,93](#)). The two tuberosities coalesce at around 5 to 7 years of age and subsequently fuse with the humeral head at 7 to 13 years of age ( [81,93](#)).

The proximal physis of the humerus continues to proliferate well into the teenage years and is ultimately responsible for approximately 80% of the overall humeral growth ( [8,85,86,105](#)). Interestingly, longitudinal growth at the proximal humeral physis changes during development such that it is responsible for only 75% of humeral growth before age 2, but up to 90% of growth after age 11 ( [8,85,86](#)). For girls, this growth continues until around 14 years of age, with subsequent fusion of the epiphysis to the shaft at 14 to 17 years of age ( [8,19,105](#)). For boys, growth continues until about age 16, when closure of the physis begins ( [8,85,86](#)). For most boys, the proximal humeral physis is closed by about 18 years of age ( [19](#)).

The articular surface of the proximal humerus covers most of the medial aspect of the epiphysis as well as the proximal medial corner of the metaphysis ( [Fig. 17-8](#)). The glenohumeral joint capsule surrounds the articular surface such that most of the medial epiphysis as well as the proximal medial corner of the metaphysis are intraarticular ( [Fig. 17-9](#)). Conversely, a predominant proportion of the physis is extracapsular and remains susceptible to injury. The periosteum is quite strong in the posteromedial aspect of the proximal humerus, but the periosteum in the anterolateral aspect is relatively weak, occasionally allowing the fractured fragment to penetrate and prevent reduction ( [22](#)).

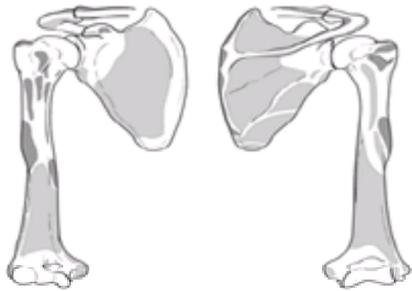


**FIGURE 17-8.** The articular surface of the proximal humerus.



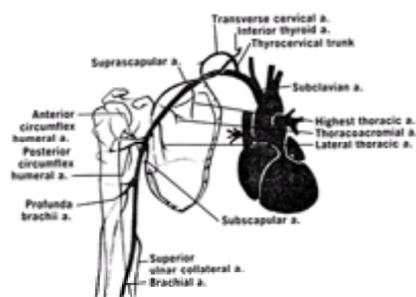
**FIGURE 17-9.** Glenohumeral joint capsule.

The proximal humerus is the site of insertion for a number of different muscles that can influence the pattern of fracture displacement. These muscles and their attachments form early during development and are grossly similar to those of an adult shoulder by the time of birth. The four muscles of the rotator cuff insert onto the epiphysis. The subscapularis muscle inserts on the anterior aspect of the epiphysis on the lesser tuberosity, whereas the teres minor, the infraspinatus, and the supraspinatus muscles insert onto the superior and posterior aspect of the epiphysis near the greater tuberosity ( [Fig. 17-10](#)). In addition to the rotator cuff muscles, other muscular attachments that can cause fracture displacement are the deltoid and pectoralis major muscles. The deltoid muscle attaches in the lateral aspect of the humeral shaft, whereas the pectoralis major muscle attaches to the anteromedial aspect of the metaphysis.



**FIGURE 17-10.** Origins and insertions of the cuff muscles in a child: subscapularis, teres minor, infraspinatus, and supraspinatus.

The vascular supply to the proximal humerus arises from the axillary artery. Distal to the pectoralis minor muscle, three different arterial branches arise from the axillary artery before it becomes the brachial artery to supply the upper extremity. One of these branches is the subscapular artery, which runs with the subscapular nerve to supply the rotator cuff muscles. The remaining two branches, the anterior and the posterior humeral circumflex arteries, supply the proximal humerus. Most of the humeral head vascularity is from the arcuate artery, which, in turn, is from the ascending branch of the anterior humeral circumflex artery (30,55). The posterior humeral circumflex artery is a less dominant vascular supplier of the proximal humerus, because it supplies a small portion of the greater tuberosity and a small portion in the posteroinferior portion of the humeral head (30) (Fig. 17-11).



**FIGURE 17-11.** The arterial anatomy of the shoulder region.

The close proximity of the axillary nerve to the proximal humerus makes this neural structure susceptible to injury during fracture and fracture dislocations of the proximal humerus (4,27,117). The axillary nerve is a branch of the posterior cord of the brachial plexus. It traverses the anterior aspect of the subscapularis muscle before passing inferior to the glenohumeral joint to the posterior aspect of the proximal humerus (Fig. 17-12). The axillary nerve provides innervation to the deltoid muscle as well as a cutaneous sensory function over the lateral aspect of the deltoid cuff. Documentation of the normal function of this nerve before the initiation of treatment is essential.



**FIGURE 17-12.** Relationship of the brachial plexus and artery to the proximal humerus and the scapula.

Most fractures of the pediatric proximal humerus involve the physis (12,22,83). The extracapsular location of the proximal humeral physis makes this structure susceptible to injury. Physeal fractures are thought to occur through the zone of hypertrophy and spare the reserve as well as the proliferative cells in the physis (92). Therefore, children with Salter-Harris type I or II fractures still maintain excellent potential for longitudinal growth and remodeling in the proximal humerus (6,22).

In children 5 to 12 years of age, a significant proportion of proximal humeral fractures involve the metaphysis rather than the physis (22). This rather unexpected finding has been attributed to the rapid metaphyseal growth that occurs during this age, which, in turn, results in a relative structural weakness of the metaphysis (22).

Various muscular attachments to the proximal humerus contribute to the degree and the overall pattern of fracture displacement. With fractures of the physis (Salter-Harris types I, II, and III), the rotator cuff muscles displace the epiphysis into abduction, flexion, and slight external rotation, whereas the metaphysis is displaced anteriorly and medially by the pectoralis major muscle. With metaphyseal fractures proximal to the pectoralis major muscle, the proximal fragment is abducted, flexed, and externally rotated by the rotator cuff muscles. The deltoid muscle displaces the distal fragment proximally, whereas the pectoralis major muscle displaces the same fragment anteriorly and medially. If the fracture occurs between the insertions of the deltoid and the pectoralis major muscles, the proximal fragment is adducted by the pull of the pectoralis major muscle, and the distal fragment will be pulled proximally by the deltoid muscle. If the fracture occurs distal to the deltoid muscle insertion, the proximal fragment is abducted by the deltoid muscle and displaced anteriorly by the pectoralis major muscle. The distal fragment is pulled proximally and medially by the biceps and the triceps muscles (21).

### Treatment Options

Because of their tremendous potential for healing and remodeling, fractures of the proximal humerus in children rarely require operative reduction and fixation (Table 17-1). This is especially true for obstetric proximal humeral fractures in infants. These fractures are quite amenable to gentle reduction with minimal or no anesthesia. If desired, the adequacy of the reduction can be evaluated via ultrasonography. With or without anatomic reduction, the affected upper extremity should then be immobilized to the body by using a safety pin to attach the shirt sleeve to the shirt (94). Proximal humeral fractures in this age group heal quite rapidly, typically within 2 to 3 weeks, and result in no residual functional or cosmetic deficits (22,36,47,62,101,122). Nondisplaced or minimally displaced proximal humeral fractures in older children also should be treated nonoperatively. Initial management of these fractures with sling-and-swathe immobilization (Fig. 17-13) followed by protected motion provides excellent long-term results (13,22,77).

	Immobilization	Operative Reduction/Immobilization	Operative Reduction/Internal Fixation
Birth fractures	X		
Chronic slipped proximal humeral epiphysis	X		
Metaphyseal fractures	X		
SHI before age 11 yr		X	
SHI after age 11 yr			X

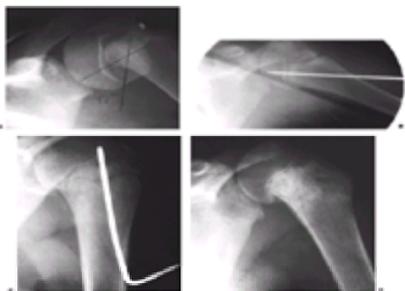
**TABLE 17-1. INTERVENTIONS FOR PROXIMAL HUMERUS FRACTURES**



**FIGURE 17-13.** Sling and swathe for immobilization of proximal humeral fracture.

Initial management of displaced proximal humeral fractures may require reduction of the fracture before immobilization. Because the remodeling potential of the fracture decreases with the increasing age of the child, the degree of acceptable displacement and angulation also changes with the age of the child. Generally, relatively greater displacement and angulation can be accepted in younger children. For fractures in children under the age of 11, good to excellent long-term outcomes can be expected regardless of the fracture displacement ([22,59,77,103](#)). Grossly displaced or angulated proximal humeral fractures in children over 11 are managed with fracture reduction and sometimes with specialized immobilization ([22,59,77,100](#)). Various types of shoulder immobilization include sling-and-swathe, thoracobrachial bandage, hanging arm cast, shoulder spica cast, salute position shoulder spica cast, and “statue of liberty” cast ([13,22,36,59,77](#)).

Some investigators recommend reducing all grade III and grade IV fractures and immobilizing grade IV fractures in salute-position shoulder spica casts ([77](#)). Other investigators suggest gentle reduction of Salter-Harris type I and II fractures in children over 11 years of age, followed by immobilization with thoracobrachial bandage or shoulder spica casts or more likely percutaneous pinning ([Fig. 17-14](#)) ([22](#)). If a Salter-Harris type II fracture cannot be adequately reduced, Dameron and Reibel recommend treatment with a salute-position shoulder spica cast with the arm positioned in external rotation, abduction, and flexion ([22](#)). An acceptable reduction of proximal humeral fractures in children over 11 years of age has been proposed by some to be less than 50% displacement and 20 degrees of angulation ([100](#)). Regardless of the specific treatment options, nonoperative treatment of pediatric proximal humeral fractures has produced good to excellent results in all age groups ([22,59,77,100](#)).



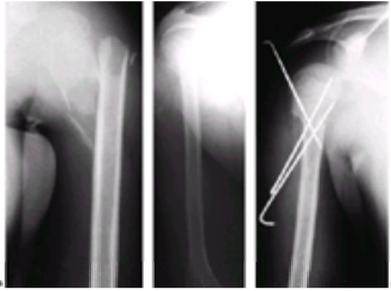
**FIGURE 17-14. A:** Salter-Harris I fracture of proximal humerus.

Multiple maneuvers exist for the reduction of pediatric proximal humeral fractures. Most fractures can be reduced by applying longitudinal traction to the arm while positioning it in abduction and flexion. If this maneuver does not sufficiently reduce the fracture, better reduction can be obtained by moderate abduction, flexion to 90 degrees, and external rotation ([77](#)). Alternatively, the fracture can be reduced by direct manual manipulation of the fragments while the arm is placed in marked abduction (~30 degrees), slight flexion (~30 degrees), and longitudinal traction ([9,47,122](#)). Despite significant efforts, however, some fractures cannot be adequately reduced because of a barrier in the fracture site. Anatomic structures that can prevent reduction of proximal humeral fractures include the periosteum, the periosteal cuff, the shoulder joint capsule, and the biceps tendon ([26,44,60,62,118](#)).

Because of the consistently good results of nonoperative treatment, indications for operative treatment of pediatric proximal humeral fractures are limited ([18](#)). Reported indications for operative interventions include open fractures, fractures associated with neurovascular injury, fractures associated with multiple trauma, displaced intraarticular fractures such as Salter-Harris type III fractures, irreducible fractures, and significantly displaced fractures in older adolescents ([12,40,60,68,73,76,98,106,120](#)).

## AUTHOR'S PREFERRED METHOD OF TREATMENT

I manage most fractures of the proximal humeral physis nonoperatively. This includes undisplaced and most displaced physeal injuries of the proximal humerus. I prefer to percutaneously pin markedly displaced fractures involving the proximal humeral physis and metaphysis, particularly those in children over 12 years of age and then apply a shoulder immobilizer or Velpeau bandage ([Fig. 17-15](#)). Almost all metaphyseal fractures are treated nonoperatively with only a few requiring closed reduction. Because of the tremendous remodeling potential of metaphyseal fractures in children, displacements in bayonet apposition of up to 1 cm are acceptable. The rationale behind closed reduction and percutaneous pinning of proximal humeral physeal fractures is a more rapid return to normal active and passive range of motion. There is a minimally increased but acceptable risk of infection. In my opinion, this approach is more acceptable than leaving the fracture unreduced. My preference is to place the pin percutaneously through the metaphyseal fragment up into the physis as in pinning of a slipped capital femoral epiphysis in the hip ([Fig. 17-14](#)). After percutaneous pinning, the arm can be brought alongside the patient's body for more acceptable and more comfortable immobilization with a shoulder immobilizer. Alternatively, intramedullary fixation with flexible nails is an effective method ([Fig. 17-16](#)).



**FIGURE 17-15. A:** Anteroposterior radiograph of displaced fracture of the proximal humerus metaphysis with shortening with apparent inferior subluxation of the humeral head with respect to the glenoid.



**FIGURE 17-16. A:** Anteroposterior radiograph of displaced, shortened fracture of the proximal humerus. **B:** Axillary image of same. **C:** Intramedullary fixation using retrograde technique. Note the satisfactory restoration of alignment. **D:** Postoperative lateral view.

Displaced fractures of the lesser tuberosity generally are treated with open reduction to restore the subscapularis tendon and anterior capsule. Lag screws or suture anchors are very useful in this region, particularly for smaller injuries.

Fractures of the greater tuberosity generally are associated with acute dislocations of the shoulder and are treated nonoperatively after following the closed reduction of the shoulder dislocation. Rarely, after closed reduction of the shoulder dislocation, the greater tuberosity fracture reduction is unacceptable, and an open approach to repair the tuberosity fracture along with the rotator cuff is required.

Fracture dislocations of the shoulder require closed reduction with appropriate anesthesia. If this is associated with a physeal injury, the physeal injury generally does not require further closed reduction or open reduction techniques. If the dislocation or fracture cannot be placed in an acceptable position, open reduction is generally done through an anterior or axillary approach to the proximal humerus.

## Complications

### Early Complications

Diagnosis of a proximal humeral fracture can be delayed in a child who is asymptomatic or minimally symptomatic. In children suffering from multiple traumas, the diagnosis can be delayed because of a low level of clinical suspicion. Even after the diagnosis of proximal humeral fracture is made, full evaluation and characterization of the fracture pattern can remain incomplete because of adequate radiographic studies. A high index of suspicion, thorough physical examination, and insistence on high-quality radiographs must all be present to ensure prompt diagnosis and treatment of proximal humeral fractures.

Neurologic injury to the brachial plexus can result from fractures and fracture–dislocations of the proximal humerus ( 4,23,27,117). Most nerve deficits can be diagnosed immediately because the clinical signs are readily apparent. Rarely, however, nerve deficits from proximal humeral fractures can evolve slowly and delay the diagnosis ( 23). Typically, these nerve deficits are transient, and full function can be expected to return in less than 6 months ( 45). If the neurologic deficit persists longer than 3 months, further evaluation with electromyography is warranted. If no evidence of nerve recovery or regeneration is present, nerve exploration, repair, and grafting can be considered ( 4,15). Salvage operations for permanent nerve deficits include proximal humeral osteotomy and muscle or tendon transfers ( 3,17,39,48,84).

Fractures of the proximal humerus in children also can be associated with other injuries, including rib fractures and pneumothorax ( 94). In adults, these fractures have been associated with disruptions and thrombosis of the axillary vessels as well ( 65,97,107,127). Operative fixation of proximal humeral fractures with pins and wires have been associated with hardware migrations that can be fatal ( 64,70). Therefore, serial radiographic monitoring of the hardware after shoulder operations is essential.

### Late Complications

Humerus varus after trauma is a rare complication that typically affects neonates and children under 5 years of age ( 25,58,69,104,113). Children with humerus varus have a significant decrease in the humeral neck–shaft angle and shortening of the upper extremity. Although shoulder abduction may be moderately limited, most children with humerus varus have only mild functional deficits and do not require surgical correction of the deformity ( 25,58,69,113). If, however, active abduction and flexion are severely limited, corrective osteotomy of the proximal humerus can produce good results ( 32,104).

Hypertrophic scarring can occur after surgical reduction of proximal humeral fractures. When the scarring is present in the anterior shoulder region after an anterior deltopectoral incision, the cosmetic deformity may be significant and psychologically damaging, especially for girls ( 26,31). Therefore, many investigators have argued for the more cosmetically appealing axillary or anterior axillary incision ( 35,63).

Limb length inequality after proximal humeral fractures occurs more frequently in children treated with surgical intervention than in those treated nonoperatively ( 6,22,91). The inequality is not significantly affected by the quality of initial fracture reduction ( 6). The discrepancy may be more pronounced in older children (1–3 cm) ( 77). Despite this inequality, however, these children rarely develop any functional deficits to warrant surgical intervention. Full arrest of physeal growth after traumatic proximal humeral fractures is extremely uncommon ( 22). Although still quite rare, it does occur more frequently in children with pathologic fractures through unicameral bone cysts ( 38,75,79). If the functional or cosmetic deficit is significant, a limb-lengthening procedure may be of benefit for these children ( 96).

Osteonecrosis of the humeral head after proximal humeral fractures occurs frequently in adults, but is rare in children ( 74,126). Even after acute disruption of the vascular supply to the proximal humeral epiphysis, subsequent remodeling and revascularization may occur in children and lead to excellent clinical results ( 120). Similarly, glenohumeral subluxation after proximal humeral fractures is a rare complication in the pediatric population that typically results in good clinical outcomes ( 126). These children are best treated with a short period of immobilization followed by early physical therapy and rehabilitation ( 94).

## FRACTURES OF THE SCAPULA

The unique anatomy of the scapula protects this bony structure from severe injury. The scapula has 17 different muscle attachments and is encased in multiple layers of muscle and other soft tissue structures. Due to this inherent protection, fractures and dislocations of the scapula are rare. In fact, it has been estimated that only 1% of all fractures involve the scapula (133,135). The significance of a scapular injury, however, is its disproportionately high association with major trauma. The high-energy traumas that cause scapular injuries also can result in significant injuries to other major organ systems (150,160,161). Therefore, all children with scapular fractures must be systematically evaluated to exclude other life-threatening injuries that require immediate medical attention and intervention.

## Diagnosis

### Mechanism of Injury

#### Glenoid

Fractures of the glenoid typically occur in a fall onto an upper extremity. This is believed to drive the humeral head onto the glenoid fossa, which, in turn, results in the fracture. Depending on the direction of the force, the fracture may only injure the rim of the glenoid or the entire glenoid fossa.

#### Scapula

Many fractures at other sites of the scapula are avulsion-type injuries from the various muscle attachments. Scapular fractures also can occur after a direct traumatic insult to the bone itself. These insults may be associated with other life-threatening injuries, such as hemothorax, pneumothorax, and cardiac contusions that require immediate medical attention (150,160). As with all other high-energy injuries, child abuse must be excluded as a cause for the scapular injury when no clear traumatic cause is evident (142).

### Signs and Symptoms

Scapular fractures or dislocations, by themselves, do not pose an immediate danger to the patient. However, over 75% of all patients with scapular fractures have other injuries (128,139,157,160). In one reported series, the rate of mortality among patients with scapular fractures was over 14% (160). Therefore, before the scapular fracture is investigated, the child must be systematically examined for other, possibly life-threatening, injuries. A full trauma evaluation should be undertaken for head, chest, abdominal, and renal injuries. If available, a consultation with the trauma service would also be prudent. Conversely, fractures of the scapula should be sought in children with life-threatening trauma.

Orthopaedic evaluation for scapular fracture should include an examination for other bony deformities and extremity tenderness. Children with scapular fractures complain of significant pain and tenderness around the shoulder girdle and decline to move the affected arm. Localized edema may obscure the overall shoulder contour. A comparison with the contralateral shoulder often is helpful in determining this loss of contour. Because of the close proximity of the scapula to the axillary artery and the brachial plexus, fractures of the scapula often are associated with neurovascular injury (160) (Fig. 17-12). The ipsilateral arm must be carefully examined to document any arterial or neurologic deficits before the initiation of treatment. When injury to axillary or distal vasculature is suspected, an angiogram should be performed to examine the integrity of the vessels.

### Radiographic Studies

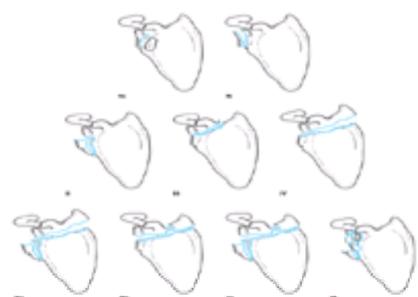
Many scapular injuries are initially identified on the AP chest radiograph of a trauma series. An AP chest radiograph also may reveal scapulothoracic dissociation. This devastating injury should be suspected if the medial border of the scapula is displaced laterally, if there is a clavicular fracture with a large displacement, or if there is a complete acromioclavicular joint separation with large displacement (155). Scapulothoracic dissociation typically occurs in patients with massive, direct trauma to the chest or proximal upper extremity and is highly associated with ipsilateral neurovascular injury (131,155).

When a scapular injury is suspected, AP and lateral view radiographs of the scapula should be immediately obtained because they are essential in characterizing the fracture. In addition to these, other views should be obtained to assess various fracture patterns. The Stryker notch view, for example, better reveals coracoid fractures, whereas the axillary lateral view is better suited to identify glenoid fractures. The axillary lateral view also is helpful in confirming the location of the humeral head on the glenoid. Because the three-dimensional structure of the scapula is complex, even high-quality radiographs may not provide all the details necessary to characterize the fracture. In such instances, CT with three-dimensional reconstructions may be required. CT also is essential in characterizing intraarticular injuries of the glenoid.

Accurate interpretation and assessment of scapular radiographs requires recognition and identification of developmental variations. Reported developmental variations of the scapula include Sprengel's anomaly, absent acromion, bipartite or tripartite acromion, bipartite coracoid, and coracoid duplication (129,140,148,156,158). Os acromiale, for example, is often mistakenly identified as a fracture. This variation occurs when the centers of ossification in the acromion fail to unite (129). Os acromiale is present in 2.7% of the general population and is bilateral in 60% (145). Typically, os acromiale is located in the anterior and inferior aspect of the distal acromion and has a smooth and uniform appearance on radiographs. If, however, radiographic studies and clinical examination cannot distinguish between a fracture and a developmental variation, further evaluation with a bone scan may be indicated (141).

### Classification

Multiple classification systems for scapular fractures have been reported. Many are descriptive and based primarily on the anatomic location. Ada and Miller, for example, classified scapular injuries into those of acromion, spine, coracoid, neck, glenoid, and body (128). In their series, fractures occurred most often in the body (35%), followed by the neck (27%). Fractures of the coracoid were least common (7%). Similarly, Thompson et al. (160) classified scapular fractures into the three broad anatomic locations: fractures of the glenoid and the glenoid neck, fractures of the acromion and the coracoid, and fractures of the body (Fig. 17-17). Other anatomic location-based scapular fracture classifications have been reported by Imatani (139) and Wilbur and Evans (161).



**FIGURE 17-17.** General classification of scapular/glenoid fractures.

The classification described below is also based on the anatomic location of the fracture, with additional subclassifications based on multiple reported studies (Fig. 17-17 and Table 17-2). It must be emphasized, however, that most of these studies are not specific for pediatric scapular fractures. Therefore, application of this classification system and its supportive studies to pediatric scapular fractures should be individualized to each child. The fracture should be adequately evaluated for its anatomic location, displacement, comminution, and articular involvement. In addition, ipsilateral neurovascular status, the overall status of the patient, and other concomitant injuries should be fully characterized.

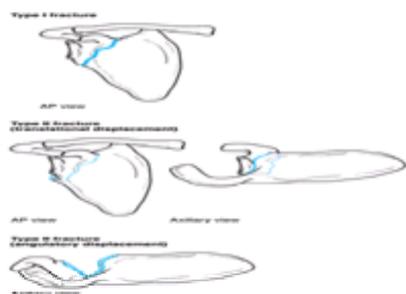
	Scapular Body Fracture	Scapular Neck Fracture—Undisplaced	Scapular Neck Fracture—Displaced	Coracoid Fracture	Acromial Fracture	Displaced Glenoid Fracture
Operation	I	X	X	X	I	
Open reduction			X			X
Open reduction/primary approach						X

**TABLE 17-2. TREATMENT OF SCAPULAR FRACTURES**

Fractures of the body and the spine of the scapula, which make up nearly 50% of all scapular fractures, are broadly categorized into those with and without displacement. Although isolated fracture of the scapular neck is believed to be a stable bony construct, ipsilateral fractures to both the scapular neck and the clavicle may lead to disruption of the suspensory mechanism of the shoulder (133,144). Therefore, fractures of the scapular neck are categorized into those with and without concomitant injury to the clavicle. For similar considerations, fractures of the coracoid process are categorized into those with and without concomitant injury to the acromioclavicular joint.

Fractures of the acromion are categorized into those with and without displacement. Displaced fractures are further subclassified based on the presence or absence of subacromial narrowing. Subacromial space narrowing may occur after inferior displacement of the acromion or after superior displacement of an ipsilateral glenoid fracture. When treated conservatively, these fracture patterns often lead to subacromial impingement in adults and result in decreased range of shoulder motion and increased shoulder pain (143). Although its applicability to acromial fractures in younger children is still debated, this finding significantly affects the treatment options for acromial fractures in older children.

Fractures of the glenoid typically occur when the humeral head is driven onto the glenoid fossa. Depending on the direction of the force applied to the humeral head, the fracture may involve the entire fossa or just the rim. If the entire fossa is involved, the fracture line may then exit in multiple locations about the scapula. Hence, fractures of the glenoid are classified into five distinct groups based on their anatomic location and course of the fracture. This system was initially proposed by Ideberg and later expanded by Goss (Fig. 17-18) (132,137,138). Type I fractures are isolated glenoid rim fractures, with Ia involving the anterior rim and Ib involving the posterior rim. Type II, III, and IV fractures are glenoid fractures with fracture lines exiting through lateral, superior, and medial aspects of the scapula, respectively. Type V fractures are various combinations of type II, III and IV fractures. Type Va, for example, is a combination of types II and IV. Type Vb is a combination of types III and IV, whereas type Vc is a combination of type II, III, and IV. Type VI fractures are comminuted fractures of the glenoid fossa. These various types of glenoid fractures are associated with distinct patterns of morbidity and treatment options.



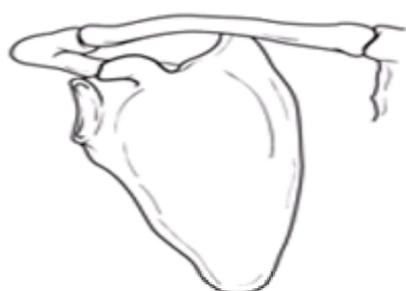
**FIGURE 17-18.** Classification of fractures of the glenoid neck.

Scapulothoracic dissociation occurs when all attachments or articulations between the thorax and the scapula are completely severed. When there is an ipsilateral neurovascular injury, it is sometimes referred to as a forequarter amputation. This is in contrast to scapulothoracic dislocation, where only the inferior scapulothoracic articulation is displaced (131). Although intrathoracic dissociations have been reported (153), scapulothoracic dissociations are typically laterally displaced. These injuries are categorized as open or closed with intact or compromised neurovascular status.

### Surgical Anatomy

During development, the scapula forms in the first trimester of gestation. It first appears near the level of lower cervical spine, C4–C7, and then descends to its final position on the lateral aspect of the upper thorax during development. Most of the scapula is formed by intramembranous ossification. Numerous centers of ossification exist for the scapula: three for the body, two for the coracoid process, two to five for the acromion (129), and one for the glenoid. These ossification centers during childhood are often mistakenly identified as fractures. In some developmental anomalies, distinct ossification centers fail to fuse and persist into adulthood (148). These conditions are also frequently characterized as fractures. With few exceptions, however, a developmental variation and a fracture can be distinguished by clinical history, physical examination, and radiographic appearance.

In its final form, the scapula is roughly triangular and has a complex three-dimensional structure. It is responsible for linking the upper extremity to the axial skeleton (Fig. 17-19) and contains attachments to 17 distinct muscles. The anterior aspect of the scapular body is a relatively flat surface, most of which is covered by the subscapularis muscle. The posterior aspect of the scapula is divided into two fossae by the scapular spine. These superior and inferior scapula fossae are mostly covered by the supraspinatus and the infraspinatus muscles, respectively. The anteromedial border of the scapular body provides attachment to the serratus anterior muscle. The posteromedial border contains the attachment sites of the levator scapulae, rhomboideus major and minor, and latissimus dorsi muscles. The omohyoid muscle attaches to the superior aspect of the scapular body, whereas the teres minor and major muscles and the triceps muscle attach to the lateral border. The scapular spine provides attachments to the trapezius and deltoid muscles, and the long head of the biceps muscle originates from the superior rim of the glenoid. Finally, the pectoralis minor muscle, as well as the conjoined tendon of the coracobrachialis and short head of the biceps muscles, attach to the coracoid process.



**FIGURE 17-19.** Relationship between the scapula, clavicle, and sternum.

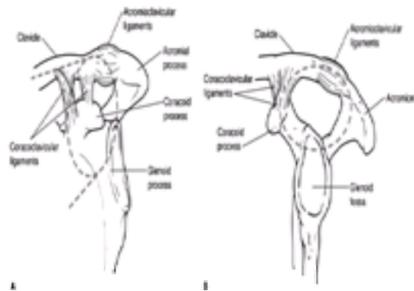
In addition to these muscle attachments, the scapula participates in the formation of both glenohumeral and acromioclavicular joints. The glenohumeral joint is stabilized by multiple dynamic and static forces about the joint, which are discussed separately. The acromioclavicular joint is stabilized in part by the presence of two coracoclavicular ligaments that position the distal clavicle immediately medial to the acromion. The two ligaments are the conoid and the trapezoid ligaments, with the conoid being the more medial of the two.

In close proximity to the scapula are a number of neurovascular structures that can be injured during a scapular fracture. Most notable are the brachial plexus and the axillary artery, which course across the anterosuperior aspect of the scapula. They are immediately posterior and inferior to the tip of the coracoid process. Medial to the base of the coracoid process is the scapular notch with the overlying transverse scapular ligament. The suprascapular nerve and artery pass under and over the ligament, respectively, in the scapular notch and are susceptible to injury with nearby fractures. The axillary nerve travels within an intermuscular interval immediately inferior to the glenoid and is also susceptible to injury with displaced fractures of the glenoid neck ( [149](#)).

### Biomechanics

The scapula can protract, retract, abduct, and elevate. Its motion augments the movements about the glenohumeral joint, including abduction, flexion, and extension. In addition, with the clavicle, the scapula acts to link the axial skeleton to the upper extremity. It provides a scaffold from which the upper extremity suspends and articulates in order to function. Therefore, its structural integrity is essential to the proper functioning of the upper extremity.

A traumatic insult may cause fractures in multiple locations about the scapula, with one fracture influencing the stability of another. Goss solidified this idea and subsequently proposed the concept of a superior shoulder suspensory complex (SSSC) ( [133](#)). The SSSC is a set of bony struts attached to a circular complex of structures at the lateral end of the scapula ( [Fig. 17-20](#)). The superior and inferior bony struts are the middle clavicle and the lateral scapula body/spine, respectively. The circular complex is composed of acromioclavicular ligament, acromion, glenoid process, coracoid process, coracoclavicular ligament, and distal clavicle. As a whole, the SSSC is responsible for linking the upper extremity to the axial skeleton. Traumatic injury to any single component of the SSSC will likely result in a minimally displaced fracture, because the inherent stability of the circular complex is still intact. However, when multiple structures of the circular complex are injured, a double disruption to the circle occurs. This, in turn, results in significant instability and displacement at one or all of the fracture sites. Similarly, injury to one of the structures of the ring complex with a concomitant injury to a bony strut also may create an unstable construct. Goss therefore proposed that the treatment decisions for scapular injuries should be based on the maintenance of SSSC integrity ( [133](#)).



**FIGURE 17-20.** Superior shoulder suspensory complex.

### Treatment Options

Isolated fractures of the scapular body do not affect the integrity of the SSSC ( [Table 17-2](#)). In addition, because of the numerous muscle attachments, fractures of the scapular body are quite stable and can be treated conservatively. Conservative treatment of nondisplaced or minimally displaced scapular body fractures in adults is generally associated with excellent results; therefore, similar treatment is recommended for equivalent fractures in the pediatric population ( [139,154,157,162](#)). In adults, scapular body fractures with significant displacement of more than 10 mm, however, resulted in unfavorable outcomes when treated conservatively ( [154](#)). Unfortunately, a comparable study of pediatric scapular body fractures has not yet been reported, and ideal treatment can only be inferred.

Nondisplaced or mildly displaced scapular neck fractures without concomitant injury to the clavicle can be treated conservatively ( [146](#)). In the presence of ipsilateral clavicular injury, however, surgical intervention generally is recommended to reestablish the SSSC ( [128,136,144,151](#)). Recommendations vary as to whether open reduction and fixation of the clavicle is sufficient to stabilize the fracture ( [136](#)) or whether the neck fracture also must be reduced in addition to the clavicle ( [144](#)). For patients in whom surgical intervention is not possible, skeletal traction may be an acceptable option ( [130](#)).

Fractures of the coracoid process typically occur at the base. Isolated fractures of the coracoid process usually are nondisplaced and can be treated conservatively with a sling and mobilization as tolerated. Displaced coracoid fractures occur with ipsilateral injury to the distal clavicle or the acromioclavicular joint. Most investigators favor open reduction and internal fixation of these fractures to restore the integrity of the SSSC ( [147,157,161](#)). Displaced coracoid fractures near the suprascapular notch with injury to the suprascapular nerve also have been described, with some investigators arguing for early exploration ( [152](#)).

Isolated fractures of the acromion in children are typically nondisplaced. In adults, acromial fractures with subacromial narrowing are associated with subsequent development of subacromial impingement when treated nonsurgically ( [143](#)). Therefore, most investigators recommend open reduction and internal fixation for displaced acromial fractures where the subacromial space has been compromised ( [143](#)). In addition, as described above, when fractures of the acromion occur with another disruption in the SSSC, the resulting deformity and instability may warrant operative fixation ( [133](#)).

Fractures of the glenoid neck typically are nondisplaced unless other elements of the SSSC are disrupted. These fractures generally have excellent outcomes with nonsurgical treatment. Significant displacement or angulation, however, may limit glenohumeral motion ( [130,154](#)). In adults, glenoid neck fractures with more than 10 mm of displacement or 40 degrees of angulation result in poor outcomes when treated without surgical reduction ( [128](#)). Therefore, it is reasonable to infer that pediatric glenoid neck fractures with significant displacement or angulation also require surgical intervention. The indications for surgery, unfortunately, remain poorly defined and must be individualized to each child.

Treatment of glenoid rim fractures (types I and II) is based on the presence or absence of shoulder instability. Closed treatment of asymptomatic glenoid rim fractures rarely result in longterm morbidity ( [163](#)). For glenoid rim fractures with resulting shoulder subluxation or instability, however, operative reduction and fixation are recommended to prevent permanent or recurrent dislocations. ( [130,134,135](#)). In adults, shoulder instability occurred when the fracture was displaced more than 10 mm or when the fracture involved more than either 25% of the anterior or 33% of the posterior aspects of the glenoid ( [130](#)). Anterior and posterior approaches to the glenoid generally are recommended for open reduction and internal fixation of anterior and posterior rim fractures, respectively ( [133](#)).

Nondisplaced glenoid fossa fractures (types III–VI) also can be successfully treated nonsurgically ( [133](#)). Displaced fractures, on the other hand, are associated with significant morbidity (pain, stiffness, and limited range of motion) when treated without surgical reduction. For glenoid fossa fractures, maximal acceptable intraarticular displacement is believed to be 5 mm ( [130,159](#)). Again, lack of definitive studies prevent the formation of absolute indications for surgery in the pediatric population. In type IV glenoid fractures, where significant comminution is present, acceptable operative reduction and fixation may be difficult to achieve ( [133,138](#)), and these fractures may be better treated with nonsurgical options ( [133,138](#)). For open reduction and internal fixation of these fractures, a posterior approach

generally provides the most acceptable exposure ([133](#)).

Initial treatment of scapular dissociations generally focuses on stabilization and repair of the neurovascular injury. If the axillary artery and the brachial plexus are completely destroyed, an early amputation should be considered ([155](#)). For most patients including children, however, the limb is salvaged whenever possible. Immediate exploration of the brachial plexus is warranted when a concomitant vascular injury requires an operative repair. In the absence of a vascular injury, however, the brachial plexus need not be acutely explored. After a period of 4 to 6 weeks, the extent of the brachial plexus injury should be documented before any surgical reconstruction, including nerve repair or musculotendinous transfer ([133,155](#)). Immediate operative stabilization of an ipsilateral clavicular fracture generally is not necessary, unless the bony instability further compromises the integrity of the neurovascular structures.

## AUTHOR'S PREFERRED TREATMENT

Most problems related to injuries of the scapula are not necessarily related to treatment but are more often related to failure to accurately evaluate associated major systems injuries ([68](#)). Therefore, once the patient is stabilized, the approach to the scapula or glenoid fractures can be more thoroughly undertaken. Fractures of the glenoid generally are treated with observation and follow-up, including a program of physical therapy and rehabilitation. For the rare displaced glenoid fracture, open reduction with internal fixation is recommended. This is generally performed via a deltopectoral approach, but posterior approaches to the scapula and glenoid may be useful in this particular injury.

Intrathoracic dislocation is rare. Most can be reduced by closed manipulative methods. In those associated with residual scapular deformity, an open approach may be required.

With scapulothoracic disassociation it is important to attend to the priorities of trauma care, including an appropriate and detailed neurovascular examination. Vascular consultation or evaluation may be required given the potential for massive injury to the brachial artery or plexus. In these instances, early or late amputation should be considered.

### Complications

Late complications associated with scapular fractures generally involve improper functioning of the upper extremity. Displaced fractures of the scapular body and spine, for example, infrequently result in upper extremity weakness and pain with movement ([128](#)). Similarly, fractures of the acromion can result in pain and decreased range of upper extremity motion secondary to subacromial impingement ([143](#)). Displaced intraarticular fractures of the glenoid are associated with glenohumeral subluxation or dislocation, as well as early progression of degenerative arthritis ([130,133,134](#) and [135](#)).

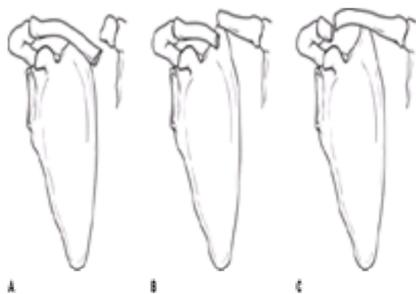
Of special consideration are the concomitant injuries frequently associated with scapular fractures ([128,139,157,160](#)). Due to their proximity, the axillary and the suprascapular nerves may be injured in association with glenoid and coracoid fractures, respectively ([149,152](#)). In addition, the energy required to create scapular fractures likely results in other injuries, such as rib fractures, pneumothorax, and vascular avulsions. All or portions of the lower brachial plexus are susceptible to injury with scapulothoracic dissociations ([131,155,158](#)). This devastating injury also has been associated with the development of compartment syndrome in the upper arm ([162](#)).

## FRACTURES OF THE CLAVICLE

The clavicle has the important function of linking the axial skeleton to the upper extremity ([Fig. 17-19](#)). It is an S-shaped bone whose medial end is connected to the axial skeleton through the sternoclavicular joint. The medial two thirds of the bone is in the shape of a tube, whereas the lateral end is flatter and is stabilized in its position by the two coracoclavicular ligaments (trapezoid and conoid). The lateral aspect of the clavicle takes part in the formation of the acromioclavicular joint, which, in effect, is connected to the upper extremity through the glenohumeral joint.

Through its joints, the sternoclavicular and the acromioclavicular, the clavicle contributes to the overall motion of the upper extremity. The clavicle can protract and retract ([228](#)). It also rotates and elevates to contribute to shoulder abduction ([164,203,228](#)). In addition, the clavicle provides the attachment site for the two predominant mobilizers of the upper extremity: the pectoralis major and the deltoid muscles. The integrity of the clavicle, therefore, is crucial to the optimal functioning of the entire upper extremity.

The clavicle is mostly subcutaneous throughout its span, being situated on the anterosuperior aspect of the thorax. Its structure is palpable and, in some cases, prominent in the upper thorax. Because of its subcutaneous location, it is one of the most frequently fractured bony structures in the body. In fact, it is the bone most commonly injured during labor and delivery, accounting for nearly 90% of all obstetrical fractures ([179,189,250](#)). In older children, clavicular fractures occur frequently, with the reported rates ranging between 8% and 15% of all pediatric fractures ([216,219,232](#)). Because of differences in the mechanism and rate of injury, prognosis, and treatment options, clavicular fractures are broadly categorized by their anatomic location: medial third, middle third, and distal third ([Fig. 17-21](#)). Most clavicular fractures occur at the middle third, with the reported rates ranging from 76% to 85% ([226,232](#)). The second most common site of clavicular injury is the distal third, with the reported rates between 10% and 21% ([226,232,243,249](#)). Fractures in the medial third of the clavicle are relatively uncommon and represent only 3% to 5% of all clavicular fractures ([232,249](#)).



**FIGURE 17-21. A:** Fracture of the medial third of the clavicle. **B:** Fracture of the middle one third of the clavicle. **C:** Fracture of the lateral third of the clavicle.

### Diagnosis

#### Mechanism of injury

The clavicle is the most common site of all obstetrical fractures, and clavicular fractures occur in 1% to 13% of all births ([177,179,189,195,196,206,237,250,251,264](#)). The incidence of obstetric clavicular fractures increases with larger fetuses and with decreasing experience of the obstetricians ([179,206,218](#)). In addition, deliveries involving the use of instruments or specialized obstetric maneuvers are more likely to result in a clavicular fracture ([179,196,206,218](#)). Therefore, it has been postulated that the fracture occurs as a result of excessive lateral to medial pressure on the shoulders during passage through the narrow birth canal. Accepting that this mechanism appears likely, it also must be noted, however, that the most clavicular fractures occur during deliveries of normal-sized to small fetuses with excellent obstetric care. Thus, it appears that obstetrical clavicle fractures are unavoidable consequences of some vaginal deliveries.

The most common mechanism of clavicular fractures in older children is a fall onto the shoulder ([215,232,260](#)). Other mechanisms include accidents where the traumatic insult is applied directly to the clavicle ([260](#)). Indirect applications of force, like falling onto an outstretched hand, are much less likely to result in clavicular

fractures (260).

A significant amount of energy can be directly applied to the clavicle during sports-related activities such as football or lacrosse. As expected, children participating in sports frequently sustain clavicular fractures as well as sternoclavicular and acromioclavicular joint dislocations (216,232). A large proportion of these injuries may be preventable with the use of protective equipment and adequate padding (258). Many children victimized by child abuse also present with clavicular fractures. Despite clinical suspicions, however, no clear pattern for isolated clavicular fractures has been associated with child abuse (201,214).

### Signs and Symptoms

Clavicular fractures in newborn infants may be difficult to identify. In the acute stage, before stabilization of the fracture, infants with clavicular fractures may voluntarily splint or not move the affected arm to minimize discomfort (180,224). Frequently, this pseudoparalysis is mistaken for a brachial plexus injury, delaying the correct diagnosis. In order to reduce the pull of the sternocleidomastoid muscle across the fracture site, these infants will also turn their heads toward the side of the fracture. In addition, infants with acute clavicular fractures typically exhibit an asymmetric Moro reflex (242,254). Generalized edema is present, which prevents the palpation of normal clavicular margins (198). Often, the diagnosis may be suspected and later confirmed only after a mass is noticed over the affected clavicle. This mass represents a healing fracture callus that forms 7 to 10 days after the initial trauma. Therefore, by the time of such delay in diagnosis, the fracture has already stabilized to the extent that it causes little discomfort to the infant.

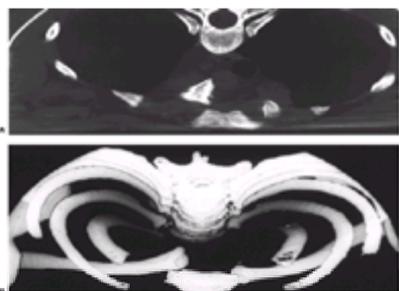
Clavicular fractures in older children typically are readily apparent and less difficult to diagnose. Children complain of moderate to severe pain around the area of the fracture and voluntarily immobilize and stop using the affected arm. Tenderness, ecchymosis, and edema, to some degree, are invariably present, and in fractures with large displacement, a bony prominence or deformity may be noted. Most children with clavicular fractures keep their heads turned to the side of the fracture in order to relax the sternocleidomastoid muscle (198). It must be cautioned that, on rare occasions, atlantoaxial subluxation and clavicular fracture may occur together. Attributing acute torticollis entirely to the clavicular fracture may delay the diagnosis of atlantoaxial subluxation, which can lead to the development of permanent atlantoaxial rotatory fixation (165).

Examination of children with either acromioclavicular or sternoclavicular joint injuries may produce crepitus and instability over the affected joint. Of special note are children with acute posterior dislocations of the sternoclavicular joint. Because of their proximity, structures of the mediastinum are susceptible to both compression and injury. Compressions of the great vessels, the esophagus, or the trachea can lead to serious and even life-threatening emergencies (193,267). Children with these injuries may complain of inability to speak, respire, or swallow. In addition, pulses in the ipsilateral upper extremity may be diminished or absent, and the neck veins may be distended. Because of the potentially life-threatening nature of this specific injury, every child with a suspected clavicular injury should be carefully evaluated for these signs and symptoms before the initiation of treatment.

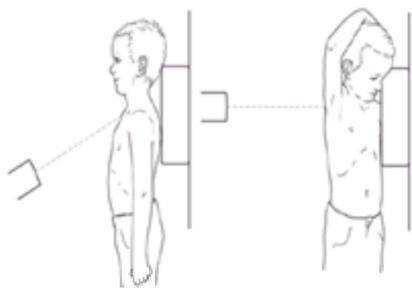
### Radiographic Studies

In all age groups, an AP radiograph is the standard study with which a clavicular fracture is initially evaluated. For neonates, however, ultrasonography has been a valuable supplement in establishing the diagnosis of clavicular fractures (199,212). Ultrasonography is particularly useful in detecting occult clavicular fractures as well as sternoclavicular joint dislocations in this early age group (199,241).

For older children, other radiographic studies may be necessary to supplement the AP radiograph in evaluating the clavicular fracture. For fractures in the middle third of the clavicle, several views may be beneficial: the cephalad-directed views, the apical oblique view, and the apical lordotic view. The cephalad-directed views are helpful in illustrating the degree of fracture displacement (Fig. 17-22). These views are taken with the x-ray beam 20 to 40 degrees cephalad to the clavicle (Fig. 17-23A). The apical oblique view is taken with the x-ray beam 45 degrees lateral to the axial axis of the body and 20 degrees cephalad to the clavicle. This view is better suited to identify fractures in the middle third of the clavicle, where significant curvature is present in the bone (265). The apical lordotic view is a perpendicular view of the anteroposterior radiograph. It is taken laterally with the shoulder abducted more than 130 degrees (Fig. 17-23B). This degree of shoulder abduction, however, can cause significant discomfort in children with acute clavicular fractures. Therefore, this radiographic view may be better suited for evaluating the healing of the clavicular fracture rather than for the initial assessment of the fracture (245).



**FIGURE 17-22. A:** Computed tomography image of the clavicle showing posterior retrosternal dislocation of the medial end of the clavicle. **B:** Three-dimensional reconstruction of image shown in A.

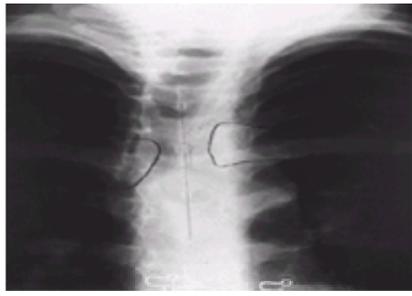


**FIGURE 17-23. A:** Cephalad-directed views. **B:** Apical lordotic view.

Fractures in the lateral aspect of the clavicle may require additional radiographic views for full assessment. In addition to the views mentioned above, an axillary lateral view is helpful in evaluating the fracture and its displacement. If the injury in this portion of the clavicle or the acromioclavicular joint is not obvious on the obtained radiographs, a radiographic stress view may provide more useful information. A radiographic stress view is an AP radiograph of the lateral clavicle with distraction on the ipsilateral upper extremity. Distraction can be achieved by asking the child to hold 5 to 10 pounds of weight with his or her hand or by simply having an assistant gently pull the arm downward. The stress view may demonstrate subtle injuries to the distal clavicle or the acromioclavicular joint. If there are concerns about the integrity of the acromioclavicular joint, computed tomography (CT scan) may be essential for full evaluation of the injury.

Fractures in the medial third of the clavicle, including sternoclavicular dislocation, are sometimes difficult to characterize even with the radiographic views mentioned above. The “serendipity” view, where a broad x-ray beam with 40 degrees of cephalic tilt projects both clavicles on the same film, is helpful for evaluating fractures in this portion of the clavicle (Fig. 17-24) (246). By comparing with the uninjured contralateral side, the location of injury and the degree of displacement often can be determined. The most rapid and effective study for evaluating injuries in the medial third of the clavicle, however, is CT. CT provides detailed information about the

morphology of the medial clavicle, the medial physis, the degree of displacement, and possible injury to the underlying intrathoracic structures. Therefore, CT is an essential part of the evaluation of injury to the medial end of the clavicle and is recommended for both acute and chronic injuries.



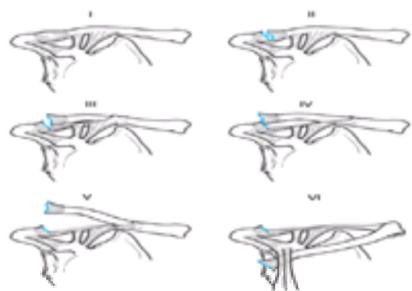
**FIGURE 17-24.** Serendipity view of the medial clavicle.

### Classification

The most widely used classification for clavicular fractures, proposed by Allman, is based on the anatomic location of the fracture. ( 166) (Fig. 17-21). Type I fractures occur in the middle third of the clavicle and generally include all fractures lateral to the sternocleidomastoid muscle and medial to the coracoclavicular ligament. Type II fractures are in the distal clavicle, including and lateral to the coracoclavicular ligament. Type III fractures, which are relatively uncommon, are medial to the sternocleidomastoid muscle. Within this general framework, further classifications exist for injuries to the distal and medial ends of the clavicle.

### Distal Clavicular Injuries

Distal clavicular injuries lateral to the coracoclavicular ligament and injuries to the acromioclavicular joint are categorized by a system proposed by Dameron and Rockwood (Fig. 17-25) (180). Although similar to the system for adult distal clavicular injuries, this classification system incorporates the observation that the distal clavicle displaces through a disruption in its periosteal sleeve rather than by true disruption of the coracoclavicular ligaments. It should be also noted that true acromioclavicular dislocations rarely occur in children. Most fractures in this region are either metaphyseal or physeal fractures ( 188,234). However, because distal clavicular epiphyseal ossification does not occur until age 18 or 19, these injuries may have the radiographic appearance of an acromioclavicular dislocation rather than a fracture (pseudodislocation) ( 188,234,261).



**FIGURE 17-25.** Dameron and Rockwood classification of distal/lateral fractures.

Type I distal clavicular injuries are caused by low-energy trauma and are characterized by mild strains of the acromioclavicular ligaments. There are no other soft tissue or bony abnormalities, and no gross changes are seen on radiographs. Type II injuries include complete disruptions of the acromioclavicular ligaments with mild damage to the superolateral aspect of the periosteal sleeve. Mild instability of the distal clavicle results, and minimal widening of the acromioclavicular joint may be seen on a radiograph. In type III injuries, complete disruptions of the acromioclavicular ligaments occur in addition to a large disruption in the periosteal sleeve. Noticeable superior displacement of the distal clavicle, with the coracoid–clavicle interval being 25% to 100% greater than the contralateral uninjured side, is seen on an AP radiograph (175,181). Similar soft tissue disruptions are seen in type IV injuries. The distal clavicle, however, is displaced posteriorly and is often imbedded in the trapezius muscle (169). Minimal changes may be noted on an AP radiograph, so an axillary lateral view is required to identify the posterior clavicular displacement. Type V injuries are similar to type III injuries. The difference lies in the fact that the superior aspect of the periosteal sleeve is completely disrupted in type V injuries. This allows subcutaneous displacement of the distal clavicle, occasionally splitting the deltoid and the trapezius muscles. On an AP radiograph, the coracoid–clavicle interval is more than 100% greater than the contralateral uninjured side. In type VI injuries, the distal clavicle is displaced inferiorly with its distal end located inferior to the coracoid process ( 194).

### Medial Clavicular Injuries

The medial physis of the clavicle is the last physis in the body to close, and the fusion of this epiphysis to the shaft occurs as late as 23 to 25 years of age ( 208,239). The sternoclavicular ligaments attach primarily to the epiphysis, leaving the physis unprotected outside the capsule ( 214). Because of its unique anatomy, traumatic insults to the medial end of the clavicle in children typically result in fractures through the physis rather than dislocations through the sternoclavicular joint. Therefore, these injuries are categorized most appropriately in the Salter-Harris classification system ( 252). Most fractures at the medial end of the clavicle are Salter-Harris type I or II fractures. These fractures are further subdivided by the direction of the clavicular displacement, either anterior or posterior. Although anterior displacement of the clavicle occurs more frequently, more attention is given to fractures with posterior displacement due to the possibility of concomitant mediastinal injuries and potential emergent nature of the injury.

### Surgical Anatomy

The clavicle appears early during embryonic development. By the 5th or 6th week of gestation, it begins ossification at two separate centers, medial and lateral (192,228,233). By the 7th or 8th week of gestation, its overall contour and shape are already formed ( 192). During childhood, approximately 80% of clavicular growth and lengthening occur at the medial physis ( 223,236). Despite this early ossification and growth, complete growth of the clavicle does not occur until early adulthood. The lateral physis continues to proliferate until 18 to 19 years of age, and the medial physis does not close until 23 to 25 years of age ( 238,239,261).

The distal clavicle articulates with the scapula through the acromioclavicular joint, a joint that lacks inherent structural stability. It is held together in part by the acromioclavicular ligaments, which are relatively weak secondary stabilizers. The primary stabilizers of the joint are the two coracoclavicular ligaments, the conoid and the trapezoid, which place the lateral end of the clavicle immediately next to the acromion. Although the distal clavicle and the coracoid process usually do not articulate, a coracoclavicular joint has been reported in adults ( 213). In children, the distal clavicle and the acromion are surrounded by thick periosteum that forms a protective tube around the bony structures. The coracoclavicular ligaments are attached to the periosteum on the inferior surface of the distal clavicle. Because these ligament attachments are stronger than the periosteum, displacement of the distal clavicle occurs through a disruption in the periosteum rather than by detachment of the ligaments. In fact, displacement of the distal clavicle through this periosteum in children has been likened to having “a banana being peeled out of its skin.” As mentioned above, the distal clavicular physis does not ossify until early adulthood ( 261). Therefore, fractures through the distal clavicular physis or metaphysis may

be mistakenly identified as acromioclavicular joint dislocations.

Medially, the clavicle articulates with the sternum and the first rib through the sternoclavicular joint. Similar to the acromioclavicular joint, this joint also lacks inherent structural stability. It is held together by a series of strong ligaments, including the intraarticular disc ligament, the anterior and posterior capsular ligaments, the interclavicular ligament, and the costoclavicular ligament ( [172](#)). In children, the medial physis of the clavicle is still open, and the capsular ligaments attach primarily to the epiphysis ( [172,208,239](#)). Therefore, injuries to the medial clavicle typically result in physeal fractures with the epiphysis attached to the sternum.

Along its course, the clavicle also serves as attachment sites for a number of different muscles. On its superior surface, the clavicular head of the sternocleidomastoid muscle is attached. On the posterior surface, the trapezius muscle is attached, whereas the pectoralis major and the deltoid muscles are attached on the anterior surface. Inferiorly, the clavicle provides attachment sites for the subclavius muscle as well as the clavipectoral fascia.

In addition to these muscle attachments, the clavicle provides protection to the subclavian vessels and the brachial plexus. These vital structures are located posterior to the clavicle, crossing the clavicle at the junction between the medial two thirds and lateral one third of the bone ( [Fig. 17-12](#)). Due to this close proximity, the neurovascular status of the ipsilateral upper extremity may be jeopardized in children with displaced clavicular shaft fractures. In addition, as discussed above, posterior dislocation of the sternoclavicular joint can lead to compression or injuries of the great vessels within the mediastinum. Therefore, neurovascular status of the ipsilateral upper extremity must be documented before the initiation of treatment for any clavicular injury.

### Biomechanics

The clavicle contributes significantly to the overall motion and optimal function of the upper extremity. In the anterior to posterior direction, the clavicle can protract and retract about 35 degrees ( [228](#)). Laterally, it can rotate and elevate to contribute approximately 30 degrees to full shoulder abduction ( [164,203,228](#)). The clavicle also provides the attachment sites for the major mobilizers of the upper arm in the pectoralis major and the deltoid muscles. Finally, together with the scapula, the distal clavicle forms the SSSC. As proposed by Goss, the SSSC provides a scaffold from which the upper extremity suspends and articulates in order to function ( [197](#)).

### Treatment Options

#### General Comments

Numerous methods of treating clavicular fractures have been described. In fact, it has been stated that over 200 methods of treatment exist ( [215](#)). For most of these pediatric fractures, nonoperative treatment should be the prevailing rule rather than the exception. In addition, the importance of prevention cannot be overemphasized, because it is certainly the best of all the treatment options. Improved obstetrical care, use of adequate equipment and padding in contact sports, and updated safety equipment and conditions at child recreational facilities should lead to significant reductions in the incidence and complications of clavicle fractures.

#### Middle Third Fractures

Treatment of obstetrical clavicular fractures is always nonoperative. For most birth-related clavicular fractures, minimal or no treatment is recommended. If the infant appears to be in significant discomfort, the affected arm can be immobilized to the body for a short period of time, typically less than 2 weeks. Immobilization of the affected arm can be easily and effectively accomplished by using a safety pin to attach the long shirt sleeve to the shirt ( [211,221,253](#)). The parents should be warned to not disturb the upper extremity by unnecessary excessive movements in the acute period. In addition, they should be informed that the infant will develop a noticeable mass over the fracture site which will typically resolve within 6 months ( [242](#)).

Good to excellent results also can be expected from nonoperative treatment of most clavicle fractures in older children. A figure-of-eight splint is an acceptable method of nonoperative treatment and has been widely used with successful outcomes ( [207,230,242,255](#)). It can be applied directly or after an attempt at closed reduction with retraction of the shoulders ( [235](#)). In general, younger children do not require reduction of the fracture because their potential for remodeling is greater ( [235](#)).

The figure-of-eight splint, unfortunately, can be uncomfortable for some children. In addition, inappropriate use of the splint, on rare occasions, can lead to a number of complications, including edema, compression of the axillary vessels, and brachial plexopathy ( [Fig. 17-26](#)) ( [191,217,230](#)). Use of a sling, on the other hand, is typically well tolerated by children and is not associated with any of these complications. Treatment of both nondisplaced and displaced clavicular fractures with a sling has shown remarkably good results. In fact, in comparison with a figure-of-eight splint, treatment of clavicular fractures with a sling resulted in similar final outcomes ( [167,207,259](#)). Therefore, it appears that nonoperative treatment of middle third clavicular fractures with a simple sling can result in excellent outcomes without compromising the child's comfort.



**FIGURE 17-26. A:** Anteroposterior radiograph of comminuted clavicular fracture. **B:** Skin erosion over fracture fragment due to figure-of-eight splint.

Reported indications for operative treatment of clavicular fractures include severely displaced and irreducible fractures that threaten skin integrity, concomitant vascular injury requiring repair, irreducible compression of the subclavian vessels, compromise of the brachial plexus, and open fractures ( [202,204,229,240,268](#)). In addition, as discussed separately in this chapter, concomitant displaced fractures in various regions of the scapula, including the acromion, the coracoid, and the scapular neck, may compromise the SSSC and require operative repair ( [197](#)).

#### Distal Third Fractures

Injuries to the distal clavicle in the pediatric population typically are pseudodislocations of the acromioclavicular joint, with fractures through the metaphysis or the physis ( [186,234](#)). The acromioclavicular joint and the coracoclavicular ligaments usually are undamaged, and most of the periosteal sleeve is intact. Therefore, exceptional potential for growth and remodeling exists for these fractures, allowing successful nonoperative treatment for most injuries to the distal clavicle.

Most investigators agree that undisplaced or minimally displaced injuries of the distal clavicle (types I, II, and III), should be treated without surgery ( [171,180,200,234,247](#)). These injuries are managed with a sling or a figure-of-eight splint immobilization followed by early rehabilitation with range of motion exercises. Most children treated with nonoperative management demonstrate no significant long-term functional or cosmetic deficits.

The treatment of displaced types IV, V, and VI distal clavicle fractures remains controversial. Some investigators report that most children experience no functional deficits regardless of the method of treatment ( [171,200](#)). Others report that distal clavicular injuries with either fixed or gross displacement should be treated with open reduction and internal fixation to prevent permanent deformity ( [168,175,180,182,188,200,234,248](#)). One report suggested that although displaced distal clavicular injuries in children under 13 may be amenable to nonoperative treatment, those in children over 13 years of age should be treated with open reduction and internal

fixation ([186](#)).

Although no clear consensus exists for the treatment of grossly displaced distal clavicular fractures in children, as long as the integrity of the SSSC is maintained, it appears that neither nonoperative or operative management results in long-term deficit in the normal function of the shoulder. Treatment options, therefore, should be individualized for each child and his or her family based on their compliance as well as their acceptance of the possible cosmetic deformity.

### **Medial Third Clavicular Injuries**

Most pediatric injuries in the medial clavicle are fractures through the physis. Similar to distal clavicular injuries, these fractures have vast potential for healing in an acceptable position, and subsequent remodeling and nonoperative management is appropriate.

Nondisplaced fractures of the medial physis do not require active intervention. Symptomatic treatment is all that is required for these stable fractures. In fact, nondisplaced fractures often are missed during initial examination and are only discovered after a mass is noted over the medial clavicle. The parents should be warned that the mass is a healing callus surrounding the fracture and that it should remodel and disappear in 4 to 8 months.

Most fractures with anterior displacement also should be treated without surgery. Open reduction and internal fixation of these injuries may lead to significant, and sometimes life-threatening, complications ([178,190,223,224](#)). Attempts at closed reduction, especially in younger children, are not required because these fractures will remodel and result in minimal or no residual deformity. The maneuver for the closed reduction requires applying longitudinal traction to the ipsilateral upper extremity while the shoulder is abducted to 90 degrees ([253](#)). Gentle posterior pressure also should be applied over the fracture to encourage reduction. After the reduction is accomplished, the clavicle should be immobilized with a figure-of-eight splint or cast ([253](#)).

Treatment of medial clavicular fractures with posterior dislocation requires immediate evaluation for the presence or absence of concomitant mediastinal injuries. If physical examination and radiographic evaluations do not reveal mediastinal injuries, these fractures are treated with a controlled closed reduction. The fractures can be expected to heal and remodel without any significant residual deformity or pain. If mediastinal structures are compromised or injured, immediate attempts at closed reduction are required. Under adequate anesthesia, the displaced medial clavicle is held manually or with a towel clip and manipulated anteriorly to reduce the fracture. This reduction maneuver should be performed while longitudinal traction is applied to the ipsilateral upper extremity ([253](#)). If closed reduction is unsuccessful, open reduction, with the assistance of a thoracic surgeon, may be required. Once reduced, these fractures typically are stable and require no internal fixation. Infrequently, however, some fractures may involve a physeal disruption of the medial border and require a suture repair. Immobilization with a figure-of-eight splint or cast for a short period of time should provide adequate environment for fracture healing.

### **AUTHOR'S PREFERRED METHOD OF TREATMENT**

The approach to neonatal or birth injuries is one of diagnosis and reassurance and education of the parents. The family is told that a bump will develop over the fracture site and that the fracture will heal uneventfully. If the infant initially demonstrates discomfort with the fracture, the long arm sleeve of the infant's shirt can be pinned to the shirt for 7 to 10 days to provide adequate immobilization.

Older children who present to the emergency room generally have a significant level of pain and discomfort. I prefer to use a figure-of-eight harness to provide retraction of the shoulder, to gain length at the level of the fracture, and to reduce pain. With the use of the figure-of-eight harness, it is important to inspect the skin on weekly follow-ups for 3 weeks to assure that no unusual sharp bone fragments create any skin problems at the site of passage of the figure-of-eight harness over the fracture ([Fig. 17-26](#)). The parents are once again informed that the bone will take a couple of months to remodel and that there may be a bump for up to a year after the fracture.

The indications for operative management for clavicular fractures in children are rare and include fractures with the potential to develop both thickness loss over the apex of a fracture or a direct impingement of the clavicle on either the brachial plexus or subclavian vessel. Even with these fractures, gentle manipulation and closed reduction should be attempted. If open repair is done, the fractured clavicle generally can be placed into the periosteal sleeve and the periosteal sleeve can be repaired over the fractured clavicle without the need for additional internal fixation.

Most fractures of the medial end of the clavicle, with or without anterior displacement or posterior retrosternal dislocations, can be treated nonoperatively. Generally 3 to 6 weeks in a figure-of-eight harness or sling is adequate after stable reduction.

Anterior displacements of the medial end of the clavicle generally are associated with a physeal disruption and usually can be treated with closed reduction. Longitudinal traction is applied to the upper extremity with moderate abduction of the humerus and general pressure is applied over the scapuloacromioclavicular joint. Persistent instability of the scapuloacromioclavicular joint is acceptable in anterior displacements because of the significant potential for remodeling in this area. After 2 to 4 weeks of immobilization, a program of progressive rehabilitation can begin.

Posterior dislocations of the medial end at the clavicle of the sternoclavicular joint may be either pure dislocations or associated with a physeal disruption and may be acute life-threatening injuries. Most of these injuries can be treated successfully with closed reduction with general anesthesia and stand-by support of the cardiovascular service. The technique for the closed reduction is quite specific and involves the placement of a bolster in the midline along the level of the spine and spinous processes. Both humeri are adducted to the level of the chest, and anterior pressure is placed over the deltoid and humeral head toward the table with a downward pressure over both proximal humeri. This is generally sufficient to provide adequate retraction of the shoulder and restore the length of the clavicle at the level of the sternoclavicular joint. Further downward pressure to the level of the table provides a fulcrum force to reduce the medial end of the clavicle anteriorly into the sternoclavicular joint. Rarely a towel clip may be required. It is placed subcutaneously through the medial third of the clavicle to aid in the reduction process. Open reduction of the medial end of the sternoclavicular joint is indicated when closed reduction fails or results in an unstable retrosternal displacement. If the dislocation is unstable, generally repair of the capsule with a nonabsorbable suture through the capsule of the joint at the level of the sternum through holes drilled into the medial end of the clavicle is sufficient to provide anterior stability of the dislocation. Internal fixation is not recommended in this location.

Most injuries to the distal end of the clavicle in children and adolescents are treated nonoperatively. These fractures heal rapidly because of the early deposition of periosteal new bone and remodeling. Generally, patients can be treated with a sling and pain management with appropriate oral analgesics and ice to control swelling. Early range-of-motion therapy is recommended at approximately 10 days to 2 weeks. Clinical union is generally seen by 4 to 6 weeks.

For the rare type IV, V, or VI displaced distal clavicular injury, an open approach can be useful in replacing the distal clavicle in its periosteal sleeve, and repair of the periosteal sleeve may be sufficient to provide adequate fixation.

### **Complications**

Implants and internal fixation devices for clavicular fractures have been associated with numerous complications, including hardware migration, infection, and nonunion ([178,190,223,224,231,256,257](#)). Although most of these complications can be adequately treated, some can have fatal results ([178](#)). Therefore, whenever possible, fixation of pediatric clavicular fractures should use minimal or no hardware.

Serious vascular injuries also have been described in association with clavicular fractures, including subclavian and axillary artery disruption, subclavian vessel compression, and arteriovenous fistula ([170,202,209,227,262](#)). In addition, displaced fractures of the medial clavicle may result in compression or injury of the great vessels within the mediastinum ([193,267](#)). Occasionally, these compressions can be relieved nonoperatively by reducing the fracture and eliminating the excessive pressure on the vessels ([202,227](#)). However, if nonoperative treatment does not alleviate the compression, operative reduction of the fracture and possible vascular repair may be required. Certainly, if the structural integrity of the vessels is compromised, operative repair by an experienced vascular or thoracic surgeon is necessary.

In addition to the compression of the great vessels, displaced medial clavicular fractures can result in compression of the trachea and esophagus, causing difficulty with respiration or swallowing ([193,267](#)). Clavicular fractures resulting from severe trauma can be associated with pneumothorax ([185,225](#)). Rarely, a pneumothorax results from obstretical clavicular fractures ([220](#)).

Neurologic deficits of the brachial plexus have been reported in association with clavicular fractures. Brachial plexus palsy may present early or late after the traumatic insult, and occasionally requires operative reduction of the fracture ( [170,183,184,202,205](#)). Rarely, such nerve deficits can result from inappropriate use of the figure-of-eight splints ( [217,230](#)). Although permanent nerve deficits have been reported, most brachial plexus injuries resolve spontaneously ( [205,210](#)).

Although malunions are frequent soon after fracture healing, most children experience no long-term deformities because of their tremendous potential for remodeling. Occasionally, however, significant deformities such as clavicular reduplication and cleidoscapular synostosis may require further intervention ( [234,244](#)).

Clavicular pseudarthrosis may be congenital, traumatic, or pathologic ( [174,176,231,238,243,263,266](#)), but most often it results from previous injuries. Clavicular pseudarthroses, especially congenital or embryonic types, often are completely asymptomatic and require no treatment. Operative indications for pseudarthrosis include unacceptable cosmetic deformity and pain ( [173,176,222,266](#)). It must be noted, however, that operative repair with grafting and internal fixation of the pseudarthroses can result in other complications, such as pneumothorax, subclavian vessel damage, air embolism, and brachial plexus deficit ( [187](#)).

## FRACTURES OF THE HUMERAL SHAFT

### Embryology and Development

The end of the embryonic period is marked by vascular invasion of the humerus at age 8 weeks. During the subsequent fetal period, the humerus resembles the adult bone in both form and muscular relationships ( [357,364](#)). A bony collar is present very early with subsequent enchondral bone formation. The secondary ossification centers at the ends are not generally ossified radiographically until after birth ( [364](#)).

### Applied Anatomy

#### Osseous

The proximal metaphysis of the humerus is wider than the thinner, triangular shaft. Distally, this flattens and widens to form the condylar region of the elbow. The deltoid inserts into a protuberance midway down the shaft known as the deltoid tuberosity. Distal to the tuberosity, the muscular spiral groove wraps posteriorly around the humerus. The groove gives origin to the uppermost fibers of the brachialis. The periosteum of the humeral diaphysis is thick and provides good remodeling potential ( [327,331](#)). The main vascular foramen is at mid-shaft, but accessory foramina are common—most enter the anterior surface usually below the main foramen, but many are posterior ( [308,364](#)).

#### Nerves

The radial nerve ordinarily lies close to the inferior lip of the spiral groove but not directly in it ( [515](#)). The profunda artery either accompanies the radial nerve or passes in a second narrower groove. The nerve is protected from the humerus by a layer of either the triceps or the brachialis until the lower margin of the spiral groove near the lateral intermuscular septum ( [515](#)). The ulnar nerve passes from anterior to posterior just distal to the humeral mid-shaft. A well-formed arcade and internal brachial ligament may hold the ulnar nerve ( [395](#)). This ligament is always posterior to the medial intermuscular septum and subsequently joins the medial intermuscular septum proximal to the medial epicondyle. A few patients with a modified arcade have only superficial fibers of the triceps medial head passing superficial to the ulnar nerve and none deep to the nerve, making the nerve very close to the bone and vulnerable during a fracture ( [395](#)).

#### Muscles

Several major muscle attachments occur throughout the metaphyseal and diaphyseal regions of the humerus. The pectoralis major muscle inserts laterally and distal to the bicipital groove along the anterior aspect of the humerus. The latissimus dorsi and teres major insert on the upper medial aspect of the humerus medial to the bicipital groove. The deltoid courses from the clavicle, acromion, and scapular spine to insert over a broad area of the deltoid tuberosity. The coracobrachialis arises from the coracoid process and inserts on the anterior medial aspect of the humerus at the junction of the middle and lower thirds. The brachialis originates from the anterior humerus about midway down the shaft. Knowledge of these muscles and their directions is essential to understand fracture displacement and treatment ( [327,383](#)).

### Mechanisms of Injury

#### Birth Injuries

Humeral fractures are more common in breech presentations and with macrosomic infants. The most difficult position is when the child's arms have gone above the head with maneuvers to bring the arm down after version and extraction ( [423](#)).

#### Child Abuse

Humeral fractures in child abuse represent 61% of all new fractures and 12% of all fractures ( [417,441](#)). Shaw et al. ( [482](#)), in a retrospective review of 34 humeral shaft fractures in children under 3 years of age, found that most occurred accidentally: only 6 were classified as caused by probable abuse. Child abuse must be part of the differential diagnosis in children with humeral diaphyseal fractures ( [441](#)). The fractures may be spiral from a twisting injury or transverse from a direct blow.

#### Older Children

Older children sustain primarily transverse fractures from direct blows to the arm, frequently from falls, pedestrian/vehicle accidents, gunshot wounds, and machinery. Sports injuries are direct from contact sports or indirect from throwing. Throwing injuries occur as a stress injury from overuse or acutely during the throwing cycle from poor mechanics ( [269,303,359,363,366,381,415,490,493,502,514](#)). A stress fracture also has been reported in an adolescent tennis player ( [458](#)). Acute throwing fractures result from a sudden external rotation torque developed on the distal humerus with concomitant proximal internal rotation from the pectoralis major between the cocking and acceleration phases ( [339](#)) as the shoulder external rotation and elbow flexion suddenly change to shoulder internal rotation and elbow extension. Humeral fractures may occur from arm wrestling in older adolescents ( [284,418,433](#)). Many humeral fractures are pathologic through simple bone cysts or through dysplastic bones from osteogenesis imperfecta or fibrous dysplasia. Occasionally, pathologic fractures occur from benign or malignant tumors.

### Classification

The simplest classification for humeral diaphyseal fractures describes the location (proximal third, middle third, or distal third, or the diaphyseal–metaphyseal junction), the pattern (spiral, short oblique, or transverse), the direction of displacement, and any tissue damage. Anatomically, the location is noted as proximal to the pectoralis major insertion, between the pectoralis major and deltoid insertions, below the deltoid insertion, or at the distal metaphyseal–diaphyseal junction ( [332](#)). Humeral shaft fractures may be segmental, with fractures of the shaft and neck ( [492](#)), or associated with shoulder dislocation ( [283](#)). If they are associated with fractures of the ipsilateral forearm, they result in the so-called “floating elbow” ( [491](#)).

The Association for the Study of Internal Fixation (AO-ASIF) has a classification for humeral shaft fractures ( [435](#)), but it is not very applicable to most children's fractures, and like most classifications, it is subject to interobserver variability ( [393](#)).

### Incidence

Fractures of the humeral shaft represent 10% or less of humerus fractures in children ( [316,392,412](#)) and 2% to 5.4% of all children's fractures ( [316,513](#)). They are most common in children under 3 and over 12 years of age ( [285](#)). The incidence is greater in children with more severe trauma ( [470](#)). The incidence is 12 to 30 per 100,000 per year ( [404,513,518](#)). Birth injuries to the humerus have a reported incidence ranging from 0.035% to 0.34% ( [306,423](#)).

## Signs and Symptoms

### *Evaluation of the Neonatal Shoulder*

#### History

The infant who does not move the shoulder poses a diagnostic challenge. Establishing and evaluating a differential diagnosis is the first concern. By history, was the delivery normal? When was the problem noticed? Does the child move any part of the extremity? Was there a history of maternal gestational diabetes or of fetal macrosomia? Does the child nurse from each breast? A broad, useful differential diagnosis consists of clavicle fracture, proximal humeral physeal fracture, humeral shaft fracture, shoulder dislocation, brachial plexus palsy, septic shoulder, osteomyelitis, hemiplegia, and child abuse.

#### Examination

Initially, the child should be observed for spontaneous motion of the upper extremity. Is there any hand or elbow motion? Are there any areas of swelling, ecchymosis, or increased warmth? Does the child move the ipsilateral lower extremity? The clinician should carefully palpate each area of the upper extremity, starting with the clavicle and comparing it carefully with the opposite side for any change in soft tissue contour or tenderness. The upper arms and shoulders should then be examined, looking for any tenderness in the supraclavicular fossa. Lastly, the spine should be examined for tenderness or swelling.

#### Imaging Studies

Radiographs may be needed of the shoulder, clavicle, humerus, and cervical spine. Often the shoulder, clavicle and humerus can be seen on a single anteroposterior view of both upper extremities and the chest. Ultrasonography can be used to identify a fracture of the clavicle or the proximal humeral epiphysis, a shoulder dislocation, or a shoulder effusion. A computed tomography (CT) scan or arthrogram may be necessary. The radiographic findings for each fracture are discussed in the particular anatomic sections.

#### Birth Fractures of the Humerus

In the newborn, a humeral fracture can simulate a brachial plexus palsy with pseudoparalysis and an asymmetric Moro reflex. The fracture site is tender and may have swelling or ecchymosis. The diagnosis is confirmed by plain radiography ([387,423](#)).

#### Older Children

In older children, the diagnosis is usually evident with pain, swelling, and unwillingness to move the arm. The arm is often supported by the opposite hand and is held tightly to the body ([Fig. 17-27](#)). It is essential to perform a complete neurologic and vascular examination of the extremity before any treatment except emergency splinting.



**FIGURE 17-27.** A young patient with a humeral shaft fracture, holding the arm tightly to his side.

Children with torus or greenstick fractures may have localized tenderness but no deformity. In multiple-trauma victims, careful evaluation should be made of the arm because the diagnosis can be missed, especially if the patient is medically unstable ([406](#)). Humeral fractures should be sought in patients with massive upper extremity trauma.

#### Radiographic Findings

Birth fractures of the humerus are usually quite apparent on anteroposterior and lateral radiographs of the humerus. In older children, radiographs should be taken in both the anteroposterior and lateral planes to obtain two films perpendicular to each other. Most fractures are easily visualized on these radiographs. A true lateral view of the distal humerus is noted by superimposition of the posterior supracondylar ridges of the medial and lateral epicondyles ([488](#)). A supracondylar process of the humerus, when present, is best seen on an oblique radiograph showing the anterior medial aspect of the distal humerus.

Displaced fractures above the pectoralis major have marked abduction of the proximal fragment with external rotation by the rotator cuff attachment ([327,332](#)). The distal fragment is pulled proximally by the deltoid and medially by the pectoralis major. Displaced fractures between the pectoralis major and deltoid insertions show adduction of the proximal fragment from the pectoralis major and shortening by pull of the deltoid on the distal fragment. Fractures below the deltoid insertion have abduction of the long proximal fragment by the deltoid, but with shortening and medial displacement of the distal fragment by the pull of the biceps and triceps ([332](#)).

Pathologic bone may be evident ([500](#)). Simple bone cysts are a common cause of fractures. Periostitis or periosteal reaction of the humerus necessitates differentiating osteomyelitis or Ewing's sarcoma from a stress fracture; every effort must be made to identify a cortical fissure using other imaging techniques ([275,299](#)).

Holstein and Lewis described a short oblique fracture of the distal third of the humerus with potential radial nerve palsy after closed reduction ([385](#)). This has been called the Holstein-Lewis fracture.

#### Treatment

##### *Birth Injuries*

Neonatal humeral shaft fractures heal and remodel quite well, with 40% to 50% remodeling within 2 years ([Fig. 17-28](#)) ([289](#)). Reported treatments include a sling and swathe ([384](#)) or a traction device using the von Rosen splint ([277](#)). The primary potential complication of birth injuries is an internal rotation deformity. Therefore, the fracture is best stabilized by splinting the arm in extension. If the parents will be moving the child, the splinted arm can be bound to the chest with a soft wrap. Children with arthrogryposis and brachial plexus palsies are prone to internal rotation contractures of the shoulder; these can be exacerbated if the birth fracture's rotation is not controlled.



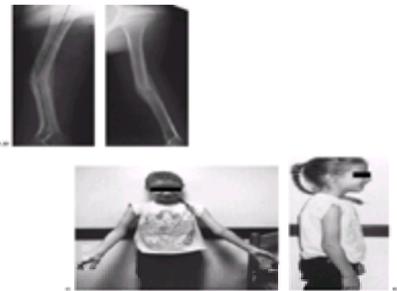
**FIGURE 17-28.** **A:** Fracture of the left humerus in a neonate that occurred during a difficult delivery. **B:** After 2 weeks of immobilization, clinical and radiographic union is evident, but with anterolateral angulation. **C:** At 2 months after injury, there is considerable remodeling. **D and E:** At 20 months, there is essentially complete remodeling of the fracture.

### Stress Fractures

Virtually all nondisplaced stress injuries heal well with temporary rest and immobilization ([269,337,359,363,415,458,493,503,514](#)). They can displace if not treated ([269](#)). Displaced stress fractures should be treated like other humerus fractures.

### Acceptable Alignment

Because the humerus is not a weight-bearing bone, it does not require the precise mechanical alignment of the lower extremity. The marked mobility of the shoulder also allows some axial and rotational deviation without functional problems. Severe internal rotation contractures can cause difficulties in some overhead activities such as ball throwing and facial hygiene. Varus of 20 to 30 degrees is necessary before becoming clinically apparent ([Fig. 17-29](#)) ([332,397,457](#)). Anterior bowing may be apparent with 20 degrees angulation ([397](#)). Functional impairment does not occur with 15 degrees or less of internal rotation deformity ([332](#)). Even adolescents can correct up to 30 degrees spontaneously ([332](#)). Beaty ([285](#)) gives guidelines based on the patient's age: children under 5 years of age tolerate 70 degrees angulation and total displacement, children 5 to 12 tolerate 40 to 70 degrees angulation, and children over 12 tolerate 40 degrees and 50% apposition. However, bayonet apposition is acceptable ([292–294](#)), with 1 to 2 cm of shortening well tolerated ([Fig. 17-30](#)). Clinical appearance is more important than radiographic alignment.



**FIGURE 17-29.** **A and B:** Radiographic appearance of a malunited humerus fracture showing 20 degrees of varus. **C and D:** The same patient with no deformity or disability, despite her thin extremities.



**FIGURE 17-30.** **A:** Humerus fracture allowed to heal in slight varus and bayonet apposition. **B and C:** The ultimate result, with essentially normal alignment.

### Nonoperative Treatment

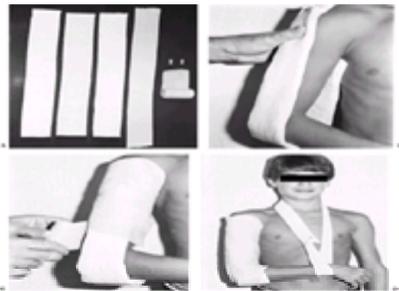
Nonoperative treatment often increases internal rotation by 3 to 12 degrees at the expense of external rotation ([332](#)). This rarely is a functional problem. Nonoperative methods include a sling and swathe, the U plaster, a hanging arm cast, a thoracobrachial cast or dressing, a coaptation splint or functional bracing, and traction.

#### Sling and Swathe

The simplest form of treatment for fractures is a sling and swathe. It is sufficient for patients with minimally displaced greenstick and torus fractures ([375,457](#)). Although this treatment may yield good results in displaced fractures ([489](#)), it can be quite difficult to control anterior angulation ([386](#)) and may be uncomfortable.

#### U Plaster-Sugartong

Böhler ([292](#)) described a U plaster similar to the sugartong splint used on forearms. Plaster of appropriate width for the upper arm is formed from over the shoulder along the lateral aspect of the arm, underneath the olecranon, and along the medial aspect of the arm to the axilla. Cotton webbing is placed between the plaster and the skin, and the plaster is secured using a wrap ([Fig. 17-31](#)). Results have been quite good ([332](#)), particularly in children ([398](#)). Holm ([384](#)) suggested applying benzoin before the cotton webbing and using a collar-and-cuff sling about the wrist. To prevent slippage, Shantharam ([477](#)) suggested applying the splint from the base of the neck, over the shoulder, and around to the axillary fold, with a strap securing the proximal end to the chest. The U plaster may not control alignment satisfactorily in more displaced fractures, which may require a thoracobrachial cast ([293,384,457](#)) or internal fixation. Böhler actually abandoned the immediate use of the U plaster for a thoracobrachial cast because of problems with early swelling ([293,294](#)).



**FIGURE 17-31.** Coaptation splints with collar and cuff. **A:** The material used for a sugartong arm splint is two pieces of cast padding rolled out to the length of the plaster-of-paris splint and applied to each side of the splint after it is wet. The splint is then brought into the tubular stockinette of the same width but 4 inches longer than the splint. **B:** The plaster splint is applied to the arm from the axilla up to the tip of the acromion. **C:** As the plaster is setting, the splint is molded to the arm. An elastic bandage holds the splint in place. **D:** Stockinette is applied and attached to the wrist to form a collar-and-cuff sling.

### Hanging Arm Cast

The hanging arm cast, described by Caldwell as a technique already in use (305), consists of a long arm cast with a sling around the neck tied to the cast along the forearm. The weight of the cast and arm provides longitudinal traction. The position of the sling is modified to correct anterior or posterior angulation and varus or valgus. Rotation is difficult to control. Stewart and Hundley suggested not using it in children under age 12 because children cannot keep their arms in a dependent position during sleep and often keep the arm supported rather than hanging while awake (495). However, excellent results are reported in patients under age 10 (516). This is probably due to the marked remodeling and potential for good results regardless of treatment in children. Possible complications of the hanging cast include inferior shoulder subluxation (516), decreased external rotation (320), and shoulder stiffness (279), but these are rarely significant in children.

### Thoracobrachial Immobilization

Severely unstable fractures uncontrollable in a hanging cast or U plaster may necessitate extending the cast to the chest as a thoracobrachial cast or splint (293,294,384,386,457). Various types of thoracobrachial dressings are often described as a Velpeau, but technically this is incorrect: Velpeau described a thoracobrachial bandage with acute elbow flexion. If a thoracobrachial cast or splint is used for a grossly unstable fracture, usually only a few degrees of abduction is necessary (384). Distal diaphyseal fractures rarely require extension to the chest.

### Functional Bracing

Functional bracing, as described by Sarmiento (473), has been quite effective in adults (280,340,354,371,408,432,437,445). It may be difficult to use in children because size differences require a customized brace for each patient or a large supply of braces; however, modern thermoplastics can keep this economical (Fig. 17-32) (286). A prefabricated brace is placed on the initial visit if possible or on subsequent visits after placement of a U plaster or sling and swathe at the initial evaluation (520). The patient must be followed closely and the splint tightened as needed. It should not be used in bedridden patients because of loss of gravity support (280). Sarmiento (270,473) noted difficulty in controlling anterior angulation and indicated that patients should not lean on the elbow. The results in adults may be functionally superior to those of the U plaster (481).



**FIGURE 17-32.** Light plastic functional braces are useful to maintain alignment and allow early restoration of motion, particularly in older children and adolescents.

### Traction

Side-arm and overhead skin and skeletal forms of traction have been described (276,384,457,509). If olecranon skeletal traction is used, the AO method of an eye screw in the olecranon is less likely to produce ulnar nerve irritation than is a transolecranon pin (435,457). Excessive traction can lead to nonunion in adults (382) and elbow dislocation in children (376).

### Operative Treatment

There are several surgical alternatives for humeral diaphyseal fractures: pinning, external fixation, intramedullary rodding, screw fixation, and compression plating. Biomechanically, interlocking rods are the stiffest in bending, and dynamic compression plating is stiffest in torsion. Flexible intramedullary rods are not as stiff as intact bone (379).

### Open Reduction and Internal Reduction

Open reduction and internal reduction can be performed through either a posterior triceps-splitting approach [as advocated by the AO group (435,468)] or through an anterior lateral approach between the brachialis and brachioradialis with extension proximally between the deltoid and pectoralis (287,348). The normal 4.5-mm dynamic compression plate does not provide adequate stability for the adult humerus shaft. The broad 4.5-mm dynamic compression plate is designed to allow compact screw placement without causing excessive stress on the humerus (435). At least six cortices of screw fixation proximal and distal to the fracture site are needed. With either the anterolateral or posterior approach, the lateral intermuscular septum should be split in the distal third to release the radial nerve's tether. Interfragmentary lag screws should be used when possible. The plate should be slipped underneath the radial nerve and vessels and some muscle placed between the plate and the nerve. Multiple screws in oblique fractures without a compression plate are unsatisfactory in adults (468) but may be sufficient in children. Extensively comminuted fractures should be grafted with autologous cancellous bone.

Generally, the results are good (287,377,378,436,517,523), and plating is particularly advocated in multiple-trauma patients to facilitate nursing care and management of other injuries (287). Potential complications include radial nerve palsy, infection, delayed union, nonunion, and failure of fixation (523).

### Intramedullary Rodding

Several types of intramedullary rods are available. Currently, there are no indications for reamed intramedullary nailing in children because of potential proximal

physeal damage and the small diaphyseal diameter. However, they may be used in older adolescents if the risk of physeal arrest is minimal and the canal has sufficient diameter (461). Reamed nailing has been reported in patients as young as 16 (326,328). The results are generally good (326,338,372,391,399,426,456,466,505,507,521), with a low risk of nonunion and infection (278).

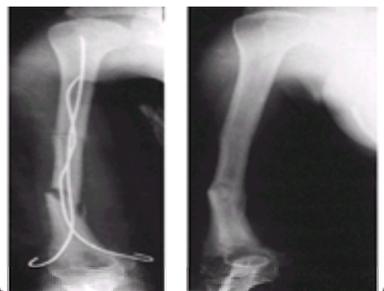
Unreamed nails, such as Ender nails, Rush rods, or flexible titanium rods have been used primarily in adults. Nails or rods can be inserted via a posterior triceps-splitting approach through a hole just above the olecranon fossa. This can be useful for rapid management of fractures, including open fractures in patients with multiple trauma (303,315,323,335,373). Rods should not be inserted through the greater tuberosity in children (except under extenuating circumstances) because of the proximal humeral physis and the potential for shoulder impingement (421,467,494). Inserting these relatively large rods through the epicondyles results in a high incidence of nail back-out (373).

There are two techniques of using small, flexible, smooth wires. In the Hackethal technique (370), fluoroscopy is used with a tourniquet placed on the upper arm. A hole is made just proximal to the olecranon fossa using a triceps-splitting approach. Smooth, blunt-tipped Steinmann pins are placed up the canal of the humerus, progressively filling the canal with smaller and smaller pins as needed (Fig. 17-33). Results are generally good (313,336,341,380,430,446,522), although pin back-out can be a problem and care must be taken not to distract the fracture site. The rods should be bent 90 degrees at the cortical window (446). This technique may be useful for segmental and pathologic fractures (414).



**FIGURE 17-33.** The Hackethal technique involves multiple smooth pins placed up the humeral shaft through a cortical window just above the olecranon fossa. The pins are placed until the canal is filled.

The other technique consists of using small smooth rods or Steinmann pins placed through the epicondyles (413,431). The tips of the rods should be blunt and slightly bent. These are placed through the lateral epicondyle or through both the medial and lateral epicondyles. A hole is made in the epicondyle, and a blunt-tipped Steinmann pin is tapped up the diaphysis using a mallet or passed by hand, with a drill chuck holding the pin. The bend on the tip of the rods facilitates crossing the fracture site and manipulating the fracture reduction. A splint is necessary postoperatively. Alignment need be only within the tolerances for a closed reduction (Fig. 17-34).



**FIGURE 17-34. A:** A segmental fracture difficult to align by nonoperative methods treated with two intramedullary smooth pins. **B:** Alignment need be only within the same tolerances as closed reduction.

### External Fixation

Both unilateral and multiplanar external fixation are occasionally useful for humeral shaft fractures (276,333,394,396). External fixators are primarily useful for severe open fractures or as an alternative to internal fixation. In patients with open fractures, immediate external fixation with subsequent bone grafting yields good results (455,486). External fixation can be combined with internal fixation for immediate stability (325) for early rehabilitation. Severe open fractures with bone loss can be treated with primary shortening followed by callus distraction (469) to provide early soft tissue coverage and subsequent restoration of humeral length. Care must be taken during pin placement to avoid radial nerve injury. If screws are used, limited open screw placement can prevent this injury (455). Ring fixators may be useful for reconstructing the injured humerus (304,310,311,321,389,390,484,485).

### Operative Versus Conservative Treatment

Because most humeral fractures are controllable nonoperatively, there are few surgical indications (288). Potential operative indications include open fractures, multiple trauma, bilateral injuries, arterial injuries, compartment syndromes, pathologic fractures, significant nerve injuries, inadequate closed reduction, and ipsilateral upper extremity injuries or paralysis.

Preadolescents can almost always be managed nonoperatively, except with severe soft tissue injury. If fracture reduction cannot obtain less than 30 degrees varus and 20 degrees anterior angulation in older children and adolescents—or more importantly, if the arm appears deformed—alternatives such as internal fixation, intramedullary rodding, external fixation, a thoracobrachial cast, or traction should be considered. Inadequate closed reduction is most common in obese patients and in thin women with large breasts (468). However, obesity tends to hide the deformity of the fracture, and large breasts are seldom encountered in children.

Open fractures may require fixation. Small, stable grade 1 wounds can still be managed using coaptation splints or other closed methods. Unstable open fractures should be stabilized with internal or external fixation to protect soft tissues (318,335,409,420,468,510).

Multiple-trauma victims are often best treated with internal or external fixation for more rapid mobilization (285,287,303,348,416,425). This is particularly true in patients with chest injuries, where thoracobrachial immobilization would compromise pulmonary care (335,416,420). Excellent results have been reported with external fixation (318,394,455,486), retrograde rodding using Ender nails or Rush rods (303), and internal fixation (287). In older adolescents, more rigid locked or unlocked intramedullary rodding can be used for patients requiring their upper extremities for mobility (348). However, this luxury does not exist for younger children.

Arterial injury and compartment syndromes requiring fasciotomy are potential indications for internal fixation (353,451,460,487). Continued fracture mobility can damage a vascular anastomosis (355,428,460,487), and fasciotomy can make the fracture less stable. Temporary vascular shunting before internal fixation allows the orthopaedist and the vascular surgeon to work under optimal conditions (317).

Most pathologic fractures in children, including those from malignancy (463), fibrous dysplasia (360,492), osteogenesis imperfecta, and simple bone cysts, can be

treated nonoperatively. Simple bone cysts are discussed later in the section on proximal humerus fractures. Occasionally, patients with osteogenesis imperfecta require intramedullary rodding using Bailey-Dubow rods. A report of a 6-year-old with progressive ossifying fibrodysplasia suggests that internal fixation may prevent stiffness after fractures in this condition ( [438](#)). In fractures secondary to malignancy, intramedullary rodding is necessary if extensive cortical loss causes instability ( [322,326,411,421,504](#)). Spontaneous fracture in a severely brain-injured or unresponsive cerebral palsy patient is best treated nonoperatively ( [508](#)).

Ipsilateral injuries, particularly fractures of the proximal or distal humerus and of the forearm, can be difficult to control. In adults with a floating elbow, internal fixation of the humeral fracture provides optimal results ( [303,405,464](#)). This is also true for supracondylar humeral fractures in children but is not documented in diaphyseal fractures ( [491](#)). The floating elbow is often associated with other organ system injuries; nerve injury occurs in up to 50% of these patients ( [448](#)).

Humeral shaft fractures with ipsilateral brachial plexus palsies in adults heal best with open reduction and internal fixation ( [300](#)). The same is true with spinal cord injuries ( [358](#)). Functional bracing is precluded in these patients because the muscles do not function. Because of the excellent healing potential in children, they may be treated nonoperatively if satisfactory alignment can be maintained. Older adolescents should be treated like adults.

### **Radial Nerve Palsies**

Radial nerve palsies with humeral shaft fractures have been reported in children ( [Fig. 17-35](#)) ( [314,420](#)). Primary radial nerve palsies occur at the time of the fracture; secondary radial nerve palsies occur after manipulation of the fracture. Many clinicians recommend exploration of primary ( [270,329,356,385,401,403,435,443,450,465,501](#)) and secondary radial nerve palsies ( [285,332,356,443,450,483,506,513](#)). The incidence of concomitant radial nerve palsy with a humeral shaft fracture ranges from 2.4% to 20.6% ( [291,336,403,420,427,449,465,479,511](#)) and has been reported in 4.4% of children's humeral shaft fractures ( [420](#)). Most occur with middle and distal humeral shaft fractures, but they may occur with more proximal fractures as well ( [427](#)). In explored primary radial nerve palsies, the incidence of complete nerve laceration is small ( [420,443,479,488,511](#)). Commonly, the nerve is tented over the bone, trapped in the fracture site, or contused. The natural history is excellent, with recovery ranging from 78% to 100% ( [271,291,296,297,338,356,361,443,449,450,470,479,511](#)). Therefore, many clinicians recommend observation rather than early exploration ( [291, 338,356,361,443,449,450,470,479,511](#)). Open fractures resulting in severe soft tissue injury requiring debridement should have the radial nerve explored and tagged ( [499](#)) or preferably repaired ( [349](#)). More severe open fractures should be stabilized using either intramedullary rodding or internal or external fixation to provide good soft tissue for radial nerve recovery. Early repair of the nerve provides the best anatomic results ( [290](#)). Bostman et al. ( [297](#)) recommended exploration and internal fixation in patients with bayonet apposition because the abundant callus may endanger nerve recovery ( [297](#)). The recommended waiting time before radial nerve exploration ranges from 8 weeks to 6 months ( [270,271,332,338,386,427,449,450,457,483,513](#)). Nerve grafting up to 18 months after the injury can provide good function ( [290,347](#)). Seddon suggested a physiologic time of allowing 1 mm per day after the 1 to 2 months of Wallerian degeneration and nerve growth through the neuroma ( [475](#)). Nerves grow 1 to 3 mm per day ( [475,476,498](#)), and this rate has been used clinically with good success ( [362,499](#)).



**FIGURE 17-35.** Radial nerve palsy secondary to a humeral shaft fracture from a low-velocity gunshot wound.

In secondary radial nerve palsies, the surgeon may feel compelled to explore the nerve because he or she “caused” the radial nerve injury. However, natural history studies of observed secondary radial nerve palsies show recovery of 80% to 100% with nonoperative treatment ( [297,356](#)). Secondary palsies occurring after manipulation may be observed ( [291,338,402,445,479](#)). If the palsy occurs after a considerable time, the nerve is probably encased in callus and further investigation, including exploration, is warranted ( [342,496](#)). Late presentation may result in an osseous foramen containing the nerve and requiring decompression ( [342](#)).

### **AUTHORS' PREFERRED METHOD OF TREATMENT**

Birth fractures have a very good prognosis for full recovery. To prevent an internal rotation contracture, I place the arm in either a U plaster or a plaster coaptation splint with the palm facing anteriorly. A soft wrap holds the arm to the body so the child can be carried. The splint can be removed in 2 weeks. On healing, the radiographic angulation can be quite worrisome to the parents. I like to show them radiographs of other infants with marked remodeling, and keep photographs handy for this purpose.

Most humeral diaphyseal fractures in children are treated nonoperatively. Torus fractures are treated with a commercial shoulder immobilizer or a sling. Greenstick fractures and displaced fractures in younger children are treated with a U plaster or a plaster coaptation splint; these are usually applied in the emergency department with mild sedation. I prefer general anesthesia if more manipulation is needed. A careful neurologic and vascular evaluation is performed before and after manipulation. I place a U plaster with Webril padding extending from over the deltoid, around the olecranon, and up to the axillary fold, and secure it with a gauze wrap followed by an elastic bandage. I have had similar results applying plaster coaptation splints on the medial and lateral aspects of the arm and rewrapping frequently with an elastic wrap. The patient is placed in a collar-and-cuff sling for forearm support. If alignment is unsatisfactory, a new splint is reapplied and molded.

In those rare fractures uncontrollable by closed means, I prefer smooth intramedullary rodding using two 2-mm rods placed retrograde through the epicondyles. For unstable fractures with extensive comminution, I prefer to use a unilateral external fixator, with small incisions made during screw placement to avoid the radial nerve. Open fractures are treated in a similar manner. Significant bone loss can be treated using bone transport techniques. I avoid plate fixation because it creates a stress riser, particularly in growing children. If a fracture occurs distal or proximal to the plate, it must be reexplored for plate removal, necessitating reexploration of the radial nerve and potential nerve damage. I observe both primary and early secondary radial nerve palsies, exploring them only after 1 to 2 months and growth of 1 mm per day if electromyography shows no return.

### **Rehabilitation**

Patients treated with closed manipulation should be followed weekly for the first few weeks to ensure that alignment is maintained. The coaptation splint or long arm cast should be replaced as needed. Patients with radial nerve palsies must be instructed in finger motion to keep the fingers supple and prevent contractures. Noncompliance requires formal hand therapy or a radial nerve outrigger. Stiffness of the shoulder and elbow is uncommon in children, but pendulum exercises are started at 3 to 4 weeks in older children and adolescents. Some form of immobilization is generally continued for 6 weeks. Patients should not return to contact sports until there is adequate healing, and the family should be cautioned that refracture may occur during the first 6 months after injury.

### **Prognosis**

The prognosis for healing and remodeling of humeral shaft fractures in children is excellent. Internal rotation deformity is usually minimal, and the outlook for radial nerve palsies is good. Loss of shoulder motion may occur but is more common in older patients ( [386](#)).

### **Complications**

### **Malunion**

Malunion is uncommon in children's humeral diaphyseal fractures. Varus of 20 to 30 degrees can be accepted ( [Fig. 17-29](#)) ([299,332,343,457](#)), but anterior bowing of 20 degrees may be apparent ([397](#)). An internal rotation deformity of 15 degrees causes no functional impairment ([332](#)). Most patients under 6 years of age grow out of angular deformities ([386](#)). Children 6 to 13 years of age may not, although some remodeling is possible even in adolescents ([386,440](#)). Obese patients are more prone to malunion, but they also hide their deformity better ([468](#)). Green and Gibbs ([365](#)) noted that the deformity visible on the anteroposterior and lateral radiographs is generally not the maximum deformity, which is the vector sum of the two deformities. This can be appreciated by obtaining a radiograph perpendicular to the plane of the deformity, similar to the Stagnara view for scoliosis.

### **Nonunion**

Primarily a problem in adults and occasionally in older adolescents, there are few reports of humeral nonunion in children—one in a child with progeria at age 4 ([351](#)), four in children with osteogenesis imperfecta ([355](#)), and three from severe trauma ([410](#)). In adults, numerous treatments have been used successfully. These include reamed nails ([319](#)) and modified flexible nails ([368,452](#)). However, the best results appear to be from ASIF techniques with the broad dynamic compression plate and autogenous bone grafting ([282,312,346,374,435,519](#)). Currently, treatment in children and adolescents must be extrapolated from adult treatment. In general, the atrophic ends of the nonunion are taken back to bleeding surfaces and apposed, a compression plate is applied with fixation of at least six cortical screws proximally and distally, and bone grafting is performed ([374](#)). The Ilizarov technique also reportedly produces good results ([275,304,310,389,390,484](#)). Electrical stimulation also has been used with success ([324,344,345,477,478,497](#)). Children with dysplastic bone, such as those with osteogenesis imperfecta, are best treated with intramedullary rodding and bone grafting ([355](#)).

### **Nerve Palsies**

Radial nerve palsies were discussed previously. They also may occur immediately after operative treatment ([334](#)), or may be delayed and occur many years after internal fixation ([352](#)). Ulnar nerve paralysis has been reported from entrapment of the nerve in the fracture site ([395](#)). A small percentage of people have an abnormal arcade of Struthers in which only superficial fibers of the triceps medial head pass superficial to the ulnar nerve and none pass deep to the nerve, making the nerve extremely close to the bone and vulnerable to an abduction extension mechanism of fracture, which opens the anterior medial aspect of the humerus ([395](#)). In about 10% of the population, the median nerve crosses posterior to the brachial artery rather than anterior, placing it closer to the humerus. Median nerve palsy has been reported from an apex anterior mid-diaphyseal fracture ([422](#)). After an easy fracture reduction, the median nerve was caught in the fracture between the coracobrachialis and brachialis muscles, where the nerve crossed anteriorly. Anterior interosseous nerve palsies have not been reported in fractures above the supracondylar region.

### **Compartment Syndrome**

The fascia of the upper arm is not as strong as it is in the lower arm, making compartment syndrome less common. Mubarak and Carroll ([434](#)) reported a dorsal forearm compartment syndrome in a 9-year-old boy with a humerus shaft fracture. Gupta and Sharma ([367](#)) reported on an adult with a triceps compartment syndrome from a middle third minimally displaced fracture; this fracture did not disrupt the intercompartmental boundaries.

### **Vascular Injuries**

Vascular injuries require a high index of suspicion and rapid treatment ([301,334,424,429](#)). The fracture should be stabilized sufficiently to prevent disruption of the vascular repair.

### **Infection**

Infections have been reported in patients undergoing surgery. They have not been reported in closed fractures of the humerus in children, but have been reported in closed fractures elsewhere ([307,512](#)).

### **Loss of Motion**

Loss of shoulder and elbow motion is more common in older patients ([272,386](#)). The joint affected is usually the one closest to the fracture site.

### **Limb-Length Discrepancy**

Overgrowth after humeral fracture occurs in about 81% of patients but is generally minimal (<1 cm) ([375](#)). Some generalized stimulus to the extremity is evident, with overgrowth of the carpals as well ([474](#)). In patients with limb-length discrepancy of 3 cm or more at maturity, lengthening may be indicated ([330,447](#)). Unilateral or ring fixators may be used with Ilizarov's principles ([310,311](#)).

### **Other Complications**

Uncommon complications include reflex sympathetic dystrophy ([350](#)) and fat embolism ([400](#)). Late refracture may occur from retained internal fixation ([298](#)).

## **DISTAL HUMERAL DIAPHYSEAL FRACTURES**

Little has been written about distal humeral diaphyseal or metaphyseal–diaphyseal junction fractures, which are much less common than supracondylar humeral fractures. Fractures in this region should not be confused with supracondylar humeral fractures. The distal diaphysis is more triangular and the periosteum is thinner than in the supracondylar region ([327,331](#)), making these fractures generally less stable than supracondylar fractures. The cortical bone also heals more slowly than metaphyseal bone, requiring longer immobilization. The mobile wad, anconeus, and flexor pronator mass originate off the epicondyles; the biceps, brachialis, and triceps all insert distally. Therefore, forearm position greatly affects the fracture position. Because the brachial artery is tethered by the lacertus fibrosus, injury to the artery is more likely than with more proximal fractures.

### **Etiology**

Distal humeral diaphyseal–metaphyseal junction fractures may be caused by transverse or longitudinal loading, torsion, or moments generated by the forearm about the elbow. They are more often caused by direct blows and twisting rather than ulnar leverage in the olecranon fossa. The diagnosis, made on plain radiographs, must be differentiated from a supracondylar humerus fracture.

### **Classification**

Most distal humeral diaphyseal fractures are transverse, spiral, or short oblique. Occasionally, an oblique or spiral fracture extends distally toward or beyond the epicondyles ([Fig. 17-36](#)). The description must include the direction of displacement, the neurologic and vascular status, and the degree of comminution. Medial column comminution predisposes to varus malunion.



**FIGURE 17-36. A and B:** Distal humeral diaphyseal fracture extending to the epicondyles. This fracture was treated by casting with the forearm in pronation.

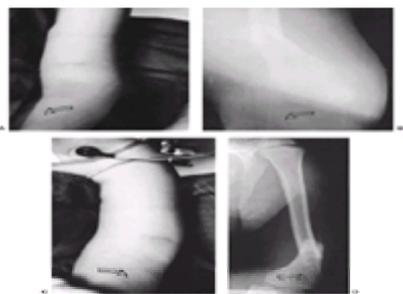
## Treatment

### Nonoperative

Closed treatment usually is possible because acute flexion of the elbow, with potential vascular compromise, is not required to maintain reduction. These fractures tend toward varus malunion ([Fig. 17-37](#)) ([302](#)), which may be cosmetically unacceptable, particularly in more distal fractures. With 20% or less of humeral growth occurring distally ([295,453,454](#)), significant remodeling may not occur. Because of the proximity to the epicondyles with their muscular origins, supination and pronation affect fracture reduction. If one cortex is open, then the muscles originating on that side should be tightened to reduce the fracture ([274](#)). Because of the varus tendency, this is usually by pronation ([292,294,487](#)). However, this is best checked radiographically ([Fig. 17-38](#) and [Fig. 17-39](#)).



**FIGURE 17-37. A and B:** Radiographs showing the tendency of distal humeral diaphyseal fractures toward varus malunion. This fracture required remanipulation.



**FIGURE 17-38.** Influence of forearm rotation. Pronation (**A and B**) of the forearm produces a valgus angulation at the fracture site (*arrows*). Supination (**C and D**) creates a varus angulation (*arrows*).



**FIGURE 17-39.** The same patient shown in [Fig. 17-38](#). **A:** The humeral coaptation splint is molded (*arrows*) with the forearm in neutral. **B:** A second forearm coaptation splint is added, and the extremity is suspended with a loop. **C:** Radiographs show satisfactory linear alignment. **D and E:** The fracture healed in bayonet apposition but with satisfactory alignment.

### Operative

Unstable fractures may require fixation ([302,457](#)) and possibly open reduction. Closed reduction and percutaneous pinning should be performed in a similar fashion to supracondylar humerus fractures. However, because the fracture is more proximal, it is difficult to get the pins into the diaphysis without crossing them at the fracture site ([Fig. 17-40](#)). Attempts should be made to pass the wires in intramedullary fashion up the lateral or medial and lateral columns separately to provide stability ([Fig. 17-41](#)) ([457](#)). This can be done by drilling the wires, but it is easier to create a starting site at the epicondyles and pass blunt-tipped wires up the columns. Holding the wires with a drill chuck helps, too. Because of the bony anatomy and the ulnar nerve, lateral wires are easier to place, particularly in younger children ([Fig. 17-42](#)). Alternatively, the fracture can be managed with skeletal traction until callus forms; then either a U plaster splint or a long-arm cast can be applied ([Fig. 17-43](#)). Brug and colleagues report the best results with flexible intramedullary rodding ([302](#)).



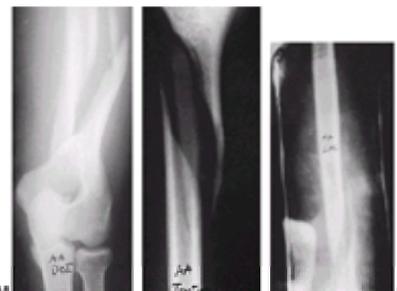
**FIGURE 17-40. A and B:** Distal humeral diaphyseal fracture in an 18-month-old child treated with closed reduction and percutaneous pinning **(C)**. The pins cross at the fracture site with decreased stability and some loss of position **D and E:** The ultimate outcome was good.



**FIGURE 17-41.** Ideally, pin fixation for distal humeral diaphyseal–metaphyseal junction fractures involves pins placed in intramedullary fashion up the medial and lateral columns.



**FIGURE 17-42.** Segmental distal humeral diaphyseal and supracondylar fracture in a 4-year-old boy. **A and B:** Both fractures could not be controlled by closed means.



**FIGURE 17-43.** A comminuted distal humeral metaphyseal–diaphyseal fracture in a 14-year-old boy. Injury films **(A)** show multiple fragments in the metaphyseal–diaphyseal area. **(B)** The patient was placed in traction for 2 weeks until callus appeared and then was transferred to a long arm cast **(C)**.

## AUTHORS' PREFERRED METHOD OF TREATMENT

For distal humeral diaphyseal–metaphyseal junction fractures, I prefer closed treatment. Nondisplaced fractures are treated with a long arm cast split to allow for swelling. A double sugartong splint is used if swelling is severe. I reduce displaced fractures under general anesthesia. Because supination and pronation of the forearm can affect the position, I use the image intensifier to determine the position best for maintaining the reduction; this is usually pronation. If the reduction obtained is unstable and cannot be held with a cast, I do not hesitate to treat it by percutaneous pinning, with small Steinmann pins placed through the medial and lateral epicondyles and up their respective columns, keeping the pins as divergent as possible at the fracture site or with lateral column pins. It is helpful to introduce them through the epicondyle and then tap rather than drill them up the column to prevent convergence at the fracture site. The pins are removed once good callus forms.

## SUPRACONDYLAR PROCESS FRACTURES

### Anatomy

Occasionally, a proboscis-like supracondylar process extends from a few centimeters above the medial epicondyle. The incidence of this process ranges from 0.1% to 2.7%, with the lower percentages in blacks and the higher percentages in whites ([281,419,439](#)). The process extends obliquely downward and may be connected with the medial epicondyle by a tough fibrous band ([281,309,394,419,439](#)). Frequently, the foramen formed between the fibrous band and the humerus is traversed by the median nerve and the brachial artery. They may be entrapped by a fracture. Anomalous attachments of the coracobrachialis and the pronator teres may occur on the process ([Fig. 17-44](#)) ([369,399](#)).



**FIGURE 17-44.** Radiographic appearance of a supracondylar process ( *arrow*).

### Etiology

Supracondylar process fractures are the result of direct blows. There are no reports of avulsion from the anomalous muscle attachments.

### Classification

Supracondylar process fractures are classified as displaced or nondisplaced, with notation of median nerve or brachial artery compromise.

### Treatment

Supracondylar process fractures have been reported in children (399) and usually are caused by direct blows to the distal humeral area. They may be quite painful and result in compression of the brachial artery or median nerve (281,309,369,399,419). The process is best seen on oblique views (439). If there are no symptoms of median nerve or brachial artery compression, they are treated by elevation, ice, and temporary immobilization for comfort. However, if a painful nonunion or neurovascular symptoms develop, the fragment should be excised (439). Fractures with neurologic signs or symptoms are treated by fragment excision and nerve and artery decompression.

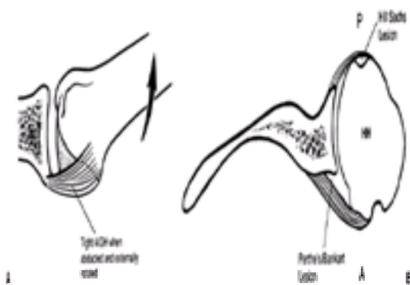
## GLENOHUMERAL SUBLUXATION AND DISLOCATION

Dislocation of the glenohumeral joint in children is rare. None of the ancient writings of Hippocrates (460–375 B.C.), Galen (A.D. 131–201), and Paul of Aegina (A.D. 625–690) made specific mention of this injury in children (601). Most textbooks that address children's shoulder problems do not even discuss dislocations of the glenohumeral joint, and others barely touch on the subject (533,599,600,607,610,615). A review of the literature would suggest that glenohumeral dislocations in children less than 12 are rare. Although several case reports have been presented, no large series of this entity are available (564,566,579,614). Rowe, in his 1956 review of 500 dislocated shoulders (604), found that only 8 patients were under 10 years of age. In this same series, 99 patients were 10 to 20 years of age, but no details on skeletal maturity were given (603,604). Rockwood reported a series of 44 patients with shoulder dislocations, predominantly adolescents (601). Many articles have been published on adolescent patients without discussing their skeletal maturity (548,557,586,591). As the child reaches adolescence, the incidence of shoulder instability increases, but in the skeletally immature patient, this injury can still be considered rare. Marans et al. (585) in 1992 presented a series of 21 patients with open physes from two major trauma centers.

### Anatomy

Developmental anatomy was discussed in the section on fractures of the proximal humerus. The glenohumeral joint consists of the articulation between the large convex humeral head and the relatively flat glenoid fossa. This joint is anatomically suited to accommodate the wide range of motion necessary to perform upper extremity function. To accomplish this range of motion, very little bony constraint is inherent in this joint. The articular surface area and radius of curvature of the humeral head are about three times the size of the relatively flat glenoid surface. Although the glenoid fossa is deepened by the labrum, the mismatch in the surface area and the radius of curvature explains the lack of joint stability.

The primary constraint for the joint is the capsular/ligamentous complex. The capsule on its inner surface is reinforced by thickened areas known as the anterior glenohumeral ligaments. This complex capsular/ligamentous structure must provide stability against abnormal translation while allowing a wide range of motion. With the arm abducted, the inferior capsule is highly redundant. The most important ligament is the anteroinferior glenohumeral ligament, located within the inferior redundant area. It is mechanically designed to tighten as the arm is abducted and externally rotated, much like the effect of wringing out a washcloth. This structure becomes the primary site of pathology in anterior shoulder instability, either when the anteroinferior glenohumeral ligament attachment to the glenoid and labrum is stripped from the anterior neck of the glenoid or as these ligaments are disrupted in substance (Fig. 17-45). Disruption of the capsular/labral attachment is known as a Perthes' or Bankart's lesion.



**FIGURE 17-45.** **A:** The tight anteroinferior glenohumeral ligament complex with the arm abducted and externally rotated. This ligament sling is the primary restraint against anterior instability of the shoulder. **B:** A cross-section in the transverse plane through the glenohumeral joint demonstrates the common lesions associated with anterior instability of the shoulder: Hill-Sachs lesion, Perthes-Bankart lesion, and redundant anteroinferior glenohumeral ligaments. *A*,anterior; *P*,posterior; *HH*, humeral head.

The humeral attachment of the capsule of the glenohumeral joint is along the anatomic neck of the humerus except medially, where the attachment is more distal along the shaft. The physis, therefore, lies in an extracapsular position except on the medial side. As in most pediatric joint injuries, the strong capsular attachment to the epiphysis makes failure through the physis a much more common injury than true capsular/ligamentous injury (542,574,611). Therefore, fracture through the physis is more common than a dislocation in the skeletally immature patient.

The rotator cuff tendons consist of the subscapularis, supraspinatus, infraspinatus, and teres minor muscles. These muscle–tendon units surround the joint anteriorly, superiorly, and posteriorly. They serve an important function as dynamic secondary stabilizers of the joint by forming a force-couple with the large shoulder muscles (deltoid, pectoralis major, and latissimus dorsi). As the glenohumeral joint moves through its range of motion, the cuff provides a dynamic stabilizing effect, preventing excessive translation of the humeral head on the glenoid. This is important when addressing rehabilitation for the prevention of recurrent glenohumeral dislocation.

## Mechanism of Injury

### Traumatic Dislocations

Anterior dislocations of the shoulder are most common in this category. Significant evidence of trauma should be present to assign patients to this grouping, as compared with the relatively minor trauma in the atraumatic group. The mechanism of injury is similar to that observed in the adult. A force applied to the outstretched hand that forces the arm and shoulder into an abducted, externally rotated position is the primary mechanism. The humeral head is levered out of the glenoid process anteriorly, with the head lodging against the anterior neck of the glenoid. This occurs commonly in contact sports, falls, fights, and motor vehicle accidents ([531,567,592](#)).

Traumatic posterior dislocations are much less common. In most series of posterior dislocations in all age groups, they represent only 2% to 4% of all dislocations. The history is one of violent trauma with the arm in a position of flexion, internal rotation, and adduction. This can occur in falls and in motor vehicle accidents as the arm braces the body against impact. The other common mechanisms that produce posterior dislocations include convulsions and electroshock. In these cases, the shoulder is dislocated posteriorly by the violent contraction of the shoulder internal rotators, which in most cases are stronger than the shoulder external rotators. The history of the injury and a high index of suspicion are necessary to avoid missing a posterior dislocation ([546,547,563,596,612](#)).

In neonates, pseudodislocation of the shoulder can occur ([561](#)). This problem represents traumatic epiphyseal separation of the proximal humerus, which is certainly much more common than a true traumatic dislocation of the shoulder in this age group. Most true traumatic dislocations of the shoulder in the neonatal period occur in babies with underlying birth trauma to the brachial plexus or central nervous system.

Laskin and Sedlin ([577](#)) reported on a 3-month-old infant with Erb-Duchenne palsy who sustained a traumatic luxatio erecta of the shoulder during a planned shoulder manipulation. Posterior dislocation of the shoulder also can occur as a secondary traumatic phenomenon in unrecognized brachial plexus injury of the upper trunk at delivery ([527,580,617,618](#)). Green and Wheelhouse ([558](#)) reported a dislocation in a 7.5-month-old infant that was secondary to a septic brain injury.

### Atraumatic Dislocations

Atraumatic shoulder instability is more common in children and adolescents than is readily recognized. The child who presents with shoulder dislocation without a clear-cut significant history of trauma should arouse suspicion that atraumatic instability may be present. These patients have inherent joint laxity that allows the shoulder to be dislocated either voluntarily or involuntarily as the result of a minimally traumatic event ([Fig. 17-46](#)) ([536](#)). Throwing, hitting an overhead tennis shot, or pushing the body up when in bed does not constitute significant trauma. A high index of suspicion should be maintained with this kind of history. In the voluntary dislocator, conscious selective firing of muscles while antagonists are inhibited, combined with arm positioning, allows the shoulder to dislocate. A key to the diagnosis is that atraumatic instability, whether voluntary or involuntary, is not associated with much pain. Even if reduction is necessary, the pain usually disappears rapidly. In most instances, spontaneous reduction occurs without manipulation ([601](#)).



**FIGURE 17-46.** Congenital laxity of the left shoulder in a 4-year-old boy who is totally asymptomatic and has a full range of motion of the left shoulder. **A:** With abduction and extension, the head subluxates anteriorly and inferiorly. **B:** An anteroposterior radiograph shows some lateral displacement of the humeral head. **C:** With overhead elevation, the humeral head is noted to displace anteriorly, laterally, and inferiorly. (Courtesy of Don Jones, M.D.)

Other causes of atraumatic shoulder instability, in addition to multidirectional joint laxity, include Ehlers-Danlos syndrome, congenital absence of the glenoid, deformities of the proximal end of the humerus, and emotional and psychiatric instability. True congenital dislocations of the shoulder are most commonly associated with developmental defects and multiple congenital abnormalities ([537,540,555,559,598](#)). Arthrogyrosis, neglected septic arthritis, and neurologic defects also have been implicated in atraumatic dislocations in the young child ([527,557,558,565,609,617](#)).

## Classification

### Etiology of Instability

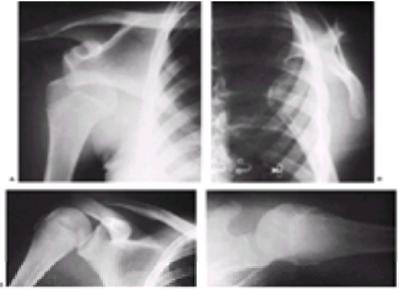
1. Traumatic dislocations
  - a. Primary trauma to the shoulder itself
  - b. Secondary to birth trauma of the brachial plexus or central nervous system.
2. Atraumatic dislocations—voluntary or involuntary
  - a. Congenital abnormalities or deficiencies of bone or soft tissue
  - b. Hereditary joint laxity problems, such as Ehlers-Danlos syndrome
  - c. Developmental joint laxity problems
  - d. Emotional and psychiatric disturbances

Because of the rarity of this injury, no consensus exists as to a classification scheme in children and adolescents. In adults, shoulder instability can be classified as to direction, degree, and chronicity. Two basic schemes have been used to classify other dislocations in children and adolescents; the more common is based on the direction or location of the dislocation. This scheme is useful in describing the clinical and radiographic features of the injury, but it does not address the underlying pathology in children ([601](#)).

### Direction of Instability

Therefore, a second classification scheme describing the etiology of the dislocation is also useful when considering treatment options for this injury in children. This second system is similar to that used for adults but takes into account congenital and developmental problems unique to children. As discussed later in the section on treatment of this problem, accurate classification is important in selecting the appropriate conservative versus surgical options ([526,542,597](#)).

There are four directions of dislocation: anterior, posterior, multidirectional, and inferior (luxatio erecta). As with shoulder dislocations in adults, anterior dislocation in children is the most common, constituting at least 90% of glenohumeral dislocations ([Fig. 17-47](#)). Several isolated reports of posterior dislocation in children and adolescents have been documented, but posterior dislocation is rare in children, as in adults ([534,553,566,587](#)). Multidirectional luxatio of the shoulder has been well described as a distinct clinical entity by Burkhead and Rockwood ([535](#)), O'Driscoll and Evans ([548](#)), and Rockwood ([601,602](#)). Luxatio erecta or inferior locked dislocations are uncommon but have been reported in children ([554,577,589](#)).



**FIGURE 17-47.** Anterior dislocation of the right shoulder in a 15-year-old girl. **A:** Note the typical subcoracoid position on the anteroposterior film. **B:** On a true scapular lateral film, note the anterior displacement of the humeral head. **C:** Postreduction film demonstrates a Hill-Sachs compression fracture in the posterolateral aspect of the humeral head. **D:** On the postreduction axillary film, note the posterolateral compression fracture of the humeral head.

### **Degree of Instability**

The degree of instability can be classified as a subluxation or a dislocation. A subluxation is an incomplete dislocation characterized by pain, a feeling of slipping, or a dead feeling in the arm. A complete dislocation of the humeral head out of the glenoid fossa is characterized by a displacement and locking of the head on the rim of the glenoid.

### **Chronicity of Instability**

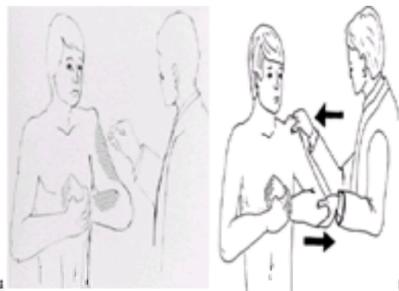
The chronicity of instability can be classified as acute, recurrent, or chronic. A single episode of instability can be described as an acute injury. As in the skeletally mature patient, an acute injury can lead to a recurrent instability, depending on the damage to the ligament and bony restraints of the joint. A chronic instability exists when an acute dislocation is not reduced, and is usually associated with congenital dislocations.

### **Signs and Symptoms**

#### **Traumatic Dislocations**

The patient with a traumatic anterior dislocation presents with a painful, swollen shoulder. Obvious deformity is present, with a prominent acromion and the lateral upper arm flattened. The arm is often supported by the opposite hand and held in an abducted and externally rotated position. Despite swelling, the humeral head can usually be palpated in a position anterior to the glenoid.

Careful examination of the neurologic and vascular status is necessary. The axillary nerve is the most commonly injured with anterior dislocation, and special attention to its function is necessary on examination (532). The sensory distribution of the axillary nerve is along the upper lateral arm; the motor innervation is to the deltoid and teres minor muscles. Light touch is adequate for sensory testing in the upper arm region. An easy way to test deltoid function is to support the involved elbow in one hand while using the opposite hand to grab the muscle belly of the deltoid. The patient is asked to abduct the arm against resistance for about 1 inch so that deltoid firing is initiated. This examination confirms the status of the axillary nerve ( Fig. 17-48).



**FIGURE 17-48.** **A:** Sensory distribution for the axillary nerve important in anterior dislocation. **B:** Deltoid muscle can be tested in acute anterior dislocation by grabbing the muscle belly with the right hand while supporting the elbow with the left. The patient then can actively contract the deltoid by pushing the elbow against the examiner's hand while the examiner feels the muscle contraction.

In recurrent anterior dislocation or subluxation, the arm is well located with an overall normal appearance of the shoulder. The shoulder demonstrates a full range of motion, although the cocking position is avoided. The apprehension test with the arm abducted above 90 degrees is positive. This is a key finding in the diagnosis of recurrent anterior instability.

Traumatic posterior dislocation is, again, much less common than anterior dislocation. The patient presents with flattening of the anterior aspect of the shoulder and posterior fullness. The arm is held at the side, with the forearm internally rotated across the chest. The patient resists any attempt at motion. A hallmark of posterior dislocation is the lack of shoulder external rotation and inability to supinate the forearm. These findings are difficult to elicit in the acute situation. It is advantageous to examine the shoulder with the patient seated so that the examiner can visualize the shoulders from above. This view accentuates the posterior fullness and anterior flattening often present. The neurovascular status should be closely checked. A history of convulsion or electrical shock should raise a high index of suspicion for posterior dislocation.

In neonates, traumatic separation of the upper humeral physis, the so-called pseudodislocation of the shoulder, can exactly mimic an anterior dislocation. The child is irritable and often holds the arm abducted and externally rotated. There is resistance to any type of motion. Deformity in dislocation or pseudodislocation in the neonate is usually absent or subtle.

#### **Atraumatic Dislocations**

The most notable finding in patients with atraumatic shoulder instability is the relative lack of pain associated with the subluxation or dislocation. Even in cases of involuntary atraumatic dislocation, the minor pain associated with the dislocation itself subsides rapidly after reduction. Episodes of atraumatic subluxation and dislocation occur much more frequently than traumatic dislocations, and in almost all cases spontaneous reduction is the rule.

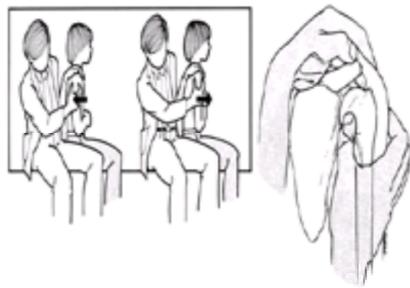
On clinical examination, there is evidence of multiple joint laxity ( 594). Also, multidirectional laxity or instability of the opposite shoulder is usually present (548,549,601,602). Characteristics of multiple joint laxity include hyperextension at the elbows, knees, and metacarpophalangeal joints. Not uncommonly, striae of the skin are present, and skin hyperelasticity is a noted characteristic of Ehlers-Danlos syndrome. Multidirectional laxity of the shoulder is characterized by a positive sulcus sign and significant translation on an anterior and posterior drawer test.

The sulcus sign is a dimpling of the skin below the acromion when manual longitudinal traction is applied to the arm ( Fig. 17-49). This produces an inferior

subluxation of the humeral head away from the acromion that enlarges the subacromial space and causes dimpling of the skin. The drawer or shift and load test is performed with the examiner seated behind the patient. The scapula is stabilized with one hand and forearm while the humeral head is manually translated anteriorly and posteriorly by the examiner's opposite hand (Fig. 17-50). Although some translation within the glenohumeral joint is expected in all patients, those with multidirectional laxity demonstrate translation of greater than 5 mm anteriorly and posteriorly from a neutral position.



**FIGURE 17-49.** Dramatic demonstration of inferior subluxation of the glenohumeral joint in a patient with multidirectional instability. The clinical correlate is the sulcus sign.



**FIGURE 17-50.** Drawer test. This technique is used to subluxate the shoulder manually both anteriorly and posteriorly to demonstrate multidirectional laxity.

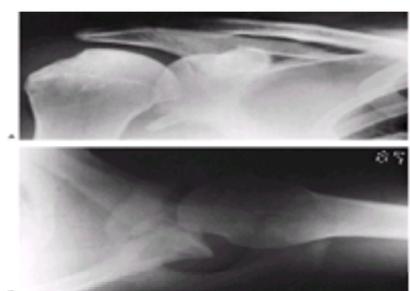
In atraumatic dislocation, the shoulder often dislocates anteriorly, posteriorly, or inferiorly. The most common direction of dislocation in voluntary instability is posterior or inferior. The patient who can voluntarily dislocate the shoulder can force the humeral head posteriorly by contracting the anterior deltoid and internal rotators while inhibiting the antagonistic muscles (Fig. 17-51). The elbow is positioned in horizontal adduction, and the head is dislocated. The arm can then be abducted and, often with an audible clunk, the shoulder reduces. Once again, the hallmark is lack of pain with the dislocation (543,550,573,578,584,605,608).



**FIGURE 17-51.** Voluntary anterior dislocation of the right shoulder in an 8-year-old boy. **A:** The patient voluntarily has dislocated the right shoulder anteriorly. **B:** The shoulder voluntarily reduced. The patient explained that he was taught to do this by an older brother who also had voluntary dislocation of the shoulders.

### Radiographic Findings

Children and adolescents with open growth plates have a low incidence of true traumatic dislocation of the shoulder. Traumatic lesions on plain radiographs are similar to those found in adults (Fig. 17-52). On the anteroposterior or internally rotated views of the proximal humerus, the Hill-Sachs compression lesion on the posterolateral aspect of the humeral head is commonly found. This injury to the proximal humerus occurs as the humeral head is impacted against the anterior rim of the glenoid during a dislocation (Fig. 17-53). Bony injury to the anterior glenoid rim can occur with dislocation as well. A variable degree of injury from small avulsion-type fragments to substantial bony fractures can occur. These anterior glenoid rim injuries are best seen as a double density on the anteroposterior view of the shoulder or as a separate fragment on the axillary and West Point lateral views. The West Point lateral view projects the anteroinferior glenoid rim and most clearly shows this lesion when it is present. In traumatic posterior dislocation, the reverse Hill-Sachs lesion can be seen on the anterior part of the humeral head in conjunction with a possible fracture of the posterior rim of the glenoid.



**FIGURE 17-52.** Traumatic anterior dislocation of the right shoulder in a 15-year-old boy. **A:** On the anteroposterior view, note the Hill-Sachs lesion as well as the anteroinferior bony fragment off the glenoid rim. **B:** Axillary radiograph made with the arm in 90 degrees of abduction demonstrates the anterior subluxation as well as the deficiency of the anterior glenoid rim.

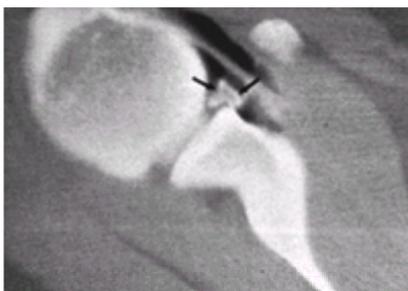


**FIGURE 17-53.** Anterior shoulder dislocation in a skeletally immature adolescent patient. **A:** Anteroposterior radiograph demonstrates the common appearance of an anterior dislocation of the shoulder. **B:** Postreduction anteroposterior radiograph of the shoulder shows a large posterolateral compression fracture of the humeral head or Hill-Sachs lesion.

In cases of traumatic subluxation of the shoulder in which the diagnosis may be unclear clinically, an arthrogram with CT scan or tomograms can sometimes better delineate the extent of capsular stripping from the anterior glenoid rim. More recently, saline arthrograms and magnetic resonance imaging have enhanced our ability to define the degree of injury to the labrum, capsule, and articular surfaces ([560,602](#)). CT scanning and magnetic resonance imaging have been useful for analyzing the significance of fractures of the glenoid rim ([Fig. 17-54](#) and [Fig. 17-55](#)). In addition, the size of the reverse Hill-Sachs lesion of the humeral head in posterior dislocations is best analyzed with a CT scan ([576,616](#)).



**FIGURE 17-54. A:** Anteroposterior radiograph of a 14-year-old boy with recurrent anterior subluxation. Notice the presence of a Hill-Sachs compression fracture on the humeral head and a subtle double density at the anteroinferior glenoid rim. **B:** Computed tomography scan shows this to be an avulsion-type bony injury of the anterior glenoid.



**FIGURE 17-55.** Magnetic resonance imaging scan in a patient with recurrent anterior instability of the shoulder. The arrows demonstrate a calcified bony Bankart's lesion.

With atraumatic dislocations in patients who do not have congenital or developmental defects, radiographs are usually normal. The most common defects seen on radiographs with atraumatic dislocations are congenital aplasia or absence of the glenoid. In patients with multidirectional laxity and atraumatic dislocation, stress radiographs can usually demonstrate instability in anterior, posterior, and inferior directions. The inferior component of multidirectional instability can be demonstrated by applying weights to the arm in an anteroposterior film. This shows the humeral head subluxating in its relation to the glenoid ([571](#)).

## Treatment

### **Traumatic Instability**

The literature on the specific treatment of shoulder instability in children is limited ([526,538,600,610](#)). Most clinicians make treatment recommendations for patients of all age groups ([524,525,530,531,545,572](#)). Therefore, most of the data presented in this section are extrapolated from the adult and adolescent literature, as well as from the experience of Dameron and Rockwood ([542](#)).

### **Acute Dislocation**

Patients with an acute dislocation of the shoulder should undergo closed reduction by one of the standard, accepted techniques. For anterior dislocation, many reduction techniques have been described. Most clinicians prefer light sedation with intravenous or intramuscular injection. The traction/countertraction method appears to be the most gentle. A sheet placed in the axilla of the affected shoulder passes above and below the patient so that countertraction can be applied to the body while longitudinal traction in line with the deformity is applied to the arm. Steady, continuous traction fatigues the muscles that lock the dislocation, and eventually reduction is accomplished by disimpacting the humerus from the glenoid. The Stimson maneuver is equally effective. The patient is placed prone on the examining table, with a weight applied to the affected arm. Both methods are gentle and effective ([591](#)). Success with gentle scapular manipulation has been reported by Kothari and Dronen ([575](#)) and McNamara ([588](#)) as a safe, effective way to reduce the dislocation.

The Steel maneuver in most cases requires no anesthesia (personal communication, J. Steel, 1975). With the patient supine on the examining table, the examiner supports the elbow in one hand while supporting the forearm and wrist in the other. The arm is gently abducted above 90 degrees and slowly externally rotated. With the arm held above 90 degrees of abduction, it is then gently adducted while further external rotation is applied. As reduction occurs, the arm can be taken down into adduction and internal rotation across the chest. Often, reduction is not even felt by the patient or physician. Caution is always used with any type of manipulative reduction, and the procedure is certainly not as safe as gentle traction/countertraction or the Stimson maneuver. Prereduction and postreduction radiographs are required to document fractures and to demonstrate the direction of dislocation. Preoperative and postoperative detailed neurologic examinations are indicated as well.

Postreduction immobilization is another highly debated point. The adult literature suggests that the period of immobilization may not be truly important in predicting recurrent dislocations. A sling or sling and swathe with the arm internally rotated is the most common method of immobilization ( [525,544](#)).

Closed reduction for acute posterior dislocations is somewhat similar to that for anterior dislocations. Traction/countertraction is the most effective. Traction is applied in line with the deformity, and the humeral head is gently lifted back into its normal relationship with the glenoid. Most clinicians agree that immobilization should be with the arm in neutral rotation or slight external rotation at the shoulder. This may require the use of a spica cast or modified shoulder spica cast, as described by Dameron and Rockwood ( [542](#)).

### **Recurrent Dislocation**

The true incidence of recurrent dislocation after traumatic shoulder dislocation in children is difficult to assess because of the rarity of the injury and the variety of reports in the literature ( [556,569](#)). In 1963, Rowe ( [603](#)) reported a 100% incidence of recurrence in children 1 to 10 years of age with anterior dislocation. He also reported a 94% incidence of recurrence in adolescents and young adults (ages 11–20) ( [603](#)). Elbaum et al. ( [551](#)) reported a recurrence rate of 71% in 9 pediatric patients with traumatic anterior dislocations. The average age was 9 years. After reduction, they were immobilized for 3 weeks and then were treated with rehabilitation. However, Rockwood ( [601](#)) reported a recurrence rate of only 50% in a series of adolescents and young adults 13.8 to 15.8 years of age. Hovelius et al. ( [569](#)) reported a 47% recurrence rate in patients less than 20 years of age. In a 10-year follow-up study, Hovelius et al. ( [568](#)) found that recurrent dislocation necessitating operative treatment had developed in 34% of shoulders in patients who were 12 to 22 years of age at the time of initial dislocation, compared with 28% in patients who were 23 to 29 and 9% in patients who were 30 to 40 years old. The type and duration of the initial treatment had no effect on the rate of recurrence. Vermeiren et al. ( [613](#)) reported a recurrence rate of 68% in patients under 20 years of age. They reported a better prognosis if the dislocation was associated with a fracture of the joint. Heck ( [564](#)) reported a case of traumatic anterior dislocation in a 7-year-old boy who remained stable at a 5-year follow-up ( [Fig. 11-56](#)). Endo et al. ( [552](#)) reported no recurrence in 2 patients, ages 3 and 9, with traumatic anterior dislocation. However, the follow-up was only 2 years in the 3-year-old and 1 year in the 9-year-old. Wagner and Lyne ( [614](#)) reported an 80% recurrence rate in 10 patients with clearly open proximal humeral epiphyses. Marans et al. ( [585](#)) reported the fate of traumatic anterior dislocations of the shoulder in 21 children (15 boys, 6 girls) in what may be the largest documented series to date. All the children had one or more documented anterior dislocations after the initial injury. Some of the children had been immobilized in a sling and swathe for 6 weeks. The literature reflects that the natural history of shoulder dislocations in adolescents and young adults demonstrates recurrence rates for dislocation of 50% to 90% despite the treatment program used after the initial dislocation.



**FIGURE 17-56.** Traumatic anterior subluxation of the left shoulder in a 7-year-old boy. **A:** Anteroposterior film of the left shoulder does not reveal any striking abnormality. **B:** An axillary film shows that the humeral head is subluxated away from the glenoid fossa. **C:** Anteroposterior film of the left shoulder after manual reduction. ( *Courtesy of Charles C. Heck.* )

Multiple surgical procedures have been described for the treatment of anterior shoulder instability. Once again, specific results for procedures such as the Putti-Platt, Bankart, and Magnuson-Stack have not been documented for children. Barry and associates ( [530](#)) described the effective use of the coracoid transfer for recurrent anterior instability in adolescents. Capsular procedures that specifically address the capsular pathology have been described by Neer and Foster ( [595](#)), Jobe ( [581](#)), and Rockwood and associates ( [602](#)), but results in children's dislocations were not documented. Goldberg and colleagues ( [557](#)) have reported on the use of arthroscopic techniques for capsular repair in adolescents.

### **Atraumatic Instability**

Treatment of patients with atraumatic dislocations of the shoulder appears more difficult than treatment for true traumatic dislocations. Emphasis should be placed on careful diagnosis in these cases. Specific congenital bony or neurologic deficits should be recognized. The sequelae of Ehlers-Danlos syndrome or other collagen deficiency syndromes should be noted.

In patients with multidirectional laxity and voluntary or involuntary dislocations, a significant history of trauma is usually lacking. These patients have minimal pain associated with the dislocation and on clinical examination usually have other signs of multidirectional laxity of the opposite shoulder. Most of these dislocations reduce spontaneously and are associated with little pain. Rowe et al. ( [605](#)), Neer ( [594](#)), and Burkhead and Rockwood ( [535,601,602](#)) have described the use of a vigorous rehabilitation program involving strengthening of the rotator cuff as the treatment of choice for these patients. Most patients who do not have significant emotional and psychiatric problems are successful in improving their shoulder stability with such a program.

Most clinicians would agree that surgical intervention is considered only if a strict 6- to 12-month rehabilitation program fails. Routine shoulder reconstructions involving subscapularis shortening, including the Magnuson-Stack and Putti-Platt procedures, or "bone blocks" such as the Bristow are not sufficient for preventing future instability. Neer and Foster ( [595](#)) described the inferior capsular shift reconstruction specifically for patients with multidirectional laxity of the shoulder with atraumatic instability. This procedure attempts to eliminate the overall capsular laxity and is used only after rehabilitation has failed. Huber and Gerber ( [570](#)) reported on 25 consecutive children with 36 involved shoulders with voluntary subluxation of the shoulder. The children managed by "skillful neglect" had a satisfactory outcome, but only 50% of those treated with an operative procedure to prevent later degenerative arthritis had good results. They concluded that voluntary subluxation of the shoulder has a favorable result and that there is no indication for surgery with this problem in children.

## **AUTHORS' PREFERRED METHOD OF TREATMENT**

The most important problem in dealing with shoulder dislocations in children is to establish whether the dislocation is truly traumatic or atraumatic in nature. This is accomplished by obtaining a careful history of the mechanism of injury and performing a physical examination designed to elicit evidence of multidirectional instability of the opposite shoulder, generalized joint laxity, or a congenital or developmental problem. Care should be taken to identify the voluntary dislocator, who should be treated nonoperatively in essentially all cases.

In the acute traumatic dislocation, whether anterior or posterior, gentle closed reduction should be performed. Prereduction and postreduction radiographs are taken, and a thorough neurologic and vascular examination is performed. The traction/countertraction method under light sedation or a gentle manipulative reduction should be used. For an anterior traumatic dislocation, we immobilize the shoulder in internal rotation for 4 weeks. For a posterior dislocation, the shoulder is immobilized for 4 weeks in a commercial splint or modified spica cast with the shoulder in neutral rotation. After this period of immobilization, a rehabilitation program stressing rotator cuff strengthening is instituted.

As discussed, the recurrence rate after a traumatic anterior dislocation is 50% or higher. Although we hesitate to intervene surgically after the initial dislocation, the patient and parents should be counseled that the recurrence rate is high and that the rehabilitation program may fail. With a second dislocation, the diagnosis of recurrent dislocation is established and surgical intervention is indicated. We use the capsular shift procedure as described by Rockwood and associates ( [602](#)). (This technique is described in detail in Vol. I of *Fractures in Adults*.) If present, the Bankart's or Perthes' lesion is repaired anatomically to the anterior glenoid rim.

Capsular shift is then performed to tighten the anteroinferior capsule.

A 6-month course of rehabilitation follows surgical intervention. For the first month, pendulums and gentle elevation exercises are performed. The shoulder is protected in a sling, especially at night. The second and third months are used to regain range of motion, including protected external rotation. This procedure is designed to address the pathology without limiting motion. The fourth through sixth months are used for a progressive strengthening program, which includes strengthening of the rotator cuff and deltoid. At 6 months, the reconstruction is mature enough to release the child to a full activity level.

For atraumatic dislocation, reduction can be accomplished if necessary after an acute dislocation in a fashion similar to that described for traumatic dislocations. Again, attention should be focused on confirming the diagnosis of atraumatic dislocation. Patients with voluntary dislocation and their families should be counseled that the dislocations can be harmful to the joint and should be discouraged.

Patients with atraumatic instability should be treated with a vigorous rehabilitation program. Only in the face of recurrence after 6 to 12 months of supervised rehabilitation should surgical intervention be considered. Great care should be taken to exclude the voluntary dislocator as a surgical candidate. Psychiatric evaluation is instituted if necessary. A capsular procedure as described by Neer or the capsular shift technique described by Rockwood can be used to eliminate laxity of the joint capsule in a circumferential manner ([594,601,602](#)). Surgical management of the atraumatic dislocator is difficult and requires meticulous attention to detail during both the surgical procedure and the postoperative rehabilitation program.

## Rehabilitation

The importance of an extensive rehabilitation program for instability about the shoulder for both traumatic and atraumatic problems has been emphasized ([525,542](#)). Specific exercises are used to strengthen the rotator cuff and deltoid muscles. The scapular stabilizers are also strengthened. Three-inch wide strips of rubber (Therabands) are used to strengthen the cuff and deltoid muscles; Theratubes are used later if necessary. The rehabilitation program is detailed in Volume I of this series (*Fractures in Adults*).

The amount of weight varies depending on the extent of the problem as well as the patient's age, size, and baseline strength. Exercises are performed four to six times a day. After basic strengthening has been accomplished, isokinetic exercises using the flexion/extension plane and the internal and external rotation plane are effective for maximizing endurance and strength in the shoulder girdle musculature.

## Complications

There is little information in the literature about the success rate of surgical reconstruction of the shoulder for recurrent dislocation in children. As discussed, traumatic dislocation in a child or adolescent can progress to recurrent dislocation in 50% to 100% of cases. Rockwood and colleagues have shown that more than 85% of atraumatic dislocators can be managed with a vigorous rehabilitation program and do not require surgery ([535,601,602](#)). Surgical treatment of these problems in children could be expected to have a success rate at least equal to that in adults. Greater than 90% success in stopping traumatic dislocations would be expected with surgical reconstruction ([524,530,572](#)).

Complications of surgical reconstruction of the shoulder have included recurrent dislocation, recurrent subluxation, painfully restricted motion, problems with metal impingement or loosening about the shoulder, and neurologic injury. Perhaps the most common problem associated with the standard reconstructions about the shoulder that include subscapularis tendon-shortening procedures (Magnuson-Stack and Putti-Platt) has been loss of external rotation. This loss in adults has been associated in some patients with a more rapid progression to glenohumeral arthritis ([562,583](#)).

Procedures that use metallic implants about the shoulder, including the Bristow and the DuToit stapling procedures, have been associated with complications of metal impingement on the humeral head or encroachment on the articular surface. Both problems can lead to pain and eventual arthritic change ([528,533,549,619](#)).

The axillary nerve is the one most commonly injured with shoulder dislocation. Fortunately, most axillary nerve injuries associated with dislocations are neurapraxic, and function returns with time and observation. In the event of complete axillary nerve palsy, significant disability can exist due to the lack of deltoid function ([529,532,539,541,551,582](#)).

Morrison and Egan (593) reported an axillary artery and a brachial plexus injury in a luxatio erecta dislocation in an 11-year-old child. The artery was rejoined with a vein graft and the brachial plexus injury fully recovered.

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## CHAPTER REFERENCES

### Proximal Humerus

1. Ahn JI, Park JS. Pathological fractures secondary to unicameral bone cysts. *Int Orthop* 1994;18:20–22.
2. Aitken AP. End results of fractures of the proximal humeral epiphysis. *J Bone Joint Surg [Am]* 1936;18:1036–1041.
3. al Zahrani S. Modified rotational osteotomy of the humerus for Erb's palsy. *Int Orthop* 1993;17:202–204.
4. Artico M, Salvati M, D'Andrea V, et al. Isolated lesion of the axillary nerve: surgical treatment and outcome in 12 cases. *Neurosurgery* 1991;29:697–700.
5. Barber DB, Janus RB, Wade WH. Neuroarthropathy: an overuse injury of the shoulder in quadriplegia. *J Spinal Cord Med* 1996;19:9–11.
6. Baxter MP, Wiley JJ. Fractures of the proximal humeral epiphysis. Their influence on humeral growth. *J Bone Joint Surg [Br]* 1986;68:570–573.
7. Berger PE, Ofstein RA, Jackson DW, et al. MRI demonstration of radiographically occult fractures: what have we been missing? *Radiographics* 1989;9:407–436.
8. Bortel DT, Pritchett JW. Straight-line graphs for the predictions of growth of the upper extremities. *J Bone Joint Surg [Am]* 1993;75:885–892.
9. Bourdillan JF. Fracture–separation of the proximal epiphysis of the humerus. *J Bone Joint Surg [Br]* 1950;32:35–37.
10. Brems-Dalgaard E, Davidsen E, Sloth C. Radiographic examination of the acute shoulder. *Eur J Radiol* 1990;11:10–14.
11. Broker FHL, Burbach T. Ultrasonic diagnosis of separation of the proximal humeral epiphysis in the newborn. *J Bone Joint Surg [Am]* 1990;72:187–191.
12. Burgos-Flores J, Gonzales-Herranz P, Lopez-Mondejar JA, et al. Fractures of the proximal humeral epiphysis. *Int Orthop* 1993;17:16–19.
13. Caldwell JA. Treatment of fractures in the Cincinnati General Hospital. *Ann Surg* 1933;97:161.
14. Camus M, Lefebvre G, Veron P, et al. Obstetrical injuries of the newborn infant. Retrospective study apropos of 20,409 births. *J Gynecol Obstet Biol Reprod (Paris)* 1985;14:1033–1043.
15. Coene LN, Narakas AO. Operative management of lesions of the axillary nerve, isolated or combined with other nerve lesions. *Clin Neurol Neurosurg* 1992;94(suppl):64–66.
16. Cohn BT, Froimson AI. Salter 3 fracture–dislocation of glenohumeral joint in a 10-year-old. *Orthop Rev* 1986;15:403–404.
17. Covey DC, Riordan DC, Milstead ME, et al. Modification of the L'Episcopo procedure for brachial plexus birth palsies. *J Bone Joint Surg [Br]* 1992;74:897–901.
18. Curtis RJ Jr. Operative management of children's fracture of the shoulder region. *Orthop Clin North Am* 1990;21:315–324.
19. Curtis RJJ, Dameron TB Jr, Rockwood CA Jr. *Fractures and dislocations of the shoulder in children*, 3rd ed. Philadelphia: JB Lippincott, 1991:829–919.
20. Dalldorf PG, Bryan WJ. Displaced Salter-Harris type I injury in a gymnast. A slipped capital humeral epiphysis? *Orthop Rev* 1994;23:538–541.
21. Dameron TB Jr, Grubb SA. Humeral shaft fractures in adults. *South Med J* 1981;74:1461–1467.
22. Dameron TB Jr, Reibel DB. Fractures involving the proximal humeral epiphyseal plate. *J Bone Joint Surg [Am]* 1969;51:289–297.
23. Drew SJ, Giddins GE, Birch R. A slowly evolving brachial plexus injury following a proximal humerus fractures in a child. *J Hand Surg [Br]* 1995;20:24–25.
24. Edeiken BS, Libshitz HI, Cohen MA. Slipped proximal humeral epiphysis: a complication of radiotherapy to the shoulder in children. *Skel Radio* 1982;9:123–125.
25. Ellefsen BK, Frierson MA, Raney EM, et al. Humerus varus: a complication of neonatal, infantile, and childhood injury and infection. *J Pediatr Orthop* 1994;14:479–486.
26. Fraser RL, Haliburton RA, Barber JR. Displaced epiphyseal fractures of the proximal humerus. *Can J Surg* 1967;10:427–430.
27. Freundlich BD. Luxatio erecta. *J Trauma* 1983;23:434–436.
28. Friedlander HL. Separation of the proximal humeral epiphysis: a case report. *Clin Orthop* 1964;35:163–170.
29. Gagnaire JC, Thoulon JM, Chappuis JP, et al. Injuries to the upper extremities in the new-born diagnosed at birth. *J Gynecol Obstet Biol Reprod (Paris)* 1975;4:245–254.
30. Gerber C, Schneeberger AC, Vinh TS. The arterial vascularization of the humeral head. an anatomical study. *J Bone Joint Surg [Am]* 1990;72:1486–1494.
31. Giebel G, Suren EG. Injuries of the proximal humeral epiphysis. Indications for surgical therapy and results. *Chirurgie* 1983;54:406–410.
32. Gill TJ, Waters P. Valgus osteotomy of the humeral neck: a technique for the treatment of humerus varus. *J Shoulder Elbow Surg* 1997;6:306–310.
33. Gregg-Smith SJ, White SH. Salter-Harris III fracture–dislocation of the proximal humeral epiphysis. *Injury* 1992;23:199–200.
34. Gross SJ, Shime J, Farine D. Shoulder dystocia: predictors and outcome. *Am J Obstet Gynecol* 1987;156:334–336.

35. Guibert L, Allouis M, Bourdelat D, et al. Fractures and slipped epiphysis of the proximal humerus in children. Place and methods of surgical treatment. *Chir Pediatr* 1983;24:197–200.
36. Haliburton RA, Barber JR, Fraser RL. Pseudodislocation: an unusual birth injury. *Can J Surg* 1967;10:455–462.
37. Harris BA. Shoulder dystocia. *Clin Obstet Gynecol* 1984;27:106–111.
38. Herring JA, Peterson HA. Simple bone cyst with growth arrest. *J Pediatr Orthop* 1987;7:231–235.
39. Hoffer MM, Phipps GJ. Closed reduction and tendon transfer for treatment of dislocations of the glenohumeral joint secondary to brachial plexus birth palsy. *J Bone Joint Surg [Am]* 1998;80:997–1001.
40. Hohl JC. Fractures of the humerus in children. *Orthop Clin North Am* 1976;7:557–571.
41. Howard CB, Shinwell E, Nyska M, et al. Ultrasound diagnosis of neonatal fracture separation of the upper humeral epiphysis. *J Bone Joint Surg [Br]* 1993;74:471–472.
42. Howard FM, Shafer SJ. Injuries to the clavicle with neurovascular complications. A study of 14 cases. *J Bone Joint Surg [Am]* 1965;47:1335–1346.
43. Iqbal QM. Long-bone fractures among children in Malaysia. *Int Surg* 1974;59:410–415.
44. Jaberg H, Warner JJ, Jakob RP. Percutaneous stabilization of unstable fractures of the humerus. *J Bone Joint Surg [Am]* 1992;74:508–515.
45. Jackson ST, Hoffer MM, Parrish N. Brachial plexus palsy in the newborn. *J Bone Joint Surg [Am]* 1988;70:1217–1220.
46. James P, Heinrich SD. Ipsilateral proximal metaphyseal and flexion supracondylar humerus fractures with an associated olecranon avulsion fracture. *Orthopedics* 1991;14:713–716.
47. Jeffrey CC. Fracture separation of the upper humeral epiphysis. *Surg Gynecol Obstet* 1953;96:205–209.
48. Kirkos JM, Papadopoulos IA. Late treatment of brachial plexus palsy secondary to birth injuries: rotational osteotomy of the proximal part of the humerus. *J Bone Joint Surg [Am]* 1998;80:1477–1483.
49. Klasson SC, Vander Schilden JL, Park JP. Late effect of isolated avulsion fractures of the lesser tubercle of the humerus in children. Report of 2 cases. *J Bone Joint Surg [Am]* 1993;75:1691–1694.
50. Kleinman PK, Akins CM. The “vanishing” epiphysis: sign of Salter type I fracture of the proximal humerus in infancy. *Br J Radiol* 1982;55:865–867.
51. Kohler R, Trillaud JM. Fracture and fracture separation of the proximal humerus in children: report of 136 cases. *J Pediatr Orthop* 1983;3:326–332.
52. Kothari K, Bernstein RM, Griffiths JJ, et al. Luxatio erecta. *Skel Radio*. 1984;11:47–49.
53. Kuhns LR, Sherman MP, Poznanaski AK, et al. Humeral head and coracoid ossification in the newborn. *Radiology* 1973;107:145–149.
54. Kumar R, Cornah MS, Morris DL. Hydatid cyst—a rare cause of pathological fracture: a case report. *Injury* 1984;15:284–285.
55. Laing PG. The arterial supply of the adult humerus. *J Bone Joint Surg [Am]* 1956;38:1105–1116.
56. Landin LA. Fracture patterns in children: analysis of 8682 fractures with special reference to incidence, etiology and secular changes in Swedish urban populations. *Acta Orthop Scand Suppl* 1983;54:1–109.
57. Landin LA. Epidemiology of children's fractures. *J Pediatr Orthop* 1997;6:79–83.
58. Langenskiöld A. Adolescent humerus varus. *Acta Chirurg Scand* 1953;105:353–363.
59. Larsen CF, Kiaer T, Lindequist S. Fractures of the proximal humerus in children: 9-year follow-up of 64 unoperated on cases. *Acta Orthop Scand* 1990;61:255–257.
60. Lee HG. Operative reduction of an unusual fracture of the upper epiphyseal plate of the humerus. *J Bone Joint Surg* 1944;26:401–404.
61. Lemperg R, Lilliequest B. Dislocation of the proximal epiphysis of the humerus in newborns. *Acta Paediatr Scand* 1970;59:377–380.
62. Lentz W, Meuser P. The treatment of fractures of the proximal humerus. *Arch Orthop Trauma Surg* 1980;96:283–285.
63. Leslie JT, Ryan TJ. The anterior axillary incision to approach the shoulder joint. *J Bone Joint Surg [Am]* 1962;44:1193–1196.
64. Liebling G, Bartel HG. Unusual migration of a Kirschner wire following drill wire fixation of a subcapital humerus fracture. *Beitr Orthop Traumatol* 1987;34:585–587.
65. Linson MA. Axillary artery thrombosis after fracture of the humerus. A case report. *J Bone Joint Surg [Am]* 1980;62:1214–1215.
66. Lipscomb AB. Baseball pitching injuries in growing athletes. *J Sports Med* 1975;3:25–34.
67. Lock TR, Aronson DD. Fractures in patients who have myelomeningocele. *J Bone Joint Surg [Am]* 1989;71:1153–1157.
68. Loder RT. Pediatric polytrauma: orthopaedic care and hospital course. *J Orthop Trauma* 1987;1:48–54.
69. Lucas LS, Gill JH. Humerus varus following birth injury to the proximal humeral epiphysis. *J Bone Joint Surg* 1947;29:367–369.
70. Lyons FA, Rockwood CA. Current concepts review. Migration of pins used in operations on the shoulder. *J Bone Joint Surg [Am]* 1990;72:1262–1267.
71. Macfarlane I, Mushayt K. Double closed fractures of the humerus in a child. A case report. *J Bone Joint Surg [Am]* 1990;72:443.
72. Madsen TE. Fractures of the extremities in the newborn. *Acta Obstet Gynecol Scand* 1955;34:41.
73. Markel DC, Donley BG, Blasier RB. Percutaneous intramedullary pinning of proximal humeral fractures. *Orthop Rev* 1994;23:667–671.
74. Martin RP, Parsons DL. Avascular necrosis of the proximal humeral epiphysis after physeal fracture. A case report. *J Bone Joint Surg [Am]* 1997;79:760–762.
75. Moed BR, LaMont RL. Unicameral bone cyst complicated by growth retardation. *J Bone Joint Surg [Am]* 1982;64:1379–1381.
76. Müller ME, Allgöwer M, Schneider R, et al. *Manual of internal fixation. Techniques recommended by the AO-ASIF Group*, 3rd ed. Berlin: Springer-Verlag, 1991.
77. Neer CS 2nd, Horowitz BS. Fractures of the proximal humeral epiphysal plate. *Clin Orthop* 1965;41:24–31.
78. Nordqvist A, Petersson CJ. Incidence and causes of shoulder girdle injuries in an urban population. *J Shoulder Elbow Surg* 1995;4:107–112.
79. Norman A, Schiffman M. Simple bone cysts: factors of age dependency. *Radiology* 1977;124:779–782.
80. Obremsky W, Routt ML Jr. Fracture–dislocation of the shoulder in a child: case report. *J Trauma* 1994;36:137–140.
81. Oden JA, Conlogue GJ, Jensen P. Radiology of postnatal skeletal development: the proximal humerus. *Skel Radio*. 1978;2:153–160.
82. Olszewski W, Popinski M. Fractures of the neck and shaft of the humerus as a rare form of double fractures in children. *Chir Narzadow Ruchu Ortop Pol* 1974;39:121–123.
83. Peterson HA, Madhok R, Benson JT, et al. Physeal fractures: Part 1. Epidemiology in Olmsted County, Minnesota, 1979–1988. *J Pediatr Orthop* 1994;14:423–430.
84. Phipps GJ, Hoffer MM. Latissimus dorsi and teres major transfer to rotator cuff for Erb's palsy. *J Shoulder Elbow Surg* 1995;4:124–129.
85. Pritchett JW. Growth and predictions of growth in the upper extremity. *J Bone Joint Surg [Am]* 1988;70:520–525.
86. Pritchett JW. Growth plate activity in the upper extremity. *Clin Orthop* 1991;268:235–242.
87. Robin GC, Kedar SS. Separation of the upper humeral epiphysis in pituitary gigantism. *J Bone Joint Surg [Am]* 1962;44:189–192.
88. Rose SH, Melton LJ 3rd, Morrey BF, et al. Epidemiologic features of humeral fractures. *Clin Orthop* 1982;168:24–30.
89. Ross GJ, Love MB. Isolated avulsion fracture of the lesser tuberosity of the humerus: report of 2 cases. *Radiology* 1989;172:833–834.
90. Runkel M, Kreitner KF, Wenda K, et al. Nuclear magnetic tomography in shoulder dislocation. *Unfallchirurgie* 1993;96:124–128.
91. Sakakida K. Clinical observations on the epiphysal separation of long bones. *Clin Orthop* 1964;34:119–141.
92. Salter RB, Harris WR. Injuries involving epiphysal plates. *J Bone Joint Surg [Am]* 1963;45:587–622.
93. Samilson RL. Congenital and developmental anomalies of the shoulder girdle. *Orthop Clin North Am* 1980;11:219–231.
94. Sanders JO, Rockwood CA Jr, Curtis RJ. Fractures and dislocations of the humeral shaft and shoulder. In: Rockwood CA Jr, Wilkins KE, Beaty JH, eds. *Fractures in children*. Philadelphia: Lippincott-Raven, 1996:905–1021.
95. Scaglietti O. The obstetrical shoulder trauma. *Surg Gynecol Obstet* 1938;66:686.
96. Schopler SA, Lawrence JF, Johnson MK. Lengthening of the humerus for upper extremity limb length discrepancy. *J Pediatr Orthop* 1986;6:477–480.
97. Seitz J, Valdes F, Kramer A. Acute ischemia of the upper extremity caused by axillary contused trauma. Report of 3 cases. *Rev Med Chila* 1991;119:567–571.
98. Sessa S, Lascombes P, Prevot J, et al. Centromedullary nailing in fractures of the upper end of the humerus in children and adolescents. *Chir Pediatr* 1990;31:43–46.
99. Shaw BA, Murphy KM, Shaw A, et al. Humerus shaft fractures in young children: accident or abuse? *J Pediatr Orthop* 1997;17:293–297.
100. Sherk HH, Probst C. Fractures of the proximal humeral epiphysis. *Orthop Clin North Am* 1975;6:401–413.
101. Shulman BH, Terhune CB. Epiphysal injuries in breech delivery. *Pediatrics* 1951;8:693–700.
102. Sloth C, Just SL. The apical oblique radiograph in examination of acute shoulder trauma. *Eur J Radio*. 1989;9:147–151.
103. Smith FM. Fracture–separation of the proximal humeral epiphysis: a study of cases seen at the Presbyterian Hospital, 1929–1953. *Am J Surg* 1956;91:627–635.
104. Solonen KA, Vastamaki M. Osteotomy of the neck of the humerus for traumatic varus deformity. *Acta Orthop Scand* 1985;56:79–80.
105. Stahl EJ, Karpman R. Normal growth and growth predictions in the upper extremity. *J Hand Surg [Am]* 1986;11:593–596.
106. Stewart MJ, Hundley JM. Fractures of the humerus: a comparative study in methods of treatment. *J Bone Joint Surg [Am]* 1955;37:681–692.
107. Stromqvist B, Lidgren L, Norgren L, et al. Neurovascular injury complicating displaced proximal fractures of the humerus. *Injury* 1987;18:423–425.
108. Szalay EA, Rockwood CA Jr. Injuries of the shoulder and arm. *Emerg Med Clin North Am* 1984;2:279–294.
109. te Slaa RL, Nollen AJ. A Salter type 3 fracture of the proximal epiphysis of the humerus. *Injury* 1987;18:429–431.
110. Tirman PF, Stauffer AE, Crues JV 3rd, et al. Saline magnetic resonance arthrography in the evaluation of glenohumeral instability. *Arthroscopy* 1993;9:550–559.
111. Torode I, Donnan L. Posterior dislocation of the humeral head in association with obstetric paralysis. *J Pediatr Orthop* 1998;18:611–615.
112. Troum S, Floyd WE 3rd, Waters PM. Posterior dislocation of the humeral head in infancy associated with obstetrical paralysis. A case report. *J Bone Joint Surg [Am]* 1993;75:1370–1375.
113. Trueta J. *Studies of the development and decay of the human frame*. Philadelphia: WB Saunders, 1968.
114. Tullos HS, Erwin WD, Woods GW, et al. Unusual lesions of the pitching arm. *Clin Orthop* 1972;88:169–182.
115. Tullos HS, Fain RH. Little league shoulder: rotational stress fracture of proximal epiphysis. *J Sports Med* 1974;2:152–153.
116. van den Broek JA, Vegter J. Echography in the diagnosis of epiphysiolysis of the proximal humerus in a new-born infant. *Ned Tijdschr Geneeskd* 1988;132:1015–1017.
117. Vastamaki M, Solonen KA. Posterior dislocation and fracture–dislocation of the shoulder. *Acta Orthop Scand* 1980;51:479–484.
118. Visser JD, Rietberg M. Interposition of the tendon of the long head of biceps in fracture separation of the proximal humeral epiphysis. *Neth J Surg* 1980;32:12–15.
119. Wadlington VR, Hendrix RW, Rogers LF. Computed tomography of posterior fracture–dislocations of the shoulder: case reports. *J Trauma* 1992;32:113–115.
120. Wang P Jr, Koval KJ, Lehman W, et al. Salter-Harris type III fracture–dislocation of the proximal humerus. *J Pediatr Orthop* 1997;6:219–222.
121. White GM, Riley LHJ. Isolated avulsion of the subscapularis insertion in a child. A case report. *J Bone Joint Surg [Am]* 1985;67:635–636.
122. Whitman RA. Treatment of epiphysal displacement and fractures of the upper extremity of the humerus designed to assure deficit adjustment and fixation of the fragments. *Ann Surg* 1908;47:706–708.
123. Williams DJ. The mechanisms producing fracture-separation of the proximal humeral epiphysis. *J Bone Joint Surg [Br]* 1981;63:102–107.

124. Wong-Chung J, O'Brien T. Salter Harris type III fracture of the proximal humeral physis. *Injury* 1988;19:453-454.
125. Worlock P, Stower M. Fracture patterns in Nottingham children. *J Pediatr Orthop* 1986;6:656-661.
126. Yosipovitch Z, Goldberg I. Inferior subluxation of the humeral head after injury to the shoulder. A brief note. *J Bone Joint Surg [Am]* 1989;71:751-753.
127. Zuckerman JD, Flugstad DL, Teitz CC, et al. Axillary artery injury as a complication of proximal humerus fractures. Two case reports and a review of the literature. *Clin Orthop* 1984;189:234-237.

#### Scapula

128. Ada JR, Miller ME. Scapular fractures: analysis of 113 cases. *Clin Orthop* 1991;269:174-180.
129. Chung SMK, Nissenbaum MM. Congenital and developmental defects of the shoulder. *Orthop Clin North Am* 1975;6:381-392.
130. DePalma AF. *Surgery of the shoulder*, 3rd ed. Philadelphia: JB Lippincott, 1983.
131. Ebraheim NA, An HS, Jackson WT, et al. Scapulothoracic dissociation. *J Bone Joint Surg [Am]* 1988;70:428-432.
132. Goss TP. Current concepts review: fractures of the glenoid cavity. *J Bone Joint Surg [Am]* 1992;72:299-305.
133. Goss TP. Scapular fracture and dislocations: diagnosis and treatment. *J Am Acad Orthop Surg* 1995;3:22-33.
134. Guttentag IJ, Rehtine GR. Fractures of the scapula: a review of the literature. *Orthop Rev* 1988;17:147-158.
135. Hardegger FH, Simpson LA, Weber BG. The operative treatment of scapula fractures. *J Bone Joint Surg [Br]* 1984;66:725-731.
136. Herscovici D Jr, Fiennes AG, Allgower M, et al. The floating shoulder: ipsilateral clavicle and scapular neck fractures. *J Bone Joint Surg [Br]* 1992;74:362-364.
137. Ideberg R. Fractures of the scapula involving the glenoid fossa. In: Bateman JE, Walsh RD, eds. *Surgery of the shoulder*. Toronto: BC Decker, 1984:63-66.
138. Ideberg R. Unusual glenoid fractures. *Acta Orthop Scand* 1987;58:191-192.
139. Imatani RJ. Fractures of the scapulae: a review of 53 fractures. *J Trauma* 1975;15:473-478.
140. Kim SJ, Min BH. Congenital bilateral absence of the acromion: a case report. *Clin Orthop* 1994;300:117-119.
141. Kleinman PK, Spevak MR. Variations in acromial ossification simulating infant abuse in victims of sudden infant death syndrome. *Radiology* 1991;180:185-187.
142. Kogutt MS, Swischuk LE, Fagan CJ. Patterns of injury and significance of uncommon fractures in the battered child syndrome. *Am J Roentgenol Radium Ther Nucl Med* 1974;121:143-149.
143. Kuhn JE, Blasler RB, Carpenter JE. Fractures of the acromion process: a proposed classification system. *J Orthop Trauma* 1994;8:6-13.
144. Leung KS, Lam TP. Open reduction and internal fixation of ipsilateral fractures of the scapular neck and clavicle. *J Bone Joint Surg [Am]* 1993;75:1015-1018.
145. Liberson F. Os acromiale? A contested anomaly. *J Joint Surg* 1937;19:683-689.
146. Lindblom A, Leven H. Prognosis in fractures of body and neck of the scapula. *Acta Chir Scand* 1974;140:33-47.
147. Montgomery SP, Loyd RDL. Avulsion fracture of the coracoid epiphysis with acromioclavicular separation. *J Bone Joint Surg [Am]* 1977;59:963-965.
148. McClure JG, Raney RB. Anomalies of the scapula. *Clin Orthop* 1975;110:22-31.
149. McGahan JP, Rab GT. Fracture of the acromion associated with an axillary nerve deficit: a case report and review of the literature. *Clin Orthop* 1980;147:216-218.
150. McGahan JP, Rab GT, Dublin A. Fractures of the scapula. *J Trauma* 1980;20:880-883.
151. Neer CS II. Fractures. In: *Shoulder reconstruction*. Philadelphia: WB Saunders, 1990:412.
152. Neer CS II. Fractures about the shoulder. In: Wood CA, Green DP, ed. *Fractures*. Philadelphia: JB Lippincott, 1984:713-721.
153. Nettrou LF, Krufky EL, Mueller RE, Raycroft JF. Locked scapula: intrathoracic dislocation of the inferior angle. *J Bone Joint Surg [Am]* 1987;54:147-155.
154. Nordqvist A, Petersson C. Fracture of the body, neck, or spine of the scapula: a long-term follow-up study. *Clin Orthop* 1992;283:139-144.
155. Oreck SL, Burgess A, Levine AM. Traumatic lateral displacement of the scapula: a radiographic sign of neurovascular disease. *J Bone Joint Surg [Am]* 1984;66:758-763.
156. Orrell KG, Bell DF. Structural abnormality of the clavicle associated with Sprengel's deformity: a case report. *Clin Orthop* 1990;258:157-159.
157. Rowe CR. Fractures of the scapula. *Surg Clin North Am* 1963;43:1565-1571.
158. Samilson RL. Congenital and developmental anomalies of the shoulder girdle. *Orthop Clin North Am* 1980;11:219-231.
159. Soslowky LJ, Flatow EL, Bigliani LU, et al. Articular geometry of the glenohumeral joint. *Clin Orthop* 1992;285:181-190.
160. Thompson DA, Flynn TC, Miller PW, et al. The significance of scapular fractures. *J Trauma* 1985;25:974-977.
161. Wilber MC, Evans EB. Fractures of the scapula: an analysis of forty cases and review of literature. *J Bone Joint Surg [Am]* 1977;59:358-362.
162. Wippermann B, Schmidt U, Nerlich M. Results of treatment of compartment syndrome of the upper arm. *Unfallchirurgie* 1991;94:231-235.
163. Zdravkovic D, Damholt VV. Comminuted and severely displaced fractures of the scapula. *Acta Orthop Scand* 1974;45:60-65.

#### Clavicle

164. Abbott L, Lucas D. Function of the clavicle: its surgical significance. *Ann Surg* 1954;140:583-599.
165. Al-Etani H, D'Astous J, Letts J, et al. Masked rotatory subluxation of the atlas associated with fracture of the clavicle: a clinical and biomechanical analysis. *J Bone Joint Surg [Am]* 1998;80:1477-1483.
166. Allman FL Jr. Fractures and ligamentous injuries of the clavicle and its articulation. *J Bone Joint Surg [Am]* 1967;49:774-784.
167. Andersen K, Jensen PO, Lauritzen J. Treatment of clavicular fractures. Figure of eight bandage versus a simple sling. *Acta Orthop Scand* 1987;58:71-74.
168. Bakalim G, Wilpulla E. Surgical or conservative treatment of total dislocation of the acromioclavicular joint. *Acta Chir Scand* 1975;141:43-47.
169. Barber FA. Complete posterior acromioclavicular dislocation. *Orthopaedics* 1987;10:493-496.
170. Bateman JE. Neurovascular syndromes related to the clavicle. *Clin Orthop* 1968;58:75-82.
171. Black GH, McPherson JA, Reed MH. Traumatic pseudodislocation of the acromioclavicular joint in children. A 15 year review. *Am J Sports Med* 1991;19:644-646.
172. Bearn JG. Direct observations on the function of the capsule of the sternoclavicular support. *J Anat* 1967;101:159-170.
173. Boehme D, Curtis RJ, DeHaan JT, et al. Nonunion of fractures of the midshaft of the clavicle. Treatment with a modified Hagie intramedullary pin and autogenous bone grafting. *J Bone Joint Surg [Am]* 1991;73:1219-1226.
174. Brooks S. Bilateral congenital pseudarthrosis of the clavicles? Case report and review of the literature. *Br J Clin Pract* 1984;38:432-433.
175. Browne JE, Stanley RF, Tullos HS, et al. Acromioclavicular joint dislocations: comparative results following operative treatment with and without primary distal clavisectomy. *Am J Sports Med* 1977;5:258-263.
176. Brunner C, Morger R. Congenital nonunion of the clavicle. *Pediatr Pado* 1981;16:137-141.
177. Camus M, Lefebvre G, Veron O, et al. Obstetrical injuries of the newborn infant: retrospective study apropos of 20409 births. *J Gynecol Obstet Biol Reprod (Paris)* 1985;14:1033-1043.
178. Clark RL, Milgram JW, Yawn DH. Fatal aortic perforation and cardiac tamponade due to a Kirschner wire migrating from the right sternoclavicular joint. *South Med J* 1974;67:316-318.
179. Cohen AW, Otto SR. Obstetric clavicular fractures: a three year analysis. *J Reprod Med* 1980;25:119-122.
180. Dameron TB, Rockwood CA. Fractures and dislocations of the shoulder. In: Rockwood CA, Wilkins KE, King RE, eds. *Fractures in children*. Philadelphia: JB Lippincott, 1984:624-653.
181. Darrow JC, Smith JA, Lockwood RC. A new conservative method for the treatment of type III acromioclavicular separations. *Orthop Clin North Am* 1980;11:727-733.
182. Dartoy C, Fenoll B, Hra B, et al. Epiphyseal fracture-avulsion of the distal extremity of the clavicle. *Ann Radiol (Paris)* 1993;36:125-128.
183. Della Santa D, Narakas A. Fractures of the clavicle and secondary lesions of the brachial plexus. *Z Unfallchir Versicherungsmed* 1992;85:58-65.
184. Della Santa D, Narakas A, Bonnard C. Late lesions of the brachial plexus after fracture of the clavicle. *Ann Chir Main Memb Super* 1991;10:531-540.
185. Dugdale TW, Fulkerson JP. Pneumothorax complicating a closed fracture of the clavicle. A case report. *Clin Orthop* 1987;221:212-214.
186. Eidman DK, Siff SJ, Tullos HS. Acromioclavicular lesions in children. *Am J Sports Med* 1981;9:150-154.
187. Eskola A, Vainionpaa S, Myllynen P, et al. Surgery for ununited clavicular fracture. *Acta Orthop Scand* 1986;57:366-367.
188. Falstie-Jensen S, Mikkelsen P. Pseudodislocation of the acromioclavicular joint. *J Bone Joint Surg [Br]* 1982;64:368-369.
189. Farkas R, Levine S. X-ray incidence of fractured clavicle in vertex presentation. *Am J Obstet Gynecol* 1950;59:204-206.
190. Fowler AW. Migration of a wire from the sternoclavicular joint to the pericardial cavity. *Injury* 1981;13:261-262.
191. Fowler AW. Treatment of fractured clavicle. *Lancet* 1968;1:46-47.
192. Gardner E. The embryology of the clavicle. *Clin Orthop* 1968;58:9-16.
193. Gardner MA, Bidstrup BP. Intrathoracic great vessel injury resulting from blunt chest trauma associated with posterior dislocation of the sternoclavicular joint. *Aust N Z J Surg* 1983;53:427-430.
194. Gerber C, Rockwood CA. Subcoracoid dislocation of the lateral end of the clavicle. A report of three cases. *J Bone Joint Surg [Am]* 1987;69:924-927.
195. Gilbert WM, Tchabo JG. Fractured clavicle in newborns. *Int Surg* 1988;73:123-125.
196. Gitche G, Schatten C. Incidence and potential factors in the genesis of birth injury induced clavicular fractures. *Zentralbl Gynakol* 1987;109:909-912.
197. Goss TP. Scapular fracture and dislocations: diagnosis and treatment. *J Am Acad Orthop Surg* 1995;3:22-33.
198. Goddard NJ, Stabler J, Albert JS. Atlantoaxial rotatory fixation and fracture of the clavicle: an association and classification. *J Bone Joint Surg [Br]* 1990;72:72-75.
199. Graif M, Stahl-Kent V, Ben-Ami T, et al. Sonographic detection of occult bone fractures. *Pediatr Radiol* 1988;18:383-385.
200. Havranek P. Injuries of distal clavicular physis in children. *J Pediatr Orthop* 1989;9:213-215.
201. Herndon WA. Child abuse in a military population. *J Pediatr Orthop* 1983;3:73-76.
202. Howard FM, Shafer SJ. Injuries to the clavicle with neurovascular complications: a study of fourteen cases. *J Bone Joint Surg [Am]* 1965;47:1335-1346.
203. Inman VT, Saunders JB, Abbott LC. Observations on the function of the shoulder joint. *J Bone Joint Surg* 1944;26:1-30.
204. Jablon M, Sutker A, Post M. Irreducible fracture of the middle third of the clavicle. Report of a case. *J Bone Joint Surg [Am]* 1979;61:296-298.
205. Jackson ST, Hoffer MM, Parrish N. Brachial plexus palsy in the newborn. *J Bone Joint Surg [Am]* 1988;70:1217-1220.
206. Jelic A, Marin L, Pracny M, et al. Fractures of the clavicle in neonates. *Lijec Vjesn* 1992;114:32-35.
207. Jensen PO, Andersen K, Lauritzen J. Treatment of midclavicular fractures. A prospective randomized trial comparing treatment with a figure of eight dressing and a simple arm sling. *Ugeskr*

- Laeger 1985;147:1986–1988.
208. Jit I, Kulkarni M. Times of appearance and fusion of epiphysis at the medial end of the clavicle. *Ind J Med Res* 1976;64:773–782.
  209. Jojart G, Nagy G. Ultrasonographic screening of neonatal adrenal apoplexy. *Int Urol Nephrol* 1992;24:591–596.
  210. Jojart G, Zubek L, Toth G. Clavicle fractures in the newborn. *Orv Hetil* 1991;132:2655–2657.
  211. Joseph PR, Rosenfeld W. Clavicular fractures in neonates. *Am J Dis Child* 1990;144:165–167.
  212. Katz R, Landman J, Dulitzky F, et al. Fracture of the clavicle in the newborn. An ultrasound diagnosis. *J Ultrasound Med* 1988;7:21–23.
  213. Kaur H, Jit I. Brief communication: coracoclavicular joint in northwest Indians. *Am J Phys Anthropol* 1991;85:457–460.
  214. Kogutt MS, Swischuk LE, Fagan CJ. Patterns of injury and significance of uncommon fractures in the battered child syndrome. *Am J Roentgenol Radium Ther Nucl Med* 1974;121:143–149.
  215. Kreisinger V. Sur le traitement des fractures de la clavicle. *Rev Chir* 1927;65:396–407.
  216. Landin LA. Fracture patterns in children: analysis of 8682 fractures with special reference to incidence, etiology, and secular changes in Swedish urban populations. *Acta Orthop Scand Suppl* 1983;54:1–109.
  217. Leffert RD. Brachial plexus injuries. *N Engl J Med* 1974;291:1059–1067.
  218. Levine MG, Holroyde J, Woods JR Jr, et al. Birth trauma: incidence and predisposing factors. *Obstet Gynecol* 1984;63:792–795.
  219. Lichtenberg RP. A study of 2532 fractures in children. *Am J Surg* 1954;87:330–338.
  220. Longo R, Ruggiero L. Left pneumothorax with subcutaneous emphysema secondary to left clavicular fracture and homolateral obstetrical paralysis of the arm. *Minerva Pediatr* 1982;34:273–276.
  221. Madsen ET. Fractures of the extremities in the newborn. *Acta Obstet Gynecol Scand* 1955;34:41–74.
  222. Manske DJ, Szabo RM. The operative treatment of midshaft clavicular nonunions. *J Bone Joint Surg [Am]* 1985;67:1367–1371.
  223. Mazet R Jr. Migration of a Kirschner wire from the shoulder region into the lung: a report of 2 cases. *J Bone Joint Surg [Am]* 1943;25:477–483.
  224. McCaughan JS Jr, Miller PR. Migration of Steinmann pin from shoulder to lung [Letter]. *JAMA* 1969;207:1917.
  225. Meeks RJ, Riebel GD. Isolated clavicle fracture with associated pneumothorax. A case report. *Am J Emerg Med* 1991;9:555–556.
  226. Miller DS, Boswick JA Jr. Lesions of the brachial plexus associated with fractures of the clavicle. *Clin Orthop* 1969;64:144–149.
  227. Mital MA, Aufranc OE. Venous occlusion following greenstick fracture of clavicle. *JAMA* 1968;206:1301–1302.
  228. Moseley HF. The clavicle: its anatomy and function. *Clin Orthop* 1968;58:17–27.
  229. Mullaji AB, Jupiter JB. Low contact dynamic compression plating of the clavicle. *Injury* 1994;25:41–45.
  230. Mullick S. Treatment of mid-clavicular fractures. *Lancet* 1967:499.
  231. Nogi J, Heckman JD, Hakala M, et al. Non union of the clavicle in a child. A case report. *Clin Orthop* 1975;110:19–21.
  232. Nordqvist A, Petersson C. The incidence of fractures of the clavicle. *Clin Orthop* 1994;300:127–132.
  233. Ogata S, Uthoff HK. The early development and ossification of the human clavicle. An embryologic study. *Acta Orthop Scand* 1990;61:330–334.
  234. Ogden JA. Distal clavicular physeal injury. *Clin Orthop* 1984;188:68–73.
  235. Ogden JA. *Skeletal injury in the child*, 2nd ed. Philadelphia: WB Saunders, 1990.
  236. Ogden JA, Conlogue GJ, Bronson ML. Radiology of postnatal skeletal development. III. The clavicle. *Skel Radio* 1979;4:196–203.
  237. Oppenheim WL, Davis A, Growdon WA, et al. Clavicle fractures in the newborn. *Clin Orthop* 1990;250:176–180.
  238. O'Rourke IC, Middleton RWD. The place and efficacy of operative management of fractured clavicle. *Injury* 1975;6:236–240.
  239. Owings-Webb PA. Epiphyseal union of the anterior iliac crest and medial clavicle in a modern multiracial sample of American males and females. *Am J Phys Anthropol* 1985;68:457–466.
  240. Poigenfurst J, Rappold G, Fischer W. Plating of fresh clavicular fractures: results of 122 operations. *Injury* 1992;23:237–241.
  241. Pollock RC, Banks MJ, Emery RJ. Diagnosis of retrosternal dislocation of the clavicle with ultrasound. *Injury* 1996;27:670–671.
  242. Post M. Current concepts in the treatment of fractures of the clavicle. *Clin Orthop* 1989;245:89–101.
  243. Pyper JB. Nonunion of fractures of the clavicle. *Injury* 1978;9:268–270.
  244. Qureshi AA, Kuo KN. Posttraumatic cleidoscapular synostosis following a fracture of the clavicle. *J Bone Joint Surg [Am]* 1999;81:256–258.
  245. Riemer BL, Butterfield SL, Daffner RH, et al. The abduction lordotic view of the clavicle: a new technique for radiographic visualization. *J Orthop Trauma* 1991;5:392–394.
  246. Rockwood CA. Dislocations of the sternoclavicular joint. *AAOS Instr Course Lect* 1975;24:144–159.
  247. Rockwood CA Jr. The shoulder: facts, confusion, and myths. *Int Orthop* 1991;15:401–405.
  248. Roper BA, Levack B. The surgical treatment of acromioclavicular dislocations. *J Bone Joint Surg [Am]* 1982;69A:1045–1051.
  249. Rowe CR. An atlas of anatomy and treatment of midclavicular fractures. *Clin Orthop* 1968;58:29–42.
  250. Rubin A. Birth injuries: incidence, mechanisms, and end results. *Obstet Gynecol* 1964;23:218–221.
  251. Salonen IS, Uusitalo R. Birth injuries: incidence and predisposing factors. *Z Kinderchir* 1990;45:133–135.
  252. Salter RB, Harris WR. Injuries involving the epiphyseal plate. *J Bone Joint Surg [Am]* 1963;45:587–622.
  253. Sanders JO, Rockwood CA Jr, Curtis RJ. Fractures and dislocations of the humeral shaft and shoulder. In: Rockwood CA Jr, Wilkins KE, Beaty JH, eds. *Fractures in children*. Philadelphia: Lippincott-Raven, 1996:905–1021.
  254. Sanford HN. The Moro reflex as a diagnostic aid in fracture of the clavicle in the newborn infant. *Am J Dis Child* 1931;41:1304–1306.
  255. Sankarankutty M, Turner BW. Fractures of the clavicle. *Injury* 1975;7:101–106.
  256. Schwarz N, Hocker K. Osteosynthesis of irreducible fractures of the clavicle with 2.7 mm ASIF plates. *J Trauma* 1992;33:179–183.
  257. Schwarz N, Leixnering M. Technique and results of clavicular medullary wiring. *Zentralbl Chir* 1986;111:640–647.
  258. Silloway KA, McLaughlin RE, Edlich RC, et al. Clavicular fractures and acromioclavicular joint dislocations in lacrosse: preventable injuries. *J Emerg Med* 1985;3:117–121.
  259. Stanley D, Norris SH. Recovery following fractures of the clavicle treated conservatively. *Injury* 1988;19:162–164.
  260. Stanley D, Trowbridge EA, Norris SH. The mechanism of clavicular fractures. A clinical and biomechanical analysis. *J Bone Joint Surg [Br]* 1988;70:461–464.
  261. Todd TW, D'Errico J Jr. The clavicular epiphyses. *Am J Anat* 1928;41:25–50.
  262. Tse DH, Slabaugh PB, Carlson PA. Injury to the axillary artery by a closed fracture of the clavicle. A case report. *J Bone Joint Surg [Am]* 1980;62:1372–1374.
  263. Wall JJ. Congenital pseudarthrosis of the clavicle. *J Bone Joint Surg [Am]* 1970;52:1003–1009.
  264. Walle T, Hartikainen-Sorri A. Obstetric shoulder injury: associated risk factors, prediction, and prognosis. *Acta Obstet Gynecol Scand* 1993;72:450–454.
  265. Weinberg B, Seife B, Alonso P. The apical oblique view of the clavicle: its usefulness in neonatal and childhood trauma. *Skel Radio* 1991;20:201–203.
  266. Wilkins RM, Johnston RM. Ununited fractures of the clavicle. *J Bone Joint Surg [Am]* 1983;65:773–778.
  267. Worman LW, Leagus C. Intrathoracic injury following retrosternal dislocation of the clavicle. *J Trauma* 1967;7:416–423.
  268. Zenni EJ Jr, Krieg JK, Rosen MJ. Open reduction and internal fixation of clavicular fractures. *J Bone Joint Surg [Am]* 1981;63:147–151.

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269. Allen ME. Stress fracture of the humerus: a case study. *Am J Sports Med* 1984;12:244–245.
270. Alnot JY, Le Reun D. Traumatic lesions of the radial nerve of the arm. *Rev Chir Orthop Reparatrice Appar Mot* 1989;75:433–442.
271. Amillo S, Barrios RH, Martinez-Peric R, et al. Surgical treatment of the radial nerve lesions associated with fractures of the humerus. *J Orthop Trauma* 1993;7:211–215.
272. Andre S, Feuillade de Chauvin P, Camilleri A, et al. Recent fractures of the humeral diaphysis in adults—comparison of orthopedic and surgical treatment apropos of 252 cases. *Rev Chir Orthop Reparatrice Appar Mot* 1984;70:49–61.
273. Ardashev IP. Treatment of pseudarthroses of the humerus after gunshot wound. *Ortop Traumatol Protez* 1990;11–13.
274. Arnold JA, Nasca RJ, Nelson CL. Supracondylar fractures of the humerus. The role of dynamic factors in prevention of deformity. *J Bone Joint Surg [Am]* 1977;59:589–595.
275. Arrive L, Sellier N, Kalifa G, et al. Diagnostic difficulties of isolated symptomatic unilamellar periosteal appositions. Uncommon form of fatigue fracture in children. *J Radio* 1988;69:351–356.
276. Asche G. Use of external fixation in pediatric fractures. *Zentralbl Chir* 1986;111:391–397.
277. Astedt B. A method for the treatment of humerus fractures in the newborn using the S. von Rosen splint. *Acta Orthop Scand* 1969;40:234–236.
278. Babin SR, Graf P, Vidal P, et al. The risk of nonunion following closed-focus nailing and reaming. Results of 1059 interventions using the Kuntscher method. *Int Orthop* 1983;7:133–143.
279. Babin SR, Steinmetz A, Wuyts JL, et al. A reliable orthopedic technic in the treatment of humeral diaphyseal fractures in the adult: the hanging plaster. Report of a series of 74 cases. *J Chir (Paris)* 1978;115:653–658.
280. Balfour GW, Mooney V, Ashby ME. Diaphyseal fractures of the humerus treated with a ready-made fracture brace. *J Bone Joint Surg [Am]* 1982;64:11–13.
281. Barnard BB, McCoy SM. The supracondyloid process of the humerus. *J Bone Joint Surg [Am]* 1946;28:845–850.
282. Barquet A, Fernandez A, Luvizio J, et al. A combined therapeutic protocol for aseptic nonunion of the humeral shaft: a report of 25 cases. *J Trauma* 1989;29:95–98.
283. Barquet A, Schimchak M, Carreras O, et al. Dislocation of the shoulder with fracture of the ipsilateral shaft of the humerus. *Injury* 1985;16:300–302.
284. Bay BH, Sit KH, Lee ST. Mechanisms of humoral fractures in arm-wrestlers [Letter; Comment]. *Br J Clin Pract* 1992;46:98–99 and *Br J Clin Pract* 1993;47:279–280.
285. Beaty JH. Fractures of the proximal humerus and shaft in children. *Instr Course Lect* 1992;41:369–372.
286. Bell CH. Construction of orthoplast splints for humeral shaft fractures. *Am J Occup Ther* 1979;33:114–115.
287. Bell MJ, Beauchamp CG, Kellam JK, et al. The results of plating humeral shaft fractures in patients with multiple injuries. The Sunnybrook experience. *J Bone Joint Surg [Br]* 1985;67:293–296.
288. Beringer DC, Weiner DS, Noble JS, et al. Severely displaced proximal humeral epiphyseal fractures: a follow-up study. *J Pediatr Orthop* 1998;18:31–37.
289. Bianco AJ, Schlein AP, Kruse RL, et al. Birth fractures. *Minn Med* 1972;55:471–474.
290. Birch R, Bonney G, Payan J, et al. Symposium: peripheral nerve injuries. *J Bone Joint Surg [Br]* 1986;68:2–21.
291. Bleeker WA, Nijsten MW, ten Duis HJ. Treatment of humeral shaft fractures related to associated injuries. A retrospective study of 237 patients. *Acta Orthop Scand* 1991;62:148–153.
292. Böhler L. *The treatment of fractures*. New York: Grune & Stratton, 1956:618–694.

293. Böhler L. Conservative treatment of fresh closed fractures of the shaft of the humerus. *J Trauma* 1965;464.
294. Böhler L. *The treatment of fractures—supplement*. New York: Grune & Stratton, 1966.
295. Bortel DT, Pritchett JW. Straight-line graphs for the predictions of growth of the upper extremities. *J Bone Joint Surg [Am]* 1993;75:885–892.
296. Bostman O, Bakalim G, Vainionpaa S, et al. Immediate radial nerve palsy complicating fracture of the shaft of the humerus: when is early exploration justified? *Injury* 1985;16:499–502.
297. Bostman O, Bakalim G, Vainionpaa S, et al. Radial palsy in shaft fracture of the humerus. *Acta Orthop Scand* 1986;57:316–319.
298. Bransby-Zachary MA, MacDonald DA, Singh I, et al. Late fracture associated with retained internal fixation. *J Bone Joint Surg [Br]* 1989;71:539.
299. Bretagne MC, Mouton JN, Pierson M, et al. Periostitis or, rather, periosteal appositions in paediatrics. *J Radiol Electrol Med Nuc.* 1977;58:119–123.
300. Brien WW, Gellman H, Becker V, et al. Management of fractures of the humerus in patients who have an injury of the ipsilateral brachial plexus. *J Bone Joint Surg [Am]* 1990;72:1208–1210.
301. Broyn T, Bie K. Peripheral arterial occlusion following traumatic intimal rupture. *Acta Chir Scand* 1966;131:167–170.
302. Brug E, Winckler S, Klein W. Distal diaphyseal fracture of the humerus. *Unfallchirurgie* 1994;97:74–77.
303. Brumback R, Bosse M, Poka A, et al. Intramedullary stabilization of humeral shaft fractures in patients with multiple trauma. *J Bone Joint Surg [Am]* 1986;68:960–970.
304. Buachidze OS, Onoprienko GA, Shternberg AA, et al. Treatment of diaphyseal pseudarthrosis with transosseous osteosynthesis. *Vestn Khir Im I I Grek* 1977;119:84–87.
305. Caldwell JA. Treatment of fractures in the Cincinnati General Hospital. *Ann Surg* 1933;97:16.
306. Camus M, Lefebvre G, Veron P, et al. Obstetrical injuries of the newborn infant. Retrospective study apropos of 20,409 births. *J Gynecol Obstet Biol Reprod (Paris)* 1985;14:1033–1043.
307. Canale ST, Puhl J, Watson FM, et al. Acute osteomyelitis following closed fractures. Report of 3 cases. *J Bone Joint Surg [Am]* 1975;57:415.
308. Carroll SE. A study of the nutrient foramina of the humeral diaphysis. *J Bone Joint Surg [Br]* 1963;45:176–181.
309. Casadei R, Ferraro A, Ferruzzi A, et al. Supracondylar process of the humerus: four cases. *Chir Organi Mov* 1990;75:265–277.
310. Cattaneo R, Catagni MA, Guerreschi F. Applications of the Ilizarov method in the humerus. Lengthenings and nonunions. *Hand Clin* 1993;9:729–739.
311. Cattaneo R, Villa A, Catagni MA, et al. Lengthening of the humerus using the Ilizarov technique. Description of the method and report of 43 cases. *Clin Orthop* 1990;250:117–124.
312. Chacha PB. Compression plating without bone grafts for delayed and nonunion of humeral shaft fractures. *Injury* 1973;5:283–290.
313. Champetier J, Brabant A, Charignon G, et al. Treatment of fractures of the humerus by intramedullary fixation. *J Chir (Paris)* 1975;109:75–82.
314. Chan D, Petricciuolo F, Maffulli N. Fracture of the humeral diaphysis with extreme rotation. *Acta Orthop Belg* 1991;57:427–429.
315. Chapman MW. Closed intramedullary nailing of the humerus. *Instr Course Lect* 1983;32:324–328.
316. Cheng JC, Shen WY. Limb fracture pattern in different pediatric age groups: a study of 3350 children. *J Orthop Trauma* 1993;7:15–22.
317. Chitwood WR Jr, Rankin JS, Bollinger RR, et al. Brachial artery reconstruction using the heparin-bonded Sundt shunt. *Surgery* 1981;89:355–358.
318. Choong PF, Griffiths JD. External fixation of complex open humeral fractures. *Aust NZ J Surg* 1988;58:137–142.
319. Christensen NO. Kuntscher intramedullary reaming and nail fixation for nonunion of the humerus. *Clin Orthop* 1976;116:222–225.
320. Ciernik IF, Meier L, Hollinger A. Humeral mobility after treatment with hanging cast. *J Trauma* 1991;31:230–233.
321. Ciuccarelli C, Cervellati C, Montanari G, et al. The Ilizarov method for the treatment of nonunion in the humerus. *Chir Organi Mov* 1990;75:115–120.
322. Colyer RA. Surgical stabilization of pathological neoplastic fractures. *Curr Probl Cancer* 1986;10:117–168.
323. Confalonieri N, Simonatti R, Ramondetta V, et al. Intramedullary nailing with a rush pin in the treatment of diaphyseal humeral fractures. *Arch Putti Chir Organi Mov* 1990;38:395–403.
324. Connolly JF. Selection, evaluation and indications for electrical stimulation of ununited fractures. *Clin Orthop* 1981;161:39–53.
325. Costa P, Giancetti F, Cavazzuti A. Internal and external fixation in complex diaphyseal and metaphyseal fractures of the humerus. *Ital J Orthop Traumatol* 1991;17:87–94.
326. Crolla RM, de Vries LS, Clevers GJ. Locked intramedullary nailing of humeral fractures. *Injury* 1993;24:403–406.
327. Curtis RJJ, Dameron TB Jr, Rockwood CA Jr, et al., eds. *Fractures in children*, 3rd ed. Philadelphia: JB Lippincott, 1991:829–919.
328. D'Ythurbide B, Augereau B, Asselineau A, et al. Closed intramedullary nailing of fractures of the shaft of the humerus. *Int Orthop* 1983;7:195–203.
329. Dabiezies EJ, Banta CJ 2nd, Murphy CP, et al. Plate fixation of the humeral shaft for acute fractures, with and without radial nerve injuries. *J Orthop Trauma* 1992;6:10–13.
330. Dal Monte A, Andrisano A, Manfrini M, et al. Humeral lengthening in hypoplasia of the upper limb. *J Pediatr Orthop* 1985;5:207.
331. Dameron TB Jr. Transverse fractures of distal humerus in children. *Instr Course Lect* 1981;30:224–235.
332. Dameron TB Jr, Grubb SA. Humeral shaft fractures in adults. *South Med J* 1981;74:1461–1467.
333. De Bastiani G, Aldegheri R, Renzi Brivio L. The treatment of fractures with a dynamic axial fixator. *J Bone Joint Surg [Br]* 1984;66:538–545.
334. de Mourgues G, Fischer LP, Gillet JP, et al. Recent fractures of the humeral diaphysis apropos of a continuous series of 200 cases, of which 107 were treated with a hanging cast alone. *Rev Chir Orthop Reparatrice Appar Mot* 1975;61:191–207.
335. DeLong WG Jr, Born CT, Marcelli E, et al. Ender nail fixation in long bone fractures: experience in a level I trauma center. *J Trauma* 1989;29:571–576.
336. Destree C, Safary A. The treatment of fractures of the neck and diaphysis of the humerus by Hackethal's bundle nailing. *Acta Orthop Belg* 1979;45:666–677.
337. Devas MB. Stress fractures in athletes. *Proc R Soc Med* 1969;62:933–937.
338. Di Filippo P, Mancini GB, Gillio A. Humeral fractures with paralysis of the radial nerve. *Arch Putti Chir Organi Mov* 1990;38:405–409.
339. DiCicco JD, Mehlman CT, Urse JS. Fracture of the shaft of the humerus secondary to muscular violence. *J Orthop Trauma* 1993;7:90–93.
340. Dufour O, Beaufils P, Ouaknine M, et al. Functional treatment of recent fractures of the humeral shaft using the Sarmiento method. *Rev Chir Orthop Reparatrice Appar Mot* 1989;75:292–300.
341. Durbin RA, Gottesman MJ, Saunders KC. Hackethal stacked nailing of humeral shaft fractures. Experience with 30 patients. *Clin Orthop* 1983;179:168–174.
342. Duthie HL. Radial nerve in osseous tunnel at humeral fracture site diagnosed radiographically. *J Bone Joint Surg [Br]* 1957;39:746–747.
343. Ellefsen BK, Frierson MA, Raney EM, et al. Humerus varus: a complication of neonatal, infantile, and childhood injury and infection. *J Pediatr Orthop* 1994;14:479–486.
344. Epps CH. Nonunion of the humerus. *Instr Course Lect* 1988;37:161–166.
345. Esterhai JL Jr, Brighton CT, Heppenstall RB, et al. Nonunion of the humerus. Clinical, roentgenographic, scintigraphic, and response characteristics to treatment with constant direct current stimulation of osteogenesis. *Clin Orthop* 1986;211:228–234.
346. Fattah HA, Halawa EE, Shafy TH. Nonunion of the humeral shaft: a report on 25 cases. *Injury* 1982;14:255–262.
347. Fisher TR, McGeorch CM. Severe injuries of the radial nerve treated by sural nerve grafting. *Injury* 1985;16:411–412.
348. Foster RJ, Dixon GL Jr, Bach AW, et al. Internal fixation of fractures and nonunions of the humeral shaft. Indications and results in a multicenter study. *J Bone Joint Surg [Am]* 1985;67:857–864.
349. Foster RJ, Swiontkowski MF, Bach AW, et al. Radial nerve palsy caused by open humeral shaft fractures. *J Hand Surg [Am]* 1993;18:121–124.
350. Fourastier J, Pialoux B, Bracq H, et al. Post-traumatic algodystrophy in children. *Chir Pediatr* 1986;27:313–317.
351. Franklyn PP. Progeria in siblings. *Clin Radio* 1976;27:327–333.
352. Friedman RJ, Smith RJ. Radial nerve laceration 26 years after screw fixation of a humeral fracture—a case report. *J Bone Joint Surg [Am]* 1984;66:959–960.
353. Gainor BJ, Metzler M. Humeral shaft fracture with brachial artery injury. *Clin Orthop* 1986;204:154–161.
354. Galasko CS. The fate of simple bone cysts which fracture. *Clin Orthop* 1974;101:302–304.
355. Gamble JG, Rinsky LA, Strudwick J, et al. Nonunion of fractures in children who have osteogenesis imperfecta. *J Bone Joint Surg [Am]* 1988;70:439–443.
356. Garcia A, Maecck BH. Radial nerve injuries in fractures of the shaft of the humerus. *Am J Surg* 1960;99:625–627.
357. Gardner E. Prenatal development of the human shoulder joint. *Surg Clin North Am* 1953;92:219–276.
358. Garland DE, Jones RC, Kunkle RW. Upper extremity fractures in the acute spinal cord injured patient. *Clin Orthop* 1988;233:110–115.
359. Garth WP Jr, Leberte MA, Cool TA. Recurrent fractures of the humerus in a baseball pitcher—a case report. *J Bone Joint Surg [Am]* 1988;70:305–306.
360. Gibson MJ, Middlemiss JH. Fibrous dysplasia of bone. *Br J Radiol* 1971;44:1–13.
361. Gjengedal E, Slungaard U. Treatment of humeral fractures with and without injury to the radial nerve—a follow-up study. *Tidsskr Nor Laegeforen* 1981;101:1746–1749.
362. Goodsell JO. The resilient radial nerve. *Mich Med* 1965;64:756–758.
363. Gore RM, Rogers LF, Bowerman J, et al. Osseous manifestations of elbow stress associated with sports activities. *AJR* 1980;134:971–977.
364. Gray DJ, Gardner E. The prenatal development of the human humerus. *Am J Anat* 1969;124:431–434.
365. Green SA, Gibbs P. The relationship of angulation to translation in fracture deformities. *J Bone Joint Surg [Am]* 1994;76:390–397.
366. Gregersen HN. Fractures of the humerus from muscular violence. *Acta Orthop Scand* 1971;42:506–512.
367. Gupta A, Sharma S. Volar compartment syndrome of the arm complicating a fracture of the humeral shaft—a case report. *Acta Orthop Scand* 1991;62:77–78.
368. Gupta RC, Gaur SC, Tiwari RC, et al. Treatment of ununited fractures of the shaft of the humerus with bent nail. *Injury* 1985;16:276–280.
369. Haagedoorn EL. Fracture of the supracondylar humeral process. *Ned Tijdschr Geneeska* 1968;112:313–316.
370. Hackethal KH. *Die Bundel-Nagelung*. Berlin: Springer-Verlag, 1961.
371. Hackstock H. Functional bracing of fractures. *Orthopadie* 1988;17:41–51.
372. Hall RF Jr. Closed intramedullary fixation of humeral shaft fractures. *Instr Course Lect* 1987;36:349–358.
373. Hall RFJ, Pankovich AM. Ender nailing of acute fractures of the humerus. A study of closed fixation by intramedullary nails without reaming. *J Bone Joint Surg [Am]* 1987;69:558–567.
374. Healy WL, White GM, Mick CA, et al. Nonunion of the humeral shaft. *Clin Orthop* 1987;219:206–213.
375. Hedstrom O. Growth stimulation of long bones after fracture or similar trauma. A clinical and experimental study. *Acta Orthop Scand Suppl* 1969;122:1–134.
376. Heilbronner DM, Manoli A 2nd, Little RE. Elbow dislocation during overhead skeletal traction therapy: a case report. *Clin Orthop* 1981;154:185–187.
377. Heim D, Herkert F, Hess P, et al. Can humerus shaft fractures be treated with osteosynthesis? *Helv Chir Acta* 1992;58:673–678.
378. Heim D, Herkert F, Hess P, et al. Surgical treatment of humeral shaft fractures—the Basel experience. *J Trauma* 1993;35:226–232.
379. Henley MB, Monroe M, Tencer AF. Biomechanical comparison of methods of fixation of a midshaft osteotomy of the humerus. *J Orthop Trauma* 1991;5:14–20.
380. Hennig F, Link W, Wofel R. Bundle nailing—an evaluation after 27 years. *Aktuel Traumatol* 1988;18:117–119.
381. Hennigan SP, Bush-Joseph CA, Kuo KN, et al. Throwing-induced humeral shaft fracture in skeletally immature adolescents. *Orthopedics* 1999;22:621–622.
382. Hermichen HG, Pfister U, Weller S. Influence of the treatment of fractures on the development of pseudoarthroses of the humerus shaft. *Aktuel Traumatol* 1980;10:137–142.
383. Hollingshead WH. *Anatomy for surgeons: the back and limbs*. New York: Harper & Row, 1982.

384. Holm CL. Management of humeral shaft fractures. Fundamental nonoperative technics. *Clin Orthop* 1970;71:132–139.
385. Holstein A, Lewis GB. Fractures of the humerus with radial nerve paralysis. *J Bone Joint Surg [Am]* 1963;45:1382–1388.
386. Hosner W. Fractures of the shaft of the humerus: an analysis of 100 consecutive cases. *Reconstr Surg Traumatol* 1974;14:38–64.
387. Howard NJ, Eloesser L. Treatment of fracture of the upper end of the humerus: an experimental and clinical study. *J Bone Joint Surg [Am]* 1934;16:1–29.
388. Hunter SG. The closed treatment of fractures of the humeral shaft. *Clin Orthop* 1982;164:192–198.
389. Ilizarov GA. *Transosseous osteosynthesis*. Berlin: Springer-Verlag, 1992.
390. Ilizarov GA, Shevtsov VI. Bloodless compression–distraction osteosynthesis in the treatment of pseudarthroses of the humerus. *Voen Med Zh* 1974;27–31.
391. Ingman AM, Waters DA. Locked intramedullary nailing of humeral shaft fractures. Implant design, surgical technique, and clinical results. *J Bone Joint Surg [Br]* 1994;76:23–29.
392. Iqbal QM. Long-bone fractures among children in Malaysia. *Int Surg* 1974;59:410–415.
393. Johnstone DJ, Radford WJ, Parnel EJ. Interobserver variation using the AO/ASIF classification of long-bone fractures. *Injury* 1993;24:163–165.
394. Kamhin M, Michaelson M, Waisbrod H. The use of external skeletal fixation in the treatment of fractures of the humeral shaft. *Injury* 1978;9:245–248.
395. Kane E, Kaplan EB, Spinner M. Observations of the course of the ulnar nerve in the arm. *Ann Chir* 1973;27:487–496.
396. Kim NH, Hahn SB, Park HW, et al. The Orthofix external fixator for fractures of long bones. *Int Orthop* 1994;18:42–46.
397. Klenerman L. Fractures of the shaft of the humerus. *J Bone Joint Surg [Br]* 1966;48B:105–111.
398. Koch G. Treatment of humeral fractures using the U splint. *Chirurgie* 1971;42:327–329.
399. Kolb LW, Moore RD. Fractures of the supracondylar process of the humerus. Report of 2 cases. *J Bone Joint Surg [Am]* 1967;49A:532–534.
400. Kretzschmar HJ. Post-traumatic fat embolism in a 13-year-old girl. *Zentralbl Chir* 1970;95:1223–1225.
401. Kulenkampff HA, Rustemeier M. Clinical experiences in the treatment of humeral shaft fractures with the Sarmiento brace. *Unfallchirurgie* 1988;14:191–198.
402. Kwasny O, Maier R, Kutscha-Lissberg F, et al. Treatment procedure in humeral shaft fractures with primary or secondary radial nerve damage. *Unfallchirurgie* 1992;18:168–173.
403. Kwasny O, Maier R, Scharf W. The surgical treatment of humeral shaft fractures. *Aktuel Traumatol* 1990;20:87–92.
404. Landin LA. Fracture patterns in children: analysis of 8682 fractures with special reference to incidence, etiology and secular changes in Swedish urban populations. *Acta Orthop Scand Suppl* 1983;54:1–109.
405. Lange RH, Foster RJ. Skeletal management of humeral shaft fractures associated with forearm fractures. *Clin Orthop* 1985;195:173–177.
406. Langenberg R. Missed humeral fracture in multiple injury of the arm. *Zentralbl Chir* 1986;111:1536–1539.
407. Langenberg R. Sarmiento functional treatment of humeral shaft fractures. Results and experiences. *Zentralbl Chir* 1987;112:1271–1277.
408. Leung KS, Kwan M, Wong J, et al. Therapeutic functional bracing in upper limb fracture–dislocations. *J Orthop Trauma* 1999;2:308–313.
409. Levin LS, Goldner RD, Urbaniak JR, et al. Management of severe musculoskeletal injuries of the upper extremity. *J Orthop Trauma* 1990;4:432–440.
410. Lewallen RP, Peterson HA. Nonunion of long bone fractures in children: a review of 30 cases. *J Pediatr Orthop* 1985;5:135–142.
411. Lewallen RP, Pritchard DJ, Sim FH. Treatment of pathologic fractures or impending fractures of the humerus with rush rods and methylmethacrylate. Experience with 55 cases in 54 patients, 1968–1977. *Clin Orthop* 1982;166:93–198.
412. Lichtenberg RP. A study of 2532 fractures in children. *Am J Surg* 1954;87:330–338.
413. Ligier JN, Metaizeau JP, Prevot J. Closed flexible medullary nailing in pediatric traumatology. *Chir Pediatr* 1983;24:383–385.
414. Link W, Herzog T, Hoffmann A. Bundle wire nailing in pathological upper arm fractures. *Zentralbl Chir* 1990;115:665–670.
415. Linn RM, Kerigshausen LA. Ball-thrower's fracture of the humerus—a case report. *Am J Sports Med* 1991;19:194–197.
416. Loder RT. Pediatric polytrauma: orthopaedic care and hospital course. *J Orthop Trauma* 1987;1:48–54.
417. Loder RT, Bookout C. Fracture patterns in battered children. *J Orthop Trauma* 1991;5:428–433.
418. Low BY, Lim J. Fracture of humerus during arm-wrestling: report of 5 cases. *Singapore Med J* 1991;32:47–49.
419. Lund HJ. Fracture of the supracondylar process of the humerus. Report of a case. *J Bone Joint Surg [Am]* 1930;12:925–928.
420. Machan FG, Vinz H. Humeral shaft fracture in childhood. *Unfallchirurgie* 1993;19:166–174.
421. Mackay I. Closed Rush pinning of fractures of the humeral shaft. *Injury* 1984;16:178–181.
422. Macnicol MF. Roentgenographic evidence of median-nerve entrapment in a greenstick humeral fracture. *J Bone Joint Surg [Am]* 1978;60:998–1000.
423. Madsen TE. Fractures of the extremities in the newborn. *Acta Obstet Gynecol Scand* 1955;34:4.
424. Makin GS, Howard JM, Green RL. Arterial injuries complicating fractures or dislocations: the necessity for a more aggressive approach. *Surgery* 1966;59:203–209.
425. Marcus RE, Mills MF, Thompson GH. Multiple injury in children. *J Bone Joint Surg [Am]* 1983;65:1290–1294.
426. Marty B, Kach K, Candinas D, et al. Results of intramedullary nailing in humerus shaft fractures. *Helv Chir Acta* 1993;59:681–685.
427. Mast JW, Spiegel PG, Harvey JP, et al. Fractures of the humeral shaft. A retrospective study of 240 adult fractures. *Clin Orthop* 1975;112:254–262.
428. McNamara JJ, Brief DK, Stremple JF, et al. Management of fractures with associated arterial injury in combat casualties. *J Trauma* 1973;13:17–19.
429. McQuillan WM, Nolan B. Ischemia complicating injury. *J Bone Joint Surg [Am]* 1968;50:482–492.
430. Menger DM, Gauger JU, Schmitt-Koppler A. Experiences with cluster nailing of humeral shaft fractures. *Unfallchirurgie* 1985;11:70–75.
431. Metaizeau JP, Ligier JN. Surgical treatment of fractures of the long bones in children. Interference between osteosynthesis and the physiological processes of consolidation. Therapeutic indications. *J Chir (Paris)* 1984;121:527–537.
432. Michiels I, Broos P, Gruwez JA. The operative treatment of humeral shaft fractures. *Acta Chir Belg* 1986;86:147–152.
433. Moon MS, Kim I, Han IH, et al. Arm-wrestler's injury: report of 7 cases. *Clin Orthop* 1977;147:219–221.
434. Mubarak SJ, Carroll NC. Volkman's contracture in children: aetiology and prevention. *J Bone Joint Surg [Br]* 1979;61:285–293.
435. Müller ME, Allgöwer M, Schneider R, et al. *Manual of internal fixation. Techniques recommended by the AO-ASIF group*, 3rd ed. Berlin: Springer-Verlag, 1991.
436. Nast-Kolb D, Knoefel WT, Schweiberer L. The treatment of humeral shaft fractures. Results of a prospective AO multicenter study. *Unfallchirurgie* 1991;94:447–454.
437. Naver L, Aalberg JR. Humeral shaft fractures treated with a ready-made fracture brace. *Arch Orthop Trauma Surg* 1986;106:20–22.
438. Nerubay J, Horoszowski H, Goodman RM. Fracture in progressive ossifying fibrodysplasia—a case report. *Acta Orthop Scand* 1987;58:289–291.
439. Newman A. The supracondylar process and its fracture. *Am J Roentgenol Radium Ther Nucl Med* 1969;105:844–849.
440. Nonnemann HC. Limits of spontaneous correction of incorrectly healed fractures in adolescence. *Langenbecks Arch Chir* 1969;324:78–86.
441. O'Neill JA Jr, Meacham WF, Griffin JP, et al. Patterns of injury in the battered child syndrome. *J Trauma* 1973;13:332–339.
442. Olszewski W, Popinski M. Fractures of the neck and shaft of the humerus as a rare form of double fractures in children. *Chir Narzadow Ruchu Ortop Poi* 1974;39:121–123.
443. Packer JW, Foster RR, Garcia A, et al. The humeral fracture with radial nerve palsy: is exploration warranted? *Clin Orthop* 1972;88:34–38.
444. Panitz K, Neundorfer B, Piotrowski W. Prognosis of nerve injuries in humeral fractures. *Chirurgie* 1975;46:392–394.
445. Peeters PM, Oostvogel HJ, Bongers KJ, van der Werken C. Early functional treatment of humerus shaft fractures by the Sarmiento method. *Aktuel Traumatol* 1987;17:150–152.
446. Peter RE, Hoffmeyer P, Henley MB. Treatment of humeral diaphyseal fractures with Hackethal stacked nailing: a report of 33 cases. *J Orthop Trauma* 1992;6:14–17.
447. Peterson HA. Surgical lengthening of the humerus: case report and review. *J Pediatr Orthop* 1989;9:596–601.
448. Pierce RO Jr, Hodurski DF. Fractures of the humerus, radius, and ulna in the same extremity. *J Trauma* 1979;19:182–185.
449. Pollock FH, Drake D, Bovill EG, et al. Treatment of radial neuropathy associated with fractures of the humerus. *J Bone Joint Surg [Am]* 1981;63:239–243.
450. Postacchini F, Morace GB. Fractures of the humerus associated with paralysis of the radial nerve. *Ital J Orthop Traumatol* 1988;14:455–465.
451. Pradhan DJ, Juanteguy JM, Wilder RJ, et al. Arterial injuries of the extremities associated with fractures. *Arch Surg* 1972;105:582–585.
452. Pritchett JW. Delayed union of humeral shaft fractures treated by closed flexible intramedullary nailing. *J Bone Joint Surg [Br]* 1985;67:715–718.
453. Pritchett JW. Growth and predictions of growth in the upper extremity. *J Bone Joint Surg [Am]* 1988;70:520–525.
454. Pritchett JW. Growth plate activity in the upper extremity. *Clin Orthop* 1991;268:235–242.
455. Putnam MD, Walsh TM. External fixation for open fractures of the upper extremity. *Hand Clin* 1993;9:613–623.
456. Putz P, Lusi K, Baillon JM, Bremen J. The treatment of fractures of the humeral diaphysis with fasciculated intramedullary pins by the Hackethal method apropos of 194 cases. *Acta Orthop Belg* 1984;50:521–538.
457. Rang M. *Anonymous children's fractures*, 2nd ed. Philadelphia: JB Lippincott, 1983:154–151.
458. Rettig AC, Beltz HF. Stress fracture in the humerus in an adolescent tennis tournament player. *Am J Sports Med* 1985;13:55–58.
459. Ricciardi-Pollini PT, Falez F. The treatment of diaphyseal fractures by functional bracing. Results in 36 cases. *Ital J Orthop Traumatol* 1985;11:199–205.
460. Rich NM, Metz CW Jr, Hutton JE Jr, et al. Internal versus external fixation of fractures with concomitant vascular injuries in Vietnam. *J Trauma* 1971;11:463–473.
461. Riemer BL, Foglesong ME, Burke CJ 3rd, et al. Complications of Seidel intramedullary nailing of narrow-diameter humeral diaphyseal fractures. *Orthopaedics* 1994;17:19–29.
462. Riska EB, von Bonsdorff H, Hakkinen S, et al. Primary operative fixation of long-bone fractures in patients with multiple injuries. *J Trauma* 1977;17:111–121.
463. Rogalsky RJ, Black GR, Reed MH. Orthopaedic manifestations of leukemia. *J Bone Joint Surg [Am]* 1986;68:494–501.
464. Rogers JF, Bennett JB, Tullos HS. Management of concomitant ipsilateral fractures of the humerus and forearm. *J Bone Joint Surg [Am]* 1984;66:552–556.
465. Rommens PM, Vansteenkiste F, Stappaerts KH, et al. Indications, dangers and results of surgical treatment of humeral shaft fractures. *Unfallchirurgie* 1989;92:565–570.
466. Rommens PM, Vergruggen J, Broos PL. Retrograde locked nailing of humeral shaft fractures. *J Bone Joint Surg [Br]* 1995;77:84–89.
467. Rose SH, Melton LJ 3rd, Morrey BF, et al. Epidemiologic features of humeral fractures. *Clin Orthop* 1982;168:24–30.
468. Rüedi T, Moshfegh A, Pfeiffer KM, et al. Fresh fractures of the shaft of the humerus—conservative or operative treatment? *Reconstr Surg Traumatol* 1974;14:65–74.
469. Sales de Gauzy J, Vidal H, Cahuzac JP. Primary shortening followed by callus distraction for the treatment of a post-traumatic bone defect: case report. *J Trauma* 1993;34:461–463.
470. Samardzic M, Grujicic D, Milinkovic ZB. Radial nerve lesions associated with fractures of the humeral shaft. *Injury* 1990;21:220–222.
471. Sarmiento A. Functional fracture bracing: an update. *Instr Course Lect* 1987;36:371–376.
472. Sarmiento A, Horowitz A, Abouafia A, et al. Functional bracing for comminuted extra-articular fractures of the distal third of the humerus. *J Bone Joint Surg [Br]* 1990;72:283–287.
473. Sarmiento A, Kinman PB, Galvin EG, et al. Functional bracing of fractures of the shaft of the humerus. *J Bone Joint Surg [Am]* 1977;59:596–601.

474. Sattel W. Effect of dia- and percondylar humeral fractures on the growth of the carpal bones in children. *Handchir Mikrochir Plast Chir* 1982;14:103–105.
475. Seddon HJ. Nerve lesions complicating certain closed bone injuries. *JAMA* 1947;135:691–694.
476. Seddon HJ, Medawar PB, Smith H. Rate of regeneration of peripheral nerves in man. *J Physiol (Paris)* 1943;102:191–215.
477. Sedel L, Christel P, Duriez J, et al. Acceleration of repair of nonunions by electromagnetic fields. *Rev Chir Orthop Reparatrice Appar Mot* 1981;67:11–23.
478. Sedel L, Christel P, Duriez J, et al. Results of nonunions treatment by pulsed electromagnetic field stimulation. *Acta Orthop Scand Suppl* 1982;196:81–91.
479. Shah JJ, Bhatti NA. Radial nerve paralysis associated with fractures of the humerus: a review of 62 cases. *Clin Orthop* 1983;172:171–176.
480. Shantharam SS. Tips of the trade: 41. Modified coaptation splint for humeral shaft fractures. *Orthop Rev* 1991;20:1033–1039.
481. Sharma VK, Jain AK, Gupta RK, et al. Nonoperative treatment of fractures of the humeral shaft: a comparative study. *J Indian Med Assoc* 1991;89:157–160.
482. Shaw BA, Murphy KM, Shaw A, et al. Humerus shaft fractures in young children: accident or abuse? *J Pediatr Orthop* 1997;17:293–297.
483. Shaw JL, Sakellarides H. Radial nerve paralysis associated with fractures of the humerus. A review of 45 cases. *J Bone Joint Surg [Am]* 1967;49:899–902.
484. Shevtsov VI. Outcome of treatment of pseudarthroses of the humerus by methods of closed perosseous osteosynthesis by the Ilizarov technic. *Sov Med* 1977;75–79.
485. Shumada IV, Zhila IS, Rybachuk OI. Compression–distraction osteosynthesis in pseudarthroses of long tubular bones. *Vestn Khir Im I I Grek* 1976;116:90–94.
486. Smith DK, Cooney WP. External fixation of high-energy upper extremity injuries. *J Orthop Trauma* 1990;4:7–18.
487. Smith RF, Szilagyi E, Elliott JP Jr. Fracture of long bones with arterial injury due to blunt trauma. Principles of management. *Arch Surg* 1969;99:315–324.
488. Sonneveld GJ, Patka P, van Mourik JC, et al. Treatment of fractures of the shaft of the humerus accompanied by paralysis of the radial nerve. *Injury* 1987;18:404–406.
489. Spak I. Humeral shaft fractures. Treatment with a simple hand sling. *Acta Orthop Scand* 1978;49:234–239.
490. Sprenger TR. Fracture of humerus from muscular violence: case report. *J Fla Med Assoc* 1985;72:101–103.
491. Stanitski CL, Micheli LJ. Simultaneous ipsilateral fractures of the arm and forearm in children. *Clin Orthop* 1980;153:218–222.
492. Stephenson RB, London MD, Harkan FM, et al. Fibrous dysplasia. an analysis of options for treatment. *J Bone Joint Surg [Am]* 1987;69:400–409.
493. Sterling JC, Calvo RD, Holden SC. An unusual stress fracture in a multiple sport athlete. *Med Sci Sports Exerc* 1991;23:298–303.
494. Stern PJ, Mattingly DA, Pomeroy DL, et al. Intramedullary fixation of humeral shaft fractures. *J Bone Joint Surg [Am]* 1984;66:639–646.
495. Stewart MJ, Hundley JM. Fractures of the humerus: a comparative study in methods of treatment. *J Bone Joint Surg [Am]* 1955;37:681–692.
496. Stimson LA. *A practical treatise on fractures and dislocations*, 3rd ed. New York: Lea Brothers & Co, 1900.
497. Sturmer KM, Schmit-Neuerburg KP. Indications and clinical results of electromagnetically induced alternating current stimulation of poorly reacting pseudarthroses. *Unfallchirurgie* 1985;11:197–203.
498. Sunderland S. Rate of regeneration in human peripheral nerves. *Arch Neurol Psych* 1947;58:251–295.
499. Szalay EA, Rockwood CA. Fractures of the distal shaft of the humerus associated with radial nerve palsy. *Orthop Trans* 1982;6:455.
500. Thomas IH, Chow CW, Cole WG. Giant cell reparative granuloma of the humerus. *J Pediatr Orthop* 1988;8:596–598.
501. Titze A. The operative treatment of fractures of the shaft of the humerus. *Reconstr Surg Traumatol* 1974;14:75–83.
502. Tullos HS, Erwin WD, Woods GW, et al. Unusual lesions of the pitching arm. *Clin Orthop* 1972;88:169–182.
503. Tullos HS, King JW. Lesions of the pitching arm in adolescents. *JAMA* 1972;220:264–271.
504. Vail TP, Harrelson JM. Treatment of pathologic fracture of the humerus. *Clin Orthop* 1991;268:197–202.
505. van der Ghinst M, De Geeter L, Thiry A. Treatment of collum and diaphyseal fractures of the humerus using Kirschner wire nailing. *Acta Chir Belg* 1972;(suppl 2):105–116.
506. Vander Griend R, Tomasin J, Ward EF. Open reduction and internal fixation of humeral shaft fractures. Results using AO plating techniques. *J Bone Joint Surg [Am]* 1986;68:430–433.
507. Vander Griend RA, Ward EF, Tomasin J. Closed Kuntscher nailing of humeral shaft fractures. *J Trauma* 1985;25:1167–1169.
508. Vastamaki M, Solonen KA. Posterior dislocation and fracture–dislocation of the shoulder. *Acta Orthop Scand* 1980;51:479–484.
509. Vichare NA. Fractures of the humeral shaft associated with multiple injuries. *Injury* 1974;5:279–282.
510. Vinz H. Treatment of open fractures in children. *Zentralbl Chir* 1980;105:1483–1493.
511. Vukadinovic S, Mikic Z, Lartey J. Humeral fractures complicated by radial nerve lesions. *Acta Chir Iugos*. 1981;28:211–217.
512. Watson FM, Whitesides TE Jr. Acute hematogenous osteomyelitis complicating closed fractures. *Clin Orthop* 1976;117:296–302.
513. Webb LX, Green NE, Swiontkowski MF, eds. *Skeletal trauma in children*. Philadelphia: WB Saunders, 1993:257–281.
514. Weseley MS, Barenfeld PA. Ball throwers' fracture of the humerus. *Clin Orthop* 1969;64:153–156.
515. Whitson RO. Relation of the radial nerve to the shaft of the humerus. *J Bone Joint Surg [Am]* 1954;36:85–88.
516. Winfield JM, Miller H, LaFerte AD. Evaluation of the "hanging cast" as a method of treating fractures of the humerus. *Am J Surg* 1942;55:228–249.
517. Winker H, Vosberg W, Cyris A. Results of treatment of humerus shaft fractures. *Aktuel Traumatol* 1993;23(suppl):36–41.
518. Worlock P, Stower M. Fracture patterns in Nottingham children. *J Pediatr Orthop* 1986;6:656–661.
519. Wright TW, Miller GJ, Vander Griend RA, et al. Reconstruction of the humerus with an intramedullary fibular graft. A clinical and biomechanical study. *J Bone Joint Surg [Br]* 1993;75:804–807.
520. Zagorski JB, Latta LL, Zych GA, et al. Diaphyseal fractures of the humerus. treatment with prefabricated braces. *J Bone Joint Surg [Am]* 1988;70:607–610.
521. Zanasi R, Romano P, Rotolo F, et al. Intramedullary osteosynthesis: 3. Kuntscher nailing in the humerus. *Ital J Orthop Traumatol* 1990;16:311–322.
522. Zifko B, Poigenfurst J. Treatment of unstable fractures of the proximal end of the humerus using elastic curved intramedullary wires. *Unfallchirurgie* 1987;13:72–81.
523. Zinghi GF, Sabetta E, Bungaro P, et al. The role of osteosynthesis in the treatment of fractures of the humerus. *Ital J Orthop Traumatol* 1988;14:67–75.

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524. Aamoth GM, O'Phelan EH. Recurrent anterior dislocation of the shoulder: a review of 40 athletes treated by subscapularis transfer (modified Magnuson-Stack procedure). *Am J Sports Med* 1977;5:188–190.
525. Aronen JG, Regan K. Decreasing the incidence of recurrence of first-time anterior shoulder dislocation with rehabilitation. *Am J Sports Med* 1984;12:283–291.
526. Asher MA. Dislocations of the upper extremity in children. *Orthop Clin North Am* 1976;7:583–591.
527. Babbitt DP, Cassidy RH. Obstetrical paralysis and dislocation of the shoulder in infancy. *J Bone Joint Surg [Am]* 1968;50A:1447–1452.
528. Bach FR, O'Brien SJ, Warren RF, et al. An unusual neurological complication of the Bristow procedure: a case report. *J Bone Joint Surg [Am]* 1988;70:458–460.
529. Barratta JB, Lim V, Mastro Monaco E, et al. Axillary artery disruption secondary to anterior dislocation of the shoulder. *J Trauma* 1983;23:1009–1011.
530. Barry TP, Lombardo SJ, Kerlan RK, et al. The coracoid transfer for recurrent anterior instability of the shoulder in adolescents. *J Bone Joint Surg [Am]* 1985;67:383–387.
531. Blazina ME, Satzman JS. Recurrent anterior subluxation of the shoulder in athletics—a distinct entity (proceedings). *J Bone Joint Surg [Am]* 1969;51:1037–1038.
532. Blom S, Dahlback LO. Nerve injuries in dislocations of the shoulder joint and fractures of the neck of the humerus. *Acta Chir Scand* 1970;136:461–466.
533. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955.
534. Boyd HB, Sisk TD. Recurrent posterior dislocation of the shoulder. *J Bone Joint Surg [Am]* 1972;54:779–786.
535. Burkhead WZ Jr, Rockwood CA Jr. Treatment of instability of the shoulder with an exercise program. *J Bone Joint Surg* 1993;75:311–312.
536. Carter C, Sweetnam R. Recurrent dislocation of the patella and of the shoulder: their association with familial joint laxity. *J Bone Joint Surg [Br]* 1960;42:721–727.
537. Chung SMK, Nissenbaum MM. Congenital and development defects of the shoulder. *Orthop Clin North Am* 1975;6:381–392.
538. Cleeman E, Flatow EL. Shoulder dislocations in the young patient. *Orthop Clin North Am* 2000;31:217–229.
539. Coene LN, Narakas AO. Operative management of lesions of the axillary nerve, isolated or combined with other nerve lesions. *Clin Neurol Neurosurg* 1992;94 (suppl):64–66.
540. Cozen L. Congenital dislocation of the shoulder and other anomalies. *Arch Surg* 1937;35:956–966.
541. Curr JF. Rupture of the axillary artery complicating dislocation of the shoulder: report of a case. *J Bone Joint Surg [Br]* 1970;52:313–317.
542. Dameron TB, Rockwood CA. Fractures and dislocations of the shoulder. In: Rockwood CA, Wilkins KE, King RE, eds. *Fractures in children*. Philadelphia: JB Lippincott, 1984: 659–676.
543. Davis AG. A conservative treatment for habitual dislocations of the shoulder. *JAMA* 1936;107:1012–1015.
544. DePalma AF, Cooke AJ, Probhakar M. The role of the subscapularis in recurrent anterior dislocations of the shoulder. *Clin Orthop* 1967;54:35–49.
545. DePalma AF, Silverstein CE. Results following a modified Magnuson Procedure in recurrent dislocation of the shoulder. *Surg Clin North Am* 1963;43:1651–1653.
546. Detenbeck LC. Posterior dislocations of the shoulder. *J Trauma* 1972;12:183–192.
547. Dimon JH III. Posterior dislocation and posterior fracture dislocation of the shoulder: a report of 25 cases. *South Med J* 1967;60:661–666.
548. O'Driscoll SW, Evans DC. Contralateral shoulder instability following anterior repair. An epidemiological investigation. *J Bone Joint Surg [Br]* 1991;73:941–946.
549. O'Driscoll SW, Evans DC. Long-term results of staple capsulorrhaphy for anterior instability of the shoulder. *J Bone Joint Surg [Am]* 1993;75:249–58.
550. Editorial. Voluntary dislocation of the shoulder. *BMJ* 1973;4:505.
551. Elbaum R, Parent H, Zeller R, et al. Traumatic scapulohumeral dislocation in children and adolescents. Apropos of 9 patients. *Acta Orthop Belg* 1994;60:204–209.
552. Endo S, Kasai T, Fujii N, et al. *Arch Arthrop Trauma Surg* 1993;112:201–202.
553. Foster WS, Ford TB, Drez D. Isolated posterior shoulder dislocation in a child. *Am J Sports Med* 1985;13:198–200.
554. Freundlich BD. Luxatio erecta. *J Trauma* 1983;23:434–436.
555. Frosch L. Congenital subluxation of the shoulder. *Klin Wochenschr* 1925;4:701–702.
556. Gartland JJ, Dowling JJ. Recurrent anterior dislocation of the shoulder joint. *Clin Orthop* 1954;3:86–91.
557. Goldberg BJ, Nirschl RP, McConnell JP, et al. Arthroscopic transglenoid suture capsulolabral repairs: preliminary results. *Am J Sports Med* 1993;21:656–665.
558. Green NE, Wheelhouse WW. Anterior subglenoid dislocation of the shoulder in an infant following pneumococcal meningitis. *Clin Orthop* 1978;135:125–127.
559. Grieg DM. On true congenital dislocation of the shoulder. *Edinburgh Med J* 1923;30:157–175.
560. Gudinchet F, Naggar L, Ginalski JM, et al. Magnetic resonance imaging of nontraumatic shoulder instability in children. *Skel Radio*. 1992;21:19–21.
561. Haliburton RA, Barber JR, Fraser RL. Pseudodislocation: an unusual birth injury. *Can J Surg* 1967;10:455–462.
562. Hawkins RJ, Angelo RL. Glenohumeral osteoarthritis: acute complications of the Putti-Platt repair. *J Bone Joint Surg [Am]* 1990;72:1193–1197.

563. Hawkins RJ, Koppert G, Johnston G. Recurrent posterior instability (subluxation) of the shoulder. *J Bone Joint Surg [Am]* 1984;66A:169–174.
564. Heck CC Jr. Anterior dislocation of the glenohumeral joint in a child. *J Trauma* 1981;21:174–175.
565. Heim M, Horoszowski H, Martinowitz U. Hemophilic arthropathy resulting in a locked shoulder. *Clin Orthop* 1986;202:169–172.
566. Hernandez A, Drez D. Operative treatment of posterior shoulder dislocations by posterior glenoidplasty, capsulorrhaphy and infraspinatus advancement. *Am J Sports Med* 1986;14:187–191.
567. Hovelius L. Anterior dislocation of the shoulder in teenagers and young adults. *J Bone Joint Surg [Am]* 1987;69A:393–399.
568. Hovelius L, Augustini BG, Fredin H, et al. Primary anterior dislocation of the shoulder in young patients: a ten-year prospective study. *J Bone Joint Surg [Am]* 1996;78:1677–1684.
569. Hovelius L, Erikson GK, Fredin FH, et al. Recurrences after initial dislocation of the shoulder. *J Bone Joint Surg* 1983;65:343–349.
570. Huber H, Gerber C. Voluntary subluxation of the shoulder in children. A long-term follow-up study of 36 shoulders. *J Bone Joint Surg* 1994;76:118–122.
571. Jalovaara P, Myllyla V, Paivansalo M. Autotractor stress roentgenography for demonstration of anterior and inferior instability of the shoulder joint. *Clin Orthop* 1992;284:136–143.
572. Karadimas J, Rentis G, Varouchas G. Repair of anterior dislocation of the shoulder using transfer of the subscapularis tendon. *J Bone Joint Surg [Am]* 1980;62:1147–1149.
573. Keiser RP, Wilson CL. Bilateral recurrent dislocation of the shoulder (atraumatic) in a 13-year-old girl. *J Bone Joint Surg [Am]* 1961;43:553–554.
574. Kleinman PK, Goss TP, Kanzaria PK, et al. Injuries of the glenoid labrum in athletic teenagers [Abstract]. *Pediatr Radio*. 1985;15:71.
575. Kothari RU, Dronen SC. Prospective evaluation of the scapular manipulation technique in reducing anterior shoulder dislocations. *Ann Emerg Med* 1992;21:1349–1352.
576. Kummel BM. Arthrography in anterior capsular derangements of the shoulder. *Clin Orthop* 1972;83:170–176.
577. Laskin RS, Sedlin ED. Luxatio erecta in infancy. *Clin Orthop* 1971;80:126–129.
578. Lawhon SM, Peoples AB, MacEwen GD. Voluntary dislocation of the shoulder. *J Pediatr Orthop* 1982;2:590.
579. Lichtblau PO. Shoulder dislocation in the infant. Case report and discussion. *J Fla Med Assoc* 1977;64:313–320.
580. Liebolt FL, Furey JG. Obstetrical paralysis with dislocation of the shoulder: a case report. *J Bone Joint Surg [Am]* 1953;35A:227–230.
581. Lombardo SJ, Kerlan RK, Jobe FW, et al. The modified Bristow procedure for recurrent dislocation of the shoulder. *J Bone Joint Surg [Am]* 1976;58:256–261.
582. Lucas GL, Peterson MD. Open anterior dislocation of the shoulder: case report. *J Trauma* 1977;17:883–884.
583. Lusard DA, Wirth MA, Wurtz D, et al. Loss of external rotation following anterior capsulorrhaphy of the shoulder. *J Bone Joint Surg [Am]* 1993;75:1185–1192.
584. Magnuson PB, Stack JK. Bilateral habitual dislocation of the shoulders in twins: a familial tendency. *JAMA* 1940;114:2103.
585. Marans HJ, Angel KR, Schemitsch EH, et al. The fate of traumatic anterior dislocation of the shoulder in children. *J Bone Joint Surg* 1992;74:1242–1244.
586. Matton D, Van Looy F, Geens S. Recurrent anterior dislocations of the shoulder joint treated by the Bristow-Latarjet procedure. *Acta Orthop Belg* 1992;58:16–22.
587. May VR Jr. Posterior dislocation of the shoulder: habitual, traumatic and obstetrical. *Orthop Clin North Am* 1980;11:271–285.
588. McNamara RM. Reduction of anterior shoulder dislocations by scapular manipulation. *Ann Emerg Med* 1993;22:1140–1144.
589. McNeil EL. Luxatio erecta [Letter]. *Ann Emerg Med* 1984;13:490–491.
590. Mirick MJ, Clinton JE, Ruiz E. External rotation method of shoulder dislocation reduction. *J Am Coll Emerg Phys* 1979;9:528–531.
591. Montgomery WH 3rd, Jobe FW. Functional outcomes in athletes after modified anterior capsulolabral reconstruction. *Am J Sports Med* 1994;22:352–358.
592. Morrey BF, Janes JM. Recurrent anterior dislocation of the shoulder. *J Bone Joint Surg [Am]* 1976;58:252–256.
593. Morrison PD, Egan TJ. Axillary artery injury in erect dislocation of the shoulder (luxatio erecta): a case report. *J Ir Orthop* 1983;260–261.
594. Neer CS II. Involuntary inferior and multidirectional instability of the shoulder: etiology, recognition, and treatment. *Instr Course Lect* 1985;34:232–238.
595. Neer CS II, Foster DR. Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder. *J Bone Joint Surg [Am]* 1980;62:897–908.
596. Norwood L, Terry GC. Shoulder posterior subluxation. *Am J Sports Med* 1984;12:25–30.
597. Ogden JA. *Skeletal injury in the child*. Philadelphia: Lea & Febiger, 1982:227–228.
598. Pettersson H. Bilateral dysplasia of the neck of the scapula and associated anomalies. *Acta Radiol Diagn (Stockh)* 1981;22:81–84.
599. Pollen AG. *Fractures and dislocations in children*. Baltimore: Williams & Wilkins, 1973.
600. Rang M. *Children's fractures*, 2nd ed. Philadelphia: JB Lippincott, 1983.
601. Rockwood CA Jr. The shoulder: facts, confusions and myths. *Int Orthop* 1991;15:401–405.
602. Rockwood CA, Matsen FA, Thomas SC. Anterior glenohumeral instability. In: Rockwood CA, Matsen FA, eds. *The shoulder*. Philadelphia: WB Saunders, 1990:592–598.
603. Rowe CR. Anterior dislocation of the shoulder: prognosis and treatment. *Surg Clin North Am* 1963;43:1609–1614.
604. Rowe CR. Prognosis in dislocation of the shoulder. *J Bone Joint Surg [Am]* 1956;38:957–977.
605. Rowe CR, Pierce DS, Clark JG. Voluntary dislocation of the shoulder. *J Bone Joint Surg [Am]* 1973;55:445–459.
606. Samilson RL, Miller E. Posterior dislocation of the shoulder. *Clin Orthop* 1964;32:69–86.
607. Sharrard WJW. *Paediatric orthopaedics and fractures*. Oxford: Blackwell, 1971.
608. Shvartzman P, Guy N. Voluntary dislocation of shoulder. *Postgrad Med* 1988;84:265–271.
609. Stern WA. Arthrogryposis multiplex congenita. *JAMA* 1923;81:1507–1510.
610. Tachdjian MO. *Paediatric orthopaedics*. Philadelphia: WB Saunders, 1990.
611. Uhthoff HK, Piscopo M. Anterior capsular redundancy of the shoulder: congenital or traumatic? An embryological study. *J Bone Joint Surg [Br]* 1985;67:363–366.
612. Vastamaki M, Solonen KA. Posterior dislocation and fracture dislocation of the shoulder. *Acta Orthop Scand* 1980;51:479–484.
613. Vermeiren J, Handelberg F, Casteleyn PP, et al. The rate of recurrence of traumatic anterior dislocation of the shoulder. *Int Orthop* 1993;17:337–341.
614. Wagner KT, Lyne ED. Adolescent traumatic dislocations of the shoulder with open epiphysis. *J Pediatr Orthop* 1983;3:61–62.
615. Weber BG, Brunner C, Freuler F. *Treatment of fractures in children and adolescents*. Berlin: Springer-Verlag, 1980:94–95.
616. White SS, Blane CE, DiPietro MA, et al. Arthrography in evaluation of birth injuries of the shoulder. *J Can Assoc Radio*. 1987;38:113–115.
617. Wickstrom J. Birth injuries of the brachial plexus treatment defects in the shoulder. *Clin Orthop* 1962;23:187–196.
618. Wickstrom J, Haslam ET, Hutchinson RH. The surgical management of residual deformities of the shoulder following birth injuries of the brachial plexus. *J Bone Joint Surg [Am]* 1955;37:27–36.
619. Zuckerman JD, Matsen FA. Complications about the glenohumeral joint related to the use of screws and staples. *J Bone Joint Surg [Am]* 1984;66:175–180.

## CERVICAL SPINE INJURIES IN CHILDREN

W. C. WARNER, JR.

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- Occiput to C2 Arthrodesis
- Fractures of the Atlas
- Atlantoaxial Injuries
- Odontoid Fractures
- Os Odontoideum
- Traumatic Ligamentous Disruption
- Atlantoaxial Instability Associated with Congenital Anomalies and Syndromes
- Atlantoaxial Rotatory Subluxation
- Hangman's Fracture
- Subaxial Injuries
  - Posterior Ligamentous Disruption
- Compression Fractures
- Unilateral and Bilateral Facet Dislocations
- Burst Fractures
- Spondylolysis and Spondylolisthesis
- Chapter References

Injuries to the cervical spine in children are uncommon, accounting for only 1% of pediatric fractures and 2% of all spinal injuries ( [4,69,70,89,107,122,139,159,173](#)). McGrory et al. ([107](#)) estimated that the incidence of pediatric cervical spine injuries was 7.41 in 100,000 per year. However, this low incidence of cervical spine injuries in children may be misleading because some injuries are not detected or detected only at autopsy. Aufdermaur ( [8](#)) examined the autopsied spines of 12 juveniles who had spinal injuries. Clinically and radiologically, a fracture was suggested in only 1, but all 12 had cartilage end plates that were separated from the vertebral bodies in the zone of columnar and calcified cartilage similar to a Salter I fracture. Only radiographs at autopsy showed the disruption, represented by a small gap or apparent widening of the intervertebral space ( [8](#)).

Pediatric cervical spine fractures differ from those in adults not only in pattern but in cause, incidence, and treatment ( [113](#)). Most cervical spine injuries in children under 11 years of age occur in the upper cervical spine, unlike in older children, adolescents, and adults, in whom fractures occur more commonly in the lower cervical spine ([55,69,89,139](#)). Upper cervical spine injuries are more predominant in young children because of the unique anatomic and biomechanical properties of the immature cervical spine. The immature spine is hypermobile because of ligamentous laxity, and the facet joints are more horizontally oriented. These horizontally oriented facets give less resistance to forward translation than the more vertically oriented facets in adults. This, combined with a young child's relatively large head, changes the fulcrum of neck motion to the upper cervical spine. Because a child's musculature is not as developed as an adult's, the cervical spine has less support during injury. As the anatomic and biomechanical aspects of the spine change with maturity, so do the level and type of injuries seen.

In younger children, most of the injuries are fractures through the vertebral end plates or synchondroses or ligamentous injuries. In adolescents, vertebral body fractures or fracture–dislocations involving the lower cervical spine are more common and have the characteristics of adult cervical spine injuries ( [Table 18-1](#)).

Age <8 yr
Upper cervical spine injuries
AOD
Odontoid fractures
Jefferson fracture
C2 spondylolisthesis
Age >8 yr
Lower cervical spine injuries
Facet fracture–dislocation
Burst fracture

**TABLE 18-1. LOCATION OF INJURIES BY AGE**

Neurologic deficits occur infrequently with pediatric cervical fractures, and when a partial deficit is present, the prognosis is better in children than in adults ( [13,47](#)). Complete neurologic deficits, however, tend to remain complete regardless of whether they occur in children or adults. This may reflect the magnitude of injury that caused the complete neurologic lesion. Orenstein et al. ( [119,120](#)) found that deaths from cervical spine injuries or associated injuries occur most commonly in children 8 years of age or younger. Nitecki and Moir ( [115](#)) found that the younger the age and the higher the level of injury, the more likely the injury was to be fatal, again reflecting the severity of the initial injury and explaining the increased incidence of death ( [72,115,119](#)).

Spinal cord injury without radiographic abnormalities (SCIWORA) has been found with increased frequency in children and may be related to the relative hypermobility of a child's spine (see later section on [SCIWORA](#)). Depending on the age at the time of injury and the level of spinal cord injury, other problems that may occur in children are the development of spinal deformities and lower extremity problems, such as contractures and progressive subluxation or dislocation of the hip joint.

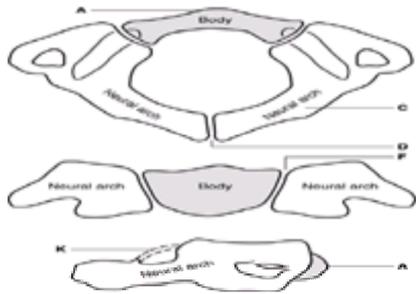
## ANATOMY

Understanding the normal growth and development of the cervical spine is essential when treating a child with a suspected cervical spine injury ( [130](#)). This will allow the physician to differentiate normal physes or synchondroses from pathologic fractures or ligamentous disruptions and will alert the physician to any possible

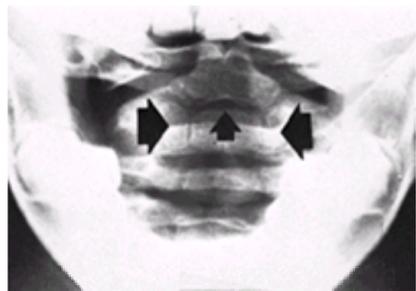
congenital anomalies that may be mistaken for a fracture.

## Upper Cervical Spine

At birth the atlas is composed of three ossification centers, one for the body and one for each of the neural arches ( Fig. 18-1). The ossification center for the anterior arch is present in approximately 20% of individuals at birth, appearing in the remainder during the first year of life. Occasionally, the anterior arch is bifid, and the body may be formed from two centers or may fail to completely appear. The posterior arches usually close by 3 years of age. Occasionally, the posterior synchondrosis between the two halves of the neural arch fail to fuse and result in a bifid arch. This common variation must be considered during any surgical dissection in this region. The neurocentral synchondroses that link the neural arches to the body are best seen on an open-mouth odontoid view. These synchondroses close by 7 years of age and should not be mistaken for fractures ( 30). The canal of the atlas is large to allow for the amount of rotation that occurs at this joint as well as some forward translation ( 31). The vertebral arteries are about 2 cm from the midline and run in a groove on the superior surface of the atlas. This must be remembered during lateral dissection at the occipital cervical junction. The ring of C1 reaches about normal adult size by four years of age. Therefore arthrodesis after 4 years of age should not cause spinal canal stenosis ( Fig. 18-2).



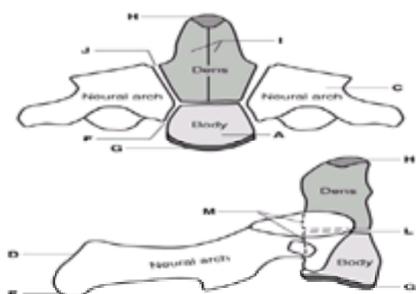
**FIGURE 18-1.** Diagram of C1 (atlas). The body (A) is not ossified at birth, and its ossification center appears during the 1st year of life. The body may fail to develop, and forward extension of neural arches (C) may take its place. Neural arches appear bilaterally about the 7th week (D), and the most anterior portion of the superior articulating surface is usually formed by the body. Synchondrosis of spinous processes unite by the 3rd year. Union may rarely be preceded by the appearance of the secondary center within the synchondrosis. Neurocentral synchondrosis (F) fuses about the 7th year. The ligament surrounding the superior vertebral notch (K) may ossify, especially in later life. (Reprinted from Bailey DK. Normal cervical spine in infants and children. *Radiology* 1952;59 : 713=714; with permission.)



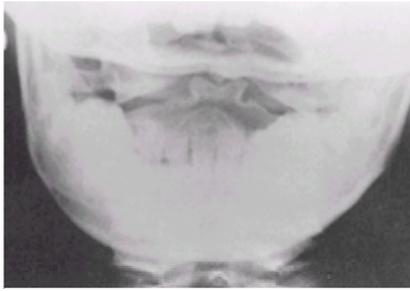
**FIGURE 18-2.** The epiphysis at the base of the odontoid process is shown by the small arrow. This is well below the level of the superior articular facet of the axis. The synchondroses between the body of the axis and the neural arches are shown by the large arrows. Just above this are the synchondroses between the odontoid process and the neural arches. The odontoid process, therefore, surmounts the body of the axis and is sandwiched between the neural arches. The physis and synchondroses combine to form the letter arches. (Reprinted from Fielding JW. Selective observations on the cervical spine in child. In: Ahstrom JP Jr, ed. *Current practice in orthopaedic surgery*. Vol. 5. St. Louis: CV Mosby, 1973; with permission.)

Occipitalization or assimilation of the atlas occurs when the first cervical vertebra fails to segment from the skull. This may lead to basilar impression and narrowing of the foramen magnum. This anomaly can increase a child's susceptibility to spinal cord injury or vertebral artery compromise caused by trauma. Vertebral artery compromise also can occur in patients with occipitalization from skull traction, resulting in brainstem ischemia ( 14).

The axis develops from at least four separate ossification centers: one for the dens, one for the body, and two for the neural arches ( Fig. 18-3). Between the odontoid and the body of the axis is a synchondrosis or vestigial disk space that often is mistaken for a fracture line. This synchondrosis runs well below the level of the articular processes of the axis and usually fuses at about 6 to 7 years of age, although it may persist as a sclerotic line until 11 years of age ( 31). The most common odontoid fracture pattern in adults and adolescents is transverse and at the level of the articular processes. This fracture should not be confused with the normal synchondrosis, which is more cup shaped, with the transverse portion below the level of the articular processes in the body of the axis. After 6 to 7 years of age, the synchondrosis should not be present on an open-mouth odontoid view. A fracture should be considered if a lucent line is present after 7 years of age. The neural arches of C2 fuse at 3 to 6 years of age. These are seen as vertical lucent lines on the open-mouth odontoid view. Occasionally, the tip of the odontoid is V shaped (dens bicornum) or a small separate summit ossification center may be present at the tip of the odontoid (ossiculum terminale) ( Fig. 18-4).

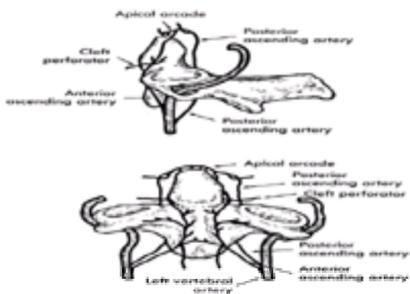


**FIGURE 18-3.** Diagram of C2 (axis). The body (A) in which one center (occasionally two) appears by the 5th fetal month. Neural arches (C) appear bilaterally by the 7th fetal month. Neural arches fuse (D) posteriorly by the 2nd or 3rd year. Bifid tip (E) of spinous process (occasionally a secondary center is present in each tip). Neurocentral synchondrosis (F) fuses at 3 to 6 years. The inferior epiphyseal ring (G) appears at puberty and fuses at about 25 years of age. The summit ossification center (H) for the odontoid appears at 3 to 6 years and fuses with the odontoid by 12 years. Odontoid (dens) (I). Two separate centers appear by the 5th fetal month and fuse with each other by the 7th fetal month. Synchondrosis between the odontoid and neural arch (J) fuses at 3 to 6 years. Synchondrosis between the odontoid and body (L) fuses at 3 to 6 years. Posterior surface of the body and odontoid (M). (Reprinted from Bailey DK. Normal cervical spine in infants and children. *Radiology* 1952;59 : 713-714; with permission.)



**FIGURE 18-4.** Anteroposterior open-mouth odontoid view demonstrating V-shaped dens bicornis and ossiculum terminale. (Reprinted from Warner WC. Cervical spine anomalies. In: Canale ST, Beaty JH, eds. *Operative pediatric orthopedics*, 2nd ed. St. Louis: CV Mosby, 1995; with permission.)

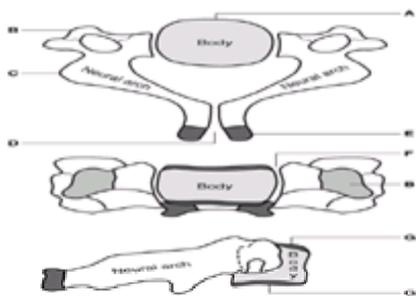
The arterial blood supply to the odontoid is derived from the vertebral and carotid arteries. The anterior and posterior ascending arteries arise from the vertebral artery at the level of C3 and ascend anteriorly and posteriorly to the odontoid, meeting superiorly to form an apical arcade. These arteries supply small penetrating branches to the body of the axis and the odontoid process. The internal carotid artery gives off cleft perforators that supply the superior portion of the odontoid. This peculiar arrangement of arteries and vessels is necessary for embryologic development and anatomic function of the odontoid. The synchondrosis prevents direct vascularization of the odontoid from C2, and vascularization from the blood supply of C1 is not possible because the synovial joint cavity surrounds the odontoid. The formation of an os odontoideum after cervical trauma may be related to this peculiar blood supply ( [Fig. 18-5](#)).



**FIGURE 18-5.** Blood supply to odontoid: posterior and anterior ascending arteries and apical arcade. (Reprinted from Schiff DC, Parke WW. The arterial supply of the odontoid process. *J Bone Joint Surg [Am]* 1973;55 : 1450-1464; with permission.)

### Lower Cervical Spine

The third and seventh cervical vertebrae share a similar ossification pattern: a single ossification center for the vertebral body and an ossification center for each neural arch ( [Fig. 18-6](#) ). The neural arch fuses posteriorly between the second and third years, and the neurocentral synchondroses between the neural arches and the vertebral body fuse by 3 to 6 years of age. These vertebrae are normally wedge shaped until 7 to 8 years of age ( [31,96,133](#) ). The vertebral bodies, neural arches, and pedicles enlarge by periosteal appositional growth, similar to that seen in long bones. By 8 to 10 years of age, a child's spine usually reaches near adult size and characteristics. There are 5 secondary ossification centers that can remain open until 25 years of age ( [96](#) ). These include one each for the spinous processes, transverse processes, and the ring apophyses about the vertebral end plates. These should not be confused with fractures.



**FIGURE 18-6.** Diagram of typical cervical vertebrae, C3 to C7. The body ( **A** ) appears by the 5th fetal month. The anterior (costal) portion of the transverse process ( **B** ) may develop from a separate center that appears by the 6th fetal month and joins the arch by the 6th year. Neural arches ( **C** ) appear by the 7th to 9th fetal week. Synchondrosis between spinous processes ( **D** ) usually unites by the 2nd or 3rd year. Secondary centers for bifid spine ( **E** ) appear at puberty and unite with spinous process at 25 years. Neurocentral synchondrosis ( **F** ) fuses at 3 to 6 years. Superior and inferior epiphyseal rings ( **G** ) appear at puberty and unite with the body at about 25 years. The seventh cervical vertebra differs slightly because of a long, powerful, nonbifid spinous process. (Reprinted from Bailey DK. Normal cervical spine in infants and children. *Radiology* 1952;59 : 713-714; with permission.)

The superior and inferior end plates are firmly bound to the adjacent disk. The junction between the vertebral body and the end plate is similar to a physis of a long bone. The vertebral body is analogous to the metaphysis, and the end plate to the physis, where longitudinal growth occurs. The junction between the vertebral body and the end plate has been demonstrated to be weaker than the adjacent vertebral body or disk, which can result in a fracture at the end plate in the area of columnar and calcified cartilage of the growth zone, similar to a Salter I fracture of a long bone ( [8](#) ). The inferior end plate may be more susceptible to this injury than the superior end plate because of the mechanical protection afforded by the developing uncinat processes ( [17,87](#) ).

The facet joints of the cervical spine change in orientation with age. The angle of the C1–C2 facet is 55 degrees in the newborn and increases to 70 degrees at maturity. In the lower cervical spine, the angle of the facet joints is 30 degrees at birth and 60 to 70 degrees at maturity. This may explain why the pediatric cervical spine may be more susceptible to injury from the increased motion or translation allowed by the facet joint orientation.

Increased ligamentous laxity in young children allows a greater degree of spinal mobility. Flexion and extension of the spine at C2–3 is 50% greater in children between the ages of 3 and 8 years than in adults. The level of the greatest mobility in the cervical spine descends with increasing age. Between 3 and 8 years of age the most mobile segment is C3–C4; from ages 9 to 11 years, C4–C5 is the most mobile segment, and from ages 12 to 15 years C5–C6 is the most mobile segment ( [34,125](#) ). This explains the tendency for craniocervical injuries in the young children.

Several anomalies of the cervical spine may influence treatment recommendations. The atlas can fail to segment from the skull, a condition called occipitalization of the atlas, and can lead to narrowing of the foramen magnum, neurologic symptoms, and increased stresses to the atlantoaxial articulation, which often causes instability. The association of occipitalization of the atlas and congenital fusion of C2 and C3 is likely to lead to atlantoaxial instability. Failure of fusion of the posterior

arc of C1 is not uncommon and should be sought before performing any surgical procedure that involves C1. Wedge-shaped vertebrae, bifid vertebrae, or a combination of these also can occur. Klippel-Feil syndrome consists of the classic triad of a short neck, low posterior hairline, and severe restriction of motion of the neck from fusion of the cervical vertebrae (77,91). Congenital fusion of the cervical spine may predispose a child to injury from trauma by concentrating stresses in the remaining mobile segments.

Hensinger et al. (76) reported congenital anomalies of the odontoid process, including aplasia (complete absence), hypoplasia (partial absence in which there is a stubby piece at the base of the odontoid located above the C1 articulation), and os odontoideum. Os odontoideum consists of a separate ossicle of the odontoid with no connection to the body of C2. The cause may be traumatic in nature (76). These anomalies of the odontoid also may predispose a child to injury or instability.

## HISTORY

Most cervical spine injuries in young children occur from falls, motor vehicle accidents, or pedestrian–vehicle accidents (36,107). In young children a fall from less than 5 feet can cause a cervical spine injury (144). Infants are at risk for cervical spine injury from lack of head control. Other causes of cervical spine injury in infants are child abuse and obstetric trauma (Table 18-1) (30,153,162). In teenagers injuries from sports and motor vehicle accidents are more common (107,166). At Children's Hospital in Boston, cervical spine injuries from unsupervised diving exceeded those occurring in organized athletics over the past 20 years. Haffner et al. (70) have reported an increased frequency of spinal injuries from gunshot wounds in teenagers.

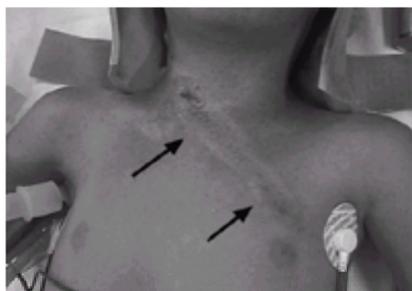
Obtaining an adequate history and physical examination from an injured child may require more time, patience, and effort by the treating physician. An adequate history and physical examination may not be obtained on the initial examination, and repeated examination may be necessary.

## SYMPTOMS

Children with cervical spine injuries have various signs and symptoms. Neck pain or localized tenderness of the cervical spine is the most common presenting symptom and should alert the physician to proceed with further diagnostic workup. The patient also may have more subtle complaints, such as headaches, occipital pain radiating to the shoulders, limited range of motion, torticollis, bowel or bladder symptoms, seizures, or a snapping sensation in the neck. In newborns, any unexplained respiratory distress, delayed motor response, hypotonia, or hypertonia should be evaluated. Occasionally, a patient may be unresponsive, so the spine must be protected until appropriate spine clearance has been obtained. Head or facial trauma, loss of consciousness, or high-speed motor vehicle accident are indications for evaluation of the cervical spine. Sometimes after multiple trauma, the only sign of a cervical spine injury is difficulty weaning from assisted ventilation. Often this is associated with a closed head injury. This can mislead the treating physician into believing that the difficulty in weaning from the ventilator is due to the head injury, and an associated cervical spine injury may be overlooked. Positive neurologic findings may be present despite a normal-appearing radiograph, a condition called SCIWORA, and should alert the treating physician to a possible occult cervical spine injury (83,122,164,169).

## EVALUATION

Examination of an injured child should begin with inspection for associated head or facial trauma. Abrasions and bruising about the neck that can be caused by direct trauma or from the shoulder harness of a seat belt may be clues to an underlying cervical spine injury (Fig. 18-7) (58,66,82,99). An unconscious patient should be treated as if a cervical spine injury is present until proved otherwise (Fig. 18-8). The spine should be palpated for tenderness, muscle spasm, and alignment of the vertebrae. The spinous processes of C2 and C3 should be prominent and easily palpable. Anterior examination of the cervical spine may be difficult in a child. The cricoid cartilage is at the C5–C6 level and can be used as an anatomic landmark. The entire spine should be palpated and examined, because 20% of patients with cervical spine injuries have injuries at multiple levels (158). A thorough neurologic examination of both the upper and lower extremities should be performed, including strength, sensation, reflexes, and proprioception. An adequate neurologic examination may not be possible initially, and repeated examinations may be necessary. Extremity weakness or subtle sensory losses may not be apparent initially and may be found only on repeated examinations. Rectal sphincter tone and perianal sensation also should be evaluated. If the child is awake and cooperative, flexion, extension, rotation, and lateral tilt can be performed and correlated with pain. However, this should not be performed if the child is not cooperative or is unresponsive and only after initial screening radiographs have been evaluated.



**FIGURE 18-7.** Clinical photograph of a patient with cervical spine injury, resulting from impact with the shoulder harness of a seat belt. Note location of skin contusions from the seat belt.



**FIGURE 18-8.** Clinical photograph demonstrating a soft tissue injury that can occur from impact with a shoulder harness of a seat belt and air bag inflation.

## RADIOGRAPHIC EVALUATION

### Plain Radiographs

When cervical spine injury is suspected, radiographs of the cervical spine are indicated. There is still no consensus on whether routine radiographs of the cervical spine should be obtained in every pediatric trauma patient. Some studies have shown that without associated facial trauma or specific physical findings that suggest cervical spine injury, routine cervical radiographs produce a low yield in pediatric trauma (42,95,133). Ratchesky et al. (133) attempted to identify which injured children required cervical spine radiographs. They reviewed a large series of patients, of whom 1.2% had documented cervical spine injuries and concluded that cervical spine radiographs were indicated if the child had a complaint of neck pain, or if head or facial trauma associated with a motor vehicle accident were present. Despite these studies, the burden is still on the treating physician to rule out cervical spine injuries. Initial plain films should include a lateral cervical spine, open-mouth odontoid view, and an anteroposterior cervical spine. Oblique radiographs may be obtained if abnormalities are present on initial radiographs, or the treating physician suspects a cervical spine injury by history or examination. If the patient is medically unstable, then a cross-table lateral radiograph is sufficient until

the patient's condition permits a complete evaluation. Significant false-negative rates of 23% to 26% for single cross-table lateral radiographs have been reported, emphasizing the need for a complete radiographic evaluation of suspected cervical spine injuries ( 9,148). Lateral flexion and extension views can be obtained, but only with careful supervision and only after static films have not shown any obvious abnormalities. The child must be alert and cooperative, so it may not be possible to obtain these studies in a young, frightened child.

Evaluation of cervical spine radiographs should proceed systematically and should include evaluation of anterior and posterior vertebral body alignment, interspinous distance, and spinolaminar line. The absence of cervical lordosis on a lateral film may be a normal finding in children and does not necessarily indicate a cervical spine injury (31). The prevertebral soft tissue space should be evaluated for increased swelling. An increase in the prevertebral soft tissue space may be a subtle finding of possible ligamentous or bony injury. This soft tissue space may be altered by inspiration, crying, and infection, but a measurement of less than 6 mm at C3 is considered normal.

The interpretation of the plain films requires knowledge of normal spine variations. This is particularly true with the neurocentral synchondrosis and growth plates. Knowing when these should be present and when they should not is important so that they are not mistaken for an occult fracture ( Table 18-2).

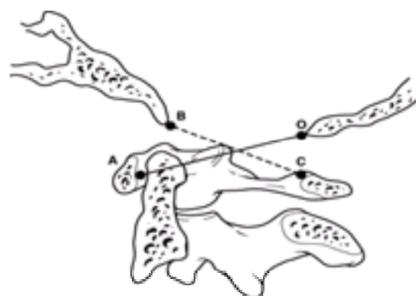
<b>Avulsion fracture</b>	Apical ossification center of the odontoid Secondary ossification centers at the tips of the transverse and spinous processes
<b>Fracture</b>	Persistence of the synchondrosis at the base of the odontoid Apparent anterior wedging of a young child's vertebral body Normal posterior angulation of the odontoid seen in 4% of normal children
<b>Instability</b>	Pseudosubluxation of C2-C3 Incomplete ossification, especially of the odontoid process, with apparent superior subluxation of the anterior arch of C1
<b>Absence of the ossification center of the anterior arch of C1 in the first year of life may suggest posterior displacement of C1 on the odontoid</b>	Increase in the atlanto-dens interval of up to 4.5 mm
<b>Miscellaneous</b>	Physiologic variations in the width of the prevertebral soft tissue due to crying misinterpreted as swelling due to edema or hemorrhage Overlying structures such as ears, braided hair, teeth, or hyoid bone. Plastic rivets used in modern emergency cervical immobilization collars can simulate fracture line Horizontally placed facets in the younger child, creating the illusion of a pillar fracture Congenital anomalies such as os odontostium, spina bifida, and congenital fusion of hemivertebrae

**TABLE 18-2. NORMAL OSSIFICATION CENTERS AND ANOMALIES FREQUENTLY CONFUSED WITH INJURY**

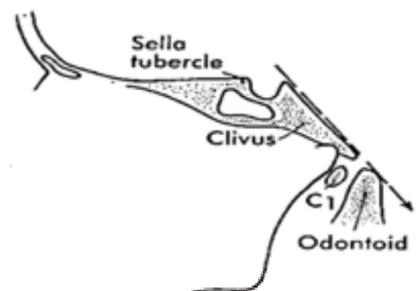
## Radiographic Evaluation of Specific Areas of the Spine

### Atlantooccipital Junction

The atlantooccipital interval is the most difficult to assess because of the lack of discreet and reproducible landmarks on radiographs. The occipital condyles should rest within the depressions of the facets of the atlas. The interval between the occipital condyles and the superior margin of the facet joints of the atlas should be approximately 3 mm, and if the interval is more than 5 mm, an atlantooccipital disruption may be present ( 43,125). The interval between the basion (the anterior cortical margin of the foramen magnum) and the tip of the dens should be less than 12 mm (25). Another measurement to evaluate the atlantooccipital junction is the Powers ratio, which assesses the relative position of the skull base to the atlas ( Fig. 18-9). A line drawn from the basion to the anterior cortex of the posterior arch of C1 is measured, and this distance is divided by the distance of a line drawn from the opisthion (the posterior cortical margin of the foramen magnum) to the posterior cortex of the anterior arch of C1. A normal value should be less than 1 and more than 0.7. A value of more than 1 suggests anterior subluxation of the atlantooccipital joint. A value of less than 0.7 suggests posterior subluxation of the atlantooccipital joint. However, the basion is not always visible on plain radiographs. The Wackenheimer line, which is drawn along the posterior aspect of the clivus, probably is the most easily identified line to determine disruption of the atlantooccipital joint (Fig. 18-10) (108). If the line does not intersect the tip of the odontoid tangentially and if this line is displaced anteriorly or posteriorly, disruption or increased laxity about the atlantooccipital joint should be suspected. Other lines such as the McGregor, McCrae, and Chamberlain are not as useful in the evaluation of trauma but have been useful for identifying congenital anomalies with regard to basilar impression.



**FIGURE 18-9.** The Powers ratio is determined by drawing a line from the basion (B) to the posterior arch of the atlas (C) and a second line from the opisthion (O) to the anterior arch of the atlas (A). The length of the line BC is divided by the length of the line OA, producing the Powers ratio. (Reprinted from Lebwohl NH, Eismont FJ. Cervical spine injuries in children. In: Weinstein SL, ed. *The pediatric spine: principles and practice*. New York: Raven, 1994; with permission.)

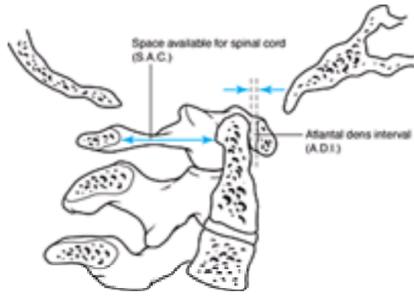


**FIGURE 18-10.** Drawing of Wackenheimer clivus-canal line. This line is drawn along the clivus into the cervical spinal canal and should pass just posterior to the tip of the odontoid. (Reprinted from Menezes AH, Ryken TC. Craniovertebral junction abnormalities. In: Weinstein SL, ed. *The pediatric spine: principles and practice*. New York: Raven, 1994; with permission.)

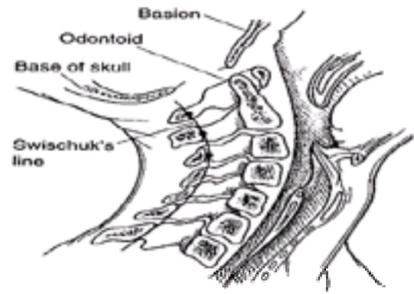
### Atlantoaxial Joint

Evaluation of the atlantoaxial joint should consist of measurement of the atlanto-dens interval and the space available for the spinal cord ( Fig. 18-11). Steel reported that the space at the atlantoaxial joint can be divided into thirds: one third is taken up by the odontoid, one third by the spinal cord, and one third is space that is available for the cord (160). Up to 4.5 mm is considered a normal atlanto-dens interval in a child, because the unossified cartilage of the odontoid apparently increases the atlanto-dens interval. Also in children, extension gives the appearance of subluxation of the anterior portion of the atlas over the unossified dens on the

lateral view. This usually is a pseudosubluxation and does not represent true instability ( [Fig. 18-12](#) ) (31,40,51,53).



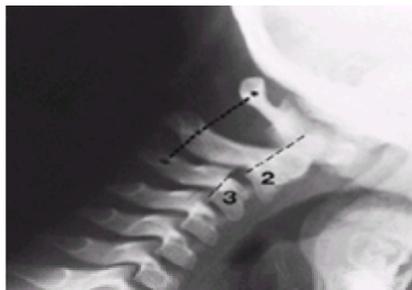
**FIGURE 18-11.** Sagittal views of the atlantoaxial joint demonstrating the atlanto-dens interval ( ADI). The space available for the cord ( SAC) is the distance between the posterior aspect of the odontoid and the posterior ring of C1. (Reprinted from Hensinger RN, Fielding JW. The cervical spine. In: Morrissy RT, ed. *Lovell and Winter's pediatric orthopaedics*, 3rd ed. Philadelphia: JB Lippincott, 1990; with permission.)



**FIGURE 18-12.** The spinolaminar line (Swischuk's line) is used to determine the presence of pseudosubluxation of C2 on C3. (Reprinted from Copley LA, Dormans JP. Cervical spine disorders in infants and children. *J Am Acad Orthop Surg* 1998;6; with permission.)

### Upper Cervical Spine

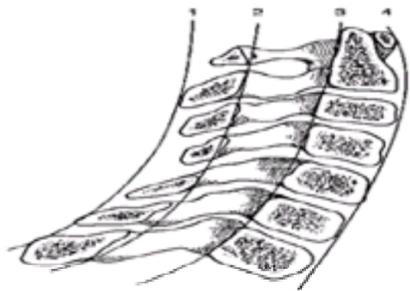
Anterior displacement of one vertebral body on another may indicate a bony or ligamentous injury, but anterior displacement of as much as 3 mm at one level may be a normal finding in the upper cervical spine of a child. This pseudosubluxation is most common at C2–C3 and at C3–C4. The anterior displacement should only be present when the radiographs are taken in flexion, and this displacement will reduce in extension. As an aid in deciding if subluxation is physiologic or pathologic, Swischuk and Rowe (161) and Von Torklus and Gehle (168) described a line drawn from the anterior cortex of the spinous process of C1 to the spinous process of C3 (Fig. 18-13). If the anterior cortex of the C2 spinous process misses this line by more than 3 mm, a true subluxation should be suspected. Also when evaluating instability caused by posterior soft tissue injuries, widening between C1 and C2 of more than 10 mm between the spinous processes is indicative of a soft tissue or ligamentous injury (2).



**FIGURE 18-13.** Pseudosubluxation of C2 on C3. Hypermobility is common in children under the age of 8 years. Specific measurements of the movement of the vertebral bodies ( *thin dotted line* ) are unreliable, whereas the relationship with the posterior elements ( *thick dotted line* ) is more consistent. In flexion, the posterior arch of C2 normally aligns itself in a straight-line fashion with C1 and C3. Note the relative horizontal nature of the facet joints, which allows greater mobility. (Reprinted from Swischuk LE. Anterior displacement of C2 in children: physiologic or pathologic? A helpful differentiating line. *Radiology* 1977;122 : 759-763; with permission.)

### Lower Cervical Spine

Evaluation of the lower cervical spine on a lateral radiograph should include examination of the alignment of the spinous processes, the spinal laminar line, the posterior vertebral body line, and the anterior vertebral body line ( Fig. 18-14 ). These should all be smooth curves with no step-offs or vertebral body translation. Loss of normal cervical lordosis may indicate spasm from a cervical injury but often is a normal finding in a child ( 169 ). Naidich's law states that at any level the interspinous distance may be one and a half times greater than the interspinous distance of the level above or the level below. A greater ratio indicates injury. Evaluation of a lateral cervical spine radiograph should include the bony anatomy as well as the soft tissue spaces. Changes in the soft tissue space may warn the physician of an underlying cervical spine injury when no abnormalities of the vertebrae or vertebral alignment are noted on plain radiographs. The normal retropharyngeal soft tissue space should be less than 6 mm at C3, and the retrotracheal space should be less than 14 mm at C6. Any increased widening may be indicative of an occult bony or ligamentous injury. Adran and Kemp ( 1 ) showed that the width of this soft tissue shadow may be markedly increased in a crying child because the pharynx is attached to the hyoid bone and any action displacing it forward artificially increases the width of this shadow. Therefore, radiographs must be taken with the patient at rest or during quiet respiration.



**FIGURE 18-14.** Normal relationships in the lateral cervical spine: 1, spinous processes; 2, spinolaminar line; 3, posterior vertebral body line; 4, anterior vertebral body line. (Reprinted from Copley LA, Dormans JP. Cervical spine disorders in infants and children. *J Am Acad Orthop Surg* 1998;6; with permission.)

### Special Imaging Studies

Special studies often are indicated for evaluation of pediatric cervical spine trauma. Most cervical spine injuries can be detected by routine plain radiographs ( 5). Flexion and extension plain radiographs often are needed to determine if any instability is present. Tomography, computed tomography (CT) scanning, CT myelography, magnetic resonance imaging (MRI), bone scanning, and cineradiography are useful adjunctive studies in the evaluation of cervical spine trauma. Of these, CT scanning to detect occult fractures is the most valuable in the trauma unit setting.

### Dynamic Studies

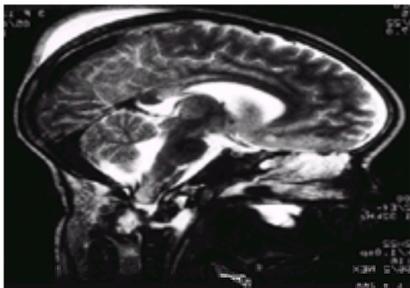
Useful tools in evaluating pediatric cervical spine problems are dynamic flexion and extension radiographs taken from a lateral view, although cineradiography occasionally may be necessary. Static lateral films should always be viewed before performing flexion and extension views. From the static radiographs the physician may suspect a cervical spine injury but the lateral flexion and extension views usually will determine the presence of any instability.

Again, it must be emphasized that these dynamic studies should only be performed under the direct supervision of the treating physician and only in a cooperative patient. Cineradiography allows evaluation of flexion and extension and rotation and tilt; however, it requires a high radiation dose and now is used only in rare cases. CT scanning and MRI have generally replaced cineradiography in the definition of cervical spine injuries in children.

Tomography can aid in identifying injuries that are not seen well on plain radiographs. Sagittal, coronal, and three-dimensional reconstructions from CT scanning can give similar information. Most of the information obtained from tomography can now be obtained with CT reconstruction views, which not only shows information in the axial plane, but also shows better detail of any fractures or bony lesions. Flexion and extension views are possible with CT scanning to evaluate any occult instability. Sagittal, coronal, or three-dimensional reconstruction views can be obtained to help identify fractures or instability patterns at sites that are not easily seen on plain radiographs. This is most useful in evaluating the lateral masses and occipital condyles for fractures and fracture–dislocations. One should take care when placing the child into a CT scanner so that the head is not inadvertently placed in flexion because of the large size of a child's head relative to the body. If this is not taken into account, the neck will be placed in flexion and potentially could increase displacement of an occult fracture. Often children under 4 years of age require sedation for CT scanning.

Magnetic resonance imaging has become a useful diagnostic study to evaluate both soft tissue or ligamentous injury and SCIWORA. MRI gives both bone and soft tissue information, but it is most useful in evaluating the spinal cord for occult injuries and soft tissue for occult swelling, edema, or ligamentous injuries. Closkey et al. described this as a useful tool for detecting cervical spine injuries in head-injured or unconscious patients when flexion and extension views cannot be safely obtained (32).

Myelography is useful in detecting abnormalities in the spinal canal but has largely been replaced by MRI ( Fig. 18-15).



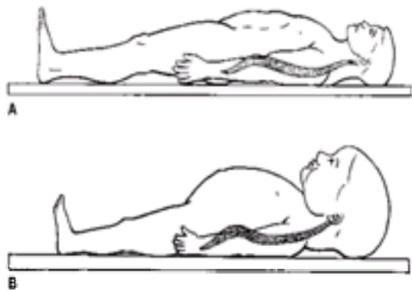
**FIGURE 18-15.** Magnetic resonance imaging depicts a ligament tear.

Another special study is arteriography, which is used to evaluate the vertebral arteries, especially in upper cervical spine trauma in young children. An MRI-enhanced technique can now be used to evaluate the vertebral arteries for injury and is not as invasive as arteriography. Therefore, cineradiography, myelography, and arteriography have been replaced by CT and MRI, which show better detail.

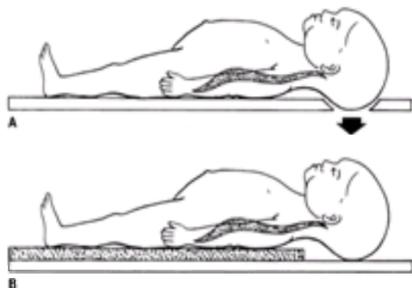
### INITIAL MANAGEMENT OF CERVICAL SPINE INJURIES

Care must be taken to immobilize the cervical spine to prevent damage to the spinal cord, especially while extracting an injured child from an automobile or during transportation. Various devices can be used to immobilize the cervical spine during extraction but should consist of splinting of the head and neck to the thorax. Any immobilization device must allow access to the patient's oropharynx and the anterior region of the neck for emergency intubation or tracheostomy if necessary.

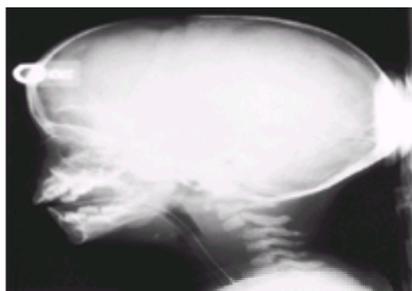
For transportation the child should be placed on a backboard. However, as Herzenberg et al. ( 79) have pointed out, the standard backboard used for adults is unsuitable for young children. Because the head of a young child is disproportionately large compared with the body, when placed on a standard board, the child's cervical spine is in flexion. This can translate the cervical spine forward into unstable fracture patterns. Herzenberg et al. ( 79) reported on 10 children under 7 years of age whose cervical spine had anterior angulation or translation on radiographs when placed on a standard backboard. They recommend a backboard with a recess so that the head can be lowered into it to obtain a neutral position of the cervical spine. This also can be accomplished with a split mattress technique, in which the body is supported by two mattresses, and the head is supported by one. Children under 6 years of age should be immobilized using a split mattress technique or a spine board with an occipital recess (Fig. 18-16 and Fig. 18-17) (79). One problem with this immobilization protocol, however, is that it tends to reduce displaced fractures and makes recognition of the injury more difficult, especially end plate fractures, which are similar to Salter I fractures in that when reduced the fracture line cannot be seen (Fig. 18-18) (78).



**FIGURE 18-16. A:** Adult immobilized on a standard backboard. **B:** Young child on a standard backboard. The relatively large head forces the neck into a kyphotic position. (Reprinted from Herzenberg JE, Hensinger RN, Dedrick DK, et al. Emergency transport and positioning of young children who have an injury of the cervical spine. The standard backboard may be hazardous. *J Bone Joint Surg [Am]* 1989;71 : 15-22; with permission.)



**FIGURE 18-17. A:** Young child on a modified backboard that has a cutout to the recess of the occiput, obtaining better supine cervical alignment. **B:** Young child on modified backboard that has a double-mattress pad to raise the chest, obtaining better supine cervical alignment. (Reprinted from Herzenberg JE, Hensinger RN, Dedrick DK, et al. Emergency transport and positioning of young children who have an injury of the cervical spine: the standard backboard may be hazardous. *J Bone Joint Surg [Am]* 1989;71 : 15-22; with permission.)



**FIGURE 18-18.** Lateral radiograph demonstrating anterior displacement of a fracture resulting from improper immobilization of a child on a flat spine board without an occipital recess.

If a child needs to be placed in a halo body jacket, this head–trunk relationship also should be remembered so that the cervical spine is not in flexion.

A large variety of pediatric cervical stabilization devices are available, including rigid cervical collars made out of plastic or soft foam ( 37,110). The rigid cervical collars give better immobilization than soft foam collars, but they may cause problems from pressure and often proper fitting may be difficult. Even the best immobilization allows 17 degrees of flexion, 19 degrees of extension, 4 degrees of rotation, and 6 degrees of lateral motion ( 110). Huerta et al. (84) recommended that these devices be supplemented with tape and sand bags on either side of the head to limit the amount of spinal motion to 3 degrees in any direction. If resuscitation is required, further displacement of an unstable cervical injury is a potential risk. A study of four patients with unstable cervical injuries who failed to be resuscitated in the emergency room showed that axial traction during the emergency treatment actually increased the deformity ( 15). Skull tongs and halo devices can be used in young children for immobilization ( 68), but care must be taken regarding the amount of pressure that can be used for the Crutchfield tongs or halo pins. There are special pediatric pin sets for the Crutchfield tongs, and if a halo device is used, then multiple pins at a lower torque are recommended.

Minerva cast immobilization is an effective means of immobilizing the cervical spine, but to place a well-fitted cast on a young child requires some attention to detail. Custom-made orthoses also can be used and have the advantages of easier care and adjustability. The disadvantage is that they do not immobilize the cervical spine as well as a properly applied Minerva cast ( Fig. 18-19). Thermoplastic materials also may cause significant skin problems from excoriation secondary to the plastic and contact dermatitis ( Fig. 18-20).



**FIGURE 18-19.** A 3-year-old with a fracture of the odontoid immobilized in a Minerva cast.

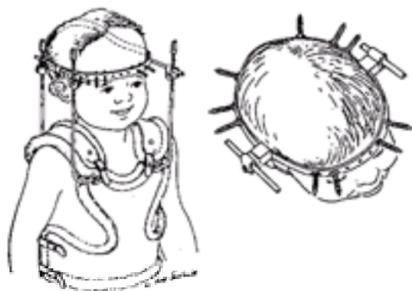


**FIGURE 18-20. A:** A child immobilized in a SOMI brace. **B:** Note the rash secondary to contact dermatitis.

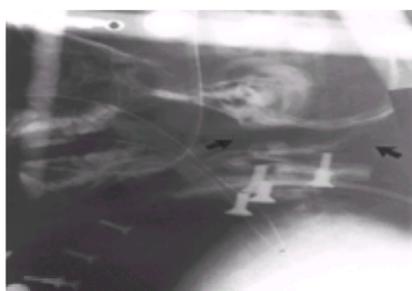
The halo device, introduced by Perry and Nickel (126) in 1959, provides immobilization of an unstable cervical spine and also can be used for traction in certain situations. The halo device can be used to immobilize the injured cervical spine in children, but the complications seen with the use of a halo device are much more frequent in children than in adults (11,44). Dormans et al. (44) reported a 68% complication rate with the use of halo immobilization in children, most commonly pin site infection and pin loosening. Other complications were dural penetration, supraorbital nerve injury, and pin site scars that were considered by the family to be objectionable. Despite this high complication rate, all patients were able to wear the halo until fracture healing or fusion occurred (45). A prefabricated halo vest has been used with good results in adults, and these vests can be easily fitted, but in children proper fit rarely is achieved and a halo cast or custom-molded halo vest is needed to obtain adequate fit and immobilization. If a cast or custom-fitted vest is not used, the head will be fixed in the halo and unwanted movement will take place in the vest (Fig. 18-21, Fig. 18-22 and Fig. 18-23).



**FIGURE 18-21.** Child immobilized in halo brace after occipital cervical arthrodesis.



**FIGURE 18-22. A:** Custom halo vest and light superstructure. **B:** In the multiple-pin, low-torque technique, 10 pins are used for an infant halo ring attachment. Usually, 4 pins are placed anteriorly, avoiding the temporal area, and the remaining 6 pins are placed in the occipital area. (Reprinted from Mubarak SJ, Camp JF, Fuleitich W, et al. Halo application in the infant. *J Pediatr Orthop* 1989;9:612–614; with permission.)



**FIGURE 18-23.** Patient with an atlantooccipital dislocation in a halo brace. Note the improper fitting of the vest and space between the vest and chest wall. This allows for significant displacement of atlantooccipital dislocation in the halo brace.

Mubarak et al. (112) recommended the following steps in the fabrication of a custom halo for a child: (a) the size and configuration of the head are obtained with the use of a flexible lead wire placed around the head; (b) the halo ring is fabricated by constructing a ring 2 cm larger in diameter than the wire model; (c) a plaster mold of the trunk is obtained for the manufacture of a custom bivalved polypropylene vest; and (d) linear measurements are made to ensure appropriate length of the superstructure.

Computed tomography scanning before halo application may help determine bone structure and skull thickness and aid in planning pin placement to avoid a suture line, congenital malformations, and sites that may be prone to pin penetration because of skull thickness. Skull thickness in children varies greatly up to 6 years of age (97) and increases between the ages of 10 and 16 years, after which time it is similar to that in adults. Letts found that a 2-mm skull could be completely penetrated with a 160-pound load, which is below the recommended torque pressure for adult skulls.

Mubarak et al. (112) recommended the use of multiple pins at lower torque pressures in infants and children. Eight to ten pins can be used and tightened to 2 to 4 inch-pounds, depending on the child's age. The use of multiple pins allows significantly less torque pressure. Although this technique is useful, skull development is an important factor to consider in halo application in patients under 2 years of age. The halo probably should not be used in children under 18 months of age: cranial suture interdigitation may be incomplete and fontanels may be open anteriorly in children under 18 months of age and posteriorly in children under 6 months of age. If

a halo is used in patients under 18 months of age, there is the potential for displacement through the unfused suture lines.

### Technique of Halo Application

Once constructed, the halo ring is applied with the patient under general anesthesia. In older children and adolescents, local anesthesia can be used. The patient is placed supine, and the head is supported by an assistant or a cupped metal extension that cradles the head. If a metal extension is used, care should be taken not to place the neck in flexion and to maintain the proper relationship of the head and neck with the trunk. The immediate areas of pin insertion are shaved and the skin is prepared with antiseptic solution. Selected areas in the skin and the periosteum are infiltrated with local anesthetic. With the help of an assistant and an application device, the halo is held around the patient's head. The halo is held below the area of greatest diameter of the skull, just above the eyebrows, and about 1 cm above the tips of the ears. The pin sites are carefully selected so that the pins enter the skull as nearly perpendicular as possible. The best position for the anterior pins is in the anterolateral aspect of the skull, above the lateral two thirds of the orbit, and below the greatest circumference of the skull; this area is a relatively safe zone. This will avoid injury to the supraorbital and supratrochlear nerves. Care should be taken to avoid the temporalis muscle because penetration of this muscle by the halo pin can be painful and may impede mandibular motion during mastication or talking; the bone in this area also is very thin, and pin loosening is likely.

The posterior pins are placed directly diagonal from the anterior pins, if possible, and inferior to the equator of the skull. The pins are introduced through the halo and the two diagonally opposed pins are tightened simultaneously. It is important that the patient's eyes are closed while the pins are tightened to ensure that the forehead skin is not anchored in such a way as to prevent the eyelids from closing after application of the halo.

In an infant or young child, 10 pins are inserted to finger tightness or 2 inch-pounds anterolaterally and posteriorly. If the skull thickness is of great concern, finger tightness only should be used to prevent penetrating the skull. In slightly older children, 2 inch-pounds of torque should be used. In adolescents near skeletal maturity whose skull thickness is nearly that of an adult (as determined by CT scan) torque pressure can be increased to as much as 6 to 8 inch-pounds. The pins are secured to the halo with the appropriate lock nuts or set screws. A custom polypropylene vest or cast is applied as well as the superstructure after the halo ring and pins are in place.

The pins should be cleaned daily at the skin interface with hydrogen peroxide or a small amount of povidone-iodine solution. The pins are tightened again 48 hours after application. If a pin becomes loose after halo application, it can be retightened as long as resistance is met. If no resistance is met, the pin should be removed and another pin inserted at another site. Superficial infections about the pin site can be treated with oral antibiotics and continued pin site care. If the cellulitis persists or an abscess forms, then the pins should be removed. Dural puncture can occur from the halo pins. When this occurs the pin should be removed and prophylactic antibiotics should be given. The dural tear usually heals in 4 to 5 days, at which time the antibiotics can be discontinued.

### SPINAL CORD INJURY WITHOUT RADIOGRAPHIC ABNORMALITIES

Spinal cord injury without radiographic abnormalities, a syndrome first brought to the attention of the medical community by Pang and Wilberger ( [122](#)), is unique to children. This condition is defined as a spinal cord injury in a patient with no visible fracture or dislocation on plain radiographs, tomograms, or CT scans.

A complete or incomplete spinal cord lesion may be present, and the injury usually results from severe flexion or distraction to the cervical spine. SCIWORA is believed to occur because the spinal column (vertebrae and disk space) in children is more elastic than the spinal cord and can undergo considerable deformation without being disrupted ( [26](#)). The spinal column can elongate up to 2 inches without disruption, whereas the spinal cord ruptures with only one fourth of an inch of elongation.

Spinal cord injury without radiographic abnormalities also may represent an ischemic injury in some patients, although most are believed to be due to a distraction-type injury in which the spinal cord has not tolerated the degree of distraction but the bony ligamentous elements have not failed. Aufdermaur suggested another possibility ( [8](#)): a fracture through a pediatric vertebral end plate reduces spontaneously (much like a Salter I fracture), giving a normal radiographic appearance, although the initial displacement could have caused spinal cord injury.

Spinal cord injury without radiographic abnormalities is more common in children under 8 years of age than in older children ( [122,124,139,169](#)). Predisposing factors that may explain the increased frequency of SCIWORA in younger children are cervical spine hypermobility, ligamentous laxity, and an immature vascular supply to the spinal cord. The reported incidence of this condition varies from 7% to 66% of patients with cervical spine injuries (Sullivan's incidence of SCIWORA was 5% to 67%) ( [121,122,175](#)).

Delayed onset of neurologic symptoms have been reported in as many as 52% of patients in some series ( [122,139](#)). Pang and Pollock reported 15 patients who had delayed paralysis after their injury ( [121](#)). Nine had transient warning signs such as paresthesia or subjective paralysis. In all patients with delayed onset of paralysis, the spine had not been immobilized after the initial trauma and all were neurologically normal before the second event. This underlines the importance of diligent immobilization of a suspected spinal cord injury in a child. Approximately half of the young children with SCIWORA in reported series had complete spinal cord injuries, whereas the older children usually had incomplete neurologic deficit injuries that involved the subaxial cervical spine ( [6,8,69,139](#)).

Careful radiographic evaluation is helpful in the workup of these patients, but MRI will show a spinal cord lesion that often is some distance from the vertebral column injury. As many as 5% to 10% of children with spinal cord injuries have normal radiographic results ( [69,75](#)).

### SPINAL CORD INJURY IN CHILDREN

Spinal cord injuries are still rare in children. Rang reviewed spinal injuries at the Toronto Hospital for Sick Children over 15 years and found that children constituted a small percentage of the patients with acquired quadriplegia or paraplegia ( [134](#)). He found that paraplegia was three times more common than quadriplegia. When a spinal cord injury is suspected, the neurologic examination must be complete and meticulous and may take several examinations of sensory and motor function. If an acute spinal cord injury is documented by examination, the administration of methylprednisolone within the first 8 hours after injury has been shown to improve the chances of neurologic recovery ( [18, 19, 20](#) and [21](#)). Methylprednisolone in the treatment of acute spinal cord injuries has been shown to improve motor and sensory recovery when evaluated 6 weeks and 6 months postinjury ( [20](#)); however, this positive effect on neurologic recovery is limited to those treated within the first 8 hours of injury. The initial loading dose of methylprednisolone is 30 mg/kg body weight. If the loading dose is given within 3 hours after injury, then a maintenance infusion of 5.4 mg/kg is given for 24 hours after injury. If the loading dose is given between 3 and 8 hours after injury, then a maintenance infusion of 5.4 mg/kg is given for 48 hours after injury. Methylprednisolone decreases edema, has an antiinflammatory effect, and protects the cell membranes from scavenging oxygen free radicals ( [18, 19, 20](#) and [21](#)).

In several series ( [18, 19, 20](#) and [21](#)) there was a slight increase in the incidence of wound infections, but no significant increase in gastrointestinal bleeding. All of these studies involved patients 13 years or older, so no documentation of the efficacy in young children exists. A combination of methylprednisolone and GM1 ganglioside is being studied for its possible beneficial effect on an injured spinal cord ( [61, 62, 63](#) and [64](#)). GM1 is a complex acidlike lipid found at high levels in the cell membrane of the central nervous system that is thought to have a neuroprotective and neurofunctional restorative potential. Early studies have shown that patients given both drugs have had improved recovery over those who had received just methylprednisolone.

Once spinal cord injury is documented, routine care includes prophylaxis for stress ulcers, routine skin care to prevent pressure sores, and initial Foley catheterization followed by intermittent catheterization and a bowel training program. With incomplete lesions, children have a better chance than adults for useful recovery. Hadley et al. ( [69](#)) noted that 89% of pediatric patients with incomplete spinal cord lesions improved, whereas only 20% of patients with complete injuries had evidence of significant recovery. Laminectomy has not been beneficial and can be harmful ( [151,174](#)) because it increases instability in the cervical spine. For example, it can cause a swan neck deformity or progressive kyphotic deformity ( [104,154](#)). The risk of spinal deformity after spinal cord injury has been investigated by several researchers ( [10,12,27,49,90,104](#)). Mayfield et al. ( [104](#)) found that patients who had a spinal cord injury before their growth spurt all developed spinal deformities, 80% of which were progressive. Ninety-three percent developed scoliosis, 57% developed kyphosis, and 18% lordosis. Sixty-one percent of these patients required spinal arthrodesis for stabilization of their curves. Orthotic management usually is unsuccessful, but in some patients it delays the age at which arthrodesis is necessary. Lower extremity deformities also may occur, such as subluxations and dislocations about the hip. Pelvic obliquity can be a significant problem and may result in pressure sores and difficulty in seating in a wheelchair.

### NEONATAL INJURY

Spinal column injury and spinal cord injury can occur during birth, especially during a breech delivery ( [117,163](#)). Injuries associated with breech delivery usually are in the lower cervical spine or upper thoracic spine and are thought to result from traction, whereas injuries associated with cephalic delivery usually occur in the upper cervical spine and are thought to result from rotation. It is unclear whether cesarean section reduces spinal injury in neonates ( [100](#)); however, Bresnan and Abrams ( [22](#)) noted that neck hyperextension *in utero* (star-gazing fetus) in breech presentations is likely to result in an estimated 25% spinal cord injury with vaginal delivery and can be prevented by delivering via cesarean section.

Distraction type injuries to the upper cervical spine have been reported in infants in forward-facing car seats. Because infants have poor head control and muscular development, if placed in a forward facing car seat and a sudden deceleration occurs, the infant's head continues forward while the remainder of the body is strapped in the car seat, resulting in a distraction-type injury ( [66](#)).

Neuromuscular control of the cervical spine in neonates and infants is underdeveloped, and a normal infant cannot adequately support his or her head until about 3 months of age. Infants, therefore, cannot protect their spines against excessive forces that may occur during delivery or during the months after birth. Skeletal injuries from obstetric trauma are probably under-reported because the infantile spine is largely cartilaginous and difficult to evaluate radiographically, especially if the injury is through the cartilage or cartilage–bone interface ( [8](#)). A cervical spine lesion should be considered in an infant who is floppy at birth, especially after a difficult delivery. Flaccid paralysis with areflexia usually is followed by a typical pattern of hyperreflexia once spinal cord shock is over. Brachial plexus palsies also warrant cervical spine radiographs. MRI can sometimes be helpful in this diagnosis.

Shulman et al. ( [153](#)) found atlantooccipital and axial dislocations at autopsy, and Tawbin ( [163](#)) found a 10% incidence of brain and spinal injuries at autopsy.

Treatment of the neonatal cervical spine is nonoperative and should consist of careful realignment and positioning of the child on a bed with neck support or a custom cervical thoracic orthosis. Healing of bony injuries is usually rapid and complete ( [159](#)).

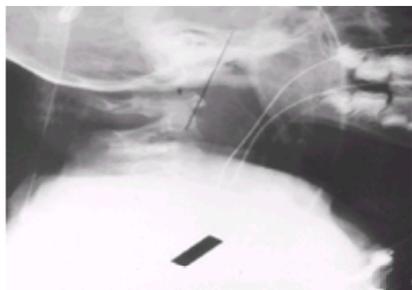
Caffey ( [30](#)) in 1974 and Swischuk ( [162](#)) in 1969 described a child abuse syndrome called the shaken infant syndrome. Children have weak and immature neck musculature and cannot support their heads when they are subjected to whiplash stresses. Intracranial and interocular hemorrhages can occur. This injury can result in death or cerebral injury with retardation and permanent visual and hearing defects. Fractures of the spinal column and spinal cord injuries can occur after the violent shaking of the child. Swischuk reported a spinal cord injury in a 2-year-old that was the result of violent shaking that produced a cervical fracture dislocation that spontaneously reduced ( [162](#)).

## OCCIPITAL CONDYLAR FRACTURE

Occipital condylar fractures are rare, and their diagnosis requires a high index of suspicion, especially in patients who are unconscious or have a closed head injury or cranial nerve injury. CT with multiplanar reconstruction usually is necessary to establish the diagnosis. Three types of occipital condylar fractures have been described: type I, impaction fracture; type II, basilar skull fracture extending into the condyle; and type III, avulsion fractures. An avulsion fracture is the only type of occipital condylar fracture that is unstable. Type I injuries are the result of axial compression. Type II injuries occur after a direct blow. Both type I and II occipital condylar fractures can be treated with a cervical orthosis. Type III or avulsion fractures require halo immobilization or occipitocervical arthrodesis ( [3](#)).

## ATLANTOCCIPITAL INSTABILITY

Atlantooccipital dislocation was once thought to be a rare fatal injury found only at the time of autopsy ( [Fig. 18-24](#)) ( [8,16,24,153](#)). This injury is now being recognized more often, and children are surviving ( [44,123,158](#)). This increase in the survival rate may be due to increased awareness and improved emergency care with resuscitation and spinal immobilization by emergency personnel. Atlantooccipital dislocation occurs in sudden deceleration accidents, such as motor vehicle or pedestrian–vehicle accidents. The head is thrown forward, and this may cause sudden craniovertebral separation.



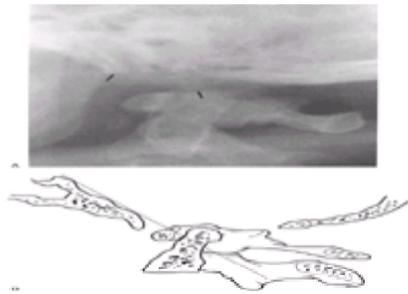
**FIGURE 18-24.** Patient with atlantooccipital dislocation. Note the forward displacement of the Wackenheim line and the significant anterior soft tissue swelling.

The atlantooccipital joint is a condylar joint that has little inherent bony stability. Stability is provided by the ligaments about the joint. The primary stabilizers are the paired alar ligaments and the tectorial membrane (a continuation of the posterior longitudinal ligament). In children, this articulation is not as well formed as in adults and it is less cup shaped. Therefore, there is less resistance to translational forces ( [8,16,24,25,153](#)).

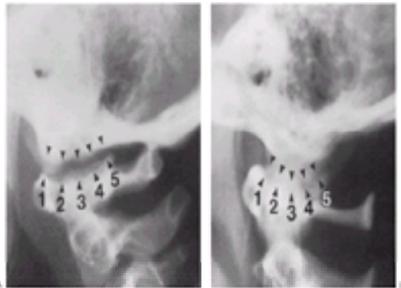
Diagnosis may be difficult, because atlantooccipital dislocation is a ligamentous injury. Although patients with this injury have a history of trauma, some may have no neurologic findings. Others, however, may have symptoms such as cranial nerve injury, vomiting, headache, torticollis, or motor or sensory deficits ( [24,29,33,74,81,123](#)). Brainstem symptoms, such as ataxia and vertigo, may be caused by verteobasilar vascular insufficiency. Unexplained weakness or difficulty in weaning off a ventilator after a closed head injury may be a sign of this injury.

The treating physician must be aware of radiographic findings associated with atlantooccipital dislocation. A significant amount of anterior soft tissue swelling usually is present.

Radiographic findings that aid in the diagnosis of atlantooccipital dislocation are the Wackenheim line, Powers ratio, dens–basion interval, and occipital condylar distance. The Wackenheim line is drawn along the clivus and should intersect tangentially the tip of the odontoid. A shift anterior or posterior of this line represents either an anterior or posterior displacement of the occiput on the atlas ( [Fig. 18-25](#)). This line is probably the most helpful because it is reproducible and easy to identify on a lateral radiograph. The Powers ratio is determined by drawing a line from the basion to the posterior arch of the atlas and a second line from the opisthion to the anterior arch of the atlas. The length of line BC is divided by the length of the line OA, producing the Powers ratio. A ratio of more than 1.0 is diagnostic of anterior atlantooccipital dislocation. A ratio of less than 0.7 is diagnostic of posterior atlantooccipital dislocation. Values between 1.0 and 0.7 are considered normal. Another radiographic measurement is the dens–basion interval. If the interval measures more than 1.2 cm, then disruption of the atlantooccipital joint has occurred ( [25,129](#)). Donahue et al. ( [43](#)) described an occipital condylar facet distance of more than 5 mm from the occipital condyle to the C1 facet as indicative of atlantooccipital injury. They recommended measuring this distance from five reference points along the occipital condyle and the C1 facet ( [Fig. 18-26](#)).



**FIGURE 18-25.** Craniovertebral dislocation. **A:** Lateral view shows extensive soft tissue swelling. The distance between the basion and the dens is 2.4 cm ( *arrows*) (normal is <1 cm). **B:** Line drawing made to demonstrate the abnormal relationship between the occiput and the upper cervical spine. (Reprinted from El-Khoury GY, Kathol MH. Radiographic evaluation of cervical trauma. *Semin Spine Surg* 1991;3:3–23; with permission.)



**FIGURE 18-26.** Atlantooccipital joint measurement points 1 through 5 demonstrated on normal cross-table lateral skull radiograph in an 8-year-old (**A**) and a 14-year-old (**B**). (Reprinted from Kaufman RA, Carroll CD, Buncher CR. Atlantooccipital junction: standards for measurement in normal children. *AJNR* 1987;8:995–999; with permission.)

Magnetic resonance imaging also is useful in diagnosing atlantooccipital dislocation by showing soft tissue edema and ligament injury or disruption.



## OPERATIVE TREATMENT

Because atlantooccipital dislocation is a ligamentous injury, nonoperative treatment usually is unsuccessful. Although Georgopoulos et al. ( [65](#)) did report successful halo stabilization, immobilization in a halo should be used with caution: if the vest or cast portion is not fitted properly, displacement can increase ( [Fig. 18-27](#)). Traction should be avoided for the same reason. Surgical stabilization is the recommended treatment ( [99](#)). Posterior arthrodesis can be performed *in situ*, with wire fixation or fixation with a contoured Luque rod and wires. If the C1–C2 articulation is stable, arthrodesis should be only from the occiput to C1 so that C1–C2 motion is preserved ( [157](#)). Some researchers have expressed reservations about the chance of obtaining fusion in the narrow atlantooccipital interval and have recommended arthrodesis from the occiput to C2. If stability of the C1–C2 articulation is questionable, arthrodesis should extend to C2 ( [106](#)).



**FIGURE 18-27.** **A:** Lateral radiograph of a patient with atlantooccipital dislocation. Note the increase in the facet condylar distance. **B:** Lateral radiograph after occipital C1 arthrodesis.

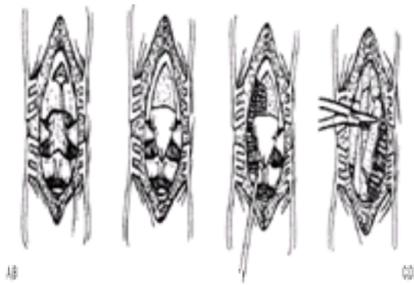
For a patient who presents very late with an unreduced dislocation, an *in situ* arthrodesis is recommended. DiBenedetto and Lee ( [41](#)) recommended arthrodesis *in situ* with a suboccipital craniectomy to relieve posterior impingement.

Instability at the atlantooccipital joint is increased in patients with Down's syndrome as well as those with a high cervical arthrodesis below the axis. These patients may be at risk of developing chronic instability patterns and are at higher risk of having instability after trauma.

### Occiput to C2 Arthrodesis

#### *Arthrodesis without Internal Fixation*

In younger children in whom the posterior elements are absent at C1 or separation is extensive in the bifid part of C1 posteriorly, posterior cervical arthrodesis from the occiput to C2 with iliac crest bone graft is performed using a periosteal flap from the occiput to provide an osteogenic tissue layer for the bone graft ( [Fig. 18-28](#)) ( [93](#)).



**FIGURE 18-28.** Technique of occipitocervical arthrodesis used when posterior arch of C1 is absent. **A:** Exposure of the occiput, atlas, and axis. **B:** Reflection of periosteal flap to cover defect in atlas. **C:** Decortication of exposed vertebral elements. **D:** Placement of autogenous cancellous iliac bone grafts. (Redrawn from Koop SE, Winter RB, Lonstein JE. The surgical treatment of instability of the upper part of the cervical spine in children and adolescents. *J Bone Joint Surg [Am]* 1984;66:403; with permission.)

A halo is applied after the patient is anesthetized, endotracheal intubation is obtained, and all anesthesia lines are in place. For younger children, 6 to 10 pins with lower pressure torque are used in the halo ([Fig. 18-21](#)); in older children, 4 pins can be used.

A radiograph is obtained to evaluate the position of the head and cervical spine in the prone position with the halo in place. The radiograph also aids in identifying landmarks and levels, although once the skin incision is made, the occiput and spinous processes can be palpated.

A straight posterior incision is made from the occiput to about C3, with care not to expose below C2 to avoid extension of the fusion to lower levels. An epinephrine and lidocaine solution is injected into the cutaneous and subcutaneous tissues to help control local skin and subcutaneous bleeding. The incision is deepened in the midline to the spinous processes of C2. Once identified, the level of the posterior elements of C1 or the dura is more easily found.

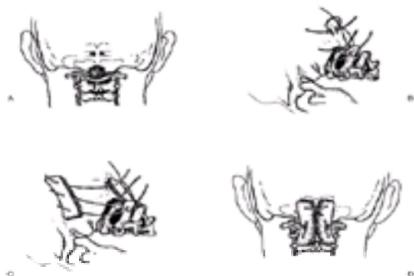
After C2 is identified, subperiosteal dissection is carried proximally. Extraperiosteal dissection is used to approach the occiput ([Fig. 18-28A](#)). The dura is not completely exposed; if possible, any fat or ligamentous tissue present is left intact. The interspinous ligaments also should be left intact.

The occipital periosteum is mobilized by making a triangular incision directly on the posterior skull, with the apex posteriorly and the broad base over the foramen magnum region. A flap of 3 or 4 cm at the base can be created. With subperiosteal elevation, the periosteum can be reflected from the occiput to the spinous processes of C2 ([Fig. 18-28B](#)). The apex of the flap is sutured to the spinous process of C2 and is attached laterally to any posterior elements that are present at C1 or other lateral soft tissues. After the periosteum is secured to the bone and any rudimentary C1 ring is exposed subperiosteally, a power bur is used to decorticate the occiput and any exposed portions of C1 and C2 ([Fig. 18-28C](#)).

Iliac crest bone graft is harvested, and struts of iliac bone are placed across the area on the periosteal flap ([Fig. 18-28D](#)). No internal fixation is used other than sutures to secure the periosteum. The wound is closed in a routine fashion, and a body jacket or cast is applied and attached to the halo. The halo cast is worn until radiographs show adequate posterior arthrodesis, usually in 8 to 12 weeks.

#### **Arthrodesis with Triple-Wire Fixation**

In older adolescents in whom the posterior elements of C1 and C2 are intact, a triple-wire technique, as described by Wertheim and Bohlman ([171](#)), can be used ([Fig. 18-29](#)). The wires are passed through the outer table of the skull at the occipital protuberance. Because the transverse and superior sagittal sinuses are cephalad to the protuberance, they are not endangered by wire passage.



**FIGURE 18-29.** Technique of occipitocervical arthrodesis used in older adolescents with intact posterior elements of C1 and C2. **A:** A bur is used to create a ridge in the external occipital protuberance, and then a hole is made in the ridge. **B:** Wires are passed through the outer table of the occiput, under the arch of the atlas, and through the spinous process of the axis. **C:** Corticocancellous bone grafts are placed on the wires. **D:** Wires are tightened to secure grafts in place. (Redrawn from Wertheim SB, Bohlman HH. Occipitocervical fusion: indications, technique, and long-term results. *J Bone Joint Surg [Am]* 1987;69:833; with permission.)

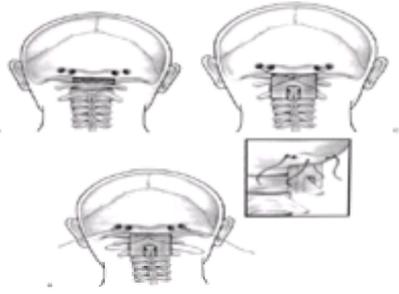
Stability of the spine is obtained preoperatively with cranial skeletal traction with the patient on a turning frame or cerebellar head rest. The patient is placed prone, and a lateral radiograph is obtained to document proper alignment. The subcutaneous tissues are injected with an epinephrine solution (1 : 500,000). A midline incision is made extending from the external occipital protruberance to the spine of the third cervical vertebra. The paraspinal muscles are sharply dissected subperiosteally with a scalpel, and a periosteal elevator is used to expose the occiput and cervical laminae, with special care to stay in the midline to avoid the paramedian venous plexus. At a point 2 cm above the rim of the foramen magnum, a high-speed diamond bur is used to create a trough on either side of the protuberance, making a ridge in the center ([Fig. 18-29A](#)). A towel clip is used to make a hole in this ridge through only the outer table of bone. A 20-gauge wire is looped through the hole and around the ridge, then another 20-gauge wire is looped around the arch of the atlas. A third wire is passed through a hole drilled in the base of the spinous process of the axis and around this structure, giving three separate wires to secure the bone grafts on each side of the spine ([Fig. 18-29B](#)).

A thick, slightly curved graft of corticocancellous bone of premeasured length and width is removed from the posterior iliac crest. The graft is divided horizontally into two pieces, and three holes are drilled into each graft ([Fig. 18-29C](#)). The occiput is decorticated and the grafts are anchored in place with the wires on both sides of the spine ([Fig. 18-29D](#)). Additional cancellous bone is packed around and between the two grafts. The wound is closed in layers over suction drains.

Either a rigid cervical orthosis or a halo cast is worn for 6 to 15 weeks, followed by a soft collar that is worn for an additional 6 weeks.

#### **Occipitocervical Arthrodesis**

The positioning of the patient and the procedure are performed with the patient under general anesthesia and with monitoring of the somatosensory-evoked potentials ([Fig. 18-30](#)). A halo ring is applied initially with the patient supine. Subsequently, the patient is carefully placed in the prone position, the halo is secured to the operating table with a halo-positioning device, and the alignment of the occiput and the cervical spine is confirmed with a lateral radiograph. The midline is exposed from the occiput to the second or third cervical vertebra. Particular care is taken to limit the lateral dissection to avoid damaging the vertebral arteries.



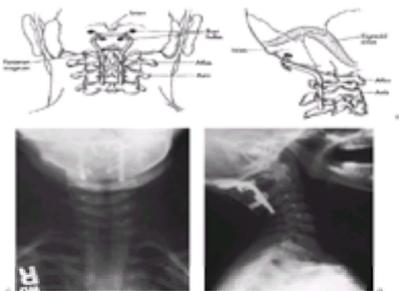
**FIGURE 18-30.** Occipitocervical arthrodesis. **A:** Four bur holes are placed into the occiput in transverse alignment, with two on each side of the midline, leaving a 1-cm osseous bridge between the 2 holes of each pair. A trough is fashioned into the base of the occiput. **B:** 16- or 18-gauge Luque wires are passed through the bur holes and looped on themselves. Wisconsin button wires are passed through the base of the spinous process of either the second or third cervical vertebra. The graft is positioned into the occipital trough and spinous process of the cervical vertebra at the caudal extent of the arthrodesis. The graft is locked into place by the precise contouring of the bone. **C:** The wires are crossed, twisted, and cut. The extension of the cervical spine can be controlled by positioning of the head with the halo frame, by adjustment of the size and shape of the bone graft, and to a lesser extent by tightening of the wires. (Reprinted from Dormans JP, Drummond DS, Sutton LN, et al. Occipitocervical arthrodesis in children. *J Bone Joint Surg [Am]* 1995;77:1234–1240; with permission.)

In patients who need decompression because of cervical stenosis or for removal of a tumor, the arch of the first or second cervical vertebra (or both) is removed, with or without removal of a portion of the occipital bone to enlarge the foramen magnum.

Four holes, aligned transversely, with two on each side of the midline, are made with a high-speed drill through both cortices of the occiput, leaving a 1-cm osseous bridge between the two holes of each pair. The holes are placed caudal to the transverse sinuses. A trough is fashioned into the base of the occiput to accept the cephalad end of the bone graft. A corticocancellous graft is obtained from the iliac crest and is shaped into a rectangle, with a notch created in the inferior base to fit around the spinous process of the second or third cervical vertebra. The caudal extent of the intended arthrodesis (the second or third cervical vertebra) is determined by the presence or absence of a previous laminectomy, congenital anomalies, or the level of the instability. On each side, a looped 16- or 18-gauge Luque wire is passed through the bur holes and looped on itself. Wisconsin button wires (Zimmer, Warsaw, Indiana) are passed through the base of the spinous process of either the second or the third cervical vertebra. The wire that is going into the left arm of the graft is passed through the spinous process from right to left. The graft is placed into the occipital trough superiorly and about the spinous process of the vertebra that is to be at the caudal level of the arthrodesis (the second or third cervical vertebrae). The graft is precisely contoured so that it fits securely into the occipital trough and around the inferior spinous process before the wires are tightened. The wires are subsequently crossed, twisted, and cut. An intraoperative radiograph is made at this point to assess the position of the graft and the wires as well as the alignment of the occiput and the cephalad cervical vertebrae. Extension of the cervical spine can be controlled by positioning of the head with the halo frame, by adjustment of the size and shape of the graft, and to a lesser extent by appropriate tightening of the wires.

#### **Occipitocervical Arthrodesis with Contoured Rod and Segmental Wire**

Occipitocervical arthrodesis using a contoured rod and segmental wire has the advantage of achieving immediate stability of the occipitocervical junction ( [Fig. 18-31](#)). This allows the patient to be in a cervical collar after surgery, avoiding the need for halo immobilization.



**FIGURE 18-31.** A–D: Occipitocervical arthrodesis using a contoured rod and segmental wire or cable fixation. ( **A** and **B** reprinted from Warner WC. Pediatric cervical spine. In: Canale ST, ed. *Campbell's operative orthopaedics*. St. Louis: Mosby Yearbook, 1998; with permission.)

The base of the occiput and the spinous processes of the upper cervical vertebrae are approached through a longitudinal midline incision, which extends deep within the relatively avascular intermuscular septum. The entire field is exposed subperiosteally. A template of the intended shape of the stainless steel rod is made with the appropriate length of Luque wire. Two bur holes are made on each side, about 2 cm lateral to the midline and 2.5 cm above the foramen magnum. Care should be taken to avoid the transverse and sigmoid sinus when making these bur holes. At least 10 mm of intact cortical bone should be left between the bur holes to ensure solid fixation. Luque wires or Songer cables are passed in an extradural plane through the two bur holes on each side of the midline. The wires or cables are passed sublaminar in the upper cervical spine. The rod is bent to match the template; this usually will have a head–neck angle of about 135 degrees and slight cervical lordosis. A Bend Meister (Sofamor/Danek, Memphis, Tennessee, U.S.A.) may be helpful in bending the rod. The wires or cables are secured to the rod. The spine and occiput are decorticated and autogenous cancellous bone grafting is performed.

## **FRACTURES OF THE ATLAS**

Fracture of the ring of C1 (Jefferson fracture) is not a common injury in children ( [15,86,102,109,114,136,165](#)). This injury is caused by an axial load applied to the head. The force is transmitted through the occipital condyles to the lateral masses of C1, causing a disruption in the ring of C1, usually in two places, with fractures occurring both in the anterior and posterior rings. In children an isolated single fracture of the ring can occur with the remaining fracture hinging on a synchondrosis. This is an important distinction in children because often fractures occur through a normal synchondrosis and there can be plastic deformation of the ring. This distinction can be seen on plain radiographs, with fractures appearing through what appears to be normal physes. As the lateral masses separate, the transverse ligament may be ruptured or avulsed, resulting in C1 and C2 instability ( [109](#)). If the two lateral masses are widened more than 7 mm beyond the borders of the axis on an anteroposterior radiograph, then an injury to the transverse ligament is presumed. Injury to the transverse ligament may be from a rupture of the ligament or an avulsion of the ligament attachment to C1. Jefferson fractures may be evident on plain radiographs, but CT scans are superior at demonstrating this injury. CT scans also may be used to follow the progress of healing.

Treatment consists of immobilization in an orthosis [Philadelphia collar or sternal occipital mandibular immobilizer (SOMI)], Minerva cast, or halo brace. If there is excessive widening (>7 mm), halo traction followed by halo brace or cast immobilization is recommended. Surgery rarely is necessary to stabilize these fractures ( [Fig. 18-32](#)).



**FIGURE 18-32.** Radiograph of an atlas fracture.

## ATLANTOAXIAL INJURIES

### Odontoid Fractures

Odontoid fractures are one of the most common fractures of the cervical spine in children (107), occurring at an average age of 4 years (45,146). The unique feature of odontoid fractures in children is that the fracture most commonly occurs through the synchondrosis of C2 distally at the base of the odontoid. This synchondrosis is a cartilage line at the base of the odontoid and looks like a physeal or Salter I injury.

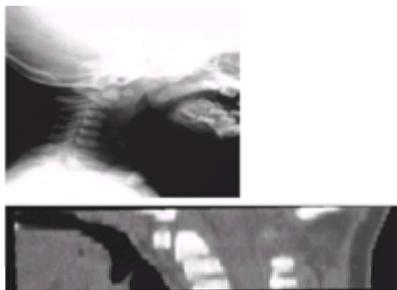
A fracture of the odontoid usually is associated with head trauma from a motor vehicle accident or a fall from a height, although it also can occur after trivial head trauma. Radiographs should be obtained in any child complaining of neck pain. Clinically, the child will complain of neck pain and resist attempts to extend the neck. Odent et al. (118) reported that 8 of 15 odontoid fractures in children were the result of motor vehicle accidents, with the child fastened in a forward-facing seat. The sudden deceleration of the body as it is strapped into the car seat while the head continues to travel forward causes this fracture.

Most odontoid injuries are anteriorly displaced and usually have an intact anterior periosteal sleeve that provides some stability to the fracture when immobilized in extension and allows excellent healing of the fracture (7,137,142,149). Growth disturbances are uncommon after this type of fracture. This synchondrosis normally closes at about 3 to 6 years of age and adds little to the longitudinal growth of C2.

Most often the diagnosis can be ascertained by viewing the plain radiographs. Anteroposterior radiographs usually appear normal, and the diagnosis must be made from lateral radiographs because displacement of the odontoid usually occurs anteriorly. Plain radiographs sometimes can be misleading when the fracture occurs through the synchondrosis and has spontaneously reduced. When this occurs the fracture has the appearance of a nondisplaced Salter I fracture. CT scans with three-dimensional reconstruction views may be needed to fully delineate the injury (150). MRI also may be useful in nondisplaced fractures by detecting edema around the injured area, indicating that a fracture may have occurred. Dynamic flexion and extension views to demonstrate instability may be obtained in a cooperative child if a nondisplaced fracture is suspected.

Odontoid fractures in children generally heal uneventfully and rarely have complications. Neurologic deficits rarely have been reported after this injury, except by Odent et al., who described neurologic injuries in 8 of 15 patients, although most were stretch injuries to the spinal cord at the cervical thoracic junction and not at the level of the odontoid fracture.

Treatment of odontoid fractures is by closed reduction (usually extension or slight hyperextension of the neck), although complete reduction of the translation is not necessary. At least 50% apposition should be obtained to provide adequate cervical alignment, then the patient should be immobilized in a Minerva or halo cast or custom orthosis. This fracture will heal in about 6 to 8 weeks. After bony healing, stability should be documented by flexion/extension lateral radiographs. Once the Minerva cast or halo is removed, a soft collar is worn for 1 to 2 weeks. If an adequate reduction cannot be obtained by recumbency and hyperextension, then head halter or halo traction is needed. Rarely, manipulation under general anesthesia is needed for irreducible fractures (Fig. 18-33). Surgery with internal fixation rarely has been reported, due to the good results that are achieved with conservative treatment in children (57,67,130,135,141).



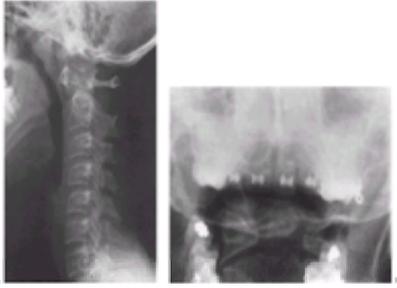
**FIGURE 18-33. A and B:** Lateral radiograph and CT reconstruction view of odontoid fracture through the synchondrosis of C2. Note the anterior displacement.

### Os Odontoideum

Os odontoideum consists of a round ossicle that is separated from the axis by a transverse gap, which leaves the apical segment without support. Fielding et al. (51,53) suggested that this was an unrecognized fracture at the base of the odontoid. Some studies have documented normal radiographs of the dens with abnormal radiographs after trivial trauma. This can be explained by a distraction force being applied by the alar ligaments, which pulls the tip of the fractured odontoid away from the base and produces a nonunion.

The presentation of an os odontoideum can be variable. Signs and symptoms can range from a minor to a frank compressive myelopathy or vertebral artery compression. Presenting symptoms may be neck pain, torticollis, or headaches caused by local irritation of the atlantoaxial joint. Neurologic symptoms can be transient or episodic after trauma to complete myelopathy caused by cord compression (46). Symptoms may consist of weakness and loss of balance with upper motor neuron signs, although upper motor neuron signs may be completely absent. Proprioceptive and sphincter dysfunction also are common.

Os odontoideum usually can be diagnosed on routine cervical spine radiographs, which include an open mouth odontoid view (Fig. 18-34). Lateral flexion and extension views should be obtained to determine if any instability is present. With os odontoideum there is a space between the body of the axis and a bony ossicle. The free ossicle of the os odontoideum usually is half the size of a normal odontoid and is oval or round with smooth sclerotic borders. The space differs from that of an acute fracture in which the space is thin and irregular instead of wide and smooth. The amount of instability should be documented on lateral flexion and extension plain radiographs that allow measurement of both the anterior and posterior displacement of the atlas on the axis. Because the ossicle is fixed to the anterior arch of C1 and moves with the anterior arch of C1 both in flexion and extension, measurement of the relationship of C1 to the free ossicle is of little value because they move as a unit. A more meaningful measurement is made by projecting lines superiorly from the body of the axis to a line projected inferiorly from the posterior border of the anterior arch of the atlas. This gives more information as to the stability of C1–C2. Another measurement that is very helpful is space available for the cord, which is the distance from the back of the dens to the anterior border of the posterior arch of C1.

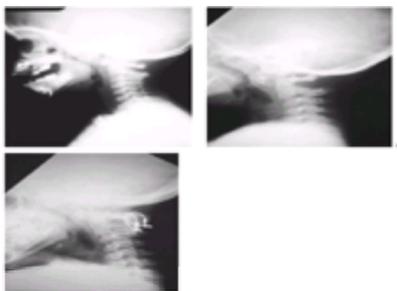


**FIGURE 18-34.** Lateral radiograph (A) and open-mouth odontoid radiograph (B) demonstrating os odontoideum. (Reprinted from Warner WC. Pediatric cervical spine. In: Canale ST, ed. *Campbell's operative orthopaedics*. St. Louis: Mosby Year Book, 1999:2817; with permission.)

Recommended treatment is posterior arthrodesis of C1 to C2. Before arthrodesis is attempted, the integrity of the arch of C1 must be documented by CT scan. Incomplete development of the posterior arch of C1 is uncommon but has been reported to occur with increased frequency in patients with os odontoideum. This may necessitate an occipital to C2 arthrodesis for stability. If a C1–C2 arthrodesis is performed, one must be careful not to overreduce the odontoid and cause posterior translation. Care also must be taken in positioning the neck at the time of arthrodesis and when tightening the wires if a Gallie or Brooks arthrodesis is performed to prevent posterior translation ([Fig. 18-35](#) and [Fig. 18-36](#)).



**FIGURE 18-35.** Posterior translation of atlas after C1–C2 posterior arthrodesis.



**FIGURE 18-36.** A: Lateral radiograph of traumatic C1–C2 instability. B: Note the increase in the atlanto-dens interval. C: Lateral radiograph after C1–C2 posterior arthrodesis.

### Traumatic Ligamentous Disruption

The transverse ligament is the primary stabilizer of an intact odontoid against forward displacement. Secondary stabilizers consist of the apical and alar ligaments, which arise from the tip of the odontoid and pass to the base of the skull. These also stabilize the atlantooccipital joint indirectly ([80](#)). The normal distance from the anterior cortex of the dens to the posterior cortex of the anterior ring of C1 is 3 mm in adults and 4.5 mm in children. In children if the distance is more than 4.5 mm, disruption of the transverse ligament is presumed. The spinal canal at C1 is large compared with other cervical segments and accommodates a large degree of rotation and some degree of pathologic displacement without compromising the spinal cord. Steel has expressed this as a rule of thirds. The spinal canal at C1 is occupied equally by the spinal cord, odontoid, and a free space, which provides a buffer zone to prevent neurologic injury. Steel found that anterior displacement of the atlas that exceeds a distance equal to the width of the odontoid may place the spinal cord at risk ([160](#)).

Acute rupture of the transverse ligament is rare and reportedly occurs in fewer than 10% of pediatric cervical spine injuries ([98,107](#)). However, avulsion of the attachment of the transverse ligament to C1 is more common.

A patient with disruption of the transverse ligament usually has a history of cervical spine trauma and complains of neck pain, often with notable muscle spasms. Diagnosis is confirmed on lateral radiographs that demonstrate an increased atlanto-dens interval. An active flexion view may be required to demonstrate instability in cooperative patients with unexplained neck pain or neurologic findings. CT scans are useful to demonstrate avulsion of the transverse ligament from its origins to the bony ring of C1.

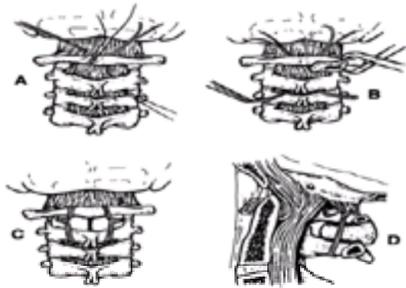
Although rarely used, conservative treatment of acute transverse ligament injuries has been reported. For acute injuries, reduction in extension is recommended followed by surgical stabilization of C1 and C2 and immobilization for 8 to 12 weeks in a Minerva cast, a halo brace, or cervical orthosis. Flexion and extension views should be obtained after stabilization to document stability.

## OPERATIVE TREATMENT

### Atlantoaxial Arthrodesis

#### *Technique of Brooks and Jenkins*

The supine patient is intubated in the supine position while still on a stretcher, and is then placed prone on the operating table, with the head supported by traction; the head-thorax relationship is maintained at all times during turning ([23](#)) ([Fig. 18-37](#)). A lateral cervical spine radiograph is obtained to ensure proper alignment before surgery. The skin is prepared and draped in a sterile fashion and a solution of epinephrine (1 : 500,000) is injected intradermally to aid hemostasis.

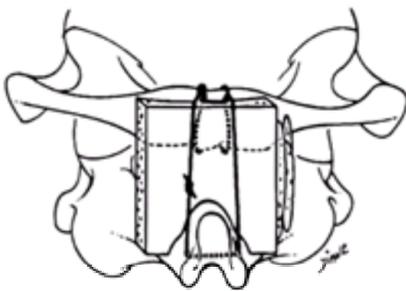


**FIGURE 18-37.** Technique of atlantoaxial arthrodesis (Brooks-Jenkins). **A:** Wires are inserted under the atlas and axis. **B:** Full-thickness bone grafts from the iliac crest are placed between the arch of the atlas and the lamina of the axis. **C and D:** The wires are tightened over the graft and twisted on each side. (Redrawn from Brooks AL, Jenkins EB. Atlantoaxial arthrodesis by the wedge compression method. *J Bone Joint Surg [Am]* 1978;60:279; with permission.)

C1 and C2 are exposed through a midline incision. Using an aneurysm needle, a mersilene suture is passed from cephalad to caudad on each side of the midline under the arch of the atlas and then beneath the lamina of C2. These serve as guides to introduce two doubled 20-gauge wires. The size of the wire used varies depending on the size and age of the patient. Two full-thickness bone grafts approximately 1.25 × 3.5 cm are harvested from the iliac crest and beveled so that the apex of the graft fits in the interval between the arch of the atlas and the lamina of the axis. Notches are fashioned in the upper and lower cortical surfaces to hold the circumferential wires and prevent them from slipping. The doubled wires are tightened over the graft and twisted on each side. The wound is irrigated and closed in layers over suction drains.

#### Technique of Gallie

The supine patient is intubated while on a stretcher (59) (Fig. 18-38). The prone patient then is placed on the operating table with the head supported by traction, maintaining the head–thorax relationship during turning. A lateral cervical spine radiograph is obtained to ensure proper alignment before surgery. The skin is prepared and draped in a sterile fashion, and a solution of epinephrine (1 : 500,000) is injected intradermally to aid hemostasis.



**FIGURE 18-38.** Wires are passed under the lamina of the atlas and through the spine of the axis and tied over the graft. This method is used most frequently. (Reprinted from Fielding JW, Hawkins RJ, Ratzan SA. *J Bone Joint Surg [Am]* 1976;58:400; with permission.)

A midline incision is made from the lower occiput to the level of the lower end of the fusion, extending deeply within the relatively avascular midline structures, the intermuscular septum, or ligamentum nuchae. Care should be taken not to expose any more than the area to be fused to decrease the chance of spontaneous extension of the fusion. By subperiosteal dissection, the posterior arch of the atlas and the lamina of C2 are exposed. The muscular and ligamentous attachments from C2 are removed with a curet. Care should be taken to dissect laterally along the atlas to prevent injury to the vertebral arteries and vertebral venous plexus that lie on the superior aspect of the ring of C1, less than 2 cm lateral to the midline. The upper surface of C1 is exposed no farther laterally than 1.5 cm from the midline in adults and 1 cm in children. Decortication of C1 and C2 generally is not necessary. From below, a wire loop of appropriate size is passed upward under the arch of the atlas either directly or with the aid of a mersilene suture. The mersilene suture can be passed with an aneurysm needle. The free ends of the wire are passed through the loop, grasping the arch of C1 in the loop.

A corticocancellous graft is taken from the iliac crest and placed against the lamina of C2 and the arch of C1 beneath the wire. One end of the wire is passed through the spinous process of C2, and the wire is twisted on itself to secure the graft in place. The wound is irrigated and closed in layers with suction drainage tubes.

#### Atlantoaxial Instability Associated with Congenital Anomalies and Syndromes

Although acute atlantoaxial instability in children is rare, chronic atlantoaxial instability occurs in certain conditions such as juvenile rheumatoid arthritis, Reiter's syndrome, Down's syndrome, and Larsen's syndrome. Bone dysplasia—such as Morquio's polysaccharidosis, spondyloepiphyseal dysplasia, and Kniest's syndrome—also may be associated with atlantoaxial instability, as well as os odontoideum, Klippel-Feil syndrome, and occipitalization of the atlas (28,73,75,92,94,111).

Certain cranial facial malformations have high incidences of associated anomalies of the cervical spine, such as Apert's syndrome, hemifacial microsomia, and Goldenhar's syndrome (152). Treatment recommendations are individualized based on the natural history of the disorder and future risk to the patient. Although there is little literature on cervical spine instability in each of these syndromes, there has been considerable interest in the incidence and treatment of atlantoaxial instability in children with Down's syndrome (35,38,131,132,167).

Some Down's syndrome patients have C1–C2 instability of more than 5 mm. The Committee on Sports Medicine of the American Academy of Pediatrics issued a policy statement (35) asserting that Down's syndrome patients with 5 to 6 mm of instability should be restricted from participating in sports that carry a risk of stress to the head and neck. The Special Olympics organization has placed even greater restrictions on particular sports (156). Davidson found that neurologic signs were more predictive of impending dislocation than the radiologic criteria. Nearly all the patients with actual dislocations that he reviewed had at least several weeks of readily detectable physical signs before dislocation occurred (38).

Surgical stabilization is indicated for patients with translation of more than 10 mm. In patients with less than 10 mm of translation and a neurologic deficit or history of neurologic symptoms, surgical stabilization also may be indicated. Once surgical stabilization is needed, the treating physician must understand the increased risk of complications (i.e., pseudarthrosis) in this patient population. Segal et al. (145) reported a high complication rate after posterior arthrodesis of the cervical spine in patients who have Down's syndrome. Six of ten patients developed resorption of the bone graft and associated pseudarthrosis. Other complications in this patient population after attempted posterior arthrodesis were wound infection, dehiscence of the operative site, instability of adjacent motion segments, and neurologic sequelae (152).

#### Atlantoaxial Rotatory Subluxation

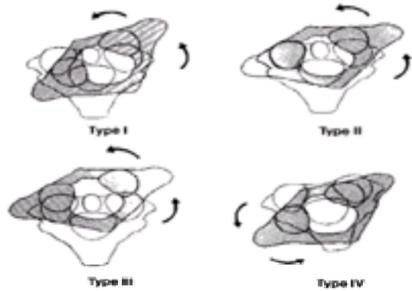
Atlantoaxial rotatory subluxation is a common cause of childhood torticollis. This condition is known by several names such as rotatory dislocation, rotatory displacement, rotatory subluxation, and rotatory fixation. Atlantoaxial rotatory subluxation probably is the most accepted term used, except for long-standing cases (3

months), which are called rotatory fixation.

A significant amount of motion occurs at the atlantoaxial joint; half of the rotation of the cervical spine occurs there. Through this range of motion at the C1–C2 articulation, some children develop atlantoaxial rotatory subluxation. The two most common causes are trauma and infection; the most common cause is an upper respiratory infection (Grisel's syndrome) (172). Subluxation also can occur after a retropharyngeal abscess, tonsillectomy, pharyngeoplasty, or trivial trauma. There is free blood flow between the veins and lymphatics draining the pharynx and the periodontoid plexus (124). Any inflammation of these structures can lead to attenuation of the synovial capsule or transverse ligament or both, with resulting instability. Another potential etiologic factor is the shape of the superior facets of the axis in children. Kawabe (88) demonstrated that the facets are smaller and more steeply inclined in children than in adults. A meniscus-like synovial fold was found between C1 and C2 that could prohibit reduction after displacement has occurred.

### Classification

Fielding and Hawkins (54) classified atlantoaxial rotatory displacements into four types based on direction and degree of rotation and translation (Fig. 18-39). Type 1 is a unilateral facet subluxation with an intact transverse ligament. This is the most common and benign type. Type 2 is a unilateral facet subluxation with anterior displacement of 3 to 5 mm. The unilateral anterior displacement of one of the lateral masses may indicate an incompetent transverse ligament with potential instability. Type 3 is bilateral anterior facet displacement with more than 5 mm of anterior displacement. This type is associated with deficiencies of the transverse and secondary ligaments, which can result in significant narrowing of the space available for the cord at the atlantoaxial level. Type 4 is an unusual type in which the atlas is displaced posteriorly. This usually is associated with a deficient dens. Although types 3 and 4 are rare, neurologic involvement may be present or instantaneous death can occur. Both types must be managed with great care.



**FIGURE 18-39.** Classification of rotatory displacement. (Reprinted from Fielding JW, Hawkins RJ. Atlantoaxial rotatory fixation. *J Bone Joint Surg [Am]* 1977;59:37; with permission.)

### Signs and Symptoms

Clinical findings include neck pain, headache, and a cock-robin position of rotating to one side as well as lateral flexion to the other (Fig. 18-40). When rotatory subluxation is acute, the child resists attempts to move the head and has pain with any attempts at correction. Usually they are able to make the deformity worse but are not able to correct it. Associated muscle spasms of the sternocleidomastoid muscle occur predominantly on the side of the long sternocleidomastoid muscle in an attempt to correct the deformity. If the deformity becomes fixed, the pain subsides but the torticollis and the decreased range of motion will persist (52). If rotatory fixation has been present for a long time in a small child, plagiocephaly sometimes can be noted. Neurologic abnormalities are extremely rare, although a few cases have been reported.

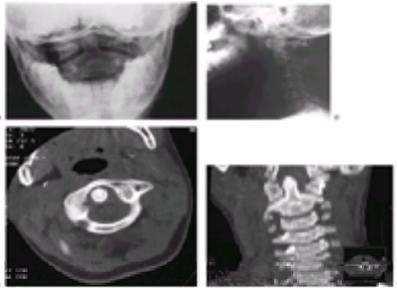


**FIGURE 18-40.** Child with rotatory subluxation of C1 on C2. Note the direction of head tilt and rotation of the neck.

### Radiographic Findings

Adequate radiographs may be difficult to obtain because of the associated torticollis and difficulty in positioning the head and neck. Anteroposterior and open mouth odontoid views should be taken with the shoulders flat and the head in as neutral a position as possible (101). Lateral masses that have rotated forward appear wider and closer to the midline, whereas the opposite lateral mass appears narrower and farther away from the midline on this view. One of the facet joints may be obscured because of apparent overlapping. The distance between the lateral mass and the dens also will be asymmetric. On the lateral view, the lateral facet appears anterior and usually appears wedge shaped instead of the normal oval shape. The posterior arches of the atlas may fail to superimpose because of head tilt, giving the appearance of fusion of C1 to the occiput (occipitalization). Flexion and extension lateral views are recommended to exclude instability.

Cineradiography has been used for the evaluation of atlantoaxial rotatory subluxation (50,54,80). This technique is limited in the acute stage, because pain restricts the motion necessary for a satisfactory study. With atlantoaxial rotatory fixation, cineradiography may be helpful in confirming the diagnosis by demonstrating that the atlas and axis are rotating as a unit. However, this technique requires high radiation exposure and generally has been replaced by CT scanning (7,45,54,60,128). CT should be performed with the head and body positioned as close to neutral as possible. This will show a superimposition of C1 on C2 in a rotated position and will allow the degree and amount of malrotation to be quantified. Some researchers have recommended dynamic CT scans taken with the patient looking to the right and the left to diagnose rotatory fixation (127). Three-dimensional CT scans also are helpful in identifying rotatory subluxation (140). MRI is of little value in this condition unless there are neurologic findings (Fig. 18-41).



**FIGURE 18-41.** **A and B:** Odontoid view and lateral cervical spine radiograph of rotary subluxation of C1 on C2. **C:** Note the asymmetry on the open-mouth odontoid view. **D:** CT and CT reconstruction documenting rotary subluxation.

### Differential Diagnoses

Differential diagnoses include torticollis caused by ophthalmologic problems, sternocleidomastoid tightness from muscular torticollis, brainstem or posterior fossa tumors or abnormalities, congenital vertebral anomalies, and infections of the vertebral column.

### Treatment

Treatment depends on the duration of the symptoms (127). Many patients probably never receive medical treatment, because symptoms may be mild and the subluxation may reduce spontaneously over a few days before medical attention is sought. If rotatory subluxation has been present for a week or less, a soft collar, anti-inflammatory medication, and exercise program are indicated. If this fails to produce improvement and the symptoms persist for more than a week, head halter traction should be initiated. This can be done either at home or in the hospital, depending on the social situation and the severity of symptoms. Muscle relaxants and analgesics also may be needed. Phillips and Hensinger (127) found that if rotatory subluxation was present for less than 1 month, head halter traction and bed rest were usually sufficient to relieve symptoms. If the subluxation is present for longer than a month, successful reduction is not very likely (29). However, halo traction can still be used to try to reduce the subluxation. The halo allows increased traction weight to be applied without interfering with opening of the jaw or causing skin pressure on the mandible. While the traction is being applied, active rotation to the right and left should be encouraged. Once the atlantoaxial rotatory subluxation has been reduced, motion has been restored, and the reduction is documented by CT scan, the patient is maintained in a halo vest for 6 weeks. If reduction cannot be maintained, posterior atlantoaxial arthrodesis is recommended. Even though internal rotation and alignment of the atlas and axis may not be restored, successful fusion should result in the appearance of normal head alignment by relieving the muscle spasms that occurred in response to the malrotation. Posterior arthrodesis also is recommended if any signs of instability or neurologic deficits secondary to the subluxation are present, if the deformity has been present for more than 3 months or if conservative treatment of 6 weeks of immobilization has failed.

### Hangman's Fracture

Bilateral spondylolisthesis of C2, or hangman's fractures, also may occur in children (170). The mechanism of injury is forced hyperextension. Most reports of this injury have been in children under the age of 2 years (48,56,128,138). This injury probably occurs more frequently in this age group because of the disproportionately large head, poor muscle control, and hypermobility. The possibility of child abuse also must be considered. Patients present with neck pain and resist any movement of the head and neck. There should be a positive history of trauma (Fig. 18-42).



**FIGURE 18-42.** Lateral radiograph of patient with traumatic C2 spondylolisthesis (Hangman's fracture).

Radiographs reveal a lucency anterior to the pedicles of the axis, usually with some forward subluxation of C2 on C3. One must be sure this is a fracture and not a persistent synchondrosis of the axis. Matthews et al. (103), Nordstrom et al. (116), and Smith et al. (155) have reported similar cases of persistent synchondrosis of the axis. CT scans showed the defect to be at the level of the neurocentral chondrosis. Later films showed ossification within the synchondrosis gap.

Treatment should be symptomatic with immobilization in a Minerva cast, halo, or cervical orthosis for 8 to 12 weeks. Traction is not needed to reduce this fracture and may even produce potentially dangerous distraction. Pizzutillo et al. (128) reported that four of five patients healed with immobilization. If union does not occur, posterior arthrodesis or anterior arthrodesis can be performed to stabilize this fracture.

## SUBAXIAL INJURIES

Fractures and dislocations involving C3 through C7 are rare in children and infants (85,87,105,151) and usually occur in teenagers or older children. Lower cervical spine injuries in children as opposed to those in adults can occur through the cartilaginous end plate (39). The end plate may break completely through the cartilaginous portion (Salter type I) or may exit through the bony edge (Salter type II). Usually the inferior end plate fractures because of the protective effect of the uncinete processes of the superior end plate (8).

### Posterior Ligamentous Disruption

Posterior ligamentous disruption can occur with a flexion or distraction injury to the cervical spine. The patient usually has point tenderness at the injury site and complains of neck pain. Initial radiographs may be normal except for loss of normal cervical lordosis. This may be a normal finding in young children, but should be evaluated for possible ligamentous injury in an adolescent. Widening of the posterior interspinous distance is suggestive of this injury. MRI may be helpful in documenting ligamentous damage.

With posterior ligamentous disruption, gradual displacement of one segment on the other can occur, and secondary adaptive changes in the growing spine may make reduction difficult. Posterior ligamentous injuries should be protected with an extension orthosis, and patients should be followed closely for the development of instability. If signs of instability are present, then a posterior arthrodesis should be performed.

### Compression Fractures

Compression fractures, the most common fractures of the subaxial spine in children, are caused by flexion and axial loading that results in loss of vertebral body height. This can be detected on a lateral radiograph. Because vertebral disks in children are more resilient than the vertebral bodies, the bone is more likely to be injured. Compression fractures are stable injuries and heal in children in 3 to 6 weeks. Many compression fractures may be overlooked because of the normal wedge shape of the vertebral bodies in young children. Immobilization in a cervical collar is recommended for 3 to 6 weeks. Flexion and extension films to confirm stability should be obtained 2 to 4 weeks after injury. In children under 8 years of age, the vertebral body may reconstitute itself with growth, although Schwarz et al. ( 143) reported that kyphosis of more than 20 degrees may not correct with growth. Associated injuries can include anterior teardrop, laminar, and spinous process fractures.

### Unilateral and Bilateral Facet Dislocations

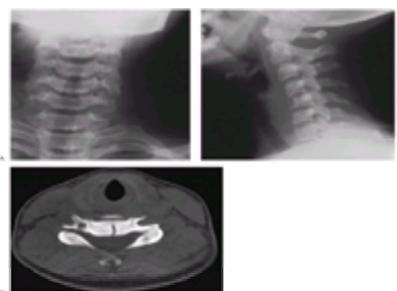
Unilateral facet dislocations and bilateral facet dislocations are the second most common injuries in the subaxial spine in children. Most occur in adolescents and are similar to adult injuries. The diagnosis usually can be made on anteroposterior and lateral radiographs. In children the so-called perched facet is a true dislocation. The cartilaginous components are overlapped and locked. On the radiograph, the facet appears perched because the overlapped cartilage cannot be seen. Unilateral facet dislocation is treated with traction and reduction. If reduction cannot be easily obtained, open reduction and arthrodesis are indicated. Complete bilateral facet dislocation, although rare, is more unstable and has a higher incidence of neurologic deficit ( Fig. 18-43). Treatment consists of reduction and stabilization with a posterior arthrodesis.



**FIGURE 18-43. A and B:** Lateral radiograph of a patient with so-called perched facets, demonstrating a facet dislocation. **C and D:** Lateral and anteroposterior radiograph after reduction and posterior arthrodesis.

### Burst Fractures

Although rare, burst fractures can occur in children. These injuries are caused by an axial load. Radiographic evaluation should consist of anteroposterior and lateral views. CT scans aid in detecting any spinal canal compromise from retropulsed fracture fragments and occult laminar fractures. If no neurologic deficit or significant canal compromise is present, then treatment consists of traction followed by halo immobilization. Anterior arthrodesis rarely is recommended in pediatric patients except in the rare patient with a burst fracture and significant canal compromise ( 147). Anterior arthrodesis destroys the anterior growth potential; as posterior growth continues, a kyphotic deformity may occur ( Fig. 18-44).



**FIGURE 18-44. A, B, and C:** Anteroposterior and lateral radiograph and CT scan of patient with a minimally displaced burst fracture of C5.

### Spondylolysis and Spondylolisthesis

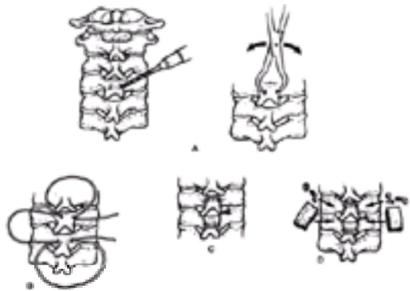
Spondylolysis and spondylolisthesis of C2 through C6 have been reported. These injuries can occur from either a hyperextension or flexion axial loading injury. Associated anterosuperior avulsion or compression fractures of the vertebral body may occur. The diagnosis usually is made on plain radiographs that show a fracture line through the pedicles. Oblique views may be necessary to better identify the fracture line. CT scanning may be useful in differentiating an acute fracture from a normal synchondrosis. Treatment consists of immobilization in a cervical orthosis or halo brace. Surgical stabilization is recommended only for truly unstable fractures or nonunions. Neurologic involvement is rare.



## OPERATIVE TREATMENT

### Posterior Arthrodesis

General anesthesia is administered with the patient supine ( Fig. 18-45). The patient is turned prone on the operating table, with care taken to maintain traction and proper alignment of the head and neck. The head may be positioned in a head rest or maintained in skeletal traction. Radiographs are obtained to confirm adequate alignment of the vertebrae and to localize the vertebrae to be exposed. Extension of the fusion mass can occur when extra vertebrae or spinous processes are exposed in the cervical spine. A midline incision is made over the chosen spinous processes, and the spinous process and lamina are exposed subperiosteally to the facet joints. If the spinous process is large enough, a hole is made in the base of the spinous process with a towel clip or Lewin clamp. An 18-gauge wire is passed through this hole, looped over the spinous process, and passed through the hole again. A similar hole is made in the base of the spinous process of the inferior vertebra to be fused, and the wire is passed through this vertebra. The wire is then passed through this hole, looped under the inferior aspect of the spinous process, and then passed back through the same hole. The wire is tightened and corticocancellous bone grafts are placed along the exposed lamina and spinous processes. The wound is closed in layers. If the spinous process is too small to pass wires, then an *in situ* arthrodesis can be performed and external immobilization used.



**FIGURE 18-45.** Technique of posterior arthrodesis in subaxial spine levels C3–C7. **A:** A hole is made in the spinous process of the vertebrae to be fused. **B:** An 18-gauge wire is passed through both holes and around the spinous processes. **C:** The wire is tightened. **D:** Corticocancellous bone grafts are placed. (Redrawn from Murphy MJ, Southwick WO. Posterior approaches and fusions. In: Cervical Spine Research Society. *The cervical spine*. Philadelphia: JB Lippincott, 1983; with permission.)

Hall et al. (71) used a 16-gauge wire and threaded Kirschner wires. The threaded Kirschner wires are passed through the bases of the spinous processes of the vertebrae to be fused. This is followed by a figure-of-eight wiring with a 16-gauge wire (Fig. 18-46). After tightening the wire about the Kirschner wires, strips of corticocancellous and cancellous bone are packed over the posterior arches of the vertebrae to be fused.



**FIGURE 18-46.** Alternative fixation method for posterior arthrodesis of C3–C7. A 16-gauge wire is placed in a figure-of-eight pattern around two threaded Kirschner wires passed through the bases of the spinous processes of the vertebrae to be fused. (Reprinted from Hall JE, Simmons ED, Danylchuk K, et al. Instability of the cervical spine and neurological involvement in Klippel-Feil syndrome: a case report. *J Bone Joint Surg [Am]* 1990;72:460; with permission.)

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## CHAPTER REFERENCES

1. Adran GM, Kemp FH. The mechanism of changes in the form of cervical airway in infancy. *Med Radiogr Photogr* 1968;44:26–38.
2. Allington JJ, Zembo M, Nadell J, Bowen JR. C1–C2 posterior soft tissue injuries with neurologic impairment in children. *J Pediatr Orthop* 1990;10:596–601.
3. Anderson PA, Montesano PX. Morphology and treatment of occipital condyle fractures. *Spine* 1988;13:731–736.
4. Anderson JM, Schutt AH. Spinal injury in children: a review of 156 cases seen from 1950 through 1978. *Mayo Clin Proc* 1980;55:499–504.
5. Anderson LD, Smith BL, DeToree J, et al. The role of polytomography in the diagnosis and treatment of cervical spine injuries. *Clin Orthop Rel Res* 1982;165:64–68.
6. Annis JA, Finlay DB, Allen MJ, et al. A review of cervical-spine radiographs in casualty patients. *Br J Radiol* 1987;60:1059–1061.
7. Apple JS, Kirks DR, Merten DF, et al. Cervical spine fractures and dislocations in children. *Pediatr Radio* 1987;17:45–49.
8. Aufdermaur M. Spinal injuries in juveniles: necropsy findings in twelve cases. *J Bone Joint Surg [Br]* 1974;56:513–519.
9. Bachulis BL, Long WB, Hynes GD, et al. Clinical indications for cervical spine radiographs in the traumatized patient. *Am J Surg* 1987;153:473–477.
10. Banniza von Bazan UK, Paeslack V. Scoliotic growth in children with acquired paraplegia. *Paraplegia* 1977;15:65–73.
11. Baum JA, Hanley EN Jr, Pulekines J. Comparison of halo complications in adults and children. *Spine* 1989;14:251–252.
12. Bedbrook GM. Correction of scoliosis due to paraplegia sustained in pediatric age group. *Paraplegia* 1977;15:90–96.
13. Birney TJ, Hanley EN. Traumatic cervical spine injuries in childhood and adolescence. *Spine* 1989;14:1277–1282.
14. Bernini EP, Elefante R, Smaltino F, Tedeschi G. Angiographic study on the vertebral artery in cases of deformities of the occipitocervical joint. *AJR* 1969;107:526–529.
15. Bivins HG, Ford S, Bezmalinovic Z, et al. The effect of axial traction during orotracheal intubation of the trauma victim with an unstable cervical spine. *Ann Emerg Med* 1988;17:25–29.
16. Bohlman HH. Acute fractures and dislocations of the cervical spine. *J Bone Joint Surg [Am]* 1969;61:1119–1142.
17. Bohn D, Armstrong D, Becker L, et al. Cervical spine injuries in children. *J Trauma* 1990;30:463–469.
18. Bracken MB. Treatment of acute spinal cord injury with methylprednisolone: results of a multi-center randomized clinical trial. *J Neurotrauma* 1991;8(suppl 1):47–50.
19. Bracken MB. Pharmacological treatment of acute spinal cord injury: current status and future projects. *J Emerg Med* 1993;11(suppl 1):43–48.
20. Bracken MB, Shepard MJ, Collins WF, et al. A randomized controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury: results of the Second National Spinal Cord Injury Study. *N Engl J Med* 1990;322:1405–1411.
21. Bracken MB, Shepard MJ, Collins WF, et al. Methylprednisolone and naloxone treatment after acute spinal cord injury: one year follow-up data. Results of the Second National Acute Spinal Cord Injury Study. *J Neurosurg* 1992;76:23–31.
22. Bresnan MJ, Abrams IF. Neonatal spinal cord transection secondary to intrauterine hyperextension of neck in breech presentation. *J Pediatr* 1974;84:734–737.
23. Brooks AL, Jenkins EB. Atlantoaxial arthrodesis by the wedge compression method. *J Bone Joint Surg [Am]* 1978;60:279–290.
24. Bucholz RW, Burkhead WZ. The pathological anatomy of fatal atlanto-occipital dislocations. *J Bone Joint Surg [Am]* 1979;61:248–250.
25. Bulas DI, Fitz CR, Johnson DL. Traumatic atlanto-occipital dislocation in children. *Radiology* 1993;188:155–158.
26. Burke DC. Spinal cord trauma in children. *Paraplegia* 1971;9:1–14.
27. Burke DC. Traumatic spinal paralysis in children. *Paraplegia* 1971;9:268–276.
28. Burke SW, French HG, Roberts JM, et al. Chronic atlantoaxial instability in Down syndrome. *J Bone Joint Surg [Am]* 1985;67:1356–1360.
29. Burkus JK, Deponte RJ. Chronic atlantoaxial rotatory fixation: correction by cervical traction, manipulation, and branching. *J Pediatr Orthop* 1986;6:631–635.
30. Caffey J. The whiplash shaken infant syndrome. *Pediatrics* 1974;54:396–403.
31. Cattell HS, Filtzer DL. Pseudosubluxation and other normal variations in the cervical spine in children. *J Bone Joint Surg [Am]* 1965;47:1295–1309.
32. Closkey R, Flynn J, Dormans J, et al. The role of MRI in the assessment of pediatric cervical spine injuries. Paper presented at the Annual Meeting of the Pediatric Orthopaedic Society of North America, Lake Buena Vista, Florida, 1999.
33. Collato PM, Demuth WW, Schwentker EP, et al. Traumatic atlanto-occipital dislocation. *J Bone Joint Surg [Am]* 1986;68:1106–1109.
34. Committee on Pediatric Orthopaedics. Trauma of the Cervical Spine. Position Statement. American Academy of Orthopaedic Surgeons, 1990.
35. Committee on Sports Medicine. Atlantoaxial instability in Down syndrome. *Pediatrics* 1984;74:152–154.
36. Conry BG, Hall CM. Cervical spine fractures and rear car seat restraints. *Arch Dis Child* 1987;62:1267–1268.
37. Curran C, Dietrich AM, Bowman MJ, et al. Pediatric cervical-spine immobilization: achieving neutral position? *J Trauma* 1995;39:729–732.
38. Davidson RG. Atlantoaxial instability in individuals with Down syndrome: a fresh look at the evidence. *Pediatrics* 1988;81:857–865.
39. Dawson EG, Smith L. Atlanto-axial subluxation in children due to vertebral anomalies. *J Bone Joint Surg [Am]* 1979;61:582–587.
40. De V de Beer J, Hoffman EB, Kieck CF. Traumatic atlantoaxial subluxation in children. *J Pediatr Orthop* 1990;10:397–400.
41. DiBenedetto T, Lee CK. Traumatic atlanto-occipital instability: a case report with follow-up and a new diagnostic technique. *Spine* 1990;15:595–597.
42. Dietrich AM, Ginn-Pease ME, Bartkowski HM, et al. Pediatric cervical spine fractures: predominately subtle presentation. *J Pediatr Surg* 1991;26:995–1000.
43. Donahue D, Muhlbauer M, Kaufman R, et al. Childhood survival of atlanto-occipital dislocation: underdiagnosis, recognition, treatment, and review of the literature. *Pediatr Neurosurg* 1994;21:105–111.

44. Dormans JP, Crisciello AA, Drummond DS, et al. Complications in children managed with immobilization in a halo vest. *J Bone Joint Surg [Am]* 1995;77:1370–1373.
45. Dvorak J, Panjabi M, Gerber M, et al. CT-functional diagnostics of the rotatory instability of the cervical spine: 1. An experimental study on cadavers. *Spine* 1987;12:197–205.
46. Dyck P. Os odontoideum in children: neurological manifestations and surgical management. *Neurosurgery* 1978;2:93–99.
47. Evans DL, Bethem D. Cervical spine injuries in children. *J Pediatr Orthop* 1989;9:563–568.
48. Fardon DF, Fielding JW. Defects of the pedicle and spondylolisthesis of the second cervical vertebra. *J Bone Joint Surg [Br]* 1981;63:526–528.
49. Farley FA, Hensinger RN, Herzenberg JE. Cervical spinal cord injury in children. *J Spinal Disord* 1992;5:410–416.
50. Fielding JW. Cineerentgenography of the normal cervical spine. *J Bone Joint Surg [Am]* 1957;39:1280–1288.
51. Fielding JW, Griffin PP. Os odontoideum: an acquired lesion. *J Bone Joint Surg [Am]* 1974;56:187–190.
52. Fielding JW, Hawkins RJ. Atlanto-axial rotary fixation (fixed rotary subluxation of the atlanto-axial joint). *J Bone Joint Surg [Am]* 1977;59:37–44.
53. Fielding JW, Hensinger RN, Hawkins RJ. Os odontoideum. *J Bone Joint Surg [Am]* 1980;62:376–383.
54. Fielding JW, Stillwell WT, Chynn KY, et al. Use of computed tomography for the diagnosis of atlanto-axial rotary fixation. *J Bone Joint Surg [Am]* 1978;60:1102–1104.
55. Finch GD, Barnes MJ. Major cervical spine injuries in children and adolescents. *J Pediatr Orthop* 1998;18:811–814.
56. Francis WR, Fielding JW, Hawkins RJ, et al. Traumatic spondylolisthesis of the axis. *J Bone Joint Surg [Br]* 1981;63:313–318.
57. Freiburger RH, Wilson PD, Nicholas JA. Acquired absence of the odontoid process. A case report. *J Bone Joint Surg [Am]* 1965;47:1231–1236.
58. Fuchs S, Barthel MJ, Flannery AM, et al. Cervical spine fractures sustained by young children in forward-facing car seats. *Pediatrics* 1989;84:348–354.
59. Gallie WE. Fractures and dislocations of the cervical spine. *Am J Surg* 1939;46:495–499.
60. Geehr RB, Rothman SLG, Kier EL. The role of computed tomography in the evaluation of upper cervical spine pathology. *Comput Tomogr* 1978;2:79–97.
61. Geisler FH, Dorsey FC, Coleman WP. Recovery of motor function after spinal cord injury—a randomized, placebo-controlled trial with GM-1 ganglioside [Erratum]. *N Engl J Med* 1991;325:1669–1670.
62. Geisler FH, Dorsey FC, Coleman WP. Recovery of motor function after spinal cord injury—a randomized, placebo-controlled trial with GM-1 ganglioside. *N Engl J Med* 1991;324:1829–1838.
63. Geisler FH, Dorsey FC, Coleman WP. GM-1 ganglioside in human spinal cord injury. *J Neurotrauma* 1992;9(suppl 1):407–416.
64. Geisler FH, Dorsey FC, Coleman WP. Past and current clinical studies with GM-1 ganglioside in acute spinal cord injury. *Rev Ann Emerg Med* 1993;22:1041–1047.
65. Georgopoulos G, Pizzutillo PD, Lee MS. Occipito-atlanto instability in children. A report of five cases and review of the literature. *J Bone Joint Surg [Am]* 1987;69:429–436.
66. Givens T, Polley KA, Smith GF, et al. Pediatric cervical spine injury: a three-year experience. *J Trauma* 1996;41:310–314.
67. Godard J, Hadji M, Raul JS. Odontoid fractures in the child with neurologic injury. Direct osteosynthesis with a cortico-spongious screw and literature review. *Childs Nerv Syst* 1997;13:105–107.
68. Griffiths SC. Fracture of the odontoid process in children. *J Pediatr Surg* 1972;7:680–683.
69. Hadley MN, Zabramski JM, Browner CM, et al. Pediatric spinal trauma: review of 122 cases of spinal cord vertebral column injuries. *J Neurosurg* 1988;68:18–24.
70. Haffner DL, Hoffer MM, Wiedebusch R. Etiology of children's spinal injuries at Rancho Los Amigos. *Spine* 1993;18:679–684.
71. Hall JE, Denis F, Murray J. Exposure of the upper cervical spine for spinal decompression by a mandible and tongue-splitting approach. Case report. *J Bone Joint Surg [Am]* 1977;59:121–125.
72. Hamilton MG, Myles ST. Pediatric spinal injury. Review of 61 deaths. *J Neurosurg* 1988;77:705–708.
73. Hammerschlag W, Ziv I, Wald U, et al. Cervical instability in an achondroplastic infant. *J Pediatr Orthop* 1988;8:481–484.
74. Harmanli O, Kaufman Y. Traumatic atlanto-occipital dislocation with survival. *Surg Neuro* 1993;39:324–330.
75. Hensinger RN, DeVito PD, Ragsdale CG. Changes in the cervical spine in juvenile rheumatoid arthritis. *J Bone Joint Surg [Am]* 1986;68:189–198.
76. Hensinger RN, Fielding JW, Hawkins RJ. Congenital anomalies of the odontoid process. *Orthop Clin North Am* 1978;9:901–912.
77. Hensinger RN, Lang JE, MacEwen GD. Klippel-Feil syndrome: a constellation of associated anomalies. *J Bone Joint Surg [Am]* 1974;56:1246–1252.
78. Herzenberg JE, Hensinger RN. Pediatric cervical spine injuries. *Trauma Q* 1989;5:73–81.
79. Herzenberg JE, Hensinger RN, Dedrick DK, et al. Emergency transport and positioning of young children who have an injury of the cervical spine: the standard backboard may be hazardous. *J Bone Joint Surg [Am]* 1989;71:15–22.
80. Hohl M, Baker HR. The atlanto-axial joint: roentgenographic and anatomical study of normal and abnormal motion. *J Bone Joint Surg [Am]* 1964;46:1739–1752.
81. Hosono N, Yonenbou K, Kazuyoshi K, et al. Traumatic anterior atlanto-occipital dislocation. *Spine* 1993;18:786–790.
82. Hoy GA, Cole WG. The paediatric cervical seat belt syndrome. *Injury* 1993;24:297–299.
83. Hubbard DD. Injuries of the spine in children and adolescents. *Clin Orthop* 1974;100:56–65.
84. Huerta C, Griffith R, Joyce SM. Cervical spine stabilization in pediatric patients. Evaluation of current techniques. *Ann Emerg Med* 1987;16:1121–1126.
85. Jacob B. Cervical fracture and dislocation (C3–7). *Clin Orthop* 1975;109:18–32.
86. Jefferson G. Fracture of the atlas vertebra: report of four cases and a review of those previously recorded. *Br J Surg* 1919–1920;7:407–422.
87. Jones ET, Hensinger RN. Cervical spine injuries in children. *Contemp Orthop* 1982;5:17–23.
88. Kawabe N, Hirotoni H, Tanaka O. Pathomechanism of atlanto-axial rotary fixation in children. *J Pediatr Orthop* 1989;9:569–574.
89. Kewalramani LS, Kraus JF, Sterling HM. Acute spinal-cord lesions in a pediatric population: epidemiological and clinical features. *Paraplegia* 1980;18:206–219.
90. Kilfoyle RM, Foley JJ, Norton PL. Spine and pelvic deformity in childhood and adolescent paraplegia. *J Bone Joint Surg [Am]* 1965;47:659–682.
91. Klippel M, Feil A. Anomalies de la colonne vertebrale par absence des vertebres cervicales; avec cage thoracique remontant jusqu'ala base du crane. *Bull Soc Anat Paris* 1912;87:185.
92. Kobori M, Takahashi H, Mikawa Y. Atlanto-axial dislocation in Down's syndrome: report of two cases requiring surgical correction. *Spine* 1986;11:195–200.
93. Koop SE, Winter RB, Lonstein JE. The surgical treatment of instability of the upper part of the cervical spine in children and adolescents. *J Bone Joint Surg [Am]* 1984;66:403–411.
94. Kransdorf MJ, Wherle PA, Moser RP Jr. Atlantoaxial subluxation in Reiter's syndrome. *Spine* 1988;13:12–14.
95. Lally KP, Senak M, Hardin WD, et al. Utility of the cervical spine radiograph in pediatric trauma. *Am J Surg* 1989;158:540–542.
96. Lawson JP, Ogden JA, Bucholz RW, et al. Physcal injuries of the cervical spine. *J Pediatr Orthop* 1987;7:428–435.
97. Letts M, Kaylor D, Gouw G. A biomechanical study of halo fixation in children. *J Bone Joint Surg [Br]* 1987;70:277–279.
98. Lui T-N, Lee S-T, Wong C-W, et al. C1–C2 fracture-dislocations in children and adolescents. *J Trauma* 1996;40:408–411.
99. Lynch JM, Meza MP, Pollack IF, et al. Direct injury to the cervical spine of a child by a lap-shoulder belt resulting in quadriplegia: case report. *J Trauma* 1996;41:747–749.
100. Maekawa K, Masaki T, Kokubun Y. Fetal spinal cord injury secondary to hyperextension of the neck: no effect of caesarean section. *Dev Med Child Neuro* 1976;18:228–232.
101. Maheshwaran S, Sgouros S, Jeyapalan K, et al. Imaging of childhood torticollis due to atlanto-axial rotary fixation. *Childs Nerv Syst* 1995;11:667–671.
102. Marlin AE, Gayle RW, Lee JF. Jefferson fractures in children. *J Neurosurg* 1983;58:277–279.
103. Matthews LS, Vetter LW, Tolo VT. Cervical anomaly stimulating hangman's fracture in a child. *J Bone Joint Surg [Am]* 1982;64:299–300.
104. Mayfield JK, Erkkila JC, Winter RB. Spine deformities subsequent to acquired childhood spinal cord injury. *Orthop Trans* 1979;3:281–282.
105. McClain RF, Clark CR, El-Khoury GY. C6–7 dislocation in a neurologically intact neonate: a case report. *Spine* 1989;14:125–126.
106. McGrory BJ, Klassen RA. Arthrodesis of the cervical spine for fractures and dislocations in children and adolescents. *J Bone Joint Surg [Am]* 1994;76:1606–1616.
107. McGrory BJ, Klassen RA, Chao EY, et al. Acute fracture and dislocations of the cervical spine in children and adolescents. *J Bone Joint Surg [Am]* 1993;75:988–995.
108. Menezes AH, Ryken JC. Craniovertebral junction abnormalities. In: Weinstein SL, ed. *The pediatric spine: principles and practice*. New York: Raven, 1994.
109. Mikawa Y, Watanabe R, Yamano Y, et al. Fractures through a synchondrosis of the anterior arch of the atlas. *J Bone Joint Surg [Br]* 1987;69:483.
110. Millington PJ, Ellingsen JM, Hauswirth BE, et al. Thermoplastic minerva body jacket—a practical alternative to current methods of cervical spine stabilization. *Phys Ther* 1987;67:223–225.
111. Miz GS, Engler GL. Atlanto-axial subluxation in Larsen's syndrome: a case report. *Spine* 1987;12:411–412.
112. Mubarak SJ, Camp JF, Vueltich W, et al. Halo application in the infant. *J Pediatr Orthop* 1989;9:612–614.
113. Murphy MJ, Ogden JA, Bucholz RW. Cervical spine injury in the child. *Contemp Orthop* 1981;3:615–623.
114. Nicholson JT. Surgical fixation of dislocation of the first cervical vertebrae in children. *NY State Med J* 1956;56:3839–3843.
115. Nitecki S, Moir CR. Predictive factors of the outcome of traumatic cervical spine fracture in children. *J Pediatr Surg* 1994;29:1409–1411.
116. Nordstrom REA, Lahdenrants TV, Kaitila II, et al. Familial spondylolisthesis of the axis is vertebra. *J Bone Joint Surg [Br]* 1986;68:704–706.
117. Norman MG, Wedderburn LC. Fetal spinal cord injury with cephalic delivery. *Obstet Gynecol* 1973;42:355–358.
118. Odent T, Langlais J, Glorion C, et al. Fractures of the odontoid process: a report of 15 cases in children younger than 6 years. *J Pediatr Orthop* 1999;19:51–54.
119. Orenstein JB, Klein BL, Gotschall CS, et al. Age and outcome in pediatric cervical spine injury: 11-year experience. *Pediatr Emerg Care* 1994;10:132–137.
120. Orenstein JB, Klein BL, Oschenslager DW. Delayed diagnosis of pediatric cervical spine injury. *Pediatrics* 1992;89:1185–1188.
121. Pang D, Pollack IF. Spinal cord injury without radiologic abnormality in children: the SCIWORA syndrome. *J Trauma* 1989;29:654–664.
122. Pang D, Wilberger JE. Spinal cord injury without radiologic abnormalities in children. *J Neurosurg* 1982;57:114–129.
123. Papadopoulos SM, Dickman CA, Sonntag VKH, et al. Traumatic atlantooccipital dislocation with survival. *Neurosurgery* 1991;28:574–579.
124. Parke WW, Rothman RH, Brown MD. The pharyngovertebral veins: an anatomical rationale for Grisel's syndrome. *J Bone Joint Surg [Am]* 1984;66:568–574.
125. Pennecot GF, Gouraud D, Hardy JR, et al. Roentgenographical study of the stability of the cervical spine in children. *J Pediatr Orthop* 1984;4:346–352.
126. Perry J, Nickel VL. Total cervical-spine fusion for neck paralysis. *J Bone Joint Surg [Am]* 1959;41:37–60.
127. Phillips WA, Hensinger RN. The management of rotatory atlanto-axial subluxation in children. *J Bone Joint Surg [Am]* 1989;71:664–668.
128. Pizzutillo PD, Rocha EF, D'Astous J, et al. Bilateral fractures of the pedicle of the second cervical vertebra in the young child. *J Bone Joint Surg [Am]* 1986;68:892–896.
129. Powers B, Milber MD, Kramer RS, et al. Traumatic anterior occipital dislocation. *Neurosurgery* 1979;4:12–17.
130. Price E. Fractured odontoid process with anterior dislocation. *J Bone Joint Surg [Br]* 1960;42:410–413.
131. Pueschel SM. Atlantoaxial subluxation in Down syndrome. *Lancet* 1983;1:980.
132. Pueschel SM, Scolia FH. Atlantoaxial instability in individuals with Down syndrome: epidemiologic, radiographic, and clinical studies. *Pediatrics* 1987;4:555–560.
133. Ratchesky I, Boyce WT, Duncan B, et al. Clinical prediction of cervical spine injuries in children: radiographic abnormalities. *Am J Dis Child* 1987;141:199–201.
134. Rang MC. *Children's fractures*. Philadelphia: JB Lippincott, 1983.
135. Reinges MHT, Mayfrank L, Royhde V, et al. Surgically treated traumatic synchondrotic disruption of the odontoid process in a 15-month-old girl. *Childs Nerv Syst* 1998;14:85–87.

136. Richards PG. Stable fractures of the atlas and axis in children. *J Neurol Neurosurg Psychiatry* 1984;47:781–783.
137. Ries MD, Ray S. Posterior displacement of an odontoid fracture in a child. *Spine* 1986;11:1043–1044.
138. Ruff SJ, Taylor TKF. Hangman's fracture in an infant. *J Bone Joint Surg [Br]* 1986;68:702–703.
139. Ruge JR, Sinson GP, McLone DG, et al. Pediatric spinal injury: the very young. *J Neurosurg* 1988;68:25–30.
140. Scapinelli R. Three-dimensional computed tomography in infantile atlantoaxial rotatory fixation. *J Bone Joint Surg [Br]* 1994;76:367–370.
141. Schippers N, Königs D, Hassler W, et al. Typical and atypical fractures of the odontoid process in young children. Report of two cases and a review of the literature. *Acta Neurochir (Wien)* 1996;138:524–530.
142. Schmidt BM, Otte D, Krettek C. Fractures of the odontoid process in small children: biomechanical analysis and report of three cases. *Eur Spine J* 1996;5:63–70.
143. Schwarz N, Genelin F, Schwarz AF. Post-traumatic cervical kyphosis in children cannot be prevented by nonoperative methods. *Injury* 1994;25:173–175.
144. Schwartz GR, Wright SW, Fein JA, et al. Pediatric cervical spine injury sustained in falls from low heights. *Ann Emerg Med* 1997;30:249–252.
145. Segal LS, Drummond DS, Zanotti RM, et al. Complications of posterior arthrodesis of the cervical spine in patients who have Down syndrome. *J Bone Joint Surg [Am]* 1991;73:1547–1560.
146. Seimon LP. Fracture of the odontoid process in young children. *J Bone Joint Surg [Am]* 1977;59:943–948.
147. Shacked I, Ram Z, Hadani M. The anterior cervical approach for traumatic injuries to the cervical spine. *Clin Orthop* 1993;292:144–150.
148. Shaffer MA, Dorris PE. Limitation of the cross table lateral view in detecting cervical spine injuries: a retrospective review. *Ann Emerg Med* 1993;10:508–512.
149. Shaw BA, Murphy KM. Displaced odontoid fracture in a 9-month-old child. *Am J Emerg Med* 1999;1:73–75.
150. Sherburn EW, Day RA, Kaufman BA, et al. Subdental synchondrosis fracture in children: the value of 3-dimensional computerized tomography. *Pediatr Neurosurg* 1996;25:256–259.
151. Sherk HH, Schut L, Lane J. Fractures and dislocations of the cervical spine in children. *Orthop Clin North Am* 1976;7:593–604.
152. Sherk HH, Whitaker LA, Pasquariello PS. Fascial malformations and spinal anomalies: a predictable relationship. *Spine* 1982;7:526–531.
153. Shulman ST, Madden JD, Esterly JR, et al. Transection of the spinal cord. A rare obstetrical complication of cephalic delivery. *Arch Dis Child* 1971;46:291–294.
154. Sim F, Svien H, Bickel W, et al. Swan neck deformity following extensive cervical laminectomy. *J Bone Joint Surg [Am]* 1974;56:564–580.
155. Smith T, Skinner SR, Shonnard NH. Persistent synchondrosis of the second cervical vertebra simulating a hangman's fracture in a child. *J Bone Joint Surg [Am]* 1993;75:1228–1230.
156. Special Olympics Bulletin. *Participation by individuals with DS who suffer from atlantoaxial dislocation*. Washington, DC: Special Olympics, 1983.
157. Sponseller PD, Cass J. Atlanto-occipital arthrodesis for instability with neurologic preservation. *Spine* 1997;22:344–347.
158. Sponseller PD, Herzenberg JE. *Cervical spine injuries in children. The cervical spine*. Philadelphia: Lippincott-Raven, 1998:357–371.
159. Stauffer ES, Mazur JM. Cervical spine injuries in children. *Pediatr Ann* 1982;11:502–511.
160. Steel HH. Anatomical and mechanical consideration of the atlanto-axial articulation. *J Bone Joint Surg [Am]* 1968;50:1481–1482.
161. Swischuk EH Jr, Rowe ML. The upper cervical spine in health and disease. *Pediatrics* 1952;10:567–572.
162. Swischuk LE. Spine and spinal cord trauma in the battered child syndrome. *Radiology* 1969;92:733–738.
163. Tawbin A. CNS damage in the human fetus and newborn infant. *Am J Dis Child* 1951;33:543–547.
164. Taylor AR. The mechanism of injury to the spinal cord in the neck without damage to the vertebral column. *J Bone Joint Surg [Br]* 1951;33:453–457.
165. Tolo VT, Weiland AJ. Unsuspected atlas fractures and instability associated with oropharyngeal injury: case report. *J Trauma* 1979;19:278–280.
166. Torg B, Das M. Trampoline and minitrampoline injuries to the cervical spine. *Clin Sports Med* 1985;4:45–60.
167. VanDyke DC, Gahagan CA. Down syndrome: cervical spine abnormalities and problems. *Clin Pediatr* 1988;27:415–418.
168. Von Torklus D, Gehle W. *The upper cervical spine*. New York: Grune & Stratton, 1972.
169. Walsh JW, Stevens DB, Young AB. Traumatic paraplegia in children without contiguous spinal fracture or dislocation. *Neurosurgery* 1983;12:439–445.
170. Weiss MH, Kaufman B. Hangman's fracture in an infant. *Am J Dis Child* 1964;126:268–269.
171. Wertheim SB, Bohlman HH. Occipitocervical fusion: indications, technique, and long-term results. *J Bone Joint Surg [Am]* 1987;69:833–836.
172. Wetzell FT, Larocca H. Grisel's syndrome. A review. *Clin Orthop* 1989;240:141–152.
173. Yashon D. *Spinal injury*, 2nd ed. Norwalk: Appleton-Century Crofts, 1986:339–352.
174. Yasuoko F, Peterson H, MacCarty C. Incidence of spinal column deformity after multiple level laminectomy in children and adults. *J Neurosurg* 1982;57:441–445.
175. Yngve DA, Harris WP, Herndon WA, et al. Spinal cord injury without osseous spine fracture. *J Pediatr Orthop* 1988;8:153–159.

## FRACTURES OF THE THORACIC AND LUMBAR SPINE

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Pediatric spinal fractures represent 2% to 5% of all spinal injuries ([49,71](#)). Most spinal fractures in children occur in the cervical spine, but severe and seriously disabling injuries do occur in the thoracolumbar spine. Fractures of the thoracic and lumbar spine are rare in children compared with their frequency in adults. The actual incidence of thoracolumbar spinal injuries, however, may be twice that reported; statistics are skewed toward more severe injuries because many patients with mild injuries are never admitted to a hospital ([5,71](#)).

The location of the fracture and the mechanism of injury vary with the age of the child. Neonates are more prone to cervical injuries than to dorsal or lumbar injuries ([74](#)). Thoracolumbar spinal fractures in infants may be caused by child abuse, whereas children in the first decade of life usually sustain these injuries from motor vehicle accidents, either as a pedestrian or passenger, or from falls from heights ([23,49,53,62](#)). Thoracolumbar spinal fractures in children over 10 years of age typically occur in sports and recreational activities (such as tobogganing and bicycling), as well as motor vehicle accidents ([57,70,71,102,124](#)). Injuries from gunshot wounds also are increasing in frequency.

### ANATOMY

Certain anatomic features in children that influence the radiographic appearance of the spine, as well as the type of fractures, include an increased cartilage–bone ratio, the presence of secondary ossification centers, and soft tissue hyperelasticity. In infancy and early childhood, the vertebral bodies are largely cartilaginous, and the intervertebral disk spaces appear radiographically to be larger than they are ([Fig. 19-1](#)). With maturation the ossification center of the centrum enlarges and the cartilage–bone ratio reverses.

**FIGURE 19-1.** The radiograph of a normal spine in a 10-week-old infant. The superior and inferior vertebral end plates and vertebral apophysis are cartilaginous. Thus, the apparent widening of the intervertebral spaces is relative to the ossific portion of the vertebrae. The anterior and posterior notching of the walls of the vertebral body are due to normal vascular channels and may be confused with fracture.

In infants, horizontal conical shadows of lessened density extend inward from both the anterior and posterior walls of the vertebral bodies and can be confused with a fracture ([Fig. 19-1](#)) ([138](#)). The posterior indentation represents the foramen for the posterior arteries and veins in the vertebral wall; it is present in all vertebrae and at all ages. The more noticeable anterior conical shadow represents a large sinusoidal space within the vertebra. This anterior notch usually disappears by 1 year of age with ossification of the anterior and lateral walls of the vertebral bodies.

The vertebral apophyses are secondary centers of ossification that develop in the cartilaginous end plates at the superior and inferior surfaces of the vertebral bodies. Because they are thicker at the periphery than at the center, they appear as rings during early ossification and are called ring apophyses. These apophyses, equivalent to the epiphyses of long bones, are separated from the vertebral bodies by narrow cartilaginous physes. Equal superior and inferior vertical growth of the vertebral bodies occurs at these physes. The vertebral physes appear radiographically between the ages of 8 and 12 years and may be confused with avulsion fractures before they fuse with the vertebral bodies by approximately 21 years of age.

In children, the elasticity of the disks and vertebral bodies far exceeds that of the neural elements. In the cervical spine, the vertebral column can be stretched up to 2 inches without disruption, whereas the spinal cord tolerates only 1/4 inch. This elasticity partially accounts for the occurrence of spinal cord injury without radiographic abnormality (SCIWORA) in younger children ([9,71,99,147](#)). By the age of 10 years, the mechanical and anatomic characteristics of the bony thoracolumbar spine approach that of an adult, and fracture patterns become the same.

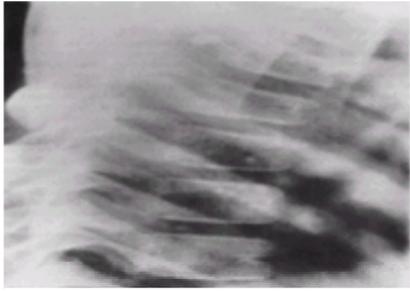
### CLASSIFICATION AND MECHANISM OF INJURY

The three main mechanisms of thoracolumbar spinal fractures are flexion with or without compression, distraction, and shear. Pure compression may cause slipping of the vertebral apophysis, which occurs only in adolescents.

#### Flexion

Hyperflexion injuries with compression are more common than distraction, shear, or subluxation–dislocation injuries ([62,63](#)). In immature spines, the intact disks are more resistant to vertical compression than are the vertebral bodies. During compression, the vertebral body collapses before the normal disk fails. Because of this, few children have posterior herniated disks, and herniation occurs only with significant loading injuries such as weight-lifting and gymnastics or repetitive trauma ([30](#)). Herniated disks associated with intervertebral disk calcification have been rarely reported ([84,103](#)).

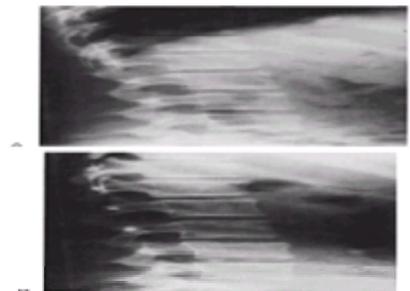
When a vertebra is slowly vertically loaded, the major distortion is a bulge in the vertebral end plate, with only a slight change of the annulus and no alteration in the shape of the nucleus pulposus ([112](#)). The bulging of the end plate forces blood out of the cancellous bone, which normally is a shock-absorbing mechanism. With further compression, the end plate fractures, nuclear material ruptures into the vertebral body, and the two vertebrae move closer ([Fig. 19-2](#)). If the spine is already flexed when compression is applied, as in tobogganing, the blood is already squeezed out of the vertebral body, the shock-absorbing properties are decreased, and less force is required to injure the spine ([Fig. 19-3](#)) ([57](#)).



**FIGURE 19-2.** Radiograph of a 5-year-old girl who sustained compression fractures of three vertebrae from a sledding accident. Note reversal of the normally convex end plate, as well as breaking and wedging of the bodies. Overlapping of the spongiosum may appear as increased density. The child was asymptomatic within 6 weeks, and complete restitution of vertebral height can be expected.

**FIGURE 19-3.** The radiograph of a 15-year-old girl who sustained an injury to the vertebral body from tobogganing, with fracture of the apophyseal ring ( *arrow*) and displacement anteriorly.

In older children, the nucleus pulposus is no longer fluid, and compression forces are transmitted through the annulus, resulting in either tearing of the annulus with a diffuse collapse of the vertebral body due to buckling of its sides or a marginal plateau fracture ( [112](#)). Stronger compression forces applied more rapidly may cause a bursting type of injury, similar to that in adults ( [Fig. 19-4](#)).

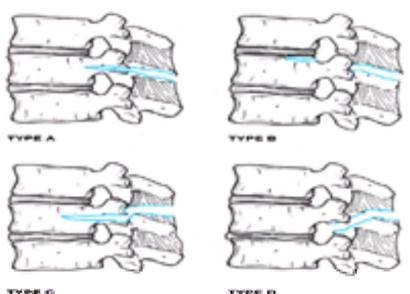


**FIGURE 19-4. A:** The radiograph of a 15-year-old girl who was involved in a motor vehicle accident, sustaining multiple compression fractures, wedging of L1, and anterior buckling of L4 and L5. **B:** The same patient 6 months later. There is persistence of disk space narrowing at T12 to L1. Complete remodeling of this injury is not expected, because she is near the end of skeletal growth. She was treated conservatively and is asymptomatic.

The vertebral disks of children are more elastic than those of adults, and transmit compression forces as a wave through multiple levels ( [63,70,112](#)). This is reflected by the clinical fact that multiple compression fractures, usually in the mid-thoracic to mid-lumbar area ( [49,53,63](#)), are more common in children ( [Fig. 19-2](#)) than in adults ( [49,53,63](#)).

Chance fractures, described by Chance in 1948 ( [25](#)), were more clearly defined by Smith and Kaufer ( [123](#)) as a lumbar spinal injury associated with seat belts. Forward flexion over the belt produces distraction of the posterior vertebral elements and anterior compression, leading to disruption of the interspinous ligaments, ligamentum flavum, and facet capsules. This injury, previously uncommon in children, is increasing with the more frequent use of lap belts because of child restraint laws ( [6,66](#)). The middle and anterior columns may fail through either soft tissues (posterior longitudinal ligament and disk) or bone (vertebral body or end plate). If the fulcrum of rotation is slightly posterior to the anterior margin of the vertebral body, there may be a slight component of vertebral body compression anteriorly. More commonly, however, the fulcrum point is anterior to the vertebral body and the anterior elements fail in distraction.

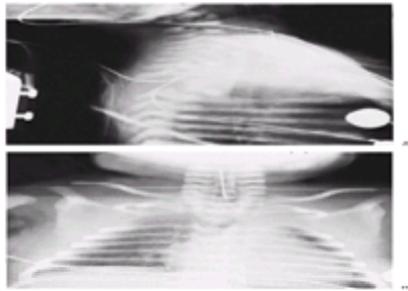
Pediatric Chance fractures are of four types ( [Fig. 19-5](#)) ( [118](#)). A reverse Chance fracture (a hyperextension injury) also has been described in infants and children ( [38,91](#)), rarely with entrapment of bowel ( [113](#)). A fracture through the posterior elements alone without lumbar body fracture also has been reported in children and adults ( [1](#)).



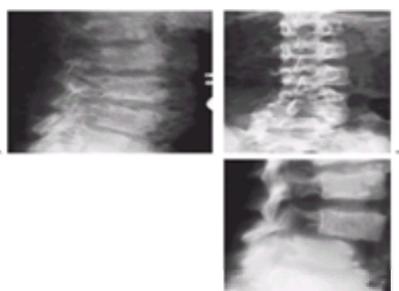
**FIGURE 19-5.** Seat-belt fracture patterns in skeletally immature children. Type A is a bony disruption of the posterior column extending just into the middle column. Type B is an avulsion of the posterior elements with facet joint disruption or fracture and extension into the apophysis of the vertebral body. Type C is a posterior ligamentous disruption with a fracture line entering the vertebra close to the pars interarticularis and extending into the middle column. Type D is a posterior ligamentous disruption with a fracture line traversing the lamina and extending into the apophysis of the adjacent vertebral body. (Adapted from Rumball K, Jarris J. Seat-belt injuries of the spine in young children. *J Bone Joint Surg [Br]* 1992;74:571–574; with permission.)

### Distraction and Shear

In children with violent injuries, such as those sustained from being hit by a car, thoracolumbar spine injuries are caused primarily by shear forces ( [7](#)). The vertebrae typically fracture through the end plate apophyses ( [Fig. 19-6](#)). These injuries also may be associated with flexion and rotation, which leads to shearing and often traumatic spondylolisthesis ( [Fig. 19-7](#)).



**FIGURE 19-6.** The radiograph of a newborn with complete disruption at T3–T4. The child was a breech delivery with cephalopelvic disproportion, leading to a difficult extraction. **A:** Anteroposterior view. **B:** Lateral view, demonstrating that the fracture extended through the cartilage end plate. The child is completely paraplegic at the T3–T4 level and has paralysis of the right hemidiaphragm.

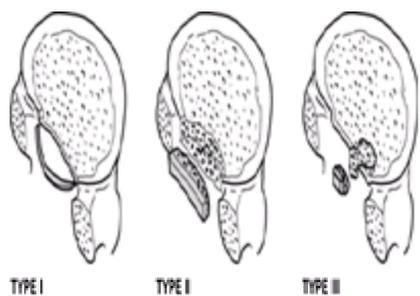


**FIGURE 19-7.** The radiograph of a 13-year-old Boy Scout who sustained this lumbar fracture when a tree fell directly across his back during a severe thunderstorm. **A:** The anteroposterior view demonstrates a shear-type injury with a fracture–dislocation of L4 to L5 lamina. **B:** The lateral view demonstrates a traumatic spondylolysis at the L4–L5 level. **C:** Lateral tomograms demonstrated severe disruption of the pedicle and facet.

### Compression (Slipping of the Vertebral Apophysis)

Slipping of the vertebral apophysis usually occurs in adolescent boys, with traumatic displacement of a lumbar vertebral ring apophysis into the spinal canal and associated disk protrusion (35,126,132). These injuries typically arise from the posterior inferior rim of L4 and less commonly from the inferior rim of L3 or L5. The age and circumstances under which this injury occurs are analogous to those in patients with slipped capital femoral epiphysis (22), which suggests that the vertebral end plate is more susceptible to shear forces during the period of rapid growth. Both chronic and acute forms have been described, and the problem is often erroneously diagnosed as a herniated disk because of the similarity of symptoms (35,126). Many patients (38%) with slipped vertebral apophyses also have lumbar Scheuermann's disease (35), perhaps because the preexisting marginal Schmorl's node weakens the edge and leads to a slip of the apophysis (126).

Slipping of the apophysis can be classified into three types according to radiographic appearance ( Fig. 19-8) (132):



**FIGURE 19-8.** Schematic representation of the three types of avulsion fractures. In type I (**left**), an arcuate fragment is found, but no osseous defect is seen at the posterior rim of the vertebral body. Type II (**middle**) is an avulsion fracture of the posterior rim of the vertebral body that includes a rim of bone. The fragment is not arcuate, and it is thicker than in type I. The sharply avulsed osseous edge is recognized on computed tomography. Type III (**right**) is a localized fracture posterior to an irregularity of the cartilage end plate. The osseous defect anterior to the fragment, as depicted on computed tomography, is larger than the fragment. (Adapted from Takata K, Inoue S-I, Takahashi K, et al. Fracture of the posterior margin of a lumbar vertebral body. *J Bone Joint Surg [Am]* 1988;70:589–594; with permission.)

Type I: Separation of the posterior rim of the involved vertebra. A calcified arc is seen on computed tomography (CT) scan with no evidence of associated large bony fracture. This type is most common in children 11 to 13 years of age.

Type II: Avulsion fracture of part of the vertebral body, annular rim, and cartilage. This type is most common in adolescents and young adults 13 to 18 years of age.

Type III: More localized injury that includes smaller posterior irregularities of the cartilaginous end plate. This is most common in young adults over the age of 18 years.

A type IV injury spans the entire length and breadth of the posterior vertebral margin between the end plates (37).

### SIGNS AND SYMPTOMS

In a child who is awake and cooperative, complaints of pain may point to spinal injury. In older children, pain and tenderness of the spine usually are present, and inability to walk and muscle spasms often are present with unstable spinal injuries. In a child with polytrauma, a significant vertebral fracture may be overlooked (80). Innocuous-appearing fractures of the lumbar or thoracic transverse processes are often associated with serious abdominal injury (20%) to the spleen and liver, pelvis, urinary tract, or chest (2,61,109,119,129,130,144); 50% to 90% of children with lap belt injuries have intraabdominal injuries (6,44,118), most commonly small bowel ruptures and traumatic pancreatitis. Some researchers (48,118) have reported that abdominal injuries are so severe as to dominate the early clinical picture and result in late detection of spinal fractures. Marked soft tissue swelling, bruising, and tenderness along the posterior spinal area are signs that a spinal fracture may have occurred. Ecchymosis in a lap belt distribution should alert the clinician to the possibility of a Chance fracture ( Fig. 19-9) (122).



**FIGURE 19-9.** An abdominal contusion from a lap belt, or the “seat belt sign.” (Reproduced from Jarvis JG. Seat belt fractures. In: Letts RM, ed. *Management of pediatric fractures*. New York: Churchill Livingstone, 1994; with permission.)

Slipping of the vertebral apophysis typically is caused by a traumatic incident such as weight lifting, gymnastics, or shoveling ( 22). Acute slipping produces signs and symptoms similar to those of a central herniated nucleus pulposus, including neurologic findings such as muscle weakness, absent reflexes, and positive straight-leg raising (126). Late findings are similar to those of spinal stenosis.

### Spinal Cord and Root Injury

The most serious diagnostic error is failure to recognize complete or partial paralysis. Neurologic evaluation of a frightened, hurt, uncooperative child is difficult. Gross flexion or reflex withdrawal of the limbs may mimic voluntary movement. Stimulation and handling of the child may produce crying, leading to a false assumption that sensation is intact. Serial observations over time may be necessary to determine the patient's true neurologic status.

Birth injuries to the spinal cord should be suspected in a floppy infant or a child with a nonprogressive neurologic lesion after a difficult delivery. The single most important finding is the demonstration of a sensory level. Somatosensory-evoked potentials may be helpful ( 11).

Nine percent to 15% of all spinal cord injuries occur in children and are twice as frequent in boys as in girls ( 33,62,71). Boys 10 to 15 years of age are at greatest risk for spinal cord injury (33). Young children usually have injuries at the cervicothoracic junction that result in more severe neurologic injury, but they also have more potential for recovery than do older children. Adolescents with thoracolumbar fractures are more likely to have transient or incomplete neurologic deficits that resolve or improve (49).

Delayed onset of paraplegia (2 hours to 4 days) may indicate a vascular insult to the spinal cord ( 26). The injury is typically at the mid-portion of the thoracic spine (watershed area) and usually is associated with a blow to the chest or abdomen, resulting in shock or profound hypotension from internal injuries. This generally results in complete and permanent paraplegia.

In older children, vertebral fractures are the most frequent cause of neurologic injury (83%) ( 71). Fracture–dislocations at the thoracolumbar junction are the most common injuries (36%), with the remainder between T4 and L2 (49,62,63,71). The risk of neurologic injury increases with canal narrowing: spinal canal stenosis of 35% at T11–T12, 45% at L1, and 55% at L2 and below are significant factors for neurologic impairment ( 52).

Recent evidence about burst fractures has suggested that the degree of neurologic injury correlates primarily with the energy of the injury, with no correlation to the amount of osseous or canal disruption. This suggests that the neurologic injury occurs at the time of trauma rather than as a result of pressure in the canal due to remaining osseous fragments (81). Further investigation is necessary to clarify the contributions of canal stenosis and the energy of the injury in the etiology of neurologic damage from thoracolumbar spine fractures.

Spinal cord injury without radiographic abnormality has been reported to occur in 5% to 55% of all pediatric spinal cord injuries, usually in children under 10 years of age (9,43,49,71,76,99,100,117,147). The immature, elastic spine is much more deformable than that of an adult. Momentary displacement from external forces can endanger the spinal cord without causing radiographic disruption of bone or ligaments. Four major factors are believed to be involved: hyperextension, flexion, distraction, and spinal cord ischemia from direct cord contusion or vascular insufficiency ( 82). The neurologic deficit ranges from complete cord transection to partial cord deficits; the cervical and thoracic areas are almost equally involved ( 33). Neurologic loss is more often complete in younger children (birth to 8 years of age), in whom thoracic lesions tend to be neurologically complete more often (92%) than in adolescents (50%). Lumbar lesions are rare and tend to be incomplete in all age groups (33). By definition, no disruption, malalignment, or other bony abnormalities are seen on plain radiographs. Physiologic disruption of the spinal cord is not necessarily associated with anatomic disruption. The exact pathoanatomy is not truly known. Magnetic resonance imaging (MRI) is most useful to study the cord and disk–ligament complexes and correlates with clinical outcome ( 45). The outcome for patients with SCIWORA is primarily determined by the initial neurologic status. Approximately 25% have late deterioration of neurologic function.

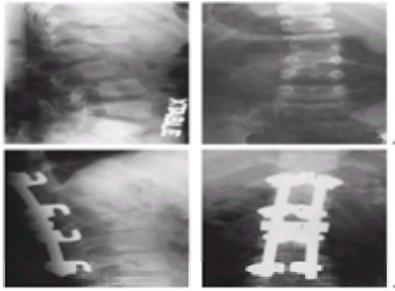
Treatment is controversial. Pang (99) recommended brace immobilization for 3 months, but in his series, no child had instability at initial evaluation and only one child later developed instability demonstrated by flexion–extension radiographs ( 99). Without documented radiographic instability, the biomechanical usefulness of brace immobilization is questionable, although Pang suggests that bracing treats “incipient instability.” In children, normal stability is seldom regained after ligamentous spinal injuries are allowed to heal with simple immobilization, and spinal fusion usually is needed. It seems unlikely that SCIWORA would behave differently regarding instability, incipient or otherwise. Regardless of whether brace immobilization is used or not, close follow-up of neurologic function is necessary. Any sign of instability on flexion–extension radiographs after 3 months of bracing is an indication for surgical stabilization.

Neurologic injuries are uncommon with Chance fractures, although in one series 3 of 10 children had paraplegia ( 118). This may be related to the higher center of gravity in children that results in an increased moment arm and greater distraction of the neurologic structures.

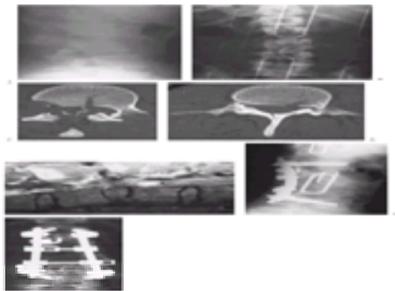
### RADIOGRAPHIC FINDINGS

The radiographic appearance of a spinal injury depends on the force and mechanism of injury. Compression can cause changes ranging from slight flattening of the normally convex end plates to frank wedging of the vertebrae. A zone of increased density in the vertebral body may overlap the trabecular bone ( 53,61). Damage to multiple vertebrae is usually present (the maximum reported number is 11), but clinically observable kyphosis is uncommon unless there is a fracture–dislocation ( 53). True fracture lines are seldom seen in prepubertal children. Avulsed vertebral corners, common in adults, are rare in children ( 43). If the amount of force is significant, the end plate ruptures and the disk is extruded into the vertebral body, forming a Schmorl's node, typically in the lower thoracic and upper lumbar vertebrae ( 10).

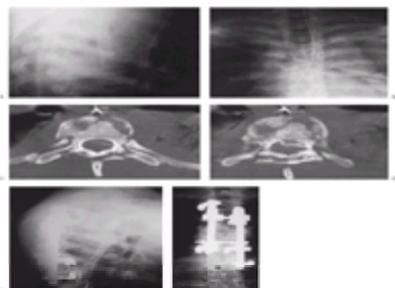
Adult fracture patterns, such as subluxation or fracture–dislocation, which are uncommon in children ( Fig. 19-10), are more common in adolescents ( Fig. 19-11). As in adults, CT scans and sagittal reconstructions are more accurate than plain x-rays in detecting posterior arch fractures ( Fig. 19-12) and retracted bone with spinal canal narrowing (15,42). The CT cuts must be at right angles to the vertebral bodies or the lesion will be confused with a pseudofracture ( 15). In children with slipped vertebral apophyses, CT scanning (35,126) shows a small bony fragment (the edge of the vertebral end plate) within the spinal canal ( Fig. 19-13). MRI shows a large anterior extradural impression (or rarely even a complete block) from the slipped apophysis and protruded disk ( 22,115).



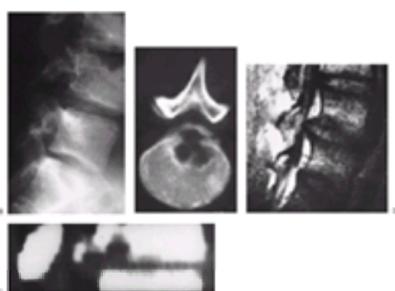
**FIGURE 19-10.** This 7-year-old boy was a restrained passenger involved in a motor vehicle accident and sustained a fracture–dislocation at L1–L2. **A:** Lateral. **B:** Anteroposterior. There was a complete absence of neurologic function below L1 on admission. He underwent open reduction and internal fixation that night with instrumentation and fusion from T10 to L3. **C:** Lateral. **D:** Anteroposterior. At follow-up some 3 years later, he has significant motor recovery, with motor strength being 4/5 psoas, quadriceps, and hamstring; 3/5 triceps surae; 2/5 extensor hallucis longus; and 1/5 tibialis anterior. He is ambulatory with ankle–foot orthoses. (Courtesy of Gregory Graziano, M.D.)



**FIGURE 19-11.** This 16-year-old girl was involved in a motor vehicle accident, sustaining a fracture–dislocation at L2–L3 with complete loss of neurologic function. The initial radiographs [lateral (**A**) and anteroposterior (**B**)] demonstrate a fracture with significant kyphosis at L2–L3. Also note that the fracture involving the posterior elements of L3 extends inferiorly nearly into L4. This created a very unstable situation, and the clinician elected to proceed with instrumentation and fusion. The preoperative computed tomography scan shows a three-column injury (**C**). Also note the L3 laminar fractures (**D**). The preoperative magnetic resonance image (T2-weighted) shows an area of increased signal (*arrow*), representing acute cord edema, as well as an area of decreased signal (*arrowhead*), representing acute intraspinal hemorrhage (**E**). At surgery, hooks could not be placed on the lamina of L3 due to fracture, as noted on the preoperative computed tomography scan. For this reason, the clinician elected to proceed with instrumentation from L1 to L4 using pedicle screws. Due to the marked disruption and instability noted at the time of posterior spinal fusion, the clinician also elected to perform an anterior fusion from L2 to L4. The postoperative radiographs are shown [lateral (**F**) and anteroposterior (**G**)]. (Courtesy of Frances Farley, M.D.)

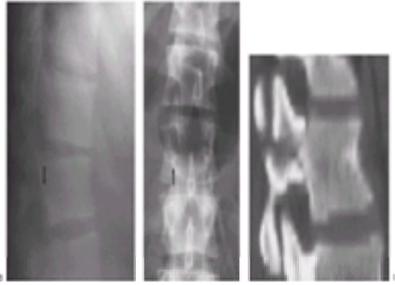


**FIGURE 19-12.** This 17-year-old girl was an unrestrained passenger in a school bus involved in a motor vehicle accident. The rear tire blew out at a speed of 70 mph, causing the bus to roll over several times. She was unconscious for a short period of time, but on arrival in the emergency department she complained of chest pain. Physical examination was pertinent for a sternal contusion, a nontender spine, and a normal neurologic examination of the lower extremities. Chest and spine radiographs demonstrated a fracture of T5. The lateral radiograph demonstrates a marked compression of T5 and kyphosis at T5–T6 (**A**). The anteroposterior radiograph demonstrates a compression of T5, with pedicular widening (**B**). The computed tomographic scan shows fractures of the laminae (**C**), along with retropulsion of bone into the canal (**D**). Due to the kyphosis, retropulsion of bone, and three-column injury, a posterior spinal fusion with distraction instrumentation was performed. Radiographs 1 year after surgery demonstrate correction of the kyphosis [lateral (**E**)] with a normal frontal alignment [anteroposterior (**F**)].



**FIGURE 19-13.** Slipped vertebral apophysis in a 15-year-old with back pain, hamstring tightness, and symptoms suggestive of spinal stenosis. **A:** Lateral radiograph at the lumbosacral junction demonstrates a Schmorl's node and bony changes in the posterior portion of the apophysis of L4. **B:** The computed tomography scan demonstrates a Schmorl's node formation and a fracture of the apophysis with subluxation into the spinal canal. **C:** The computed tomography scan reconstruction of the same lesion. **D:** Magnetic resonance imaging demonstrates slipping of the vertebral apophysis as well as the intervertebral disk, with encroachment of the spinal canal.

Chance fractures in children usually are in the mid-lumbar spine (L1–L3) instead of the thoracolumbar junction as in adults. Routine lateral radiographs are best for making the diagnosis (Fig. 19-14) and should be obtained in any child believed to have abdominal injury caused by a seat belt (139). Avulsion of spinous processes often extends over several vertebral levels, often with anterior compression fractures. CT scans typically do not demonstrate this injury because the cuts are in the same plane as the horizontal fracture–dislocations but can provide further information about the presence of intracanal bony fragments or injury to the osseous posterior arch. If injury to the gastrointestinal tract is suspected, appropriate imaging should be performed.



**FIGURE 19-14.** This 17-year-old boy was involved in a motor vehicle accident sustaining a bony Chance fracture. **A:** The lateral spine radiograph shows the fracture line (*arrow*) through the posterior elements and the vertebral body. **B:** The anteroposterior radiograph also shows the fracture and gap through the posterior elements (*arrow*). **C:** The sagittal reconstruction from the computed tomography scan clearly shows the fracture line through the posterior elements.

Magnetic resonance imaging is better than CT scanning or myelography for evaluation of spinal cord or cauda equina injuries in children ( [13,16,69,133](#)), and obviates the need for intrathecal injection of contrast dye that is necessary for a myelogram. However, false-negative and false-positive MRI results may occur. The presence of spinal instrumentation is not a contraindication to the MRI scan. Three types of MRI patterns are seen on T2-weighted images of acute spinal cord injuries. Type I is a decreased signal consistent with acute intraspinal hemorrhage, type II is a bright signal consistent with acute cord edema, and type III is a mixed signal of central hypointensity and peripheral hyperintensity, consistent with contusion. Patients with type I patterns rarely have improvement in Frankel grade, whereas those with types II and III patterns frequently improve at least one Frankel grade ( [13](#)).

## TREATMENT

### Spinal Cord and Root Injury

The second National Acute Spinal Cord Injury study published in 1990 ( [18](#)) recommended that methylprednisolone be given within the first 8 hours of injury to improve neurologic recovery. Unfortunately, the youngest patient in that study was 13 years old, so the outcome of these recommendations in younger children is unknown. The follow-up study ( [19](#)) further defined the doses and time intervals, but here again the youngest patient in that study was 14 years old. Nevertheless, we do recommend the administration of methylprednisolone in patients with acute, blunt spinal cord injuries. The initial loading dose is 30 mg/kg body weight. If the loading dose is given within 3 hours after injury, then a maintenance infusion of 5.4 mg/kg is given for 24 hours after injury; if the loading dose is given 3 to 8 hours after injury, then a maintenance infusion of 5.4 mg/kg is given for 48 hours after injury. It must be remembered that these recommendations were developed for those who had sustained blunt trauma. There is some evidence that the administration of methylprednisolone in penetrating spinal cord injury may actually impair recovery of neurologic function ( [107](#)). Finally, the administration of methylprednisolone is associated with a higher complication rate, especially pneumonia and sepsis ( [19,41](#)), but without an increase in mortality ( [19](#)).

### Fracture Treatment

Permanent disk space narrowing and spontaneous interbody fusion are uncommon after spinal injuries in children, because the healthy intervertebral disks typically transmit forces to the vertebral bodies ( [65](#)). This, along with the presence of the rib cage in the thoracic spine, makes a stronger case for nonoperative treatment of spinal injuries in children than in adults. Approximately two thirds of spinal fractures in children are stable.

### Flexion Injuries

Simple compression fractures heal quickly with little tendency for further progression. Symptomatic treatment with a short period of bed rest or immobilization with a cast or orthosis usually is sufficient for mild injuries. In studies comparing casting with bed rest, the type of treatment did not affect the outcome, and most children were asymptomatic in 1 to 2 weeks ( [62,63,88](#)). Posterior tenderness in the area of the fracture occasionally persists but usually does not pose any serious problem ( [62](#)). Symptoms may persist for some time after end plate fracture and disk herniation into the vertebral body but usually resolve with conservative treatment.

The brace used for treatment of a flexion injury (compression fracture) can be of two types. The first is a Jewitt brace made of a metal frame with a sternal and pubic anterior pad and a posterior vertebral pad. The lateral screw connection is used to secure and adjust the brace. The second type is a polypropylene "clamshell" brace with Velcro closure and foam lining. The brace is molded to fit the patient, and the anterior shell contacts the sternum and pubis to prevent kyphosis.

If hyperextension is required, it can be best obtained in a cast with extension across the hip joint to a "pantaloon," if extension of the lower lumbar spine is required. A Risser table or adapted spica table is needed to apply these body casts.

### Chance Fractures

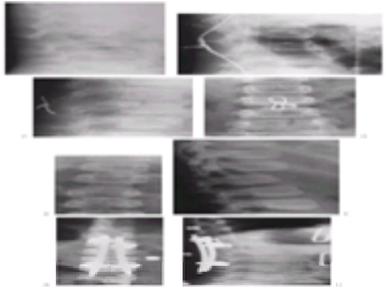
If the injury is truly bony in all columns, closed reduction with reconstitution of lordosis and cast immobilization are appropriate. If the injury is ligamentous, operative reduction with fusion is indicated, because ligamentous disruptions do not heal without instability. Glassman et al. ( [46](#)) reported that brace treatment failed only in patients with an initial kyphosis of more than 20 degrees, and they advocate immobilization if kyphosis is less than 20 degrees. In their series, all children with successful brace treatment had a decrease in kyphosis over time because the potential for anterior growth remained. When operative treatment is required, the type of fixation depends on the age of the child. In small children, simple interspinous wiring can be supplemented by postoperative cast immobilization. In adolescents, standard compression instrumentation can be used.

## OPERATIVE TREATMENT

### Interspinous Process Wiring of Ligamentous Chance (Lap-Belt) Injury

Anesthesia is induced with the patient supine, and appropriate spinal cord monitoring leads are placed. The child is then carefully log-rolled prone onto rolls on a standard operating table. Suspension-type frames (such as the Hall-Relton frame) should not be used because this may allow excessive lumbar sag in an already unstable spine and possibly increase neurologic damage. The legs and distal lumbar spine should be placed directly over the break of the operating table to allow some extension of the legs and distal lumbar spine during the reduction maneuver.

The entire spine and iliac crests are prepared and draped in standard fashion. A posterior midline incision is made over the injured vertebral levels. Often the gap between the spinous processes is easily palpable or ecchymosis is seen. If the level cannot be localized clinically, an intraoperative lateral radiograph should be taken before the incision is made ( [Fig. 19-15A](#)). Dissection is carried sharply down to the level of the thoracolumbar fascia. Dissection must be executed carefully because the injury causes severe disruption of the thoracolumbar fascia and paravertebral musculature. Incising the subcutaneous fat alone often makes the dura visible through the disrupted area.



**FIGURE 19-15.** Chance fracture. **A–E:** This 5-year-old girl was a back-seat passenger involved in a motor vehicle accident at a high rate of speed. She wore a seat belt and sustained a type B Chance fracture. The lateral radiograph intraoperatively obtained for localization of the level for purposes of incision placement (**A**). Note the overlying marker. At L2–L3 note the increased disk space posteriorly with kyphosis and interspinous distance widening, as well as the avulsion fractures of the spinous processes of L1 and L2. The intraoperative lateral radiograph after interspinous wiring, confirming reduction of the kyphosis, interspinous widening, and facet joints (**B**). Lateral (**C**) and anteroposterior (**D**) radiographs 9 months later showing maintenance of the reduction and a solid fusion mass. **E–H:** This 9-year-old child was a restrained passenger in a motor vehicle crash and sustained a type B Chance fracture [anteroposterior (**E**) and lateral (**F**)], which was also associated with a small element of shear, as noted by the slight lateral and more obvious posterior displacement of L1 on L2. He was completely hemiparetic. He underwent a posterior compression type of instrumentation [anteroposterior (**G**) lateral (**H**)]. Two months later, he had complete neurologic recovery. (**E–H** courtesy of William Warner, Jr., M.D.)

When the thoracolumbar fascia is reached, the paravertebral muscles are carefully elevated subperiosteally from the spinous processes of the two disrupted vertebrae. The laminae and disrupted facet joints are exposed by careful lateral dissection. Only the injured levels should be exposed to prevent fusion of uninjured levels or iatrogenic instability. The interspinous ligaments connecting the disrupted vertebrae to the normal cranial and caudal vertebrae should not be removed or violated in any way (removing these ligaments can create an iatrogenic ligamentous instability at the uninjured levels). Normal facet joints also must not be violated. Large hematomas are often encountered during this portion of the procedure; frequent use of warm saline irrigation with careful dissection allows safe exposure of the disrupted facet joints. Small pieces of bone avulsed from the spinous processes and a completely torn ligamentum flavum are often encountered in the disrupted area. The disrupted ligamentum flavum is carefully removed without violating the dura. Here again, frequent and copious use of warm saline irrigation will make the dissection easier by lysing the hematoma and elevating tissues off the dura. The disrupted and frayed facet capsules are removed, as well as any bony fragments in the area of the facets that might prevent anatomic reduction.

A gradual and gentle reduction is begun by grasping the separated spinous processes with towel clips and gradually reducing the kyphosis. The reduction of the disrupted facets is evaluated by visual inspection. Spinal cord-evoked potentials must be carefully monitored during the reduction maneuver. Reduction often can be facilitated by extending the leg portion of the table and bringing the distal lumbar fragment into lordosis. With small awls or towel clips, holes are made at the bases of the spinous processes of the two disrupted vertebrae. The holes should be carefully placed to ensure that the dural sac is not penetrated. A figure-of-eight wire is passed through the holes. The wire is tightened and a lateral radiograph is taken to confirm reduction, using both the facet joints and interspinous distance to confirm anatomic reduction ([Fig. 19-15B](#)). If the reduction is not adequate, the wire is further tightened until an anatomic reduction is achieved. The wire is then cut and bent. The articular cartilage of the facet joints is destroyed at the level of the fusion, and an autogenous bone graft is placed at the levels to be fused. To minimize the risk of stenosis, either a local fat graft or gelatin sponge is placed over the exposed dural sac before the bone graft is placed.

If the spinous processes are too small to place the wire through drill holes, the wire can be placed around the spinous processes. With a small Kerrison rongeur, a small trough is made in the base of the spinous process, superiorly at the superior vertebrae and inferiorly at the inferior vertebra. The interspinous ligaments connecting the disrupted vertebrae to the normal cranial and caudal vertebrae should not be removed or violated in any way. The spinous processes are then wired together in a figure-of-eight fashion by placing the wire in the troughs in the spinous processes.

After routine closure, the child is carefully rolled supine and a pantaloons cast is applied, with care to maintain lumbar lordosis. Radiographs are taken after the cast has hardened, and then the child is awakened.

## OPERATIVE TREATMENT

### Compression Instrumentation of Ligamentous Chance (Lap-Belt) Injury

In an older child or adolescent, compression instrumentation can be used ([Fig. 19-15C](#)), usually obviating the need for postoperative cast immobilization. The same intraoperative positioning and exposure as for interspinous wiring are used. After removal of the disrupted and frayed facet capsules, ligamentum flavum, and bone fragments in the area of the facets, instrumentation is begun. A claw-type construct is made using two laminar hooks (one on the right and one on the left of the spinous process) in a downgoing fashion over the lamina at the most superior level, and two laminar hooks (again one on the right and one on the left of the spinous process) in an upgoing fashion over the lamina at the most inferior level. If the superior hooks are at the level of the spinal cord or conus, the right and left hooks can be placed at successive laminar levels instead of at the same level to minimize the decrease in intracanal space owing to the sublaminar hooks. Again, the interspinous ligaments connecting the disrupted vertebrae to the normal cranial and caudal vertebrae should not be removed or violated in any way. If the instrumentation is of the Cotrel-Dubousset type, all the hooks should be closed.

Rods of appropriate length are measured and inserted into the hooks. Gradual compression is applied, alternately compressing right and left sides. The adequacy of reduction of the disrupted facets is evaluated by visual inspection. Spinal cord-evoked potentials are carefully monitored during the reduction maneuver. A lateral radiograph is taken to confirm reduction, using both the facet joints and interspinous distance to verify anatomic reduction ([Fig. 19-15E](#)). After confirmation of reduction, the hooks are firmly tightened onto the rods, and a fusion is performed as previously described. When possible, crosslinking should be used between the two rods.

After routine closure, the patient is carefully rolled supine and radiographs are taken to confirm maintenance of the reduction and instrumentation position. Depending on the degree of intraoperative stability and compliance of the patient, either no immobilization or a thoracolumbosacral orthosis is used.

### **Distraction and Shear Injuries**

Unstable injuries, such as vertebral subluxation or fracture–dislocation, should be reduced ([21,63,65,88](#)). The child should be placed on a turning frame or at complete bed rest with log rolling until the acute symptoms subside, and then operative reduction and fixation should be performed. In children with neurologic injury, the fracture should be reduced promptly, especially if the neurologic injury involves the conus medullaris and nerve roots. Children with burst fractures that result in kyphosis and spinal canal narrowing of more than 25% are at risk of further canal compromise, and early correction and decompression should be considered ([21](#)).

These adult fracture patterns usually occur in adolescents. Spontaneous interbody fusion seldom occurs; clinicians should not rely on such fusion to provide long-term stability ([63,88](#)). The spinal canal and vertebral elements are the same size as in adults, and adult instrumentation is used for reduction and stabilization ([Fig. 19-11](#) and [Fig. 19-12](#)). All instrumentation must be accompanied by posterior spinal fusion at least one level above and below the level or levels of injury. In older children with severe neurologic deficits, extending the fusion to the sacrum to prevent late onset of paralytic scoliosis is rarely advocated ([76](#)).

Some injuries that are stable in adults may be progressive in children. Severe crushing of the vertebral body and end plate (burst fractures), with or without disruption of the posterior supporting ligaments or laminectomy, is analogous to a Salter-Harris type IV injury of the vertebral apophysis. Growth arrest leads to progressive kyphosis ([88](#)). Early recognition, reduction, stabilization, and fusion prevent late deformity and neurologic compromise.

The indications for immediate surgical decompression ([49](#)) are the same as in adults: an open wound and progressive neurologic deficit in an incomplete spinal cord

injury. Reduction of an unstable fracture–dislocation is a relative indication. Laminectomy is seldom helpful, especially in children without bony injury ( 49,65). It accentuates an already unstable condition, which may lead to progressive deformity ( 146) such as kyphosis, which is difficult to manage ( 72,76). If laminectomy is necessary, it should be accompanied by a short segment fusion.

### Slipped Vertebral Apophysis

Surgical treatment is nearly always needed. Laminectomy and decompression with removal of the bony ridge and disk give excellent results ( 22,126). Disk removal alone is not sufficient to relieve the nerve root impingement ( 22).

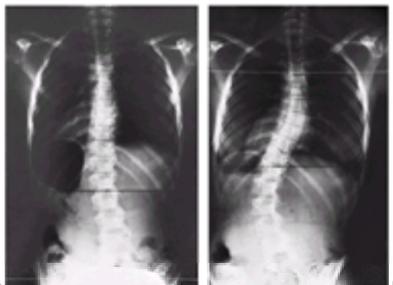
## PROGNOSIS AND COMPLICATIONS

### Growth Arrest

Progression of the vertebral body deformity is uncommon in children, unless the injury is unstable, such as a fracture–dislocation, or a neurologic deficit is present. The vertebral bodies have great potential for restoration because of the stimulation of vertebral growth and overgrowth ( 53,63). In children who are under 10 years of age or have a Risser sign of 0 or 1 at the time of injury ( 106), the vertebral body tends to return to its normal shape, even after multiple compression fractures, and kyphosis is uncommon (62,63,65,69,70,71). However, complete reconstitution is possible only if the nucleus pulposus does not protrude into the vertebra ( 62). The vertebral end plate is the area of active growth, and if it is damaged, little subsequent correction of the deformity will occur. The undamaged adjacent vertebra usually compensates for asymmetric growth of a damaged vertebra, especially in the thoracolumbar area, and significant scoliosis seldom occurs ( 62). Spontaneous interbody fusion is rare in children; the interposed, undamaged, intervertebral disk is thought to block this process ( 63,71,112).

### Spinal Cord and Root Injury

The most devastating complication of thoracolumbar spinal injuries is paraplegia. A child with a spinal cord injury has the same problems as an adult with spinal cord injury: increased susceptibility to long bone fractures, hip dislocation, pressure sores, joint contractures, and genitourinary complications ( 9). In addition, a child is likely to develop progressive spinal deformity (scoliosis, kyphosis, and lordosis) ( Fig. 19-16) (9). For many children, the original vertebral injury often is overshadowed by the severity of these late spinal deformities (9). Scoliosis erodes the ability to sit easily, and in young children pelvic obliquity may lead to subluxation of the hip and ischial pressure sores ( 72,76).



**FIGURE 19-16.** The radiograph of a 9-year-old girl who sustained a motor vehicle accident and polytrauma. There were no radiographic vertebral fractures, but she was a complete paraplegic at T10, believed to be due to a vascular injury and hypotension. **A:** The anteroposterior radiograph at 11 years of age demonstrates a mild collapsing type of scoliosis. **B:** By 13 years and 9 months of age, the curve had increased to 50 degrees despite aggressive orthotic management. This scoliosis required surgical instrumentation and fusion.

In children (girls under 12 and boys under 14 years of age), the incidence of progressive spinal deformity after traumatic paraplegia is 86% to 100% ( 9,86). The onset of curvature has been reported to occur in children as young as 3 years ( 9). The fracture seldom determines the direction of curvature; rather, most children develop a long paralytic thoracolumbar curve thought to be caused by the influence of gravity and the uneven forces of spasticity ( 72). A long thoracolumbar kyphosis reverses the normal lumbar lordosis ( 76,86). Increased lumbar lordosis is less common (18%) and usually is associated with hip flexion contractures in an ambulatory patient ( 72,86). Progression of the spinal curvature is directly related to the age of the child, the degree of spasticity, and the level of the lesion ( 76,86). Children with more proximal injuries are more likely to have a progressive deformity than are those injured at or below the level of the conus medullaris ( 9).

In adolescents who are near skeletal maturity at the time of injury, spinal deformity is more often caused by the fracture–dislocation itself ( 86). Progressive kyphosis and pain at the fracture site are common (42%), especially after laminectomy ( 72,86). If the kyphosis is progressive, long-term neurologic sequelae may develop, with further loss of function from tenting of the neural structures over the kyphos. This is another reason for early surgical stabilization of these fractures in adolescents ( 86).

Treatment of scoliosis should be initiated soon after the injury, before a severe curve develops. Total-contact underarm plastic orthoses have been helpful in at least temporarily controlling the collapsing paralytic curve. Treatment recommendations are similar to those for idiopathic scoliosis. Curves of less than 40 to 45 degrees may be controlled by bracing, or at least surgery can be delayed until further spinal growth has occurred and the child is of optimal age ( 74,76). For curves of more than 45 to 50 degrees, surgical stabilization should be performed. In one series ( 86), 68% of children required surgical correction. Children with severe or rigid curves may require anterior release with or without halo-wheelchair traction. Segmental instrumentation, such as Harrington or Luque rods with sublaminar wiring, or the newer rotational systems (such as Cotrel-Dubousset) are used, along with arthrodesis. The child should be quickly mobilized after surgery to avoid the problems associated with long periods of bed rest (such as pneumonia and decubiti in insensate areas).

With the advent of MRI, the “rare” posttraumatic syringomyelia is being discovered with increasing frequency ( 13). Symptoms can develop many years after injury (4.5 years average), even as late as 15 years ( 141). Pain is the initial symptom in over half of the children, followed by progressive neurologic loss as demonstrated by sweating below the level of the original lesion, loss of motor function, and changes in the deep tendon reflexes ( 141). The best method to detect syringomyelia is MRI, and some researchers have recommended that an initial baseline MRI be obtained to make later detection of syringomyelia easier ( 13,83).

### Other Spinal Injuries

The fractures discussed in the preceding sections are acute traumatic injuries. Fractures or fracture-like conditions of the thoracic and lumbar spine in children can result from causes other than acute trauma, including repetitive stress, systemic disease that creates pathologic bone, societal mechanisms of trauma (such as child abuse and gunshot wounds), spondylolysis and spondylolisthesis, and lumbar and dorsolumbar Scheuermann's disease.

### Stress Fractures

Stress fractures ( 31) are caused by abnormal or repetitive loading of normal bone. They typically occur in young active individuals, most often in the metatarsals and tibia in children. Grier et al. ( 47) reported stress fractures of the sacrum in two children, both of whom had pelvic pain and positive FABER (flexion, abduction, external rotation) tests. A bone scan can be used to localize the pain, and the diagnosis is confirmed by CT scanning. Treatment is nonoperative.

### Systemic Diseases

Spontaneous collapse of a single vertebral body, especially in a child between the ages of 2 and 6 years ( 96), suggests the possibility of an eosinophilic granuloma ( Fig. 19-17). Usually there is complete collapse of the body (vertebra plana), and the lytic appearance typical of this lesion in other areas of the skeleton is rarely

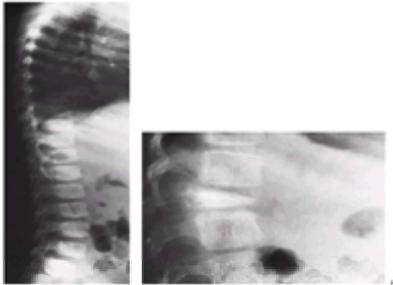
seen. The intervertebral disk is not affected. An adjacent soft tissue mass is uncommon and, if present, suggests an infectious process (such as tuberculosis or a bacterial process) or Ewing's sarcoma, instead of eosinophilic granuloma. The prognosis is excellent, with some reconstitution of the height of the vertebral body and little residual deformity. However, complete reconstitution rarely occurs.



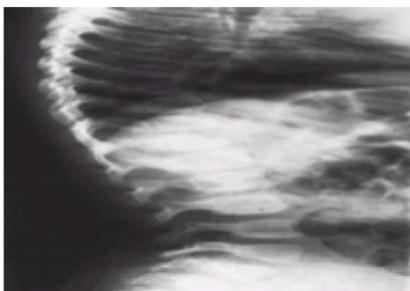
**FIGURE 19-17.** The lateral radiograph of a girl 2 years and 1 month of age with eosinophilic granuloma of T12. Note the vertebra plana with maintenance of the disk space height.

Multiple vertebral collapse is common in patients with Gaucher's disease, mucopolysaccharidoses, leukemia, and neuroblastoma ([4,40,89,108](#)). The abnormal cells displace the normal bone-forming elements, and the vertebra becomes structurally weak and collapses with minor trauma. Usually these children have visceral as well as skeletal involvement at other sites. A bone scan and skeletal survey should be obtained to identify these other sites. Typical symptoms are persistent pain in the region of the collapse. Neurologic complications are rare. In children with leukemia the peripheral leukocyte count may be normal despite vertebral involvement and collapse ([89](#)).

In patients with Gaucher's disease, the bone-forming elements are replaced by an infiltrate of glucocerebroside-containing reticulum cells. One or more vertebrae may collapse, but a gibbus is rare ([Fig. 19-18](#)). The mucopolysaccharidoses, chondrodystrophies, and lipidoses are similar to Gaucher's disease, with cellular storage of an abnormal metabolite and structural weakness. Vertebral changes are typically first noted at the thoracolumbar junction, with anterior herniation of the nucleus pulposus, and appear on radiographs as a "beaking" of the vertebral body ([Fig. 19-19](#)). Depending on the severity of the disease, the process may involve the entire spine ([4](#)).

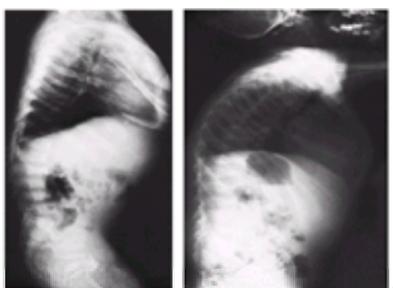


**FIGURE 19-18.** Gaucher's disease. The illness is progressive with increased storage of the abnormal metabolite and structural weakening of the vertebral elements. **A:** Normal appearance of the thoracolumbar spine at 5 years of age. **B:** At 8 years of age a spontaneous compression fracture of L1 has occurred. The patient sustained many similar fractures until her death 3 years later.



**FIGURE 19-19.** The lateral radiograph of a 13-month-old infant evaluated for delay in motor milestones and subluxation of the hips. Note the beaking of the T12 vertebral body. This finding is compatible with storage diseases such as mucopolysaccharidoses, mucopolipidoses, or hypothyroidism. In this situation, further clinical and laboratory investigation is required. This particular child was diagnosed with Hurler's syndrome on the basis of laboratory studies.

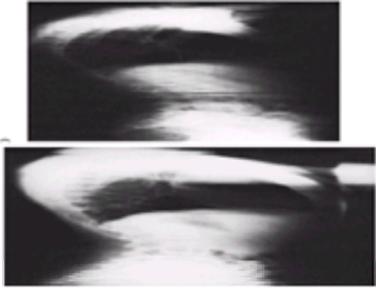
Serial compression fractures are common in patients with osteogenesis imperfecta ([Fig. 19-20](#)). However, these children usually have the typical stigmata, such as long bone fragility, extremity deformity, and blue sclera. Progressive spinal deformities of scoliosis and kyphosis are common in severely involved children. Compression fractures with kyphosis also can be caused by certain conditions associated with systemic osteoporosis, such as cystic fibrosis, or as a consequence of treatment, such as corticosteroid-induced osteoporosis in children with severe juvenile rheumatoid arthritis ([114,137](#)).



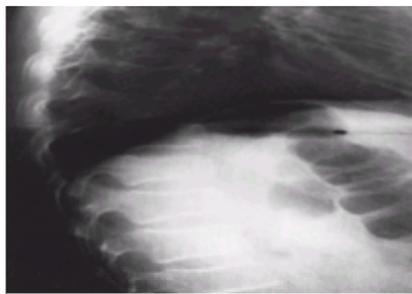
**FIGURE 19-20.** Compression fractures seen in osteogenesis imperfecta. **A:** The lateral radiograph of the spine at 3 years of age with compression fractures of the lumbar vertebrae. **B:** At age 7, compression fractures of every vertebrae, with narrowing of the vertebral body and apparent increase of the intervertebral disk spaces

representing disks that have retained their normal elasticity.

Compression fractures also occur in patients with idiopathic juvenile osteoporosis ( [Fig. 19-21](#) ), an unusual acquired systemic condition characterized by profound osteoporosis in otherwise normal prepubertal children, typically between the ages of 8 and 15 years ( [68](#) ). The condition may be confused with osteogenesis imperfecta; however, it is of limited duration, usually 1 to 4 years, and is followed by nearly normal restoration of the skeleton. Osteoporosis is evident radiologically, and fractures occur with minimal trauma. There is no family history, and patients do not have the blue sclera, wormian bone skull changes, or poor teeth characteristic of osteogenesis imperfecta ( [68](#) ). Initial complaints are usually related to the spine, and if the condition is recognized, early treatment with a Milwaukee brace improves the appearance and reduces the amount of kyphosis and residual deformity. An even rarer osteoporosis is associated with heparin use, and compression fractures rarely occur in children with severe cardiac disease that requires long-term heparin treatment to prevent thromboembolic phenomena ( [Fig. 19-22](#) ).



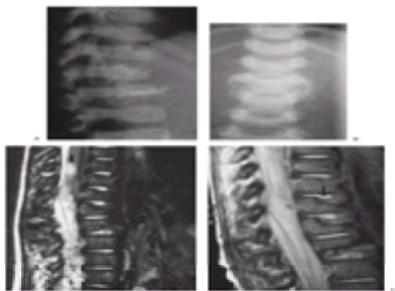
**FIGURE 19-21.** Idiopathic juvenile osteoporosis. This is a systemic condition characterized by the onset of osteoporosis in an otherwise normal prepubertal child, typically between the ages of 8 and 15. **A:** The patient has sustained multiple spontaneous compression fractures with considerable kyphosis and deformity of the vertebrae. **B:** Early treatment with a Milwaukee brace improved his appearance and reduced the degree of kyphosis and residual deformity.



**FIGURE 19-22.** The lateral radiograph of a 15-year-old boy on long-term subcutaneous heparin anticoagulation for a congenital cardiac condition. Note the osteopenia of the vertebrae, the central compression fractures of the vertebral bodies, and the preservation of the intervertebral disk height.

### **Spinal Fractures Caused by Child Abuse or Gunshot Wounds**

Vertebral injuries caused by child abuse are less common than injuries of the extremities ( [131](#) ). Spinal injuries are usually caused by hyperflexion and may be simple compression of the vertebral bodies, avulsion of the secondary centers of ossification of the spinous processes, or, less frequently, herniation of the nucleus pulposus into the vertebral body. Fracture–dislocations ( [Fig. 19-23](#) ) or kyphosis secondary to severe disruptions are rare, ( [27,34,73,131](#) ), although there are striking examples of T12–L1 subluxation–dislocation with complete paraplegia from spanking ( [32,110](#) ). These are actually Salter-Harris type I fractures of the vertebral centrum from the cartilaginous end plate and may mimic a central nervous system tumor ( [24](#) ). There are no radiographic vertebral changes specific for the battered child syndrome. Kleinman and Marks ( [73](#) ) found three main patterns: (a) compression, usually no more than 25% of the anterior half of the vertebral body; (b) fracture of the anterosuperior aspect of the vertebral body with extension into the anterior aspect of the end plate; and (c) combined lesions. Similarly, the classic corner fractures of the long bones are present in only 15% of abused children. A high index of suspicion is more helpful than a specific radiographic finding. The children may exhibit other signs of neglect, such as poor nutrition and poor hygiene.



**FIGURE 19-23.** The radiographs of a 10-month-old girl with paraplegia from a thoracolumbar fracture due to child abuse. The child presented to the emergency department with “acute onset” paraplegia. A skeletal survey also discovered healing clavicle and rib fractures, consistent with abuse. After investigation by the Department of Social Services, the father was criminally prosecuted and subsequently incarcerated. **A:** The lateral radiograph shows anterosuperior Schmorl's nodes at T12 to L2, as well as compression of the vertebral body of L2. There is callus formation at the inferior aspect of the L2 vertebral body. Note that the disk height is relatively preserved. **B:** The anteroposterior radiograph shows callus between L2 and L3 and compression of L2. **C:** The magnetic resonance image (T2-weighted) shows an area of increased signal intensity, representing cord edema ( *arrow* ). **D:** The magnetic resonance image (T1-weighted) shows the protrusion of the intervertebral disk into the anterior margin of the vertebra ( *arrow* ) with its associated compression of the body. Note the multiple levels of disk disruption and vertebral body compression.

Spinal injuries caused by gunshot wounds are increasing in frequency. At Rancho Los Amigos Medical Center, between 1985 and 1989, gunshot wounds in children accounted for the same number of spinal cord injuries as did motor vehicle accidents (38% each). Before 1970, gunshot wounds accounted for only 6% of spinal injuries ( [50](#) ). Laminectomy does not affect the outcome of patients with complete lesions and is detrimental to those with incomplete lesions ( [127](#) ), resulting in cerebrospinal fluid fistulas, infection, and late instability. Thus, laminectomy is rarely needed ( [28,127](#) ), and the only indication for surgery is a progressive neurologic deficit ( [54](#) ). One exception may be a motor lesion between T12 and L4, where one series showed a significantly better motor recovery after bullet removal ( [140](#) ).

## Spondylolysis and Spondylolisthesis

Spondylolysis occurs in approximately 5% of the general population. Its origin is controversial, with data supporting both developmental and congenital origins. Congenital deficiency of the sacrum and lack of integrity of the posterior structures, on a genetic basis, may predispose to spondylolysis. Developmental factors such as trauma, posture, or certain repetitive activities may cause a stress fracture of the pars interarticularis.

Spondylolysis occurs in children after walking age. It is rare in children younger than 5 years of age and has been reported in only one infant ( 17). It is most common in children aged 7 or 8 years, suggesting that trauma is a prominent factor in the etiology ( 8,143). Spondylolysis can only be produced by forced hyperextension, and laboratory tests indicate that a high degree of force is required ( 60). Although minor trauma is common (50% of males and 25% of females) and often initiates the onset of symptoms, seldom is the injury severe (56). Rather, the onset of symptoms coincides closely with the adolescent growth spurt ( 135).

Spondylolysis at L5–S1 is often categorized as a congenital anomaly of the spine. Although there is no supporting embryologic or anatomic evidence for this designation, several findings favor a congenital origin. A high rate of occurrence among family members and certain ethnic groups has been reported by numerous researchers, with an incidence of 27% to 69% in near relatives versus an expected frequency of 4% to 8% in the general population ( 145). There are also racial and sex differences, with the lowest incidence in black females and the highest (6.4%) in white males. Persons with spondylolysis have an increased incidence of sacral spina bifida (28% to 42%) and congenital lack of development of the proximal sacrum and superior sacral facets ( 145).

Substantial documentation supports the theory that spondylolysis represents a stress fracture of the pars interarticularis ( 77,143). Some researchers have postulated that lumbar lordosis is accentuated by the normal hip flexion contractures of childhood, focusing the force of weight bearing on the pars interarticularis and leading to gradual disruption (75). Anatomic studies suggest that shear stresses are greater on the pars interarticularis when the spine is extended ( 75,77). In children, the pars interarticularis is thin, the neural arch has not reached its maximal strength, and the intervertebral disk is less resistant to shear ( 29). A fatigue fracture can occur at physiologic loads during cyclic flexion–extension motion of the lumbar spine ( 77). These stresses may be further accentuated by lateral flexion movements on the extended spine, as may occur during a back walk-over in gymnastics (75,77). Spondylolysis is four times more frequent (11%) in female gymnasts than in other girls of the same age (64); many initially have normal radiographic results and later develop spondylolysis. Recent evidence has demonstrated the presence of synovial pseudarthroses at the site of the spondylolysis, which also communicates with the facet joint just superior to it. This suggests that stress fractures of the pars interarticularis due to repetitive trauma fail to heal because of the presence of synovial fluid from a nearby facet joint; this eventually results in a spondylolysis ( 121).

Acute spondylolysis has been reported in soldiers who carry heavy backpacks or perform exercises to which they are unaccustomed ( 143). In many, the defect healed with conservative treatment, whereas others developed pseudarthrosis, persistent symptoms, and the typical radiographic appearance of spondylolysis. An increased frequency of spondylolysis has been noted in teenagers with thoracolumbar Scheuermann's disease, a condition believed to be caused by excessive and repetitive mechanical loading on the immature spine (98). Similarly, thoracolumbar kyphosis is often associated with a compensatory increase in lumbar lordosis. Those performing heavy physical work, such as weight-lifters, lumberjacks, and football lineman, also have been reported to be more at risk for spondylolysis ( 79).

### Signs and Symptoms

Although spondylolysis commonly occurs in late childhood or early adolescence, symptoms are relatively uncommon in children and rarely are severe enough to require medical attention during the teenage years ( 39,55). In a prospective longitudinal survey, only 13% of children known to have spondylolysis developed symptoms before 18 years of age (39). In the occasional child who develops symptoms, the onset usually coincides with the adolescent growth spurt ( 56,120).

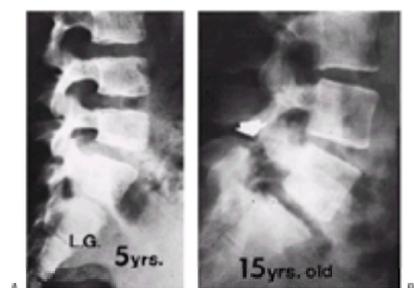
Although pain is the major complaint in adults, most children and adolescents do not have pain and seek medical attention only because of a postural deformity or gait abnormality caused by hamstring tightness. Pain generally is localized to the low back and, to a lesser extent, the posterior buttocks and thighs ( 56,120). Symptoms usually are initiated or aggravated by strenuous activity of the spine common to oarsmen, gymnasts, divers, hockey players, tennis players, and baseball pitchers. A combination of these factors are present when too much weight is used by weight-lifters performing the military press, when incorrect technique is used by gymnasts, and when swimmers or runners make too rapid advances. Symptoms usually are decreased by rest and limitation of activities ( 56,64,77).

Physical examination may demonstrate some tenderness to palpation in the low back. There may be some splinting, guarding, and restriction of side-to-side motion in the low back, especially if onset of the condition is acute. If hamstring tightness is present, forward flexion of the hips is markedly restricted. Eighty percent of symptomatic patients have hamstring tightness (so-called spasm), and this has been attributed to nerve root irritation; however, no objective evidence supports this contention ( 8,105,135). Hamstring tightness is seldom accompanied by neurologic signs ( 8,120).

Children, unlike adults, seldom have objective signs of nerve root compression, such as motor weakness, reflex change, or sensory deficit ( 135). Examination, however, should include a careful search for sacral anesthesia and bladder dysfunction. Similarly, children with spondylolysis rarely have myelographic evidence of disk protrusion or evidence of herniation at surgical exploration ( 56,135).

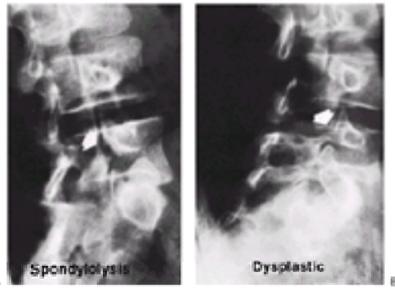
### Radiographic Findings

If the radiolucent defect in the pars interarticularis is large ( Fig. 19-24), it can be seen on nearly all radiographs of the lumbar spine. If the defect is unilateral, as it is in 20% of patients, or is not accompanied by spondylolisthesis, special imaging techniques may be required ( 78,85).

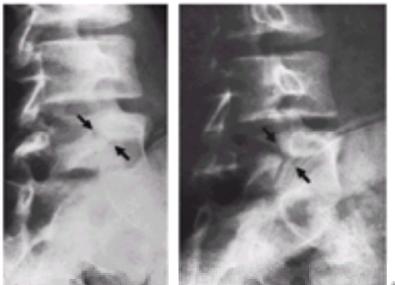


**FIGURE 19-24. A:** The radiograph of a 5-year-old girl with normal appearance of the lumbosacral junction. **B:** The same person at age 15. Spondylolysis and grade I spondylolisthesis have developed in the interim with symptoms referable to the low back. Spondylolysis in children occurs after walking age, but rarely before 5 years, and more commonly at 7 or 8 years. Onset of symptoms coincides closely with the adolescent growth spurt.

Oblique views show this area in relief and apart from overlying bony elements; the diagnosis is missed in 20% of young patients if oblique views are not obtained ( 78). The “Scotty dog” of Lachapelle, with the defect appearing at the terrier's neck, is a helpful visual aid to those inexperienced with oblique radiographs ( Fig. 19-25). In an acute injury, the gap is narrow with irregular edges, whereas in a long-standing lesion, the edges are smooth and rounded, suggesting a pseudarthrosis ( Fig. 19-26). The width of the gap depends on the amount of bone resorption after the fracture and the degree of spondylolisthesis.



**FIGURE 19-25. A:** Pars interarticularis defect, or spondylolysis, as seen on an oblique view ( *arrow*), typically found with the isthmic type of spondylolisthesis. **B:** Similar oblique view of a patient with dysplastic (congenital) spondylolisthesis, demonstrating elongation and attenuation of the pars interarticularis, perhaps a prespondylolytic defect, and may represent a stress or fatigue fracture of the pars intrarticularis.



**FIGURE 19-26.** The radiograph of a 13-year-old boy who felt a snap in the low back during a swimming race turn. **A:** Spondylolysis of the pars interarticularis is seen (*arrow*). The narrow, irregular appearance suggests recent injury. **B:** Despite cast immobilization, the fracture did not heal. The appearance 6 months later demonstrates blunting of the bone ends and widening of the gap. However, the patient was asymptomatic.

Less commonly, symptomatic children (26%–35%) have poorly developed or dysplastic (Wiltse type I) posterior structures ( [55,142](#)). Because of anomalous development, the posterior facets appear to subluxate on the sacral facets. In children with dysplastic posterior structures, rather than a gap or defect in the pars interarticularis, the facets appear to subluxate and the pars interarticularis may become attenuated like pulled taffy. This has been called the “greyhound” of Hensinger ( [56](#)); a defect may later appear in the center. This may be a different manifestation of the same disease process, because both lesions are present in members of the same family ( [145](#)), suggesting that the spondylolytic type represents an acute stress fracture of the pars interarticularis and the dysplastic or elongated type represents a chronic stress reaction with gradual attenuation of the pars.

Deficiency of the posterior elements is common in patients with spondylolysis. Easily observable defects, such as dysraphic or malformed laminae, have been reported in 32% to 94% of these patients, and, if discovered on routine lumbosacral views, should prompt a more detailed radiographic investigation ( [145](#)). CT scanning is rarely indicated in acute fracture of the pars interarticularis because a combination of bone scanning and oblique x-rays is more reliable in detecting the lesion ( [116](#)). Spondylolysis at L4 or L3 is difficult to diagnose with CT scans because the plane of scanning is parallel to the fracture ( [116](#)). MRI should be considered if the patient’s symptoms or neurologic signs do not resolve with bed rest, or if bladder or bowel dysfunction or perineal hypesthesia is present.

When the diagnosis is suspected clinically but cannot be confirmed radiographically (especially in the stress reaction stage before fracture), a bone scan will be helpful, especially a single photon emission CT scan ( [12,85](#)). Small, partial, or unilateral fractures can be overlooked on x-rays, but bone scans demonstrate an area of increased bone turnover caused by healing of the fracture. Bone scanning may demonstrate increased uptake in patients with only 5 to 7 days of symptoms ( [101,136](#)). Later, up to 1 year after onset of symptoms, bone scanning is helpful to distinguish between patients with an established nonunion (cold bone scan) and those in whom healing is still progressing (hot bone scan) and who may benefit from immobilization ( [77,136](#)). Bone scanning is not recommended in patients whose symptoms are of more than 1 year’s duration or who are asymptomatic ( [136](#)), unless a bone tumor (such as osteoid osteoma), infection, or malignancy is suspected.

Bone scanning is especially helpful in young athletes whose activities are highly associated with spondylolysis, such as gymnastics ( [64](#)). Early detection of the stress reaction allows early treatment, which can shorten the recovery period. Bone scanning also can be used to evaluate recovery and determine when an athlete can return to competition ( [64](#)).

### Treatment

In some children and adolescents, the spondylolytic defect may heal with 3 to 6 months’ casting or bracing (thoracolumbosacral orthosis) if there is an acute, clearly documented onset with injury ( [135,136,143](#)) or if the lesion is early in its course ( [93](#)). Healing is more likely with unilateral defects than bilateral defects. A bone scan may be helpful to differentiate a continuing process from one of a long duration ( [77](#)).

In children in whom the spondylolysis is of long duration, healing is unlikely, but symptoms usually respond to conservative measures ( [56](#)). Restriction of vigorous activities and back and abdominal strengthening exercises usually are successful in controlling mild backache and hamstring tightness ( [55](#)). Patients with more severe or persistent complaints may require bed rest, cast or brace immobilization, and nonnarcotic analgesics ( [128](#)). Hamstring tightness is an excellent clinical guide to the success or failure of the treatment. Most children have complete relief of symptoms or only minimal discomfort at long-term follow-up ( [135](#)).

Any child or adolescent with symptoms caused by spondylolysis, especially those under 10 years of age, should be closely followed for progression to spondylolisthesis ( [143](#)). We do not advise those with asymptomatic spondylolysis or those with minimal symptoms to restrict their activities; 7.2% of asymptomatic young men 18 to 30 years of age have pars defects and relatively few have persistent symptoms ( [92](#)). Progression is unlikely with competitive sports activities ( [94](#)). Thus, limitation of activity in a growing child does not seem justified ( [55](#)). It must be emphasized that spondylolysis is rarely symptomatic in adolescence, and caution is advisable in treating a child whose symptoms do not respond to bed rest or who has objective neurologic findings. In this situation, MRI and possibly electromyography should be considered.

A small percentage of young persons with spondylolysis do not respond to conservative measures or are unwilling to curtail their activities and may require surgical stabilization. If surgery is necessary, a posterolateral fusion from L5 to S1 with autogenous bone graft usually is sufficient. If spondylolisthesis is of grade III or greater, the fusion usually is extended to L4 ( [56](#)). Nachemson ( [95](#)) reported solid healing of the defect after bone grafting and intertransverse process fusion.

In patients with small defects (6–7 mm) and only slight spondylolisthesis, a variety of techniques have been described for direct reduction of the defect, including wiring of the transverse process or placement of a screw across the pars, coupled with a bone graft (such as in a pseudarthrosis repair) ( [20,36,97,104](#)). These procedures usually are recommended for older adolescents and young adults (<30 years of age) with minimal displacement and degenerative change ( [95,97](#)). The best candidates for this method of treatment are those with defects between L1 and L4 or those with multiple defects. This is an attractive alternative to the traditional transverse process fusion because it repairs the defect at one vertebral level rather than involving a second nonaffected vertebrae ( [20,104](#)). In properly selected patients, 80% to 90% obtain a solid fusion with good to excellent results. In children, the Gill procedure or laminectomy should never be performed without an associated fusion because removal of the posterior elements may lead to increased instability and spondylolisthesis.

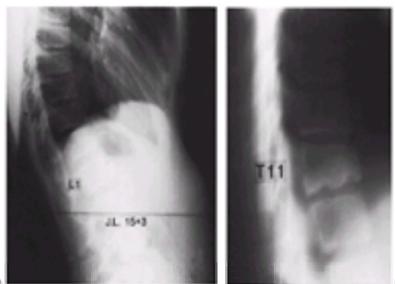
A rare type of spondylolisthesis, acute spondylolytic spondylolisthesis, has been described ( [58](#)). This injury is caused by high-energy trauma and is an unstable spinal

injury, similar to a fracture–dislocation, usually in the pars interarticularis of L5. The so-called traumatic spondylolisthesis (type IV) is caused by a fracture in areas of the bony hook other than the pars interarticularis, differentiating these two acute types of spondylolistheses. The deformity typically progresses, and some patients develop neurologic compromise. Because of their instability, high risk of progression, and possibility of developing neurologic compromise, these injuries should be treated operatively. Grade I deformities can be adequately treated with posterior arthrodesis; more severe deformities may require both anterior and posterior arthrodeses because of the greater disruption of secondary ligamentous restraints ( 58).

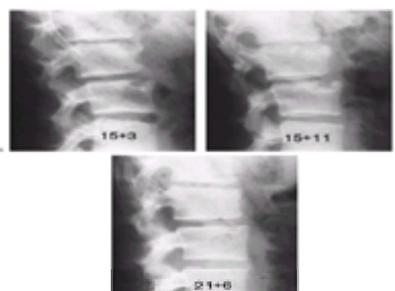
### Lumbar and Thoracolumbar Scheuermann's Disease

Scheuermann's disease is a common cause of thoracic kyphosis and is seldom painful. Children present with cosmetic concerns and are subsequently found to have the characteristic vertebral changes. Sorenson's radiographic criterion of three or more adjacent vertebrae wedged more than 5 degrees ( 125) confirms the diagnosis. End plate irregularity, Schmorl's node formation, and narrowing of the disk space are common but are not in themselves diagnostic. Thoracic Scheuermann's disease is typically limited to the thoracic vertebrae, spontaneous in onset, and due to hereditary influences. Lumbar or thoracolumbar osteochondritis is less common but is more often accompanied by pain ( 14,46,139). Several researchers suggest that the lumbar vertebral changes are the result of trauma ( 14,46,139). Lumbar or thoracolumbar Scheuermann's disease primarily affects adolescents (13–17 years of age) and is accompanied by a period of moderately severe pain with activity. Patients usually have a history of acute back strain or injury ( 14,46,87). Wassmann noted that the condition was eight times more common in "lads from the country" (139), and others have reported that the lumbar vertebral changes often are associated with hard physical labor before the age of 16, suggesting that the maturing end plate and vertebral body are more vulnerable to increased mechanical strain during this period of rapid growth ( 14,46,139). Hafner (51) coined the term *apprentice kyphosis*, or *kyphosis muscularis*, and he found that it occurred more commonly in boys (2:1) between the ages of 15 and 17 years, during the growth spurt. Micheli (90) found similar lumbar changes in young athletes and suggested that the cause was a localized stress injury to the vertebral physes.

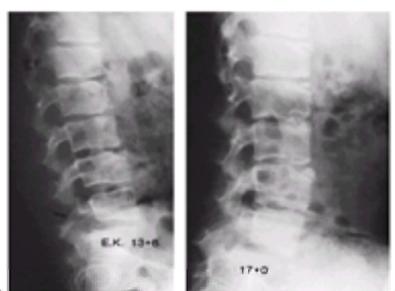
The apophyseal ossification centers first appear in the lower thoracic region at approximately 9 years of age and fuse with the vertebral bodies between the ages of 17 and 22 years. The ring is thinner in the middle than in the periphery. Increasing the pressure in the intervertebral disk forces it through the center of the end plate and into cancellous bone of the vertebral body ( Fig. 19-27) (67). The mechanism is analogous to the production of Schmorl's nodes and is accompanied by narrowing of the disk space (59,67,111). Similarly, metabolic and neoplastic diseases that lead to structural weakening of the bone are often associated with Schmorl's node formation (111). Marginal Schmorl's nodes are more often associated with trauma, and central nodes are more frequent and consistent with thoracic Scheuermann's disease (3). In experimental models, heavy lifting, especially when seated and bending forward, increased the intranuclear pressures to the lower end of the range necessary to produce fracture through a normal vertebral end plate ( Fig. 19-28) (67). Flexion–extension motion of the spine, as in rowers, weight lifters, and gymnasts, can produce these same forces ( Fig. 19-29) (90).



**FIGURE 19-27.** Radiographs of a 15-year-old boy with persistent back pain in the mid-lumbar region following a weight-lifting program. **A:** The routine lateral view suggests mild end plate changes. **B:** A lateral tomogram demonstrates Schmorl's node formation and end plate irregularity of T11. Subtle changes on routine films may belie significant end plate changes or disk protrusion into the vertebral body.

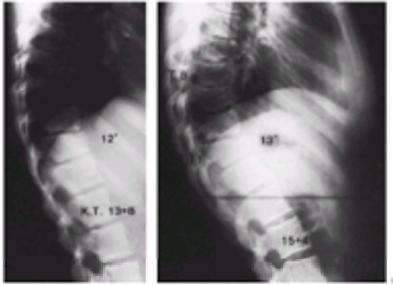


**FIGURE 19-28. A:** Acute herniated Schmorl's node in a 15-year-old weight lifter with back pain and nodes at L2, L3, and L4. **B:** Eight months later there has been continued growth of the vertebrae without progressive deformity. **C:** At 21 years of age the sites of the Schmorl's nodes are still seen, although they appear smaller. The minimal wedging at L2 has not changed in the preceding 6 years.



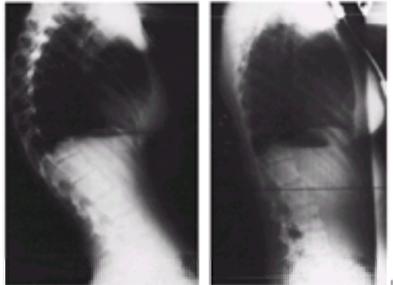
**FIGURE 19-29. A:** Radiographs of a 13-year-old boy who sustained vertebral end plate changes at several levels while playing football. Note the associated spondylolysis ( arrow) and mild spondylolisthesis. The patient's discomfort responded satisfactorily to bracing. **B:** The radiographs at age 17 years demonstrate the continued free fragment appearance of the vertebral apophysis at several levels and persistence of Schmorl's nodes.

Back pain may be present from 2 to 6 months and is increased with activity, accentuated by forward flexion, and relieved by rest ( 14,46). Conservative treatment such as bracing usually is sufficient (67,125). Occasionally, bed rest or plaster immobilization is required. There are no reports of children needing surgery for symptoms, and only a few have required correction for deformity. The vertebral changes progress slowly toward healing during the time of remaining growth. Schmorl's node formation and disk space narrowing generally persist ( Fig. 19-28 and Fig. 19-30) (87). The apophyseal fragment at the anterior margin of the vertebral body seldom heals.



**FIGURE 19-30. A:** Radiographs of a 13-year-old girl who sustained anterior compression of the L1 vertebra during gymnastics, with 12 degrees of wedging. She had persistent discomfort over several months that responded to bracing. **B:** At 15 years of age she was asymptomatic; there has been no remodeling of the deformity.

The kyphotic deformity may progress because of the anterior deformation of the vertebral bodies from the original injury or because of increased pressure on the anterior margin with cessation of growth in that region. The kyphosis generally is not severe and seldom requires any specific treatment. For severe deformity in a skeletally immature patient with kyphosis exceeding 50 to 60 degrees, a Milwaukee brace is recommended (Fig. 19-31). Surgery is rarely needed.



**FIGURE 19-31. Radiographs of a 15-year-old girl with persistent back pain and deformity following gymnastics. A:** Vertebral end plate changes, Schmorl's node, and gibbus at the thoracolumbar junction. **B:** Treatment with the Milwaukee brace improved alignment by reducing the deformity.

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## CHAPTER REFERENCES

1. Abel MS. Transverse posterior element fractures associated with torsion. *Skel Radio*. 1989;17:556–560.
2. Agran PF, Dunkle DE, Winn DG. Injuries to a sample of seatbelted children evaluated and treated in a hospital emergency room. *J Trauma* 1987;27:58–64.
3. Alexander CJ. Scheuermann's disease: a traumatic spondylodystrophy. *Skel Radio*. 1977;1:209–221.
4. Amstutz HC, Carey EJ. Skeletal manifestations and treatment of Gaucher's disease. *J Bone Joint Surg [Am]* 1966;48:670–701.
5. Anderson JM, Schutt AH. Spinal injury in children. A review of 156 cases seen from 1950 through 1978. *Mayo Clin Proc* 1980;55:499–504.
6. Anderson PA, Henley MB, Rivara FP, et al. Flexion distraction and Chance injuries to the thoracolumbar spine. *J Orthop Trauma* 1991;5:153–160.
7. Aufdermaur M. Spinal injuries in juveniles. Necropsy findings in twelve cases. *J Bone Joint Surg [Br]* 1974;56:513–519.
8. Baker DR, McHollick W. Spondyloschisis and spondylolisthesis in children. *J Bone Joint Surg [Am]* 1956;38:933–934.
9. Banniza von Bazan UK, Paeslack V. Scoliotic growth in children with acquired paraplegia. *Paraplegia* 1977–1978;15:65–73.
10. Begg AC. Nuclear herniations of the intervertebral disc. *J Bone Joint Surg [Br]* 1954;36:180–194.
11. Bell HJ, Dykstra DD. Somatosensory evoked potentials as an adjunct to diagnosis of neonatal spinal cord injury. *J Pediatr* 1985;106:298–301.
12. Bellah RD, Summerville DA, Treves ST, et al. Low-back pain in adolescent athletes: detection of stress injury to the pars interarticularis with SPECT. *Radiology* 1991;180:509–512.
13. Betz RR, Gelman AJ, DeFilipp GJ, et al. Magnetic resonance imaging (MRI) in the evaluation of spinal cord injured children and adolescents. *Paraplegia* 1987;25:92–99.
14. Blumenthal SL, Roach J, Herring JA. Lumbar Scheuermann's: a clinical series and classification. *Spine* 1987;12:929–932.
15. Boechat MI. Spinal deformities and pseudofractures. *AJR* 1987;148:97–98.
16. Bondurant FJ, Cotler HB, Kulkarni MV, et al. Acute spinal cord injury. A study using physical examination and magnetic resonance imaging. *Spine* 1990;15:161–168.
17. Borkow SE, Kleiger B. Spondylolisthesis in the newborn. *Clin Orthop Rel Res* 1971;82:73–76.
18. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury. Results of the second National Acute Spinal Cord Injury Study. *N Engl J Med* 1990;322:1405–1411.
19. Bracken MB, Shepard MJ, Holford TR, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the third National Acute Spinal Cord Injury randomized controlled trial. *JAMA* 1997;277:1597–1604.
20. Bradford DS, Iza J. Repair of the defect in spondylolysis or minimal degrees of spondylolisthesis by segmental wire fixation and bone grafting. *Spine* 1985;10:673–679.
21. Bradford DS, McBride CG. Surgical management of thoracolumbar spine fractures with incomplete neurologic deficits. *Clin Orthop Rel Res* 1987;218:201–216.
22. Callahan DJ, Pack LL, Bream RC, et al. Intervertebral disc impingement syndrome in a child. Report of a case and suggest pathology. *Spine* 1986;11:402–404.
23. Campbell J, Bonnett C. Spinal cord injury in children. *Clin Orthop Rel Res* 1975;112:114–123.
24. Carrion WV, Dormans JP, Drummond DS, et al. Circumferential growth plate fracture of the thoracolumbar spine from child abuse. *J Pediatr Orthop* 1996;16:210–214.
25. Chance GQ. Note on a type of flexion fracture of the spine. *Br J Radiol* 1948;21:452–453.
26. Choi JU, Hoffman HJ, Hendrick EB, et al. Traumatic infarction of the spinal cord in children. *J Neurosurg* 1986;65:608–610.
27. Cullen JC. Spinal lesions in battered babies. *J Bone Joint Surg [Br]* 1975;57:364–366.
28. Cybulski GR, Stone JL, Kant R. Outcome of laminectomy for civilian gunshot injuries of the terminal spinal cord and cauda equina: review of 88 cases. *Neurosurgery* 1989;24:392–397.
29. Cyron BM, Hutton WC. Variations in the amount and distribution of cortical bone across the pars interarticularis of L5. *Spine* 1979;4:163–167.
30. DeOrto JK, Bianco AJ Jr. Lumbar disc excision in children and adolescents. *J Bone Joint Surg [Am]* 1982;64:991–996.
31. Devas MB. Stress fractures in children. *J Bone Joint Surg [Br]* 1963;45:528–541.
32. Diamond P, Hansen CM, Christofersen MR. Child abuse presenting as a thoracolumbar spinal fracture dislocation: a case report. *Pediatr Emerg Care* 1994;10:83–86.
33. Dickman CA, Rekeate HL, Sonntag VKH, et al. Pediatric spinal trauma: vertebral column and spinal cord injuries in children. *Pediatr Neurosci* 1989;15:237–256.
34. Dickson RA, Leatherman KD. Spinal injuries in child abuse: case report. *J Trauma* 1978;18:811–812.
35. Dietemann JL, Runge M, Badoz A, et al. Radiology of posterior lumbar apophyseal ring fractures: report of 13 cases. *Neuroradiology* 1988;30:337–344.
36. Eingorn D, Pizzutillo PD. Pars interarticularis fusion of multiple levels of lumbar spondylolysis. *Spine* 1985;10:250–252.
37. Epstein NE, Epstein JA. Limbus lumbar vertebral fractures in 27 adolescents and adults. *Spine* 1991;16:962–966.
38. Ferrandez L, Usabiaga J, Curto JM, et al. Atypical multivertebral fracture due to hyperextension in an adolescent girl. *Spine* 1989;14:645–646.
39. Fredrickson BE, Baker D, McHollick WJ, et al. The natural history of spondylolysis and spondylolisthesis. *J Bone Joint Surg [Am]* 1984;66:699–707.
40. Freiberg AA, Graziano GP, Loder RT, et al. Metastatic vertebral disease in children. *J Pediatr Orthop* 1993;13:148–153.
41. Galandiuk S, Raque G, Appel S, et al. The two-edged sword of large-dose steroids for spinal cord trauma. *Ann Surg* 1993;218:419–427.
42. Gellad FE, Levine AM, Joslyn JN, et al. Pure thoracolumbar facet dislocation: clinical features and CT appearance. *Radiology* 1986;161:505–508.

43. Glasauer FE, Cares HL. Biomechanical features of traumatic paraplegia in infancy. *J Trauma* 1973;13:166–170.
44. Glassman SD, Johnson JR, Holt RT. Seatbelt injuries in children. *J Trauma* 1992;33:882–886.
45. Grabb PA, Pang D. Magnetic resonance imaging in the evaluation of spinal cord injury without radiographic abnormality in children. *Neurosurgery* 1994;35:406–414.
46. Greene TL, Hensinger RN, Hunter YL. Back pain and vertebral changes simulating Scheuermann's disease. *J Pediatr Orthop* 1985;5:1–7.
47. Grier D, Wardell S, Sarwark J, et al. Fatigue fractures of the sacrum in children: two case reports and a review of the literature. *Skel Radio*. 1993;22:515–518.
48. Gumley G, Taylor TKF, Ryan MD. Distraction fractures of the lumbar spine. *J Bone Joint Surg [Br]* 1982;64:520–525.
49. Hadley MN, Zabramski JM, Browner CM, et al. Pediatric spinal trauma. Review of 122 cases of spinal cord and vertebral column injuries. *J Neurosurg* 1988;68:18–24.
50. Haffner DL, Hoffer MM, Wiedbusch R. Etiology of children's spinal injuries at Rancho Los Amigos. *Spine* 1993;18:679–684.
51. Hafner RVH. Localized osteochondritis (Scheuermann's disease). *J Bone Joint Surg [Br]* 1952;34:38–40.
52. Hashimoto T, Kaneda K, Abumi K. Relationship between traumatic spinal canal stenosis and neurologic deficits in thoracolumbar burst fractures. *Spine* 1988;13:1268–1272.
53. Hegenbarth R, Ebel KD. Roentgen findings in fractures of the vertebral column in childhood: examination of 35 patients and its results. *Pediatr Radio*. 1976;5:34–39.
54. Heiden JS, Weiss MH, Rosenberg AW, et al. Penetrating gunshot wounds of the cervical spine in civilians. Review of 38 cases. *J Neurosurg* 1975;42:575–579.
55. Hensinger RN. Spondylolysis and spondylolisthesis in children. *Instruct Course Lect AAOS* 1983;32:132–150.
56. Hensinger RN, Lang JR, MacEwen GD. Surgical management of spondylolisthesis in children and adolescents. *Spine* 1976;1:207–216.
57. Herkowitz HN, Samberg C. Vertebral column injuries associated with tobogganing. *J Trauma* 1978;18:806–810.
58. Hilibrand AS, Urquhart AG, Graziano GP, et al. Acute spondylolytic spondylolisthesis: risk for progression and neurologic complications. *J Bone Joint Surg [Am]* 1995;77:190–196.
59. Hilton RC, Ball J, Benn RT. Vertebral end-plate lesions (Schmorl's nodes) in the dorsolumbar spine. *Ann Rheum Dis* 1976;35:127–132.
60. Hitchcock HH. Spondylolisthesis: observations on its development, progression, and genesis. *J Bone Joint Surg* 1940;22:1–16.
61. Hoffman MA, Spence LJ, Wesson DE, et al. The pediatric passenger: trends in seatbelt use and injury patterns. *J Trauma* 1987;27:974–976.
62. Horal J, Nachemson A, Scheller S. Clinical and radiological long term follow-up of vertebral fractures in children. *Acta Orthop Scand* 1972;43:491–503.
63. Hubbard DD. Injuries of the spine in children and adolescents. *Clin Orthop Rel Res* 1974;100:56–65.
64. Jackson DW, Wiltse LL, Cirincione RJ. Spondylolysis in the female gymnast. *Clin Orthop Rel Res* 1976;117:68–73.
65. Jackson RW. Surgical stabilization of the spine. *Paraplegia* 1975;13:71–74.
66. Jarvis JG. Seat belt fractures. In: Letts RM, ed. *Management of pediatric fractures*. New York: Churchill Livingstone, 1994;887.
67. Jayson MIV, Herbert CM, Barks SJ. Intervertebral discs: nuclear morphology and bursting pressures. *Ann Rheum Dis* 1973;32:308–315.
68. Jones ET, Hensinger RN. Spinal deformity in idiopathic juvenile osteoporosis. *Spine* 1981;6:1–4.
69. Kerlake RW, Jaspan T, Worthington BS. Magnetic resonance imaging of spinal trauma. *Br J Radio*. 1991;64:386–402.
70. Kewalramani LS, Krause JF, Sterling HM. Acute spinal-cord lesions in a pediatric population: epidemiological and clinical features. *Paraplegia* 1980;18:206–219.
71. Kewalramani LS, Tori JA. Spinal cord trauma in children. Neurologic patterns, radiologic features, and pathomechanics of injury. *Spine* 1980;5:11–18.
72. Kilfoyle RM, Foley JJ, Norton PL. Spine and pelvic deformity in childhood and adolescent paraplegia. A study of 104 cases. *J Bone Joint Surg [Am]* 1965;47:659–682.
73. Kleinman PK, Marks SC. Vertebral body fractures in child abuse: radiologic–histopathologic correlates. *Invest Radio*. 1992;27:715–722.
74. Koch BM, Eng GM. Neonatal spinal cord injury. *Arch Phys Med Rehabil* 1979;60:378–381.
75. Krenz J, Troup JDG. The structure of the pars interarticularis of the lower lumbar vertebrae and its relation to the etiology of spondylolysis. *J Bone Joint Surg [Br]* 1973;55:735–741.
76. Lancourt JE, Dickson JH, Carter RE. Paralytic spinal deformity following traumatic spinal-cord injury in children and adolescents. *J Bone Joint Surg [Am]* 1981;63:47–53.
77. Letts M, Smallman T, Afanasiev R, et al. Fracture of the pars interarticularis in adolescent athletes: a clinical-biomechanical analysis. *J Pediatr Orthop* 1986;6:40–46.
78. Libson E, Bloom RA, Dinari G, et al. Oblique lumbar spine radiographs: importance in young patients. *Radiology* 1984;151:89–90.
79. Libson E, Bloom RA, Shapiro Y. Scoliosis in young men with spondylolysis or spondylolisthesis. *Spine* 1984;9:445–447.
80. Lim LH, Lam LK, Moore MH, et al. Associated injuries in facial fractures: review of 839 patients. *Br J Plast Surg* 1993;46:635–638.
81. Limb D, Shaw DL, Dickson RA. Neurological injury in thoracolumbar burst fractures. *J Bone Joint Surg [Br]* 1995;77:774–777.
82. Linsens WHJP, Praamstra P, Babreels FJM, et al. Vascular insufficiency of the cervical cord due to hyperextension of the spine. *Pediatr Neuro* 1990;6:123–125.
83. Lyons BM, Brown DJ, Calvert JM, et al. The diagnosis and management of post-traumatic syringomyelia. *Paraplegia* 1987;25:340–350.
84. Maccartee CC Jr, Griffin PO, Byrd EB. Ruptured calcified thoracic disc in a child. *J Bone Joint Surg [Am]* 1972;54:1272–1274.
85. Mandell GA, Harcke HT. Scintigraphy of spinal disorders in adolescents. *Skel Radio*. 1993;22:393–401.
86. Mayfield JK, Erkkila JC, Winter RB. Spine deformity subsequent to acquired childhood spinal cord injury. *J Bone Joint Surg [Am]* 1981;63:1401–1411.
87. McCall IW, Park WM, O'Brien JP, et al. Acute traumatic intraosseous disc herniation. *Spine* 1985;10:134–137.
88. McPhee IB. Spinal fractures and dislocations in children and adolescents. *Spine* 1981;6:533–537.
89. Meehan PL, Viroslav S, Schmitt Jr EW. Vertebral collapse in childhood leukemia. *J Pediatr Orthop* 1995;15:592–595.
90. Micheli LJ. Low back pain in the adolescent: differential diagnosis. *Am J Sports Med* 1979;7:362–364.
91. Miller JA, Smith TH. Seatbelt induced Chance fracture in an infant. Case report and literature review. *Pediatr Radiol* 1991;21:575–577.
92. Moreton RD. So-called normal backs. *Indust Med Surg* 1969;38:216–219.
93. Morita T, Ikata T, Katoh S, et al. Lumbar spondylolysis in children and adolescents. *J Bone Joint Surg [Br]* 1995;77:620–625.
94. Muschik M, Hähnel H, Robinson PN, et al. Competitive sports and progression of spondylolisthesis. *J Pediatr Orthop* 1996;16:364–369.
95. Nachemson A. Repair of the spondylolisthesis defect and intertransverse fusion for young patients. *Clin Orthop Rel Res* 1976;117:101–105.
96. Nesbit ME, Kieffer S, D'Angio GJ. Reconstruction of vertebral height in Histiocytosis X: a long-term follow-up. *J Bone Joint Surg [Am]* 1969;51:1360–1368.
97. Nicol RO, Scott JHS. Lytic spondylolysis: repair by wiring. *Spine* 1986;11:1027–1030.
98. Ogilvie JW, Sherman J. Spondylolysis in Scheuermann's disease. *Spine* 1987;12:251–253.
99. Pang D, Pollack IF. Spinal cord injury without radiographic abnormality in children—the SCIWORA syndrome. *J Trauma* 1989;1989:654–664.
100. Pang D, Wilberger JE Jr. Spinal cord injury without radiographic abnormalities in children. *J Neurosurg* 1982;57:114–129.
101. Papanicolaou N, Wilkinson RH, Emans JB, et al. Bone scintigraphy and radiography in young athletes with low back pain. *AJR* 1985;145:1039–1044.
102. Paulson JA. The epidemiology of injuries in adolescents. *Pediatr Ann* 1988;17:84–96.
103. Peck FC. A calcified thoracic intervertebral disk with herniation and spinal cord compression in a child. *J Neurosurg* 1957;14:105–109.
104. Pedersen AK, Hagen R. Spondylolysis and spondylolisthesis: treatment by internal fixation and bone grafting the defect. *J Bone Joint Surg [Am]* 1988;70:15–24.
105. Phalen GS, Dickson JA. Spondylolisthesis and tight hamstrings. *J Bone Joint Surg [Am]* 1961;43:505–512.
106. Poulouen JC, Kassis B, Glorion C, et al. Vertebral growth after thoracic or lumbar fracture of the spine in children. *J Pediatr Orthop* 1997;17:115–120.
107. Prendergast MR, Saxe JM, Ledgerwood AM, et al. Massive steroids do not reduce the zone of injury after penetrating spinal cord injury. *J Trauma* 1994;37:576–580.
108. Rebeiro RC, Pui CH, Schell MJ. Vertebral compression fracture as a presenting feature of acute lymphoblastic leukemia in children. *Cancer* 1988;61:589–592.
109. Reid RB, Letts RM, Black GB. Pediatric Chance fractures: association with intra-abdominal injuries and seatbelt use. *J Trauma* 1990;30:384–391.
110. Renard M, Tridon P, Kuhnast M, et al. Three unusual cases of spinal cord injury in childhood. *Paraplegia* 1979;16:130–134.
111. Resnick D, Niwayama G. Intravertebral disc herniations: cartilaginous (Schmorl's) nodes. *Radiology* 1978;126:57–65.
112. Roaf R. A study of the mechanics of spinal injuries. *J Bone Joint Surg [Br]* 1969;42:810–823.
113. Rodger RM, Missiuna P, Ein S. Entrapment of bowel within a spinal fracture. *J Pediatr Orthop* 1991;11:783–785.
114. Rose J, Gamble J, Schultz A, et al. Back pain and spinal deformity in cystic fibrosis. *Am J Dis Child* 1987;141:1313–1316.
115. Rothfus WE, Goldberg AL, Deeb ZL, Daffner RH. MR recognition of posterior lumbar vertebral ring fracture. *J Comput Assist Tomogr* 1990;14:790–794.
116. Rothman SLG. Computed tomography of the spine in older children and teenagers. *Clin Sports Med* 1986;2:247–270.
117. Ruge JR, Sinson GP, McLone DG, et al. Pediatric spinal injury: the very young. *J Neurosurg* 1988;68:25–30.
118. Rumball K, Jarvis J. Seat-belt injuries of the spine in young children. *J Bone Joint Surg [Br]* 1992;74:571–574.
119. Sclafani SJA, Florence LO, Phillips TF, et al. Lumbar arterial injury: radiologic diagnosis and management. *Radiology* 1987;165:709–714.
120. Sherman FC, Rosenthal RK, Hall JE. Spine fusion for spondylolysis and spondylolisthesis in children. *Spine* 1979;4:59–67.
121. Shipley JA, Beukes CA. The nature of the spondylolytic defect. *J Bone Joint Surg [Br]* 1998;80:662–664.
122. Sivit CJ, Taylor GA, Newman KD, et al. Safety-belt injuries in children with lap-belt ecchymosis: CT findings in 61 patients. *AJR* 1991;157:111–114.
123. Smith WS, Kaufer H. Patterns and mechanisms of lumbar injuries associated with lap seat belts. *J Bone Joint Surg [Am]* 1969;51:239–254.
124. Sneed RC, Stover SL, Fine PR. Spinal cord injury associated with all-terrain vehicle accidents. *Pediatrics* 1986;77:271–274.
125. Sorensen KH. *Scheuermann's juvenile kyphosis. Clinical appearances, radiography, aetiology, and prognosis*. Copenhagen: Munksgaard, 1964.
126. Sovio OM, Bell HM, Beauchamp RD, et al. Fracture of the lumbar vertebral apophysis. *J Pediatr Orthop* 1985;5:550–552.
127. Stauffer ES, Wood RW, Kelly EG. Gunshot wounds of the spine: the effects of laminectomy. *J Bone Joint Surg [Am]* 1979;61:389–392.
128. Steiner ME, Micheli LJ. Treatment of symptomatic spondylolysis and spondylolisthesis with the modified Boston brace system. *Spine* 1985;10:937–943.
129. Sturm JT, Hines JT, Perry JF Jr. Thoracic spinal fractures and aortic rupture: a significant and fatal association. *Ann Thorac Surg* 1990;50:931–933.
130. Sturm JT, Perry JF Jr. Injuries associated with fractures of the transverse processes of the thoracic and lumbar vertebrae. *J Trauma* 1984;24:597–599.
131. Swischuk LE. Spine and spinal cord trauma in the battered child syndrome. *Radiology* 1969;92:733–738.
132. Takata K, Inoue S-I, Takahashi K, et al. Fracture of the posterior margin of a lumbar vertebral body. *J Bone Joint Surg [Am]* 1988;70:589–594.
133. Tarr RW, Drolshagen LF, Kerner TC, et al. MR imaging of recent spinal trauma. *J Comput Assist Tomogr* 1987;11:412–417.
134. Taylor DA, Eggli KD. Lap-belt injuries of the lumbar spine in children: a pitfall in CT diagnosis. *AJR* 1988;150:1355–1358.
135. Turner RH, Bianco AJ Jr. Spondylolysis and spondylolisthesis in children and teenagers. *J Bone Joint Surg [Am]* 1971;53:1298–1306.

136. van den Oever M, Merrick MV, Scott JHS. Bone scintigraphy in symptomatic spondylolysis. *J Bone Joint Surg [Br]* 1987;69:453–456.
137. Varonos S, Ansell BM, Reeve J. Vertebral collapse in juvenile chronic arthritis: its relationship with glucocorticoid therapy. *Calcif Tissue Int* 1987;41:75–78.
138. Wagoner G, Pendergrass EP. The anterior and posterior “notch” shadows seen in lateral roentgenograms of the vertebrae in infants: an anatomic explanation. *AJR* 1939;42:663–670.
139. Wassmann K. Kyphosis juvenilis Scheuermann—an occupational disorder. *Acta Orthop Scand* 1951;21:65–74.
140. Waters RL, Adkins RH. The effects of removal of bullet fragments retained in the spinal canal. A collaborative study by the National Spinal Cord Injury Model Systems. *Spine* 1991;16:934–939.
141. Williams B, Terry AF, Jones HWF, et al. Syringomyelia as a sequel to traumatic paraplegia. *Paraplegia* 1981;19:67–80.
142. Wiltse LL, Newman PH, Macnab I. Classification of spondylolysis and spondylolisthesis. *Clin Orthop Rel Res* 1976;117:23–29.
143. Wiltse LL, Widell EH Jr, Jackson DW. Fatigue fracture: the basic lesion in isthmic spondylolisthesis. *J Bone Joint Surg [Am]* 1975;57:17–22.
144. Woelfel GF, Moore EE, Cogbill TG, et al. Severe thoracic and abdominal injuries associated with lap-harness seatbelts. *J Trauma* 1984;24:166–167.
145. Wynne-Davies R. Inheritance and spondylolisthesis. A radiographic family study. *J Bone Joint Surg [Br]* 1979;61:301–305.
146. Yasuoka S, Peterson HA, MacCarty CS. Incidence of spinal column deformity after multilevel laminectomy in children and adults. *J Neurosurg* 1982;57:441–445.
147. Yngve DA, Harris WP, Herndon WH, et al. Spinal cord injury without osseous spine fracture. *J Pediatr Orthop* 1988;8:153–159.

## 20 FRACTURES OF THE PELVIS

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Pelvic fractures constitute only 1% to 3% of all fractures in children. The most important aspect of treatment of these fractures is the realization that such a fracture is an indication that significant trauma has occurred and other injuries to neurovascular structures, abdominal viscera, the genitourinary system, and the musculoskeletal system also may have occurred. Fractures of the pelvis and spine are associated with the longest hospital stays, the most admissions to the intensive care unit, and the highest rates of mortality in patients with multiple injuries (9,60). When a fracture of a child's pelvis is seen on an initial radiograph, it should be an indication that associated life-threatening soft tissue injuries may be present, and the treatment of these injuries should take priority over management of the fracture. Mortality rates in children with pelvic fractures have been reported to be as high as 25% (49), but more recent series report mortality rates of 2% to 12% (18,27,33,52,61,63). In 1,254 patients with pelvic fractures, Ismail et al. (27) reported an overall mortality rate of 5% for children and 17% for adults. In children, fracture-related exsanguination was the cause of death in only 0.3%, whereas 3.4% of deaths in adults were due to pelvic fracture exsanguination.

### DIAGNOSIS

#### Mechanisms of Injury

Most pelvic fractures in children are caused by vehicular accidents. From Rang's early reports (50) to more recent series (3,4,5,14,27,30,33,40,52,54,61), 70% to 90% of reported pelvic fractures in children were caused by traffic accidents. Most often the injured child is a pedestrian struck by a car. Avulsion injuries usually occur in athletic activities, especially gymnastics, track, and soccer. Child abuse is a rare etiology (34,44).

#### Signs and Symptoms

The evaluation of a child with a suspected pelvic fracture begins with a thorough history and physical examination. Often a child with a pelvic fracture has concomitant multiple injuries about the head, chest, abdomen, and genitourinary tract, and these other injuries take precedence concerning operative intervention to stabilize the child's condition. The pelvic fracture itself often has low priority in the critical care of a child with polytrauma.

The examination of the pelvic area begins with a visual inspection. Areas of contusion, abrasion, laceration, ecchymosis, or hematoma, especially in the perineal and pelvic areas, should be recorded.

Landmarks such as the anterior superior iliac spine, crest of the ilium, sacroiliac joints, and symphysis pubis should be palpated. Exerting posterior pressure on the anterior superior iliac crest produces pain at the fracture site as the pelvic ring is opened. Compressing the pelvic ring at the iliac crest from the lateral to the medial direction also causes pain, and crepitation may be felt if a pelvic fracture is present. Pressure downward on the symphysis pubis and posteriorly on the sacroiliac joints causes pain and motion if there is a fracture in the pelvic ring. The range of motion of the extremities, especially of the hip joint, should be determined. Occasionally, pain in the inguinal area is noted during flexion and extension of the hip if there is a pelvic fracture.

#### Associated Injuries

Because most pelvic fractures in children result from high-velocity trauma, injuries other than the pelvic fracture usually are present. Of the 43 children with pelvic fractures reported by Blasier et al. (3), 25 (58%) had associated injuries, most often multiple fractures, closed head injuries, and spine injuries. The incidence of concomitant injuries increases with the severity of the pelvic fracture. Bond et al. (5) noted that the location and number of pelvic fractures were strongly associated with probability of abdominal injury: 1% for isolated pubic fractures, 15% for iliac or sacral fractures, and 60% for multiple fractures of the pelvic ring. Poole et al. (47) suggested that the outcomes of patients with pelvic fractures were determined by the associated injuries rather than by the pelvic fracture.

As in adults, retroperitoneal hemorrhage is the most serious life-threatening complication in children with unstable pelvic ring disruptions. Marked cephalad displacement of the hemipelvis can injure the superior and inferior gluteal arteries at the sciatic notch. Injury of the iliac or femoral artery is uncommon except with open fractures involving the anterior pelvic ring (60). McIntyre et al. (33) correlated the risk of life-threatening hemorrhage to pelvic fracture complexity. Patients with bilateral anterior and posterior fractures required transfusions significantly more often than those with other types of fractures. Ultrasonography or peritoneal lavage help rule out intraabdominal injury.

Hematuria has been reported in from 34% to 57% of children with pelvic fractures, usually without any significant urinary tract injury. When hematuria is accompanied by an inability to void, blood at the urethral meatus, or abnormality of the prostate, genitourinary consultation should be obtained. Most urologic injuries occur with fractures of the anterior pelvic ring (60). The reported incidence of genitourinary injuries (bladder or urethral rupture, vaginal or scrotal tear, kidney injury) averages approximately 5%.

Children with pelvic fractures are more likely to have head injuries than are adults with pelvic fractures. Rieger and Brug ( 52) reported head injuries ranging from mild concussion to brain death in 48% of their 54 patients, and other series have reported closed head injuries in approximately 30% of patients ( 30,33,61). Although injury to the brain has a higher priority and needs more immediate attention than fractures in a child with polytrauma, inadequately treated pelvic fractures can result in permanent disability and should be treated with expectation of full neurologic recovery.

Pelvic fractures with posterior displacement of the hemipelvis or iliac wing can damage the lumbosacral plexus, as well as the sciatic, femoral, and obturator nerves. Neurologic examination of the lower extremities should be routine, and documentation of any neurologic deficit is essential.

Fractures of other bones are present in approximately half of children with pelvic fractures ( 30,33,52,54,61). The most frequently fractured bones are the femur, tibia, and fibula. Vazquez and Garcia (63), in a study of 79 children with pelvic fractures, found that the presence of any additional fracture was a significant indication that head or abdominal injury also was present and that transfusion would be required in the first 24 hours after injury. Death, thoracic injuries, and patients requiring laparotomy or an additional nonorthopaedic procedure were twice as frequent in the group with additional fractures as in the group with pelvic fractures only. Vazquez and Garcia (63) suggested that this easily identifiable risk factor can help identify patients who may benefit from early transfer to a regional pediatric trauma center.

### Radiographic Studies and Other Imaging

All too often in an emergency situation, the child's well-being is jeopardized in one of the least desirable emergency areas of the hospital, the radiology department. Radiographs should be obtained only after the patient is stabilized. Rarely do radiographs on an emergency basis help the physician stabilize the child. Once the patient is clinically stable, only pertinent radiographs should be ordered by the physician in charge. If special views are necessary, then the physician ordering these films should be in attendance. Scout views of the skull, chest, abdomen, pelvis, and long bones should be procured quickly.

In a child with a pelvic fracture, unless there is a significant fracture–dislocation, multiple radiographic views can be deferred. When the child is stable, multiple views of the pelvis should be taken (29). With the patient supine, the pelvis has a normal tilt of 25 degrees posteriorly. Thus, taking only an anteroposterior film of the pelvis gives, in reality, an oblique projection, which, as in radiographs of other long bones, is not the most desirable view. As an aid to diagnosis, a 25-degree caudad (inlet) view reveals the amount of internal or external rotation deformity or the amount of displacement. Also useful in determining the amount of rotation in the anteroposterior plane is the 35-degree cephalad (outlet) view. The pelvis can be tilted into internal and external rotation to determine fractures of the acetabulum and pelvic outlet. A description of these views recommended by Judet is given in Chapter 35 of *Fractures in Adults* (Volume 2 of this series). Comparison views of the contralateral apophysis may be helpful in evaluating avulsion fractures.

Computed tomography (CT) scanning helps determine the presence of fractures and any disruption or incongruity of the sacroiliac joint, sacrum, or acetabulum. Newer, rapid CT scanners can obtain a full pelvic scan in the initial evaluation. Three-dimensional CT images show more clearly superior or inferior displacement of the hemipelvis if a pelvic ring injury is present and help define the degree and direction of any acetabular displacement or rotational deformity ( 20,35). Magnetic resonance imaging (MRI) has the same benefits and also can delineate soft tissue injuries in the pelvis ( 35). Occasionally, a radioisotopic bone scan is useful for evaluating nondisplaced fractures and the rare stress fracture ( 58,59).

### CLASSIFICATION

Quinby (49) and Rang (50) classified pelvic fractures in children into three categories: uncomplicated fractures, fractures with visceral injuries requiring surgical exploration, and fractures associated with immediate, massive hemorrhage. The prognostic importance of this classification is apparent, and it is useful with regard to the patient's ultimate outcome; however, its emphasis, as it should be in an emergency situation, is on associated soft tissue injuries rather than on the pelvic fracture itself. Watts (64) classified pediatric pelvic fractures according to the severity of skeletal injury: (a) avulsion, such as epiphysiolysis (caused by violent muscular activity); (b) fracture of the pelvic ring (crushing injury), stable or unstable; and (c) acetabular fracture (associated with hip dislocation). Torode and Zieg ( 61), in a retrospective analysis of 141 children with pelvic fractures, also classified the injuries based on the severity of the fracture; their classification does not include acetabular fractures (Fig. 20-1, Table 20-1). Tile (59) modified Pennal's classification of pelvic fractures in adults to include three types based on mechanism of injury: anteroposterior compression, lateral compression, and vertical shear ( Table 20-2). This classification system has been incorporated into the Orthopaedic Trauma Association classification, which also consists of three main types: A, lesion sparing (or with no displacement of) posterior arch; B, incomplete disruption of posterior arch, partially stable; and C, complete disruption of posterior arch, unstable ( Table 20-3). Numerous subtypes also are included. This detailed system is described in the chapter on pelvic fractures in adults in *Fractures in Adults* (Volume 2, Chapter 35). Silber and Flynn (55) reviewed radiographs of 133 children and adolescents with pelvic fractures and classified them into two groups: immature (all physes open) and mature (closed triradiate cartilage). They suggested that in the immature group, management should focus primarily on associated injuries, and in the mature group adult pelvic fracture classifications and principles are appropriate.

- I. Avulsion fractures
- II. Iliac wing fractures
  - IIa. Separation of the iliac apophysis
  - IIb. Fracture of the bony iliac wing
- III. Simple ring fractures
  - IIIa. Fractures of the pubis and disruption of the pubic symphysis; the posterior structures remain stable
  - IIIb. Fractures involving the acetabulum, without a concomitant ring fracture
- IV. Fractures producing an unstable segment (ring disruption fracture)
  - IVa. "Straddle" fractures, characterized by bilateral inferior and superior pubic rami fractures
  - IVb. Fractures involving the anterior pubic rami or pubic symphysis and the posterior elements (e.g., sacroiliac joint, sacral ala)
  - IVc. Fractures that create an unstable segment between the anterior ring of the pelvis and the acetabulum

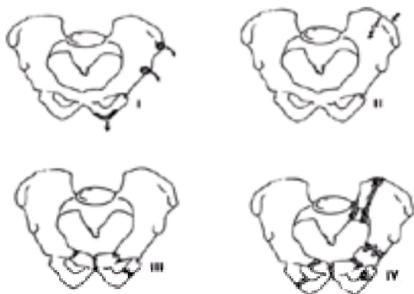
TABLE 20-1. TORODE AND ZIEG CLASSIFICATION OF PELVIC FRACTURES IN CHILDREN

- A. Stable fractures
  - A1: Avulsion fractures
  - A2: Undisplaced pelvic ring or iliac wing fractures
  - A3: Transverse fractures of the sacrum and coccyx
- B. Partially unstable fractures
  - B1: Open-book injuries
  - B2: Lateral compression injuries (includes triradiate injury)
  - B3: Bilateral type B injuries
- C. Unstable fractures of the pelvic ring
  - C1: Unilateral fractures
    - C1-1: Fractures of the ilium
    - C1-2: Dislocation or fracture–dislocation of the sacroiliac joint
    - C1-3: Fractures of the sacrum
  - C2: Bilateral fractures, one type B and one type C
  - C3: Bilateral type C fractures

TABLE 20-2. TILE AND PENNAL CLASSIFICATION OF PELVIC FRACTURES

- A. Stable fractures
- B. Rotationally unstable fractures, vertically stable
- C. Rotationally and vertically unstable fractures
  - C1.1 Iliac fracture
  - C1.2 Sacroiliac fracture–dislocation
  - C1.3 Sacral fracture
  - C3 Associated with an acetabular fracture
- Isolated acetabular fractures

**TABLE 20-3. AO/ASIF CLASSIFICATION OF PELVIC FRACTURES**



**FIGURE 20-1.** Torode and Zieg classification of pelvic fractures in children. Type I, avulsion fractures; type II, iliac wing fractures; type III, simple ring fractures; type IV, ring disruption fractures.

This multitude of classification systems makes comparison of incidence, results, and complications difficult among studies using different systems. Many recent studies of children's pelvic fractures in the literature use the Torode and Zieg (61) or Tile (2,59) classifications, or both, but the most basic classification—stable or unstable fracture—in general is the most useful information for making treatment decisions. Most pelvic fractures in children are stable injuries.

### APPLIED ANATOMY

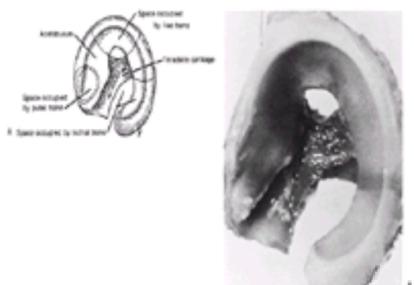
There are several important anatomic differences between the pelvis of a child and that of an adult (Table 20-4). First, a child's pelvis is more malleable because of the nature of the bone itself, the increased elasticity of the joints, and the ability of the more cartilaginous structures to absorb energy (43). Second, the elasticity of the joints may allow significant displacement and resultant fracture in only one area rather than the traditional concept of a mandatory “double break” in the ring for a displaced fracture (43,50). Third, avulsion fractures of an apophysis occur more often in children and adolescents than in adults because of the inherent weakness of cartilage compared with bone; fractures of the acetabulum into the triradiate cartilage also occur more often for the same reason (50,64). Fourth, fractures through physeal cartilage in children can ultimately cause growth arrest, leg-length discrepancy, and faulty development (e.g., a fracture through the triradiate cartilage with resultant “bony bar” formation and ultimately a deficient acetabulum) (1,21).

	Individual	Single Break	Double Break	Acetabulum
<b>Completed Case Series (204 Patients)<sup>a</sup></b>				
Avulsion (13.4%)	Two isolated sites: 1.2%	Isolated: 2%	Small fragment: 0.7%	
Pubis or ischium: 11.6%	1.2%	Wings: 1.2%	Linear fractures (stable): 6%	
Wing of ilium: 10%	Symphysis pubis: 2%	Joints, multiple: 0.7%	Linear fractures (unstable): 6	
Sacrospinous ligament: 1.3%	Sacroiliac joint: 0.7%	0.7%	Secondary to central dislocation: 5%	
<b>Total: 48.3%</b>	<b>Total: 11.8%</b>	<b>Total: 11.3%</b>	<b>Total: 6.7%</b>	
<b>Comparison with Other Series</b>				
<b>Seale</b>				
Danzon (115 patients)	30% (stable)	30% (unstable)	Not included	
Arthur (188 patients)	10%	20%	24%	
Reed (84 patients)	40.7%	1.2%	2%	
McE. Casson, Fother (204 patients)	24.5%	18.1%	11.5%	1.8% (17.2% acetabulum and joint)

**TABLE 20-4. DISTRIBUTION OF PELVIC FRACTURES IN CHILDREN**

### Ossification Centers

The pelvis in a child consists of three primary ossification centers: the ilium, ischium, and pubis. The three centers meet at the triradiate cartilage and fuse at approximately 16 to 18 years of age (43) (Fig. 20-2). The pubis and ischium fuse inferiorly at the pubic rami at 6 or 7 years of age. Occasionally, at approximately the time of fusion of the ischium to the pubis, an asymptomatic mass, the ischiopubic synchondrosis, is noted radiographically in this area. The child should be treated expectantly, and this should not be confused with a fracture of the pelvis.



**FIGURE 20-2. A:** Triradiate–acetabular cartilage complex viewed from the lateral side, showing the sites occupied by the iliac, ischial, and pubic bones. **B:** Normal acetabular cartilage complex of a 1-day-old infant. The ilium, ischium, and pubis have been removed with a curet. The lateral view shows the cup-shaped acetabulum. (From Ponseti IV. Growth and development of the acetabulum in the normal child. *J Bone Joint Surg Am* 1978;60:575–585; with permission.)

The secondary centers of ossification include the iliac crest, ischial apophysis, anterior inferior iliac spine, pubic tubercle, angle of the pubis, ischial spine, and lateral

wing of the sacrum. The iliac crest is first seen at 13 to 15 years and fuses at 15 to 17 years of age. The secondary ossification of the ischium is first seen at 15 to 17 years and fuses at 19 years of age, although fusion may be as late as 25 years of age. A center of ossification may be present at the anterior inferior iliac spine at approximately 14 years, fusing at 16 years of age (43,64). These secondary centers of ossification and the age of appearance and fusion are described so they will not be confused with avulsion fractures.

The acetabulum contains the physes of the ilium, ischium, and pubis that merge to become the triradiate cartilage. Interstitial growth in the triradiate part of the cartilage complex causes the acetabulum to expand during growth and causes the pubis, ischium, and ilium to enlarge as well. The concavity of the acetabulum develops in response to the presence of a spherical femoral head. The depth of the acetabulum increases during development as the result of interstitial growth in the acetabular cartilage, of appositional growth of the periphery of this cartilage, and of periosteal new bone formation at the acetabular margin (48). At puberty, three secondary centers of ossification appear in the hyaline cartilage surrounding the acetabular cavity. The *os acetabuli*, which is the epiphysis of the pubis, forms the anterior wall of the acetabulum. The epiphysis of the ilium, the *acetabular epiphysis*, (48,64) forms a large part of the superior wall of the acetabulum. The small secondary center of the ischium is rarely seen. The *os acetabuli*, the largest part, starts to develop at approximately 8 years of age and forms a significant part of the anterior wall of the acetabulum; it unites with the pubis at approximately 18 years of age. The acetabular epiphysis develops in the iliac acetabular cartilage at approximately 8 years and fuses with the ilium at 18 years of age, forming a substantial part of the superior acetabular joint surface (Fig. 20-3). The *secondary center of the ischium*, the smallest of the three, develops in the ischial acetabular cartilage at approximately the ninth year, unites with the ischium at 17 years, and contributes very little to acetabular development. These secondary centers should not be confused with avulsion fractures or loose bodies in the hip joint.



**FIGURE 20-3.** Right innominate bone of an adolescent. The *os acetabuli* (OA) is shown within the acetabular cartilage adjoining the public bone (PB); the acetabular epiphysis (AE), within the acetabular cartilage adjoining the iliac bone; and another small epiphysis (not labeled), within the acetabular cartilage adjoining the ischium (left). (From Ponseti IV. Growth and development of the acetabulum in the normal child. *J Bone Joint Surg Am* 1978;60:575–585; with permission.)

## AVULSION FRACTURES

Avulsion fractures of the pelvis usually occur in adolescents or young adults participating in sports (13,19,51) and usually are caused by powerful contraction of the attached muscle, although they can result from chronic repetitive traction on a developing apophysis, without acute trauma (13,19). The sartorius muscle attaches to the anterior superior iliac spine, the direct head of the rectus femoris to the anterior inferior iliac spine, and the hamstrings and adductors to the ischial tuberosity.

Of 91 pelvic avulsion fractures reported in 4 series (17,37,53,57), 38% were ischial avulsions, 32% were avulsions of the anterior superior iliac spine, and 18% were avulsions of the anterior inferior iliac spine (Fig. 20-4). Avulsions of the iliac crest (3%) and the lesser trochanter of the femur (9%) accounted for the rest.



**FIGURE 20-4.** Location of pelvic avulsion fractures in four series. (Modified from Ferbach SK, Wilkinson RH. Avulsion injuries to the pelvis and proximal femur. *Am J Radiol* 1981;137:581–584; with permission.)

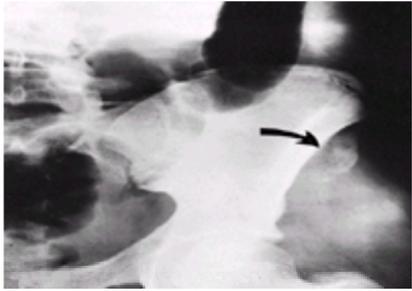
### Mechanism of Injury

Overpull of the sartorius muscle, especially with the hip in extension and the knee flexed, can result in avulsion fracture of the anterior superior iliac spine. Avulsion of the anterior inferior iliac spine can be caused by excessive exertion on the direct head of the rectus femoris muscle, which has its origin at the inferior iliac spine. Maximal exertion of the muscle occurs when the hip is hyperextended and the knee is flexed. This injury occurs most frequently in sports that involve kicking (e.g., soccer, rugby, football) and is much less common than avulsion of the anterior superior iliac spine, possibly because the anterior inferior iliac spine apophysis fuses earlier than the anterior superior iliac spine. Avulsion fractures of the ischial tuberosity can occur with maximal exertion by the hamstring muscles at their origin (38). These injuries occur most often in adolescents and young adults during sports, especially gymnastics, football, and track.

### Diagnosis

Symptoms usually include localized swelling and tenderness about the site of the avulsion fracture. Motion is limited, and pain may be mild or marked. In patients with chronic avulsions caused by repetitive activity, pain and limitation of motion usually are gradually progressive. In patients with ischial avulsions, pain at the ischial tuberosity can be elicited by flexing the hip and extending the knee. In this position, if the hip is moved into abduction, more pain is elicited. Patients also may have pain while sitting or moving on the involved tuberosity.

In patients with anterior superior iliac spine avulsions, radiographs show slight displacement of the apophysis (Fig. 20-5). In patients with anterior inferior iliac spine avulsions, radiographs show minimal distal displacement of the fragment. Further displacement is probably prevented because this is a conjoint tendon, and the reflected head of the rectus femoris muscle is intact. Contralateral views can be obtained and compared to ensure that this fragment is not actually a secondary center of ossification, either the *os acetabuli* or acetabular epiphysis (Fig. 20-6). With ischial tuberosity avulsions, radiographs reveal a large fragment displaced distally compared with the opposite ischial tuberosity (Fig. 20-7). Significant displacement is resisted by the intact sacrotuberous ligament.



**FIGURE 20-5.** Displaced fracture of the anterosuperior iliac spine.



**FIGURE 20-6. A:** Anteroposterior radiograph of an anterior inferior iliac spine fracture with displacement. **B:** Three-year follow-up shows union with no displacement and no pain.



**FIGURE 20-7.** Large ischial tuberosity fracture.

Because these avulsion fractures occur primarily through secondary centers of ossification before the center is fused with the pelvis, primarily in adolescents and young adults 14 to 25 years of age (64), comparison views of the contralateral apophysis should be taken to ensure that what appears to be an avulsion fracture is not in reality a normal adolescent anatomic variant. The exuberant callus formation can occasionally mimic an osteosarcoma. Recognition of initial deformity and fracture pattern is important to avoid unnecessary evaluation, such as CT and radionuclide scans, and inappropriate treatment.

#### Treatment and Prognosis

Usually, the only treatment necessary is a short period of rest with positioning of the hip to lessen stretch on the involved muscle and subsequent guarded weight bearing on crutches for 2 weeks or more. Fernbach and Wilkinson (17) found no decrease in athletic ability in their 20 patients treated conservatively, and they recommended surgery only for symptomatic nonunions. Although operative treatment has not been proven to improve results, Gordon et al. (20) recommended open reduction of all “widely displaced” avulsion fractures and fixation with a threaded Kirschner wire or lag screw, depending on the size of the fragment. Lynch and Renström (32) also recommended open reduction and internal fixation of large fragments displaced more than 2 cm.

Excessive callus formation after healing, especially of ischial avulsions, may be painful and impair sports activities ( Fig. 20-8), occasionally requiring excision of the callus formation and the apophysis. Sundar and Carty (57), at 44-month follow-up of sports-related avulsion pelvic fractures in 24 adolescents, found that 8 of 12 patients with ischial avulsions had significant limitation of athletic ability, and 5 had persistent symptoms. Of the 12 patients with avulsions of the anterior superior or anterior inferior iliac spine, 11 recovered completely and returned to active competition. Two of our patients with ischial tuberosity avulsions had chronic pain and disability that resolved after excision of the ischial apophysis; both returned to athletic competition and were pain free at 5-year follow-up.

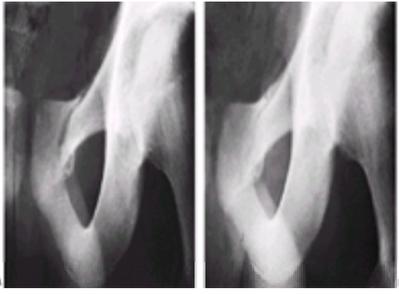


**FIGURE 20-8.** Ischial tuberosity fracture at time of fracture (A) and at 6-month follow-up (B), showing abundant callus formation.

#### FRACTURES OF THE PUBIS OR ISCHIUM

In children, pelvic rami fractures usually are caused by high-velocity trauma and have a significant number of associated injuries. In our series of pelvic fractures, of 45 pubic and ischial fractures, 38 were caused by vehicular accidents, most in either pedestrians or passengers in automobiles. Reed (51) reported that 45% of the pelvic fractures in the children in his series were rami fractures, and we noted a 33.6% incidence. Rieger and Brug (52) reported 20 (37%) “simple ring fractures” in their series of 54 pelvic fractures in children, and McIntyre et al. (33) reported that 23 (40%) of 57 pelvic fractures were “type I” (unilateral anterior) fractures. Single

ramus fractures are more common than multiple rami fractures, and the superior ramus is fractured more often than the inferior ramus ([7,16,51,61](#)) ([Fig. 20-9](#)).

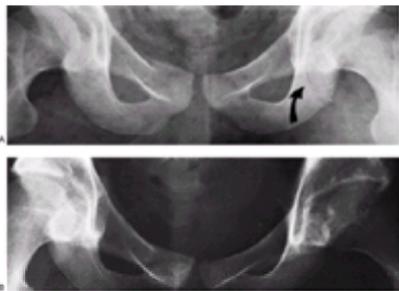


**FIGURE 20-9. A:** Stable superior pubic ramus fracture. The patient was allowed full weight bearing at 4 weeks postfracture. **B:** He was asymptomatic and radiography showed early callus formation.

In patients with isolated pubic fractures, clinical examination reveals pain and possible crepitus at the fracture site; however, there should be little or no motion on deep palpation. Inlet and outlet radiographic views or CT scanning are helpful in determining if any other pelvic fractures are present. If there is a significant displacement of the pubic rami, a second fracture through the pelvic ring should be suspected, although because of the plasticity of bone and elasticity of the symphysis and sacroiliac joints in children, more displacement can be expected than in adults with the same injury. Bed rest until pain subsides, followed by progressive weight bearing, usually is sufficient treatment.

#### Fractures of the Body of the Ischium

Fracture of the body of the ischium near the acetabulum is extremely rare in children. The fracture occurs from external force to the ischium, most commonly in a fall from a considerable height. The fracture usually is minimally displaced, and treatment consists of bed rest and progressive weight bearing ([Fig. 20-10](#)).



**FIGURE 20-10. A:** Nondisplaced fracture through the left ischium and contralateral superior pubic ramus fracture. **B:** Follow-up radiograph shows mild displacement and incongruity of the acetabulum and complete healing of the superior pubic ramus fracture. Either displacement of the fracture fragments or premature closure of the triradiate cartilage could have contributed to the incongruity of the femoral head in the acetabulum.

#### Stress Fractures of the Pubis or Ischium

Stress fractures are rare in small children, but they do occur in adolescents and young adults from chronic, repetitive stress to a bony area, and during the last trimester of pregnancy. Stress fractures of the pubis are likewise uncommon, but a small series of stress fractures, primarily in the inferior pubic rami, has been reported. Chronic symptoms and pain increased by stress may be noted in the inferior pubic area. Radiographs may show no evidence of a fracture for as long as 4 to 6 weeks, and then only faint callus formation may be visible; however, a technetium bone scan may reveal increased uptake ([26](#)), indicating a stress fracture, 3 to 4 weeks before changes on radiography. Treatment should consist of discontinuing the activity causing the repetitive stress to the area, and guarded weight bearing on crutches for 4 to 6 weeks.

At approximately the time of fusion of the ischiopubic ossification centers (age 6 to 7 years), the ischiopubic synchondrosis has been noted on radiographs and may persist for 2 to 3 years ([10,64](#)). Radiographs of the ischiopubic junction are, at best, difficult to interpret and may be misinterpreted as a fracture. Caffey ([10](#)) noted this radiographically in 57% of his pediatric patients at approximately 7 years of age. Quite often, the synchondrosis is bilateral (40%). An asymptomatic synchondrosis in a child 6 to 10 years of age should be treated as a variant of normal development (Van Neck's disease) ([62](#)). If it causes pain in a child older than 10 years of age, a fracture should be suspected and treated as such ([Fig. 20-11](#)).



**FIGURE 20-11. A:** Posterior hip dislocation in a 5-year-old girl. **B:** Nonconcentric reduction. Note widened joint space on left compared with right hip. Posterior open reduction revealed infolded labrum and a flap of capsule.

#### Fractures of the Wing of the Ilium (Duverney Fracture)

Direct trauma may cause a fracture of the wing of the ilium, but isolated iliac wing fractures are relatively rare. Reed ([51](#)) reported an incidence of 12% in children with fractures of the pelvis. Rieger and Burg ([52](#)) reported iliac wing fractures in only 3 (5.6%) of their 54 patients, and McIntyre et al. ([33](#)) reported only 7 (1.2%) in 57 fractures. However, this fracture can occur with other fractures of the pelvis, and the incidence may be slightly misleading: for example, in our series of 24 fractures of the wing of the ilium in children, only 11 were isolated fractures.

Displacement of the fracture usually occurs laterally, but it can occur medially or proximally. Severe displacement is prevented by preservation of some of the

attachments of the abdominal muscles and the hip abductors. Pain is located over the wing of the ilium, and motion at the fracture site also may be noted. A painful Trendelenburg gait may be present because of spasm of the hip abductor muscles.

A fracture of the wing of the ilium may be overlooked on an underexposed radiograph of the pelvis where the ilium is poorly seen as a large area of radiolucency. Use of a "hot light" is helpful in making the diagnosis ( [Fig. 20-12](#)).



**FIGURE 20-12.** Minimally displaced fracture of the left iliac wing.

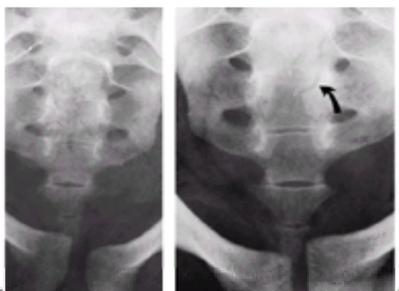
Treatment of an iliac wing fracture usually is dictated by the associated injuries. Bed rest in a comfortable position, usually with the leg abducted, is all that is necessary for treatment of the fracture itself. This should be followed by partial weight bearing on crutches until the symptoms are completely resolved. Regardless of the amount of comminution or displacement, these fractures usually unite without complications or sequelae ( [Fig. 20-13](#)).



**FIGURE 20-13. A:** Severely comminuted fracture of the left iliac wing. **B:** Radiograph at 3-month follow-up shows fracture healed with displacement, but the patient was asymptomatic.

## FRACTURES OF THE SACRUM

Sacral fractures constitute a small portion of pelvic fractures in children. Rieger and Brug ( [52](#)) reported 2 sacral fractures and 7 sacroiliac fracture–dislocations in their 54 patients. Our series of pelvic fractures in children included two patients with sacral fractures (4%), 15 and 17 years of age. Sacral fractures may be slightly more common than reported, but because they are obscured by the bony pelvis and soft tissue shadows of the abdominal viscera, and because they are rarely displaced, they may be overlooked ( [Fig. 20-14](#)). These fractures are significant because they may damage the sacral nerves, resulting in loss of bladder and bowel function.



**FIGURE 20-14. A:** Radiograph suggesting comminuted nondisplaced linear sacral fracture on the left. **B:** At 6-week follow-up, radiograph shows definite evidence of linear sacral fracture.

Sacral fractures are best diagnosed clinically. Pain and swelling may be present, usually over the lower part of the sacrum. Rectal examination causes pain anterior to the sacrum. Occasionally, the fracture fragments may be felt. Repeated bimanual rectal examination with attempts at reduction should be avoided because a tear in the rectum may occur.

The fractures are difficult to see on radiographs. The fracture can be oblique, but most are transverse with minimal displacement and occur through a sacral foramen, which is the weakest part of the body of the sacrum. Minimal offset of the foramen or offset of the lateral edge of the body of the sacrum is an indication of sacral fracture. Lateral views are helpful only if there is anterior displacement, which is rare. A 35-degree caudad view of the pelvis may reveal a fracture of the body of the sacrum. CT scans may be useful for determining the amount of anterior displacement, if any, in these fractures ( [Fig. 20-15](#)).



**FIGURE 20-15. A:** A 15-year-old boy with an obvious fracture of the left acetabulum and symphysis pubis diastasis, and a questionable and ill-defined sacral fracture. **B:** Computed tomography scan reveals a comminuted displaced sacral fracture.

## FRACTURES OF THE COCCYX

Significant soft tissue injury to the coccyx makes it difficult to determine on radiograph if a coccygeal fracture has occurred, especially in a child. However, historically, trauma to the coccyx is often refractory to treatment. For this reason, if clinical symptoms are sufficient, an injury to this area in a child should be considered a fracture regardless of whether a fracture can be seen on radiographs.

Coccygeal fractures are not included in most large series of fractures of the pelvis in adults and children, although the coccyx is part of the pelvis. Of our nine patients with coccygeal fractures, in eight the mechanism of injury was similar to that in adults: a direct fall onto the buttocks in the sitting position. There were no associated injuries with these nine fractures. Clinically, there was immediate, severe pain in the area of the coccyx in all patients. Pain on defecation may be present as well as pain on rectal examination. Because radiographic identification is difficult, the diagnosis should be made clinically by digital rectal examination. Exquisite pain may be elicited, and an abnormal mobility of the coccygeal fragments may be noted. Acute symptoms may abate at 1 to 2 weeks, but in our patients symptoms while sitting persisted for an average of 4 weeks.

Lateral radiographs of the coccyx with the hips flexed maximally may reveal the fracture ([Fig. 20-16](#)). The coccyx may appear to be acutely angulated as a variant of normal, and a fracture may not be seen, or the normal acute angulation may be interpreted as a fracture or dislocation.



**FIGURE 20-16.** Lateral radiograph with the hips maximally flexed reveals displaced coccygeal fracture in a 14-year-old boy.

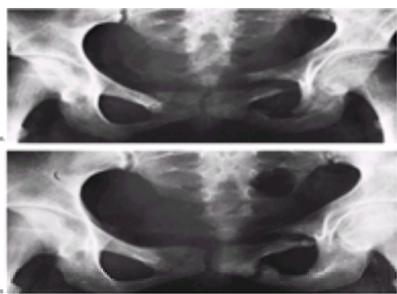
In all nine of our patients, the fracture was identified. Treatment consisted of an inflated doughnut cushion in three patients, decreased activity in three patients, and no treatment in three patients. The longest period of active treatment was 6 weeks (average, 4 weeks). Unlike adults with trauma to the coccyx, none of these children had prolonged impairment or psychological overtones.

## FRACTURES OF TWO IPSILATERAL RAMI

Fractures of the pubic rami, which are more common than any other pelvic fracture in children, may be stable bilateral fractures or ipsilateral fractures of the superior and inferior rami. Ipsilateral fractures, although stable, may produce an increased incidence of associated injuries to abdominal viscera, especially the bladder ([16](#)).

Considerable force is necessary to cause this fracture pattern, and other associated fractures should be expected. A general evaluation should be followed by examination of the pelvis and lower extremities, with special attention to abrasions, contusions, lacerations, and ecchymosis about the pelvis. Palpation reveals discomfort anteriorly, and crepitus at the fracture site may be noted.

Various methods of treatment have been advocated for adults, such as bed rest, lower limb traction, and pelvic sling. However, in children, the fracture almost always unites with adequate remodeling of even the most displaced fractures. For this reason, short-term bed rest followed by progressive weight bearing on crutches on the involved side is all that is necessary ([Fig. 20-17](#)).



**FIGURE 20-17. A:** Ipsilateral left pubic rami fractures with a contralateral right superior rami fracture. **B:** At 2-year follow-up, nonunion of the ipsilateral rami fractures is evident, but the patient is asymptomatic.

## FRACTURES NEAR OR SUBLUXATION OF THE SYMPHYSIS PUBIS

Isolated injuries in the symphysis pubis area are rare, primarily because they usually occur in association with disruption of posterior structures such as the sacroiliac joint. Although significant force appears to be necessary to disrupt or fracture the symphysis pubis, isolated disruption of the symphysis can occur ([64](#)). Usually, there is some normal elasticity at the symphysis in adults (0.5 mm in men, 1.5 mm in women), and there is probably even more in children, depending on skeletal maturity.

Clinically, exquisite pain is present anteriorly at the symphysis; the legs are externally rotated and often pain is worse in the supine position than in the side-lying position ([64](#)). Motion of the hips in flexion, abduction, external rotation, and extension is restricted and painful (fabere sign).

Radiographs may reveal subluxation and widening of the symphysis pubis, as if opening a book. Offset may be superior, inferior, anterior, or posterior ([Fig. 20-18](#)). Furthermore, a fracture near or into the symphysis may produce an equivalent subluxation of the symphysis pubis ([Fig. 20-19](#)). Because of the variable normal separation of the symphysis in children of different ages, the amount of traumatic separation may be difficult to evaluate. Watts ([64](#)) suggested radiographs with and without lateral compression of the pelvis. More than 1 cm of difference in the width of the symphysis pubis between the two views suggests a symphysis pubis separation.



**FIGURE 20-18.** Mild symphysis pubis subluxation with superior displacement. At 4-year follow-up, the patient is asymptomatic.



**FIGURE 20-19.** Fracture adjacent to the symphysis pubis with equivalent symphysis pubis separation.

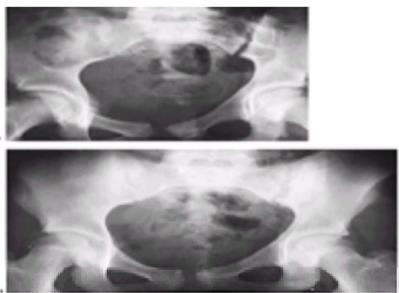
We saw a child with anterior pubic pain and obvious widening of the symphysis pubis after a moving vehicle accident. On radiographs, it appeared that the child had an isolated disruption of the symphysis pubis; however, when an adequate history was obtained and physical examination was performed, it was apparent that the child had been treated for exstrophy of the bladder with congenital widening of the symphysis. The lesson learned should be obvious: “If all else fails, examine the patient.”

Treatment of isolated fractures or subluxations of the symphysis pubis should consist of bed rest, usually in a side-lying position, especially if other injuries are present. Unilateral Buck’s traction may relieve pain, but it rarely improves alignment of the fracture or subluxation. Application of a spica cast in the lateral position with lateral compression may also reduce the displacement and decrease hospitalization ( [64](#) )

### FRACTURES NEAR OR SUBLUXATION OF THE SACROILIAC JOINT

Fractures near or subluxation of the sacroiliac joint are rare, isolated injuries, probably even less common than isolated fractures at the weaker symphysis pubis. More commonly, disruption of the sacroiliac joint occurs with fractures or dislocations of the anterior portion of the pelvis, causing an unstable injury of the pelvis. Sacroiliac dislocations differ from those in adults in several ways. In children, fractures tend to be incomplete because of partial tearing of the anterior sacroiliac ligaments and the thick posterior periosteum ( [43](#) ). A subchondral fracture through structurally weak zones of cartilage may leave the sacroiliac joint intact ( [15](#) ). Associated vascular and neurologic injuries are common ( [23](#) ).

Subluxation or fracture of the sacroiliac joint should be suspected with high-velocity trauma and injury to the posterior aspect of the pelvis near the sacroiliac joint. In patients with these injuries, the fabere sign is markedly positive on the ipsilateral side ( [15,23](#) ). Comparison views of both sacroiliac joints should be carefully evaluated to determine any asymmetry of the wings of the ilium with more separation at the sacroiliac joint ( [Fig. 20-20](#) ). Any offset of the distal articular surface of the sacrum and ilium on radiography is an indication of sacroiliac joint disruption. Oblique views for comparison of both sacroiliac joints often are beneficial. Because of the rarity of this subluxation or fracture, multiple views, including inlet and outlet views, and axial CT scan may be necessary to ensure that there is no anterior fracture ( [Fig. 20-21](#) ).



**FIGURE 20-20.** Separation of the left sacroiliac joint with asymmetry of the wings of the ilium. **A:** Careful scrutiny of the radiograph reveals contralateral pubic rami fractures. **B:** At 2-year follow-up, the sacroiliac joint is slightly wide, but the patient is asymptomatic.



**FIGURE 20-21.** Fracture of the superior pubic ramus; occult fractures of the ipsilateral sacrum and the sacroiliac joint at the distal articular surface also are present.

Bed rest and guarded weight bearing on crutches is probably all the treatment needed for isolated subluxations or fractures. Heeg and Klasen ( 23) reported sacroiliac joint dislocations in 18 children, 10 of whom had extensive degloving injuries of the posterior pelvis. Ten were treated nonoperatively, six with open reduction and internal fixation, one with open reduction but no internal fixation, and one with external fixation. Disabling long-term symptoms persisted from incomplete neurologic recovery in six.

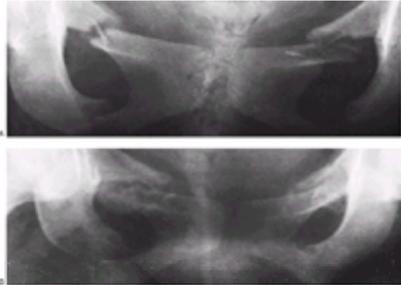
## UNSTABLE FRACTURE PATTERNS

Unstable pelvic fracture combinations usually are of three types:

- Double vertical pubic rami fractures (straddle or floating fractures) or dislocations of the pubis that occur as an anterior double break in the pelvic ring anteriorly
- Double fractures in the pelvic ring anteriorly and posteriorly, through the bony pelvis, sacroiliac joint, or symphysis pubis (Malgaigne fractures)
- Multiple crushing injuries that produce at least two severely comminuted fractures in the pelvic ring

### Bilateral Fractures of the Inferior and Superior Pubic Rami

Bilateral fractures of both the inferior and superior pubic rami (straddle fractures) cause a floating anterior arch of the pelvic ring that is inherently unstable ( Fig. 20-22), as does dislocation of the symphysis pubis with fractures of both ipsilateral pubic rami. This fracture pattern frequently is associated with bladder or urethra disruption.



**FIGURE 20-22.** A: Classic example of a straddle fracture in a 16-year-old girl. B: At 6 weeks after injury, abundant callus formation is present and the fractures have healed.

Bilateral fractures of the inferior and superior pubic rami can occur in a fall while straddling a hard object or by lateral compression on the pelvis. The floating fragment usually is displaced superiorly, being pulled in this direction by the rectus abdominis muscles ( 64). Radiographically, an inlet view most accurately determines the amount of true displacement of the floating fragment.

In a child, regardless of the amount of displacement, the fracture should heal and remodeling can be expected. Because this fracture does not involve the weight-bearing portion of the pelvis, it does not cause leg-length discrepancy. Skeletal traction is unnecessary, and a pelvic sling is contraindicated because of the possibility that compression will cause medial displacement of the ilium ( 64).

Treatment should consist simply of bed rest in the semi-Fowler position with flexion of the hips to relax the abdominal musculature. If the fracture was caused by lateral compression forces, the lateral decubitus position is contraindicated for fear of medial displacement of the ilium.

### Complex Fracture Patterns

Fractures and dislocations of the posterior arch (posterior to the acetabulum) combined with anterior ipsilateral or contralateral fractures or dislocations of the anterior arch [Malgaigne (36) fractures; Fig. 20-23] result in instability of the hemipelvis and acetabulum. These unstable fractures are associated with retroperitoneal and intraperitoneal bleeding. Bilateral anterior and posterior fractures are the most likely fracture pattern to cause severe hemorrhage. Initial treatment usually involves replacement of blood volume and stabilization of the child's overall condition before treatment of the pelvic fractures ( 60).



**FIGURE 20-23.** An unusual Malgaigne fracture; fracture extends through the ilium into the sacroiliac joint with ipsilateral pubic rami fractures.

Three mechanisms of injury have been implicated in these fractures and fracture–dislocations: anteroposterior compression forces, lateral compression forces, and, with the hip fixed in extension and abduction, indirect forces transmitted proximally along the femoral shaft.

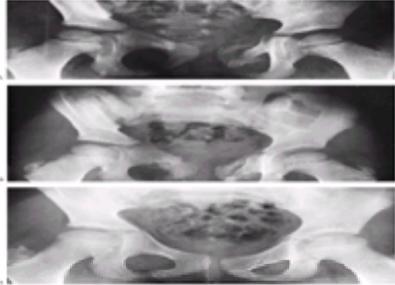
Aside from the physical signs usually associated with pelvic fractures, leg-length discrepancy and asymmetry of the pelvis also may be present because of the displacement of the hemipelvis. If the measured distance from the umbilicus to the medial malleolus is unequal for the two extremities, and the distance from the anterior superior iliac spine to the medial malleolus is the same, pelvic obliquity or displacement is present rather than true leg-length discrepancy. Inlet and outlet radiographic views and CT scanning reveal the amount of pelvic displacement.

Numerous treatment regimens have been successful, depending on the type of fracture and the amount of displacement. For fractures with mild displacement, bed rest in the lateral recumbent position may be all that is necessary. If lateral displacement is severe, closed manipulation in the lateral decubitus position and spica casting can be used, as described in Chapter 35 in Volume 2 of this series. If the displacement is cephalad only, skeletal traction or even skin traction can be used in a small child. Occasionally, manipulation under anesthesia may be required. After successful manipulation of the fragments, traction on the involved side can be used to maintain the reduction. Open or percutaneous external fixation of the pelvis with pins incorporated in a distraction or compression device has been advocated to allow accurate reduction of the fracture or dislocation, early ambulation (non-weight bearing), and decreased pain secondary to instability.

Schwarz et al. (54), in a long-term (2 to 25 years) follow-up of 17 children with nonoperatively treated unstable pelvic fractures, reported unsatisfactory results in 8 due to pelvic asymmetry. They emphasized that reduction of pelvic ring fractures should be as anatomic as possible because healing in malposition causes poor results.

Nierenberg et al. (42), however, reported excellent or good results after conservative treatment of 20 unstable pelvic fractures in children despite radiographic evidence of deformity. They suggested that treatment guidelines for unstable pelvic fractures are not the same for children as for adults, and recommended that external or internal fixation should be used only when conservative methods fail.

Operative treatment of pelvic fractures in children is not routinely recommended (3) because (a) exsanguinating hemorrhage is unusual in children, so operative pelvic stabilization to control bleeding rarely is necessary (3,41); (b) pseudarthrosis is rare in children and fixation is not necessary to promote healing (51); (c) the thick periosteum in children tends to help stabilize the fracture, so surgery usually is not necessary to obtain stability (51); (d) prolonged immobilization usually is not necessary for fracture healing (42); (e) significant remodeling can occur in skeletally immature patients (18) (Fig. 20-24); and (f) long-term morbidity after pelvic fracture is rare in children (18,41). Operative fixation may be indicated to facilitate wound treatment in open fractures, control hemorrhage during resuscitation, allow patient mobility and make nursing care easier, prevent deformity in severely displaced fractures that may not heal or adequately remodel, improve overall patient care in patients with polytrauma, minimize risk of growth disruption, or restore articular congruity.

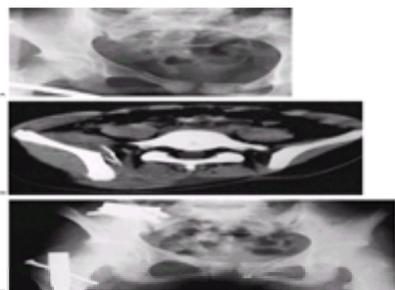


**FIGURE 20-24.** **A:** A 6-year-old child with a Malgaigne fracture with right sacroiliac joint displacement and multiple (four) pubic rami fractures. **B:** Four weeks after injury. **C:** At 5-year follow-up, complete remodeling is present.

Keshishyan et al. (29) advocated external fixation of complex pelvic fractures, especially in children with polytrauma, and Gordon et al. (20) suggested external fixation or open reduction and internal fixation in children older than 8 years of age because hip spica casting is poorly tolerated in older children. Stiletto et al. (56) reported good results after open reduction and internal fixation of unstable pelvic fracture in two toddlers. AO small-fragment instrumentation was used in both. These surgeons recommended protection in a spica cast for 6 to 8 weeks, with removal of the implants at that time. Occasionally, open reduction and internal fixation of severely malaligned fractures may be indicated. However, because of the potential surgical complications, open reduction of pelvic fractures in children should not be undertaken casually by the inexperienced surgeon.

## AUTHORS' PREFERRED TREATMENT

Treatment is more likely to be conservative in younger children and operative in juveniles and adolescents. For toddlers, we prefer to use bed rest and distal femoral skeletal traction on the displaced side of the hemipelvis. The younger the child, the more likely that traction will be adequate treatment and the pelvis will remodel. Open reduction and internal fixation rarely are required in a young child unless severe (>3 cm) displacement of the sacroiliac joint cannot be corrected with traction. Combining open reduction and internal fixation with external fixation may be necessary in a child older than 8 to 10 years of age with an unstable fracture and severe polytrauma (Fig. 20-25). The technical principles are identical to those used for unstable pelvic fractures in adults (see Chapter 35, Volume 2). In older adolescents, treatment should follow the guidelines for the treatment of adult fractures (see Chapter 35, Volume 2), including a combination of internal and external fixation for fracture stabilization and early mobilization.



**FIGURE 20-25.** **A:** Multiple trauma in this 12-year-old child included three fractures of the pubic rami, disruption and fracture of the sacroiliac joint on the right, and a femoral shaft fracture on the right. **B:** Computed tomography scan shows fracture of the ilium and disruption of the sacroiliac joint. **C:** After open reduction and internal fixation of the sacroiliac joint and closed intramedullary nailing of the femoral shaft fracture. Note femoral nail inserted through the greater trochanter.

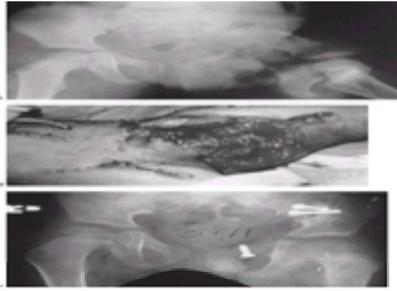
### Severe Multiple or Open Fractures

In patients with crushing injuries, distortion of the pelvis is severe and, in addition to multiple breaks in the pelvic ring, apparent or occult fractures of the sacrum may be present, with or without neurologic involvement. Massive hemorrhage is common, and only rarely does a child survive this major insult (39,64). The patient is usually in hypovolemic shock, and emergency measures outlined previously in this chapter may be necessary.

Although total disruption of the pelvis is apparent on radiographs, usually one hemipelvis is partially intact. In general, each complex fracture needs a special treatment regimen; however, the treatment outlined for unstable fracture patterns usually can align the pelvis. Treatment may have to be by trial and error, with serial radiographs and CT scans to evaluate the progress of realignment.

The patient should be stable without evidence of a drop in blood volume before any operative intervention, either closed or open external fixation or open reduction, is undertaken.

These severe multiple fractures about the pelvis are important because the mobile fracture fragments may penetrate visceral organs (e.g., the bladder or abdominal viscera), lacerate the abdominal vascular tree, or cause neurologic involvement (Fig. 20-26). These acute injuries should take precedence over realignment of the pelvic architecture, although if possible during emergency surgery such as a laparotomy, pelvic stabilization should be achieved quickly with a combination of internal and external fixation as needed while the patient is under general anesthesia. In particular, the application of an external fixator may decrease blood loss by stabilizing mobile, bleeding bone fragments (1,58,59).



**FIGURE 20-26. A and B:** Complex open type IIIC pelvic fracture in a 3-year-old boy. Vascular (femoral artery and vein) and neurologic injuries also were present. Multiple debridements were required, as were colostomy and vesicostomy. The fractures of the wing of the ilium and pubic rami were fixed with small screws. **C:** One year after injury. (Courtesy of Dr. Gerry Clancy, Children's Hospital, Denver, CO.)

Open pelvic fractures are rare in children. Mosheiff et al. (40) reported that 13% of 116 pediatric pelvic fractures seen over a 12-year period were open injuries. Fourteen of the 15 children were struck by motor vehicles and one sustained a gunshot wound. Five children with stable fractures were treated nonoperatively, and 10 with unstable fractures were treated with external fixation (5), internal fixation (2), or combined external and internal fixation (3). Three of the children died because of uncontrollable hemorrhage (two patients) and chest injury (one patient). Eleven of the 12 surviving children had deep wound infection or sepsis, and 3 had premature physeal closure. Mosheiff et al. (40) emphasized that the treatment of the soft tissue injuries depends on stabilization of the pelvis and that external fixation alone is insufficient for most open pelvic fractures.

## ACETABULAR FRACTURES

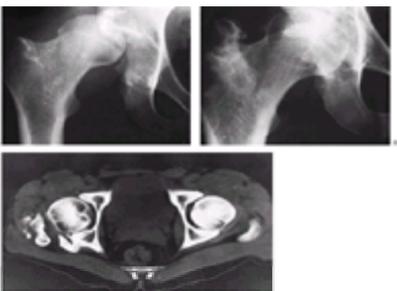
Acetabular fractures constitute only 1% to 15% of pelvic fractures in children, making them very uncommon. The mechanism of injury of acetabular fractures in children is similar to that in adults: the fracture occurs from a force transmitted through the femoral head. The position of the leg with respect to the pelvis and the location of the impact determine the fracture pattern; the magnitude of the force determines the severity of the fracture or fracture–dislocation. Patients with high-energy injuries usually have major associated injuries, whereas isolated acetabular fractures can occur from low-energy forces.

### Classification

Watts (64) described a classification of acetabular fractures in children that consisted of four types: (a) small fragments that most often occur with dislocation of the hip, (b) linear fractures that occur in association with pelvic fractures without displacement and usually are stable, (c) linear fractures with hip joint instability, and (d) fractures secondary to central fracture–dislocation of the hip. More recently, however, acetabular fractures in both adults and children usually are classified by the system of Letournel and Judet (28,31). A more comprehensive classification is based on the AO comprehensive fractures classification, which groups all fractures into A, B, and C types with increasing severity. Type A acetabular fractures involve a single wall or column; type B fractures involve both columns (transverse or T-types) and a portion of the dome remains attached to the intact ilium; and type C fractures involve both columns and separate the dome fragment from the axial skeleton by a fracture through the ilium. Both of these classification systems are discussed in more detail in Chapter 35, Volume 2 of this series (*Fractures in Adults*).

### Radiographic Evaluation

Anteroposterior and lateral views may not adequately show the amount of displacement of acetabular fragments after fracture. Inlet, outlet, and 45-degree oblique (Judet) views often are necessary to appreciate the amount of displacement. CT scanning can be used to determine the amount of acetabular displacement (Fig. 20-27) and to determine if any retained fragments in the acetabulum are preventing an accurate concentric reduction (11). Three-dimensional CT reconstructions can give an excellent view of the overall fracture pattern, but often underestimate minimally displaced fractures.



**FIGURE 20-27. A:** Traumatic dislocation with a small acetabular fragment. **B:** After reduction, a small fragment is visible, but it is not impeding hip congruity or function. **C:** At 12 weeks after injury, computed tomography scan reveals that the fragment is from the posterior acetabulum, with mild displacement of the acetabulum posteriorly.

After reduction of a hip dislocation, radiographs of both hips should be carefully compared to ensure that the reduction is not incongruous. Subtle signs of an incongruous reduction with retained osseous or cartilaginous fragments, an inverted limbus, or entrapped soft tissues include minimal widening of the joint (without traction applied) and asymmetry of Shenton's line compared with the opposite hip (11) (Fig. 20-28). If there is any doubt about the concentricity of the reduction, CT or MRI is indicated.



**FIGURE 20-28. A:** Posterior hip dislocation in a 5-year-old girl. **B:** Nonconcentric reduction. Note widened joint space on left compared with right hip. Posterior open reduction revealed infolded labrum and a flap of capsule.

## Treatment

The aim of treatment for acetabular fractures in children is the same as for adults: to restore joint congruity and hip stability. Treatment guidelines in general follow those for adults. Bed rest or non-weight-bearing ambulation with crutches can be used for nondisplaced or minimally ( $\leq 1$  mm) displaced fractures. Because weight-bearing forces must not be transmitted across the fracture, crutch ambulation is appropriate only for older children who can be relied on to avoid putting weight on the injured limb. Non-weight bearing usually is continued for 6 to 8 weeks. In younger children, this may be shortened to 5 or 6 weeks, and in adolescents (older than 12 years of age), partial weight bearing should be continued for 3 to 4 more weeks. For fractures in which displacement can be reduced to less than 2 mm, skeletal traction with a traction pin in the distal femur can be used. Because traction must be maintained for 5 to 6 weeks, this option usually is not feasible in older children or adolescents.

Gordon et al. (20) recommended accurate reduction and internal fixation of any displaced acetabular fracture in a child. They noted that the presence of incomplete fractures and plastic deformation may make accurate reduction difficult or impossible; they recommended that incomplete fractures be completed and that osteotomies of the pubis, ilium, or ischium be made if necessary for accurate reduction of the acetabulum. In children with open physes, all periacetabular metallic implants should be removed 6 to 18 months after surgery.

Improved outcomes with early (<24 hours) fixation of acetabular fractures in adults has been reported (46), and Gordon et al. (20) noted that early fixation is especially important to prevent malunion in young patients in whom healing is rapid.

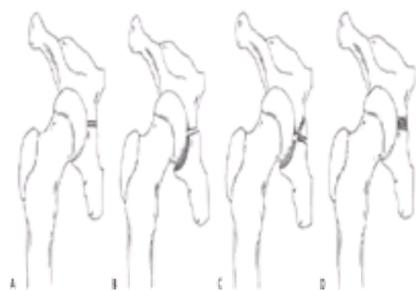
In addition, anatomic alignment of the triradiate cartilage should be obtained in children. Linear growth of the acetabulum occurs by interstitial growth in the triradiate part of the cartilage complex, causing the pubis, ischium, and ilium to enlarge. The depth of concavity of the acetabulum is in response to the presence of a spherical femoral head and increases during development as a result of interstitial growth in the acetabular cartilage. Cessation of growth of all or part of the triradiate cartilage occurring secondary to fracture may result in a dysplastic acetabulum.

Acetabular dysplasia secondary to growth arrest (bony bridge) of the triradiate cartilage has been reported after trauma to the acetabulum (Fig. 20-29). Heeg et al. (22,24,25) reported acetabular deformity and subluxation of the hip in two of three patients with premature fusion of the triradiate cartilage. Peterson and Robertson (45) reported formation of a physeal osseous bar in a 7-year-old boy 2 years after fracture of the lateral portion of the superior ramus at the junction with the triradiate cartilage. After excision of the osseous bridge, the physis remained open. Although the injured physis closed earlier than the contralateral side, there was only a slight increase in the thickness of the acetabular wall and lateral displacement of the femoral head. Peterson and Robertson emphasized that early recognition and treatment are essential, before premature closure of the entire physis and development of permanent osseous deformity.



**FIGURE 20-29.** A: Fractures of the left superior and inferior pubic rami and the left ilium with injury to the right triradiate cartilage in a 5-year-old boy. B: Three years after injury, the pubic rami fractures are healed and remodeling has occurred, but acetabular dysplasia, widening of the “teardrop,” and mild subluxation are evident in the right hip. C: At 15 years of age, the sequelae of the mild triradiate cartilage injury are still apparent, but he is not symptomatic.

Bucholz et al. (8) noted two main patterns of physeal disturbance in nine patients with triradiate cartilage injury: a Salter-Harris type I or II injury, which had a favorable prognosis for continued normal acetabular growth, and a Salter-Harris type V crush injury, which had a poor prognosis with premature closure of the triradiate cartilage caused by formation of a medial osseous bridge (Fig. 20-30). In either pattern, the prognosis depended on the child's age at the time of injury. In young children, especially those younger than 10 years of age, acetabular growth abnormality was common and resulted in a dysplastic acetabulum. By the time of skeletal maturity, disparate growth increased the incongruity of the hip joint and led to progressive subluxation. These researchers found that acetabular reconstruction was frequently necessary to correct the gradual subluxation of the femoral head.



**FIGURE 20-30.** Types of triradiate cartilage fractures. A: Normal triradiate cartilage. B: Salter-Harris type I fracture. C: Salter-Harris type II fracture. D: Salter-Harris type V (compression) fracture. (Redrawn from Scuderi G, Bronson MJ. Triradiate cartilage injury: report of two cases and review of the literature. *Clin Orthop* 1987;217:179–189; with permission.)

## Surgical Treatment

The surgical approach varies according to the pattern of the fracture and the direction of the displacement as determined on the preoperative radiographs and CT scans (20) (Table 20-5). Fractures of the posterior wall or posterior column can be approached through a Kocher-Langenbeck approach with the patient either in the lateral decubitus position (isolated posterior wall fracture) or supine (associated posterior column fracture). Anterior column injuries can be approached through an ilioinguinal approach. Some transverse fractures may require an extended iliofemoral approach, but this is rare in children (12). The extended lateral approaches, which include the extended iliofemoral and triradiate approaches, should be avoided as much as possible because of the risk of devascularization of the ileum and heterotopic bone formation (21).

The surgeon should be familiar with Judet's (29) treatise on the operative reduction of acetabular fractures and with Letournel's works before performing this surgery. For smaller children and smaller fragments, Watts (64) recommended threaded Kirschner wires for reduction. In larger children, cannulated screws may aid in reduction and provide secure fixation (Fig. 20-31). Small-fragment reconstruction plates, appropriately contoured, also can be used. Gordon et al. (20) described the addition of a small (two- or three-hole) “hook plate” for small or comminuted fragments (Fig. 20-32). Because operative procedures about the hip may be necessary later, the hardware in a child may be removed in this situa- tion.

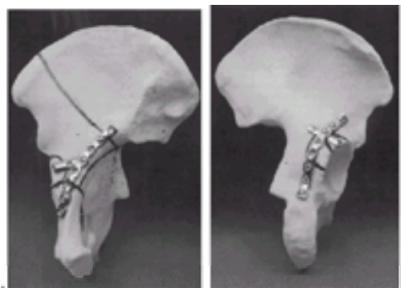
Fracture Type	Exposure
Anterior column or wall	Iliinguinal
Posterior column or wall	Kocher-Langenbeck
Transverse	Iliinguinal (or extended lateral)
T-shaped	Iliinguinal and Kocher-Langenbeck (or extended lateral)
Anterior column and posterior hemitransverse	Iliinguinal
Both columns	Iliinguinal (or extended lateral)

From Gordon RG, Karpik K, Hardy S, et al. Techniques of operative reduction and fixation of pediatric and adolescent pelvic fractures. *Oper Tech Orthop* 1995;5:95-114; with permission.

**TABLE 20-5. SURGICAL EXPOSURE FOR OPERATIVE FIXATION OF ACETABULAR FRACTURES**



**FIGURE 20-31. A:** Fracture of the wing of the ilium with extension into the dome of the acetabulum in a 3-year-old boy. **B:** After reduction and fixation with two cannulated screws. (From Habacker T, Heinrich SD, Dehne R. Fracture of the superior pelvic quadrant in a child. *J Pediatr Orthop* 1995;15:69-72; with permission.)



**FIGURE 20-32. A:** Anterior column plate and additional anterior wall "hook" plate. **B:** Posterior wall buttress plate and hook plate. (From Gordon RG, Karpik K, Mears DC. Techniques of operative reduction and fixation of pediatric and adolescent pelvic fractures. *Oper Tech Orthop* 1995;5:95-114; with permission.)

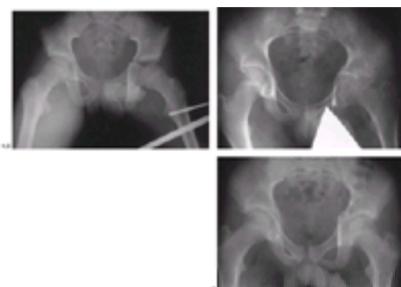
Brown et al. (6) described the use of CT image-guided fixation of acetabular fractures in 10 patients, including bilateral posterior wall fractures in a 14-year-old girl. They cite as advantages of image-guided surgery reduced operating time (approximately 20% reduction), less extensive surgical dissection, reduced fluoroscopic time, and compatibility with traditional fixation techniques. Most important, it allows accurate and safe placement of screws and pins for acetabular fixation.

### Postoperative Management

Small children can be immobilized in a spica cast for 6 weeks. If radiographs show adequate healing at that time, the cast is removed and free mobility is allowed. In an older child with stable fixation, crutches are used for protected weight bearing for 6 to 8 weeks. If radiographs show satisfactory healing, weight bearing is progressed as tolerated. Return to vigorous activities, especially competitive sports, is delayed for at least 6 months.

### COMPLICATIONS

Because of the remodeling potential in young children, loss of reduction and malunion usually are not problems. Reported complications include premature triradiate cartilage closure, avascular necrosis, traumatic arthritis, sciatic nerve palsy, heterotopic myositis ossificans about the acetabulum and pelvis after acetabular fractures (Fig. 20-33), and pelvic asymmetry at long-term follow-up of female patients. Because this asymmetry may cause maternal dystocia during childbearing, pelvimetry is recommended before pregnancy. Rieger and Brug (52) reported one female patient who required cesarean section because of ossification of the symphysis pubis after nonoperative treatment of an open-book fracture. Schwarz et al. (54) reported leg-length discrepancies of 1 to 5 cm in 10 of 17 patients after nonoperative treatment of unstable pelvic fractures; 5 complained of low back pain at long-term follow-up. Nine of 10 patients with lumbar scoliosis also had low back pain.



**FIGURE 20-33. Central fracture–dislocation of the hip with injury to the triradiate cartilage in a 15-year-old boy. Note distraction and incongruous reduction. A:** During skeletal traction after reduction of the hip dislocation.

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## CHAPTER REFERENCES

1. Alonzo JE, Horowitz M. Use of the AO-ASIF external fixator in children. *J Pediatr Orthop* 1987;7:594–600.
2. Batislam E, Ates Y, Germiyanoglu C, et al. Role of Tile classification in predicting urethral injuries in pediatric pelvic fractures. *J Trauma* 1997;42:285–287.
3. Blasler RD, McAtee J, White R, et al. Disruption of the pelvic ring in pediatric patients. *Clin Orthop* 2000;376:87–95.
4. Blount WP. *Fractures in children*, 2nd ed. Baltimore: Williams & Wilkins, 1965.
5. Bond SJ, Gotschall CS, Eichelberger MR. Predictors of abdominal injury in children with pelvic fracture. *J Trauma* 1991;31:1169–1173.
6. Brown GA, Willis MC, Firoozbakhsh K, et al. Computed tomography image guided surgery in complex acetabular fractures. *Clin Orthop* 2000;370:219–226.
7. Bryan WJ, Tullos HS. Pediatric pelvic fractures: review of 52 patients. *J Trauma* 1979;19:799–805.
8. Bucholz RW, Ezaki M, Ogden JA. Injury to the acetabular triradiate physeal cartilage. *J Bone Joint Surg Am* 1982;64:600–609.
9. Buckley SL, Gotshall CS, Eichelberger MR. Predictors of abdominal injury in children with pelvic fractures. *J Trauma* 1991;31:1169–1173.
10. Caffey JP. *Pediatric x-ray diagnosis: textbook for students and practitioners of pediatrics, surgery and radiology*, 7th ed. Chicago: Year Book Medical Publishers, 1978.
11. Canale ST, Manugian AH. Irreducible traumatic dislocations of the hip. *J Bone Joint Surg Am* 1979;61:7–14.
12. Carnesale PG. Extensile approach to the acetabulum. In: Crenshaw AH, ed. *Campbell's operative orthopaedics*, 8th ed. St. Louis: Mosby-Year Book, 1992:83–85.
13. Clancy WG, Foltz AS. Iliac apophysis and stress fractures in adolescent runners. *Am J Sports Med* 1976;4:214.
14. Conway FM. Fractures of the pelvis: a clinical study of 56 cases. *Am J Surg* 1935;30:69–82.
15. Donoghue V, Daneman A, Krajchik I, et al. CT appearance of sacro-iliac joint trauma in children. *J Comput Assist Tomogr* 1985;9:352–356.
16. Dunn AW, Morris HD. Fractures and dislocations of the pelvis. *J Bone Joint Surg Am* 1968;50:1639–1648.
17. Fernbach SK, Wilkinson RH. Avulsion injuries to the pelvis and proximal femur. *Am J Radiol* 1981;137:581–584.
18. Garvin KL, McCarthy RE, Barnes CL, et al. Pediatric pelvic ring fractures. *J Pediatr Orthop* 1990;10:577–582.
19. Godshall RW, Hansen CA. Incomplete avulsion of a portion of the iliac epiphysis: an injury of young athletes. *J Bone Joint Surg Am* 1973;55:1301–1302.
20. Gordon RG, Karpik K, Hardy S, et al. Techniques of operative reduction and fixation of pediatric and adolescent pelvic fractures. *Oper Tech Orthop* 1995;5:95–114.
21. Hall BB, Klassen RA, Ilstrup DM. Pelvic fractures in children: a long-term follow-up study. (Unpublished.)
22. Heeg M, de Ridder VA, Tornetta P III, et al. Acetabular fractures in children and adolescents. *Clin Orthop* 2000;376:80–86.
23. Heeg M, Klasen JH. Long-term outcome of sacroiliac disruptions in children. *J Pediatr Orthop* 1997;17:337–341.
24. Heeg M, Visser JD, Oostvogel HJM. Injuries of the acetabular triradiate cartilage and sacroiliac joint. *J Bone Joint Surg Br* 1988;70:34–37.
25. Heeg M, Klassen HJ, Visser JD. Acetabular fractures in children and adolescents. *J Bone Joint Surg Br* 1989;71:418–421.
26. Heinrich SD, Gallagher D, Harris M, et al. Undiagnosed fractures in severely injured children and young adults: identification with technetium imaging. *J Bone Joint Surg Am* 1994;76:561–572.
27. Ismail N, Bellemare JF, Mollitt DL, et al. Death from pelvic fracture: children are different. *J Pediatr Surg* 1996;31:82–85.
28. Judet R, Judet J, Letournel E. Fractures of the acetabulum: classification and surgical approaches for open reduction. *J Bone Joint Surg Am* 1964;46:1615–1646.
29. Keshishyan RA, Rozinov VM, Malakhov OA, et al. Pelvic polyfractures in children: radiographic diagnosis and treatment. *Clin Orthop* 1995;320:28–33.
30. Lane-O'Kelly A, Fogarty E, Dowling F. The pelvic fracture in childhood: a report supporting nonoperative management. *Injury* 1995;26:327–329.
31. Letournel E, Judet R. *Fractures of the acetabulum*. New York: Springer-Verlag, 1981.
32. Lynch SA, Renström PAFH. Groin injuries in sport: treatment strategies. *Sports Med* 1999;28:137–131.
33. McIntyre RC Jr, Bensard DD, Moore EE, et al. Pelvic fracture geometry predicts risk of life-threatening hemorrhage in children. *J Trauma* 1993;35:423–429.
34. McNeese MC, Hebler JR. *The abused child: a clinical approach to identification and management*. Clinical Symposia, Vol. 29(5). Summit, NJ: CIBA Pharmaceutical Company, 1977.
35. Magid D, Fishman EK, Ney DR, et al. Acetabular and pelvic fractures in the pediatric patient: value of two- and three-dimensional imaging. *J Pediatr Orthop* 1992;12:621–625.
36. Malgaigne JF. *Treatise on fractures*. Philadelphia: JB Lippincott, 1859.
37. Metzmaker JN, Pappas AM. Avulsion fractures of the pelvis. *Orthop Trans* 1980;4:52(abstr).
38. Milch H. Avulsion fracture of the tuberosity of the ischium. *J Bone Joint Surg* 1926;8:832–838.
39. Moreno C, Moore EE, Rosenberg A, et al. Hemorrhage associated with pelvic fracture: a multispecialty challenge. *J Trauma* 1986;26:987–991.
40. Mosheiff R, Suchar A, Porat S, et al. The “crushed open pelvis” in children. *Injury* 1999;30:S-B14–S-B18.
41. Musemeche CA, Fischer RP, Cotler HB, et al. Selective management of pediatric pelvic fractures: a conservative approach. *J Pediatr Surg* 1987;22:538–540.
42. Nierenberg G, Volpin G, Bialik V, et al. Pelvic fractures in children: a follow-up in 20 children treated conservatively. *J Pediatr Orthop B* 1993;1:140–142.
43. Ogden JA. *Skeletal injury in the child*, 2nd ed. Philadelphia: WB Saunders, 1990.
44. Pendergrast NC, deRoux SJ, Adsay NV. Non-accidental pediatric pelvic fracture: a case report. *Pediatr Radio*. 1998;28:344–346.
45. Peterson HA, Robertson RC. Premature partial closure of the triradiate cartilage treated with excision of a physeal osseous bar: case report with a fourteen-year follow-up. *J Bone Joint Surg Am* 1997;79:767–770.
46. Plaiser BR, Meldon SW, Super DM, et al. Improved outcome after early fixation of acetabular fractures. *Injury* 2000;31:81–84.
47. Poole GV, Ward EF, Griswold JA, et al. Complications of pelvic fracture from blunt trauma. *Am Surg* 1992;58:225–231.
48. Ponseti IV. Growth and development of the acetabulum in the normal child. *J Bone Joint Surg Am* 1978;60:575–585.
49. Quinby WC Jr. Fractures of the pelvis and associated injuries in children. *J Pediatr Surg* 1966;1:353–364.
50. Rang M. *Children's fractures*, 2nd ed. Philadelphia: JB Lippincott, 1983.
51. Reed MH. Pelvic fractures in children. *J Can Assoc Radio* 1976;27:255–261.
52. Rieger H Brug E. Fractures of the pelvis in children. *Clin Orthop* 1997;336:226–239.
53. Rosenberg N, Noiman M, Edleson G. Avulsion fractures of the anterior superior iliac spine in adolescents. *J Orthop Trauma* 1996;10:440–443.
54. Schwarz N, Posch E, Mayr J, et al. Long-term results of unstable pelvic ring fractures in children. *Injury* 1998;29:431–433.
55. Silber JS, Flynn JM. Changing patterns of pediatric pelvic fractures with skeletal maturation: implications for classification and management. Presented at the 67th Annual Meeting of the American Academy of Orthopaedic Surgeons, Orlando, Florida, March 16, 2000.
56. Stiletto RJ, Baacke M, Gotzen L. Comminuted pelvic ring disruption in toddlers: management of a rare injury. *J Trauma* 2000;48:161–164.
57. Sundar M, Carty H. Avulsion fractures of the pelvis in children: a report of 32 fractures and their outcome. *Skeletal Radio*. 1994;23:85–90.
58. Tile M. Pelvic fractures. *Orthop Clin North Am* 1980;11:423–464.
59. Tile M. *Fractures of the pelvis and acetabulum*. Baltimore: Williams & Wilkins, 1984.
60. Tolo VT. Orthopaedic treatment of fractures of the long bones and pelvis in children who have multiple injuries. *J Bone Joint Surg Am* 2000;82:272–280.
61. Torode I, Zieg D. Pelvic fractures in children. *J Pediatr Orthop* 1985;5:76–84.
62. Van Neck M. *Arch Provence Chir* 1924;238.
63. Vazquez WD, Garcia VF. Pediatric pelvic fractures combined with an additional skeletal injury is an indicator of significant injury. *Surg Gynecol Obstet* 1993;177:468–472.
64. Watts HG. Fractures of the pelvis in children. *Orthop Clin North Am* 1976;7:615–624.

## FRACTURES AND TRAUMATIC DISLOCATIONS OF THE HIP IN CHILDREN

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### Fractures of the HIP

#### Diagnosis

#### Classification

#### Unusual Fracture Patterns

#### Surgical and Applied Anatomy

#### Treatment

#### Surgical Procedures

#### Postoperative Fracture Care

#### Complications

#### Stress Fractures

### HIP Dislocations in Children

#### Diagnosis

#### Classification

#### Unusual Fracture Patterns Associated with Hip Dislocation

#### Surgical and Applied Anatomy

#### Treatment Options

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## FRACTURES OF THE HIP

Fractures of the head and neck of the femur in children are exceedingly rare, accounting for fewer than 1% of all pediatric fractures ( 24). In comparison, the prevalence of fractures of the hip in children is less than 1% of that in adults. Therefore, most orthopaedic surgeons will treat only a few such fractures in a lifetime (16).

The pattern of fracture and thus the classification in children differ from those in adults. Because of the weak proximal femoral physis, a transphyseal separation can occur in children. Transcervical and cervicotrochanteric fractures have an extremely high risk for avascular necrosis (AVN) and coxa vara compared with their adult counterparts. Intertrochanteric fractures are mechanically similar in both groups, although in children involvement of the greater trochanteric apophysis can result in premature closure.

The proximal femoral physis is at risk in hip fracture and has obvious implications for fracture care and prognosis. If the proximal physis is damaged, coxa vara or coxa breva may develop with further growth regardless of fracture alignment. Conversely, if the greater trochanter apophysis fuses prematurely as a result of trauma, coxa valga may develop (4).

Although hip fractures in children can generally be expected to heal, their importance lies in the frequency and severity of complications, including AVN, coxa vara, premature physeal closure, limb length discrepancy and occasionally nonunion. Because the hip is developing in the growing child, deformities can progress with age.

### Diagnosis

#### *Mechanism of Injury*

Hip fractures in children can be caused by axial loading, torsion, hyperabduction, or a direct blow to the hip. Almost all hip fractures in children are caused by severe, high-energy trauma. This is in marked contrast to hip fractures in the elderly, in whom minor torsional forces acting on osteoporotic bone cause most hip fractures. The proximal femur in children, except for the proximal femoral physis, is extremely strong, and high-energy forces, such as from moving vehicle accidents and high falls, are necessary to cause fracture (9).

#### *Examination*

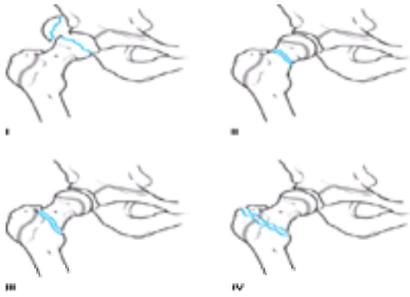
Clinical examination usually reveals pain in the hip and a shortened, externally rotated extremity. With a nondisplaced or stress fracture of the femoral neck, the patient may be able to bear weight with a limp and may only demonstrate hip or knee pain with extremes of range of motion, especially internal rotation. A good quality anteroposterior pelvic radiograph will provide a comparison view of the opposite hip if a displaced fracture is suspected. A cross-table lateral radiograph should be considered to avoid further displacement and unnecessary discomfort to the patient from an attempt at a frog-leg lateral view. Any break or offset of the bony trabeculae near Ward's triangle is evidence of a nondisplaced or impacted fracture.

Not all fractures can be detected on plain radiographs early. Special studies may be required to reveal an occult fracture. A radioisotopic bone scan 48 hours after the onset of symptoms may show increased uptake at the fracture site. The typical magnetic resonance imaging (MRI) appearance of a fracture is a linear black line (low signal) on all sequences surrounded by a high signal band of bone marrow edema and hemorrhage. The low signal represents trabecular impaction. MRI may detect an occult hip fracture within the first 24 hours after injury ( 17).

In a patient with posttraumatic hip pain without evidence of a fracture, other diagnoses must be considered, including synovitis, hemarthrosis, and infection. A complete blood count, erythrocyte sedimentation rate, C-reactive protein, and temperature are helpful. Ultrasonography can be used to detect fluid in the joint. If necessary, aspiration should be performed. A bloody aspirate establishes the diagnosis of fracture, whereas a serous or suppurative aspirate suggests synovitis or infection, respectively. In children under 5 years of age, developmental coxa vara can be confused with an old hip fracture ( 4).

### Classification

Pediatric hip fractures are classified by the method of Delbet. Its simplicity and uniformity allow accurate description and reporting of results of each fracture by type. Type I is a transepiphyseal separation, with (type IA) or without (type IB) dislocation of the femoral head from the acetabulum. Type II is a transcervical fracture. Type III is a cervicotrochanteric fracture. Type IV is an intertrochanteric fracture ( 8) (Fig. 21-1). Subtrochanteric fractures are not included in this classification and are discussed in [Chapter 22](#).



**FIGURE 21-1.** Delbet classification of hip fractures in children. *I*, transepiphyseal, with or without dislocation from the acetabulum; *II*, transcervical; *III*, cervicotrochanteric; and *IV*, intertrochanteric.

### **Type I**

Transphyseal fractures occur through the proximal femoral physis (Fig. 21-2). Such fractures are rare, constituting 8% of femoral neck fractures in children (16). True transphyseal fractures tend to occur in young children after high-energy trauma (5,12) and are different from unstable slipped capital femoral epiphyses of the preadolescent, which probably occur as a result of a subtle endocrinopathy. In the absence of a history of significant trauma in a young child, battered child syndrome should be suspected (28). Rarely, this injury occurs during a difficult delivery or attempted closed reduction of a traumatic dislocation of the hip in adolescents (16). Approximately half of type I fractures are associated with a dislocation of the capital femoral epiphysis. In such cases, the outcome is dismal because of AVN and premature physeal closure in virtually 100% of patients (5,12).



**FIGURE 21-2.** Type I transphyseal fracture of the left proximal femur in a 3-year-old patient with spina bifida. Superior translation of the metaphysis with the head remaining in the acetabulum is typical of a type IA fracture.

Type I fractures in children under 2 or 3 years of age have a better prognosis than in older children. AVN is unlikely, although coxa vara, coxa breva, and premature physeal closure can cause subsequent leg length discrepancy (18,23).

### **Type II**

Transcervical fractures account for 46% of fractures of the head and neck of the femur in children (16). In three large series, 77% of all type II fractures were displaced (16). Nondisplaced fractures have a better prognosis and a lower rate of AVN than displaced fractures, regardless of treatment (5,24). The risk of AVN is thought by most investigators to be directly related to the initial displacement of the fracture, although a few have hypothesized an intraarticular hemarthrosis with tamponade (5,16) as the etiology of vascular impairment.

### **Type III**

Cervicotrochanteric fractures are, by definition, located at or above the anterior intertrochanteric line and are the second most common type of hip fracture in children, comprising about 34% of fractures (16). The incidence of AVN is 20% to 30%, and, as in type II fractures, the risk of AVN is directly related to the degree of displacement at the time of injury (16). Premature physeal closure occurs in 25% of patients and coxa vara in 14% (16). Displaced type III fractures are similar to type II fractures in regard to the development of complications. Nondisplaced type III fractures have a much lower complication rate than displaced fractures.

### **Type IV**

Intertrochanteric fractures account for only 12% of fractures of the head and neck of the femur in children (16). This fracture has the lowest complication rate of all four types. Nonunion and AVN after this fracture are exceedingly rare. Coxa vara and premature physeal closure have rarely been reported (5,16,19,24,25).

### **Unusual Fracture Patterns**

Type I fracture in a neonate deserves special attention. This injury is exceedingly rare, and, because the femoral head is not visible on plain radiographs, the index of suspicion must be high. An affected infant holds the extremity flexed, abducted, and externally rotated. A strong suspicion, pseudoparalysis, and shortening are keys to the diagnosis. The differential diagnosis includes septic arthritis and hip dislocation. Plain radiographs may be of assistance, but ultrasonography is useful if the diagnosis remains in doubt. Plain radiographs may show a high riding proximal femoral metaphysis on the involved side, resembling a dislocation. Ultrasonography shows the cartilaginous head in the acetabulum with dissociation from the femoral shaft. The diagnosis can be missed if there is no history of trauma (such as in child abuse) or if there is an ipsilateral fracture of the femoral shaft (1).

Stress fractures are caused by repetitive injury and result in hip or knee pain and a limp. Pain associated with long distance running, marching, or a recent increase in physical activity is suggestive of stress fracture. Close scrutiny of high-quality radiographs may identify sclerosis, cortical thickening, or new bone formation. Undisplaced fractures may appear as faint radiolucencies (Fig. 21-3).



**FIGURE 21-3. A:** This 5-year-old boy jumped off his bunk bed and subsequently complained of right hip pain and limp. Anteroposterior radiography yielded normal findings. **B:** Careful examination of the frog-leg lateral radiograph revealed a nondisplaced femoral neck fracture. Symptoms resolved after 4 weeks in a spica cast.

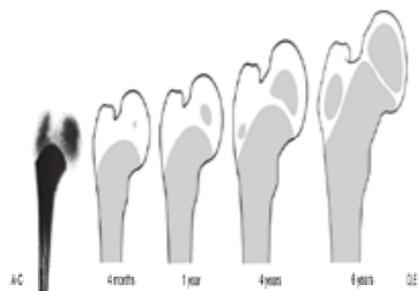
An acute unstable slipped capital femoral epiphysis may be confused with an acute type I fracture of the proximal femur; however, a slipped capital femoral epiphysis is caused by an underlying abnormality of the physis and occurs after trivial trauma, usually in preadolescents, whereas type I fractures usually occur in young children.

Fracture after minor trauma suggests weakened bone. Intrinsic bone disease, tumors, cysts, and infections may weaken the bone. If the trauma is significant, but the history is not consistent, nonaccidental trauma must always be considered ( 2,28).

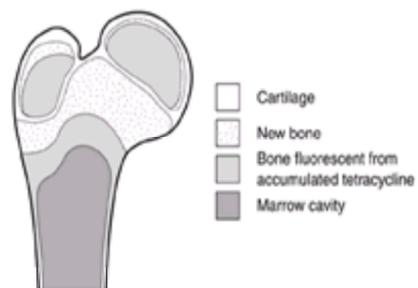
It is easy to miss hip fractures that are overshadowed by more dramatic or painful injuries. Radiographs of the proximal femur should be examined carefully in patients with femoral shaft fractures because ipsilateral fracture or dislocation of the hip is not unusual ( 1).

### Surgical and Applied Anatomy

Ossification of the femur begins in the 7th fetal week ( 12). In early childhood, only a single proximal femoral physis exists ( Fig. 21-4A and Fig. 21-4B). During the first year of life, the medial portion of this physis grows faster than the lateral, creating an elongated femoral neck by 1 year of age ( Fig. 21-4C). The capital femoral epiphysis begins to ossify at approximately 4 months in girls and 5 to 6 months in boys. The ossification center of the trochanteric apophysis appears at 4 years in boys and girls ( 16) ( Fig. 21-4D). The proximal femoral physis is responsible for the metaphyseal growth in the femoral neck, whereas the trochanteric apophysis contributes to the appositional growth of the greater trochanter and less to the metaphyseal growth of the femur ( 20) ( Fig. 21-5). Fusion of the proximal femoral and trochanteric physes occurs at about the age of 14 in girls and 16 in boys ( 15). The confluence of the greater trochanteric physis with the capital femoral physis along the superior femoral neck ( Fig. 21-4E) and the unique vascular supply to the capital femoral epiphysis make the immature hip vulnerable to growth derangement and subsequent deformity after a fracture.



**FIGURE 21-4.** The transformation of the preplate to separate growth zones for the femoral head and greater trochanter. The diagram shows development of the epiphyseal nucleus in the proximal end of the femur. **A:** Radiograph of the proximal end of the femur of a stillborn female, weight 325 g. **B–E:** Drawings made on the basis of radiographs. (Reprinted from Edgren W. Coxa plana. A clinical and radiological investigation with particular reference to the importance of the metaphyseal changes for the final shape of the proximal part of the femur. *Acta Orthop Scand* 1965;84(suppl):24; with permission.)

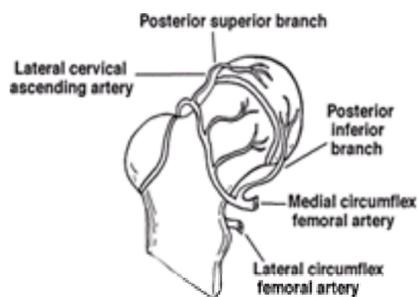


**FIGURE 21-5.** Drawing of a frontal section through the upper end of the femur of a 14-week-old pig injected 8 weeks previously with tetracycline (50 mg/kg). Diagram depicts the contribution of the capital femoral physis and greater trochanteric physis to new bone formation. (Reprinted from Edgren W. Coxa plana. A clinical and radiological investigation with particular reference to the importance of the metaphyseal changes for the final shape of the proximal part of the femur. *Acta Orthop Scand* 1965;84(suppl):24; with permission.)

### Vascular Anatomy

Because of the frequency and sequelae of AVN of the hip in children, the blood supply has been studied extensively ( 7,23,29,30). Postmortem injection and microangiographic studies have provided clues to the vascular changes with age. These observations are enumerated as follows:

1. The vessels of the ligamentum teres are of virtually no importance. They contribute little blood supply to the femoral head until age 8, and then only about 20% as an adult.
2. At birth, the branches of the medial and lateral circumflex arteries (metaphyseal vessels) traversing the femoral neck predominately supply the femoral head. These arteries gradually diminish in size as the cartilaginous physis develops and forms a barrier that prevents penetration of these vessels into the femoral head. This metaphyseal blood supply is virtually nonexistent by age 4.
3. When the metaphyseal vessels diminish, the lateral epiphyseal vessels predominate and the femoral head is primarily supplied by these vessels, which bypass the physal barrier.
4. Ogden noted that the lateral epiphyseal vessels consist of two branches, the posterosuperior and posteroinferior branches of the medial circumflex artery ( Fig. 21-6). At the level of the intertrochanteric groove, the medial circumflex artery branches into a retinacular arterial system (the posterosuperior and posteroinferior arteries). These arteries penetrate the capsule and traverse proximally (covered by the retinacular folds) along the neck of the femur to supply the femoral head peripherally and proximally to the physis.



**FIGURE 21-6.** Arterial supply of the proximal femur. The capital femoral epiphysis and physis are supplied by the medial circumflex artery through two retinacular vessel systems: the posterosuperior and posteroinferior. The lateral circumflex artery supplies the greater trochanter and the lateral portion of the proximal femoral physis and a small area of the anteromedial metaphysis.

5. Capsulotomy does not damage the blood supply to the femoral head, but violation of the intertrochanteric notch or the lateral ascending cervical vessels may render the head avascular.
6. At about 3 to 4 years of age, the lateral posterosuperior vessels appear to predominate and supply the entire anterior lateral portion of the capital femoral epiphysis.
7. The posteroinferior and posterosuperior arteries persist throughout life and supply the femoral head.
8. The multiple small vessels of the young coalesce with age to a limited number of larger vessels. As a result, damage to a single vessel can have serious consequences; for example, occlusion of the posterosuperior branch of the medial circumflex artery can cause AVN of the anterior lateral portion of the femoral head (4).

### Soft Tissue Anatomy

The hip joint is enclosed by a thick fibrous capsule. Tense hemarthrosis after intracapsular fracture may tamponade the ascending cervical vessels and may have implications in the development of AVN. The hip joint is surrounded on all sides by a protective cuff of musculature. Open hip fracture is rare. In the absence of associated hip dislocation, neurovascular injuries are rare after hip fracture and are more likely to occur during surgery.

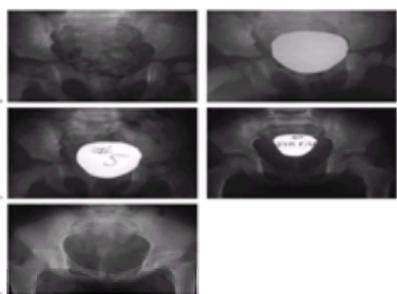
The lateral femoral cutaneous nerve lies in the interval between the tensor and sartorius muscles and supplies sensation to the lateral thigh. This nerve must be identified and preserved during an anterolateral approach to the hip. The femoral neurovascular bundle is separated from the anterior hip joint by the iliopsoas. Thus, any retractor placed on the anterior acetabular rim should be carefully placed deep to the iliopsoas to protect the femoral bundle. Inferior and medial to the hip capsule, coursing from the deep femoral artery toward the posterior hip joint, is the medial femoral circumflex artery. Placement of a distal Hohmann retractor too deeply can tear this artery, and control of the bleeding may be difficult.

The sciatic nerve emerges from the sciatic notch beneath the piriformis and courses superficial to the external rotators and the quadratus medial to the greater trochanter. The nerve is rarely seen at hip fracture surgery, but placement of a Hohmann retractor dorsally and distally, simultaneous with external rotation of the leg, can damage it.

### Treatment

#### Type I

Fracture treatment is based on the age of the child and fracture stability after reduction. In toddlers under 2 years of age with nondisplaced or minimally displaced fractures, simple spica cast immobilization is likely to be successful. Because the fracture tends to displace into varus and external rotation, the limb should be casted in mild abduction and neutral rotation to prevent displacement. Displaced fractures in toddlers should be reduced closed by gentle traction, abduction, and internal rotation. If the fracture “locks on” and is stable, casting without fixation is indicated. If the fracture is not stable, it should be fixed with small smooth pins that access the femoral neck and cross the physis. If casting without fixation is done, repeat radiographs should be taken within days to look for displacement because the likelihood of successful repeat reduction decreases rapidly with time and healing in a young child ( Fig. 21-7).



**FIGURE 21-7.** **A:** A 16-month-old boy with a type I fracture of the left hip. Note widening of the left femoral physis. **B:** Lateral radiograph at injury. Instability of the physis is apparent. **C:** Anteroposterior radiograph after 1 month in spica cast. **D:** At 2 years postinjury, the patient has mild coxa valga and coxa breva. **E:** At 10 years postinjury, coxa breva secondary to premature physeal closure is evident. Note the overgrowth of the greater trochanter.

Older children should always have operative fixation even if the fracture is undisplaced because the complications of late displacement may be great. Smooth pins can be used in young children, but cannulated screws are better for older, larger children. Fixation should cross the physis into the capital femoral epiphysis. Irreducibility mandates an open reduction and internal fixation. Postoperative spica cast immobilization is mandatory in all but the oldest and most reliable adolescents. Fixation may be removed shortly after fracture healing (8–12 weeks) to enable further growth in younger patients.

Closed reduction of type IB fracture–dislocations should be attempted, with immediate open reduction if unsuccessful. Internal fixation is mandatory. The surgical approach should be from the side to which the head is dislocated, generally posterolateral. Parents must be advised in advance about the risk of AVN.

#### Type II and Type III

Displaced neck fractures should always be treated with anatomic reduction and stable internal fixation to minimize the risk of late complications. In two large series, the prevalences of coxa vara and nonunion were high in displaced transcervical fractures treated with immobilization but without internal fixation ( 5,19). Much lower prevalences of these two complications have been documented in patients treated with anatomic closed or open reduction and internal fixation ( 5).

Internal fixation is also recommended by most investigators for nondisplaced transcervical fractures ( 16), because the risk of late displacement in such fractures far outweighs the risk of percutaneous screw fixation, especially in young children ( 3). Nondisplaced type II fractures in children under 5 years of age can be managed with spica casting and close follow-up ( 10,19), but fixation remains preferable. Even then, close follow-up is necessary to prevent varus displacement in the cast.

Gentle closed reduction of displaced fractures is accomplished with the use of longitudinal traction, abduction, and internal rotation. Open reduction is frequently

necessary for displaced fractures and should be performed via a Watson-Jones surgical approach.

Internal fixation with cannulated screws is performed through a small lateral incision. Three screws should be placed if possible. One screw should be placed low along the calcar, and two above, spaced as widely as possible (3). Occasionally, the small size of the child's femoral neck will accommodate only two screws. Care should be taken to minimize drill holes in the subtrochanteric region because they increase the risk of subtrochanteric fracture. If possible, screws should be inserted short of the physis in type III fractures; however, if physeal penetration is necessary for purchase, it must be done ( Fig. 21-8 and Fig. 21-9). The risks of premature physeal closure and trochanteric overgrowth are much less than those of nonunion, pin breakage, and AVN. Treatment of the fracture is the first priority, and any subsequent growth disturbance and leg length discrepancy (LLD) are secondary.

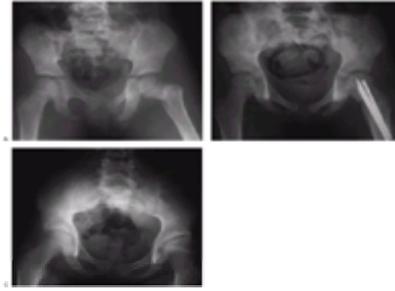


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**FIGURE 21-8. A:** A girl 4 years and 4 months of age with a type II fracture of the left femoral neck. Anteroposterior pelvis film at presentation shows a displaced femoral neck fracture. **B:** Three weeks postinjury. Smooth pins were chosen to minimize the risk of physeal damage. **C:** Six years after fracture. Mild coxa valga is evident, possibly from damage to the growth zone on the superior femoral neck in this young child.



**FIGURE 21-9. A:** This boy 4 years and 9 months of age sustained a type III femoral neck fracture in a motor vehicle accident. **B:** Lateral radiograph at presentation. **C:** Three months after anatomic open reduction and internal fixation with two screws, sparing the physis. **D:** Lateral radiograph on the same date.

Nondisplaced cervicotrochanteric fractures can be treated adequately in an abduction one and one-half spica cast with close follow-up (16). Displaced cervicotrochanteric fractures have been shown to have a complication rate similar to that for type II fractures and should be treated similarly. Fixation generally does not need to cross the physis in type III fractures.

In a more distal cervicotrochanteric fracture, especially in a child over 5 years of age, a pediatric hip compression screw can be used for more secure fixation. Consideration may be given to capsulotomy or aspiration of the joint to eliminate tense hemarthrosis at the time of surgery. Spica casting is routine, except in older reliable children. Hardware removal at 6 to 12 months after fracture union will avoid bony overgrowth of the hardware.

**Type IV**

Good results can be expected after closed treatment of most intertrochanteric fractures, regardless of displacement. Traction and spica cast immobilization are effective (16). Failure to maintain adequate reduction and polytrauma are indications for internal fixation. Children old enough to use crutches or those with multiple injuries can be treated with open reduction and internal fixation ( Fig. 21-10 and Fig. 21-11). A pediatric hip screw provides the most rigid internal fixation for this purpose.



**FIGURE 21-10. A:** A girl 3 years and 7 months of age with type IV intertrochanteric right femur fracture. **B:** Three months after fixation with a pediatric sliding hip screw.



**FIGURE 21-11. A:** A 14-year-old boy who fell from a tree swing sustained this nondisplaced left intertrochanteric hip fracture. **B:** Lateral radiograph shows the long

spiral fracture line. **C:** Three months postfixation with an adult sliding hip screw.

## Surgical Procedures

When an operation is indicated, several factors must be evaluated before the method of internal fixation and the operative procedure are selected ( [Table 21-1](#)). Perhaps the most important consideration is the age of the patient. For discussion, three age-groups have been established arbitrarily: infantile (younger than three years), juvenile (three to eight years), and adolescent (older than eight years).

Anterolateral approach  
Age 0–3 yr—smooth pins, 5/64-inch or 3/32-inch  
Age 3–8 yr—cannulated 4.5-mm screws  
Age 8+ yr—6.5 or 7.3-mm cannulated screws  
Type IV fractures  
<8 yr—pedi hip compression screw  
>8 yr—adult hip compression screw  
Always predrill and tap before inserting screws  
Avoid crossing physis if possible, but cross physis if necessary for stability  
Age <10 yr—hip spica for 6–12 wk

**TABLE 21-1. SURGICAL TIPS AND PEARLS FOR FRACTURES IN CHILDREN**

For younger and smaller patients, the operation should be done on a radiolucent operating table rather than on a fracture table, which is more appropriate for older and larger adolescents.

For internal fixation of types I, II, and III fractures of the femoral neck, smooth pins may be used in infants; cannulated 4.0-millimeter screws, in children; and cannulated 6.5-millimeter screws, in adolescents. For fixation of type-IV fractures, pediatric-size hip-compression screws should be used in children and adult-size hip-compression screws, in adolescents.

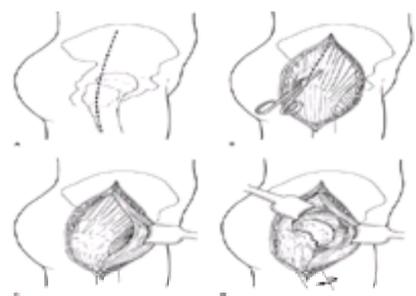
A hip-spica cast should supplement internal fixation in all patients who are less than ten years old. Fractures of the neck of the femur in children who are twelve years old or more are treated in a manner similar to that used in adults: no postoperative cast is used and early walking with crutches is encouraged. For patients who are between the ages of ten and twelve years, the necessity for a postoperative cast is less clear-cut. If stability of the fracture fixation is questionable, or if compliance of the patient is doubtful, a hip-spica cast should be used.

Because the femoral bone in children is harder than the osteoporotic bone in elderly patients, pre-drilling and pre-tapping are necessary for insertion of all screws.

Finally, growth of the femur and the contribution of the proximal femoral physis are important; however, this physeal contribution to growth is only 13 per cent of the entire extremity, or three to four millimeters per year on the average. Once the decision for internal fixation of a fracture of the head or neck of the femur is made, stable fixation of the fracture is a higher priority than preservation of the physis. If stability is questionable, the internal fixation device should extend into the femoral head for rigid, stable fixation, regardless of the type of fracture or the age of the child.

### Anterolateral Approach

If closed reduction is successful, a 5- to 4.5-cm lateral incision is made distal to the greater trochanter apophysis for insertion of pins or cannulated screws. An anterior approach through the Watson-Jones interval is often used for open reduction. A lateral incision is made over the proximal femur, slightly anterior to the greater trochanter ([Fig. 21-12A](#)). The fascia lata is incised longitudinally ([Fig. 21-12B](#)). The innervation of the tensor muscle by the superior gluteal nerve is 2 to 5 cm above the greater trochanter and care should be taken not to damage this innervation. The tensor muscle is reflected anteriorly. The interval between the gluteus medius and the tensor muscles will be used ([Fig. 21-12C](#)). The plane is developed between the muscles and the underlying hip capsule. If necessary, the anteriormost fibers of the gluteus medius tendon can be detached from the trochanter for wider exposure. After clearing the anterior hip capsule, longitudinal capsulotomy is made along the anterosuperior femoral neck. A transverse incision may be added superiorly for wider visualization ([Fig. 21-12D](#)).



**FIGURE 21-12.** Watson-Jones lateral approach to the hip joint for open reduction of femoral neck fractures in children. **A:** Skin incision. **B:** Interval between gluteus medius and tensor fasciae. **C:** Dissection carried proximally. **D:** Completed exposure.

Alternatively, a bikini approach can be used through the Smith-Petersen interval. Care should be taken to identify and protect the lateral femoral cutaneous nerve. The sartorius and rectus muscles can be detached to expose the hip capsule. Medial and inferior retractors should be carefully placed to avoid damage to the femoral neurovascular bundle and medial femoral circumflex artery, respectively. Care must be taken not to violate the intertrochanteric notch and the lateral ascending vessels.

Under direct vision, the fracture is reduced and guidewires are passed from the lateral aspect of the proximal femur up the neck perpendicular to the fracture. Wires are passed either through the incision or percutaneously if a bikini approach is used. Smooth wires can be used as definitive fixation in toddlers or as guides for drilling if cannulated screws are used. The choice of internal fixation should consider the child's size and age. For internal fixation of types I, II, and III fractures, smooth pins are appropriate in children under 3 years of age, cannulated 4.5-mm screws in children 3 to 8 years of age, and 6.5-mm cannulated screws in children over 8 years of age. For fixation of type IV fractures, simple screw fixation is inappropriate. A pediatric-size hip compression screw is appropriate in patients under 8 years of age, and an adult-size hip compression screw may be used in older children and adolescents. Because the femoral neck in children is denser and harder than the osteoporotic bone in elderly patients, predrilling and tapping are necessary before the insertion of screws.

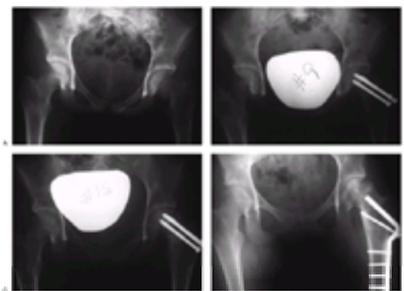
### Postoperative Fracture Care

Hip spica casting is used after internal fixation in most patients under 10 years of age. The cast should remain in place for 6 to 12 weeks depending on age. For children over 12 years of age, no postoperative cast is used, and early walking with crutches is encouraged, as in adults. For children 10 to 12 years of age, the use of a postoperative cast depends on the stability of fracture fixation and the patient's compliance. If either is in doubt, a single hip spica cast is used. Formal rehabilitation usually is unnecessary unless there is a severe persistent limp, which may be due to abductor weakness. Stiffness rarely is a problem in the absence of AVN.

## Complications

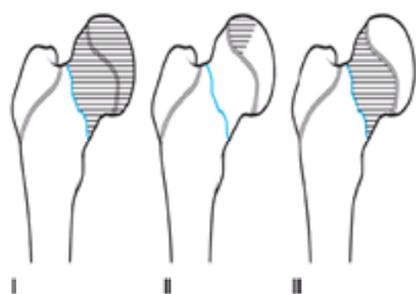
### Avascular Necrosis

Avascular necrosis is the most serious and frequent complication of hip fractures in children. Its overall prevalence is approximately 30% based on nine series in the literature (6,16,22). It is the primary cause of poor results after fractures of the hip in children. The risk of AVN is related to the extent of initial displacement of the fracture and to the damage to the blood supply at injury. The risk of AVN is highest after displaced type IB, type II, and type III fractures (16) (Fig. 21-13). Although prompt reduction of displaced fractures may be of some benefit, its worth has not been proven. Increased intraarticular pressure caused by fracture hematoma may be related to AVN after intracapsular fracture, and evacuation of this hemothrosis may decrease the AVN rate (16,22,27). Aspirating the hematoma from the hip capsule may decrease the intracapsular pressure and increase blood flow to the femoral head (6,22) or may have no effect (16,21). If a child is going to have an anesthetic for treatment of a fracture, aspiration of the hematoma can easily be accomplished. Open reduction results in capsular evacuation at capsulotomy, or a small capsulotomy can be made if a straight lateral incision is used after unsuccessful closed reduction.



**FIGURE 21-13.** A: A 14-year-old girl with a type II fracture of the left femoral neck. B: After fixation with three cannulated screws. C: Seven months after injury. Avascular necrosis with collapse of the superolateral portion of the femoral head. D: After treatment with valgus osteotomy.

Avascular necrosis has been classified by Ratliff as follows: type I, involvement of the whole head; type II, partial involvement of the head; and type III, an area of AVN from the fracture line to the physis (24) (Fig. 21-14). Type I is the most severe and most common form and has the poorest prognosis. Type I probably results from damage to all of the lateral epiphyseal vessels, type II from localized damage to one or more of the lateral epiphyseal vessels near their insertion into the anterolateral aspect of the femoral head, and type III from damage to the superior metaphyseal vessels. Type III is rare but has a good prognosis (24).



**FIGURE 21-14.** Three types of avascular necrosis. (Reprinted from Ratliff AHC. Fractures of the neck of the femur in children. *J Bone Joint Surg [Br]* 1962;44:528; with permission.)

Avascular necrosis causes pain and limitation of motion. As early as 6 weeks after injury, plain radiographs may reveal decreased density of the femoral head with widening of the joint space. Fragmentation and collapse of the femoral head occur late. Technetium bone scanning with pinhole collimation may show decreased uptake in the involved femoral head early in the course of AVN. With revascularization, changes may be variable. Signs and symptoms of AVN usually develop within the first year after injury, but sometimes as late as 2 years (16,25). Patients should be followed with plain radiographs for at least 2 years after fracture to rule out late onset of AVN. MRI reveals AVN within a few days of injury (see the subsection on [Avascular Necrosis](#) later under Hip Dislocations in Children). If MRI does not reveal AVN within 6 weeks of injury, it is unlikely to develop. The long-term results of AVN are poor in over 60% of patients (5,10,13). There is no clearly effective treatment for posttraumatic AVN in children (16,25). Older children (>10 years of age) tend to have worse outcomes than younger children. Ongoing investigative research includes the role of core decompression, vascularized fibular grafting, and the trapdoor procedure. Results of the procedures in few reported patients must ultimately be compared with the natural healing of untreated AVN. Remodeling can occur over many years and is more likely in younger children than in older ones. Degenerative arthritis in older children is generally irreversible. Valgus intertrochanteric osteotomy may improve coxa vara and leg length discrepancy if there is reasonable congruence in adduction of the hip preoperatively (Fig. 21-15).



**FIGURE 21-15.** A: A girl 9 years and 4 months of age with Ratliff type I avascular necrosis of the femoral head. B: Lateral radiograph on the same date. The patient has had a previous acetabular osteotomy for containment. C: Two years after valgus osteotomy. D: Lateral radiograph on same date. Note that some remodeling of the femoral head has occurred.

### Coxa Vara

The prevalence of coxa vara has been reported to be approximately 20% to 30% in nine series (16), although it is significantly lower in series in which internal fixation was used after reduction of displaced fractures (5). Coxa vara may be caused by malunion, AVN, premature physal closure, or a combination of these problems (Fig. 21-16). Severe coxa vara raises the greater trochanter in relation to the femoral head, causing shortening of the extremity and mechanical disadvantage of the abductors. The result is an abductor lurch. If the child is over 8 years of age, the neck shaft angle is 110 degrees or less, and coxa vara has been persistent for more than 2 years, subtrochanteric valgus osteotomy may be considered to restore limb length and abductor strength (16).



**FIGURE 21-16.** **A:** A 12-year-old boy with a type III left hip fracture. Poor pin placement and varus malposition are evident. **B:** The fracture united in mild varus after hardware revision. **C:** Fourteen months after injury. Collapse of the weight-bearing segment is evident (Ratliff type II avascular necrosis). **D:** Six years postinjury. Coxa breva and trochanteric overgrowth are seen secondary to avascular necrosis, malunion, and premature physal closure.

### Premature Physal Closure

Premature physal closure has occurred after approximately 28% of fractures (16). The risk of premature physal closure increases with penetration by fixation devices or when AVN is present. It is most frequent in patients who have type II or III AVN (24,25) (Fig. 21-16).

The capital femoral physis contributes only 13% of the growth of the entire extremity and normally closes earlier than most of the other physes in the lower extremity. As a result, shortening due to premature physal closure is not significant except in very young children (16,18). Treatment for leg length discrepancy is only indicated for significant discrepancy (2.5 cm or more projected at maturity) (16). Rarely, trochanteric epiphysiodesis may be used in progressive coxa vara.

### Nonunion

Nonunion occurs infrequently, with an overall incidence of 7% of hip fractures in children (16). Nonunion is a complication of femoral neck fracture and is not generally seen after type I or type IV fractures. The primary cause of nonunion is failure to obtain or maintain an anatomic reduction (5,16). After femoral neck fracture in a child, pain should be gone and bridging new bone should be seen at the fracture site by 3 months after injury. A computed tomography (CT) scan may be helpful to look for bridging bone. If no or minimal healing is seen by 3 months, the diagnosis of nonunion is established. Nonunion should be treated operatively as soon as possible. Either rigid internal fixation or subtrochanteric valgus osteotomy should be performed to allow compression across the fracture (Fig. 21-17). Because the approach necessary for bone grafting is extensive, it should be reserved for recalcitrant cases. Internal fixation should extend across the site of the nonunion, and spica cast immobilization should be used in all but the most mature and cooperative adolescents.



**FIGURE 21-17.** **A:** A 15-year-old girl with a markedly displaced type II femoral neck fracture. **B:** She underwent open reduction and internal fixation with two 7.3-mm cannulated screws and one 4.5-mm cannulated screw. Primary bone grafting of a large defect in the superior neck also was performed. **C:** Radiograph at 5 months showing a persistent fracture line. **D:** Six weeks after valgus intertrochanteric osteotomy. The fracture is healing.

### Other Complications

Infection is uncommon after hip fractures in children. The reported incidence of 1% (5,19,24) is consistent with the expected infection rate in any closed fracture treated surgically with open reduction and internal fixation.

Chondrolysis is exceedingly rare and has been reported only in one series at a rate of 50% (13). Care must be taken to avoid persistent placement of hardware into the joint, which can cause this condition.

### Stress Fractures

Stress fractures of the femoral neck are unusual in children. Only 13 cases have been reported in the English-language literature. The rarity of such fractures underscores the need for a high index of suspicion when a child has unexplained hip pain, because early diagnosis and treatment are essential to avoid complete fracture with displacement.

### Mechanism

Stress fractures of the femoral neck in children result from repetitive cyclic loading of the hip, such as that produced by a new or increased activity. A recent increase in the repetitive activity is highly suggestive of the diagnosis. Long distance running, trampoline use, and scooter use are examples of such activities. Underlying metabolic disorders that weaken the bone may predispose to stress fracture. In adolescent female athletes, amenorrhea, anorexia nervosa, and osteoporosis have been implicated in the development of stress fractures of the femoral neck (14).

The usual presentation is that of progressive hip or groin pain with or without a limp. The pain may be perceived in the thigh or knee, and the pain may not be so severe as to preclude the offending activity. In the absence of displacement, examination typically reveals slight limitation of hip motion with increased pain, especially with internal rotation. Usually, plain radiographs reveal the fracture, but in the first 4 to 6 weeks after presentation, plain films may be negative. If there are no changes

or only linear sclerosis, bone scan will help identify the fracture. MRI has been documented as a sensitive test for undisplaced fractures of the femoral neck because impaction of the bony trabeculae appears on both T1 and T2 images as a linear black signal ( 21). If a sclerotic lesion is seen on plain radiographs, the differential diagnosis should include osteoid osteoma, chronic sclerosing osteomyelitis, bone infarct, and osteosarcoma. Other causes of hip pain, including slipped capital femoral epiphysis, Legg-Calve-Perthes disease, infection, avulsion injuries of the pelvis, and eosinophilic granuloma should be considered. Stress fracture unrelieved by rest or treatment may progress with activity to complete fracture with displacement ( 26). For this reason, prompt diagnosis and treatment are important.

### Classification

Devas classified femoral neck stress fractures into two types: compression fractures and tension fractures. The compression type appears as reactive bone formation on the inferior cortex without cortical disruption. This type rarely becomes completely displaced but may collapse into a mild varus deformity ( 11). Compression types have recently been reported to progress to complete fracture without early treatment ( 26). The tension type is a transverse fracture line appearing on the superior portion of the femoral neck (11). This type is inherently unstable because the fracture line is perpendicular to the lines of tension and fractures heal best under compression. Tension stress fractures have not been reported in children but may occur in the skeletally mature teenager ( 26).

### Treatment

Compression type fractures generally can be treated with a period of non-weight bearing on crutches. Partial weight bearing can be allowed at 6 weeks and full weight bearing at 12 weeks if pain is resolved and there is radiographic evidence of healing. In small or uncooperative children, spica casting may be necessary. Displacement into varus, however minimal, mandates internal fixation. Tension fractures are at high risk for displacement and should be treated with *in situ* compression fixation using cannulated screws ( Fig. 21-18).



**FIGURE 21-18.** Treatment algorithm for hip pain in children and adolescents with a history and physical examination compatible with a stress fracture. (Reprinted from St. Pierre P, Staheli LT, Smith JB, et al. Femoral neck stress fractures in children and adolescents. *J Pediatr Orthop* 1995;15:470–473.

### Complications

Coxa vara is the most common complication of untreated compression type fractures. Acute displacement of this type also has been described. Once displaced, the stress fracture is subject to all the complications of type II and type III displaced femoral neck fractures.

## HIP DISLOCATIONS IN CHILDREN

Hip dislocations in children are relatively uncommon. They can occur in young children under age 5 as a result of seemingly trivial trauma ( 32,37,43,46,51). The child's acetabulum at that age is primarily soft pliable cartilage, and there is generalized ligamentous laxity that allows hip dislocation. Dislocations in older children usually require significant trauma because the acetabulum is bony and less resilient and the ligaments are stiffer ( 37,43,46,51). Most hip dislocations in children can be reduced easily and will heal satisfactorily ( 46,51), although the possibility of late problems exists ( 50). Difficulties after hip dislocation can include neurovascular injury, concomitant fracture, irreducibility, nonconcentric reduction, AVN, coxa magna, arthrosis, and recurrent dislocation.

### Diagnosis

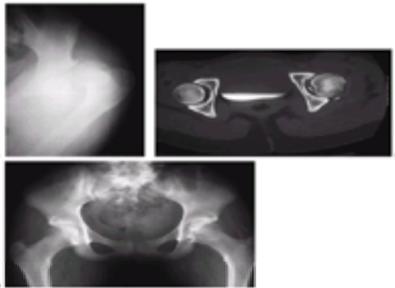
The mechanism of injury dictates the direction of hip dislocation. Posterior dislocations are the most common ( 32,50,51) and generally occur when a force is applied to the leg with the hip flexed. Anterior dislocations generally occur through a combination of external rotation and abduction.

The affected child has pain and inability to ambulate. Children sometimes feel the pain in the knee rather than in the hip ( Fig. 21-19). The hallmark of the clinical diagnosis of dislocation of the hip is abnormal positioning of the limb, which is not seen in fracture of the femur. With posterior dislocation, the thigh tends to be flexed, adducted, and internally rotated. The greater trochanter is proximal to its normal position, and the femoral head is often palpable in the gluteal region. If the hip is dislocated anteriorly, the extremity is generally extended, abducted, and externally rotated. Posterior dislocations of the femoral head can damage the sciatic nerve, and function of this nerve should be specifically tested after injury. Anterior dislocations can damage the femoral neurovascular bundle, and femoral nerve function and perfusion of the limb should be assessed.



**FIGURE 21-19.** **A:** Not all children have severe pain with dislocation. An 8-year-old complained of pain and had difficulty walking after wrestling. Because of knee pain, a knee immobilizer was placed at an outside facility. **B:** The leg length discrepancy had gone unnoticed. **C:** The thigh was markedly shortened on the dislocated right side. **D:** Closed reduction was easily achieved under anesthesia. Thigh length was restored. She made an unremarkable recovery.

Plain radiographs usually confirm the diagnosis. Radiographs should be examined for fracture of the acetabular rim and proximal femur, which may be associated with dislocation. Ipsilateral femoral fracture has been described in a few patients ( 50). CT scanning is useful for evaluating the acetabulum and may be useful in localizing intraarticular bony fragments after reduction ( 44) ( Fig. 21-20). The identification of nonbony fragments is difficult by CT without the use of concomitant arthrography ( 44). MRI is useful for evaluating soft tissues that may be interposed between the femoral head and acetabulum. MRI is especially helpful in nonconcentric reductions when the initial direction of dislocation is unknown. Soft tissue injury will dictate the surgical approach.

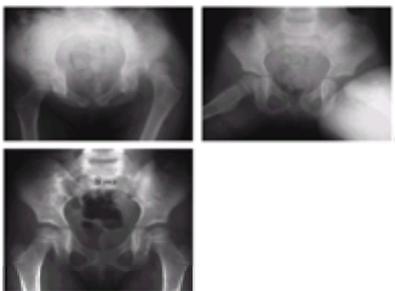


**FIGURE 21-20. A:** A girl 13 years and 11 months of age sustained a left posterior hip dislocation in a motor vehicle accident. **B:** Computed tomography scan after reduction showed intraarticular bony fragments. **C:** At open reduction and capsulorrhaphy, the bony fragments were removed. Suture anchors were used to reattach capsule to bone. Ten months postinjury, there is no sign of avascular necrosis. Heterotopic ossification is seen.

Spontaneous reduction may occur after hip dislocation (47), and the diagnosis will be missed if it is not considered. The presence of air in the hip joint, which may be detectable on CT scan of the pelvis, is evidence that a hip dislocation has occurred (36). Dislocation and spontaneous reduction with interposed tissue can occur and lead to late arthropathy if untreated (47). Widening of the joint space on plain radiographs suggests the diagnosis. In patients with hip pain, a history of trauma, and widening of the joint space, consideration should be given to MRI or arthrography to rule out dislocation with spontaneous relocation incarcerating soft tissue. If incarcerated soft tissues or osseous cartilage fragments are found, open reduction is required to obtain concentric reduction of the hip.

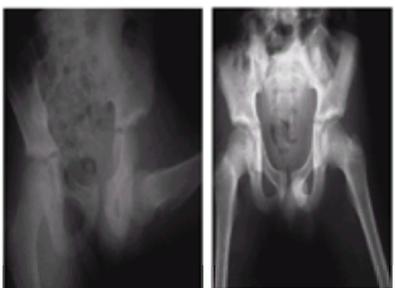
### Classification

Hip dislocations in children generally are classified as anterior or posterior depending on where the femoral head lies after dislocation. Posterior dislocations are much more common than anterior dislocations and tend to occur as a result of an axial force on the femur applied toward the hip with the hip in flexion. Dashboard injury is a frequent cause. The limb assumes a position of shortening, internal rotation, and adduction (Fig. 21-21).



**FIGURE 21-21. A:** A girl 4 years and 7 months of age presented with a posterior dislocation of the left hip. **B:** Frog-leg lateral radiograph at injury is shown. **C:** Eight months after successful closed reduction, radiographic appearance is normal.

Anterior dislocations can occur superiorly or inferiorly and result from forced abduction and external rotation. In extension, the hip tends to dislocate anteriorly and superiorly. The limb appears shortened, the thigh is positioned in external rotation and extension and the femoral head is palpable in the groin. If the hip dislocates with the leg flexed, the femoral head tends to dislocate inferiorly. The leg is held in abduction, external rotation, and flexion, and the femoral head is palpable near the obturator foramen (Fig. 21-22).

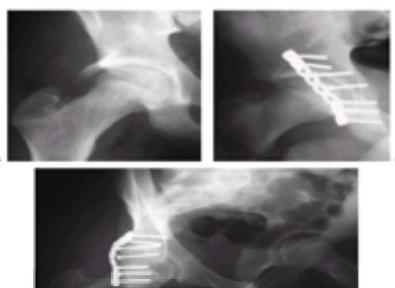


**FIGURE 21-22. A:** An 11-year-old girl sustained anterior inferior dislocation of the hip. **B:** Immediate closed reduction was concentric.

Fracture–dislocation of the hip involving the femoral head or the acetabulum is much more unusual in children than in adults. Older adolescents may sustain adult-type fracture–dislocations of the hip, and these are best classified by the methods of Thompson and Epstein and of Pipkin.

### Unusual Fracture Patterns Associated with Hip Dislocation

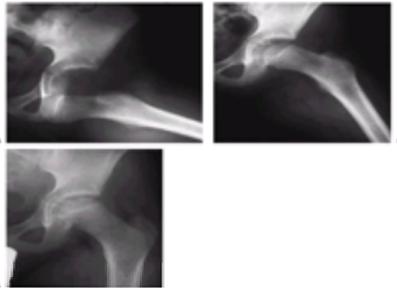
There are several pitfalls in the diagnosis of hip dislocations in children. It is always important to look for associated fractures. In older children, it is important to evaluate the posterior rim of the acetabulum after posterior dislocation to rule out fracture (Fig. 21-23). Fractures at other sites in the femur must be considered. It is important to obtain radiographs that show the entire femur to rule out ipsilateral fracture. Fractures of the femoral head are distinctly unusual in children, but separation of the capital femoral epiphysis and femoral neck fracture have been reported in association with dislocation of the hip.



**FIGURE 21-23. A:** A 12-year-old boy was tackled from behind in football. The right hip was dislocated. Reduction was easily achieved, but the hip was unstable posteriorly as a result of fracture of the posterior rim of the acetabulum. **B:** The fracture and capsule were fixed via a posterior approach. **C:** Oblique view shows reconstitution of the posterior rim.

Another pitfall is the possibility of spontaneous relocation of a dislocation of the hip. Failure to appreciate the presence of hip dislocation may lead to inadequate treatment. If soft tissue has been interposed in the hip joint, chronic arthropathy may result. In a child with posttraumatic hip pain without obvious deformity, the possibility of dislocation–relocation must be considered. Radiographs should be obtained to rule out joint space widening and undisplaced fracture.

Another consideration after reduction of hip dislocations is interposed tissue. After reduction, hemarthrosis may initially cause the hip joint to appear slightly wide ( 49). With time, the hip should seat and the increased iliofemoral distance should subside ( Fig. 21-24). If it fails to appear concentric after a few days, the possibility of interposed soft tissue must be considered (39,43,47,52).



**FIGURE 21-24. A:** A boy 10 years and 3 months of age sustained an anterior dislocation of the hip in football. **B:** The hip was easily reduced. The joint space was initially widened, probably due to hemarthrosis but normalized in a few days. **C:** At 4 months postinjury, the radiographic appearance was normal.

### Surgical and Applied Anatomy

The hip joint is highly specialized. Although capable of bearing body weight, the hip still provides a tremendous range of motion, surpassed only by the range of motion of the shoulder. The architecture of the hip joint is highly specialized and is centered on the spherical femoral head, which resides in the bony acetabulum. The relatively narrow femoral neck increases the range of motion possible at the hip joint in flexion, extension, abduction, and circumduction. A larger diameter neck would impinge on the acetabular rim at extremes of motion.

Containment of the hip joint is assured by several factors. The bony socket physically constrains the femoral head and is further deepened by the surrounding fibrocartilaginous acetabular labrum. In young children, the socket and labrum are largely cartilaginous and flexible. In older children, a larger proportion of the socket and rim is hard bone. Intimate contact between the cartilaginous surfaces of the round head and the socket, in the presence of joint fluid, provides a suction fit. In an intact joint, considerable force is required to disrupt this union. The strong fibrous joint capsule further contains the hip joint. The capsule is flexible enough to allow excellent range of motion, but secure enough to maintain the hip reduced except for extreme circumstances. The ligamentum teres does not provide any stability to the hip. The muscles that span the hip joint further provide active extrinsic stability by maintaining constant tension across the hip joint, which pushes the head into the acetabulum. These muscles, which provide the power for standing and locomotion, act around the fulcrum centered at the hip. Efficient transmission of muscle forces requires hip stability.

In order for the hip to dislocate, considerable force or mechanical advantage is required to overcome these restraints. The capsule must be torn or stretched. This will be deformed or disrupted at the time of dislocation. The ligamentum teres is likely to be torn, but this does not appear to result in any long-term sequelae.

### Treatment Options

The immediate goal in the treatment of a dislocated hip is to obtain concentric reduction as soon as practically possible. Generally, closed reduction should be attempted initially. Successful closed reduction can be achieved with intravenous or intramuscular sedation in the emergency room in many patients ( 49). Complete muscle relaxation is required for others, and this is best provided in the operating room with a general anesthetic. Open reduction is indicated if closed reduction is unsuccessful or incomplete.

Several methods of closed reduction have been described. Stimson described a maneuver for reduction of posterior dislocation of the hip. It is also referred to as the gravity method of Stimson. In this method, the patient is placed prone with the lower limbs hanging over the edge of a table. Two persons are required to perform this maneuver. An assistant stabilizes the pelvis by applying pressure downward from above. The manipulator holds the affected knee and hip flexed 90 degrees and applies gentle downward pressure in an attempt to bring the posteriorly dislocated head over the posterior rim of the acetabulum and back into the socket. Gentle internal and external rotation may assist in the reduction.

Allis described a maneuver in which the patient is placed supine and the reducing surgeon stands above the patient. For this reason, either the patient must be placed on the floor or the surgeon must climb onto the operating table. The knee is flexed to relax the hamstrings. While an assistant stabilizes the pelvis, the surgeon applies longitudinal traction along the axis of the femur and gently manipulates the femoral head over the rim of the acetabulum and back into the socket.

Bigelow described a manipulative reduction in which the patient lies supine and an assistant provides downward pressure on the pelvis. The surgeon grasps the ipsilateral limb at the ankle with one hand, puts the opposite forearm behind the knee, and applies longitudinal traction in the axis of the femur. Internal rotation, adduction, and flexion of 90 degrees or more take the tension off the \*Y\* ligament and allow the surgeon to bring the femoral head to the level of the acetabulum, posteriorly. The femoral head is then levered into the acetabulum by abducting, externally rotating, and extending the hip. This is a more forceful maneuver than the others and may cause damage to the articular surfaces of hip or even fracture the femoral neck, so it should be used with great caution.

A technique called the reverse Bigelow maneuver can be used for anterior dislocation. In this technique, the hip is held in partial flexion and abduction. One of two reduction methods may be used. The first is a lifting method in which a firm jerk is applied to the thigh, which may result in reduction. If that fails, traction is applied in the line of the thigh and the hip is then sharply internally rotated, adducted, and extended. This manipulative method may result in reduction but also risks fracture of the femoral neck.

With any type of dislocation, traction along the axis of the thigh coupled with gentle manipulation of the hip often effects reduction after satisfactory relaxation of the surrounding muscles.

If satisfactory closed reduction cannot be obtained using reasonable measures, it is appropriate to proceed with open reduction to remove any obstructing soft tissues.

### Surgical Procedures

Open reduction of a posterior dislocation should be performed through a posterolateral approach. The patient is positioned in the lateral decubitus position with the

dislocated side upward. The incision is centered on and just posterior to the greater trochanter and goes up into the buttock. Generally a straight incision can be made with the hip flexed approximately 90 degrees. Once the gluteal fascia lata is incised, the femoral head can be palpated beneath or within the substance of the gluteus maximus muscle. The fibers of the gluteus maximus can then be divided by blunt dissection, exposing the femoral head. The path of dislocation is followed through the short external rotator muscles and capsule down to the acetabulum. The sciatic nerve lies on the short external rotators and should be inspected. It may be necessary to detach the short external rotators in order to see inside the joint capsule.

Anterior dislocations should be approached through an anterior approach. This can be done through a bikini incision that uses the interval between sartorius and tensor fascia lata. The deep dissection follows the defect created by the femoral head down to the level of the acetabulum.

At the time of open reduction, the femoral head should be inspected for damage, scuffing, or fracturing. Before reduction, the acetabulum should be inspected and palpated for similar damage. Any intraarticular fragments should be removed. The labrum and capsule should be inspected for repairable tears. Labral fragments that cannot be securely replaced should be excised, but repair should be attempted. Frequently, the labrum or hip capsule is entrapped in the joint. The femoral head should be dislocated and any interposed soft tissue extracted. The labrum or capsule may be tied for ease in removal. Obstacles to reduction should be teased out of the way and the traumatic defect enlarged if necessary. The hip joint is then reduced under direct vision. Radiographs should be taken to confirm concentric reduction. If the joint appears slightly widened, repeat investigation must rule out interposed tissue. Slight widening may be due to fluid in the hip joint and this should settle out over the next few days. The capsule is repaired if possible. Closure is routine.

Open fractures should be treated with immediate irrigation and debridement. The surgical incision should incorporate and enlarge the traumatic wound. Inspection should proceed as detailed above. Capsular repair should be attempted if the hip joint is not contaminated. The wound should be left open or should be well drained to prevent invasive infection. As in all open fractures, intravenous antibiotics should be administered and repeat wound care performed as needed.

### **Postreduction Care**

After reduction, treatment should be symptomatic. Generally a short period of recumbency, until the pain subsides, can be followed by return to ambulation with crutches if necessary. Bed rest, spica casting, skin traction, and non-weight bearing have not been proven to be beneficial ( 43,50,51). After open reduction with substantial capsulorrhaphy, immobilization or a spica cast may be indicated for a period of 6 weeks to allow capsular healing. Physical therapy is not routinely necessary. Return to full activities is encouraged.

### **Complications**

Most hip dislocations in children will be treated and resolved without sequelae. Complications are rare.

#### ***Vascular Injury***

Impingement on the femoral neurovascular bundle has been described after anterior hip dislocation in children, and this may occur in 25% of patients ( 50). If there is femoral artery occlusion, the hip should be relocated as soon as possible to remove the offending pressure from the femoral vessels. If relocation of the hip fails to restore perfusion, immediate exploration of the femoral vessels by a vascular surgeon is indicated.

#### ***Nerve Injury***

The sciatic nerve may be damaged after a posterior dislocation of the hip in 2% to 13% of patients ( 35,50,51). Usually the nerve is directly compressed by the femoral head. The treatment is expedient relocation of the hip. Nerve function returns spontaneously in most patients ( 35,43). The nerve does not need to be explored unless open reduction is required for other reasons. If sciatic nerve function is demonstrated to be intact and is lost during the reduction maneuver, the nerve should be explored to ensure that it has not displaced into the joint. Other nerves around the hip joint rarely are injured at dislocation. Treatment is generally expectant unless laceration or incarceration is suspected. If so, exploration is indicated.

#### ***Avascular Necrosis***

Avascular necrosis occurs in about 10% of hip dislocations in children ( 40,50). Prompt relocation of the hip, especially within 24 hours, may decrease the incidence of this complication (37,50). The risk of AVN is probably related to the severity of initial trauma ( 50). The cause is unknown. It may result from damage to ascending vessels or increased intracapsular pressure ( 49). The type of postreduction care has not been shown to influence the rate of AVN.

Early technetium bone scanning detects AVN as an area of decreased uptake. This is best seen on pinhole collimated images. After a few weeks, with the onset of revascularization and reossification, the uptake may appear normal or even increased.

Magnetic resonance imaging detects avascularity of the capital femoral epiphysis as loss of signal on T1-weighted images ( 48). Findings on T2-weighted images are abnormal but of variable signal intensity.

After hip dislocation, routine screening for AVN by bone scan or MRI cannot be strongly recommended for several reasons. Even if a perfusion defect is detected, there is no known treatment that will reverse it. Secondly, MRI may be falsely negative if performed within a few days of injury ( 48). Furthermore, hips with abnormal bone scan and MRI weeks after injury may not develop symptomatic AVN. In fact, a large proportion of perfusion defects seen on MRI spontaneously resolve after several months (41,48).

If hips are followed by serial radiographs for AVN, it is recommended that they be studied for at least 2 years after dislocation because radiographic changes may appear late (32). If MRI yields normal findings 4 to 6 weeks after injury, no further study is necessary because the risk of developing symptomatic AVN is miniscule (48).

If AVN develops, pain, loss of motion, and deformity of the femoral head are likely ( 31). AVN in a young child resembles Perthes' disease and may be treated like Perthes' disease (31). Priorities are to maintain mobility and containment of the femoral head to maximize congruity after resolution. AVN in older children should be treated as in adults and may require hip fusion, osteotomy, or reconstruction, as discussed following femoral neck fractures.

### **Recurrent Dislocation**

Recurrence after traumatic hip dislocation is rare but occurs most frequently after posterior dislocation in children under 8 years of age ( 33,38) or in children with known hyperlaxity (Down's syndrome, Ehlers-Danlos disease). The incidence is estimated at no more than 3% ( 46). At surgical exploration of these hips, recurrence has been found to result from either laxity or a defect in the capsule ( 33). Recurrence can be quite disabling and in the long term may result in damage to the articular surfaces due to scuffing. Arthrography is recommended to identify a capsular defect or redundancy ( 33). Prolonged spica casting (at least 3 months) may stop recurrence (53), but exploration with capsulorrhaphy is a more rapid and reliable solution ( 33,38,53). In older children, recurrent dislocation can occur as a result of a bony defect in the posterior rim of the acetabulum similar to that in adults and may require posterior acetabular reconstruction.

### **Chondrolysis**

Chondrolysis has been reported after hip dislocation in up to 6% of children ( 40,43,45,46) and probably occurs as a result of articular damage at the time of dislocation. Chondrolysis cannot be reversed by medical means, and treatment should be symptomatic. Antiinflammatory medicines and weight-relieving devices should be used as needed. If the joint fails to reconstitute, fusion or reconstruction should be considered.

### **Coxa Magna**

Coxa magna occasionally occurs after hip dislocation. The reported incidence ranges from 0% to 47% ( 40,45,46). It is believed to occur as a result of posttraumatic

hyperemia (46). In most children, this condition is asymptomatic and does not require any treatment (46). There is no intervention that will prevent coxa magna.

### Late Presentation

Not all hip dislocations in children cause severe or incapacitating symptoms. Ambulation may even be possible (Fig. 21-19). As a result, treatment may be delayed or the diagnosis missed, and shortening of the limb and contracture are well-established, making reduction difficult. Prolonged heavy traction will sometimes effect reduction (42). Preoperative traction, extensive soft tissue release, or primary femoral shortening should be considered if open reduction is required. Open reduction will likely be difficult and will not always be successful. Even if the hip stays reduced, progressive arthropathy may lead to a stiff and painful hip. The likelihood of a good result decreases with the duration of dislocation.

### Interposed Soft Tissue

Interposed tissues may cause nonconcentric reduction or result in complete failure of closed reduction. Muscle, bone, articular cartilage and labrum have been implicated (34,37,39,43,52). CT arthrography or MRI provides information on obstacles to complete reduction and the direction of the initial dislocation (39,52). Open reduction generally is necessary to clear impeding tissues from the joint (34,39,43,46,52). Untreated nonconcentric reduction may lead to permanent degenerative arthropathy (47).

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## CHAPTER REFERENCES

### Hip Fractures in Children

1. Alho A. Concurrent ipsilateral fractures of the hip and femoral shaft. *Acta Orthop Scand* 1996;67:19–28.
2. Ashwood N, Wojcik AS. Traumatic separation of the upper femoral epiphysis in a 15-month-old girl: an unusual mechanism of injury. *Injury* 1995;26:695–696.
3. Bray TJ. Femoral neck fracture fixation. *Clin Orthop Rel Res* 1997;339:20–31.
4. Canale ST, Beaty JH. Pelvic and hip fractures. In: Rockwood CA Jr, Wilkins KE, Beaty JH, eds. *Fractures in children*, 4th ed. Philadelphia: Lippincott-Raven, 1996:1109–1193.
5. Canale ST, Bourland WL. Fracture of the neck and intertrochanteric region of the femur in children. *J Bone Joint Surg [Am]* 1977;59:431–443.
6. Cheng JCY, Tang N. Decompression and stable internal fixation of femoral neck fractures in children can affect the outcome. *J Pediatr Orthop* 1999;19:338–343.
7. Chung SMD. The arterial supply of the developing proximal end of the human femur in childhood. A report of six cases. *Ann Surg* 1928;88:902–907.
8. Colonna PC. Fracture of the neck of the femur. *J Bone Joint Surg [Am]* 1976;58:961–970.
9. Currey JD, Butler G. Mechanical properties of bone tissue in children. *J Bone Joint Surg [Am]* 1975;57:810–814.
10. Davison BL, Weinstein SL. Hip fractures in children: a long-term follow-up study. *J Pediatr Orthop* 1992;12:355–358.
11. Devas MB. Stress fractures of the femoral neck. *J Bone Joint Surg [Br]* 1965;47:728–738.
12. Edgren W. Coxa plana. A clinical and radiological investigation with particular reference to the importance of the metaphyseal changes for the final shape of the proximal part of the femur. *Acta Orthop Scand Suppl* 1965;84:1–129.
13. Forlin E, Guille JT, Kumar SJ, et al. Transepiphyseal fractures of the neck of the femur in very young children. *J Pediatr Orthop* 1992;12:503–509.
14. Haddad FS, Bann S, Hill RA, et al. Displaced stress fracture of the femoral neck in an active amenorrhoeic adolescent. *Br J Sports Med* 1997;31:70–75.
15. Hansman CF. Appearance and fusion of ossification centers in the human skeleton. *AJR* 1962;88:476–482.
16. Hughes LO, Beaty JH. Current concepts review: fractures of the head and neck of the femur in children. *J Bone Joint Surg [Am]* 1994;76:283–292.
17. Ingari JV, Smith DK, Aufdemorte TB, et al. Anatomic significance of magnetic resonance imaging findings in hip fracture. *Clin Orthop Rel Res* 1996;332:209–214.
18. Jerre R, Karlsson J. Outcome after transphyseal hip fractures. *Acta Orthop Scand* 1997;68:235–238.
19. Lam SF. Fractures of the neck of the femur in children. *J Bone Joint Surg [Am]* 1971;53:1165–1179.
20. Langenskiold A, Salenius P. Epiphyseodesis of the greater trochanter. *Acta Orthop Scand* 1967;38:199–219.
21. Maruenda JI, Barrios C, Gomar-Sancho F. Intracapsular hip pressure after femoral neck fracture. *Clin Orthop Rel Res* 1997;340:172–180.
22. Ng GP, Cole WG. Effect of early hip decompression on the frequency of avascular necrosis in children with fractures of the neck of the femur. *Injury* 1996;27:419–421.
23. Ogden JA. Changing patterns of proximal femoral vascularity. *J Bone Joint Surg [Am]* 1974;56:941–950.
24. Ratliff AHC. Fractures of the neck of the femur in children. *J Bone Joint Surg [Br]* 1962;44:528–542.
25. Ratliff AHC. Complications after fractures of the femoral neck in children and their treatment. In Proceedings of the British Orthopaedic Association. *J Bone Joint Surg [Br]* 1970;52:175.
26. St. Pierre P, Staheli LT, Smith JB, et al. Femoral neck stress fractures in children and adolescents. *J Pediatr Orthop* 1995;15:470–473.
27. Soto-Hall R, Johnson LH, Johnson RA. Variations in the intra-articular pressure of the hip joint in injury and disease. *J Bone Joint Surg [Am]* 1964;46:509–516.
28. Swischuk LE. Irritable infant and left lower extremity pain. *Pediatr Emerg Care* 1997;13:147–148.
29. Trueta J. The normal vascular anatomy of the human femoral head during growth. *J Bone Joint Surg [Br]* 1957;39:358–393.
30. Trueta J, Morgan JD. The vascular contribution to osteogenesis. *J Bone Joint Surg [Br]* 1960;42:97–109.

### Hip Dislocations in Children

31. Barquet A. Natural history of avascular necrosis following traumatic hip dislocation in childhood. A review of 145 cases. *Acta Orthop Scand* 1982;53:815–820.
32. Barquet A. Traumatic hip dislocation in childhood. *Acta Orthop Scand* 1979;50:549–553.
33. Barquet A. Recurrent traumatic dislocation of the hip in childhood. *J Trauma* 1980;20:1003–1006.
34. Canale ST, Manugian AH. Irreducible traumatic dislocations of the hip. *J Bone Joint Surg [Am]* 1979;61:7–14.
35. Epstein HC. Traumatic dislocations of the hip. *Clin Orthop Rel Res* 1973;92:116–142.
36. Fairbairn KJ, Mulligan ME, Murphey MD, et al. Gas bubbles in the hip joint on CT: an indication of recent dislocation. *AJR* 1996;166:472–473.
37. Funk FJ. Traumatic dislocation of the hip in children. *J Bone Joint Surg [Am]* 1962;44:1135–1145.
38. Gaul RW. Recurrent traumatic dislocation of the hip in children. *Clin Orthop Rel Res* 1973;90:107–109.
39. Gennari JM, Merrot T, Bergoin V, et al. X-ray transparency interpositions after reduction of traumatic dislocations of the hip in children. *Eur J Pediatr Surg* 1996;6:288–293.
40. Glass A, Powell HDW. Traumatic dislocation of the hip in children. An analysis of forty-seven patients. *J Bone Joint Surg [Br]* 1961;43:29–37.
41. Godley DR, Williams RA. Traumatic dislocation of the hip in a child: usefulness of MRI. *Orthopedics* 1993;16:1145–1147.
42. Gupta RC, Shrivast BP. Reduction of neglected traumatic dislocation of the hip by heavy traction. *J Bone Joint Surg [Am]* 1977;59:249–251.
43. Hamilton PR, Broughton NS. Traumatic hip dislocation in childhood. *J Pediatr Orthop* 1988;18:691–694.
44. Hernandez RJ, Poznanski AK. CT evaluation of pediatric hip disorders. *Orthop Clin North Am* 1985;16:513–541.
45. Hougard K, Thomsen PB. Traumatic hip dislocation in children. Follow up of 13 cases. *Orthopedics* 1989;12:375–378.
46. Offierski CM. Traumatic dislocation of the hip in children. *J Bone Joint Surg [Br]* 1981;63:194–197.
47. Olsson O, Landin LA, Johansson A. Traumatic hip dislocation with spontaneous reduction and capsular interposition. *Acta Orthop Scand* 1994;65:476–479.
48. Poggi JJ, Callaghan JJ, Spritzer CE, et al. Changes on magnetic resonance images after traumatic hip dislocation. *Clin Orthop Rel Res* 1995;319:249–259.
49. Rieger H, Pennig D, Klein W, Grunert J. Traumatic dislocation of the hip in young children. *Arch Orthop Trauma Surg* 1991;110:114–117.
50. The Scientific Research Committee of the Pennsylvania Orthopaedic Society. Traumatic dislocation of the hip in children. Final report. *J Bone Joint Surg [Am]* 1968;50:79–88.
51. Schlonsky J, Miller PR. Traumatic hip dislocations in children. *J Bone Joint Surg [Am]* 1973;55:1057–1063.
52. Shea KP, Kalamachi A, Thompson GH. Acetabular epiphysis–labrum entrapment following traumatic anterior dislocation of the hip in children. *J Pediatr Orthop* 1986;6:215–219.
53. Wilchinsky ME, Pappas AM. Unusual complications in traumatic dislocation of the hip in children. *J Pediatr Orthop* 1985;5:534–539.

## 22 FEMORAL SHAFT FRACTURES

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JAMES H. BEATY

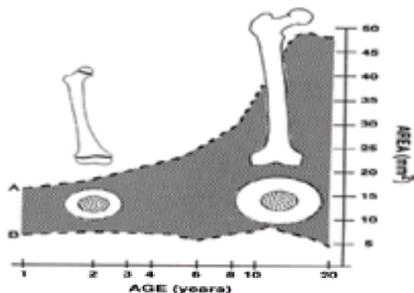
[Anatomy](#)  
[Mechanism of Injury](#)  
[Diagnosis](#)  
[Radiographic Findings](#)  
[Classification](#)  
[Treatment](#)  
[Pavlik Harness](#)  
[Immediate Spica Casting](#)  
[Traction and Casting](#)  
[External Fixation](#)  
[Intramedullary Fixation](#)  
[Open Reduction and Internal Fixation with Plate and Screws](#)  
[Complications of Femoral Shaft Fractures](#)  
[Leg Length Discrepancy](#)  
[Angular Deformity](#)  
[Rotational Deformity](#)  
[Delayed Union](#)  
[Nonunion](#)  
[Muscle Weakness](#)  
[Infection](#)  
[Neurovascular Injury](#)  
[Compartment Syndrome](#)  
[Special Fractures of the Femoral Shaft](#)  
[Metaphyseal \(Subtrochanteric and Supracondylar\) Fractures](#)  
[Open Femoral Fractures](#)  
[Femoral Fractures in Patients with Metabolic or Neuromuscular Disorders](#)  
[Floating Knee Injuries](#)  
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Femoral shaft fractures, including subtrochanteric and supracondylar fractures, represent approximately 1.6% of all bony injuries in children. The male:female ratio of femoral fracture is 2.6:1 with a bimodal distribution (10,12,17). The first peak occurs in early childhood, the second in mid-adolescence. A review of the Maryland Hospital Discharge Database by Hinton et al. (14) confirmed the bimodal distribution with peak incidences at 2 and 12 years of age. The annual rate of femoral shaft fracture in children was 1 per 5,000. In Switzerland, the incidence of pediatric femoral shaft fracture is 1 per 2,000 per year (5).

Although femoral shaft fractures are dramatic and disabling injuries, both to the patient and the family, most unite rapidly without significant complications or sequelae. Not many years ago, traction and casting were standard treatment for all femoral shaft fractures in children, and femoral fractures ranked high in duration of hospitalization for a single diagnosis (13). More recently, a variety of therapeutic alternatives, such as external fixation, compression plating, and flexible or locked intramedullary nailing, have become available to help decrease impairment, increase convenience, and decrease cost of care.

### ANATOMY

Children's bone changes from primarily weak woven bone to stronger lamellar bone through remodeling during childhood (26). Strength also is increased by a change in geometry (Fig. 22-1). The increasing diameter and area of bone result in a markedly increased area moment of inertia, leading to marked increase in strength. This progressive increase in bone strength helps explain the bimodal distribution of femoral fractures. In early childhood, the femur is relatively weak and breaks under load conditions reached in normal play. In adolescence, high-velocity trauma is required to reach the stresses necessary for fracture.



**FIGURE 22-1.** The shaded area represents cortical thickness by age group. This rapid increase in cortical thickness may contribute to the diminishing incidence of femoral fractures during late childhood. (Redrawn from Netter FH. *The Ciba collection of medical illustrations*. Vol. 8. Musculoskeletal system. Part I. Anatomy, physiology, and metabolic disorders. Summit, NJ: Ciba-Geigy, 1987; with permission.)

### MECHANISM OF INJURY

The etiology of femoral fractures in children varies with the age of the child. In children younger than walking age, up to 80% of femoral fractures may be caused by abuse (1,11,27); in children under 4 years of age, up to 30% of femoral fractures may be caused by abuse. Blakemore et al. (3) reported that in 42 children 1 to 5 years of age who had femoral fractures, a history of a fall was present in 34. Although the history was considered suspicious for intentional injury in 14 patients, only 1 patient had a documented abuse-related injury. Older children are unlikely to have a femoral shaft fracture caused by abuse, because their bone is sufficiently strong to tolerate forceful blows or is able to resist torque without fracture. In older children, femoral fractures are most likely to be caused by high-energy injuries, such as motor vehicle accidents, which account for over 90% of femoral fractures in this age group (9,12,20). Gunshot wounds are an increasingly common cause of femoral fractures.

Pathologic femoral fractures are relatively rare in children, but they may occur because of generalized osteopenia in infants or young children with osteogenesis imperfecta. A femoral fracture in a young child with no history suggestive of abuse or significant trauma should suggest the possibility of osteogenesis imperfecta (19). Radiologic evaluation is often insufficient to diagnose osteogenesis imperfecta, and skin biopsy, collagen analysis, and bone biopsy may be required to make a definitive diagnosis. Generalized osteopenia also may accompany neurologic diseases, such as cerebral palsy or myelomeningocele, leading to fracture with minor trauma in osteopenic bone (10,16,25). Pathologic fractures may occur in patients with neoplasms, most often benign lesions such as nonossifying fibroma, aneurysmal bone cyst, unicameral cyst, or eosinophilic granuloma. Pathologic femoral fractures rarely occur in patients with osteosarcoma or Ewing's sarcoma (Fig. 22-2).



**FIGURE 22-2. A:** Femoral fracture through a poorly demarcated, mixed, osteoblastic, osteolytic lesion—an osteosarcoma. **B:** Sclerotic borders of this lesion in the distal femur are typical of a pathologic fracture through a nonossifying fibroma.

Stress fractures may occur in any location in the femoral shaft (6,15,22). Most occur in adolescents involved in sports activity, such as football, lacrosse, and track. Although uncommon (4% of all stress fractures in children), femoral shaft stress fracture should be considered in a child with thigh pain because an unrecognized stress fracture may progress to a displaced femoral fracture. As the number of young children involved in athletics increases, stress fractures will become more frequent (4). Bilateral femoral stress fractures were reported in a rollerblade enthusiast, reflecting a departure from the traditional sports recognized as being related to this injury (28).

An unusual femoral fracture reported in infants is a greenstick fracture of the medial distal femoral metaphysis that occurs when the parent falls on a child who is straddling the parent's hip. It is important to recognize this fracture because it occurs in infants at an age when abuse is the leading cause of femoral fracture. The fracture is caused by bending of the femur, which produces a compression injury to the medial cortex. This injury is not consistent with abuse and may confirm a parent's description of a fall as the cause.

## DIAGNOSIS

Most patients with femoral shaft fractures are unable to walk and are in extreme pain with an obvious fracture. The diagnosis is more difficult in patients with multiple trauma or head injury, and in nonambulatory, severely disabled children. A physical examination usually is sufficient to document the presence of a femoral fracture. Swelling, instability, crepitation, and tenderness usually are present. In patients lacking sensation (myelomeningocele), swelling and redness may simulate infection.

The entire child must be carefully examined. Hypotension rarely results from an isolated femoral fracture. Waddell's triad of femoral fracture, intraabdominal or intrathoracic injury, and head injury are associated with high-velocity automobile injuries. Multiple trauma with head injury may dictate a change in management of the femoral shaft fracture (20,23).

The hemodynamic significance of femoral fracture has been studied recently by two groups (8,21). Hematocrit levels below 30% rarely occurred in the absence of multiple system injury. Hemodynamic instability was always associated with multisystem injury. A declining hematocrit should not be attributed to closed femoral fracture until other sources of blood loss have been eliminated (8,21).

## RADIOGRAPHIC FINDINGS

Radiographic evaluation should include the entire femur, as well as the hip and knee, because injury of the adjacent joints is common. Femoral shaft fractures may occur with intertrochanteric fractures of the hip, fractures of the femoral neck, and dislocation of the upper end of the femur (2,7). Distal femoral fractures may be associated with physeal injury about the knee, knee ligament injury, meniscal tears (29), and tibial fractures (18).

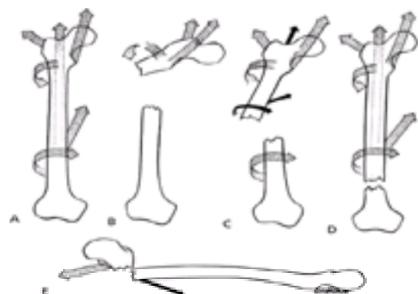
Plain radiographs generally are sufficient for making the diagnosis. Bone scanning and magnetic resonance imaging (MRI) occasionally may be helpful in the diagnosis of small buckle fractures in limping children or stress fractures but usually are not necessary. Comminution or nondisplaced "butterfly" fragments, second fractures, joint dislocations, and pathologic lesions should be considered.

Roach and Hoschl (24) described diffuse uptake of radionuclide tracer throughout an entire femoral shaft in an 18-month-old child. This is significant because it demonstrates that radionuclide uptake may not be limited to the isolated area of the fracture but may involve the entire shaft of the bone in a very young child. A similar finding has been shown in plastic deformation of the forearm in young children.

## CLASSIFICATION

Femoral fractures are classified as (a) transverse, spiral, or oblique; (b) comminuted or noncomminuted; and (c) open or closed. Open fractures are classified according to Gustilo's system (13). The presence or absence of vascular and neurologic injury is documented and is part of the description of the fracture. The most common femoral fracture in children (over 50%) is a simple transverse, closed, noncomminuted injury.

The level of the fracture (Fig. 22-3) leads to characteristic displacement of the fragments based on the attached muscles. With subtrochanteric fractures, the proximal fragment lies in abduction, flexion, and external rotation. The pull of the gastrocnemius on the distal fragment in a supracondylar fracture produces an extension deformity, which may make the femur difficult to align.



**FIGURE 22-3.** The relationship of fracture level and position of the proximal fragment. **A:** In the resting unfractured state, the position of the femur is relatively neutral due to balanced muscle pull. **B:** In proximal shaft fractures the proximal fragment assumes a position of flexion (iliopsoas), abduction (abductor muscle group), and lateral rotation (short external rotators). **C:** In mid-shaft fractures the effect is less extreme because there is compensation by the adductors and extensor attachments on the proximal fragment. **D:** Distal shaft fractures produce little alteration in the proximal fragment position because most muscles are attached to the same fragment providing balance. **E:** Supracondylar fractures often assume a position of hyperextension of the distal fragment due to the pull of the gastrocnemius.

## TREATMENT

Treatment of femoral shaft fractures in children is age dependent, with considerable overlap between age groups ( [Table 22-1](#)). The child's size and bone age also must be considered, as well as the cause of the injury. Whether the femoral fracture is an isolated injury or part of polytrauma influences treatment choices. Economic concerns, the family's ability to care for a child in a spica cast or external fixator, and the advantages and disadvantages of any operative procedure also are important factors. In adolescents, the psychologic implications of treatment should be considered. Prolonged hospitalization alters the adolescent's self-image and interrupts social and educational development. Especially in older children, the disadvantages of nonoperative treatment—time in traction or a cast, economic and social impact on the family—must be carefully weighed against the potential complications of a surgical procedure: infection, refracture after removal of fixation, neurologic injury, limb shortening or overgrowth, and avascular necrosis of the femoral head.

Age	Treatment
Birth to 24 mo	Pavlik harness (newborn to 6 mo) Immediate spica cast Traction → spica cast
24 mo to 5 yr	Immediate spica cast Traction → spica cast External fixation (rare) Flexible intramedullary rod (rare)
6–11 yr	Traction → spica cast Flexible intramedullary rod Compression plate External fixation
12 yr to maturity	Flexible intramedullary rod Compression plate Locked intramedullary rod External fixation

Treatment choices are influenced by polytrauma (vs. an isolated femoral shaft fracture) or open fractures with soft tissue trauma.

**TABLE 22-1. TREATMENT OPTIONS FOR FEMORAL SHAFT FRACTURES IN CHILDREN AND ADOLESCENTS**

The comparative economics of nonoperative and operative treatment of femoral shaft fractures have been evaluated by several researchers, but no clear consensus has been reached. Reeves et al. ([57](#)) reported that the cost of nonoperative treatment was 46% higher than that of operative treatment, even considering the necessity for a second surgical procedure for implant removal. Newton and Mubarak ([53](#)) analyzed the financial aspects of femoral shaft fracture treatment in 58 children and adolescents and determined that total charges were lowest for those treated with early spica casting and highest for those treated with skeletal traction or intramedullary nailing. Similarly, Coyte et al. ([35](#)) found the cost of surgical treatment (external fixation) to exceed that of early spica casting in all cases. Stans and Morrissy ([65](#)), in evaluating the cost of treating femoral fractures in children 6 to 16 years of age, found that all surgical treatments cost approximately the same. This cost was three times that of early spica cast management and essentially equivalent to traction, followed by spica cast. In all cost studies related to femoral fracture, the determinants of increased cost are (a) cost of fixation device, (b) cost of the operating room, and (c) cost of hospital days for recovery. Nork and Hoffinger ([55](#)) showed that hospital profit was highest in the traction group, despite charges being equivalent to the surgical group, because the actual hospital resources required were significantly less. In a study by Yandow et al. ([70](#)) comparing cost of traction to immediate spica in young children, equivalent results occurred with 83% greater charges in the traction group. Certainly, cost is a factor, but it should not be the overriding consideration in discussions of treatment options with the family.

In infants, newborn to 6 months of age, femoral fractures usually are reasonably stable because of thick periosteum. For stable proximal or mid-shaft femoral fractures, simple splinting or a Pavlik harness is all that is required. For unstable fractures in infancy, a simple splint can be tried, but usually the femur cannot be adequately treated in this manner. We have found that a Pavlik harness with a wrap around the thigh, as described by Wilkins, is beneficial. For femoral fractures with excessive shortening (>1–2 cm) or angulation (>30 degrees), spica casting may be required. Traction rarely is necessary in this age group.

In children 6 months to 6 years of age, immediate or early spica casting is the treatment of choice for femoral fractures with less than 2 cm of initial shortening ( [Fig. 22-4](#)). Femoral fractures with more than 2 cm of initial shortening or marked instability and fractures that cannot be reduced with immediate spica casting require 3 to 10 days of skin or skeletal traction. Skeletal stabilization by external fixation generally is reserved for children with open fractures or multiple trauma. Intramedullary rodding is used in children with metabolic bone disease that predisposes to fracture or after multiple fractures, such as in osteogenesis imperfecta. Larger children in whom reduction cannot be maintained with a spica cast occasionally may benefit from flexible intramedullary rodding or traction.



**FIGURE 22-4. A:** Three weeks after immediate spica casting of isolated femoral shaft fracture in 3-year-old child. **B:** Three months after injury, fracture is healed in good position.

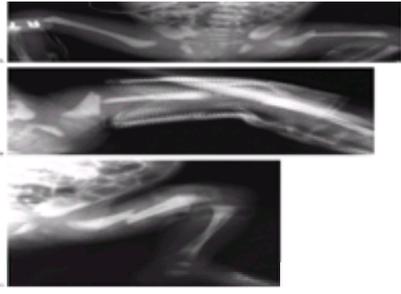
Treatment of femoral fractures in children 6 to 11 years of age is highly controversial. For stable, minimally displaced fractures, immediate spica casting usually produces satisfactory results; however, in large children with unstable comminuted fractures, traction followed by application of a cast brace or spica cast may be necessary. Because of the cost and the social problems that may accompany management of a child in a spica cast, enthusiasm for skeletal fixation has increased in recent years. Skeletal fixation frequently is used in children with multiple trauma, head injury, vascular compromise, floating knee injuries, or multiple fractures. Treatment options should be discussed with the parents before choosing the method of fracture fixation, even for isolated fractures.

Enthusiasm for treatment that decreases hospital stay has led to the use of external fixation and flexible intramedullary nails in children 6 years of age through maturity. Compression plating has been reintroduced as a technique with low risks and significant benefit in the management of pediatric femoral fractures. Even home traction has been recommended as a low-cost alternative for management with satisfactory outcome. In older children and adolescents, antegrade rodding has been recommended as a standard procedure, but the recognized risks of avascular necrosis and growth disturbance have led to limited use of this as a standard technique.

### Pavlik Harness

In a newborn with a femoral fracture, one is faced with a very small child, a thick periosteum, and a remarkable remodeling potential. Stannard, Christensen, and Wilkins ([64](#)) popularized the use of the Pavlik harness for the treatment of this fracture. This treatment is ideal for a proximal or mid-shaft femur fracture that occurs as a birth-related injury. In a newborn infant in whom a femoral fracture is noted in the intensive care unit or nursery, simple padding or immobilization of the femoral with a soft splint can be tried. For a stable fracture, this approach may be sufficient and will allow intravenous access to the feet if needed. If the fracture remains angulated, a Pavlik harness can be applied with the hip in moderate flexion and abduction. This often helps align the distal fragment with the proximal fragment ( [Fig. 22-5](#)). Reduction can be aided by a wrap around the thigh if greater stability is needed. The reduction is easily evaluated in the sagittal plane with a lateral radiograph, but evaluation of angulation in the coronal plane (varus-valgus) is more difficult because of hyperflexion. Stannard et al. ([64](#)) reported acceptable alignment in all patients with less than 1 cm of shortening. In fractures occurring in infancy, management may include evaluation for underlying metabolic bone abnormality or abuse. Once these have been ruled out, a stable fracture should be managed using a simple splint. Unstable fractures should be managed using a Pavlik harness and a small wrap around the thigh, with immediate spica or traction reserved for the rare fracture that cannot be managed with simpler means because of failure to align the

fracture or excessive shortening.



**FIGURE 22-5.** **A:** This infant had a birth-related left femoral fracture. **B:** An anterior-posterior splint was used but ended at the fracture site, only increasing the angulation. **C:** A Pavlik harness reduces the fracture by flexing the distal fragment.

### Immediate Spica Casting

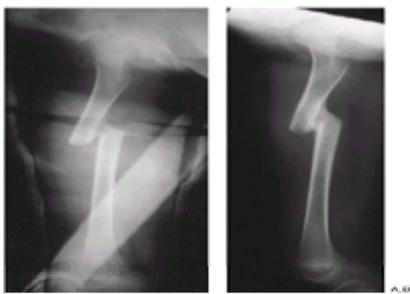
Immediate spica casting, popularized by Irani et al. (46) and Staheli et al. (62), is indicated for isolated femoral shaft fractures in children under 6 years of age unless (a) shortening of more than 2 cm is present, (b) massive swelling of the thigh is noted, or (c) associated injuries are present. Its primary advantages are simplicity, low cost, and generally good results based on leg length equality, healing time, and motion (38,45).

Hughes et al. (42) evaluated 23 children ranging in age from 2 through 10 years who had femoral fractures treated with immediate spica casting to determine the impact of treatment on the patients and their families. The greatest problems encountered by the family in caring for a child in a spica cast were transportation, cast intolerance by the child, and keeping the child clean. Although most children did not attend school while in the cast, no child was required to repeat a grade and no permanent psychological effects were reported by the parents. The researchers found that overall treatment in a spica cast was much easier for families of preschool children than for those with school-age children: healing time was faster, younger children were lighter, time until independent walking was shorter, and families of younger children had more preexisting care arrangements for them. They recommended counseling and planning with the family before cast application if possible.

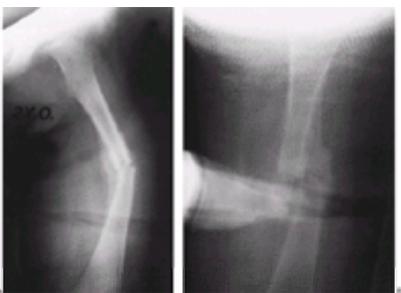
Ilgen et al. (44), in a series of 114 isolated femoral fractures in children under 6 years of age, found that 90 degree/90 degree (90/90) spica casting was successful in 86% without cast change or wedging, based on tolerance of shortening less than 1.5 cm and angulation less than 10 degrees. Of the 20 patients requiring spica change, only 2 healed with unacceptable position (>2 cm of leg length discrepancy). One of these overgrew by 1.5 cm and the other was lost to follow-up. Ilgen et al. used an immediate spica regardless of initial shortening and placed the child in traction only if unacceptable shortening occurred. Shortening requiring spica cast change was associated with a knee flexion angle of less than 50 degrees. Similar excellent results have been reported by Czertak and Henrikus (36) using the 90/90 spica cast.

Thompson et al. (67) described the telescope test in which patients were examined with fluoroscopy at the time of reduction and casting. If more than 3 cm of shortening could be demonstrated with gentle axial compression, traction was used rather than immediate spica casting. By using the telescope test, these researchers decreased unacceptable results (>2.5 cm of shortening) from 18% to 5%.

Martinez et al. (47) reported excessive shortening and angular deformity in 26 of 51 patients after immediate spica casting, especially in comminuted fractures. Although shortening and angulation can occur in a spica cast, excessive deformity can be detected with weekly radiologic and clinical evaluations during the first 2 to 3 weeks after injury. Minimal shortening is acceptable but should not exceed 2 cm (Fig. 22-6). This is best measured on a lateral radiograph taken through the cast. If follow-up radiographs reveal significant varus (>10 degrees) or anterior angulation (>30 degrees), the cast may be wedged. However, Weiss et al. (69) noted that wedging of 90/90 spica casts can cause peroneal nerve palsy, especially during correction of valgus angulation. For unacceptable position, the fracture can be manipulated and a new cast applied, or the cast can be removed and the patient placed in traction to regain or maintain length (Fig. 22-7). Angular deformity of up to 15 degrees in the coronal plane and up to 30 degrees in the sagittal plane may be acceptable, depending on the patient's age (Table 22-2). Finally, if shortening exceeds 2 cm, traction or an external fixator can be used.



**FIGURE 22-6.** **A:** Immediate spica casting of isolated femoral shaft fracture in an 8-year-old child; shortening on initial radiograph was 2 cm. **B:** Four months after injury, shortening of 2 cm is seen with this healed fracture.



**FIGURE 22-7.** **A:** Unacceptable position of fracture in spica cast in a 2-year-old child. **B:** With manipulation, application of a new spica cast, and cast wedging, this angulation is corrected.

Age	Varus/ Valgus (degrees)	Anterior/ Posterior (degrees)	Shortening (mm)
Birth to 2 yr	30	30	15
2-5 yr	15	20	20
6-10 yr	10	15	15
11 yr to maturity	5	10	10

**TABLE 22-2. ACCEPTABLE ANGULATION**

Shortening and angulation occur most often in fractures associated with polytrauma and those with loss of the periosteal sleeve ( 9,10). Fry et al. (10) found that 50% (12 of 23) of closed femoral shaft fractures caused by high-energy trauma in children under 10 years of age required repeat reduction or other treatment to correct excessive shortening or angulation that occurred after initial reduction; only 8% (2 of 24) of low-energy fractures required repeat closed reduction.

The position of the hips and knees in the spica cast is controversial. Spica cast application with the hip and knee extended and the bottom of the foot cut out to prevent excessive shortening has been described ( 49). Varying the amounts of hip and knee flexion in the spica cast based on the position of the fracture also has been recommended: the more proximal the fracture, the more flexed the hip should be (62).

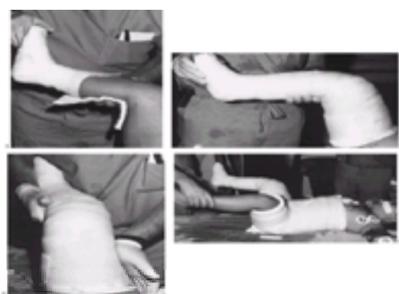
An alternative to the standard hip-knee extended spica is the 90/90 spica cast or the sitting spica cast ( 48,50). A sitting spica cast (Fig. 22-8), with the hips and knees set in about 90 degrees of flexion, is the easiest and perhaps the most effective cast for femoral fractures in preschool-aged children, unless the fracture cannot be maintained in this position. The child is placed in a sitting position with the legs abducted about 30 degrees on either side. The synthetic material used for the cast gives it sufficient strength so that no bar is required between the legs. This not only allows the child to be carried on the parent's hip but also aids in toiletry needs, making bedpans unnecessary. Also, the child can sit upright during the day and can attend school in a wheelchair.



**FIGURE 22-8.** A 90 degree/90 degree spica cast allows a small child to be carried on the parent's hip. The child can be placed in a sitting position in a chair or on the toilet in this type of cast.

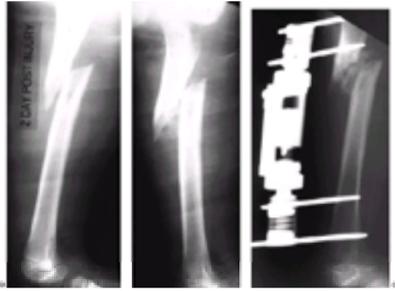
#### **Spica Cast Application: Technique**

The child is taken to the operating room or plaster room where anesthesia or sedation is administered. A short leg cast is applied with the foot in neutral position ( Fig. 22-9A). Extra padding and a felt pad are placed in the area of the popliteal fossa. The cast is then extended to a long leg cast with the knee held in 90 degrees of flexion (Fig. 22-9B). Because most diaphyseal fractures tend to fall into varus angulation while in a spica cast, a valgus mold is necessary ( Fig. 22-9C). The patient is then placed on a spica table, supporting the weight of the legs with manual traction, and the remainder of the cast is applied with the hips in 90 degrees of flexion and 30 degrees of abduction, holding the fracture out to length ( Fig. 22-9D). The leg should be placed in 15 degrees of external rotation to align the distal fragment with the external rotation of the proximal fragment. After the spica cast is in place, anteroposterior (AP) and lateral radiographs are obtained to ensure that length and angular and rotational alignment are maintained.



**FIGURE 22-9.** Application of a 90 degree/90 degree spica cast. **A:** Generous padding is applied over the foot, and a pad is placed on the popliteal fossa to prevent injury to the peroneal nerve and popliteal vessels. **B:** A long leg cast is applied with the knee flexed 90 degrees. **C:** A mold is placed over the apex of the fracture, generally correcting a varus deformity into slight valgus. **D:** Using a standard spica table, a one-and-a-half spica cast is applied with the hip flexed 90 degrees and abducted 30 degrees.

Gortex liners can be used to decrease the skin problems of diaper rash and superficial infection. This has been beneficial and has justified the cost of a Gortex liner. If angulation of more than acceptable limits occurs, the cast should be changed with manipulation in the operating room. Casts can be wedged for less than 15 degrees of angulation. If shortening of more than 2 cm is documented, the child should be treated either with cast change, traction, or conversion to external fixation with lengthening. When conversion to external fixation is required, we recommend osteoclasis at the time of the application of the external fixator with slow lengthening over a period of several weeks (1 mm per day) to reestablish acceptable length ( Fig. 22-10).



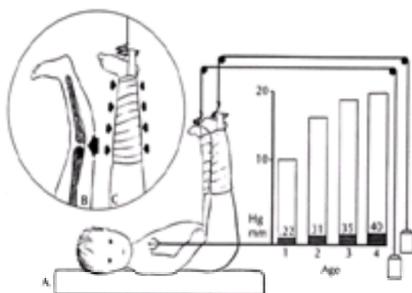
**FIGURE 22-10.** **A:** This 8-year-old child with a femur fracture was treated with an immediate spica cast. **B:** After 3 weeks of immobilization, unacceptable shortening (2.2 cm) was noted. **C:** Closed osteoclysis and lengthening with an external fixation device resolved the problem with length achieved by callus distraction.

Generally, the spica cast is worn for 4 to 8 weeks, depending on the age of the child and the severity of the soft tissue damage accompanying the fracture. After the cast has been removed, management should include skin care in young children and crutch-assisted or walker-assisted ambulation in older children. Ambulation is accomplished with weight bearing as tolerated. Stiff knees and weak hamstrings and quadriceps should be identified after the child begins to walk, and a physical therapy program should be started if needed. Long-term follow-up for leg length evaluation, range of motion, and quadriceps weakness should be maintained.

An alternative to extend the use of early spica cast treatment into an older age group and unstable fractures is the incorporation of a supracondylar traction pin into the spica cast. In a series reported by Sahin et al. (60), this technique was demonstrated to be effective in children 1 to 10 years of age. The clinicians accepted no more than 15 mm of shortening at the time of spica cast application, and obtained satisfactory results in all patients.

### Traction and Casting

Since as early as the eighteenth century, traction has been used for management of femoral fractures. The French used the Hippocratic method of traction and coaptation splinting in extension. The English, influenced by Pott (56), treated patients on their side with the hip and knee flexed. In 1861, Buck (32) introduced simple horizontal traction. Vertical overhead traction with the hip flexed 90 degrees and the knee straight was introduced by Bryant in 1873 (31,33), but this often resulted in vascular insufficiency (Fig. 22-11) (54), and it is now rarely used for treatment of femoral fractures, except occasionally in infants under 2 years of age and weighing less than 20 pounds. However, even in this infantile age group, split Russell's traction (58) or immediate spica casting is preferable. Modified Bryant's traction, in which the knee is flexed 45 degrees, increases the safety of overhead skin traction (39). Gallows traction also has been used (41).



**FIGURE 22-11.** Adverse effect on limb circulation by overhead traction as detailed by Nicholson. **A:** Incremental increase in hydrostatic pressure necessary for limb perfusion due to increasing limb length. **B:** Hyperextension of the knee occurs if the infant moves to the foot of the bed, stretching the popliteal artery. **C:** Extrinsic compression of wrapping to maintain traction tapes aggravates the problem. Any or all of these factors contribute to the risk of overhead traction. (Redrawn from Nicholson J, Foster R, Heath R. Bryant's traction: a provocative cause of circulatory complications. *JAMA* 1955;157:4118; with permission.)

The indications for skin or skeletal traction include (a) unstable femoral fracture in a child under 6 years of age with more than 2 to 3 cm of shortening, (b) femoral fracture that fails to maintain proper length and alignment in a spica cast in a child under 6 years of age, and (c) femoral fracture in a child 6 to 11 years of age, without multiple fractures, head trauma, or severe soft tissue or vascular injuries, who is able to cooperate with a period of bed rest and spica cast immobilization and whose family prefers no surgery. The limit of skin traction is the interface between skin and tape or skin and foam traction boot. Skin complications, such as slough and blistering, usually occur when more than 5 pounds of traction is applied. When more than 5 pounds of traction is required, or simply for ease in patient management, skeletal traction can be used to maintain alignment (30).

Skeletal traction also can be used in adolescents with comminuted proximal femoral shaft and intertrochanteric fractures in whom secure fixation cannot be obtained without risk of vascular compromise to the proximal femur. In general, however, skeletal traction is not recommended for children 12 years of age or older because of significant incidences of shortening and angular malunion.

The distal femur is the location of choice for a traction pin (30,37,59). Although proximal tibial traction pins have been recommended by some clinicians (43), growth arrest in the proximal tibial physis and subsequent recurvatum deformity have been associated with their use (Fig. 22-12). Also, knee ligament and meniscal injuries that sometimes accompany femoral fractures are aggravated by the chronic pull of traction across the knee. The rare indication for a tibial traction pin is a child in whom fracture configuration or skin problems prevent placement of a femoral traction wire and in whom no knee injury is present.

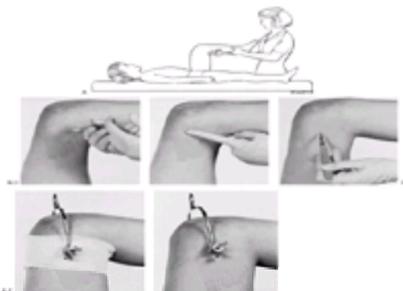


**FIGURE 22-12.** Tibial epiphyseal injury in association with tibial pin traction treatment for a femoral fracture. A 14-year-old boy sustained a femoral fracture that was treated by tibial skeletal traction. Two years later the fracture was well healed but 2.5 cm short. A recurvatum deformity of the same side was apparent. **A:** An apparent fusion of the tibial tubercle. **B:** The bridge was confirmed by tomography. **C:** Bridge resection was performed with free fat interposition. A marker was placed to facilitate subsequent evaluation of growth. A tibial pin, if used, should be inserted posterior to the anterior aspect of the tibial tubercle.

In a group of 45 patients with femoral fractures, Stanitski et al. (63) calculated the radiation dose received by the patient while in traction. The average dose before casting was 0.699 rad. Although this is a significant radiation exposure, it was not excessive compared to that received by patients managed with other methods of treatment, particularly when intraoperative exposures are considered. However, radiation exposure during treatment must be considered.

### **Femoral Traction Pin Insertion: Technique**

After preparation of the thigh circumferentially from the knee to the mid-thigh, the limb is draped in a sterile manner. The knee is held in the position in which it will remain during traction; that is, if 90/90 traction is being used, the traction pin should be inserted with the knee bent 90 degrees ( Fig. 22-13A). The patient either should be sedated and the wound treated with a local anesthetic or general anesthesia should be given before the traction pin is inserted ( Fig. 22-13B). The location of pin insertion is one finger breadth above the patella with the knee extended or just above the flare of the distal femur ( Fig. 22-13C). A small puncture wound is made over the medial side of the femur (Fig. 22-13D). A medial-to-lateral approach is used so that the traction pin does not migrate into the area of the femoral artery that runs through Hunter's canal on the medial side of the femur. A traction pin between 3/32 inch and 1/8 inch is chosen depending on the size of the child. The pin is placed parallel to the joint surface (30) to help maintain alignment while in traction. After the pin protrudes through the lateral cortex of the femur, a small incision is made over the tip of the pin. The pin is then driven far enough through the skin to allow fixation with a traction bow ( Fig. 22-13F). If 90/90 traction is used, a short leg cast is laced with a ring through its mid-portion to support the leg. Alternatively, a sling to support the calf may be used. If a sling is used, heelcord stretching should be performed while in traction.



**FIGURE 22-13.** Insertion of femoral skeletal traction pin. **A:** The limb is held in the 90 degree/90 degree position by an assistant. **B:** A site just superior to the adductor tubercle is surgically prepared, both medially and laterally. **C:** Local anesthesia is applied in the skin and periosteum on both sides of the limb. **D:** The skin is penetrated with a scalpel. **E:** A threaded Steinmann pin is introduced at the level 1 cm proximal to the adductor tubercle. **F:** Dressing and traction bow in place.

After the skeletal traction pin has been placed in the distal femur, traction is applied in a 90/90 position (the hip and knee flexed 90 degrees) ( Fig. 22-14) or in an oblique position (the hip flexed 20–60 degrees). If the oblique position is chosen, a Thomas splint or sling is necessary to support the leg. The fracture may be allowed to begin healing in traction, and radiographs should be obtained once or twice a week to monitor alignment and length. In a child under 10 years of age, the ideal fracture position in traction should be up to 1 cm of shortening and slight valgus alignment to counteract the tendency to fall into varus in the cast and the eventual overgrowth that may occur (average 0.9 cm). If this method is used for adolescents (11 years or older), normal length should be maintained.



**FIGURE 22-14.** In 90 degree/90 degree traction, a femoral pin is used and the lower leg and foot are supported with a short leg cast or a sling.

### **Technique Tips**

Threaded pins, although more difficult to remove, are preferable to smooth pins because of their secure fixation within the bone without side-to-side movement; however, they have a slightly higher incidence of skin interface complications.

Aronson et al. (30) found that obliquely placed femoral traction pins were associated with an increased incidence of varus or valgus angulation. Pins for skeletal traction should be placed parallel to the axis of the knee joint, and in children over 11 years of age the fracture should be reduced without shortening.

### **Postoperative Care**

Prolonged traction of 3 weeks or more followed by knee immobilization in a spica cast is well tolerated by young children. In children over 12 years of age or in children weighing over 100 pounds, maintaining the knee in 90 degrees of flexion for a prolonged period of time may lead to knee stiffness and a difficult period of rehabilitation (43). Cast braces, either with a traction pin or after a period of traction, allow early knee motion. Polycentric hinges, fiberglass rods, or hinge cables should be used to prevent malalignment between the cast brace and the knee that restricts motion.

Alternatives for treatment after traction application include (a) a 2- to 3-week period of traction until callus formation is apparent and the fracture is stable before pin removal and application of a spica cast or cast brace (32,34,49,52,61); (b) early incorporation of a traction pin into a spica cast; and (c) incorporation of a traction pin into a cast brace with intermittent traction to maintain length until callus formation is present (40). Each of these methods has advantages, costs, and risks, and a clear “best method” cannot be identified.

### **Results of Traction and Casting**

In a study by Gross et al. (40), 72 children with femoral fractures were treated with early cast brace/traction management. In this technique, a traction pin is placed in the distal femur and then incorporated in a cast brace. The traction pin is left long enough to be used for maintaining traction while the patient is in the cast brace or traction is applied directly to the cast. The patient is allowed to ambulate in the cast brace starting 3 days after application. Radiographs are taken of the fracture in the cast brace to document that excessive shortening is not occurring. The patient then is returned to traction in the cast brace until satisfactory callus is present to prevent shortening or angular deformity with weight bearing. The technique was not effective in older adolescents with mid-shaft fractures but achieved excellent results in children 5 to 12 years of age. The average hospital stay was 17 days.

## Complications of Traction and Casting

Comparative studies and retrospective reviews have demonstrated unsatisfactory results in a small, yet significant, percentage of patients treated with skeletal traction (43,57,97,102). It is clear that traction remains an excellent method of treatment of femoral shaft fractures in children under 12 years of age, but other methods are easier or more convenient with the same outcome.

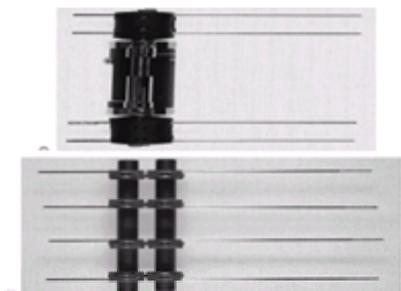
## External Fixation

External fixation of femoral shaft fractures offers a fast convenient method to align and hold the fractured femur. It is the method of choice when severe soft tissue injury is present and may be considered in any patient where traditional closed methods of management are not appropriate (104). In head-injured or multiply injured patients and those with open fractures, external fixation offers an excellent method of management. Wagner was an early advocate of monolateral frame fixation and developed an external fixation and lengthening device. Aronson and Tursky (71) in 1987 reported their early experience with 44 femoral fractures treated with primary external fixation and early weight bearing. Most patients returned to school by 4 weeks after fracture and had full knee motion by 6 weeks after the fixator was removed. In this early study, end-on alignment was the goal and overgrowth was minimal. Following early enthusiasm for the use of external devices, the 1990s saw some waning interest in their use because of complications with pin track infections, scarring, and refracture. With other methods of skeletal fixation such as flexible intramedullary rodding, the use of external fixation devices for primary treatment of femoral fractures has declined.

Bar-On et al. (72) compared external fixation with flexible intramedullary rodding in a prospective randomized study. They found that the early postoperative course was similar but that the time to return to school and to resume full activity was less with intramedullary fixation. Muscle strength was better in the flexible intramedullary fixation group at 14 months after fracture. Parental satisfaction was also significantly better in the flexible intramedullary rodding group. Bar-On et al. (72) recommended that external fixation be reserved for open or severely comminuted fractures.

## Fixator Design

The design of external fixator frames is important in determining the end result. In general, circular fixation devices are rarely, if ever, indicated for femoral fractures. External fixation should be performed using a monolateral or cantilever type system. The monolateral devices are of two types (Fig. 22-15). The AO system, in which pins can be placed at any point along a bar with a special clamp holding the pins at a right angle to the bar, has been in common use (Fig. 22-16). The advantage of this system is that the stability of fixation is increased if the two pins on each side of the fracture are spread widely with one pin close to the fracture and one quite distant from it. A second longitudinal rod can be added to this system to increase its rigidity. A second type of external fixation system has pin clamps at the end of a telescopic tube. The pin clamps provide easy application, but the stability of the fixation device is decreased because the pins are widely separated from the fracture. The pin clamps may be constrained to rotation only (Wagner) or attached with a universal joint to the barrel of the device (Orthofix) (Fig. 22-17). The telescoping barrel provides lengthening or dynamization, and the universal joints provide adjustment. Sola, Schoenecker, and Gordon (119) found that results were improved significantly by adding an auxiliary pin to the standard Orthofix type frame, providing better fixation near the fracture site. They reviewed 38 fractures in 37 children 5 to 18 years of age. Six of 22 femurs treated without auxiliary pins required remanipulations for loss of reduction, whereas only 1 of the 16 frames with an auxiliary pin required remanipulation. Although these researchers provided auxiliary pin fixation by attaching a pin to the barrel of the external fixator with methylmethacrylate, other methods of intermediate pin fixation are available at this time.



**FIGURE 22-15.** Monolateral external fixation devices have (A) fixed end clamps with universal joints (Orthofix type) or (B) pins that can be secured at various positions on a simple rod that positions close to and distant from the fracture site (AO), achieving greater stability of fracture fixation.



**FIGURE 22-16.** Application of AO external fixator. The most proximal and distal pins are placed a few centimeters from the fracture site to improve frame stability.



**FIGURE 22-17.** A: Two weeks after unilateral external fixation of a comminuted femoral shaft fracture in an 8-year-old child. B: Clinical photograph of external fixator in place. C: Four months after fracture, immediately after removal of the external fixator, fracture has healed in good position.

A new variation of external fixation device, the Orthofix device, uses a bar with pin clamps and intermediary fixation. The sliding clamps allow auxiliary pin fixation near the fracture site, as well as distal pin fixation. Dynamization is allowed with this device as well. Series reporting experience with this treatment of femoral

fractures are not yet available, and because enthusiasm currently is shifting toward flexible intermedullary rodding, large series treated with this device may not be routinely reported.

Another technical development in external fixation has been the use of hydroxyapatite-coated pins. The pin–bone interface was a problem with loosening with some devices. The strength of the pin–bone interface with hydroxyapatite-coated pins appears to be somewhat better, although large series with this device have not yet been reported.

Price and others have anecdotally recommended application of a frame with a more flexible construct. Pins are more closely clustered and placed farther from the fracture site, and the frame itself is placed more laterally away from the femur, which does not need to be anatomically reduced.

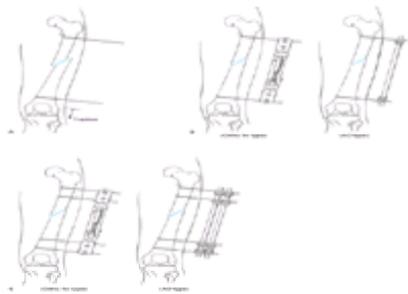
### **Unilateral Frame Application: Technique**

Preoperative planning is mandatory. Fracture lines indicative of comminution must be recognized. There must be room between the trochanteric and the distal femoral physes for the device chosen.

After appropriate anesthesia, the leg is prepared and the patient is placed on a radiolucent table or a fracture table, depending on the preference of the surgeon and the size of the child. Either the fracture table or Fluoro table works well. We make the decision based on the size of the patient and the ease with which reduction can be obtained, as well as the help available. In general, a fracture table facilitates reduction and application of the external fixator.

First we try to reduce the fracture both in length and alignment. If the fracture is open, it should be irrigated and debrided before application of the external fixation device. With the fracture maximally aligned, fixation is begun. The minimal and maximal length constraints characteristic of all external fixation systems must be kept in mind. The angular adjustment intrinsic to the fixation device needs to be determined. If a universal joint at the end of the Orthofix device only allows for 15 degrees of angular correction, one cannot expect 40 degrees of adjustment after application of the device. Rotation in general is constrained with all external fixation systems once the first pins are placed. That is, if parallel pins are placed with the fracture in 40 degrees of malrotation, a 40 degree malalignment will exist. Rotational correction must be obtained before placing the pins in the proximal and distal shafts of the femur.

Application of the fixator is similar no matter what device is chosen ( [Fig. 22-18](#)). One pin is placed proximally in the shaft, and another pin is placed distally perpendicular to the long axis of the shaft. Alignment is always to the long axis of the shaft rather than to the joint surface. Rotation should be checked before the second pin is placed because it constrains rotation but not angulation or length. After pins are correctly placed, all fixation nuts are secured and sterile dressings are applied to pins.



**FIGURE 22-18.** Technique of external fixation. **A:** With traction applied and provisional reduction of the fracture obtained, the proximal and distal pins are placed perpendicular to the long axis of the bone, being certain that rotational alignment is satisfactory. **B:** Provisional fixation is obtained by attaching the external fixation device with single pins in place. **C:** The second pin is added proximal and distal, providing secure fixation.

### **Technique Tips**

Pin sizes vary with manufacturers, as do drill sizes. In general the pins are placed through predrilled holes to avoid thermal necrosis of bone. Sharp drills should be used. The manufacturer's recommendation for drill and screw sizes should be checked before starting the procedure. At least two pins should be placed proximally and two distally. An intermediate pin may be beneficial.

### **Postoperative Care**

Pin care is critical, and avoiding tension at the skin–pin interface is beneficial. Large pin sites are cleaned with peroxide daily. Showering is allowed once the wound is stable and there is no communication between the pin and the fracture hematoma. Antibiotics are used liberally because pin site infections are common and easily resolved with antibiotic treatment, usually cephalosporin. The external fixation device remains until the fracture is healed. Dynamization is performed before removal, allowing the bone to be stressed to full body weight. The device should not be removed until three or four cortices demonstrate bridging bone continuous on AP and lateral radiographs, typically 3 to 4 months after injury.

### **Complications of External Fixation**

The most common complication of external fixation is pin track infection, which has been reported to occur in up to 72% of patients ( [111](#)). This complication generally is mild and easily treated with oral antibiotics and local pin site care. Sola et al. ( [119](#)) reported a decreased number of pin track infections after changing their pin care protocol from cleansing with peroxide to simply having the patient shower daily. Superficial infections should be treated aggressively with pin track releases and antibiotics. Deep infections are rare, but if present, drainage and antibiotic therapy are mandatory. Any skin tenting over the pins should be released at the time of application or at follow-up.

In a study of complications of external fixators for femoral fractures, Gregory et al. ( [91](#)) reported a 30% major complication rate and a 107% minor complication rate. Among the major complications were five refractures or fractures through pin sites. Skaggs et al. ( [117](#)), in reviewing the use of external fixation devices for femoral fractures at Los Angeles Children's Hospital, found a 12% rate of secondary fractures in 66 patients. Multivariate linear regression analysis showed no correlation between the incidence of refracture and the fracture pattern, percentage of bone contact after fixator application, type of external fixator used, or dynamization of the fracture. A statistically significant association was found between the number of cortices demonstrating bridging callus on both the AP and lateral views at the time of fixator removal and refracture. Fractures with fewer than three cortices with bridging callus had a 33% risk of refracture, whereas those with three or four cortices showing bridging callus had only a 4% rate of refracture. Other reports in the literature with smaller numbers, but still substantial experience, document refracture rates as high as 20% with more significant complications ( [84,92,99,113,115,117](#)).

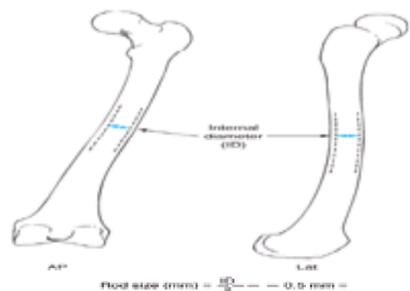
In 1997, in a follow-up of the original article by Aronson and Tursky ( [71](#)), Blasier et al. ( [76](#)) reported 139 femoral fractures treated with external fixation between 1984 and 1993. The average age at treatment was 8.9 years, and the average time to healing was 11.4 weeks. Only 18 patients had definitive radiographic follow-up, with 15 patients demonstrating overgrowth averaging 8.7 mm. In the series of Blasier et al., pin track infection was common, and there was a 2% incidence of fracture after removal of the device.

Although joint stiffness has been noted in some patients treated with external fixation, it is relatively uncommon in children with femoral fractures unless major soft tissue injury is present ( [85](#)).





**FIGURE 22-19.** **A:** The initial anteroposterior radiography of the 8-year-old child shows an oblique femoral fracture, but there is also a nondisplaced butterfly fragment (*arrows*). **B:** Flexible rods may be used in such a case, being careful not to displace the fragment and **(C)** burying the rods deep in the proximal femoral metaphysis to provide satisfactory fixation.



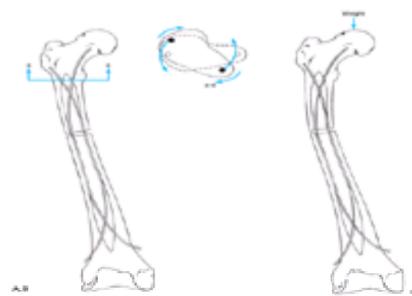
**FIGURE 22-20.** To determine the size of titanium flexible rods to be used, measure the diaphyseal internal diameter on both the anteroposterior and lateral views, divide by 2, and subtract 0.5 mm. Use the smaller of the number obtained from the anteroposterior and lateral views. AO rods range from 2.0 to 4.0 mm in diameter, in 0.5-mm increments.

The procedure can be performed either on a fracture table or on a fluoroscopy table, but reduction must be documented with fluoroscopy before insertion of the rods. The procedure is described with the use of a fracture table, but in small children (<80 pounds), we find this easier to do without a fracture table. The procedure has been described with AO titanium elastic rods, but other devices are available and can be used with slight variations in procedure.

### Rod Bending

The distance from the top of the inserted rod to the level of the fracture site is measured, and a gentle 30-degree bend is placed in the nail with the apex at what will be the level of the fracture. Next the rod tip is bent to facilitate placement, and to allow the rod to bounce off the opposite cortex at the time of insertion. This also facilitates spreading of the rods in the proximal metaphysis, either in the femoral neck or in the greater trochanter. Some surgeons prefer to avoid the second bend at the level of the fracture and use these rods as straight rods, much as in traditional intramedullary fixation. Although not consistent with the recommendation of Lascombes, satisfactory results have been achieved with this technique, especially with the stronger stainless-steel rods. The rods used generally are 3.0 to 4.0 mm in diameter, depending on the size of the bone and the child. Two rods of the same size should be used, and varying sizes should be avoided.

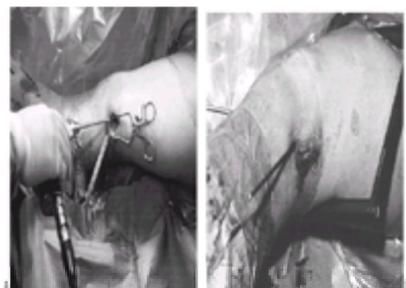
The technique of elastic fixation of femoral fractures as described by Ligier et al. ([105](#)) requires that a bend be placed in the mid-portion of the rod at the level of the fracture site. This produces a spring effect ([Fig. 22-21](#)) that adds to the rigidity of the fracture fixation. The spread of the rods in opposite directions provides a “prestressed” fixation, which increases resistance to bending. The opposite bends of the two rods at the level of the fracture significantly increase resistance to varus and valgus stress, as well as torsion.



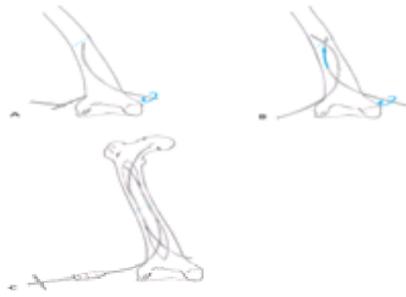
**FIGURE 22-21.** **A:** Stability from flexible rods comes from proper technique. **B:** Torsional stability results from divergence of the rods in the metaphysis. **C:** Resistance to sagittal and coronal bending results from spreading of the prebent rods through the diaphysis, as well as the size and material properties of the rods. Elastic rods return to their predetermined alignment when loaded unless plastic deformation occurs.

### Retrograde Insertion

After the child is placed on the fracture table, the leg is prepared and draped with the thigh (hip to knee) exposed ([Fig. 22-22](#)). The image intensifier is used to localize the placement of skin incisions by viewing the distal femur in the AP and lateral planes. Incisions are made on the medial and lateral side distal to the insertion site in the bone. The top of the 3-cm incision should be at or just distal to the level of the insertion site, which is about 2.5 to 3 cm proximal to the distal femoral physis ([Fig. 22-23](#)). A 4.5-mm drill bit or awl is used to make a cortical hole in the bone.



**FIGURE 22-22. A:** In children weighing more than 80 pounds, the fracture table may be used to provide traction and reduction of a femoral fracture. The leg is draped free, allowing medial and lateral rods to be placed. **B:** A radiolucent reduction bar may be beneficial to reduce angular deformity when passing the rods.



**FIGURE 22-23. Retrograde insertion. A:** The rods are inserted through a distal cortical hole 4.5 mm in diameter made either with a drill or an awl 2.5 cm above the physis. **B:** Rods are advanced to the level of the fracture site by rotating (reciprocally) or pounding with an inserter and mallet. The fracture is reduced and one rod is driven across the fracture site 3 to 5 cm. **C:** Then the second rod is driven into the proximal fragment.

Rods are inserted from the medial and lateral side and driven up to the level of the fracture. The distal femoral metaphysis is opened using a drill or awl at a point 2.5 cm proximal to the distal femoral physis. The drill is then inclined 10 degrees anteriorly and steeply angled in the frontal plane to facilitate passage of the nail through the dense pediatric metaphyseal bone. Upon insertion the rod glances off the cortex as it advances toward the fracture site. Both medial and lateral rods are inserted to the level of the fracture. At this point the fracture is reduced using longitudinal traction, as well as a fracture reduction tool ( Fig. 22-22B). This tool is radiolucent and holds the unstable femoral fracture in the appropriate position to allow fixation. After the first rod is driven across the fracture, approximately 2 to 3 cm, the second rod is driven across the fracture. The two rods then are driven into the proximal end of the femur with one driven toward the femoral neck and the other toward the greater trochanter. When placing the second rod across the fracture site and rotating it, care must be taken not to wind one rod around the other. After the rods are driven across the fracture and before they are seated, fluoroscopy is used to confirm satisfactory reduction of the fracture and to ensure that the rods have not comminuted the fracture as they were driven into the proximal fragment.

The rods are pulled back approximately 2 cm, the end of each rod is cut, and the rods are driven back securely into the femur. The end of the rod should lie adjacent to the bone of the metaphysis but should be at least 1 cm distal to the insertion hole to allow ease in later removal. Bending the rod ends should be avoided because it can cause a painful bursa over the rod end.

If the fracture is in the lower portion of the femur and perhaps even if it is diaphyseal, a proximal insertion site should be considered. The insertion site may be anterolateral just below the greater trochanter or through the lateral border of the trochanter. Through a skin incision at the level of the trochanter, two 4.5-mm holes are drilled into the metaphysis just below the apophysis and connected with a rongeur. The rods are inserted antegrade. The sizing of the rods and postoperative management are the same as in the retrograde technique.

#### Technique Tips

Mazda et al. (109) emphasized that for insertion of titanium elastic nails, the nails have to be bent into an even curve over the entire length, and the summit of the curve has to be at the level of the fracture or very close to it in comminuted fractures. The depth of curvature should be about three times the diameter of the femoral canal. Flynn et al. (87) also stressed the importance of contouring both nails with similar gentle curvatures, choosing nails that are 40% of the narrowest diaphyseal diameter, and using medial and lateral starting points that are at the same level in the metaphysis.

#### Postoperative Management

For unstable long oblique fractures or comminuted fractures, immobilization with a cast or traction may be of benefit. To decrease knee pain and quadriceps spasm, we have found the use of a knee immobilizer to be beneficial in the early postoperative course. Physical therapy with touchdown weight bearing should begin as soon as the patient is comfortable. Gentle knee exercises and quadriceps strengthening can be begun, but there should be no aggressive passive motion of the knee, which increases the motion at the fracture site and increases quadriceps spasm. Postoperative knee motion does return to normal, but this requires time. Full weight bearing generally is tolerated by 6 weeks.

The rod may be removed 6 to 12 months after injury when the fracture is fully healed, usually as an outpatient procedure.

#### Complications of Flexible Intramedullary Nailing

Complications are relatively infrequent after flexible intramedullary nailing. In 308 fractures reported in six studies ( 72,81,86,96,105,108), no nonunions, infections, or occurrences of avascular necrosis were reported. Approximately 12% of patients had malunions, most often mild valgus or varus deformities, and approximately 3% had clinically significant leg length discrepancies from either overgrowth or shortening. Mazda et al. ( 108) pointed out a technique-related complication that occurred in 10 of their 34 patients: rods were left too long and caused painful bursae and limited knee flexion. All 10 patients had the nails removed 2 to 5 months after surgery. In a multicenter study that included 58 femoral fractures stabilized with titanium elastic nails, irritation of the soft tissue near the knee by the nail tip occurred in 4 patients (7%), leading to a deeper infection in 2 patients. This study also reported one refracture after premature nail removal, leading to a recommendation that nail removal be delayed until callus is solid and the fracture line is no longer visible.

#### Rigid Intramedullary Rod Fixation

Problems with angular malalignment and maintenance of length can be avoided with the use of rigid intramedullary fixation. Interlocking proximally and distally to maintain length and rotational alignment in unstable fractures, as in adults, appears to be of benefit to some adolescents ( 74,78,93). Beaty et al. (73) reported the use of interlocking intramedullary nails for the treatment of 31 femoral shaft fractures in 30 patients 10 to 15 years of age. All fractures united, and the average leg length discrepancy was 0.51 cm. No angular or rotational malunions occurred. All nails were removed at an average of 14 months after injury; no refracture or femoral neck fracture occurred after nail removal. One case of avascular necrosis of the femoral head occurred, which was thought to be secondary to injury to the ascending cervical artery during nail insertion. A poll of the members of the Pediatric Orthopaedic Society disclosed 14 patients with avascular necrosis in approximately 1,600 femoral fractures. Despite the use of a "safe" transtrochanteric insertion site for antegrade femoral rodding, a case of avascular necrosis has been reported. Buford et al. (79) showed in their MRI study of hips after antegrade rodding that subclinical avascular necrosis may be present. Antegrade rodding through the trochanter or the upper end of the femur appears to be associated with a risk of avascular necrosis in children with open physes, regardless of chronologic age.

Reamed antegrade nailing in children under 12 years of age is not recommended because of proximal femoral growth abnormalities ( 163), the risk of avascular necrosis of the femoral head (56,73,110,121), the size of the proximal femur, and the relative success of other treatment methods. However, Maruenda-Paulino et al. (107) reported good results using 9-mm Kuntscher rods in children 7 to 12 years of age, and Beaty et al. ( 73) reported the use of newer pediatric "intermediate" interlocking nails for femoral canals with diameters as small as 8 mm. In children 12 to 17 years of age, antegrade locked transtrochanteric fixation may occasionally be indicated for an unstable fracture in a large adolescent.

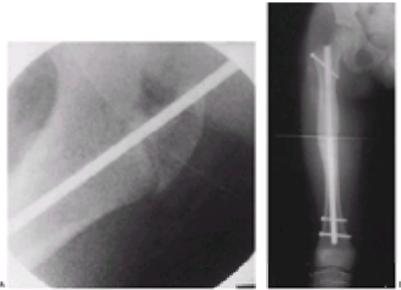
Open fractures in older adolescents can be effectively treated with intramedullary rodding, either as delayed or primary treatment, including those caused by gunshot wounds and high-velocity injuries ( 75,123). Antegrade intramedullary rod insertion maintains length, prevents angular malunion and nonunion, and allows the patient

to be rapidly mobilized and discharged from the hospital. However, other techniques with fewer potential risks should be considered.

### **Antegrade Intramedullary Nailing: Technique**

The child is placed either supine or in the lateral decubitus position on a fracture table. The upper end of the femur is approached through a 5-cm longitudinal incision proximal to the greater trochanter and in line with the femoral shaft. The skin incision can be precisely placed after localization on both the AP and lateral views. The gluteus maximus muscle is spread, and the gluteus medius muscle identified. The rod should be inserted through the gluteus medius muscle insertion in the tip of the trochanter, because posterior dissection may place the vasculature proximally at risk for injury. Identifying the piriformis fossa with vigorous dissection and placement of anterior and posterior retractors to the femoral neck also may cause injury to the lateral ascending cervical artery and should be avoided. Dissection should be limited to the lateral aspect of the greater trochanter and not into the piriformis fossa. This prevents dissection near the origin of the lateral ascending cervical artery medial to the piriformis fossa.

A threaded-tip guidewire is inserted into the proximal edge of the greater trochanter, followed by reaming with a 9-mm reamer ( Fig. 22-24). A ball-tip guidewire then can be inserted into the proximal femur. No dissection should be performed medial or posterior to the insertion site. The ball-tip guidewire is driven across the fracture site and into the distal fragment to a level just proximal to the distal femoral physis. Progressive reaming is performed before a reamed rod or a nonreamed rod is chosen. The smallest rod that maintains contact with the femoral cortices is used (generally 8 or 9 mm) and is locked proximally and distally. Only one distal locking screw is necessary, but two can be used (66). Rods that have an expanded proximal cross-section should be avoided. The proximal end of the nail should be left slightly long (up to 1 cm) to make later removal easier.



**FIGURE 22-24.** Antegrade insertion of intramedullary nail. **A:** Guidewire is placed at the medial edge of the greater trochanter, piercing the gluteus medius muscle and avoiding posterior insertion. **B:** Three months after fixation of a femoral shaft fracture in an 11-year-old child with multiple trauma and a closed head injury. A 9-mm pediatric locked intramedullary nail was used.

### **Technique Tips**

Dissection should be limited to the tip of the greater trochanter, without extending to the capsule or mid-portion of the femoral neck. Leaving the proximal end of the nail up to 1 cm long makes later removal easier.

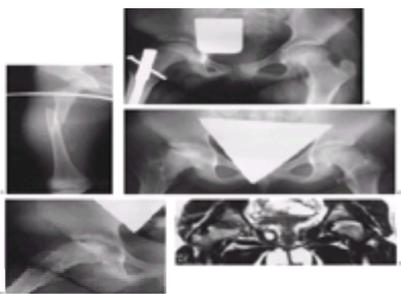
### **Postoperative Management**

Nails can be removed 9 to 18 months after radiographic union to prevent bony overgrowth over the proximal tip of the nail. Dynamization with removal of the proximal or distal screw generally is not necessary.

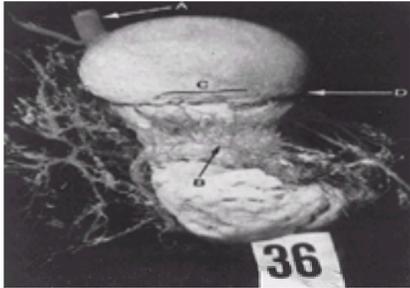
### **Complications of Locked Intramedullary Nailing**

Although good results have been reported with this technique and patient satisfaction is high, problems with proximal femoral growth, avascular necrosis, and leg length discrepancy cannot be ignored.

In a series of intramedullary nailing of 31 fractures, Beaty et al. ( 73) reported one patient with segmental avascular necrosis of the femoral head ( Fig. 22-25), which was not seen radiographically until 15 months after injury. Kaweblum et al. ( 101) reported a patient with avascular necrosis of the proximal femoral epiphysis after a greater trochanteric fracture, suggesting that the blood supply to the proximal femur may have been compromised by vascular disruption at the level of the greater trochanter during rod insertion. Other researchers have reported single patients with avascular necrosis of the femoral head after intramedullary nailing ( 110,112,121). Chung (82) noted the absence of transphyseal vessels to the proximal femoral epiphysis and demonstrated that the singular lateral ascending cervical artery predominantly supplies blood to the capital femoral epiphysis ( Fig. 22-26). He stated that all of the epiphyseal and metaphyseal branches of the lateral ascending cervical artery originate from a single stem that crosses the capsule at the trochanteric notch. Because the space between the trochanter and the femoral head is extremely narrow in children under 8 years of age, this single artery is vulnerable to injury and appears to be so until skeletal maturity, regardless of chronologic age.



**FIGURE 22-25.** **A:** Isolated femoral shaft fracture in 11½-year-old child. **B:** One month after fixation with an intramedullary nail, femoral head appears normal. **C:** Eight months after injury, fracture is healed; note early signs of avascular necrosis of right femoral head. **D:** Fifteen months after injury, segmental avascular necrosis of the femoral head is evident on radiographs. **E:** Magnetic resonance image shows extent of avascular necrosis of right femoral head. (D reprinted from Beaty JH, Austin SM, Warner WC, et al. Interlocking intramedullary nailing of femoral-shaft fractures in adolescents: preliminary results and complications. *J Pediatr Orthop* 1994;14:178–183; with permission.)



**FIGURE 22-26.** The single ascending cervical artery (A) is the predominant blood supply to the femoral head. The vessel is at risk during antegrade insertion of an intramedullary rod. (Reprinted from Chung S. The arterial supply of the developing proximal end of the femur. *J Bone Joint Surg [Am]* 1976;58:961; with permission.)

Townsend and Hoffinger (124) reported no avascular necrosis in 34 patients in whom a trochanteric tip starting point was used.

Skak et al. (118), in a long-term study of 52 children and adolescents with femoral fractures treated with plating and intramedullary rodding, found that excessive overgrowth was less common with nailing than with plating. Three patients had moderate valgus deformities of the hip and one had late arthrosis after intramedullary rodding despite using a transtrochanteric approach. Skak et al. (118) concluded that open physes were a contraindication to antegrade rodding even with a transtrochanteric approach. Thometz and Handen (121) also documented an association between avascular necrosis and antegrade rodding in an adolescent.

Growth abnormality in the proximal femur may occur with arrest of the greater trochanteric physis. Although most growth from the greater trochanter after 8 years of age is appositional, Raney et al. (163) reported five patients who developed coxa valga and mild hip subluxation from trochanteric physal arrest after antegrade nailing. These patients ranged in age from 9 to 13 years, suggesting that even in older children greater trochanteric physal arrest occasionally can produce clinical problems. Beaty et al. (73) reported two patients with overgrowth of more than 2.5 cm that required epiphysiodesis because of leg length discrepancy.

The proximal femoral physis is a continuous cartilaginous plate between the greater trochanter and the proximal femur in young children. Interference with the physis may result in atrophy of the femoral neck, placing the child at a small risk for subsequent femoral neck fracture (116). Antegrade rodding with reaming of a large defect also may result in growth disturbance in the proximal femur as well as femoral neck fracture (Fig. 22-27). Beaty et al. (73) reported no “thinning” of the femoral neck in their patients, which they attributed to an older patient group (10–15 years of age) and design changes in the femoral nail that allowed a decrease in the cross-sectional diameter of the proximal portion to 10 mm.

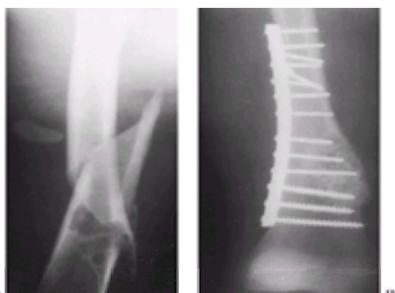


**FIGURE 22-27.** Fifteen-year-old boy 3 years after intramedullary nailing of the right femur. Acetabular to trochanter distance increased by 1.5 cm; note partial trochanteric epiphysiodesis (arrow) with mild overgrowth of the femoral neck. (Reprinted from Beaty JH, Austin SM, Warner WC, et al. Interlocking intramedullary nailing of femoral-shaft fractures in adolescents: preliminary results and complications. *Pediatr Orthop* 1994;14:178–183; with permission.)

### Open Reduction and Internal Fixation with Plate and Screws

Ward et al. (125), Kregor et al. (103), Fyodorov et al. (88), and Hansen (94) recommended this form of treatment in selected femoral fractures. Ward et al. (125) reported the use of AO compression plates for the treatment of femoral shaft fractures in 25 children 6 to 16 years of age, 22 of whom had associated fractures or multisystem injuries. Follow-up was available for 24 patients, 23 of whom (96%) had healed fractures at an average of 11 weeks. According to these investigators, plate fixation offers the advantages of anatomic reduction, ease of insertion, simplified nursing care, rapid mobilization without casting, and applicability to any size femoral shaft. Disadvantages of plate fixation include the long incision necessary and the risks of plate breakage and stress fracture after plate removal. They recommend plate fixation only for children under 11 years of age with closed-head injuries or multiple trauma. All the patients reported by Kregor et al. (103) had multiple trauma, and this form of treatment was selected to stabilize the femoral fracture quickly and mobilize the patient. Fyodorov et al. (88) reviewed 21 patients in whom 4.5-mm DCP plates were used for fixation. Patients were kept non-weight bearing on crutches for 8 weeks. There were two plate breakages requiring revision and one requiring spica casting to achieve healing. Modern techniques of femoral plating (114) with maintenance of the periosteum may lead to greater acceptance of this method of management.

Pathologic fractures, especially in the distal femoral metaphysis, create larger areas of bone loss that can be treated with plate fixation and immediate bone grafting to avoid protracted periods of traction and immobilization (Fig. 22-28).



**FIGURE 22-28.** A: Pathologic fracture (nonossifying fibroma) of the femoral diaphysis. B: Plate fixation at the time of biopsy and bone grafting.

### Technique

In a sterile operating room environment after general anesthesia has been given, the patient is placed on a radiolucent operating table and the entire extremity is prepared and draped. The femur is approached laterally. The vastus lateralis is retracted anteriorly to expose the femur. Soft tissue attachment to the bone is

preserved to the extent possible. Fragments are lagged into place and secured with a dynamic compression plate. A 4.5-mm or larger compression plate is used in children (Fig. 22-29). If the fracture is at either end of the bone, leaving insufficient bone for cortical fixation, fully threaded cancellous screws add to the stability of fixation and are preferable to either cortical or partially threaded cancellous screws. Both interfragmentary compression and dynamic compression techniques can be used to achieve stability and anatomic alignment.



**FIGURE 22-29. A:** This oblique mid-shaft femoral fracture with some comminution in a 200-pound male occurred after an automobile accident. **B:** Stable fixation with plate and lag screw construction is highly successful and carries no risk of avascular necrosis.

### Technique Tips

Soft tissue stripping should be limited, and three screws should be used proximal and distal to the fracture site.

### Postoperative Management

Protected weight bearing is progressed to weight bearing to tolerance. Active range of motion of the hip and knee is encouraged.

### Complications of Plate Fixation

Extensive dissection and periosteal stripping during plate application may lead to overgrowth. Overgrowth was not a significant problem in the series of Kregor et al. (103), with an average increase in length of 0.9 cm (range 0.5–1.5 cm), but Ward et al. (125) reported several patients with considerable overgrowth (approximately 1 inch), and Hansen (94) reported overgrowth of an inch in a 12-year-old boy, suggesting that overgrowth is possible in children over 10 years of age. More data are necessary on this issue to make a definitive statement.

Fyodorov, Sturm, and Robertson (88) reported hardware failure in 2 of 23 femoral shaft fractures treated with dynamic compression plating. One was treated with revision plating and the other with spica cast; both fractures healed uneventfully. No other complications were noted in their patients.

Refracture is rare, and whether bone atrophy under a plate is caused by stress shielding or by avascularity of the cortex is unknown. Although still somewhat controversial, the plate and screws are generally removed at 1 year after fracture to avoid fracture at the end of the plate.

Quadriceps strength after plate fixation appears not to be compromised, (147) relative to intramedullary fixation or cast immobilization.

## AUTHORS' PREFERRED METHOD OF TREATMENT

For stable femoral fractures in children under 6 months of age, we use a posterior splint or Pavlik harness. If the fracture is unstable, usually the proximal fragment is flexed and a Pavlik harness is the ideal device for reducing and holding the fracture. The use of a Pavlik harness requires an attentive and compliant caregiver. Traction with a spica cast occasionally is needed in this group. Abuse and metabolic bone disease must be considered in an infant with a femoral fracture.

In children 6 months to 6 years of age, an immediate spica cast with Gortex liner is almost always the treatment of choice. If length or alignment cannot be maintained in an immediate spica cast, traction followed by casting can be used. We usually use a distal femoral traction pin and place the child in a 90/90 or oblique position in the bed for traction. It is of note, however, that over 95% of young children with femoral fractures can be managed with immediate spicas with a low complication rate and low cost. In children with multiple trauma, either flexible intramedullary rodding or external fixation is chosen, based on the fracture anatomy and the soft tissue injury.

In children 6 to 12 years of age, flexible intramedullary rodding is frequently used, most often with retrograde insertion. External fixation is used for unstable fracture patterns, comminuted fractures, and fractures with severe soft tissue injury. Immediate spica casts may be used for the most stable fractures in this age group. In children 12 years to maturity, we use flexible intramedullary rods as our treatment of choice. In a patient with an unstable fracture configuration, we would use either a plate or an antegrade rod, recognizing the low but significant risk of avascular necrosis of the femoral head. Plating is preferable for subtrochanteric and supracondylar fractures of the femur, whereas intramedullary nails are ideal for mid-shaft fractures. If antegrade rodding is chosen, a transtrochanteric approach is used. New methods are under development for fixation, using an infratrochanteric approach with a locked type of flexible rod, but such devices are not available yet.

## COMPLICATIONS OF FEMORAL SHAFT FRACTURES

### Leg Length Discrepancy

The most common sequela after femoral shaft fractures in children is leg length discrepancy. The fractured femur may be initially short from overriding of the fragments at union; growth acceleration occurs to “make up” the difference, but often this acceleration continues and overgrowth occurs. The potential for growth stimulation from femoral fractures has long been recognized, but the exact cause of this phenomenon is still unknown. Growth acceleration has been attributed to age, sex, fracture type, fracture level, handedness, and the amount of overriding of the fracture fragments. Age seems to be the most constant factor, but fractures in the proximal third of the femur and oblique comminuted fractures also have been associated with relatively greater growth acceleration. According to Staheli (170), in patients over 10 years of age, shortening is more likely; in patients 2 to 10 years of age, overgrowth is more likely, especially if traction has been used.

### Shortening

Because the average overgrowth after femoral fracture is approximately 1.5 cm, shortening of 2 to 3 cm in the cast is the maximal acceptable amount. The maximal acceptable shortening depends on the age of the child; for example, in a 6-year-old child, 2.5 cm may be acceptable, whereas only 1 to 2 cm should be accepted in a 14-year-old approaching skeletal maturity. In patients 2 to 10 years of age with more than 3 cm of shortening after immediate spica casting, the cast is removed, traction is reapplied until acceptable length is obtained, and then a new cast is applied. For early shortening of more than 3 cm in a patient 11 or 12 years old, a reinstatement of traction and reapplication of the cast also may be appropriate. If, however, the shortening is unacceptable at 6 weeks after fracture, the decision must be made as to whether osteoclasis and distraction with external fixation is preferable to a later limb equalization procedure (lengthening or shortening). The trend is to correct the shortening immediately with external fixation if possible.

### Overgrowth

Overgrowth after femoral fracture is common in children 2 to 10 years of age. The average overgrowth is 0.9 cm, with a range of 0.4 to 2.5 cm (169). Overgrowth

occurs whether the fracture is short, at length, or overpulled in traction at the time of healing. In general, overgrowth occurs most rapidly during the first 2 years after fracture and to a much lesser degree for the next year or so (148).

Truesdell (173) first reported the phenomenon of overgrowth in 1921, and many researchers since have verified the existence of growth stimulation after fracture (129,130,131,132,138,139,142,145,158,159). The relationship of the location of the fracture to growth is somewhat controversial. Staheli (170) and Malkawi (154) reported that overgrowth was greatest if the fracture occurred in the proximal third of the femur, whereas Henry (151) stated that the most overgrowth occurred in fractures in the distal third of the femur. Other investigators have found no relationship between fracture location and growth stimulation (145,151,164); Shapiro's data (169) support the lack of relationship. The relationship between fracture type and overgrowth also is controversial. In general, most researchers believe that no specific relationship exists between fracture type and overgrowth, but some have reported overgrowth to be more frequent after spiral, oblique, and comminuted fractures associated with greater trauma.

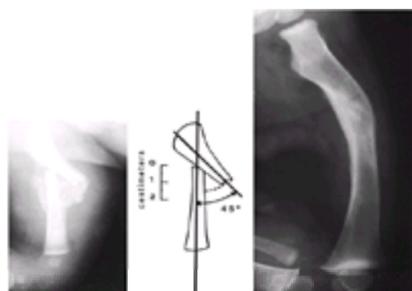
### Angular Deformity

Some degree of angular deformity is frequent after femoral shaft fractures in children, but this usually remodels with growth. Angular remodeling occurs at the site of fracture with appositional new bone formation in the concavity of the long bone. Differential physeal growth also occurs in response to diaphyseal angular deformity. Wallace and Hoffman (176) stated that 74% of the remodeling that occurs is physeal, and appositional remodeling at the fracture site occurs to a much lesser degree. However, this appears to be somewhat age dependent. It is clear that angular remodeling occurs best in the direction of motion at the adjacent joint (176). That is, anterior and posterior remodeling in the femur occurs rapidly and with little residual deformity. In contrast, remodeling of a varus or valgus deformity occurs more slowly. The differential physeal growth in a varus or valgus direction in the distal femur causes compensatory deformity, which is usually insignificant. In severe varus bowing, however, a hypoplastic lateral condyle results, which may cause a distal femoral valgus deformity if the varus bow is corrected (Fig. 22-30).



**FIGURE 22-30.** Long-standing varus deformity in the femoral shaft may lead to hypoplasia of the lateral condyle and a compensatory distal femoral valgus deformity after correction of the varus of the shaft.

Guidelines for acceptable alignment vary widely. The range of acceptable anterior and posterior angulation varies from 30 to 40 degrees in children up to 2 years of age to 10 degrees in older children and adolescents (Fig. 22-31) (157). The range of acceptable varus and valgus angulation also becomes smaller with age. Varus angulation in infants and children should be between 10 and 15 degrees, although greater degrees of angulation may have a satisfactory outcome. Acceptable valgus angulation is 20 to 30 degrees in infants, 15 to 20 degrees in children up to 5 years of age, and 10 degrees in older children and adolescents. The muscle mass of the femur generally hides femoral deformity from direct observation. The acceptability of femoral deformity, in general, is a direct function of the degree of difficulty in changing the deformity and the appearance of the leg.



**FIGURE 22-31.** Remodeling potential of the femur during infancy. This infant sustained a femoral fracture during a breech delivery and was placed in a spica cast but with insufficient flexion of the hip. **Left:** At 3 weeks union is evident with about 45 degrees of angulation in the sagittal plane and 1.5 cm of overriding. **Center:** Line drawing demonstrating true angulation. **Right:** Twelve months later the anterior angulation has reduced to a level such that it was not apparent to the family, and the shortening has reduced to less than 1 cm.

Late development of genu recurvatum deformity of the proximal tibia after femoral shaft fracture has been most often reported as a complication of traction pin or wire placement through or near the anterior aspect of the proximal tibial physis, excessive traction, pin track infection, or prolonged cast immobilization (68). However, proximal tibial growth arrest may complicate femoral shaft fracture, presumably as a result of occult injury (152). Femoral pins are preferred for traction, but if tibial pins are required, the proximal anterior tibial physis must be avoided (51). Femoral traction pins should be placed one or two finger breadths proximal to the superior pole of the patella to avoid the distal femoral physis.

If significant angular deformity is present after fracture union, corrective osteotomy should be delayed for at least a year unless the deformity is severe enough to impair function. This will allow determination of remodeling potential before deciding that surgical correction is necessary. The ideal osteotomy corrects the deformity at the site of fracture. In juvenile patients, however, metaphyseal osteotomy of the proximal or distal femur may be necessary. In adolescents with mid-shaft deformities, diaphyseal osteotomy and fixation with an interlocking intramedullary nail are preferable.

### Rotational Deformity

According to Verbeek (174), rotational deformities of 10 degrees to more than 30 degrees occur in one third of children after conservative treatment of femoral shaft fractures. Malkawi et al. (158) found asymptomatic rotational deformities of less than 10 degrees in two thirds of their 31 patients. Torsional deformity usually is expressed as increased femoral anteversion on the fractured side compared with the opposite side, as demonstrated by anteversion views; a difference of more than 10 degrees has been the criterion of significant deformity. However, Brouwer et al. (136) challenged this criterion, citing differences of 0 to 15 degrees in a control group of 100 normal volunteers. The accuracy of measurements from plain radiographs also has been disputed, and Norbeck et al. suggested the use of computed tomographic (CT) scanning for greater accuracy.

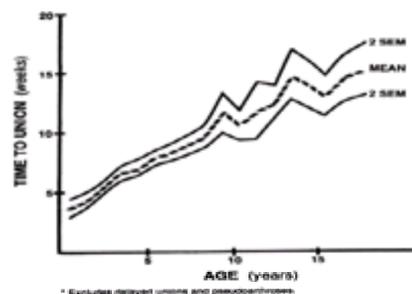
Rotational remodeling in childhood femoral fractures is another controversy in the search for criteria on which to base therapeutic judgments. According to Davids (143) and Braten et al. (135), up to 25 degrees of rotational malalignment at the time of healing of femoral fractures appears to be well tolerated in children. In their patients with more than 25 degrees of rotational malalignment, however, deformity caused clinical complaints. Davids found no spontaneous correction in his study of malunions based on CT measurements, but the length of follow-up is insufficient to state that no rotational remodeling occurs. Brouwer et al. (136) and others

(133,149,162,174) reported slow rotational correction over time.

Certainly, in older adolescents, no significant rotational remodeling will occur. In infants and juveniles, some rotational deformity can be accepted ( 146), because either true rotational remodeling occurs or functional adaptation allows resumption of normal gait. Up to 30 degrees of angular malrotation in the femur should result in no functional impairment unless there is preexisting rotational malalignment. The goal, however, should be to reduce a rotational deformity to 10 degrees, based on alignment of the proximal and distal femur radiographically, interpretation of skin and soft tissue envelope alignment, and correct positioning within a cast, based on the muscle pull on the proximal fragment. The distal fragment should be lined up with the position of the proximal fragment determined by the muscles inserted upon it (Fig. 22-3).

### Delayed Union

Delayed union of femoral shaft fractures is uncommon in children. The time to fracture union in most children is rapid and age dependent. In children under 5 years of age, healing usually occurs in 4 to 6 weeks. In children 5 to 10 years of age, fracture healing is somewhat slower, requiring 8 to 10 weeks. Throughout adolescence, the time to healing continues to lengthen. By the age of 15 years, the mean time to healing is about 13 weeks, with a range from 10 to 15 weeks ( Fig. 22-32). The rate of healing also is related to soft tissue injury and type of treatment. Application of an external fixation device appears to delay callus formation and slow the rate of healing.

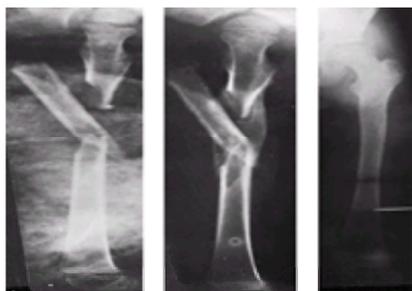


**FIGURE 22-32.** Time required for union of femoral shaft fractures in childhood and adolescence. (Redrawn from Skak SV, Jensen TT. Femoral shaft fracture in 265 children. *Acta Orthop Scand* 1988;59:704–707; with permission.)

Bone grafting and internal fixation with either a compression plate or locked intramedullary nail is the usual treatment for delayed union in older children and adolescents. Delayed union of a femoral fracture treated with casting in a child 1 to 6 years of age is probably best treated by continuing cast immobilization until bridging callus forms or (rarely) by additional bone grafting.

### Nonunion

Nonunions of pediatric femoral fractures are rare ( 156). They tend to occur in adolescents, in infected fractures, or in fractures with segmental bone loss or severe soft tissue loss. Most nonunions in children are of tibial fractures; femoral fractures account for only 15% of nonunions in children. Even in segmental fractures with bone loss, young children may have sufficient osteogenic potential to fill in a significant fracture gap ( Fig. 22-33) (160). For the rare femoral shaft nonunion in a child 5 to 10 years of age, bone grafting and plate-and-screw fixation have been traditional treatment methods, but more recently insertion of an interlocking intramedullary nail and bone grafting have been preferred, especially in children over 10 to 12 years of age.



**FIGURE 22-33.** The effectiveness of remodeling of the femur in a child. **Left:** Comminuted fracture in an 8-year-old child managed with a femoral pin incorporated in a spica cast. The mid-fragment is markedly angulated. **Center:** Fracture after union 12 weeks later with filling in of the defect and early absorption of the protruding fragment. **Right:** Appearance at age 12 with only a minimal degree of irregularity of the upper femur remaining.

Robertson et al. (166) reported the use of external fixators in 11 open femoral fractures. The time to union was delayed, but a satisfactory outcome occurred without subsequent procedures. This supports the belief that the rates of delayed union and nonunion are low in pediatric femoral fractures, because open fractures would have the highest rates of delayed union.

### Muscle Weakness

Weakness after femoral fracture has been described in the hip abductor musculature, quadriceps, and hamstrings, but persistent weakness in some or all of these muscle groups seldom causes a clinical problem. Hennrikus et al. ( 150) found that quadriceps strength was decreased in 30% of his patients and 18% had a significant decrease demonstrated by a one-leg hop test. Thigh atrophy of 1 cm was present in 42% of patients. These deficits appeared to be primarily related to the degree of initial displacement of the fracture. Finsen et al. ( 147) found hamstring and quadriceps deficits in patients with femoral shaft fractures treated with either rods or plates.

Damholt and Zdravkovic (141) documented quadriceps weakness in approximately one third of patients with femoral fractures, and Viljanto et al. ( 175) reported that this weakness was present whether patients were treated operatively or nonoperatively. Biyani ( 134) found that hip abductor weakness was related to ipsilateral fracture magnitude, long intramedullary rods, and, to a lesser degree, heterotopic ossification from intramedullary rodding.

Injury to the quadriceps muscle probably occurs at the time of femoral fracture, and long-term muscle deficits may persist in some patients regardless of treatment. Severe scarring and contracture of the quadriceps occasionally require quadricepsplasty ( 154).

### Infection

Infection may rarely complicate a closed femoral shaft fracture, with hematogenous seeding of the hematoma and subsequent osteomyelitis. Fever is commonly associated with femoral fractures during the first week after injury ( 171), but persistent fever or fever that spikes exceedingly high may be an indication of infection.

Pin-track infections occasionally occur with the use of skeletal traction, but most are superficial infections that resolve with local wound care and antibiotic therapy. Occasionally, however, the infections may lead to osteomyelitis of the femoral metaphysis or a ring sequestrum that requires surgical debridement.

### Neurovascular Injury

Nerve and vascular injuries are uncommon with femoral fractures in children (140,144,153,167). An estimated 1.3% of femoral fractures in children are accompanied by vascular injury (140,144,153,167), such as intimal tears, total disruptions, or injuries resulting in the formation of pseudoaneurysms (168). Vascular injury occurs most frequently with displaced Salter-Harris physeal fractures of the distal femur or distal metaphyseal fractures. If arteriography indicates that vascular repair is necessary after femoral shaft fracture, open reduction and internal fixation or external fixation of the fracture are recommended. Secondary limb ischemia also has been reported after the use of both skin and skeletal traction. Documentation of peripheral pulses at the time of presentation, as well as throughout treatment, is necessary.

Nerve abnormalities reported with femoral fractures in children include those caused by direct trauma to the sciatic or femoral nerve at the time of fracture and injuries to the peroneal nerve during treatment. Weiss et al. (69) reported peroneal nerve palsies in 4 of 110 children with femoral fractures treated with early 90/90 hip spica casting. They recommended extending the initial short leg portion of the cast above the knee to decrease tension on the peroneal nerve.

Riew et al. (165) reported 8 nerve palsies in 35 consecutive patients treated with locked intramedullary rodding. The nerve injuries were associated with delay in treatment, preoperative shortening, and boot traction. Resolution occurred in less than one week in 6 of 8 patients.

The natural history of peroneal nerve injury with femoral shaft fractures in children seems to be spontaneous correction. In infants, however, the development of an early contracture of the Achilles tendon is more likely. Because of the rapid growth in younger children, this contracture can develop quite early; and if peroneal nerve injury is suspected, the extremity should be braced until the peroneal nerve recovers. If peroneal, femoral, or sciatic nerve deficit is present at initial evaluation of a closed fracture, no exploration is indicated. If a nerve deficit occurs during reduction or treatment, the nerve should be explored. Persistent nerve loss without recovery over a 4- to 6-month period is an indication for exploration.

### Compartment Syndrome

Compartment syndromes of the thigh musculature are rare, but have been reported in patients with massive thigh swelling after femoral fracture and in patients treated with intramedullary rod fixation (161). If massive swelling of thigh musculature occurs and pain is out of proportion to that expected from a femoral fracture, compartment pressure measurements should be obtained and decompression by fasciotomy should be considered. It is probable that some patients with quadriceps fibrosis (164) and quadriceps weakness (150,172) after femoral fracture had intracompartmental pressure phenomenon. Vascular insufficiency related to Bryant's traction may produce signs of compartment syndrome with muscle ischemia (137). Janzing (155) reported the occurrence of compartment syndrome using skin traction for treatment of femoral fractures. Skin traction has been associated with compartment syndrome in the lower leg in both the fractured and nonfractured side. The association of compartment syndrome with Bryant's traction is well recognized. It is important to realize that in a traumatized limb, circumferential traction needs to be monitored closely and is contraindicated in the multiply injured or head-injured child.

## SPECIAL FRACTURES OF THE FEMORAL SHAFT

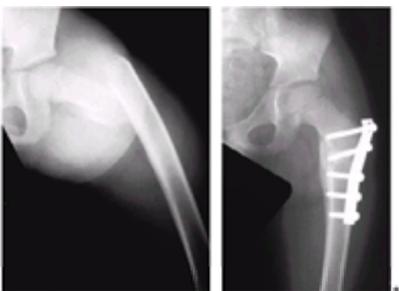
### Metaphyseal (Subtrochanteric and Supracondylar) Fractures

Subtrochanteric fractures can be treated in traction, followed by either a cast brace or single spica cast (Fig. 22-34) (177). Sponseller (181) reported satisfactory results with conservative treatment of subtrochanteric fractures in children. Internal fixation with plate and screw devices also can produce satisfactory results (179). Intramedullary fixation of subtrochanteric fractures in children is generally not indicated. In adolescents, there is insufficient experience with this fracture to determine at what age intramedullary fixation with a reconstruction-type nail and an angled transfixion screw into the femoral neck is indicated. Antegrade intramedullary nail systems place significant holes in the upper femoral neck and should be avoided. Unlike subtrochanteric fractures in adults, nonunions are rare in children with any treatment method.



**FIGURE 22-34.** Subtrochanteric fractures (A) may be treated with traction followed by a one-legged ambulatory spica cast (B).

With the admonition against intramedullary fixation in children with open physes combined with the fact that nonunions are rare, our recommendation is for traction followed by spica casting in young children up to approximately age 5 or 6, with plate and screw fixation in older children. The method of plate and screw fixation may be either a straight plate with angled screws in the proximal fragment (Fig. 22-35) or hip screw configuration systems appropriately sized to a small child.



**FIGURE 22-35.** A: This 6-year-old boy sustained a subtrochanteric fracture of the femur. B: He was treated with plate and screw fixation supplemented with a spica cast.

The traction technique should be that of 90/90 traction in which the distal fragment is flexed to align with the proximal fragment and spica cast treatment may be either in an ambulatory one-legged spica cast or a flexed hip/flexed knee spica cast, depending on the fracture and healing.

Supracondylar fractures are difficult to treat because the gastrocnemius muscle inserts just above the femoral condyles and pulls the distal fragment into a position of extension (178), making alignment difficult (Fig. 22-3). The traditional methods of casting and single-pin traction may be satisfactory (Fig. 22-36). If alignment cannot be achieved using these methods, however, open reduction and internal fixation or combined epiphyseal-metaphyseal traction can be used. Generally, internal fixation is preferable, either with plates and fully threaded cancellous screws if there is sufficient metaphyseal length to allow this or with crossed smooth Kirschner wires transfixing the fracture from the epiphysis to the metaphysis, as described for distal femoral physeal separations (180). If there is sufficient metaphyseal length, antegrade flexible rods can be used.



**FIGURE 22-36. A:** This 6-year-old patient sustained an unstable supracondylar fracture of the femur. **B:** The fracture was managed with immediate spica casting with the knee in 90 degrees of flexion, mandatory in such a case to prevent posterior angulation. **C:** Bayonet apposition is acceptable in a child this age.

### Open Femoral Fractures

Open femoral fractures are uncommon in children because of the large soft tissue compartment around the femur. Proper wound care, debridement, stabilization, and antibiotic therapy are required to reduce the chance of infection (178).

External fixation of open femoral shaft fractures simplifies wound care and allows early mobilization. The configuration of the external fixator is determined by the child's size and the fracture pattern. Generally, monolateral half-pin frames are satisfactory, but thin-wire circular frames may be necessary if bone loss is extensive. External fixation provides good fracture control, but as always, family cooperation is required to manage pin and fixator care.

Plate fixation also allows early mobilization, as well as anatomic reduction of the femoral fracture. Wound care and treatment of other injuries are made easier in children with multiple trauma. However, this is an invasive technique with the potential for infection and additional injury to the already traumatized soft tissues in the area of the fracture. In emergency situations, plate fixation can be used for Gustilo-Anderson type I and II fractures; type III fractures in older adolescents are better suited for external fixation or intramedullary nailing. Plate breakage can occur if bone grafting is not used for severe medial cortex comminution.

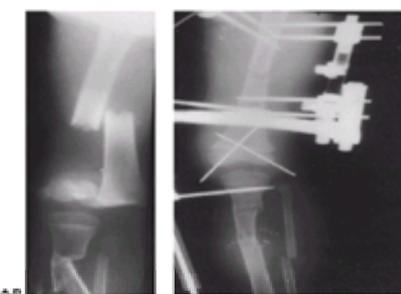
In older adolescents, plating or flexible intramedullary nailing is especially useful. Closed nailing after irrigation and drainage of the fracture allows early mobilization and easy wound care in patients with Gustilo-Anderson type I, II, IIIA, and IIIB injuries, but the risk of avascular necrosis must be recognized.

### Femoral Fractures in Patients with Metabolic or Neuromuscular Disorders

For patients with osteogenesis imperfecta who have potential for ambulation, surgical treatment with Rush or Bailey-Dubow rods is recommended for repeated fractures or angular deformity. Cast immobilization is usually avoided in patients with myelomeningocele or cerebral palsy because of the frequency of osteoporosis and refracture in these patients. If possible, existing leg braces are modified for treatment of the femoral fracture. In nonambulatory patients, a simple pillow splint is used.

### Floating Knee Injuries

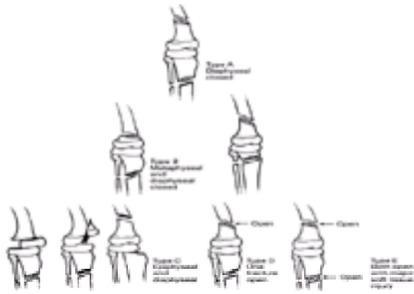
These rare injuries occur when ipsilateral fractures of the femoral and tibial shafts leave the knee joint "floating" without distal or proximal bony attachments (Fig. 22-37A). They are high-velocity injuries, usually resulting from collision between a child pedestrian or cyclist and a motor vehicle. Most children with floating knee injuries have multiple trauma, including severe soft tissue damage, open fractures, and often head injuries.



**FIGURE 22-37. A:** Floating knee injury in a 4-year-old girl: femoral shaft fracture, displaced physeal fracture of the distal femur, and open (type IIIB) proximal tibial fracture. **B:** After external fixation of femoral shaft fracture, closed reduction and percutaneous pinning of the distal femoral physeal fracture, and external fixation of the open tibial fracture; a free-flap was required for skin coverage.

In general, at least one of the fractures, usually the tibia, should be fixed. The femoral fracture can usually then be treated by the most appropriate option. If both fractures are open, external fixation of both the tibial and femoral fractures may be appropriate. If immediate mobilization is necessary, fixation of both fractures with external fixation, intramedullary nails, compression plates, or any combination of these may be indicated (Fig. 22-37B).

Letts and Vincent (18) described five patterns of ipsilateral tibial and femoral fractures and made treatment recommendations based on those patterns (Fig. 22-38). Because of the high prevalence of complications after closed treatment, Bohn and Durbin recommended open or closed reduction and internal fixation of the femoral fracture in older children. In children under 10 years of age, these investigators recommend 90/90 femoral pin traction for 4 weeks, followed by 1-½ spica casting until fracture union. The tibial fracture was immobilized in a below-knee cast during the traction. Bohn and Durbin listed four situations in which operative stabilization of the femoral fracture is indicated: (a) a child with severe head trauma with signs of posturing, (b) an adolescent near skeletal maturity, (c) a child with severe soft tissue injury, and (d) an inability to obtain acceptable closed reduction. Stabilization of the tibial fracture by external fixation is indicated if satisfactory closed reduction cannot be obtained or maintained and for open fractures with severe soft tissue injury, regardless of the age of the child.



**FIGURE 22-38.** Classification of floating knee injuries in children. (Redrawn from Letts M, Vincent N, Gouw G. The “floating knee” in children. *J Bone Joint Surg [Br]* 1986;68:442; with permission.)

Letts and Vincent (18) reported that maintaining adequate reduction by traction was difficult in children over 12 years of age and that complications were more frequent in children treated by traction alone. Complications in their patients treated by traction alone included pin track infection, osteomyelitis, limitation of knee motion, malunion, and leg length discrepancies of 1.5 cm or more. Bohn and Durbin reported that of 19 patients with floating knee injuries, at long-term follow-up 11 had limb length discrepancy secondary to either overgrowth of the bone after the fracture or premature closure of the ipsilateral physis (7 patients), genu valgum associated with fracture of the proximal tibial metaphysis (3 patients), or physeal arrest (1 patient). Four patients had late diagnoses of ligamentous laxity of the knee that required operation. Other complications included peroneal nerve palsy, infection, nonunion, malunion, and refracture.

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## CHAPTER REFERENCES

### Etiology, Diagnosis, and Classification

1. Beals RK, Tufts E. Fractured femur in infancy: the role of child abuse. *J Pediatr Orthop* 1983;3:583–586.
2. Bennett FS, Zinar DM, Kilgus DJ. Ipsilateral hip and femoral shaft fractures. *Clin Orthop* 1993;296:168–177.
3. Blakemore LC, Loder RT, Hensinger RN. Role of intentional abuse in children 1 to 5 years old with isolated femoral shaft fractures. *J Pediatr Orthop* 1996;16:585–588.
4. Buckley SL, Robertson WW Jr, Shalaby-Rana E. Stress fractures of the femoral diaphysis in young children. A report of 2 cases. *Clin Orthop Rel Res* 1995;310:165–169.
5. Buess E, Kaelin A. One hundred pediatric femoral fractures: epidemiology, treatment, attitudes, and early complications. *J Pediatr Orthop* 1998;7:186–192.
6. Burks RT, Sutherland DH. Stress fracture of the femoral shaft in children: report of two cases and discussion. *J Pediatr Orthop* 1984;4:614–616.
7. Cannon SR, Pool CJF. Traumatic separation of the proximal femoral epiphysis and fracture of the mid-shaft of the ipsilateral femur in a child: a case report and review of the literature. *Injury* 1984;15:156–158.
8. Ciarallo L, Fleisher G. Femoral fractures: are children at risk for significant blood loss? *Pediatr Emerg Care* 1996;12:343–346.
9. Daly KE, Calvert PT. Accidental femoral fracture in infants. *Injury* 1991;22:337–338.
10. Fry K, Hoffer M, Brink J. Femoral shaft fractures in brain-injured children. *J Trauma* 1976;16:371–373.
11. Gross RH, Stranger M. Causative factors responsible for femoral fractures in infants and young children. *J Pediatr Orthop* 1983;3:341–343.
12. Hedlund R, Lindgren U. The incidence of femoral shaft fractures in children and adolescents. *J Pediatr Orthop* 1986;6:47–50.
13. Henderson J, Goldacre MJ, Fairweather JM, et al. Conditions accounting for substantial time spent in hospital in children aged 1–14 years. *Arch Dis Child* 1992;67:83–86.
14. Hinton RY, Lincoln A, Crockett MM, et al. Fractures of the femoral shaft in children. Incidence, mechanism, and sociodemographic risk factors. *J Bone Joint Surg [Am]* 1999;81:500–509.
15. Johnson AW, Weiss CB Jr, Wheeler DL. Stress fractures of the femoral shaft in athletes—more common than expected: a new clinical test. *Am J Sports Med* 1994;22:248–256.
16. Katz JF. Spontaneous fractures in paraplegic children. *J Bone Joint Surg [Am]* 1953;35:220–226.
17. Landin LA. Fracture patterns in children: analysis of 8,682 fractures with special reference to incidence, etiology and secular changes in a Swedish urban population 1950–1979. *Acta Orthop Scand Suppl* 1986;202:54.
18. Letts M, Vincent N. The floating knee in children. *J Bone Joint Surg [Br]* 1986;68:442–446.
19. Leventhal JM, Thomas SA, Rosenfield NS, et al. Fractures in young children: distinguishing child abuse from unintentional injuries. *Am J Dis Child* 1993;147:87–92.
20. Loder RT. Pediatric polytrauma: orthopaedic care and hospital course. *J Orthop Trauma* 1987;1:48–54.
21. Lynch JM, Gardner MJ, Gains B. Hemodynamic significance of pediatric femur fractures. *J Pediatr Surg* 1996;31:1358–1361.
22. Meaney JE, Carty H. Femoral stress fractures in children. *Skel Radio* 1992;21:173–176.
23. Porat S, Migrom C, Nyska M, et al. Femoral fracture treatment in head-injured children: use of external fixation. *J Trauma* 1986;26:81–84.
24. Roach J, Hoschl R. Diffuse femoral uptake on bone scan after fracture in an infant. *Q J Nucl Med* 1996;40:194–196.
25. Robin GC. Fractures in poliomyelitis in children. *J Bone Joint Surg [Am]* 1966;48:1048–1054.
26. Schenk R. Basic histomorphology and physiology of skeletal growth. In: Weber B, Brunner C, Frueher F, eds. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980:3–19.
27. Silverman FN. Radiological Aspects of the Battered Child Syndrome. In: *The battered child*, 4th ed. Chicago: University of Chicago Press, 1987:214–246.
28. Toren A, Goshen E, Katz M, et al. Bilateral femoral stress fractures in a child due to in-line (roller) skating. *Acta Paediatr* 1997;86:332–333.
29. Vangsness CT Jr, Decampos J, Merritt PO, et al. Meniscal injuries associated with femoral shaft fractures. *J Bone Joint Surg [Br]* 1993;75:207–209.

### Treatment

#### Traction and Casting

30. Aronson DD, Singer RM, Higgins RF. Skeletal traction for fractures of the femoral shaft in children. *J Bone Joint Surg [Am]* 1987;69:1435–1439.
31. Bryant T. *The practice of surgery*. Philadelphia: HC Lee, 1873.
32. Buck G. An improved method of treating fractures of the thigh illustrated by cases and a drawing. *Trans NY Acad Sci* 1861;2:232–250.
33. Cole WH. Results of treatment of fractured femurs in children with special reference to Bryant's overhead traction. *Arch Surg* 1922;5:702–716.
34. Connolly JF, Dehne E, LaFollett B. Closed reduction and early cast brace ambulation in the treatment of femoral shaft fractures: II. Results in one hundred and forty-three fractures. *J Bone Joint Surg [Am]* 1973;55:1581.
35. Coyte PC, Bronskill SE, Hirji ZZ, et al. Economic evaluation of 2 treatments for pediatric femoral shaft fractures. *Clin Orthop Rel Res* 1997;336:205–215.
36. Czertak DJ, Hennrikus WL. The treatment of pediatric femur fractures with early 90-90 spica casting. *J Pediatr Orthop* 1999;19:229–232.
37. Dencker H. Wire traction complications associated with treatment of femoral shaft fractures. *Acta Orthop Scand* 1964;35:158.
38. Ferguson J, Nicol RO. Early spical treatment of pediatric femoral shaft fractures. *J Pediatr Orthop* 2000;20:189–192.
39. Ferry AM, Edgar MS. Modified Bryant's traction. *J Bone Joint Surg [Am]* 1966;48:533–536.
40. Gross RH, Davidson R, Sullivan JA, et al. Cast brace management of the femoral shaft fracture in children and young adults. *J Pediatr Orthop* 1983;3:572–582.
41. Holmes SJK, Sedgwick DM, Scobie WG. Domiciliary gallows traction for femoral shaft fractures in young children: feasibility, safety and advantages. *J Bone Joint Surg [Br]* 1983;65:288–290.
42. Hughes BF, Sponseller PD, Thompson JD. Pediatric femur fractures: effects of spica cast treatment on family and community. *J Pediatr Orthop* 1995;15:457–460.
43. Humberger FW, Eyring EJ. Proximal tibial 90-90 traction in treatment of children with femoral shaft fractures. *J Bone Joint Surg [Am]* 1969;51:499–504, 1969.
44. Illgen R 2nd, Rodgers WB, Hresko MT, et al. Femur fractures in children: treatment with early sitting spica casting. *J Pediatr Orthop* 1998;18:481–487.
45. Infante AF, Albert MC, Jennings WB, et al. Immediate hip spica casting for femur fractures in pediatric patients: a review of 175 patients. *Clin Orthop* 2000;376:106–112.
46. Irani RN, Nicholson JT, Chung SMK. Long-term results in the treatment of femoral shaft fractures in young children by immediate spica immobilization. *J Bone Joint Surg [Am]* 1976;58:945–951.
47. Martinez AG, Carroll NC, Sarwark JF, et al. Femoral shaft fractures in children treated with early spica cast. *J Pediatr Orthop* 1991;11:712–716.
48. McCarthy RE. A method for early spica cast application in treatment of pediatric femoral shaft fractures. *J Pediatr Orthop* 1986;6:89–91.
49. McCullough N, Vinsant J, Sarmiento A. Functional fracture-bracing of long-bone fractures of the lower extremity in children. *J Bone Joint Surg [Am]* 1978;60:314.
50. Miller ME, Bramlett KW, Kissell EU, et al. Improved treatment of femoral shaft fractures in children: the “Pontoon” 90-90 spica cast. *Clin Orthop* 1987;219:140–146.
51. Miller PR, Welch MC. The hazards of tibial pin replacement in 90-90 skeletal traction. *Clin Orthop* 1978;135:97–100.
52. Mital MA, Cashman WF. Fresh ambulatory approach to treatment of femoral shaft fractures in children: a comparison with traditional conservative methods. *J Bone Joint Surg [Am]*

1976;58:285.

53. Newton P, Mubarak S. Financial aspects of femoral shaft fractures. *J Pediatr Orthop* 1994;14:508–512.
54. Nicholson JT, Foster RM, Heath RD. Bryant's traction: a provocative cause of circulatory complications. *JAMA* 1955;157:415.
55. Nork SE, Hoffinger SA. Skeletal traction versus external fixation for pediatric femoral shaft fractures: a comparison of hospital costs and charges. *J Orthop Trauma* 1998;12:563–568.
56. Pott P. *Some few general remarks on fractures and dislocations*. London: L. Hawes, E. Clarke, and R. Collins, 1769.
57. Reeves RB, Ballard RI, Hughes JL. Internal fixation versus traction and casting of adolescent femoral shaft fractures. *J Pediatr Orthop* 1990;10:592–595.
58. Russell RH. Theory and method in extension of the thigh. *BMJ* 1921;2:637–639.
59. Ryan JR. 90-90 Skeletal femoral traction for femoral shaft fractures in children. *J Trauma* 1981;21:46–48.
60. Sahin V, Baktir A, Turk CY, et al. Femoral shaft fractures in children treated by closed reduction and early spica cast with incorporated supracondylar Kirschner wires: a long-term follow up results. *Injury* 1999;30:121–128.
61. Scott J, Wardlaw D, McLauchlan J. Cast bracing of femoral shaft fractures in children: a preliminary report. *J Pediatr Orthop* 1981;1:199–201.
62. Staheli LT, Sheridan GW. Early spica cast management of femoral shaft fractures in young children. *Clin Orthop* 1977;126:162–166.
63. Stanitski CL, Monroe MT, Stanitski DF, et al. Radiation exposure during skeletal traction treatment of pediatric femoral fractures. *J Pediatr Orthop* 1998;18:271–272.
64. Stannard JP, Christensen KP, Wilkins KE. Femur fractures in infants: a new therapeutic approach. *J Pediatr Orthop* 1995;15:461–466.
65. Stans AA, Morrissy RT, Renwick SE. Femoral shaft fracture treatment in patients age 6 to 16 years. *J Pediatr Orthop* 1999;19:222–228.
66. Sugi M, Cole WG. Early plaster treatment for fractures of the femoral shaft in childhood. *J Bone Joint Surg [Br]* 1987;69:743–745.
67. Thompson JD, Buehler KC, Sponseller PD, et al. Shortening in femoral shaft fractures in children treated with spica cast. *Clin Orthop Rel Res* 1997;338:74–78.
68. Van Meter JW, Branick RI. Bilateral genu recurvatum after skeletal traction. *J Bone Joint Surg [Am]* 1980;62:837–839.
69. Weiss AP, Schenck RC Jr, Sponseller PD, et al. Peroneal nerve palsy after early cast application for femoral fractures in children. *J Pediatr Orthop* 1992;12:25–28.
70. Yandow SM, Archibeck MJ, Stevens PM, et al. Femoral-shaft fractures in children: a comparison of immediate casting and traction. *J Pediatr Orthop* 1999;19:55–59.

### **Rodding, External Fixation, and Plating**

71. Aronson J, Tursky RN. External fixation of femur fractures in children. *J Bone Joint Surg [Am]* 1987;69:1435–1439.
72. Bar-On E, Sagiv S, Porat S. External fixation or flexible intramedullary nailing for femoral shaft fractures in children. A prospective, randomised study. *J Bone Joint Surg [Br]* 1997;79:975–978.
73. Beaty JH, Austin SM, Warner WC, et al. Interlocking intramedullary nailing of femoral shaft fractures in adolescents: preliminary results and complications. *J Pediatr Orthop* 1994;14:178–183.
74. Benirschke SK, Melder I, Henley MD, et al. Closed interlocking nailing of femoral shaft fractures: assessment of technical complications and functional outcomes by comparison of a prospective database with retrospective review. *J Orthop Trauma* 1993;7:118–122.
75. Bergman M, Tornetta P, Kerina M, et al. Femur fractures caused by gunshots: treatment by immediate reamed intramedullary nailing. *J Trauma* 1993;34:783–785.
76. Blasier RD, Aronson J, Tursky EA. External fixation of pediatric femur fractures. *J Pediatr Orthop* 1997;17:342–346.
77. Bourdelat D. Fracture of the femoral shaft in children: advantages of the descending medullary nailing. *J Pediatr Orthop* 1996;5:110–114.
78. Brumback RJ, Ellison TS, Poka A, et al. Intramedullary nailing of femoral shaft fractures: III. Long-term effects of static interlocking fixation. *J Bone Joint Surg* 1992;74:106–112.
79. Buford D Jr, Christensen K, Weatherall P. Intramedullary nailing of femoral fractures in adolescents. *Clin Orthop Rel Res* 1998;350:85–89.
80. Cameron CD, Meek RN, Blachut PA, et al. Intramedullary nailing of the femoral shaft: a prospective, randomized study. *J Orthop Trauma* 1992;6:448–451.
81. Carey TP, Galpin RD. Flexible intramedullary nail fixation of pediatric femoral fractures. *Clin Orthop Rel Res* 1996;332:110–118.
82. Chung SM. The arterial supply of the developing proximal end of the human femur. *J Bone Joint Surg [Am]* 1976;58:961–970.
83. Cramer KE, Tornetta P III, Spero CR, et al. Ender rod fixation of femoral shaft fractures in children. *Clin Orthop* 2000;376:119–123.
84. Davis TJ, Topping RE, Blanco JS. External fixation of pediatric femoral fractures. *Clin Orthop Rel Res* 1995;318:191–198.
85. Evanoff M, Strong ML, MacIntosh R. External fixation maintained until fracture consolidation in the skeletally immature. *J Pediatr Orthop* 1993;13:98–101.
86. Fein LH, Pankovich AM, Spero CM, et al. Closed flexible intramedullary nailing of adolescent femoral shaft fractures. *J Orthop Trauma* 1989;3:133–141.
87. Flynn JM, Hresko T, Reynolds RAK, et al. Titanium elastic nails for pediatric femur fractures: a multicenter study of early results with analysis of complications. *J Pediatr Orthop* 2001;21:4–8.
88. Fyodorov I, Sturm PF, Robertson WW Jr. Compression plate fixation of femoral shaft fractures in children aged 8 to 12 years. *J Pediatr Orthop* 1999;19:578–581.
89. Galpin RD, Willis RB, Sabano N. Intramedullary nailing of pediatric femoral fractures. *J Pediatr Orthop* 1994;14:184–189.
90. Gregory RJ, Cubison TC, Pinder IM, et al. External fixation of lower limb fractures in children. *J Trauma* 1992;33:691–693.
91. Gregory P, Pevny T, Teague D. Early complications with external fixation of pediatric femoral shaft fractures. *J Orthop Trauma* 1996;10:191–198.
92. Gregory P, Sullivan JA, Herndon WA. Adolescent femoral shaft fractures: rigid versus flexible nails. *Orthopaedics (Thorofare, NJ)* 1995;18:645–649.
93. Hajek PD, Bicknell HR Jr, Bronson WE, et al. The use of one compared with two distal screws in the treatment of femoral shaft fractures with interlocking intramedullary nailing: a clinical and biomechanical analysis. *J Bone Joint Surg* 1993;75:519–525.
94. Hansen TB. Fractures of the femoral shaft in children treated with an AO-compression plate: report of 12 cases followed until adulthood. *Acta Orthop Scand* 1992;63:50–52.
95. Heinrich SD, Drvaric D, Darr K, et al. Stabilization of pediatric diaphyseal femur fractures with flexible intramedullary nails (a technique paper). *J Orthop Trauma* 1992;6:452–459.
96. Heinrich SD, Drvaric DM, Darr K, et al. The operative stabilization of pediatric diaphyseal femur fractures with flexible intramedullary nails: a prospective analysis. *J Pediatr Orthop* 1994;14:501–507.
97. Herndon WA, Mahnken RF, Yngve DA, et al. Management of femoral shaft fractures in the adolescent. *J Pediatr Orthop* 1989;9:29–32.
98. Hrescovic D Jr, Scott DM, Behrens F, et al. The use of Ender nails in femoral shaft fractures: what are the remaining indications? *J Orthop Trauma* 1992;6:314–317.
99. Hull JB, Sanderson PL, Rickman M, et al. External fixation of children's fractures: use of the Orthofix dynamic axial fixator. *J Pediatr Orthop* 1997;6:203–206.
100. Karaoglu S, Baktir A, Tuncel M, et al. Closed Ender nailing of adolescent femoral shaft fractures. *Injury* 1994;25:501–506.
101. Kaweblum M, Lehman WR, Grant AD, et al. Avascular necrosis of the femoral head as sequela of fracture of the greater trochanter. *Clin Orthop Rel Res* 1993;294:193–195.
102. Kirby RM, Winquist RA, Hansen ST Jr. Femoral shaft fractures in adolescents: a comparison between traction plus cast treatment and closed intramedullary nailing. *J Pediatr Orthop* 1981;1:193–197.
103. Kregor PJ, Song KM, Routt ML Jr, et al. Plate fixation of femoral shaft fractures in multiply injured children. *J Bone Joint Surg* 1993;75:1774–1780.
104. Krettek C, Haas N, Walker J, et al. Treatment of femoral shaft fractures in children by external fixation. *Injury* 1991;22:263–266.
105. Ligier JN, Metaizeau JP, Prevot J, et al. Elastic stable intramedullary nailing of femoral shaft fractures in children. *J Bone Joint Surg [Br]* 1988;70:74–77.
106. Mann DC, Weddington J, Davenport K. Closed Ender nailing of femoral shaft fractures in adolescents. *J Pediatr Orthop* 1986;6:651–655.
107. Maruenda-Paulino JI, Sanchis-Alfonso V, Gomar-Sancho F, et al. *Int Orthop* 1993;17:158–161.
108. Mazda K, Khairouni A, Pennecot GF, et al. Closed flexible intramedullary nailing of the femoral shaft fractures in children. *J Pediatr Orthop* 1997;6:198–202.
109. McGraw JJ, Gregory SK. Ender nails: an alternative for intramedullary fixation of femoral shaft fractures in children and adolescents. *South Med J* 1997;90:694–696.
110. Mileski RA, Garvin KL, Huurman WW. Avascular necrosis of the femoral head after closed intramedullary shortening in an adolescent. *J Pediatr Orthop* 1995;15:24–26.
111. Miner T, Carroll KL. Outcomes of external fixation of pediatric femoral shaft fractures. *J Pediatr Orthop* 2000;20:405–410.
112. O'Malley DE, Mazur JM, Cummings RJ. Femoral head avascular necrosis associated with intramedullary nailing in an adolescent. *J Pediatr Orthop* 1995;15:21–23.
113. Probe R, Lindsey RW, Hadley NA, et al. Refracture of adolescent femoral shaft fractures: a complication of external fixation: a report of two cases. *J Pediatr Orthop* 1993;13:102–105.
114. Rozbruch SR, Muller U, Gautier E, et al. The evolution of femoral shaft plating technique. *Clin Orthop Rel Res* 1998;354:195–208.
115. de Sanctis N, Gambardella A, Pempinello C, et al. The use of external fixators in femur fractures in children. *J Pediatr Orthop* 1996;16:613–620.
116. Simonian PT, Chapman JR, Selznick HS, et al. Iatrogenic fractures of the femoral neck during closed nailing of the femoral shaft. *J Bone Joint Surg [Br]* 1994;76:293–296.
117. Skaggs DL, Leet AI, Money MD, et al. Secondary fractures associated with external fixation in pediatric femur fractures. *J Pediatr Orthop* 1999;19:582–586.
118. Skak SV, Ovoergaard S, Nielsen JD, et al. Internal fixation of femoral shaft fractures in children and adolescents: a ten- to twenty-one-year follow up of 52 fractures. *J Pediatr Orthop* 1996;5:195–199.
119. Sola J, Schoenecker PL, Gordon JE. External fixation of femoral shaft fractures in children: enhanced stability with the use of an auxiliary pin. *J Pediatr Orthop* 1999;19:587–591.
120. Speed K. Analysis of the results of treatment of fractures of the femoral diaphysis in children under twelve years of age. *Surg Gynecol Obstet* 1921;32:527–534.
121. Thometz JG, Lamdan R. Osteonecrosis of the femoral head after intramedullary nailing of a fracture of the femoral shaft in an adolescent. A case report. *J Bone Joint Surg [Am]* 1995;77:1423–1426.
122. Timmerman LA, Rab GT. Intramedullary nailing of femoral shaft fractures in adolescents. *J Orthop Trauma* 1993;7:331–337.
123. Tolo VT. External fixation in multiply injured children. *Orthop Clin North Am* 1990;21:393–400.
124. Townsend DR, Hoffinger S. Intramedullary nailing of femoral shaft fractures in children via the trochanter tip. *Clin Orthop* 2000;376:113–118.
125. Ward WT, Levy J, Kaye A. Compression plating for child and adolescent femur fractures. *J Pediatr Orthop* 1992;12:626–632.
126. Winquist R, Hansen S, Clawson DK. Closed intramedullary nailing of femoral fractures. *J Bone Joint Surg [Am]* 1984;66:529–539.
127. Wiss DA, Brien WW, Stetson WB. Interlocked nailing for treatment of segmental fractures of the femur. *J Bone Joint Surg [Am]* 1990;72:724–728.
128. Ziv I, Blackburn N, Rang M. Femoral intramedullary nailing in the growing child. *J Trauma* 1984;24:432–434.

### **Complications**

129. Aitken AP, Blackett CW, Cincotti JJ. Overgrowth of the femoral shaft following fracture in childhood. *J Bone Joint Surg* 1939;21:334–338.
130. Aitken AP. Overgrowth of the femoral shaft following fracture in children. *Am J Surg* 1940;49:147–148.
131. Anderson M, Green WT. Lengths of the femur and the tibia: norms derived from orthoroentgenograms of children from five years of age until epiphyseal closure. *Am J Dis Child* 1948;75:279–290.
132. Barfod B, Christensen J. Fractures of the femoral shaft in children with special reference to subsequent overgrowth. *Acta Chir Scand* 1958;116:235–250.

133. Benum P, Ertresvag K, Hiseth K. Torsion deformities after traction treatment of femoral fractures in children. *Acta Orthop Scand* 1979;50:87–91.
134. Biyani A, Jones DA, Daniel CL, et al. *Assessment of hip abductor function in relation to peritrochanteric heterotopic ossification after closed femoral nailing*. Swansea, England: Department of Orthopaedics, Morriston Hospital.
135. Braten M, Terjesen T, Rossvoll I. Torsional deformity after intramedullary nailing of femoral shaft fractures: measurement of anteversion angles in 110 patients. *J Bone Joint Surg [Br]* 1993;75:799–803.
136. Brouwer KJ, Molenaar JC, Van Linge B. Rotational deformities after femoral shaft fractures in childhood. *Acta Orthop Scand* 1981;52:81–89.
137. Clark MW, D'Ambrosia RD, Roberts JM. Equinus contracture following Bryant's traction. *Orthopaedics* 1978;1:311–312.
138. Clement DA, Colton CL. Overgrowth of the femur after fracture in childhood: an increased effect in boys. *J Bone Joint Surg [Br]* 1986;68:534–536.
139. Cole WH. Compensatory lengthening of the femur in children after fracture. *Ann Surg* 1925;82:609–616.
140. Connolly JF, Whittaker D, Williams E. Femoral and tibial fractures combined with injuries to the femoral or popliteal artery: a review of the literature and analysis of fourteen cases. *J Bone Joint Surg [Am]* 1971;53:56.
141. Damholt B, Zdravkovic D. Quadriceps function following fractures of the femoral shaft in children. *Acta Orthop Scand* 1974;45:756.
142. David VC. Shortening and compensatory overgrowth following fractures of the femur in children. *Arch Surg* 1924;9:438–449.
143. Davids JR. Rotational deformity and remodeling after fracture of the femur in children. *Clin Orthop* 1994;302:27–35.
144. Dehne E, Kriz FK Jr. Slow arterial leak consequent to unrecognized arterial laceration: report of five cases. *J Bone Joint Surg [Am]* 1967;49:372.
145. Edvardsen P, Syversen SM. Overgrowth of the femur after fracture of the shaft in childhood. *J Bone Joint Surg [Br]* 1976;58:339–342.
146. Engel GM, Staheli LT. The natural history of torsion and other factors influencing gait in childhood: a study of the angle of gait, tibial torsion, knee angle, hip rotation and development of the arch in normal children. *Clin Orthop* 1974;99:12–17.
147. Finsen V, Harness OB, Ness O, et al. Muscle function after plated and nailed femoral shaft fractures. *Injury* 1993;24:531–534.
148. Griffin PP, Anderson M, Green WT. Fractures of the shaft of the femur in children: treatment and results. *Orthop Clin North Am* 1972;3:213–224.
149. Hagglund G, Hansson LI, Norman O. Correction by growth of rotational deformity after femoral fractures in children. *Acta Orthop Scand* 1983;54:858.
150. Hennrikus WL, Kasser JR, Rand F, et al. The function of the quadriceps muscle after a fracture of the femur in patients who are less than seventeen years old. *J Bone Joint Surg* 1993;75:508–513.
151. Henry AN. Overgrowth after femoral shaft fractures in children. *J Bone Joint Surg [Br]* 1963;45:222.
152. Hresko MT, Kasser JR. Physeal arrest about the knee associated with non-physeal fractures in the lower extremity. *J Bone Joint Surg [Am]* 1989;71:698–703.
153. Isaacson J, Louis DS, Costenbader JM. Arterial injury associated with closed femoral shaft fracture: report of five cases. *J Bone Joint Surg [Am]* 1975;57:1147.
154. Ikpeme JO. Quadricepsplasty following femoral shaft fractures. *Injury* 1993;24:104–108.
155. Janzing H, Broos P, Rommens P. Compartment syndrome as a complication of skin traction in children with femoral fractures. *J Trauma Injury Infect Crit Care* 1996;41:156–158.
156. Lewallen RP, Peterson HA. Nonunion of long bone fractures in children: a review of 30 cases. *J Pediatr Orthop* 1985;5:135–142.
157. MacEwen GD, Kasser JR, Heinrich SD. *Pediatric fractures*. Baltimore: Williams & Wilkins, 1993:281.
158. Malkawi H, Shannak A, Hadidi S. Remodeling after femoral shaft fractures in children treated by the modified Blount method. *J Pediatr Orthop* 1986;6:421–429.
159. Meals RA. Overgrowth of the femur following fractures in children: influence of handedness. *J Bone Joint Surg [Am]* 1979;61:381–384.
160. Mesko JW, DeRosa GP, Lindseth RE. Segmental femur loss in children. *J Pediatr Orthop* 1985;5:471–474.
161. Miller DS, Martin L, Grossman E. Ischemic fibrosis of the lower extremity in children. *Am J Surg* 1972;84:317.
162. Oberhammer J. Degree and frequency of rotational deformities after infant femoral fractures and their spontaneous correction. *Arch Orthop Traumatol Surg* 1980;97:249–255.
163. Raney EM, Ogden JA, Grogan DP. Premature greater trochanteric epiphysiodesis secondary to intramedullary femoral rodding. *J Pediatr Orthop* 1993;13:516–520.
164. Reynolds DA. Growth change in fractured long bones: a study of 126 children. *J Bone Joint Surg [Br]* 1981;63:83–88.
165. Riew KD, Sturm PF, Rosenbaum D, et al. Neurologic complications of pediatric femoral nailing. *J Pediatr Orthop* 1996;16:606–612.
166. Robertson P, Karol LA, Rab GT. Open fractures of the tibia and femur in children. *J Pediatr Orthop* 1996;16:621–626.
167. Rosenthal JJ, Gasper MR, Gjerdrum TC, et al. Vascular injuries associated with fractures of the femur. *Arch Surg* 1975;110:494–499.
168. Shah A, Ellis RD. False aneurysm complicating closed femoral fracture in a child. *Orthop Rev* 1993;22:1265–1267.
169. Shapiro F. Fractures of the femoral shaft in children: the overgrowth phenomenon. *Acta Orthop Scand* 1981;52:649–655.
170. Staheli LT. Femoral and tibial growth following femoral shaft fracture in childhood. *Clin Orthop* 1967;55:159–163.
171. Staheli LT. Fever following trauma in childhood. *JAMA* 1967;199:503–504.
172. Thomson SA, Mahoney LJ. Volkmann's ischemic contracture and its relationship to fracture of the femur. *J Bone Joint Surg [Br]* 1951;33:336–347.
173. Truesdell ED. Inequality of lower extremity following fracture of the femoral shaft in childhood. *Ann Surg* 1921;74:498–500.
174. Verbeek H. Does rotational deformity following femur shaft fracture correct during growth? *Reconstr Surg Traumatol* 1979;17:77–81.
175. Viljanto J, Kiviluoto H, Paananen M. Remodeling after femoral shaft fracture in children. *Acta Chir Scand* 1975;141:360–365.
176. Wallace ME, Hoffman EB. Remodelling of angular deformity after femoral shaft fractures in children. *J Bone Joint Surg [Br]* 1992;74:765–769.

#### Subtrochanteric and Supracondylar Fracture

177. DeLee J, Clanton TO, Rockwood CA Jr. Closed treatment of subtrochanteric fractures of the femur in a modified cast brace. *J Bone Joint Surg [Am]* 1981;63:773.
178. Gustillo RB. Current concepts in the management of open fractures. *Instr Course Lect* 1989;36:359–366.
179. Ireland DCR, Fisher RL. Subtrochanteric fractures of the femur in children. *Clin Orthop* 1975;110:157.
180. Shahcheraghi GH, Doroodchi HR. Supracondylar fracture of the femur: closed or open reduction? *J Trauma* 1993;34:499–502.
181. Sponsellor P. Personal communication, series in preparation.

## FRACTURES AND DISLOCATIONS ABOUT THE KNEE

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## Part I: Extraarticular Fractures

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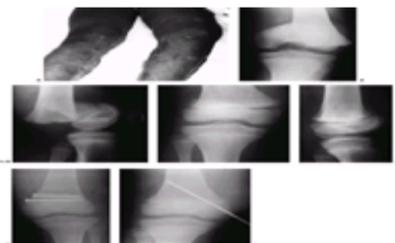
## PART I: EXTRAARTICULAR FRACTURES

Fractures involving the physes around the knee are far from “routine” injuries because they have a significant risk of growth disturbance, which can result in shortening or angulation, or both. Vascular injury occasionally occurs, especially with proximal tibial injuries, and nerves and ligaments also may be damaged.

### DISTAL FEMORAL EPIPHYSEAL FRACTURES

#### Historical Review

The cause of a distal femoral epiphyseal fracture can help determine the method of treatment and identify potential problems. In hyperextension fractures, the epiphysis is displaced anteriorly and the metaphysis is displaced into the popliteal fossa, making neurovascular injury possible. Reduction often is unstable because ligamentotaxis rarely is effective. Extreme knee flexion may be required to tighten the anterior soft tissue hinge. Varus–valgus fractures ( [Fig. 23-1](#)) result from an abduction or adduction force, and the posterior periosteal hinge is intact. In either type of fracture, reduction should be precise because any residual varus or valgus about the knee in older children has limited remodeling potential.



**FIGURE 23-1.** Appearance at presentation of an 11-year-old boy who was hit by a car from the front on the right side, sustaining a fracture-separation of both distal femoral epiphyses (**A**). There was apex-posterior angulation of both sides, with varus angulation on the left side (**B and C**) and valgus on the right (**D and E**). Both sides were treated by closed reduction, with stabilization using cannulated screws on the left (**F**) and a percutaneous smooth pin on the right (**G**).

Many clinical studies of distal femoral physeal fractures report high incidences of growth disturbance, resulting in asymmetry of length or angulation, or both ( [6,63,77](#)). Growth disturbance is caused by bony bridging resulting from direct physeal trauma or from lack of anatomic reduction of the physis. Several authors have shown that the likelihood of physeal disturbance is greater with significant (>50% of the width of the physis) initial displacement of the fracture ( [42,54,78](#)). The complex contour of the physis makes it possible for shearing of the fracture line to occur across several zones of the physis at a microscopic level, even in fracture patterns that are typically considered to be benign, such as Salter-Harris type I and II injuries.

Although it has been thought that knee ligament injury does not occur with fracture through the distal femoral physis, Bertin and Goble ( [7](#)) found that 14 of 29 patients with physeal injuries about the knee had ligamentous instability at follow-up. Brone and Wroble ( [15](#)) reported three patients with Salter-Harris type III fractures of the femoral condyle associated with anterior cruciate ligament (ACL) tears, and found two more reported in the literature. All were near skeletal maturity. An awareness of this possibility and a careful examination will allow timely treatment of ligamentous injury.

Distal femoral physeal injuries account for 1% to 6% of all physeal injuries ( [Table 23-1](#)) and for fewer than 1% of all fractures in children. They are much less common than physeal injuries of the ankle or upper extremity. Most are Salter-Harris type II injuries. Although separations through the cartilaginous physis would seem to be more likely than fractures through the hard cortical bone of the femoral shaft, the physis is protected by its large surface area and its undulating shape.

Location	Neer <sup>a</sup>	Peterson <sup>b</sup>	Mann <sup>c</sup>
Proximal femur		1 (5%)	4 (1%)
Distal femur	28 (5%)	13 (5%)	29 (9%)
Posterosal tibia	17 (3%)	8 (4%)	12 (4%)
Distal tibia	238 (41%)	104 (52%)	282 (86%)
Proximal fibula	2 (0.3%)	1 (5%)	
Distal fibula	352 (51%)	58 (24%)	
Totals	587	195	327

<sup>a</sup>Neer CS Jr, Horwitz ES. Fractures of the proximal humeral epiphyseal plate. *Clin Orthop* 1962;41:24–31.  
<sup>b</sup>Peterson HA, Wadlock R, Benson JT et al. Physeal fractures: Part I. Epidemiology in Olmsted County, Minn., 1959–1988. *J Pediatr Orthop* 1994;14:423–436.  
<sup>c</sup>Mann DC, Rajamann S. Distribution of physeal and nonphyseal fractures in 2,859 long-bone fractures in children age 0–4 years. *J Pediatr Orthop* 1990;10:713–716.

**TABLE 23-1. COMPARTIVE INCIDENCE OF EPIPHYSEAL SEPARATIONS IN THE LOWER EXTREMITIES**

In 1898, Hutchinson and Barnard (36) published one of the earliest reports of this injury. The results were dismal: of 58 patients with distal femoral physeal injuries, 22 required amputation and 10 died. The worst results were in patients with hyperextension injuries that caused popliteal ischemia and uncontrollable infection. Also in 1898, Poland (61) reported 114 patients with injuries to the distal femoral epiphysis, 24 of them boys whose legs were caught in the spokes of a moving carriage wheel as they swung themselves up over the tailgate. With the anterior thigh stopped against the wagon bed, the lower leg was hyperextended by the revolving wheel. The result was often an open injury with neurovascular compromise. Appropriately, separation of the distal femoral epiphysis came to be known as the *wagon-wheel injury* by the end of the 19th century.

After the beginning of the 20th century, little was written about injuries to the distal femoral epiphysis until Aitken and Magill's 1952 report of 15 patients (2). Ten of the 15 separations occurred in patients 13 to 17 years of age; all 10 were football injuries. Only 1 of the 15 patients had a hyperextension injury so common in the days of Hutchinson and Poland. Aitken and Magill wrote, "The horse and wagon has been replaced by the football field as the source of this type of injury."

In 1960, Neer (51) reviewed 2,368 epiphyseal fractures and found that only 1% involved the distal femur. Seven patients (25%) had resultant growth disturbance. He related the severity of initial displacement to early physeal closure. Neer thought that delayed closure resulted from injury to the epiphyseal artery. In 1962, Bassett and Goldner (4) reported 25 patients with distal femoral physeal injuries, 40% of whom experienced significant shortening or angular deformity.

In 1973, Roberts (64) reported 100 patients with injuries to the distal femoral epiphysis. Two thirds of the separations resulted from sports. Of 50 patients followed an average of 7 years, 11 had shortening of more than 2.5 cm. Of the entire group of 100 fractures, no vascular injury was documented, and only 1 patient had an associated nerve injury (a transient peroneal deficit).

In 1974, in a retrospective review of 20 patients with separation of the distal femoral epiphysis, Stephens et al. (76) found a positive correlation between the severity of injury and premature closure of the physis. Substantial shortening developed in five patients despite satisfactory reduction. Growth inhibition usually was evident by 6 months after injury. Because six of their patients who had shortening had Salter-Harris type I or II injuries, the authors questioned the prognostic significance of the Salter-Harris classification as it pertained to separations of the distal femoral epiphysis.

In 1983, Riseborough et al. (63) in a follow-up study of 66 distal femoral separations reported that 17 patients subsequently underwent osteotomy for angular deformity. They identified a pattern of central growth arrest that supported Brashear's hypothesis (12-14) of damage to the germinal cells from the grinding of the central ridges on the proximal surface of the epiphysis as it displaced with injury.

In 1995, Thomson et al. (78) reported 30 fractures of the distal femoral physis and found that many displaced fractures lost reduction when immobilized in a cast. They recommended internal fixation of displaced fractures.

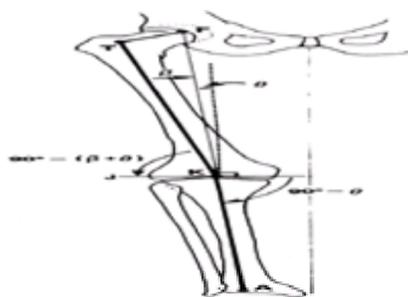
From one century to the next, the clinical presentation of distal femoral epiphyseal separation has changed considerably. The 19th-century surgeon most often found anterior displacement of the epiphysis from forced hyperextension, an open injury, neurovascular compromise, and, later, life-threatening infection. The 21st-century orthopaedic surgeon usually is presented with a closed injury, with displacement in either the coronal or sagittal plane, caused by either a motor vehicle accident or an athletic injury. Currently, the main concern is premature closure of the physis leading to progressive angulation or shortening, or both.

### Surgical Anatomy

The epiphysis of the distal femur is larger and grows more rapidly than any other epiphysis in the lower extremity. It develops from a single ossific nucleus. It is the first epiphysis to ossify and the last to fuse. From birth to skeletal maturity, the distal femoral physis contributes 70% of the growth of the femur and 37% of the growth of the lower extremity. The annual rate of growth is approximately  $\frac{3}{8}$  in. or 9 mm. The growth rate slows at a mean skeletal age of 13 years in girls and 15 years in boys (3,31).

### Bony Anatomy

Immediately above the medial border of the medial condyle, the metaphysis of the distal femur widens sharply to the adductor tubercle. In contrast, the metaphysis flares minimally on the lateral side to produce the lateral epicondyle. A line tangential to the distal surfaces of the two condyles (the joint line) is approximately horizontal in an upright stance. The longitudinal axis of the diaphysis of the femur inclines medially downward, with an angle of 9 degrees from vertical. The mechanical axis of the femur, formed by a line between the centers of the hip and knee joints, is 3 degrees from vertical (Fig. 23-2).



**FIGURE 23-2.** Relation between anatomic and mechanical axes of lower extremity and joint line. Normal value for theta is 3 degrees and for beta 6 degrees. (From Krackow KA. *Adv Orthop Surg* 1983;69-88; with permission.)

Viewed from behind, the condyles are separated by a deep intercondylar notch. In the coronal plane, the medial condyle has a larger cross section; in the transverse plane, the lateral condyle is larger.

A large part of the surface of the distal femoral epiphysis is covered by cartilage for articulation with the proximal tibia and patella. The anterior or patellar surface has a shallow midline concavity to accommodate the longitudinal ridge on the undersurface of the patella. The distal or tibial surface of each condyle extends on either side of the intercondylar notch far around onto the posterior surface. Here, the articular cartilage nearly reaches the posterior margin of the physis. Sometimes, there is a shallow horizontal groove on the articular surface of each condyle, marking the border between the patellar and tibial surfaces. This groove is more constant on the lateral condyle.

The contour of the distal femoral physis undulates from side to side, as well as from front to back. Although less pronounced in humans than in some animals, there is a distinctly quadrupedal configuration. On the distal surface of the metaphysis, facing the physis, a midline groove from anterior to posterior lies just above the intercondylar notch. A similar central groove is present in the midline from the medial to the lateral margins. The intersecting grooves divide the distal surface of the metaphysis into four mamillary processes. The corresponding proximal surface of the epiphysis has four valleys. The interdigitating configuration between the distal surface of the metaphysis and the proximal surface of the epiphysis may help to resist shear and torsion. When subject to trauma, however, the epiphyseal ridges may grind against the metaphyseal projections as a displaced separation occurs. In this way, germinal cells in the deeper layers of the intervening cartilaginous physis may be damaged. Brashear (12,13) has shown this to occur experimentally in simulated separations of the distal epiphyses in rat femora.

### Soft Tissue Anatomy

The distal femoral physis is completely extraarticular. Medially and laterally, the physis is exactly at the level of the epicondyles, which serve as a landmark for it. Anteriorly and posteriorly, the synovial membrane and joint capsule of the knee attach to the femoral epiphysis close to the perimeter of the distal femoral physis.

Anteriorly, the suprapatellar pouch balloons up over the anterior surface of the metaphysis. On the medial and lateral surfaces of the epiphysis, the proximal attachment of the synovium and capsule is separated from the physis by the insertions of the collateral ligaments.

The strong posterior capsule, as well as all major supporting ligaments of the knee, are attached to the epiphysis of the femur distal to the physis. Both cruciate ligaments originate in the upward-sloping roof of the intercondylar notch distal to the physis. Compression and tension forces can be transmitted across the extended knee to the epiphysis of the femur by taut ligaments (57).

The medial head of the gastrocnemius and the plantaris muscles originate from the popliteal surface of the distal femoral metaphysis proximal to the physis. There is some difference of opinion among anatomists (11,29,30), as to whether the lateral head of the gastrocnemius originates from the metaphysis or the epiphysis. Muscle pull would not seem to be as much of a factor as the pull of the ligaments in the initial displacement of the epiphysis at the time of injury. Posterior displacement or angulation is not common, in contrast to displaced supracondylar femoral fractures. Also in contrast, separation of the distal femoral epiphysis with posterior displacement can be reduced and held by the extended knee, with the tightened gastrocnemius acting as an internal splint against the posterior aspect of the epiphysis (2).

### Vascular Anatomy

The popliteal artery is close to the posterior surface of the distal femur: only a thin layer of fat separates the artery from the popliteal surface of the metaphysis. Directly above the femoral condyles, the superior geniculate arteries pass medially and laterally to lie between the femoral metaphysis and the overlying muscles. The terminal branches of the superior geniculate arteries enter the distal femoral epiphysis near the medial and lateral epicondyles. As the popliteal artery continues distally, it lies on the posterior capsule of the knee joint between the femoral condyles. At this level, the middle geniculate artery branches directly forward to enter the posterior aspect of the distal femoral epiphysis. The popliteal artery and its branches are vulnerable to injury from the distal femoral metaphysis at the time of hyperextension injury. An intimal tear of the artery is possible. It is unlikely that the distal femoral epiphysis would be completely shorn of its blood supply because of the rich anastomosis supplied, in part, by the superior geniculate branches.

Above the popliteal space, the sciatic nerve divides into the peroneal and tibial nerves. The peroneal nerve descends posteriorly between the biceps femoris muscle and the lateral head of the gastrocnemius muscle to a point just distal to the head of the fibula. The nerve is subject to stretch if the distal femoral epiphysis is tilted into varus or rotated medially. Moreover, the superficial course of the peroneal nerve makes it vulnerable to direct pressure on the posterolateral aspect of the knee.

### Mechanism of Injury

The pattern of injury is determined by the direction of applied force as well as any other superimposed compressive or distraction loads. The epiphysis may be subject to distraction by a lateral force to the distal femur against a fixed foot, or by hyperextending the lower leg against a fixed thigh. An example of the former injury is a football player whose foot is fixed to the ground by his cleated shoe and who is hit from the side by another player. The classic example of the hyperextending mechanism is, as mentioned earlier, a boy who catches his lower leg in the spokes of a revolving wheel. A more modern example is a child descending on a trampoline while it is recoiling from another child (41). Both compression and distraction forces may be imposed simultaneously on the physis at the time of injury. Physeal separation begins on the tension side and ends with an oblique fracture through the metaphysis on the compression side (a Salter-Harris type II injury) (68). On the tension side, the cartilaginous matrix is more vulnerable to distraction or avulsion force. On the compression side, the osseous matrix of the metaphysis is more vulnerable to shear failure from loading.

Separation of the distal femoral epiphysis also can be caused by a direct blow. The anterior surface of the epiphysis may be struck when a patient falls forward on a bent knee. One reported distal femoral physeal separation was in a 14-year-old boy who struck his knee on the door frame as he galloped his horse through a barn (35).

Specific mechanisms of injury in recent clinical reviews are summarized in Table 23-2. In one series, all but 2 of 15 children hit by automobiles were 6 to 12 years of age. In contrast, separations from sports injuries usually occur between 10 and 19 years of age.

	Neer <sup>a</sup>	Bassett <sup>b</sup>	Roberts <sup>c</sup>	Stephens <sup>d</sup>	Total
Sports injury	5	4	64	8	81 (89%)
Hit by automobile	9	8	15	10	42 (25%)
Falls	3	3	12	1	19 (12%)
Auto accident	—	3	5	1	9 (5%)
Other	4	7	4	—	15 (9%)

<sup>a</sup>Neer CS II. Separation of the lower femoral epiphyses. *Am J Surg* 1962;99:754-761.  
<sup>b</sup>Bassett FH II, Goldner JL. Fractures involving the distal femoral epiphyseal growth line. *South Med J* 1962;55:545-557.  
<sup>c</sup>Roberts JM. Fracture separation of the distal femoral epiphysis. *J Bone Joint Surg Am* 1973;55A:1124.  
<sup>d</sup>Stephens DC, Lusk DS, Louis E. Traumatic separation of the distal femoral epiphyseal cartilage plate. *J Bone Joint Surg Am* 1974;56:1381-1390.

**TABLE 23-2. MECHANISM OF INJURY IN CLINICAL REVIEWS OF SEPARATION OF THE DISTAL FEMORAL EPIPHYSIS**

Unusual mechanisms or underlying conditions also may cause distal femoral physeal injuries (4,73,74,77,80). Unilateral or bilateral separations can occur from birth injury (16). Separation of this epiphysis also has been noted in infants with arthrogryposis multiplex congenita, presumably occurring as stiff knees are passively manipulated (26). Epiphyseal separation also is a risk of manipulating a stiff knee in a normal child (71). Separations of the distal femoral epiphysis may occur in association with osteomyelitis, leukemia, hemophilia, osteosarcoma, and myelomeningocele (27,75,82). Rodgers et al. (66) reported four chronic physeal fractures of the distal femur in patients with myelomeningocele; they attributed them to chronic stress to a poorly sensate limb. Kumar et al. (39) noted that physeal injuries in children with myelomeningocele may heal more slowly than metaphyseal fractures and may require splinting or casting for a longer period. Separation through a normal physis has been reported in children with congenital absence of pain (53).

### Classification

Separations of the distal femoral epiphysis have been classified according to the pattern of fracture, the direction of displacement, or the mechanism of injury. Each has specific implications (Table 23-3). The Salter-Harris classification (68) is useful for description and treatment planning. The direction and degree of displacement may help predict the type and severity of complications. Mechanisms of injury, as well as the implications of growth disturbance, vary with the patient's age.

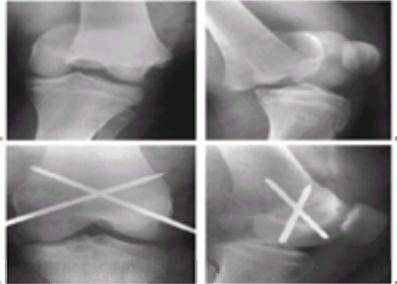
Parameter	Implications
<b>Anatomic</b> Salter I-IV (I most common)	Directs method of stabilization, if displaced (does not strongly predict growth disturbance)
<b>Displacement</b> Medial Lateral Anterior Posterior	Predicts risk of neurovascular injury
<b>Age</b> Infantile Juvenile Adolescent	Predicts severity of growth disturbance—infantile and juvenile have risk of greatest disturbance

**TABLE 23-3. CLASSIFICATION OF DISTAL FEMORAL EPIPHYSEAL FRACTURES: PARAMETERS AND IMPLICATIONS**

### Classification by Fracture Pattern

The Salter-Harris type I pattern is a separation through the distal femoral physis, without fracture through the adjacent epiphysis or metaphysis. It occurs in newborns with birth injury and in adolescents, often as a nondisplaced separation. Either may go undetected. Sometimes the diagnosis is made only in retrospect, when subperiosteal new bone formation occurs along the adjacent metaphysis. If displacement is present, it is usually in the sagittal plane. Growth disturbance may occur after Salter-Harris type I distal femoral injuries, contrary to the usual expectations for Salter-Harris type I fractures.

The Salter-Harris type II pattern, characterized by an oblique extension of the fracture across one corner of the adjacent metaphysis, is the most common type of separation at the distal femur and usually occurs in adolescents. Displacement usually is toward the side of the metaphyseal fragment ( Fig. 23-3). The incidence of premature growth arrest, even with satisfactory reduction, is significant. If asymmetric growth follows a type II separation, the portion of the physis underneath the metaphyseal fracture usually is spared ( 78). Therefore, if the metaphyseal fracture is medial, deformity is more likely to be valgus than varus. If the metaphyseal fracture is lateral, varus angulation is more likely. The size of the metaphyseal fragment is proportional to the amount of compression force at the time of injury.



**FIGURE 23-3. A and B:** Anteriorly and medially displaced Salter-Harris type II fracture of the distal femoral epiphysis in a 16-year-old football player struck on the outside of the left knee. **C and D:** After closed reduction and percutaneous fixation with crossed Kirschner wires.

A Salter-Harris type III injury consists of a partial separation of the physis, with a vertical fracture line extending from the physis down to the articular surface of the epiphysis ( Fig. 23-4). The vertical fracture line is usually in line with the intercondylar notch. Salter-Harris type III injuries most often are caused by valgus stress in sports and usually involve the medial condyle. The fracture may be nondisplaced and detected only with a stress radiograph. If displaced, reduction usually is unstable and requires internal fixation. A displaced type III fracture may cause incongruity of the joint surface, especially of the patellofemoral articulation. Hemarthrosis occurs from direct bleeding into the joint from the articular fracture.



**FIGURE 23-4. A:** Minimally displaced Salter-Harris type III fracture—separation of the distal femoral epiphysis in a 15-year-old football player hit from the front on the left knee. **B:** Fifteen years after injury.

Salter-Harris type IV injuries of the distal femur are uncommon. A sagittal fracture line extends from the metaphyseal cortex down across the physis and enters the articular surface of the epiphysis. Even slight displacement of the fracture fragment may produce growth disturbance from formation of a bony bridge from the displaced epiphysis to the metaphysis. Therefore, anatomic reduction and internal fixation are advised.

Salter-Harris type V injuries (without fracture) are rare. Most commonly, the diagnosis is made in retrospect at the time of evaluation for premature growth arrest and limb-length discrepancy or angular deformity.

Even less common is an avulsion injury to the edge of the physis. A small fragment, including a portion of the perichondrium and underlying bone, may be torn off when the proximal attachment of the collateral ligament is avulsed. This uncommon injury may also lead to localized premature growth arrest and progressive angular deformity ( 34). The bony bridge usually is small, localized, and surgically accessible, and excision is appropriate.

A triplane fracture infrequently occurs in the distal femur. Computed tomographic (CT) scans with three-dimensional modeling may be helpful in identifying and analyzing ( 44) this injury.

### Classification by Displacement

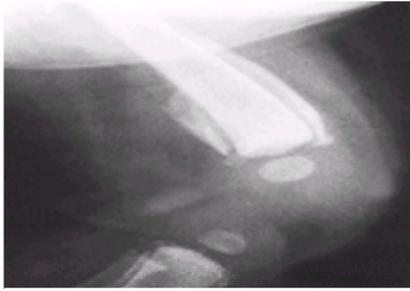
Anterior displacement of the epiphysis results from hyperextension of the knee. The extension force on the distal femur is transmitted through the posterior capsule of the knee joint. The mechanism is similar to that of knee dislocations in adults. There is an increased risk of neurovascular injury ( 77).

Posterior displacement of the epiphysis on the femur is uncommon. It has been reported in birth injuries and in older children struck on the front of the flexed knee. Reduction is obtained and maintained by extending the knee.

Medial/lateral displacement currently is most common, usually with an associated fracture of the adjacent metaphysis (Salter-Harris type II).

### Classification According to Age

Separations of the distal femoral epiphysis in infants may be associated with breech birth or child abuse ( 64) ( Fig. 23-5). Most are Salter-Harris type I injuries. Clinically differentiating between an epiphyseal separation and hematogenous osteomyelitis may be difficult. Ultrasonography may be used to confirm fracture. Older children and preadolescents usually are injured in high-energy accidents such as a fall or being hit by a vehicle. Associated musculoskeletal and visceral injuries are common in this group.



**FIGURE 23-5.** Lateral radiograph of a swollen knee in a 3-month-old girl who reportedly fell out of her crib 8 days earlier. Subperiosteal ossification along the distal femoral shaft indicates separation of the distal femoral epiphysis. Note evidence of fracture–separation of the proximal tibial epiphysis as well. Final diagnosis: abused child.

In most recent reviews, approximately two thirds of distal femoral epiphyseal separations occur in adolescents, often from contact sports ( [45,51,64](#)). The most common patterns are Salter-Harris types I and II. The potential for growth disturbance is lowest in this group ( [Fig. 23-6](#)).



**FIGURE 23-6. A:** The initial anteroposterior radiograph of a 12-year-boy who sustained an injury to the left knee while playing football shows widening of the lateral aspect of the distal femoral physis. **B:** With continued play, a second injury was sustained with anterior displacement of the distal femoral epiphysis. **C:** Open reduction and fixation with crossed Kirschner wires was necessary because of late presentation of injury. **D:** At follow-up, early closure of the distal femoral physis is apparent. (Courtesy of Stephen Cope, MD, Mobile, Alabama.)

## Signs and Symptoms

### Physical Findings

The patient usually cannot walk or bear weight on the injured limb immediately after sustaining a displaced separation of the distal femoral epiphysis. However, with a nondisplaced separation, such as in athletic injuries, the patient may be able to walk. He or she may have felt a “pop” at the injury and presumed a ligament tear. Effusion of the knee and soft tissue swelling develop rapidly. Abrasion or laceration of the overlying soft tissues may be a clue to the mechanism of injury or to an open fracture.

If the secondary hamstring spasm can be relaxed, instability just above the knee joint may be felt. Complete separation of the epiphysis and tear of adjacent ligaments can occur simultaneously. A soft end point or crepitus may accompany the abnormal motion after separation of the physis. Crepitus sometimes is absent because the periosteum is interposed between the metaphysis and the epiphysis. Abnormal laxity in a patient with negative radiographs may be caused by a nondisplaced physeal injury rather than by a ligamentous tear. If the diagnosis of physeal injury is confirmed radiographically, further stress or displacement of the epiphysis should be minimized.

It sometimes is possible to localize tenderness to the level of the physis, which is at approximately the same level as the upper pole of the patella and the adductor tubercle, both of which can be used as reference points. In Salter-Harris type III and IV fractures, tenderness is greater on the involved side of the distal femur.

Separation of the distal femoral epiphysis usually is accompanied by effusion of the knee joint. This is logical in Salter-Harris type III and IV separations, in which the articular surface is fractured. The hemarthrosis associated with other patterns of separation may be caused by bleeding through the synovial suprapatellar pouch. The knee may feel tense to palpation. Aspiration nearly always produces sanguineous fluid. Extravasation of blood into the soft tissues of the distal thigh and popliteal fossa produces ecchymosis that becomes apparent within 72 hours after injury.

### Direction of Displacement

Most commonly, displacement of the epiphysis occurs in the coronal plane, producing varus or valgus deformity. The protruding end of the metaphysis can be palpated through the distal portion of the vastus medialis with valgus injuries, or through the vastus lateralis with varus injuries.

With anterior displacement, the patella, riding forward on the femoral epiphysis, becomes extremely prominent. There is a palpable depression across the anterior thigh just proximal to the patella, and fullness in the popliteal fossa is produced by posterior displacement of the distal end of the metaphysis. Posterior protrusion of the metaphysis may put pressure on the popliteal artery, so that pulsations become palpable in the subcutaneous region of the popliteal fossa. Anterior displacement of the distal femoral epiphysis may be indicated by prominence of the adductor tubercle, which is displaced with the epiphysis anterior to the metaphysis.

With posterior displacement of the distal femoral epiphysis, the distal end of the metaphysis is prominent on the anterior aspect of the distal thigh directly above the patella, and the epiphysis can be felt as a palpable fullness in the popliteal fossa. Whatever the direction of displacement, the patella and femoral condyles remain in line with the proximal tibia, a point useful in differentiating epiphyseal separation from dislocation of the knee.

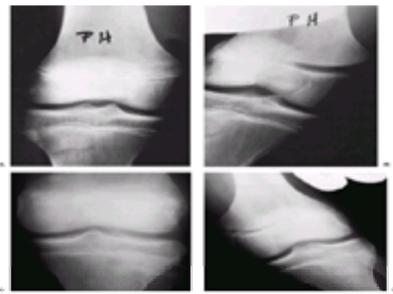
### Polytrauma

With an open injury, the distal end of the metaphysis may cause a transverse laceration, most often in the lateral portion of the popliteal fossa. The apex of the protruding metaphysis is denuded of periosteum.

Whenever a diagnosis of epiphyseal separation is suspected, careful neurovascular examination of the lower leg and foot should be done, including pulses, color, temperature, capillary refill, and motor and sensory status. The extremity may become cyanotic if venous return is impaired. The use of the Doppler flow meter may be helpful in evaluating circulation distal to the injury. Compartmental pressure recordings should be obtained if there are clinical findings of compartment syndrome ( [50](#)). If the separation occurred in a vehicular accident, associated injuries may be present ( [4](#)).

## Radiographic Findings

Because the physis normally is radiolucent, injury is diagnosed by displacement, widening, or adjacent bony disruption ( 54). However, a nondisplaced Salter-Harris type I or III fracture without separation can be easily overlooked. Oblique views of the distal femur may reveal an occult fracture through the epiphysis or metaphysis. Stress views should be considered if multiple plain films are negative in a patient with an effusion or tenderness localized to the physis ( Fig. 23-7).



**FIGURE 23-7.** A 14-year-old football player sustained a knee injury when he was tackled. **A:** The initial anteroposterior radiograph shows no fracture or separation. **B:** Anteroposterior valgus-stress radiograph reveals separation of the distal femoral epiphysis. **C and D:** Another example, age 16: such injuries may occur even when the physes are nearly closed.

In a Salter-Harris type II injury, the most common pattern, a fracture line extends from the radiolucent physis obliquely through the distal femoral metaphysis. The metaphyseal fracture line and spared segment of physis outline a triangular fragment of bone that remains in position relative to the epiphysis. The size of the metaphyseal triangle tends to be larger if displacement has occurred in the coronal plane.

A vertical fracture line extending from the articular surface of the distal femoral epiphysis into the radiolucent physis is diagnostic of a Salter-Harris type III fracture. Usually, the epiphyseal fracture line is best seen on an anteroposterior view because it is oriented in the sagittal plane. The degree of displacement in this fracture pattern may be difficult to measure unless the radiographic projection is precisely in line with the plane of fracture. If the fracture pattern requires further definition to determine treatment, multiple oblique views or CT scanning may be helpful to demonstrate the fracture plane or measure the gap in the articular surface of the femoral portion of the patellofemoral joint. Less commonly, the vertical fracture extends through the articular surface in the weight-bearing portion of the femoral condyle. If the weight-bearing surface is involved, it is even more important to determine the degree of displacement.

A fracture line extending from the epiphyseal surface across the physis and up through the metaphysis is characteristic of a Salter-Harris type IV injury. Even 1 to 2 mm of displacement is significant (21). Occasionally, the pattern of fracture separation may be difficult to outline. If needed, CT can be useful.

Anterior or posterior displacement of the epiphysis is best appreciated on the lateral projection. The anteriorly displaced epiphysis usually is tilted so that the distal articular surface faces anteriorly. The posteriorly displaced epiphysis is rotated so that the distal articular surface faces the popliteal fossa.

Separation of the distal femoral epiphysis in an infant is difficult to see on initial radiographs unless there is displacement because only the center of the epiphysis is ossified at birth. This ossicle should be in line with the axis of the femoral shaft on both anteroposterior and lateral views ( 17,18). Comparative views of the opposite knee may be helpful. Magnetic resonance imaging (MRI), ultrasonography, or arthrography of the knee may help to identify a separation of the relatively unossified femoral epiphysis.

Stress views are indicated when the initial radiographic appearance is negative but the history and physical signs suggest epiphyseal separation. Stress radiographs may be falsely negative if there is associated muscle spasm. Adequate analgesia relaxes muscle spasm and helps protect the physis from further injury during examination. Some traction should be applied to the lower leg as the knee is angulated for radiographic examination. Smith ( 72) reported two 15-year-old boys injured on the football field for whom the diagnosis of distal femoral epiphyseal separation could be made only on stress views.

The radiolucent line representing the physis on the anteroposterior projection is approximately 3 mm thick until adolescence. Diminution of the distance between the line of provisional calcification at the lower edge of the metaphysis and the bony plate on the upper border of the epiphysis suggests a compression injury to the physis, especially if reinforced by the clinical findings. Neer ( 51) pointed out that radiographic signs of premature closure usually become evident within 6 months after injury.

### Treatment—Overview

The objectives of treatment of separation of the distal femoral epiphysis are to obtain and maintain satisfactory reduction, to regain a functional range of motion of the knee joint, to regain normal strength of the quadriceps and hamstring muscles, and to avoid further damage to the physis.

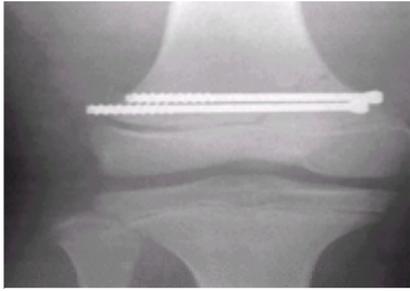
Anatomic reduction of a displaced separation of the distal femoral epiphysis is desirable, and the closer the patient is to skeletal maturity, the greater the need for exact realignment. Blount (8) pointed out that residual varus or valgus deformity after reduction usually does not remodel with further growth. Reduction of displacement in the sagittal plane may be slightly less precise. Sharrard ( 70) stated that angulation of up to 15 degrees with apex anterior or posterior is well tolerated. Blount wrote that a child younger than 10 years of age with posterior angulation of less than 20 degrees will not have permanent genu recurvatum, and the deformity remodels with growth. The remodeling potential in infants is so great that considerable displacement can be accepted. Burman and Langsam ( 16) reported good results in displaced birth fractures that were merely splinted. Most infants can be treated by supportive traction or splinting, no matter how great the displacement.

Closed reduction usually can be performed in older children up to 10 days after injury. Böhler ( 9) reported successful reduction 12 days after injury, and Patterson (57) reported successful reduction in a 9-year-old girl 11 days after injury.

Closed reduction may fail for a number of reasons, making open reduction necessary. A Salter-Harris type I or II separation may be irreducible by closed methods because of interposed soft tissue, usually a flap of torn periosteum or muscle that curls up inside the fracture cleft. Displaced Salter-Harris type III or IV separations almost always require open reduction and internal fixation to minimize disruption of the articular surface and decrease the likelihood of premature growth arrest. In an open injury, open reduction and fixation may be accomplished at the time of wound debridement.

Although growth may be adversely affected by the injury itself, further damage to the physis should be avoided during diagnostic stress radiography, closed reduction, or open reduction. The use of general anesthesia decreases the forces across the physis. Kurlander ( 40) reported a patient in whom the popliteal artery was injured at the time of closed reduction. Salter et al. (67) stated that it is better to accept a less-than-perfect reduction and do a corrective osteotomy later than to cause further damage by rough handling.

Thomson et al. (78) showed that significantly displaced physal fractures of the distal femur had a better outcome if they were internally fixed. Whenever possible, fixation devices are placed to avoid crossing the physis. In Salter-Harris type III separations, pins or screws may be placed transversely through the epiphysis. In Salter-Harris type II or IV separations, pins or screws are placed transversely through the metaphysis, engaging the triangular fragment if it is large enough (2 to 3 cm tall; Fig. 23-8). If traversing the physis is unavoidable, smooth, small-diameter pins are used.



**FIGURE 23-8.** Previously reduced Salter-Harris type II fracture of the distal femoral epiphysis in a 13-year-old boy. After reduction, two 6.5-mm cannulated screws were inserted through the large medial metaphyseal fragment for fixation.

External fixation rarely is indicated for the treatment of physeal injuries about the knee because of the danger of secondary knee joint infection ( [79](#)).

At the time of initial evaluation and treatment planning, the short- and long-term problems and complications are explained to the patient and parents. The need for long-term follow-up is stressed from the beginning of treatment. The family is better able to comprehend these facts if they are presented from the beginning.

### **Nonoperative or Conservative Treatment**

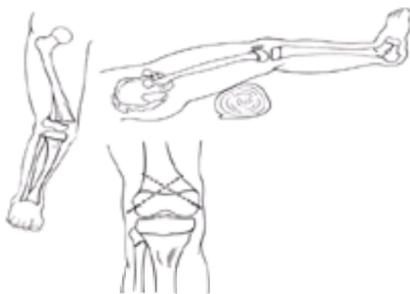
#### **Immobilization**

A nondisplaced separation is immobilized to prevent displacement and relieve pain. If there is tense effusion of the knee joint, aspiration under sterile precautions may be done. A well-molded long leg cast is applied with the knee in approximately 15 to 20 degrees of flexion. The forces that caused the injury should be reversed and the intact periosteal hinge tightened. Thus, if the metaphyseal fragment of a nondisplaced Salter-Harris type II separation is on the lateral side of the metaphysis, the cast is applied with three-point molding into slight valgus. If the metaphyseal fragment is medial, the cast is molded into slight varus. Alternative methods of immobilization include a Jones dressing, posterior splint, cylinder cast from high thigh to supramalleolar level, or, most often, a single hip spica cast. The more secure form of immobilization should be used if the patient is obese or of uncertain reliability. It must be stressed that there is a high chance of loss of reduction if a displaced fracture is reduced and not internally fixed ( [78](#)). Radiographs are made 1 week after immobilization to ensure that displacement has not occurred.

Isometric exercises are started as soon as symptoms permit. Straight-leg raises usually are possible approximately a week after injury. Ambulation on crutches with touchdown gait is continued for 3 to 6 weeks after injury. By 4 to 8 weeks after injury, depending on the patient's age, the cast is removed or converted to a removable posterior splint. Thereafter, active range-of-motion exercises are done twice daily until normal strength and knee motion are regained. Return to athletic activities may be permitted when symptoms are resolved, knee range of motion is normal, and quadriceps mass is symmetric with the opposite side. Even with a nondisplaced fracture, growth inhibition may be caused by a compression force at the time of injury, and the patient should be followed for at least 6 months and preferably for 12 to 24 months.

#### **Closed Reduction and Fixation**

The technique of closed reduction depends on the direction and degree of displacement of the epiphysis ( [Fig. 23-9](#)). General anesthesia often is indicated to decrease associated muscle spasm and diminish the risk of further injury to the physis. Joint aspiration may precede manipulation. Keeping in mind the intact tether of periosteum on the side to which the epiphysis is displaced, the first maneuver increases the deformity slightly with traction. The proximal edge of the displaced epiphysis can then be brought to the edge of the metaphysis on the same side of the periosteal tether. Reduction is then completed by realignment of the angular deformity. Grinding of the physeal cartilage against the metaphysis is thereby avoided. The sequence of events is to pull, tip, and close the separation. The maneuver should be 90% traction and 10% leverage.



**FIGURE 23-9.** Closed reduction and stabilization of a Salter-Harris type I or II fracture. **A:** With medial or lateral displacement, traction is applied longitudinally along the axis of the deformity to bring the fragments out to length. **B:** For anterior displacement, the reduction can be done with the patient prone or supine. Length is gained first; then, using a large bolster as a fulcrum, a flexion moment is added. **C:** Fixation is with smooth 3-mm pins in most adolescents; the angle must be oblique so that the pins cross proximal to the physis. Pins should engage the far cortex and may be cut off under the skin.

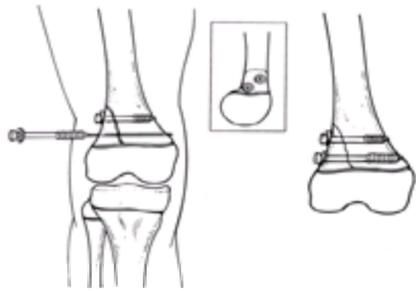
**Medial or Lateral Displacement.** To reduce either medial or lateral displacement of the epiphysis, the patient is placed supine. The leg is grasped with the knee in extension and the hip in slight flexion. The thigh is fixed by an assistant. Moderate longitudinal traction is exerted by a handhold on the leg above the ankle. If the displacement of the epiphysis is medial, varus is increased gently and cautiously to avoid stretching the peroneal nerve. With one hand holding traction on the leg, the palm of the other hand is placed against the concave surface of the angulated distal femur. The epiphysis is pushed toward the metaphysis as the leg is realigned with the thigh. Once reduction is obtained, longitudinal traction is released. If traction on the leg is precluded by an associated injury to the femur or a tear of a knee joint ligament, a pin can be inserted transversely across the proximal tibia to be used as a handle in the maneuver. Usually, however, these associated injuries increase the risk of loss of reduction and are indications for primary internal fixation in adolescents. The reduction is checked by anteroposterior and lateral radiographs. A long leg cast or hip spica cast is then applied, with the knee in slight flexion. External immobilization is continued for 5 to 6 weeks. Thereafter, the care is similar to that for a nondisplaced separation.

**Anterior Displacement.** Anterior displacement of the epiphysis can be reduced with the patient either supine ( [82](#)) or prone ( [62,68](#)). Hutchinson and Barnard ( [36](#)) showed that reduction of this displacement could be more easily and surely obtained by applying traction to the leg with the knee flexed. With the patient supine, the hip is flexed approximately 60 degrees and the thigh is fixed by an assistant. Longitudinal traction is applied, with the knee in partial flexion. Downward pressure on the epiphysis is exerted manually. With continuing traction on the leg, the knee is flexed 45 to 90 degrees. Prone reduction requires fewer assistants. If the surgeon chooses to perform the reduction with the patient prone, traction is applied to the limb, an assistant pushes down on the posterior aspect of the thigh, and the knee is flexed further until approximately 110 degrees of flexion is reached. This sequence is similar to that for reduction of a supracondylar humerus fracture of the elbow.

After reduction of an anteriorly displaced epiphysis, it is important to check the pulses in the foot and ankle. Flexion of a swollen knee to beyond 90 degrees may compromise the popliteal vessels. The position is maintained by temporary splinting while images are obtained. If reduction is adequate, the knee is immobilized in flexion by a long leg or hip spica cast. Bellin ( [5](#)) and Griswold ( [32](#)) noted difficulty regaining extension of the knee after prolonged immobilization in flexion. Two weeks after injury, the cast is changed or modified so that the knee can be brought out to 45 degrees flexion. It is important to increase the range of extension gradually

during the 6- to 8-week period of immobilization.

**Pin Fixation.** The larger the metaphyseal fragment and the greater the displacement, the less stable the closed reduction. If reduction of the anterior displacement is unstable, percutaneous pin fixation is recommended. An image intensifier may be used. If the metaphyseal fragment is large enough, threaded pins or screws can be directed transversely across the metaphysis after reduction ([Fig. 23-10](#)). In the absence of a substantial metaphyseal fragment, smooth pins are directed through the side of each condyle to cross in the metaphysis proximal to the central third of the physis. The closer the pins are to crossing at the fracture site, the less stable they are. To make the pins cross at a point proximal to the fracture site, they should come in at a "high" angle, less than 45 degrees to the long axis of the femur. The pins are cut off under the skin before application of the cast, with the knee in slight flexion. Infection is frequent if pins in this region are left out through the skin for a long time.



**FIGURE 23-10.** Reduction and percutaneous screw fixation of Salter-Harris type II fracture with a large metaphyseal fragment. **A:** Cannulated screws are placed closer to the physis than to the fracture line. Two screws may be placed anterior and posterior to each other. A washer helps increase compression. **B:** After both screws are in place, reduction should be maintained when pressure is removed. If deformity recurs, the metaphyseal fragment may be unstable or the periosteum may be infolded on the contralateral side.

**Posterior Displacement.** To reduce posterior displacement of the distal femoral epiphysis, the patient is placed supine. The surgeon grasps the leg and exerts downward longitudinal traction while the knee is held partly flexed. Longitudinal traction is continued as the leg is brought up to extend the knee. An assistant pulls up directly under the distal femoral epiphysis with one hand and pushes down on the distal metaphysis of the femur with the other. Heller ([33](#)) advised placing a pin transversely distal to the tibial tubercle. Upward pull on the tibial pin is continued while applying longitudinal traction to the leg. Aitken and Magill ([2](#)) pointed out that reduction of this type of separation was best maintained by leaving the knee in extension. The medial head of the gastrocnemius originates from the metaphysis proximal to the distal femoral physis and acts as an internal splint when held taut against the posterior aspect of the epiphysis by the extended position of the knee. Burman and Langsam ([16](#)) advised immobilization without reduction for posterior displacement in a newborn after breech delivery. They reported that adequate remodeling occurred, even with severe displacement.

#### Operative Treatment

##### Open Reduction of Physeal Fractures

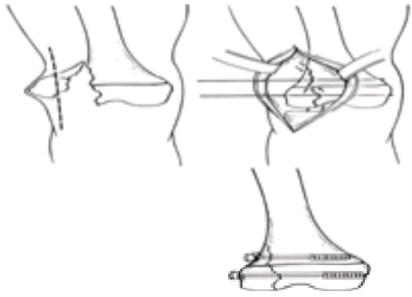
Open reduction is indicated for displaced Salter-Harris type III or IV fractures, for all other types in which satisfactory alignment cannot be obtained by closed means, or when associated injuries mandate it (i.e., a "floating knee" or ligament injury). A tourniquet around the proximal thigh may be used for temporary hemostasis if it is placed proximally enough to avoid binding the thigh muscles under the inflated tourniquet.

For an irreducible Salter-Harris type II separation in the coronal plane, a longitudinal incision is centered over the palpable, prominent end of the metaphysis. The approach from this direction gives direct exposure of any obstacles to reduction and avoids disruption of the periosteal hinge. If the displacement of the epiphysis is lateral, the metaphysis protrudes medially under the distal portion of the vastus medialis. If the displacement of the epiphysis is medial, the metaphysis protrudes laterally under the distal edge of the vastus lateralis. If the displacement is anterior, the procedure is done with the patient prone. After incision of the deep fascia, dissection is continued, extending the plane of injury bluntly by spreading the muscle fibers to expose the end of the metaphysis. Irrigation and careful removal of clotted blood permit better inspection of the separation. An interposed flap of periosteum may be identified between the epiphysis and metaphysis. The periosteal flap is gently pulled out of the separation with forceps or a large skin hook. Special care is taken to avoid any additional damage to the physis covering the proximal surface of the epiphysis. Once the muscle and periosteal flap are removed, reduction is carried out by gentle realignment. To avoid damage to the physis, instruments should not be placed in the physeal interval. Again, for unstable or irreducible fractures, percutaneous pinning is indicated to stabilize the fracture ([56](#)) ([Fig. 23-11](#)). After closure of the wound, a long leg or hip spica cast is applied.



**FIGURE 23-11. A:** Completely displaced Salter-Harris type II fracture of the distal femur in a 6-year-old girl whose foot was on the back of the driver's headrest when the automobile in which she was riding was involved in an accident. **B:** Ecchymosis in the popliteal fossa and anterior displacement of the distal femur are evident. Clinical examination revealed absence of peroneal nerve function and a cold, pulseless foot. The fracture was irreducible by closed methods and required open reduction, internal fixation, and repair of a popliteal artery laceration. **C and D:** Three months after injury; note incomplete reduction, 25 degrees of posterior angulation, and abundant callus formation about the fracture. **E and F:** Four years after injury; note remodeling about the distal femur with normal growth of the distal femoral physis.

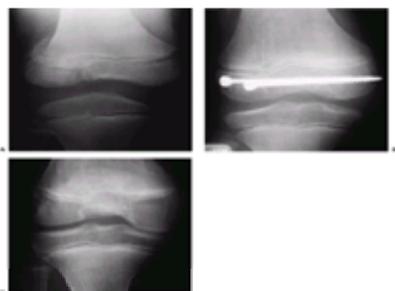
For open reduction and internal fixation of a Salter-Harris type III or IV separation, an anteromedial or anterolateral longitudinal incision is used ([Fig. 23-12](#)). The anterior physeal and articular margins of the fracture are exposed. If the fracture is nondisplaced, percutaneous cannulated screw fixation is indicated. In displaced fractures, arthrotomy allows inspection of the articular surface of the epiphysis. Reduction is checked by noting the apposition of the articular surfaces, the physeal line anteriorly, and the fracture pattern ([Fig. 23-12](#), arrows) and can be confirmed with fluoroscopy. Provisional stabilization is obtained with Kirschner guidewires. When reduction is accomplished, threaded pins or screws are directed transversely across the epiphysis in Salter-Harris type III separations ([Fig. 23-13](#)), or across the metaphysis and epiphysis in Salter-Harris type IV injuries ([Fig. 23-14](#)). If crossing the physis with fixation is unavoidable, smooth pins or wires should be used. Fixation pins usually are introduced through stab wounds adjacent to the incision. The pins are cut off beneath the skin. After reduction and fixation are checked by intraoperative radiographs, the knee joint is thoroughly irrigated and inspected for other fractures and ligament disruption. After surgery, the reduction is protected by a long leg or hip spica cast.



**FIGURE 23-12.** Open reduction of displaced lateral Salter-Harris type IV fracture of the distal femur. **A:** Longitudinal skin incision. **B:** Provisional stabilization with Kirschner wires. Cortex, physis, and joint surfaces are aligned (arrows). **C:** Fixation screws are inserted parallel to the physis.



**FIGURE 23-13. A:** Salter-Harris type III fracture—separation of the distal femur. Note the vertical fracture line extending from the physis distally into the intercondylar notch with displacement. **B:** After reduction and fixation with two compression screws extending transversely across the epiphyseal fragments. Note closure and healing of the vertical fracture line in the epiphysis, with restoration of the articular surface. **C:** Placement of screws in the epiphysis distal to the physis and proximal to the articular cartilage.



**FIGURE 23-14. A:** Salter-Harris type IV fracture of the distal femur in a 7-year-old boy who was struck by a car. The fracture plane was complex and it was significantly displaced. **B:** Two years after open reduction and internal fixation, growth was maintained, although flexion was limited to 110 degrees. **C:** Six years later, limb lengths and angulation were equal.

If an associated collateral ligament injury is found, it can be repaired at the time of open reduction. Internal fixation is used to allow early mobilization and rehabilitation of both the physeal separation and the ligamentous injury.

If vascular repair is indicated, a posterior modified S-shaped incision or posteromedial incision is used to follow the course of the femoral artery ( Fig. 23-15). Care should be taken during incision because the vessel may be superficial beneath the skin. The hamstring tendons may be “bowstrung” around the femoral metaphysis. The artery may be in spasm, occluded by intimal tear, or torn. After the vascular structures are identified, the fracture is reduced and stabilized before vascular repair.



**FIGURE 23-15.** Open reduction of distal femoral fracture with vascular repair. **A:** Lateral view showing site of vascular injury. **B:** Modified S-shaped incision follows the course of the femoral artery.

Open separations of the distal femoral epiphysis usually are caused by hyperextension, with anterior displacement of the epiphysis. A wound may be present in the popliteal fossa, overlying the posterior protruding end of the metaphysis. The patient is placed prone with the knee slightly flexed and the skin is thoroughly irrigated and debrided. The wound is enlarged to allow inspection of the contents of the popliteal fossa. Muscle or periosteum or both may be interposed in the fracture site. Because the patient is prone, reduction is obtained by bringing the leg up against downward pressure on the distal end of the femoral shaft, while maintaining longitudinal traction. Internal fixation is used to stabilize the fracture, especially if vascular or ligamentous repair is to be done. The wound is packed open, and the knee immobilized in slight flexion with a long leg or hip spica cast. Rarely, if extensive soft tissue reconstruction is required, an external fixator spanning the knee may be considered to facilitate dressing changes and soft tissue reconstruction.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

Nondisplaced fractures are treated with above-the-knee cast immobilization for 4 to 6 weeks. For displaced Salter-Harris type I or II fractures, we perform gentle

closed reduction with the patient under general anesthesia, using traction and gentle manipulation. Extreme flexion should not be necessary to maintain reduction and should be avoided to prevent injury to vascular structures. In children younger than 10 years of age, as much as 20 degrees of posterior angulation is acceptable, but in patients closer to adolescence, we accept only minimal anteroposterior angulation and no more than 5 degrees of varus–valgus angulation.

If the displacement is anterior, we perform the reduction with the patient supine. An assistant holds the thigh with the hip partly flexed over a bolster. The surgeon grasps the leg from behind the calf with one hand and pulls down in line with the thigh as he or she tries to tip and close the epiphysis against the metaphysis with the other hand. We check for pulses after this maneuver. For all but minimally displaced and completely stable fractures, we use internal fixation with percutaneous pins or screws. Smooth pins  $\frac{5}{64}$  or  $\frac{3}{32}$  in.) are placed so that they cross in the metaphysis, proximal to the fracture, to prevent the epiphysis from rotating at the fracture site. In an adolescent with a large (2 to 3 cm high) triangular metaphyseal spike (Thurston-Holland fragment), we insert two cannulated 4.0- or 6.5-mm screws transversely to fix the spike to the metaphysis without crossing the physis. An above-the-knee cast is applied with the knee in 5 to 10 degrees of flexion.

We prefer open anatomic reduction for all displaced type III and IV fractures to prevent the formation of a bony bar, which causes limb-length discrepancy and angular deformity. The fracture line, the physis, and the joint surface are observed to confirm anatomic reduction ( [Fig. 23-16](#)). Cannulated screws are then inserted with either an open technique or percutaneously with the aid of image intensification.



**FIGURE 23-16.** **A:** Comminuted Salter-Harris type III fracture of the distal femoral epiphysis with large osteochondral fragments. **B:** Axial plane computed tomography scan demonstrates intraarticular osteochondral fragments. **C and D:** After open reduction and internal fixation with Herbert screws. On the lateral view, the screws appear to be protruding anteriorly in the distal femoral epiphysis, but they actually are buried in articular cartilage. (Courtesy of Dr. William C. Warner, Jr, Campbell Clinic, Memphis, Tennessee.)

Open fractures or fractures caused by massive penetrating trauma require meticulous debridement. We try to save and stabilize any viable fragments of epiphysis or articular surface, but totally free fragments are removed. Debridement is repeated as necessary, usually at 48 hours, and soft tissue coverage is accomplished as soon as feasible.

#### Postreduction Care

Ambulation on crutches usually is possible within a few days. At 1 week after reduction, the patient returns for radiographs taken through the cast. If displacement has occurred, it is not too late to obtain reduction by remanipulation. If reduction is maintained, straight-leg exercises are begun four times daily. At 4 weeks after injury, the patient returns for radiographs. If subperiosteal new bone formation is present adjacent to the metaphysis, the cast is bivalved, and the posterior portion may then be removed twice daily for active muscle-strengthening and range-of-motion exercises, done both supine and prone. The splint may be reapplied for ambulation with crutches. Partial weight bearing is gradually increased. At 6 to 8 weeks after injury, and at regular intervals thereafter, the patient returns for reexamination to evaluate healing, range of knee motion, and the strength of thigh muscles. When range of motion and thigh muscle strength are fully recovered, the patient may return to normal activities. At 6 months after injury, alignment, leg length, and gait are evaluated with comparative radiographs of the lower extremities. If alignment, leg length, and gait are within normal limits, the patient is dismissed from routine care but is counseled to return for evaluation of growth 12 and 24 months after injury.

#### Prognosis

The prognosis for separation of the distal femoral epiphysis usually is good. Over two thirds of the patients with this injury are healthy adolescents ( [64](#)). With appropriate treatment, they return to normal activities within 4 to 6 months. Although as many as a third of patients may sustain some damage to the physis at the time of injury, they usually are close enough to the end of growth to make shortening or angulation insignificant. For younger children with more remaining growth, the potential for angular and length deformity is significant.

The outlook for newborns with separations sustained at the time of delivery in general is good. Remodeling occurs rapidly. If part of the physis is damaged, however, the consequence is major deformity (a rare event). In some patients, nearly normal growth continues for a decade until the adolescent growth spurt makes a growth disturbance obvious ( [25](#)).

#### Complications

Early complications of separations of the distal femoral epiphysis may include injury to the popliteal artery, neurapraxia of the peroneal nerve, associated ligamentous injury, and recurrent displacement of the epiphysis ( [Table 23-4](#)). Complications that occur later include angular deformity, leg-length discrepancy, stiffness, quadriceps weakness, and persistent instability of the knee. Delayed union or nonunion is rarely a problem, except in patients with an underlying neuropathy such as meningomyelocele. Avascular necrosis of the epiphysis has not been reported after distal femoral physeal injuries, in contrast to proximal femoral physeal injuries. The reported incidences of complications in five clinical reviews are summarized in [Table 23-5](#).

Pitfall	Preventive Strategy
Missed diagnosis	Consider undiagnosed physical injury in athletes with tenderness at physis or laxity on stress. Careful physical examination of polytrauma patients for tenderness, or consider bone scan if unresponsive.
Vascular impairment	Check pulses, temperature, muscle function; angiogram if abnormal.
Peroneal nerve injury	Avoid excessive stretch through traction or excessive varus stress at reduction.
Redisplacement	Apply spica cast if needed; internally fix if displaced or unstable; early follow-up to allow correction (within 1 week after injury).
Knee joint instability	Check ligaments when fracture stabilized or healed; tailor treatment to age and activity.
Progressive angulation	Minimize trauma at reduction; magnetic resonance imaging may help to make earliest diagnosis; follow-up at 6 months after trauma to detect growth disturbances.

**TABLE 23-4. DISTAL FEMORAL PHYSEAL FRACTURES: PITFALLS AND PREVENTION**

Reference	Number Patients	Popliteal Artery Injury	Peroneal Nerve Injury	Angular Deformity	Leg Length Discrepancy	Knee Stiffness
Aitken <sup>2</sup>	15	—	—	1	1	4
Lombardo <sup>3</sup>	34	—	1	11	19	11
Stephens <sup>4</sup>	20	1	2	4	8	4
Roberts <sup>5</sup>	38	—	1	9	18	4
Neer <sup>6</sup>	21	1	—	—	—	—
<b>Total</b>	<b>148</b>	<b>2 (1%)</b>	<b>4 (3%)</b>	<b>26 (18%)</b>	<b>38 (26%)</b>	<b>23 (16%)</b>

<sup>2</sup>Aitken DJ, Magill RL. Fractures involving the distal femoral epiphyseal cartilage. *J Bone Joint Surg Am* 1952;34:108-108.  
<sup>3</sup>Lombardo L, Harvey P. Fractures of the distal femoral epiphysis. Factors influencing prognosis: a review of 34 cases. *J Bone Joint Surg Am* 1971;53:742-751.  
<sup>4</sup>Stephens JC, Linn S, Linn E. Traumatic separation of the distal femoral epiphyseal cartilage plate. *J Bone Joint Surg Am* 1974;56:1333-1336.  
<sup>5</sup>Roberts BK. Fracture separation of the distal femoral epiphysis. *J Bone Joint Surg Am* 1972;54:1523-1524.  
<sup>6</sup>Neer CS. Separation of the lower femoral epiphysis. *Am J Surg* 1926;9:750-751.

**TABLE 23-5. INCIDENCE OF COMPLICATIONS AFTER SEPARATION OF THE DISTAL FEMORAL EPIPHYSIS IN CLINICAL REVIEWS**

### Vascular Impairment

Intimal tear and thrombosis in the popliteal artery may be caused by trauma from the distal end of the metaphysis when the epiphysis is displaced anteriorly with a hyperextension injury (41,45). The current incidence of vascular injury is approximately 1%. A single vascular deficit occurred in each of two reviews (51,76). In another four reviews, there were no vascular injuries (2,45,64).

Neer (51) found vascular insufficiency in 1 of 21 patients with displaced separations of the lower femoral epiphysis; this was promptly relieved by reduction of the separation. Bassett and Goldner (4) reported on a patient with arterial spasm associated with displacement in the sagittal plane. At the time of initial examination, shortly after injury, the foot was pale and cold. Pulses were not palpable. After reduction, the color improved and the pulses returned. There were no sequelae to the temporary vascular occlusion.

If vascular impingement occurs but is relieved by prompt reduction of the displaced epiphysis, the patient must be observed for 48 to 72 hours to rule out an intimal tear with thrombosis. Vascular impairment may develop slowly from increasing compartmental pressure. If the patient has inordinate persistent pain, with a cool and pale foot, a femoral arteriogram and compartment pressure measurement should be considered, even if peripheral pulses are present. An arteriogram is not indicated for routine, closed hyperextension epiphyseal displacements, as long as the clinical examination is negative after reduction (38,81). In a patient with an acute fracture with vascular injury, in whom the foot is pale, cool, and nonviable, popliteal artery exploration is indicated after fracture reduction and stabilization. Arteriography is not mandatory for an isolated vascular injury because the site of the lesion is known and can be addressed at the time of reduction. If there is an associated fracture of the pelvis or femoral shaft, arteriography may be necessary to localize the vascular injury.

### Peroneal Nerve Injury

The peroneal nerve is the only nerve injured with any appreciable frequency in this type of fracture. It may be stretched by anterior or medial displacement of the epiphysis.

Lombardo and Harvey (45) reported a patient with an associated peroneal nerve palsy that resolved spontaneously over 6 months. Stephens et al. (76) reported two patients with peroneal nerve palsies who recovered completely. Both were adolescents with Salter-Harris type II separations. Roberts (64) found one patient with neurapraxia of the peroneal nerve associated with medial displacement of a Salter-Harris type II separation. The separation was satisfactorily reduced by closed means, and the nerve injury spontaneously resolved in several months.

Peroneal nerve injury is reported to occur in approximately 3% of separations of the distal femoral epiphysis. It rarely requires treatment other than reduction of the separation. The exception to this is a transected nerve in association with an open injury, which may be treated with repair or grafting. Persistent neurologic deficit after 3 to 6 months warrants electromyographic examination. If the conduction time is prolonged and fibrillation or denervation is present in distal muscles, exploration and microneural reanastomosis or resection of any neuroma may be indicated.

### Recurrent Displacement

Separation of the distal femoral epiphysis may be quite unstable after reduction. Bassett and Goldner (4) found that 10 of 25 patients had loss of reduction after the initial manipulation. Thomson (78) found that 6 of 30 had either an unacceptable reduction or loss of reduction. After reduction of anterior displacement, the epiphysis may tilt forward again as swelling subsides. On the other hand, immobilization in extension stabilizes reduction of posterior or mediolateral displacement. Aitken and Magill (2) pointed out that the gastrocnemius acts as a strap against the posterior aspect of the distal femoral epiphysis when the knee is extended. If reduction is lost so that a second manipulation is required, internal fixation should be used.

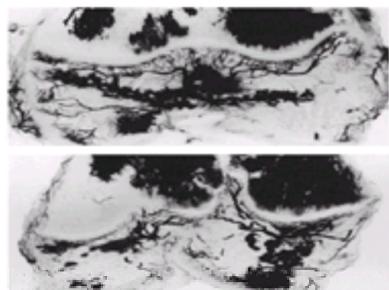
### Knee Joint Instability

Symptomatic knee joint instability may persist after the epiphyseal separation has healed. This finding at follow-up implies concomitant injury to knee ligaments, often unappreciated at the time of initial management of the epiphyseal separation. Aitken and Magill (2) found that four of nine patients had "some evidence of relaxation of the anterior cruciate ligament." In the 20 patients reviewed by Stephens et al. (76), 5 had persistent instability of the knee. Lombardo and Harvey (45) found knee laxity in 8 of 34 patients. Brone and Wroble (15) found three ACL tears associated with Salter-Harris type III fractures of the medial femoral condyle.

Bertin and Goble (7) found that 6 of 16 patients seen in follow-up for distal femoral physeal fractures had positive anterior drawer and Lachman tests; 1 patient had laxity to valgus stress. They surmised that during injury, knee ligaments stretch in series just before fracture-separation of the adjacent cartilaginous physis. They emphasized the importance of early diagnosis of associated ligament injury. If there is no meniscal injury, a rehabilitation program is indicated initially. If there is a reparable meniscal tear, cruciate reconstruction at the time of meniscal repair after physeal healing may be indicated, depending on the patient's age and activity level.

### Progressive Angulation

Progressive angulation after separation of the distal femoral epiphysis usually is caused by asymmetric growth (1,8,60) from either trauma to the physis at the initial injury (Salter-Harris type I or II) or physeal offset with bony bar formation after healing (28) (Salter-Harris type III or IV; Fig. 23-17). Occasionally, progressive angulation follows nonphyseal fractures (Fig. 23-18), in which an associated Salter-Harris type V physeal injury presumably was not noted (34). The risk of angular disturbance is highest in juveniles. If the growth defect is secondary to a persistently displaced Salter-Harris type IV injury, evidence of bony union is present between the displaced epiphysis and the metaphysis across the fracture line.



**FIGURE 23-17.** Mechanism of physeal bar formation after Salter-Harris type III and IV fractures. Physeal vessels that normally are distal **(A)** may anastomose with metaphyseal vessels because of vertical translation after Salter-Harris type IV fracture **(B)**. (From Gomes LSM, Volpon JB. Experimental physeal fracture separations treated with rigid internal fixation. *J Bone Joint Surg Am* 1993;75:1756–1764; with permission.)



**FIGURE 23-18. A:** Genu valgum deformity of the right knee in a 14-year-old boy who sustained a right femoral shaft fracture at 10 years of age. **B and C:** Anteroposterior and lateral tomograms demonstrate bony bar formation in the posterior and lateral aspects of the distal femoral epiphysis. **D:** After distal femoral supracondylar osteotomy and bony bar resection of the distal femur.

If the separation is a Salter-Harris type II injury, the physis distal to the triangular metaphyseal fragment usually is spared. The localized area of growth inhibition occurs in that portion of the physis not protected by the metaphyseal fragment. If progressive angulation occurs after a Salter-Harris type II separation with lateral displacement, the subsequent deformity usually is varus. Conversely, if a Salter-Harris type II separation occurs with initial medial displacement, the subsequent deformity often is valgus.

If a localized area of premature arrest constitutes less than 25% to 30% of the total area of the physis and if at least 2 years of growth remain, excision of the bony bridge should be considered (65). The bridge can be accurately defined by MRI, but the study must be ordered with attention called to the physis, rather than to the knee itself. The characteristics of partially bound water in physeal cartilage are distinct from those of free synovial fluid, and excellent contrast can be obtained with proper planning. The surgeon should request proton-density sequences with fat suppression or T2-weighted gradient-echo images (fast-field echo) (19). Alternatively, helical CT scans may be used to create a map of the injured physis (20,44). The technique of excision is well described by Peterson (60). A peripheral bridge can be approached directly. A central bridge is approached through a metaphyseal window. The area of union between the epiphysis and the metaphysis is carefully removed with a curet and power bur. The defect is filled with autogenous fat (42,43) or cranioplastic methacrylate.

Hemiepiphysiodesis may be considered in maturing adolescents with progressive varus or valgus angulation associated with a central bony bridge with some remaining growth medially or laterally. Enough growth may remain in the segment of physis between the bony bridge and the perimeter on the same side to correct the deformity. Either stapling (85) or percutaneous epiphysiodesis (65) can be done at the edge of the physis opposite the bony bridge, often with satisfactory results. Bowen et al. (10) devised a chart based on skeletal age that is useful in estimating the amount of correction of angular deformity that hemiepiphysiodesis will effect. Nevertheless, because of the physeal bar, this procedure has an element of unpredictability that counterbalances its simplicity. If hemiepiphysiodesis is used to correct angular deformity, timely epiphysiodesis of the opposite limb may be required to prevent significant limb-length discrepancy ( Fig. 23-19).



**FIGURE 23-19. A and B:** Comminuted, open, T-condylar fracture of the distal femoral metaphysis and epiphysis in a 10-year-old girl. **C and D:** After irrigation and debridement and cannulated screw fixation of the metaphyseal and epiphyseal fractures. **E:** One year after injury, the articular surface is restored but the distal femoral physis is closed.

If the patient is within 2 years of skeletal maturity, correction of significant angular deformity by epiphysiodesis with an external fixator may be considered (23,25,48,58,59). The pins can be held by one of the many types of adjustable lengthening fixators (24,25,37,55). Gradual distraction and realignment are done with careful attention to the possibility of neurologic compromise. These techniques are demanding and are best done by those with experience.

If the patient is approaching skeletal maturity, corrective osteotomy is the preferred treatment. For associated shortening, an opening-wedge osteotomy is preferable. The technique for correcting deformity has been described and illustrated by Abbott and Gill (1) and by Scheffer and Peterson (69). An osteotomy is made at the supracondylar level, parallel to the articular surface of the condyles. The hinge of the osteotomy should be in metaphyseal bone, which is better able to deform plastically than is cortical bone. Usually no more than 15 degrees of correction can be obtained without cracking the hinge. The distal fragment is then tilted to place the articular surface in horizontal alignment. A triangular fragment of autogenous iliac bone is inserted in the opening. If the hinge is strong and the patient is slender, immobilization in a cast may be all that is needed. Otherwise, fixation with crossed pins or plate and screws may be necessary.

An alternative method of fixation for an opening-wedge osteotomy or for lengthening and angular correction of the distal femur is external fixation. The DeBastiani device or an Ilizarov construct (33,55) is useful because the amount of lengthening or angulation can be adjusted postoperatively, and significant angulation and length deformities can be corrected.

Abbott and Gill (1) noted that a compensatory deformity may occur in the proximal tibia, and if angulation of the femur is corrected, subsequent osteotomy for correction of the tibial deformity may be indicated. Although no long-term data exist, any coexisting tibial deformity of more than 10 degrees probably should be corrected to maintain a horizontal knee joint.

### Leg-Length Discrepancy

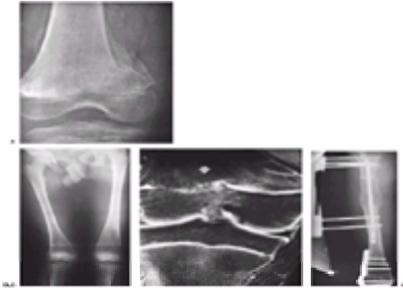
Progressive leg-length discrepancy may follow a separation of the distal femoral epiphysis if premature arrest of the physis occurs (22). If the patient is within 2 years of skeletal maturity at the time of injury, the shortening probably will be insignificant. If there are more than 2 years from the time of injury to skeletal maturity, the leg-length discrepancy may progress at a rate of 1 cm ( $\frac{3}{8}$  in.) per year.

Sometimes the growth disturbance is not a discrete bar but a partial physeal slowing. The progression of leg-length discrepancy is best followed by serial

examinations. Every 6 months, a scanogram and bone age are obtained and the clinical discrepancy is measured. The leg lengths can be plotted on the Moseley straight-line graph (49). After three sequential scanograms over a period of 12 to 18 months, discrepancy at skeletal maturity can be estimated by extrapolation according to the rate of growth of each limb.

If the estimated discrepancy at skeletal maturity is less than 2.5 cm (1 in.), no definitive treatment is indicated. If the estimated discrepancy is 2.5 to 5 cm, surgical closure of the contralateral femoral or tibial physes in the opposite extremity at the appropriate time should be considered (47). In older patients close to skeletal maturity, shortening of the contralateral femur at the subtrochanteric or mid-diaphyseal level is an option.

If the estimated discrepancy at maturity exceeds 5 cm, lengthening of the short femur by corticotomy and slow distraction with an external fixator should be considered (Fig. 23-20). Large discrepancies after injury early in life can be equalized by repeated lengthenings, combined if needed with appropriate epiphysiodesis or shortening of the opposite limb. Epiphysiodesis should be timed by either the Moseley method (49) or Green-Anderson method (31).



**FIGURE 23-20.** **A:** Salter-Harris type II fracture of the distal femur with medial metaphyseal fragment in an 8-year-old boy. **B:** After being lost to follow-up for 4 years, he presented with 6 cm of shortening and a mild varus deformity. **C:** Magnetic resonance image with gradient-echo sequence shows physeal bar formation involving approximately 40% of the physis. **D:** Distal femoral osteotomy and proximal femoral lengthening of 9 cm to match the existing and anticipated future discrepancy produced satisfactory results.

### Stiffness

Limitation of knee motion after separation of the distal femoral epiphysis may be caused by intraarticular adhesions, capsular contracture, or muscular contracture. Regaining knee motion is difficult after immobilization in flexion for reduction of an anteriorly displaced epiphysis. As soon as possible, the knee should be gradually extended. Limitation of knee extension usually is caused by contracture of the posterior capsule and a weak, stretched-out quadriceps muscle. This should be treated with active and active-assistive range-of-motion exercises, with emphasis on quadriceps strengthening. Internal fixation with early motion may be considered. For patients with stiff knees in whom conservative treatment has failed, surgical release of contractures and adhesions, followed by continuous passive motion, may regain significant motion (21).

Intraarticular adhesions may form in the knee with hemarthrosis. An irregular articular surface from a Salter-Harris type III or IV injury may contribute to early degenerative changes. The incidence of permanent persistent stiffness is relatively low [23 of 140 patients (16%) (2,45,51,65,76); see Table 23-5].

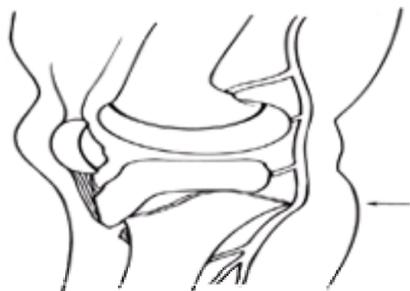
## FRACTURE OF THE PROXIMAL TIBIAL EPIPHYSIS

### Historical Review

The physis of the proximal tibia is well protected, in contrast to the distal femoral physis. On the lateral aspect, the proximal tibial epiphysis is buttressed by the upper end of the fibula. Anteriorly, the tubercle projects down from the epiphysis to overhang the adjacent metaphysis. The superficial portion of the medial collateral ligament (MCL) extends beyond the physis to insert into the upper metaphysis. In the posteromedial corner, the insertion of the semimembranosus muscle spans the physis. Thus, there is nearly circumferential reinforcement to the perichondrium.

Because of this protection, separation of the proximal tibial epiphysis is relatively rare (see Table 23-1). Neer and Horwitz (52) reported an incidence of 0.8% in 2,500 consecutive physeal fractures. Mann and Rajmaira (46) reviewed 2,650 long bone fractures in children; 15 (0.6%) were physeal separations of the proximal tibia, mostly in adolescent boys.

The most serious complication of injury to the proximal tibia is vascular compromise. The popliteal artery is tethered by its major branches near the posterior surface of the proximal tibial epiphysis. The posterior tibial branch passes under the arching fibers of the soleus. The anterior tibial artery passes forward through an aperture above the proximal border of the interosseous membrane. A hyperextension injury that results in posterior displacement of the upper end of the metaphysis may stretch and tear the bound popliteal artery (Fig. 23-21).



**FIGURE 23-21.** Medial view of fracture—separation of the proximal tibial epiphysis resulting from hyperextension of the right knee. Posteriorly displaced proximal tibial metaphysis impinges on the adjacent popliteal artery.

Similar to separations of the distal femoral epiphysis, injuries to the proximal tibial epiphysis may cause shortening or angulation from subsequent growth inhibition. This inhibition can occur after Salter-Harris type I and II injuries (76,82,89). Presumably, a concurrent longitudinal compression force damages the deeper proliferative zone of the physis.

Some authors (87,93) include complete avulsions of the tibial tubercle in reviews of fracture-separations of the proximal tibial epiphysis. However, avulsion fractures of the tibial tubercle constitute a specific subgroup considered in a separate section of this chapter.

Poland (61) reported 24 children with fracture-separations of the proximal tibial epiphysis, most of them younger than 9 years of age. He stated that the usual mechanism of injury was a direct crushing force, such as a wagon wheel rolling over a child's leg. In Poland's day, such an injury was often open, and infection frequently led to amputation, if not death. Poland found that experimental separation of the upper tibial epiphysis could be produced most easily on cadavers between

the ages of 8 and 15 years, and most injuries to the proximal tibial epiphysis occur in this age group.

Nicholson (90) reported four separations of the proximal tibial epiphysis, all in children struck by automobiles. In three of the four patients, the displacement was posterior. Burkhart and Peterson's (87) clinical review of 28 fracture-separations included four avulsion fractures of the tibial tubercle and five open injuries to small children from rotary lawn mowers. Of the 19 remaining injuries, half occurred during athletics and half in motor vehicle accidents. Corrective surgery for subsequent growth disturbance was required in a third of the patients. Two patients presented late with associated arterial insufficiency.

Shelton and Canale (93) reported 39 injuries to the proximal tibial epiphysis. Posterior displacement of the shaft with disruption of the popliteal artery was found in two patients. Unsatisfactory results because of chronic vascular insufficiency, growth disturbance, or traumatic arthritis occurred in four patients.

In 1983, Bertin and Goble (7) reported a retrospective study of physeal fractures about the knee in which 8 of 13 patients with separations of the proximal tibial epiphysis had ligamentous laxity manifested by positive anterior drawer and Lachman tests. These authors described a subset of three patients with almost identical Salter-Harris type III fracture-separations with associated MCL tears.

In 1989, Poulsen et al. (91) reviewed 15 patients with proximal tibial epiphyseal fractures. Concomitant avulsion fractures of the tibial insertion of the ACL occurred in five patients. One patient had primary repair of the tibial collateral ligament. At follow-up, two patients had combined anterior and medial laxity, and another two patients had isolated anterior laxity.

In 1991, Wozasek et al. (96) reported 31 injuries of the proximal tibial epiphysis. Four patients had signs of distal ischemia before reduction that resolved after reduction. One patient had a delayed diagnosis of ischemia that required amputation, and one had peroneal palsy.

In 1998, Gautier et al. (89) performed a meta-analysis of the literature including their own cases, and found a 25% incidence of significant growth disturbance.

## Surgical Anatomy

### Bony Anatomy

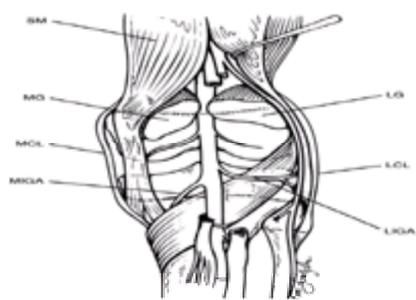
The ossific nucleus of the proximal tibial epiphysis appears by 2 months of age. It lies in the center of the cartilaginous anlage, somewhat closer to the metaphysis than to the articular surface. Occasionally, the ossification center is double. The secondary center in the tubercle appears between the 9th and 14th years. By the 15th year, the upper epiphysis unites with the tubercle and is almost completely ossified.

The distal surface of the epiphysis is concave to match the convex upper surface of the metaphysis. There is a slight central notch in the surface of the metaphysis. The epiphysis is higher on the lateral surface than on the medial surface. The physis slopes down somewhat farther on the lateral side than on the medial side. In the posterolateral corner, the physeal surface is immediately inferior to the upper tibiofibular joint. From this point, the perimeter of the physis extends across the posterior aspect of the upper tibia proximal to the origin of the popliteus muscle. On the medial side, the physis is proximal to the insertion of the superficial MCL. On the anteromedial and anterolateral surfaces of the upper tibia, the physis creates a circumferential ridge between the vertical surface of the epiphysis and the sloping of the metaphysis. This ridge is palpable through the overlying skin and subcutaneous tissue. In the midline anteriorly, the physis dips down underneath the tibial tubercle.

The lateral edge of the physis is separated from the proximal tibiofibular joint by a thin layer of joint capsule. Hemorrhage from a separation may extend into the adjacent joint cavity and through it into the knee joint itself (92). The physis closes slightly earlier posteriorly than anteriorly (86).

### Soft Tissue Anatomy

The synovium and the capsule of the knee joint insert into the proximal tibial epiphysis well above the physis. There is a defect in the capsule where the popliteus tendon runs over the posterolateral corner of the tibia. The arcuate ligament arches over the popliteus tendon from the proximal tibia to the posterior aspect of the epiphysis. The capsular ligament anchors the menisci to the tibial epiphysis medially and laterally. The lateral collateral ligament (LCL) of the knee inserts into the fibula (Fig. 23-22). The MCL inserts beyond the physis into the upper metaphysis of the tibia. The patellar ligament inserts, for the most part, into the secondary ossification center of the tubercle, although some fibers extend beyond the physis into the anterior aspect of the upper tibial diaphysis. The semimembranosus muscle inserts above and below the physis in the posteromedial corner. As this tendon approaches the proximal tibia, it divides into four insertions: the posterior capsule of the knee joint, the posterior surface of the tibial epiphysis, the medial surface of the tibial epiphysis, and the medial surface of the tibial metaphysis.



**FIGURE 23-22.** Posterior anatomy of the right popliteal region. Note that the popliteal vessels are protected from bone (especially the tibia) only by the popliteus muscle. The vessels are tethered by the geniculate branches and by the trifurcation. MG and LG, medial and lateral gastrocnemius heads; SM, semimembranosus; MCL, medial collateral ligament; LCL, lateral collateral ligament; MIGA, medial inferior geniculate artery; LIGA, lateral inferior geniculate artery; ATA, anterior tibial artery.

### Vascular Anatomy

The distal portion of the popliteal artery lies close to the posterior aspect of the upper tibia. Firm connective tissue septa hold the vessel against the knee capsule. The popliteus muscle intervenes between the artery and bone. The lateral inferior geniculate artery runs across the surface of the popliteus muscle, anterior to the lateral head of the gastrocnemius, and turns forward underneath the LCL. The medial inferior geniculate artery passes along the proximal border of the popliteus muscle, anterior to the medial head of the gastrocnemius, and extends forward along the medial aspect of the upper tibia. Beneath the soleal arch, the popliteal artery divides into the anterior tibial and posterior tibial branches.

The proximal tibial epiphysis derives much of its blood supply from an anastomosis between the geniculate arteries posterior to the patellar ligament. Multiple small vessels pass backward and downward into the anterior aspect of the epiphysis. Other small vessels enter the epiphysis on the posterior surface, adjacent to the attachment of the posterior cruciate ligament (PCL). More vessels enter the medial and lateral surface of the epiphysis. The diffuse extraarticular blood supply to this epiphysis makes it less vulnerable to ischemia from injury.

### Mechanism of Injury

Fracture-separation of the proximal tibial epiphysis can be caused by a direct or indirect force. A direct force may be imposed when a child's leg is run over by the wheels of a vehicle or when it is caught between bumpers of two automobiles (Fig. 23-23). More often, separation of the proximal tibial epiphysis is caused by an indirect mechanism. The lower leg is forced into abduction or hyperextension against the fixed knee. An apex of deformity on the medial side implies a partial tear of

the superficial portion of the MCL. Indirect injuries to adolescents occur during sports, motor vehicle accidents, or falls. Less frequently, flexion injuries of the proximal tibial physis have been described, all in boys 15 or 16 years of age as they started or landed from a jump ( 95). Many had closure of the physis posteriorly, resulting in genu recurvatum deformity. Vascular injury has not been reported with this injury (96). These fractures represent a transition between tibial tubercle fractures and tibial epiphyseal separations, in comparing the mechanism of injury and fracture anatomy.



**FIGURE 23-23. A:** Comminuted Salter-Harris type II fracture of the proximal tibial epiphysis in a 9-year-old boy. Additional injuries included peroneal nerve injury, popliteal artery laceration, and compartment syndrome of the tibia. **B:** At follow-up, note valgus deformity of the proximal tibia with delayed union of the proximal tibial metaphysis.

Injury to the proximal tibial epiphysis can occur from passive extension of the legs of a newborn at the time of a difficult breech delivery ( 74). Separations of this epiphysis have occurred during passive manipulation of the lower limbs in infants with arthrogryposis ( 26), and attempts at closed manipulation of valgus deformities have produced this lesion. Pathologic separations have been reported in association with osteomyelitis of the proximal tibia or meningomyelocele ( 27,84).

### Classification

Most separations of the proximal tibial epiphysis are Salter-Harris type I and II injuries ( Table 23-6 and Table 23-7). The frequency of Salter-Harris type III injuries may be skewed by the inclusion of displaced avulsion fractures of the tibial tubercle, and the incidence of Salter-Harris type IV injuries depends on whether open injuries to the knee (i.e., lawn mower) are included, as in the series by Burkhart and Peterson ( 87).

Classification	Implications
<b>Mechanism of injury</b>	
I. Hyperextension	Risk of vascular disturbance
II. Varus/valgus	Usually results from jumping; very near maturity
III. Flexion	Pes anserinus or periosteum may be entrapped
<b>Salter-Harris pattern</b>	
I	Fifty percent nondisplaced
II	Thirty percent nondisplaced
III	Associated collateral ligament injury possible
IV	Rare
V	Has been reported; diagnosis usually late

**TABLE 23-6. CLASSIFICATIONS AND IMPLICATIONS OF PROXIMAL TIBIAL PHYSEAL FRACTURES**

Salter-Harris Classification	Aitken <sup>a</sup>	Burkhart <sup>b</sup>	Shelton <sup>c</sup>	Total
I	—	3	9	12 (15%)
II	9	9	17	35 (43%)
III	2	4 <sup>d</sup>	10 <sup>e</sup>	16 (22%)
IV	3	0 <sup>f</sup>	3	6 (7%)
V	—	2	—	2 (2%)
				81

<sup>a</sup>Aitken AP. Fractures of the proximal tibial epiphyseal cartilage. *Clin Orthop* 1962;41:52-57.  
<sup>b</sup>Burkhart OS, Peterson PA. Fractures of the proximal tibial epiphysis. *J Bone Joint Surg Am* 1976;58:896-902.  
<sup>c</sup>Shelton WR, Canale ST. Fractures of the tibia through the proximal tibial epiphyseal cartilage. *J Bone Joint Surg Am* 1976;58:163-172.  
<sup>d</sup>Includes avulsion fractures of the tubercle extending up into the proximal tibial epiphysis.  
<sup>e</sup>Includes five open injuries from rotary lawn mowers.

**TABLE 23-7. FREQUENCY OF TYPES OF FRACTURE-SEPARATION OF THE PROXIMAL TIBIAL EPIPHYSIS**

Fifty percent of Salter-Harris type I separations of the proximal tibia are nondisplaced. Stress radiographs may reveal widening on the medial or posterior aspect. If displaced, the metaphysis is medial or posterior relative to the epiphysis. Presumably, the overhanging tubercle prevents anterior displacement and the fibula prevents lateral displacement of the metaphysis. There may be an associated fracture of the proximal diaphysis of the fibula or a separation of the proximal fibular epiphysis.

Two thirds of Salter-Harris type II fracture-separations of the proximal tibial epiphysis are displaced. Displacement of the tibial metaphysis usually is medial ( Fig. 23-24), and the associated metaphyseal fracture usually is lateral, resulting in valgus deformity. The proximal fibula also may be fractured.

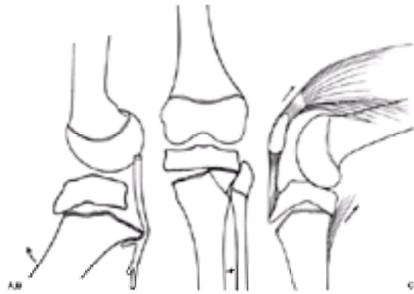


**FIGURE 23-24. A:** Salter-Harris type II separation of the proximal tibial metaphysis, with medial displacement of the proximal tibial metaphysis and complete fracture of the upper third of the fibula with complex soft tissue injury. **B:** After reduction and percutaneous fixation with a 4.5-mm cannulated screw.

Salter-Harris type III separations have a vertical fracture line through the proximal epiphysis from the articular surface to the physis. Most commonly, the lateral epiphysis is fractured, and the MCL frequently is torn. This lateral fragment may require internal fixation, and the MCL may require surgical repair.

Salter-Harris type IV injuries can involve the medial or lateral tibial plateau. Salter-Harris type V injuries are rare but have been reported in the proximal tibia. Usually, the diagnosis is made in retrospect when progressive angulation or leg-length discrepancy is noted. A type V injury localized to the anterior portion of the physis may cause genu recurvatum. A triplane fracture–separation may rarely involve the proximal tibial epiphysis.

Proximal tibial fractures also can be classified by the direction of deformity ( Fig. 23-25). The classic hyperextension type has an apex-posterior angulation and results from forced hyperextension. The flexion type results from internally generated forces of jumping or landing and has an anterior apex. Varus and valgus types result from abduction or adduction forces.



**FIGURE 23-25.** Classification of proximal tibial physeal fractures by direction of displacement. **A:** Type I: hyperextension type, usually caused by direct force. Risk of vascular damage exists. **B:** Type II: varus or valgus type. Less risk of vascular injury. Reduction may be inhibited by interposition of pes anserinus or periosteum. **C:** Type III: flexion type (rare), usually caused by internal forces as in jumping. Carries least risk of vascular injury; occurs near end of growth.

### Signs and Symptoms

P>A patient with a separation of the proximal tibial epiphysis usually has a knee joint tense with hemarthrosis. Extension is limited because of hamstring spasm. Typically, tenderness is present over the proximal tibial physis 1 to 1.5 cm distal to the joint line.

If the proximal end of the metaphysis is displaced posteriorly, a concavity is seen and felt anteriorly at the level of the tibial tubercle. If the metaphysis is displaced medially, a valgus deformity is present. There may be tenderness or angulation of the proximal fibula as well. If the proximal end of the metaphysis protrudes under the subcutaneous tissues on the medial aspect of the knee, a tear of the distal end of the MCL should be suspected.

Vascular status must be carefully documented. Routine angiography is not mandatory because ischemia usually resolves with reduction, but motor function, pulses, warmth, and color should be checked frequently during the initial 48 to 72 hours. Welch and Wynne ( 94) described a nondisplaced separation of the proximal tibial epiphysis that was initially misdiagnosed as an isolated tear of the MCL because tenderness over the medial aspect of the proximal tibia was interpreted as evidence of an avulsed ligament. Operative exploration showed the distal attachment of the ligament was intact below the level of the physis. Stress radiographs revealed an epiphyseal separation.

Fracture–separations of the ipsilateral distal femoral and proximal tibial epiphyses may occur simultaneously ( Fig. 23-26). This injury in children is analogous to the floating knee injury in adults. Internal derangement of the knee joint may occur with separation of the proximal tibial epiphysis. Concomitant avulsion of the tibial eminence also has been reported.



**FIGURE 23-26.** **A:** Salter-Harris type IV fracture of the proximal tibia and Salter-Harris type III fracture of the distal femur treated with open reduction and internal fixation. **B:** Four years later, growth is restored.

### Radiographic Findings

Nondisplaced separations may not be visible radiographically. Swelling about the proximal tibia may obscure soft tissue shadows. An associated hemarthrosis is manifested by an increased space between the patella and distal femur. Stress radiographs in both coronal and sagittal planes should be obtained, but hyperextension of the knee should be avoided because of the possibility of injury to the popliteal vessels.

The radiographs are scanned for evidence of fracture lines extending proximally through the epiphysis or distally through the metaphysis. A small bony fragment at the periphery of the metaphysis may be the only clue to the diagnosis. Fracture lines may be visible only on oblique views.

Most patients with separations of the proximal tibial epiphysis are adolescents in whom the secondary ossification of the tibial tubercle has appeared. A smooth, horizontal radiolucent line through the base of the tubercle should not be confused with an epiphyseal fracture. It may represent an incomplete fusion of the two secondary ossification centers: the tubercle ossicle and the main portion of the proximal tibial epiphysis.

Computed tomographic scans may be helpful in determining the treatment of Salter-Harris type III and IV fracture–separations. MRI may identify soft tissue interposition in displaced proximal tibial epiphyses, which are difficult to reduce by closed methods. Wood et al. ( 95) reported a patient in whom the pes anserinus was folded into the fracture site.

### Treatment

#### Nonoperative Treatment

Hyperextension fractures are reduced with a longitudinal force on the tibia combined with a gentle, anterior translating force on the proximal metaphysis. Countertraction on the femoral shaft is applied by an assistant.

Nicholson (90) reported reduction of posterior displacement of the metaphysis by pulling the proximal tibia forward and flexing the knee to 90 degrees with mild internal rotation of the tibia.

Böhler (9) advised placing a calcaneal pin. The leg is then flexed over the surgeon's forearm, which is placed in the popliteal fossa. Longitudinal traction is applied to the pin while the surgeon's arm lifts up. Upward traction on the proximal tibial metaphysis may be accomplished with a second pin inserted under the tibial tubercle. A long leg cast is applied after reduction is obtained.

An abduction fracture with valgus angulation usually can be reduced by adducting the leg on the extended knee. This should be a gentle manipulation to decrease the risk of injury to the peroneal nerve. After reduction, a long leg cast with varus molding is applied with the knee in slight flexion.

### **Indications for Operative Treatment**

Separations of the proximal tibial epiphysis may be surprisingly unstable. Smooth pins can be inserted percutaneously, crossing distal to the physis to maintain reduction. The proximal ends should not protrude into the knee joint. An image intensifier makes percutaneous fixation easier.

Open reduction is indicated for displaced Salter-Harris type III injuries. An anterior incision is used to allow inspection of the articular surface. A pin is inserted in the displaced fragment and is used to guide it toward reduction. Other pins or screws are then inserted horizontally across the epiphysis. Small cannulated screws often are helpful in this type of fracture.

A similar technique is used for displaced Salter-Harris type IV injuries. Fixation can be obtained by placing screws across the larger metaphyseal, as well as the epiphyseal, fragments (Fig. 23-26). If there is a linear peripheral tear of the meniscus overlying the injured condyle, repair is performed. Primary repair of concomitant complete ligamentous injury also should be considered.

Operative fixation of a hyperextension injury may be indicated for stabilization if an associated popliteal artery injury is repaired. Immediate exploration is not indicated for symptoms of peroneal nerve injury. Peroneal neuropathy associated with a separation of the proximal tibial epiphysis usually recovers spontaneously with time.

### **AUTHOR'S PREFERRED METHOD OF TREATMENT**

If a proximal tibial epiphysis is nondisplaced, we place the patient in a long leg cast with the knee flexed 30 degrees. The cast and underlying padding are initially bivalved from top to bottom. Anteroposterior and lateral radiographs are repeated 1 week after injury to confirm acceptable position. The cast is removed 6 to 8 weeks after injury. If subperiosteal new bone formation has appeared and if the leg is nontender, active range-of-motion and muscle-strengthening exercises are begun. We do not allow the patient to return to vigorous activities until the region is nontender and knee range of motion and quadriceps strength are near normal.

Before reducing a Salter-Harris type I hyperextension injury, we check for signs of neurologic or circulatory impairment. If the displacement is significant, we prefer to use general anesthesia for reduction. With the patient supine, the fracture is reduced by flexing the hip and knee to 45 degrees while applying longitudinal traction. The upper leg is grasped behind the calf. The distal tibia is stabilized, usually by holding it between the surgeon's knees or between his or her elbow and chest. The proximal metaphysis is pulled gently forward. At this point, flexion of the knee is increased to 90 degrees, the peripheral pulses are checked again, and a lateral radiograph is obtained. If reduction is satisfactory, a long leg cast with the knee flexed at 60 degrees is applied, and the cast is bivalved from top to bottom. Direct pressure over the proximal posterior tibia should be minimal. Radiographs are obtained the next day and 1 week later. The cast is changed to extend the knee to 30 degrees at 3 to 4 weeks. At 6 to 8 weeks after injury, the cast is converted to a posterior splint, and active range-of-motion exercises are begun.

For an abduction injury with valgus angulation, we sedate the patient, aspirate any significant knee effusion under sterile precautions, and test carefully for a concomitant tear of the MCL or cruciate ligament. Valgus angulation is corrected by putting manual longitudinal traction on the leg. The knee is held in almost full extension. With an assistant stabilizing the distal thigh and knee, the valgus angulation is corrected by guiding the leg into adduction. Traction is not released until reduction is accomplished. If the radiographs confirm reduction, the extremity is immobilized in a long leg cast, molded into varus, with the knee flexed 15 to 20 degrees. Neurovascular status is checked before and after reduction. The cast is removed in 6 to 8 weeks, and a knee immobilizer may be used for support for an additional 2 to 4 weeks.

For flexion injuries, reduction usually is obtained by applying longitudinal traction with the knee in full extension. An extension cast is worn for 4 to 6 weeks, after which muscle-strengthening and range-of-motion exercises are begun. If a Salter-Harris type I or II fracture is unstable after reduction, percutaneous crossed Kirschner wires should be used for fixation. These can be inserted from a distal-to-proximal direction to avoid passing through the joint capsule. Smooth  $\frac{5}{64}$ -or  $\frac{3}{32}$ -in. Kirschner wires can be used and removed 6 to 8 weeks after insertion.

For nondisplaced Salter-Harris type III separations, a long leg cast is worn for 6 to 8 weeks. If displacement exceeds 2 mm, we perform closed or open reduction and fixation with percutaneous pins or cannulated screws. After the patient has been anesthetized, a Steinmann pin is inserted into the fragment and used to guide the fragment into position with the help of an image intensifier. After reduction, one or two smooth pins or small cannulated screws are inserted transversely across the epiphysis, taking care not to cross the physis in a young child. After internal fixation has been obtained, the knee is again carefully stressed into valgus to see if the MCL is intact. Similarly, a lateral view with stress in the sagittal plane is obtained to rule out associated cruciate injury. If a tear of the MCL is detected, we may proceed with primary ligamentous repair, especially in an adolescent.

A similar technique is used for reduction and fixation of Salter-Harris type IV separations. If reduction is not anatomic, we perform an open reduction under direct vision. Fixation pins may be removed under brief general anesthesia when the cast is removed, 6 to 8 weeks after injury. If the fracture is open and the fragment is devitalized (i.e., lawn mower injury), the avascular piece is removed. Non-weight bearing is advised for 2 months after Salter-Harris type III and IV injuries, but active range-of-motion exercises should be started immediately after removal of the cast.

The patient is followed for 2 years to watch for signs of angular deformity or persistent instability.

### **Prognosis**

The overall prognosis for separations of the proximal tibial epiphysis is fairly good. Shelton and Canale (93) found that 24 of 28 patients (86%) followed until after skeletal maturity had satisfactory results. If a small group of lawn mower injuries is separated from the series of Burkhart and Peterson (87), 76% of the remaining 21 patients did well after initial reduction. Most of the fractures in both series occurred in adolescents, and most injuries were closed.

Open injuries of the proximal tibia have a much worse prognosis. All the lawn mower injuries reported by Burkhart and Peterson (87) had adverse sequelae. Four patients had significant angulation (Fig. 23-27), two patients had major leg-length discrepancies, one contracted osteomyelitis, and one had a severed peroneal nerve. Direct crushing injuries to the knee with extensive bruising or laceration of overlying soft tissue also have a poor prognosis.



**FIGURE 23-27.** Despite anatomic reduction, growth disturbance (*solid arrow*) and valgus deformity occurred after metaphyseal fracture of the proximal tibia caused by a lawn mower accident. The fracture proved to be a Salter-Harris type IV injury because of fracture extension through the unossified epiphysis.

Bertin and Goble (7) warned that nearly half the patients with proximal tibial physeal fractures can be expected to have ligamentous instability 5 years after injury. They noted that persistent MCL insufficiency, genu valgum, and early degenerative changes in the knee could follow Salter-Harris type III injuries. Poulsen et al. (91) found that 6 of 11 patients complained of pain or discomfort at an average follow-up of 7 years. Four patients had persistent ligamentous laxity, and three patients had degenerative changes on radiographs.

The Salter-Harris classification is not always useful in predicting risk of growth disturbance after separation of the proximal tibial epiphysis. Complete or partial growth arrest can occur after Salter-Harris type I, II, and III injuries, as well as after type IV and V injuries. Burkhart and Peterson (87) reported two patients with significant leg-length discrepancies and angular deformities after Salter-Harris type I and II injuries. Shelton and Canale (93) reported that all seven patients in their series with growth disturbances had fractures classified as Salter-Harris type I or II. No significant growth disturbances were associated with 13 Salter-Harris type III or IV injuries. Subsequent osteoarthritic changes in the knee were related to fracture patterns with intraarticular extension.

The direction and degree of displacement are factors in the frequency of associated neurovascular or ligamentous injuries. Posterior displacement of the metaphysis relative to the epiphysis is more likely to cause neurovascular or ligamentous injury than is displacement in the medial or anterior direction. Hyperextension injuries often cause neurovascular deficits because of impingement and stretch on structures running through the popliteal fossa.

### Complications

Complications of proximal tibial fractures may be early or late. Table 23-8 shows the incidences of the most common complications from major series.

Complication	Incidence
Vascular impairment	10%
Peroneal palsy	3%
Knee instability/degenerative joint disease	33%
Growth disturbance	10%-25%

Data from references 53, 87, 89, 90, 93, and 96.

**TABLE 23-8. COMPLICATIONS OF PROXIMAL TIBIAL FRACTURES**

### Instability

Separations of the proximal tibial epiphysis are surprisingly unstable, regardless of the Salter-Harris type. This is because of loss of periosteal stability or ineffective splinting from surrounding soft tissues. The fact that the collateral ligaments do not attach to the proximal tibial epiphysis also may be a factor in instability. It is wise to obtain radiographs 1 week after injury to check reduction. At that point, it still is not too late to remanipulate the extremity if necessary. If reduction cannot be maintained initially, percutaneous pin fixation or open reduction with internal fixation may be indicated, especially with displaced fractures. In unstable fractures with vascular injury, reduction and pin fixation should precede exploration of the popliteal artery. If the knee is swollen, sufficient flexion to maintain reduction of an anteriorly displaced separation may compromise circulation of the lower leg. Internal fixation allows flexibility in positioning the knee in a cast after injury.

### Vascular Injury

Nicholson's report (90) of a patient with associated arterial injury is notable. The patient was seen less than an hour after an injury sustained while playing football. The knee was tense with effusion and extremely tender. There was a deep depression in the area of the tibial tubercle, indicating posterior displacement of the metaphysis relative to the epiphysis. The foot was warm, but the patient could not move his toes actively. Both pulses in the foot were absent and did not return after reduction of the fracture. Exploration of the popliteal fossa revealed that both the popliteal artery and vein were completely severed. An attempt at anastomosis failed and amputation was performed.

Burkhart and Peterson (87) reported a patient with vascular occlusion. A 12-year-old hurdler sustained a closed Salter-Harris type III injury that was treated with closed reduction and a long leg cast. Increased pressure in both the anterior and posterior muscle compartments caused narrowing of the terminal branches of the popliteal artery, although arteriography showed that the popliteal artery itself remained patent. Fasciotomies and a sympathectomy failed to save the leg.

Arterial insufficiency may result from either a tear in the popliteal artery at the time of epiphyseal separation ( Fig. 23-28) or from a compartment syndrome. Delay in recognition results in delay of treatment, which is potentially catastrophic. Arteriography is not mandatory for isolated injuries but may be helpful if the vascular supply is questionable. Fracture fixation should be followed by arterial exploration (posterior or posteromedial approach) and repair or vein grafting. Compartment syndrome is treated by fasciotomy and delayed wound closure or skin grafting.



**FIGURE 23-28.** Anteroposterior radiograph at the time of arteriogram shows a Salter-Harris type III fracture–separation of the proximal tibial epiphysis. The arterial flow through the popliteal vessels is blocked.

### Premature Growth Arrest

Angulation from a localized area of growth inhibition may occur after any type of proximal tibial separation. The degree of progressive angulation depends on the proximity of the area of growth arrest to the periphery of the physis. The amount of angulation also depends on the years left for growth after injury.

If the area of growth arrest is localized to less than a third of the total area of the physis and at least 2 years of growth remain, resection of the bony bridge may be attempted. Alternatively, epiphysiodesis or stapling of the side of the physis opposite a peripheral bar may stop progression of the deformity ( 88). Existing deformity at the time of surgery may require proximal tibial osteotomy. If there is significant valgus deformity, an opening-wedge osteotomy to correct valgus deformity and gain length may injure the peroneal nerve. Slow correction after corticotomy, with guided lengthening using an external fixator by surgeons familiar with this technique, is an option in patients close to skeletal maturity who have severe angular deformity and a large leg-length discrepancy.

### Leg-Length Discrepancy

The proximal tibial physis grows approximately ¼ in. per year. If complete growth arrest follows an epiphyseal separation at this level and the patient is within 3 years of the end of growth, an equalization procedure is unnecessary. If more years of growth remain, epiphysiodesis of the opposite extremity may be considered. If arrest occurs before age 6 to 8 years, leg lengthening at a later stage may be indicated. If the growth arrest is partial, it is helpful to measure with sequential scanograms the relative growth rates of the two extremities so that the discrepancy at skeletal maturity can be predicted. If the predicted discrepancy is less than 2.5 cm, no equalization procedure is planned. If the expected discrepancy is 2.5 to 5 cm, epiphysiodesis of the opposite lower extremity is appropriate. If the expected discrepancy exceeds 5 cm, limb lengthening should be considered.

## AVULSION OF THE TIBIAL TUBERCLE

Considering the force with which the contracting quadriceps muscle pulls on its insertion, especially during jumping (concentrically) or landing (eccentrically), it is surprising that the tibial tubercle is not avulsed more frequently. The incidence relative to injuries of other apophyses remains ill defined for two reasons. First, the severe type of avulsion of the tibial tubercle that extends proximally through the upper epiphysis of the tibia into the knee joint is sometimes reported as a Salter-Harris type III separation of the proximal tibial epiphysis. Second, an acute localized avulsion fracture of the tibial tubercle is sometimes thought to be a variant of the Osgood-Schlatter (OS) lesion. For example, Shelton and Canale ( 93) and Burkhart and Peterson ( 87) included extraarticular tubercle avulsions in their reviews of proximal tibial physeal separations, whereas Ogden ( 53) included in his series of tibial tubercle avulsions some cases that overlap with OS lesion.

The incidence of avulsion fractures of the tibial tubercle, compared with all epiphyseal injuries, is probably between the 0.4% reported by Burkhart and Peterson ( 87) and the 2.7% reported by Ogden et al. ( 117). The difficulty in separating avulsion fractures of the tibial tubercle from OS lesions stems largely from the lack of precise definition of either entity. Some differences are listed in Table 23-9, but some clinical features are similar. Some patients with OS lesions have a history of specific trauma. Avulsion fractures of the tibial tubercle localized to the distal fragment of the tubercle (type I) occasionally cause little more than point tenderness.

Osgood-Schlatter Lesion	Acute Traumatic Avulsion of the Tibial Tubercle
Onset often insidious	Acute injury (often in athletics)
Intermittent mild symptoms	Immediate marked pain and swelling
Partial disability	Often unable to stand or walk
Symptomatic and supportive treatment	Often open reduction and internal fixation
Prognosis fairly good (occasionally long-term symptoms with ununited ossicle)	Rapid healing and return to full activities

**TABLE 23-9. COMPARISON OF THE CLINICAL FEATURES OF OSGOOD-SCHLATTER LESION AND ACUTE TRAUMATIC AVULSION OF THE TIBIAL TUBERCLE**

An OS lesion may precede an acute avulsion of the tibial tubercle. Deliyannis ( 106) reported a complete avulsion of the tibial tuberosity in a 16-year-old boy who had been seen previously for an OS lesion. Levi and Coleman ( 115) stated that 4 of 15 patients with fractures of the tibial tubercle had histories of OS lesions. Ogden et al. ( 117) reported that 9 of their 14 patients with fractures of the tibial tuberosity had preexisting OS lesions, 7 in the contralateral knee and 2 in the ipsilateral knee. Ogden ( 53) defined an OS lesion as an avulsion of the anterior surface of the apophysis. He stated that there is no separation between the ossific nucleus of the apophysis and the adjacent tibial metaphysis. If there is a separation through the physis deep to the ossific nucleus of the tubercle, an avulsion fracture of the tubercle has occurred. This distinction often is difficult to recognize on radiographs of an incompletely ossified tibial tubercle.

In an adolescent, the patellar ligament inserts not only into the developing tibial tubercle, but beyond into the adjacent perichondrium of the physis and the periosteum of the adjacent metaphysis ( 107, 117). With an OS lesion, the central fibers of the patellar ligament avulse a localized fragment of surface cartilage and bone from the secondary ossification center. The displacement of this fragment is not severe because the remaining fibers of the patellar ligament that fan out medially and laterally remain intact. With a limited, minimally displaced avulsion fracture of the tibial tubercle, a cleavage plane is present through part of the physis between the secondary ossification center of the tubercle and the adjacent metaphysis of the proximal tibia. The perichondrium is torn adjacent to the separated portion of the physis. A severely displaced avulsion fracture of the tibial tubercle implies a more extensive tear of the fibrous expansion of the insertion of the patellar ligament.

In 1971, Hand et al. ( 109) reported seven avulsion fractures of the tibial tubercle, all in boys 14 to 16 years of age and all resulting from sports or play activities. Six of the seven patients were treated successfully by open reduction and internal fixation. These authors noted that a large periosteal flap could become folded into the gap underneath the avulsed tubercle.

Levi and Coleman ( 115) reported uniformly good results in 14 patients with avulsions of the tibial tubercle. All but one of the patients were boys, with an average age of 14 years, and all injuries were sustained during sports or play activities. All were treated by open reduction, with various forms of fixation. The authors warned that the size of the avulsed fragment often is underestimated because the tibial tubercle is incompletely ossified at the time of injury.

In an in-depth study of 14 fractures of the tibial tubercle by Ogden et al. ( 117), the sequence of events in the morphologic development of the tibial tuberosity was related to the narrow age range in which these fractures occur. An analogy was drawn to the fracture of Tillaux because both injuries occur at an age when physiologic epiphysiodesis has begun. Two pathologic fractures were included in this report.

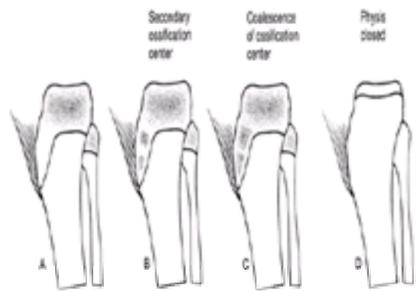
In 1981, Christie and Dvonch ( 103) reported eight avulsion fractures of the tibial tubercle in seven patients. All the injuries occurred in boys 13 to 17 years of age who were playing basketball. The degree of displacement varied. The authors recommended an attempt at closed reduction, even in severely displaced fractures, before resorting to open reduction and internal fixation. They pointed out that the healing of this injury occurs by epiphysiodesis, but genu recurvatum rarely results because the patients are close to skeletal maturity.

## Surgical Anatomy

### Bony Anatomy

In its final adult form, the tibial tubercle is a bony prominence on the anterior aspect of the proximal tibia. It lies approximately one to two fingerbreadths distal to the proximal articular surface of the tibia and forward of the anterior rim of the proximal articular surface. The tubercle is at a point where the broad, flat anterior surface of the tibial condyles narrows sharply to become the anterior border of the diaphysis. Helfet ( [110](#)) showed that the tibial tubercle is in line with the medial half of the patella when the knee is flexed and with the lateral half when the knee is extended. Thus, the position of the tibial tubercle in the coronal plane relative to the patella is a function of the position of the knee joint. This is because the proximal end of the tibia rotates relative to the distal end of the femur with flexion and extension movements of the knee.

Ehrenborg ([107](#)) divided the postnatal development of the tibial tubercle into four stages ( [Fig. 23-29](#)). The cartilaginous stage occurs before the secondary ossification center appears and persists in girls until 9 years and in boys until 10 years of age. The apophyseal stage, in which the ossification center appears in the tongue of cartilage, occurs between 8 and 12 years in girls and between 9 and 14 years of age in boys. The epiphyseal stage, in which the secondary ossification centers coalesce to form a tongue of bone continuous with the proximal tibial epiphysis, occurs in girls between 10 and 15 years and in boys between 11 and 17 years of age. In the final bony stage, the epiphyseal line is closed between the fully ossified tuberosity and the tibial metaphysis.



**FIGURE 23-29.** Development of the tibial tubercle. **A:** In the cartilaginous stage, there is no ossification center in the cartilaginous anlage of the tibial tubercle. **B:** In the apophyseal stage, the secondary ossification center or centers forms in the cartilaginous anlage of the tibial tubercle. **C:** In the epiphyseal stage, the primary and secondary ossification centers of the proximal tibial epiphysis have coalesced. **D:** In the bony stage, the proximal tibial physis has closed.

### Soft Tissue Anatomy

The patellar ligament, which lies between the distal pole of the patella and the tibial tubercle, is the terminal portion of the inserting tendon of the powerful quadriceps muscle. During the apophyseal stage of development of the tubercle, the patellar ligament inserts into an area approximately 10 mm long, corresponding to the fibrous cartilage proximal and anterior to the secondary ossification center. The main attachment is in the proximal area of this insertion zone, at the level of the cartilage lying between the secondary ossification centers of the tubercle and the main portion of the proximal tibial epiphysis. The fibrocartilaginous tissue lying anterior to the secondary ossification center receives only the distal part of the insertion. During the epiphyseal stage, the patellar ligament inserts through fibrocartilage on the anterior aspect of the downward-projecting tongue of the proximal tibial epiphysis. The inserting fibers merge distally into deep fascia after spanning the physis. With traumatic avulsion of the tibial tubercle in this stage of development, a broad flap of adjacent periosteum is attached to the displaced fragment. In the final bony stage, the tendon fibers insert directly into bone. After physiologic epiphysiodesis has occurred, the tibial tubercle rarely is avulsed if the patient has normal bone.

Although the patellar ligament represents the main insertion of the quadriceps muscle onto the leg beyond the knee joint, it is reinforced by retinacular fibers radiating from the medial and lateral margins of the patella obliquely down to the respective tibial condyles. Also, longitudinally oriented retinacular fibers extend from the distal margin of the vastus lateralis down to the anterior aspect of the lateral tibial condyle, partly merging with the inserting fibers of the iliotibial band ( [120](#)). Similarly, longitudinal fibers extend from the distal margin of the vastus medialis down to the anterior aspect of the medial tibial condyle. Frazer ( [100](#)) showed that the insertion of the medial retinaculum extends beyond the proximal tibial physis into the metaphysis. After traumatic avulsion of the tibial tuberosity, a limited amount of active extension of the knee still is possible through the retinacular extensions of the extensor mechanism. However, patella alta and an extensor lag are present.

The anatomic position of the tibial tubercle is biomechanically important ( [102,105,114](#)). It is one factor that determines the length of the moment arm from the patellar ligament to the center of rotation of the knee ( [113](#)). This moment arm helps determine the amount of patellofemoral reaction force developed by extending the knee against resistance ( [116](#)).

### Vascular Anatomy

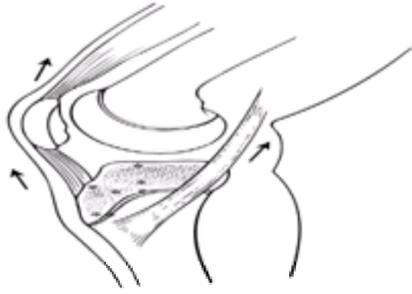
The tibial tubercle receives its main blood supply from an anastomosis behind the quadriceps tendon. In particular, a prominent leash of vessels bilaterally arises from the anterior tibial recurrent artery and may be torn with this fracture ( [118,122](#)). Several small branches extend down into the secondary ossification center. A smaller part of the blood supply enters the superficial surface of the tubercle from adjacent periosteal vessels. Trueta ( [121](#)) showed in injection studies that a few of the longitudinal vessels in the patellar ligament itself extend into the tubercle. Ogden et al. ( [117](#)) showed blood vessels entering the medial and lateral aspects of the tubercle. They demonstrated cartilage canals in the physis carrying these branches of the metaphyseal vessels in children up to 10 to 12 years of age.

### Mechanism of Injury

Acute traumatic avulsions of the tibial tubercle occur most often during sports or play activities ( [97,104,108,112,119](#)). Böhler ( [9](#)) stated that most avulsions originated from "a leap over a wooden horse" in physical training. Of the seven avulsions reported by Hand et al. ( [109](#)), four occurred while playing football and three while jumping during play. Of the 15 injuries reported by Levi and Coleman ( [115](#)), 5 occurred while playing basketball, 2 while diving from a springboard, 2 during competitive running, and 1 during a high jump. All eight injuries reported by Christie and Dvonch ( [103](#)) occurred while playing basketball.

Avulsion of the tibial tubercle occurs when the patellar ligament traction exceeds the combined strength of the physis underlying the tubercle, the surrounding perichondrium, and the adjacent periosteum. This can occur in two ways. The first mechanism is violent contraction of the quadriceps muscle against a fixed tibia. This can happen when an athlete jumps or lands, as in basketball or track. The second mechanism is acute passive flexion of the knee against the contracted quadriceps. This can happen when a person makes a bad landing at the end of a jump or fall. In other words, avulsion of the tubercle can result from sudden acceleration or deceleration of the knee extensor mechanism.

Some predisposing factors to avulsion of the tibial tubercle have been identified. Patella infera, with a relatively short patellar ligament, can impose increased tension on the distal insertion of the quadriceps mechanism. Tight hamstrings can increase flexion torque, opposing active extension of the knee ( [Fig. 23-30](#)). As mentioned earlier, the weak link in the extensor mechanism in adolescents is the changing physis beneath the tibial tubercle. Acute disruptions are more common in knees with preexisting OS disease, which indicates a mechanical vulnerability from either overdevelopment of the quadriceps mechanism, lack of flexibility of the knee flexors, or impaired coordination between opposing torques about the knee. In addition, Ogden et al. ( [117](#)) reported two patients with what were probably pathologic fractures. One injury occurred in a child with myelomeningocele, and the other patient had metaphyseal dysplasia with preexisting varus deformity of the injured knee.

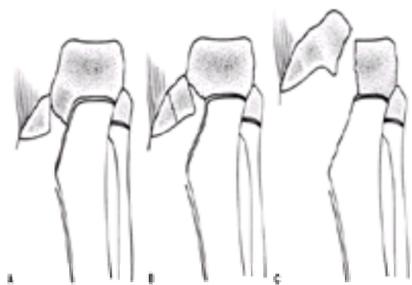


**FIGURE 23-30.** Contracting quadriceps mechanism exerts extension torque on the knee joint, whereas contracting hamstring muscles exert flexion torque on the knee joint. Asterisks indicate points of possible failure when the knee is jammed by opposing torques.

### Classification

Watson-Jones (82) described three types of avulsion fractures of the tibial tubercle. In the first type, a small fragment, representing part of the tubercle, is avulsed and displaced upward. In the second type, the secondary center of ossification in the tubercle has already coalesced with the rest of the proximal tibial epiphysis, and the entire lip formed by the front of the upper tibial epiphysis is hinged upward. In the third type, the line of fracture passes proximally across the proximal physis of the tibia.

Ogden and associates (117) refined the classification and described three types, depending on the distance of the fracture from the distal tip of the tubercle ( Fig. 23-31). Each type is divided into two subtypes, depending on the severity of displacement and comminution:



**FIGURE 23-31.** Classification of avulsion fractures of the tibial tubercle. **A:** Type I fracture across the secondary ossification center level with the posterior border of the inserting patellar ligament. **B:** Type II fracture at the junction of the primary and secondary ossification centers of the proximal tibial epiphysis. **C:** Type III fracture propagates upward across the primary ossification center of the proximal tibial epiphysis into the knee joint. This fracture is a variant of the Salter-Harris type III separation and is analogous to a Tillaux fracture of the ankle because the posterior portion of the proximal tibial physis is closing. (From Ogden JA, Tross RB, Murphy MJ. Fractures of the tibial tuberosity in adolescents. *J Bone Joint Surg Am* 1980;62:205–215; with permission.)

Type I—The separation through the distal portion of the physis under the tubercle breaks up proximally through the secondary ossification center of the tubercle.

Type II—The separation extends anteriorly through the area bridging the ossification centers of the tibial tubercle and the proximal tibial epiphysis.

Type III—The separation beneath the tubercle propagates through the proximally proximal tibial epiphysis into the knee joint under the anterior attachments of the menisci.

Ogden noted that the degree of displacement depends on the severity of injury to adjacent soft tissue attachments.

### Signs and Symptoms

Swelling and tenderness are centered over the anterior aspect of the proximal tibia. Joint effusion and tense hemarthrosis may be present. A freely movable triangular fragment of bone may be palpated subcutaneously between the proximal tibia and the femoral condyles. This fragment may have rotated so that the distal end projects forward, tenting the overlying skin. If the avulsed fragment is held and moved passively, crepitus may be felt between the fragment and the proximal tibia. A palpable defect on the anterior aspect of the tibia at the upper end of the anterior border of the diaphysis represents the bed from which the fragment was avulsed. In a markedly displaced Watson-Jones type III avulsion, the palpable defect can be as much as 3 cm long.[nb

The injured knee is held in 20 to 40 degrees of flexion by hamstring spasm. The amount of patella alta is proportional to the severity of displacement of the tibial tubercle. The patella may be displaced proximally as much as 10 cm. With associated joint effusion, the patella may seem to float off the anterior aspect of the distal femur. With a type I avulsion, the patient usually can actively extend the knee, although not completely. With type II and III lesions, full extension is impossible.

### Radiographic Findings

Because the tubercle lies just lateral to the midline of the tibia, the best profile can be obtained by a lateral projection, with the tibia rotated slightly internally. Soft tissue technique helps define the margins of the patellar ligament and smaller fragments of bone. In children and adolescents 9 to 17 years of age, the normal tubercle is ossified to varying degrees. One or more secondary ossification centers form at the distal end of the tubercle during the apophyseal stage of development. The presence of several centers does not represent an abnormality. Multiple ossification centers coalesce and join with the proximal tibial epiphysis during the epiphyseal stage of development. At this time, a horizontal radiolucent band becomes apparent on the anteroposterior radiograph at the distal end of the tubercle. The band represents a groove filled with cartilage between the tip of the tibial tubercle and the upper end of the anterior border of the diaphysis. During the epiphyseal stage of development, the borders of the tubercle are sharply defined.

After an avulsion of the tibial tubercle, the size and degree of displacement of the fragment are best seen on the lateral radiograph ( Fig. 23-32). In a type I injury, the distal end of the tubercle is displaced upward and forward to varying degrees. The avulsed fragment corresponds to that part of the tubercle between the proximal border of the inserting patellar ligament and the distal end of the secondary ossification center. In the second type of avulsion of the tubercle, the fragment is larger, hinging or separating at the level of the horizontal portion of the proximal tibial physis. In type III, the fracture line extends proximally from beneath the tubercle through the proximal tibial epiphysis and exits the upper surface of the epiphysis ( Fig. 23-33). The fracture line emerges anterior to the tibial spine. If this type of injury is comminuted, fragmentation occurs at the level of the horizontal component of the proximal tibial physis. In a type III lesion, the central portion of the physis beneath the proximal tibial epiphysis may have already closed. Bruijn et al. (101) reported a “sleeve” fracture of the tibial tubercle in a 14-year-old gymnast; his radiographs appeared normal, but 6 months later a 4-cm, ossified, proximally displaced sleeve representing the base of the patellar tendon was seen.



**FIGURE 23-32.** Three variations of avulsions of the tibial tubercle. **A:** Anterior injury with avulsion of the tubercle and anterior aspect of the epiphysis. **B:** Classic Salter-Harris type III fracture, beginning in the physis and extending into the articular surface of the epiphysis. **C:** Comminuted epiphyseal-metaphyseal fracture of the proximal tibia.



**FIGURE 23-33.** **A:** A 15-year-old boy with a comminuted, displaced tibial tuberosity avulsion sustained while jumping in a basketball game. **B:** After open reduction and internal fixation with multiple cancellous screws.

Closure of the gap between the avulsed fragment and its bed indicates satisfactory reduction. Persistence of even a small gap between the distal end of the tubercle and the adjacent metaphysis may indicate an interposed flap of periosteum.

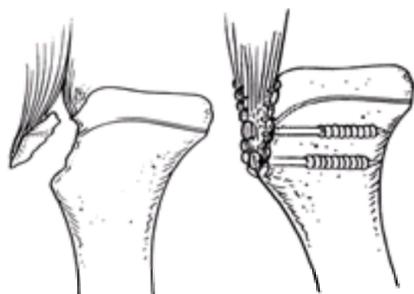
Before reduction of an avulsed tibial tubercle, the degree of patella alta and, therefore, the severity of displacement can be determined by one of several radiographic methods (98,99,111). Because the tibial tubercle is displaced proximally to the same extent as the patella, the ratio between the overall length of the patella and the distance between the patella and tibial tubercle would not change. The best method for estimating patella alta after avulsion of the tibial tubercle is that described by Blackburne and Peel (98): the position of the patella is compared with a line extrapolated forward from a tangent across the upper surface of the tibia. Even this method gives varying degrees of patellar displacement, depending on contraction or spasm in the quadriceps muscle.

### Treatment

Minimally displaced, small avulsion fragments have been treated successfully by closed methods. The leg is positioned with the knee extended and the hip slightly flexed. Reduction can be held by a long leg cast that is well molded above the proximal pole of the patella. Most authors advise open reduction and internal fixation of type II and III fractures. A midline vertical incision is recommended to facilitate any possible knee surgery in the future. The fracture bed is carefully cleared of debris. If a periosteal flap is folded under the avulsed fragment, it is extracted and held spread out while the fragment is reduced with the knee extended. Depending on the size of the avulsion fragment and the patient's age, fixation is obtained with transfixing pins or screws and is reinforced by repair of the torn periosteum.

### Operative Treatment

We recommend open reduction and internal fixation of all but the smallest undisplaced fragments. Closed methods can be used in minimally displaced fractures if the knee can be actively extended to 0 degrees. For open reduction, we prefer a vertical incision centered over the tibial tubercle, along either its medial or lateral border. We are careful to clear the gap between the displaced fragment and its bed of any soft tissue interposition. With type III fractures, the menisci should be inspected for tears. If the reduced fragment is large enough, we prefer to use one or two cancellous screws, extending through the tubercle, parallel to the joint into the metaphysis. If the patient is more than 3 years from skeletal maturity, smooth pins are used (Fig. 23-34). If the fracture is comminuted, we prefer multiple screws or threaded Steinmann pins reinforced by periosteal sutures. A strong, tension-holding suture such as the Krackow (running locking) suture may add strength to the repair; sutures also may be used in the periosteum and lateral tendon expansion. After wound closure, a well-fitting long leg or cylinder cast is applied. The cast is carefully molded on either side of the reduced tibial tubercle and proximal to the patella so that the latter is held downward.



**FIGURE 23-34.** Open reduction and internal fixation of type II tibial tubercle fracture **(A)**. After anatomic reduction **(B)**, a large fragment is stabilized with two screws.

### Postreduction Care

The cylinder or long leg cast applied after reduction of the avulsed tubercle is worn for 4 to 6 weeks and then is bivalved. For the next 2 weeks, the posterior half of the cylinder cast is worn as a splint between exercise periods. Gentle active range-of-motion and quadriceps-strengthening exercises are begun three times a day. At 6 weeks after injury, exercise of the quadriceps against some resistance can be started if there is no tenderness over the tibial tubercle. The patient is permitted to return to athletics and vigorous play only after the quadriceps strength equals that of the opposite side and range of motion has been regained.

### Prognosis

The outcome of a well-reduced avulsion fracture of the tibial tubercle is consistently good. Most patients return to normal function, including sports. All 15 patients reported by Levi and Coleman (115) had returned to normal activity without deformity or restriction 1 to 18 years after injury. All but one of the patients reported by

Ogden et al. (117) were completely asymptomatic, with no disability. All seven patients reported by Christie and Dvonch (103) had returned to normal function at an average of 3 years after injury.

### Complications

The early and late complications of avulsion of the tibial tubercle are listed in [Table 23-10](#).

#### Early

Compartment syndrome

Meniscal tears

Infection

#### Late

Genu recurvatum

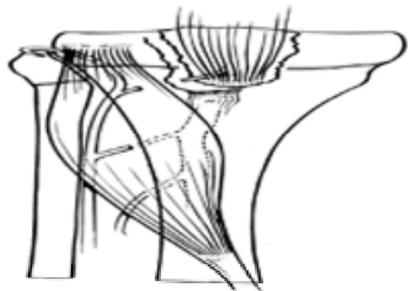
Loss of flexion

Refracture

**TABLE 23-10. COMPLICATIONS OF TIBIAL TUBERCLE AVULSION**

Blount (8) noted that genu recurvatum caused by premature closure of the anterior portion of the physis is likely to occur after avulsion of the tibial tubercle in skeletally immature children, usually those younger than 11 years of age. Genu recurvatum is rare after avulsion fracture of the tibial tubercle, despite the fact that healing actually occurs by epiphysiodesis. The explanation of this apparent paradox is that avulsions of the tubercle almost always occur in patients very close to skeletal maturity.

Compartment syndrome has been reported in seven patients with type III fractures, presumably because of tearing of anterior tibial recurrent vessels, which fan out at the tubercle but retract into the anterior compartment when torn (118,122) ([Fig. 23-35](#)). Close monitoring is necessary for patients treated nonoperatively and careful inspection, possibly with prophylactic anterior fasciotomy, is recommended for patients treated operatively (118,122).



**FIGURE 23-35.** Probable mechanism of development of compartment syndrome after tibial tubercle avulsion. The anterior tibial recurrent artery is torn and retracts into the anterior compartment musculature.

Minor complications also have been reported. Christie and Dvonch (103) reported a patient with persistent loss of 25 degrees of knee flexion 19 months after a type III injury. Hand et al. (109) reported a patient with persistent quadriceps atrophy, and Levi and Coleman (115) reported a patient with a persistent prominence 7 years after open reduction and internal fixation. To this short list could be added the expected complication of patella alta if a displaced tibial tubercle is not reduced adequately.

### OSGOOD-SCHLATTER LESION

Osgood-Schlatter (OS) lesions are considered separately from acute traumatic avulsions of the tibial tubercle because the pathophysiologic process, treatment, and outcome differ somewhat (121,129). Although acute avulsion of the tubercle is almost always related to a specific injury, the patient with an OS lesion often can give only a vague history about the onset of symptoms. Whereas an adolescent with a displaced acute avulsion of the tibial tubercle is immediately unable to stand or walk, an adolescent with OS disease often tries to continue sports and play activities despite discomfort. Treatment of acute avulsion of the tubercle depends on the size of the avulsed fragment and the degree of displacement, but usually open reduction and internal fixation are indicated. Treatment of OS lesions usually is symptomatic and supportive. The prognosis for a reduced and healed avulsed tubercle is excellent. The patient makes a rapid return to full activities within several months. On the other hand, it usually takes up to 2 years for resolution of the symptoms of an OS lesion, and occasionally disability persists into adulthood.

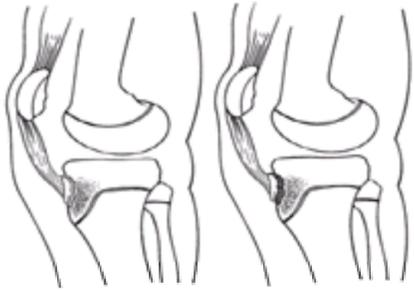
Despite these differences, there is convincing evidence that the OS lesion and acute traction of the tibial tubercle represent a spectrum of avulsion phenomena. The contrasting clinical picture probably is due to the fact that a limited amount of tissue is displaced to a limited degree in the OS lesion (see [Table 23-9](#)).

In 1891, Paget (62) described the clinical syndrome that later became known as OS disease. In 1903, Osgood (132) and Schlatter (136) published separate papers on the subject. Beginning with Osgood's article, three timely papers on the subject have in common the conviction that the etiology is trauma.

Osgood (132) reported 10 patients in whom he observed that function was not completely lost, although recurrent soreness and weakness caused some disability in athletic participation. In a dissected specimen, Osgood observed that after simulating the lesion with localized osteotomy of the tibial tubercle at the insertion of the central ligament, repeated traction on the quadriceps muscle produced upward displacement of the detached fragment of no more than ¼ in. The knee still could be extended with the tibial tubercle detached if the retinaculum was intact. Osgood concluded that the clinical lesion represented a localized rupture of the central insertion of the quadriceps mechanism.

In 1962, Ehrenborg (125) published a comprehensive study of OS disease that included 218 affected knees in 170 patients. No patient was older than 15 years of age at the time of initial examination, and the age of onset of the clinical syndrome coincided with the apophyseal stage of development of the tubercle. He explained the relatively painless onset of the clinical course by noting that the avulsed cartilaginous tissue contained very few pain fibers.

In 1976, Ogden and Southwick (131) reported 53 patients with OS lesions; 41 were in the apophyseal stage of tibial tubercle development at the onset of symptoms. They observed new bone formation anterior to the secondary ossification center and concluded that the weak link that failed under tension was the developing ossification center with overlying hyaline cartilage rather than the cartilage at the base of the tubercle apophysis ([Fig. 23-36](#)).



**FIGURE 23-36.** Development of Osgood-Schlatter lesion. **A:** Avulsion of osteochondral fragment that includes surface cartilage and a portion of the secondary ossification center of the tibial tubercle. **B:** New bone fills in the gap between the avulsed osteochondral fragment and the tibial tubercle.

### Surgical Anatomy

The age range in which each of the two principal types of avulsion injuries occurs is related to the specific stage of development of the tuberosity at that time ( 110). The OS lesion occurs when the tibial tubercle is in the apophyseal stage and the secondary ossification center has appeared. The cartilage overlying the ossification center anteriorly and underlying it posteriorly is fibrocartilaginous and can resist tension forces better than the bone in the secondary ossification center itself, which may give way during contraction of the extensor mechanism. During the epiphyseal stage of tibial tubercle development, the ossification center has become stronger by enlargement and coalescence with the main ossification center of the proximal tibial epiphysis. During this stage of development, violent contraction of the quadriceps mechanism produces the acute fracture–separation through the tubercle physis that was discussed in the preceding section.

### Mechanism of Injury

Usually no specific injury or event can be directly related to the onset of symptoms; however, repeated normal stresses or overuse can produce a limited or localized disruption ( 117,130,131). Bilateral lesions are found in approximately one fourth of patients.

Other anatomic variants may predispose to the development of OS lesions. Lancourt and Cristini ( 114) found patella infera in a small series of adolescents with OS lesions before physeal closure. The low-lying patella, with shortening of the patellar ligament, may explain the increased stress on the tibial tubercle. Jakob et al. ( 128) found patella alta in 185 knees in 125 patients with OS disease. In most, the lesion had healed. These findings also were reported by Aparicio ( 123). They believed that patella alta after OS disease had run its course implied a preexisting quadriceps contracture, especially of the rectus femoris. They repeatedly observed that children with OS disease had a well-developed, even hypertrophic quadriceps muscle with decreased elasticity. Willner ( 139) found genu valgum, pronated feet, and internal rotation of the knee in all of his 78 patients with OS lesions. Turner and Smillie ( 138) found that patients with the OS lesion had a substantial increase in lateral tibial torsion. Also, as in acute avulsion of the tibial tubercle, tight hamstrings may increase the resistance of the knee to extension by quadriceps action, thereby increasing the tension force on the patellar ligament. Patients with cerebral spastic diplegia, characterized by hamstring contracture, are vulnerable to OS lesions in the appropriate age group. Rosenthal and Levine ( 134) showed a relatively high incidence of the OS lesion in patients with cerebral palsy.

### Signs and Symptoms

The patient with an OS lesion usually is between 11 and 15 years of age. Boys are more frequently affected than girls. Approximately half the patients give a history of precipitating trauma. The pain usually has been present intermittently over a period of several months before the patient is seen by the physician. The pain is aggravated by running, jumping, kneeling, squatting, and climbing or descending stairs. Rest or restriction of strenuous activities usually brings relief.

Physical findings are localized swelling and tenderness at the insertion of the patellar ligament. There is no effusion of the knee joint. Pain is reproduced by extension against forced resistance. There may be some quadriceps atrophy.

### Radiographic Findings

Radiographs are not indicated in all patients with OS lesions. The diagnosis is clinical, but plain films may be helpful if other bony entities must be ruled out, if symptoms are prolonged, or if the patient is older. The OS lesion is best seen on a lateral projection taken with the knee in slight internal rotation. A film taken with soft tissue technique may confirm the diagnosis by showing that the edges of the patellar ligament are blurred. Small, flakelike fragments of the secondary ossification center of the tubercle may be displaced slightly anteriorly and superiorly, but these are difficult to distinguish from normal multicentric ossification of the tubercle. Later in the course of the disease, the ossicles may enlarge, presumably because of further ossification of the cartilaginous component of the avulsed fragment. Also, the gap between the avulsed fragments and the tibial tuberosity may fill in with bone. The displaced fragments sometimes fail to unite and remain as separate ossicles detached from the anterior border of the maturing tubercle ( Fig. 23-37). An undue prominence of the tubercle itself may be present in the residual stages. Occasionally it is helpful to compare lateral radiographs of the affected knee with those of the normal side.



**FIGURE 23-37.** Extreme example of Osgood-Schlatter lesion. Lateral radiograph shows enlargement of the tibial tubercle, ununited ossicles, and patella alta.

A prospective study ( 133) of OS lesions done with serial MRI, CT, and bone scans showed that the most striking feature was soft tissue inflammation, not bony avulsion. Tendon thickening was seen in all patients and a distended deep infrapatellar bursa was present in two thirds. An ossicle was seen in only one third of the patients, with both early and late disease. Scintigraphic uptake rarely exceeded normal.

### Treatment

The most important thing a physician can do is to explain the natural history of the disease to the family, because OS disease is related to activity and resolves with rest and maturation. Many are relieved to know that it is not related to any intraarticular derangement or risk of arthritis. Treatment measures most often recommended for OS lesions are symptomatic and supportive ( 79,124,125 and 126). The goal of such treatment is to ease pain and swelling. A temporary restriction of athletics or vigorous activities may be sufficient. In the acute stages of OS disease, activities should be restricted to mild forms of athletic participation, such as walking or swimming. As symptoms improve, moderate activities such as tennis, baseball, swimming, and roller skating can be allowed. Jakob et al. ( 128) proposed stretching exercises for the quadriceps to relieve tension on the tibial tubercle. Stretching exercises also are indicated for the hamstrings, which commonly are tight.

If severe symptoms interfere with normal activities, a knee immobilizer or a cylinder cast may be used for 6 weeks. Turner and Smillie ( [138](#)) suggested multiple drilling in patients with enlargement and irregularity of the tubercle, and they advised excision of ununited fragments or ossicles after maturity.

Some investigators ( [135,139](#)) recommend against treating OS lesions with local injections of steroids because this may impair healing and result in scarring of the overlying skin, with significant cosmetic deformity.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

When we see an adolescent early in the course of symptoms from an OS lesion, we discuss the natural history with the patient and parents. We tell them that symptoms may wax and wane with activities for several years, but this is not a sign of serious internal derangement of the joint. If symptoms are markedly severe, we advise a knee immobilizer or cylinder cast for weight-bearing activities. When there is no acute tenderness, quadriceps-strengthening exercises and hamstring-stretching exercises are begun. Restriction of athletic activities is advised according to the severity of symptoms. The patient often can tell which activities cause recurrent discomfort—usually those that involve jumping, such as basketball or hurdling, and those that require repetitive knee flexion and extension, such as bicycling. Nonsteroidal antiinflammatory medications and icing after sports may relieve symptoms.

Operative treatment is reserved for ununited ossicles with persistent symptoms after the physes have closed. Excision is performed through a short incision by longitudinally splitting the distal fibers of the patellar ligament. The ossicle can be shelled out without extensive dissection. Postoperative casting for 6 weeks may be necessary if more dissection is performed.

### Prognosis

The overall prognosis for OS lesions is excellent. After the symptoms run their course, the patient usually returns to full activity, including participation in athletics. Some prominence of the tibial tubercle may persist, and rarely kneeling may be difficult ( [140](#)).

Symptoms persisting beyond the normal course of the lesion usually are associated with an ununited ossicle ( [127,137](#)). Mital et al. ( [130](#)) found ununited ossicles in 14 of 118 patients with OS lesions. Painful ossicles in adults are successfully treated with simple excision.

An OS lesion occasionally predisposes to an avulsion fracture of the tibial tubercle. Patients and their parents should be informed about this if they choose to continue recreational or sports activities despite persistent discomfort.

## FRACTURES OF THE PATELLA

Patellar fractures are much less common in children than in adults ( [143,152,153](#)). Bostrom ( [145](#)), in his discussion of the incidence of patellar fractures in children, cited three large series: Diebold ( [148](#)), who reported that 16 of 1,200 patients were younger than 16 years of age; Schoenbauer ( [163](#)), who reported 5 of 578 patients to be younger than 16 years of age; and Ray and Hendrix ( [159](#)), who reported 12 of 185 patients younger than 16 years of age. In these three series, less than 2% of patellar fractures occurred in skeletally immature patients.

The low incidence of fractures of the patella in children may be due to the fact that the osseous portion of the patella is less subject to both impact and tensile forces in children than in adults. It is surrounded by a thick layer of cartilage that acts as a cushion against a direct blow ( [146](#)). The relative magnitude of forces generated in the extensor mechanism of a child's knee is no doubt less than an adult's knee because of the smaller muscle mass and shorter moment arm in children. A child's patella also has greater mobility in the coronal plane.

Although the patella may escape fracture from direct injury in children, it is more vulnerable to osteochondral fracture or avulsion of the medial margin associated with lateral patellar dislocation ( [160](#)).

Avulsion fractures of the patella are classified according to location. A superior avulsion involves the superior pole of the patella and appears to be the least common pattern. An inferior avulsion involves the lower pole of the patella and usually is caused by an acute injury ( [142](#)), however, it should be distinguished from a Sinding-Larsen–Johansson lesion ( [164](#)), an incomplete avulsion caused by repetitive cyclic stress. A medial avulsion involves most of the medial margin of the patella and accompanies acute lateral dislocation of the patella. Lateral avulsion fracture usually involves the superolateral margin of the patella and usually is described as a bipartite patella ( [149,150,161](#)) or dorsal defect of the patella.

The greatest difficulty with fractures of the patella in children is diagnosis. Developmental anomalies may be confused with fractures ( [141,155,158](#)), and the size of fracture fragments, especially in sleeve fractures, may be underestimated in a growing child because the patella is partly cartilaginous ( [153](#)). Belman and Neviasser ( [143](#)) pointed out that it is not uncommon for the diagnosis of patellar fracture in a child to be missed or substantially delayed.

### Anatomy

#### Bony Anatomy

The anlage for the patella develops in the ninth embryonic week. It lies deep to the tendon, not embedded in it. At birth, the shape is well defined in cartilage form. Ossification of the cartilaginous anlage begins between 3 and 6 years of age. Often there is more than one central ossicle—there may be as many as six irregular centers. Gradually, the ossicles coalesce, and ossification proceeds peripherally until all but the articular surface is replaced by bone. Until ossification is complete, the edges of the enlarging ossific nucleus may appear irregular on a radiograph ( [18](#)). The pattern of bony development is similar in this respect to the growing epiphysis of the distal femur ( [Fig. 23-38](#)). Ossification of the patella usually is complete by the beginning of the second decade.



**FIGURE 23-38.** Normal knee in a 6-year-old child. Note irregular ossification of the patella and the distal femoral condyle.

Congenital absence ( [144](#)) and congenital hypoplasia ( [163](#)) of the patella are uncommon. Although these variations in development have been reported as isolated findings, they also occur as part of the hereditary symptom complex onychoosteodysplasia or nail-patella syndrome ( [144](#)).

An unossified patella may be bipartite or multipartite. Partition of the patella into almost equal anterior and posterior portions has been reported, often in patients with multiple epiphyseal dysplasia ( [149](#)). The overall incidence of bipartite patella in adolescents is reported to range from 0.2% to 6%. It is more common in boys than in

girls. Although Kohler and Zimmer (155) stated that unilateral bipartite patella is rare, Green (150) found that 57% of his patients had unilateral bipartite patella.

### Vascular Anatomy

Scapinelli (162) studied the blood supply to the human patella in specimens ranging from birth to old age. Because neither he nor Crock (146) noted differences between immature and mature specimens, it is presumed that the pattern of blood supply in children is similar to that in adults. An anastomotic circle surrounds the patella. Contributions to the anastomosis derive from the paired superior and inferior geniculate arteries, as well as from the supreme geniculate artery above and the anterior tibial recurrent artery below. From the anastomotic ring, branches converge centripetally toward the anterior surface of the patella and enter through foramina in the middle third of this surface. Additional blood supply to the patella enters the distal pole behind the patellar ligament. Thus, virtually the entire blood supply to the patella comes from the anterior surface or distal pole, with essentially no penetration of vessels from the medial, proximal, or lateral margins of the patella. Scapinelli (162) noted that these findings correlate with the fact that marginal fractures of the patella rarely unite. Also, injury to blood vessels entering the anterior aspect of the patella may lead to osteonecrosis of the proximal pole.

### Mechanism of Injury

As in adults, fractures of the patella in children result from a direct blow (most common), sudden contraction of the extensor mechanism, or a combination of both. Direct impact may cause a linear or comminuted fracture pattern.

Avulsion of the distal pole of the patella can occur from tensile loading. Houghton and Ackroyd (153) reported three displaced avulsion fractures sustained in this way. Two patients were jumping, and the third was propelling a skateboard. Each injury was witnessed by a parent or teacher, and in none was there direct trauma to the knee.

Preexisting abnormalities in the extensor mechanism may predispose to avulsion fractures (Fig. 23-39). Blount (8) reported a displaced transverse fracture of the patella in a 6-year-old boy who had sustained a previous laceration of the quadriceps mechanism that resulted in limited knee flexion. The acute patellar fracture was produced by sudden forceful flexion against the scarred quadriceps. Rosenthal and Levine (134) found fragmentation of the distal pole of the patella in seven patients with spastic cerebral palsy involving the lower extremities. They believed that the fragmentation represented stress fractures caused by excessive tension in the muscle associated with a flexed-knee gait. Three of the fragmented patellae healed after hamstring lengthening. Kaye and Freiburger (154) found either elongation or fragmentation of the patella in nearly a third of patients with cerebral palsy and fragmentation of the patella in a patient with arthrogryposis.



**FIGURE 23-39.** A 15-year-old male renal transplant recipient felt a “pop” in the knee after a fall down stairs. Lateral radiograph demonstrates avulsion of the patellar tendon from the interior pole of the patella with proximal migration of the patella and quadriceps muscle.

### Classification

Fractures of the patella usually are classified according to location, pattern, and displacement of the fracture. To the anatomic classification should be added the sleeve fracture, which occurs in younger children (Fig. 23-40). A sleeve fracture is an avulsion of a small bony fragment from the distal pole of the patella, together with a sleeve of articular cartilage, periosteum, and retinaculum pulled off the remaining main body of the patella. The extent of injury is not appreciated on initial radiographs. If allowed to heal without reduction and fixation, this gap may fill in completely or partially with bone and fibrous tissue, with enlargement of the patella. This injury has been reported in children 8 to 12 years of age (153).



**FIGURE 23-40.** Sleeve fracture of the patella. **A:** A small segment of the distal pole of the patella is avulsed with a relatively large portion of the articular surface. **B:** On lateral view, the small osseous portion of the displaced fragment is visible, but the cartilaginous portion is not seen. **C:** Healed sleeve fracture after open reduction and internal fixation.

### Signs and Symptoms

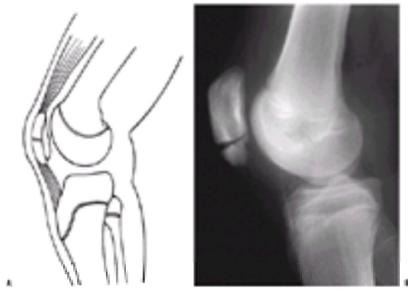
The injured knee is swollen and tender, often with tense hemarthrosis. Full knee extension is difficult, and weight bearing often is impossible. Palpation may reveal a high-riding patella or a palpable defect in the extensor mechanism. If the distal pole is avulsed, voluntary contraction of the quadriceps muscle draws the patella upward, but the patellar ligament remains lax.

With marginal fractures, there may be little more than tenderness and localized swelling over the lateral or medial margin of the patella, and straight-leg raising may be possible (147). An avulsion fragment adjacent to the medial margin of the patella may indicate that an acute lateral dislocation of the patella has occurred. The patella may have reduced spontaneously after the injury. If dislocation has occurred, the apprehension test is positive. The patient either resists passive manipulation by contracting the quadriceps or may even grasp the examiner's hand to prevent further passive displacement.

### Radiographic Findings

Transverse fractures are best seen on lateral radiographs. The amount of displacement varies. In a child, the major fragments may tilt away from one another, with the maximal gap anteriorly and minimal gap posteriorly (143). This may signify that the articular cartilage remains intact, even with a complete fracture through the bony

portion of the patella ([Fig. 23-41](#)). The extent of displacement may not be fully appreciated unless the knee is flexed to 30 degrees when the radiograph is made.



**FIGURE 23-41. A:** Incomplete transverse fracture of the patella. The articular cartilage of the patella remains intact, but the fracture gaps anteriorly. **B:** Lateral radiograph shows incomplete transverse fracture of the patella.

Longitudinally oriented marginal fractures are best seen on axial or skyline views. It is important to differentiate a medial marginal fracture that traverses the entire thickness of the bone from a medial tangential osteochondral fracture. The latter may be difficult to appreciate on an overpenetrated radiograph. An osteochondral fracture may include a substantial amount of cartilage not visible on plain radiographs. Lateral marginal fractures usually are vertically oriented and include the entire thickness of the patella. If a marginal fracture is old, the edges of the fragment are sclerotic. Lateral tilt, lateral subluxation, and lateral overhang of the patella relative to the femoral condyles also are best evaluated on the axial or skyline view.

Small flecks of bone adjacent to the distal pole may be noteworthy ([142](#)). If the fragment is scalelike and closely approximated to the anterior surface of the patella, it may represent an accessory ossification center. A symptomatic, small, visible radiodensity after specific injury or overuse may represent a Sinding-Larsen-Johansson lesion ([164](#)). Small and similarly benign-looking ossicles may signify a sleeve fracture if a good history of acute avulsion injury is given and associated clinical signs are present. Fragmentation or elongation of the distal pole associated with patella alta in a child with cerebral palsy indicates long-standing extensor mechanism stress.

A bipartite patella is best seen on the anteroposterior radiograph, which shows a crescent-shaped radiolucent line in the superolateral quadrant of the patella and rounded margins of the accessory ossicle. If symptoms are confusing, comparison radiographs of the opposite knee are helpful. A similar radiographic appearance of the contralateral patella supports the diagnosis of bipartite patella.

### Treatment

Closed treatment is recommended for nondisplaced fractures. Aspiration of hemarthrosis relieves pain. A well-molded cylinder cast in extension is applied, and progressive weight bearing is permitted. Immobilization may be continued for 4 to 6 weeks.

### Operative Treatment

Operative treatment is reserved for displaced fractures that exhibit 3 mm or more of articular displacement or fractures that disrupt the extensor mechanism. Reduction and internal fixation are preferable to patellectomy. Fixation techniques include a circumferential wire loop, nonabsorbable sutures through longitudinally drilled holes, the AO tension-band technique, or screws or threaded pins. Ogden ([53](#)) believes that to avoid growth disturbance, circumferential wiring through adjacent soft tissue around the patella is preferable to wiring through the patella. Blount ([8](#)) also advised a circular turn of wire about the patella. Others have pointed out that circular wire fixation may threaten the blood supply to the patella. However, Maguire and Canale reported no growth disturbance after the use of cerclage wires. Weber et al. ([83](#)) advised tension-band wiring through a transverse incision, and Rang ([62](#)) advised tension-band wiring for displaced transverse fractures in older children. An experimental study by Weber et al. ([165](#)) supported fixation by either modified tension-band wiring or by the Magnusson wiring method. The former consists of a wire loop passed beyond the tips of two longitudinal fixation wires and over the anterior surface of the patella ([Fig. 23-42](#)). The Magnusson technique consists of a wire loop passed through longitudinal drill holes in the two apposed fragments. All authors agree that meticulous repair of any adjacent retinacular tear is as important as accurate approximation and stable fixation of bony fragments ([9,145,151](#)).



**FIGURE 23-42. A:** Displaced transverse fracture of the patella; note open distal femoral and proximal tibial physes. **B and C:** After reduction and internal fixation with tension-band technique using figure-of-eight wire over parallel longitudinal pins.

Sleeve fractures require careful approximation of the distal pole to prevent persistent deformity and extensor lag. Houghton and Ackroyd ([153](#)) and Wu et al. ([166](#)) advised careful reduction of the distal pole with the attached sleeve of cartilage to the main body of the patella. Modified tension-band wiring around two longitudinal Kirschner wires helps centralize fixation in the small fragment. The extensor retinaculum should be repaired.

For medial marginal fractures, Rorabeck and Bobechko ([160](#)) advised excising the osteochondral fragment and repairing the extensor apparatus in acute dislocation of the patella. Griswold ([151](#)) suggested that marginal fractures of the patella do best with surgery. He advised excision because fibrous union can cause persistent pain. Peterson and Stener ([158](#)) described treatment of concurrent avulsions of the medial and lateral margins of the patella in a 12-year-old boy; a good result was obtained with operative repair.

### Operative Versus Conservative Treatment

Indications for open treatment of fractures of the patella in children are essentially the same as for fractures of the patella in adults. A diastasis of 3 mm or more on radiographs or a step-off on the articular surface of 3 mm or more is an indication for open reduction and internal fixation. It may be more difficult to estimate the amount of gap or the degree of step-off in a younger child because of the partial ossification of the patella. An inability voluntarily to extend the knee fully indicates an associated tear of the retinaculum that should be repaired. An open fracture requires appropriate debridement and irrigation, as well as internal fixation when indicated.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

We advise closed treatment if the fracture is not displaced. A well-molded cylinder or long leg cast is applied to hold the knee in extension. Partial weight bearing is allowed as soon as symptoms permit, usually within the first several days. Straight-leg raising should be possible by 1 week after injury.

For displaced transverse fractures, we perform open reduction with internal fixation using the modified tension-band technique with a wire loop over parallel Kirschner wires. A longitudinal midline incision is preferred to facilitate possible future surgery about the knee. The retinaculum on both the medial and lateral sides is repaired meticulously from outward in toward the margins of the patella.

We advise excising displaced marginal fractures, whether they are on the medial or lateral margin. The exception is a marginal fracture that includes a significant part of the articular surface. This lesion is more properly considered an osteochondral fracture, and a fragment larger than 1 cm should be replaced and fixed by Herbert screws.

### Postoperative and Postfracture Care

Immobilization in a cylinder cast is continued for 4 to 6 weeks after either closed treatment or open reduction and internal fixation. Quadriceps-strengthening exercises are begun as soon as possible. The cast may have to be changed as swelling diminishes. After removal of the cast, quadriceps-strengthening exercises are continued and range-of-motion exercises are begun. It is helpful to begin flexion exercises with the patient prone. Return to vigorous activities or athletics is not permitted until muscle mass and range of motion are equal to those in the opposite extremity.

### Prognosis

The prognosis is good for nondisplaced fractures in which accurate apposition of fragments, with healing, has been achieved. Comminuted, displaced patellar fractures are uncommon in growing children. Those treated without reduction may heal with elongation of the patella and diminution of function of the extensor mechanism. Comminuted fractures in adolescents approaching skeletal maturity are best treated by patellectomy. Symptoms that persist after marginal fractures usually can be treated by simple excision of the fragment.

### Complications

Complications of unreduced displaced fractures include patella alta, extensor lag, and quadriceps atrophy. Houghton and Ackroyd ( [153](#)) reported these findings at short-term follow-up of a displaced sleeve fracture in a 9-year-old girl treated by aspiration and immobilization alone. The fragments had remained separated and the bone had filled the defect. Two years later, moderate quadriceps atrophy and an extensor lag persisted, along with a subchondral defect in the central area of the articular surface. Failure to restore length after injury precludes return to optimal function ( [156,157](#)).

## PART II: INTRAARTICULAR AND LIGAMENOUS INJURIES ABOUT THE KNEE

### OSTEOCHONDRAL FRACTURES

Osteochondral fractures of the knee usually involve the medial or lateral femoral condyle or the patella. The diagnosis often is difficult because even a large osteochondral fragment may contain only a small ossified portion that is visible on plain radiographs. Differential diagnoses include meniscal injury and osteochondritis dissecans ( [169,174,176](#)).

Coleman ( [173](#)) reported five osteochondral fractures of the patella in children and recommended removal of loose bodies, repair of articular defects, and repair of the medial retinaculum to prevent recurrent patellar dislocation. Rosenberg ( [186](#)) reported 15 osteochondral fractures of the lateral femoral condyle caused by flexion-rotation forces to the knee rather than by direct blows. He concluded that osteochondral fractures of the lateral femoral condyle are caused by direct force from the patella, although not all patients had patellar dislocations. He also recommended removal of the loose osteochondral fragments. Rorabeck and Bobeck ( [185](#)) reported 18 children with acute patellar dislocations and osteochondral fractures of the patella or lateral femoral condyle. They recommended repair of the medial retinaculum in addition to removal of the osteochondral fragment.

### Surgical Anatomy

The patella tracks in the intercondylar notch between the medial and lateral femoral condyles during flexion and extension of the knee ( [175,178](#)). With increasing knee flexion, the contact area on the articular surface of the patella moves to the proximal patella. Between 90 and 135 degrees of flexion, the patella glides into the intercondylar notch between the femoral condyles. The two primary areas of contact are the medial patellar facet with the medial femoral condyle and the superolateral quadrant of the lateral patellar facet with the lateral femoral condyle. Soft tissue support for the patellofemoral joint includes the quadriceps muscle, the patellar ligament, and the vastus medialis and lateralis muscles.

Dislocation of the patella may tear the medial retinaculum, but the rest of the quadriceps muscle-patellar ligament complex continues to apply significant compression forces as the patella dislocates laterally. These forces are believed to cause fracture of the medial patellar facet, the lateral femoral condyle articular rim, or both ( [180,184,186](#)) ( [Fig. 23-43](#)). Osteochondral fractures are uncommon with chronic recurrent subluxation or dislocation of the patella because of laxity of the medial retinaculum and lesser compressive forces on the patella and the lateral femoral condyle. In adults, compressive or rotary forces at the knee are dissipated between calcified and uncalcified cartilage without involvement of the subchondral bone. In adolescents, osteochondral fracture extends into the subchondral bone.



**FIGURE 23-43.** Osteochondral fractures associated with dislocation of the right patella. **A:** Medial facet. **B:** Lateral femoral condyle.

### Mechanism of Injury

Kennedy ( [180](#)) described the two mechanisms of osteochondral fractures: exogenous, a direct blow and a shearing force to either the medial or lateral condyle, and endogenous, a flexion-rotation injury of the knee in which contact between the tibia and femoral condyle causes the fracture. The endogenous mechanism is also the cause of osteochondral fracture with patellar dislocation secondary to the shearing force of the patella against the lateral femoral condyle ( [172,182](#)). Ahstrom ( [168](#)) reported 18 osteochondral fractures; 14 occurred during sports-related activities. Most patients give a history of a twisting injury consistent with acute patellar dislocation, but a few report a direct blow to the lateral or medial femoral condyle, accounting for a shear injury. Nietosvaara et al. ( [183](#)) reported that of 69 acute

patellar dislocations in children and adolescents, 62 (90%) occurred in falls; 39% also had osteochondral fractures.

### Classification

The classification of osteochondral fractures of the knee is based on the site, the type, and the mechanism of injury. The classification outlined in [Table 23-11](#) is based on the descriptions of osteochondral fractures by Kennedy ([180](#)) and Smillie ([188](#)).

Site	Mechanism
Medial femoral condyle	Direct blow (fall) Compression and rotation (tibiofemoral)
Lateral condyle	Direct blow (kick) Compression and rotation (tibiofemoral)
Patella (medial margin)	Acute patellar dislocation Acute patellar dislocation

**TABLE 23-11. MECHANISM OF OSTEOCHONDRAL FRACTURES**

### Signs and Symptoms

Most children and adolescents with osteochondral fractures of the knee give a history of a flexion–rotation injury consistent with patellar dislocation. Rarely, patients with multiple trauma have an osteochondral fracture from a severe direct blow or open injury of the knee. Severe pain, swelling, and difficulty in weight bearing are immediate after osteochondral fracture.

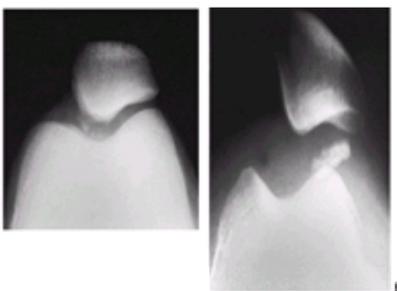
Physical findings may include knee effusion and tenderness over the medial or lateral femoral condyle or the medial patella. The knee usually is held in 15 to 20 degrees of flexion, and movement in any direction is resisted. Attempts at flexion or extension of the knee and manipulation of the patella elicit pain. Sterile aspiration of the knee using local anesthesia usually obtains a bloody aspirate, occasionally with fat globules suggestive of an intraarticular fracture. Late physical findings are identical to those of a loose body: intermittent locking and catching of the knee joint or a palpable fragment beneath the skin.

### Radiographic Findings

Because the osteochondral fragment may be difficult to see on plain radiographs, anteroposterior and lateral views must be carefully assessed for even the smallest ossified fragment ([Fig. 23-44](#)). A tunnel view may help locate a fragment in the region of the intercondylar notch. Osteochondral fragments also may be located at the extreme periphery of the joint in the suprapatellar pouch or beneath the MCL or LCL ([Fig. 23-45](#)). The tibial tuberosity should not be confused with an osteochondral fragment, as noted by Ogden ([184](#)). Occasionally, a large osteochondral fragment with a small ossified portion can be seen more clearly with arthrography, CT arthrography, or MRI.



**FIGURE 23-44.** Osteochondral fracture of lateral femoral condyle after patellar dislocation. **A:** Fragment seen in lateral joint space. **B:** Lateral view.



**FIGURE 23-45.** Osteochondral fractures associated with dislocation of the patella. **A:** Medial facet of patella. **B:** Lateral femoral condyle.

### Methods of Treatment

Most authors recommend operative treatment of acute osteochondral fractures of the knee, either excision of the fragment or fixation to its anatomic location. Kennedy ([180](#)) and Smillie ([188](#)) recommend replacement in acute injuries if the fragment is large, easily accessible, and in a weight-bearing area of the knee. If arthrotomy is delayed for 10 days or more, fixation is contraindicated because the articular surface defect has already begun to fill in with fibrocartilage. Other authors recommend excision of small fragments from the medial or lateral condyle or the patella ([167,177,179,187](#)). Excision or fixation of the fragment may be done with arthroscopy or arthrotomy, and fixation devices vary from small, smooth or threaded Steinmann pins to countersunk screws ([171,190](#)). Lewis and Foster ([181](#)) reported good results in eight osteochondral fractures after fixation with Herbert bone screws. They cite as advantages of this technique:

1. Little cartilage damage is done during insertion.
2. Because the screw is entirely buried, it does not interfere with soft tissues.
3. Fixation is rigid enough to allow the use of continuous passive motion.
4. Reoperation for screw removal is unnecessary.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

Treatment of the osteochondral fragment depends on five factors: size, site, stability, surface, and subchondral status of the lesion ( [189,191](#)). If the fragment is from a non-weight-bearing area, arthroscopic excision is recommended with debridement of the fragment's crater to stable edges and perforation of the underlying subchondral bone to encourage cell ingrowth. With unstable or loose lesions larger than 3 mm in weight-bearing contact areas, fragment salvage is attempted if the fragment can be reduced, its articular surface is pristine and not crenellated, and its subchondral segment is adequate for fixation, usually 3 mm in depth. Bioabsorbable fixation devices can be used but carry a risk of sterile synovitis with recurrent effusions. Most osteochondral fractures associated with patellar dislocation, either from the patella or lateral femoral condyle, can be removed arthroscopically. Rarely, after a severe direct blow, a large osteochondral fracture of the femoral condyle requires open reduction and internal fixation.

### Operative Treatment

The first step in treatment is to obtain adequate radiographic studies to assess the size and origin of the fragment accurately. If the fragment is small (<2 cm) and from a non-weight-bearing area, we prefer arthroscopy for inspection of the joint and removal of the fragment. Arthroscopic examination also may be done if imaging studies do not clearly reveal the size and origin of the fragment. If the fragment is larger than 2 cm and from a weight-bearing surface, either medial or lateral arthrotomy may be necessary, as indicated by the location of the fragment, to return the fragment to its bed and fix it with small, threaded Steinmann pins placed in a retrograde or transverse direction through the femoral condyle into the subcutaneous tissue medially or laterally. The pins should not be left protruding into the joint, but should be buried beneath the articular cartilage and cut off in the subcutaneous tissue for easy removal after fracture healing. For very large fragments, especially in adolescents nearing skeletal maturity, we use countersunk minifragment screws or Herbert screws. These screws may require later removal even if buried beneath articular cartilage. Regardless of the fixation device chosen, care should be taken to avoid penetration of the distal femoral physis, especially in younger children.

### Postoperative Care

After fixation of a large osteochondral fragment with pins or screws, a long leg, bent-knee cast is applied with the knee flexed to 30 degrees. Non-weight-bearing ambulation is allowed on crutches, and quadriceps muscle-strengthening exercises are begun. The cast is removed at 6 weeks after surgery, quadriceps exercises are continued, and weight bearing gradually progresses. Full weight bearing is allowed after swelling has subsided and radiographs show healing of the fracture fragment. Return to athletic activities is permitted when knee range of motion has recovered and quadriceps strength is equal to that of the uninvolved extremity.

After arthroscopic excision of a small fragment, a knee immobilizer splint is fitted, quadriceps-strengthening exercises are begun, and weight bearing is allowed as tolerated. The immobilizer is discontinued 2 weeks after arthroscopy.

### Prognosis

Osteochondral fractures with small fragments not involving the weight-bearing portion of the joint usually have a good prognosis. The prognosis is more uncertain, however, if the fracture fragment involves a large portion of the weight-bearing surface. When acute patellar dislocation requires arthrotomy for fixation or removal of a large osteochondral fragment, Rorabeck and Bobechko ([185](#)) and Bassett ([170](#)) recommend operative repair of the medial retinaculum. Ahstrom ([168](#)) noted poor results after osteochondral fracture associated with recurrent patellar dislocation, but believed these were related more to the chronic dislocation or subluxation than to the articular injury. Even after surgical fixation, large osteochondral fractures with incongruous articular surfaces may cause loss of motion and late osteoarthritic changes ([Fig. 23-46](#)).



**FIGURE 23-46.** Comminuted osteochondral fracture of the patella after acute lateral dislocation of the patella at the time of a karate kick. (Courtesy of John Roberts, MD, Bangor, Maine.)

### Complications

Complications of surgical treatment, especially with large fragments, include adhesions in the suprapatellar pouch, quadriceps insufficiency, and malalignment of the quadriceps-patellar ligament complex. Threaded Steinmann pins or countersunk screws that protrude into the joint may cause synovitis and loss of knee joint motion.

## FRACTURE OF THE TIBIAL SPINE (INTERCONDYLAR EMINENCE)

Avulsion fracture of the tibial spine is a relatively rare injury in children: Skak et al. ( [219](#)) reported that it occurred in 3 per 100,000 children each year. The most common causes of these fractures are bicycle accidents and athletic activities ( [210,211](#)). A stable, painless knee can be obtained in many patients with closed treatment ([190](#)), but displaced fractures irreducible by closed means require open reduction.

Garcia and Neer ([199](#)) reported 42 fractures of the tibial spine in patients ranging in age from 7 to 60 years, 6 of whom had positive anterior drawer signs indicating associated collateral ligament injuries. They reported successful closed management in half their patients. Meyers and McKeever ( [209](#)), however, recommended arthrotomy and open reduction for all displaced fractures, followed by cast immobilization with the knee in 20 degrees of flexion rather than hyperextension, believing that hyperextension aggravated the injury in one of their patients. Gronkvist et al. ( [201](#)) reported late instability in 16 of 32 children with tibial spine fractures, and recommended surgery for all displaced tibial spine fractures, especially in children older than 10 years because "the older the patient the more the demand on the anterior cruciate ligament-tibial spine complex." Because of elongation of the anterior cruciate ligament (ACL), which contributes to instability, they recommended countersinking the tibial spine-proximal tibial epiphyseal fragment during open reduction. Baxter and Wiley ( [193](#)) noted mild to moderate knee laxity at follow-up in 45 patients, even after anatomic reduction of the tibial spine.

McLennan ([208](#)) reported 10 patients with type III intercondylar eminence fractures treated with closed reduction and with arthroscopic reduction with or without internal fixation. At second-look arthroscopy 6 years after the initial injury, those treated with closed reduction had more knee laxity than those treated arthroscopically. In a long-term (average 16 years) follow-up study of 61 children with anterior tibial spine fractures, Janarv et al. ( [203](#)) found pathologic knee laxity from 3 to 9 mm in 18 (38%) of the 47 patients clinically tested.

Falstie-Jensen and Søndergard-Petersen ( [197](#)) reported four patients with moderate or complete displacement of the fracture and incarceration of the medial meniscus beneath the tibial spine. They recommended arthroscopic release of the incarcerated meniscus and open reduction and fixation of the tibial spine fracture if necessary ([206,207](#)). Hayes and Masear ([202](#)) reported one patient with an anterior tibial spine fracture associated with injury of the MCL. For patients with a positive anterior drawer sign or other evidence of ligamentous instability, they recommended stress radiographs and, if necessary, examination under anesthesia, followed by repair of any injury to the ACL or collateral ligament. Robinson and Driscoll ( [217](#)) reported one patient with avulsions of the femoral and tibial insertions of the ACL.

and associated MCL injury.

The posterior intercondylar eminence is even more rarely injured in children than the anterior eminence ( [222](#)) ([Fig. 23-47](#)). Roberts and Lovell ([216](#)) found the anterior tibial spine to be injured 10 times more frequently than the posterior tibial spine. Goodrich and Ballard ( [200](#)) and Ross and Chesterman ([218](#)) reported isolated avulsion of the posterior tibial spine at the insertion of the PCL.



**FIGURE 23-47.** Posterior tibial spine fracture. **A:** Lateral radiograph of the left knee of a 10-year-old girl with an avulsion of the posterior tibial spine. **B:** Magnetic resonance image shows that the posterior cruciate ligament (*solid arrow*) is attached to the posterior tibial spine fragment (*open arrow*). (Courtesy of Claiborne A. Christian, MD, Memphis, Tennessee.)

### Surgical Anatomy

The ACL attaches distally to the anterior tibial spine and the anterior horn of the medial meniscus, with separate slips anterior and lateral as well. The ligament rests against the lateral roof of the intercondylar notch of the femur. The intercondylar eminence is that part of the proximal surface of the tibia lying between the anterior poles of the menisci forward to the anterior tibial spine. It is triangular, with its base at the anterior border of the proximal tibia. In the immature skeleton, the proximal surface of the eminence is covered entirely with cartilage. Matz et al. ( [266](#)) found the medial meniscus to be trapped under the fragment in 8 of 10 type III lesions.

In a cadaver study by Roberts and Lovell ([215,216](#)), fracture of the anterior intercondylar eminence was simulated by oblique osteotomy beneath the eminence and traction on the ACL. In each specimen, the displaced fragment could be reduced into its bed by extension of the knee. In adults, the same stress might cause an isolated tear of the ACL, but in children the incompletely ossified tibial spine is weaker to tensile stress than the ligament, so failure occurs through the cancellous bone beneath the subchondral bone of the tibial spine.

### Mechanism of Injury

A fall from a bicycle was the most common mechanism of injury in the series of Meyers and McKeever ([210](#)) and Roberts and Lovell ([216](#)), but anterior tibial spine fractures also have been reported in children participating in athletic activities or with multiple trauma ( [213,214](#)). The injury probably occurs as the tibia is rotated relative to the femur and forced into hyperextension.

### Classification

Meyers and McKeever ([210](#)) proposed a classification of tibial spine fractures based on the degree of displacement ( [Fig. 23-48](#)):



**FIGURE 23-48.** Classification of tibial spine fractures. **A:** Type I—minimal displacement. **B:** Type II—hinged posteriorly. **C:** Type III—complete separation. (Modified from Meyers MH, McKeever FM. Fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg Am* 1959;41:209–222; with permission.)

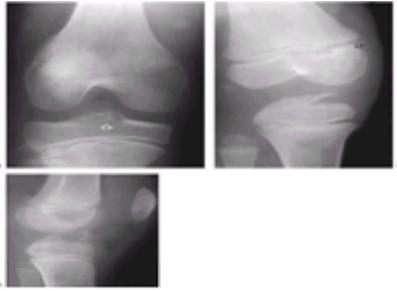
1. Type I—minimal displacement of the fragment from the rest of the proximal tibial epiphysis
2. Type II—displacement of the anterior third to half of the avulsed fragment, which is lifted upward but remains hinged on its posterior border in contact with the proximal tibial epiphysis
3. Type III—complete separation of the avulsed fragment from the proximal tibial epiphysis, with upward displacement and rotation

### Signs and Symptoms

Physical findings in acute injury include pain, effusion from associated hemarthrosis, and reluctance to bear weight. Extremes of motion are resisted because of increasing pain. Gentle stress testing should be performed to detect any tear of the MCL or LCL or physeal fracture of the distal femur or proximal tibia. Patients with late malunion of a displaced tibial spine fracture may lack full extension because of a mechanical bony block.

### Radiographic Findings

Adequate anteroposterior and lateral radiographs are essential to evaluate the degree of displacement of the anterior tibial spine, although the fracture is best seen on the lateral view. Both views should be carefully scrutinized: the avulsed fragment may be mostly nonossified cartilage with only a small, thin ossified portion visible on the lateral view. Obtaining the anteroposterior radiograph more parallel to the normal posterior tilt of the proximal tibial articular surface may better reveal the fragment ([Fig. 23-49](#)).



**FIGURE 23-49.** Stages of displacement of tibial spine fractures. **A:** Type I fracture, minimal displacement (*open arrow*). **B:** Type II fracture, posterior hinge intact. **C:** Type III fracture, complete displacement and proximal migration.

If the fragment appears narrow and confined to the central portion of the intercondylar eminence, closed reduction may be successful. If the fragment is wider and extends medially or laterally beneath the articular surface of the tibial epiphysis, the anterior horn of a meniscus may be interposed beneath the fracture fragment.

Stress views should be obtained if collateral ligament injury or physeal fracture is suggested by physical examination. These can be done with the patient sedated or, if necessary, under general anesthesia. Abnormal widening of the joint space confirms an associated tear of the collateral ligament or may indicate occult fracture of the distal femur or proximal tibial physis.

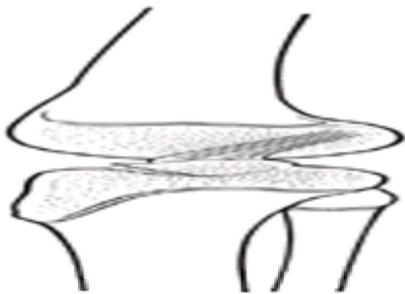
A malunited displaced fragment appears on the lateral view as a large intraarticular extension into the intercondylar notch. A lateral view taken with the knee in hyperextension may reveal impingement of the proximal portion of the malunited fragment on the articular surface of the distal femur.

### Methods of Treatment

For nondisplaced or minimally displaced (types I and II) fractures of the anterior tibial spine, most investigators recommend closed treatment. The position of immobilization, however, is controversial; some recommend hyperextension, others 10 to 20 degrees of flexion. For type III displaced fractures, most authorities recommend open reduction and internal fixation ([194,209,222,223,226](#)). Bakalim and Wilpulla ([192](#)) reported successful closed reduction in 10 patients, none of whom had evidence of laxity of the ACL at follow-up, although some complained of anteroposterior instability. Smillie ([186](#)) suggested that closed reduction by hyperextension can be accomplished only with a large fragment. Meyers and McKeever ([209](#)) recommended cast immobilization with the knee in 20 degrees of flexion for all type I and II fractures and open reduction or arthroscopic treatment of all type III fractures.

### AUTHOR'S PREFERRED METHOD OF TREATMENT

We prefer closed treatment of most type I and II fractures. If hemarthrosis causes severe pain, aspiration can be done under sterile conditions, after which the child is placed in a long leg cast with the knee in a position of neutral to 10 degrees of flexion ([Fig. 23-50](#)). Anteroposterior and lateral radiographs are obtained to confirm satisfactory reduction of the tibial spine fragments and are repeated in 1 to 2 weeks to ensure that the fragment has not become displaced. The cast is removed in 6 weeks and physical therapy is continued to strengthen the quadriceps and hamstring muscles and to increase range of motion.

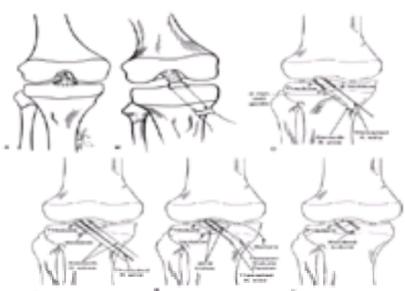


**FIGURE 23-50.** Reduction of type II tibial fracture with knee in 10 to 20 degrees of flexion.

### Operative Treatment

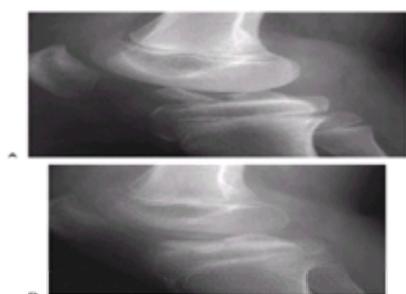
Although closed reduction of type III fractures can be attempted by placing the knee in neutral to slight flexion, we usually have not found this successful. We prefer open reduction through an anteromedial arthrotomy or arthroscopic treatment. This approach allows inspection of the anterior horns of both menisci and distraction of the meniscus if its interposition beneath the anterior tibial spine fragment prevents anatomic reduction. The fragment is reduced with the knee in neutral or 10 to 20 degrees of flexion.

If open surgery is used, numerous fixation techniques are available ([206,207](#) and [208](#)) ([Fig. 23-51](#)). In a young child in whom the fragment often is large and mostly cartilaginous, the fragment can be reduced in the tibial epiphyseal crater and secured with peripheral absorbable sutures. In an adolescent with a small fragment, a nonabsorbable suture can be woven through the ACL and out the distal end of the tibial spine fragment. From an anteromedial starting point on the tibial metaphysis, drill holes are placed obliquely into the crater in the tibial epiphysis, carefully avoiding the remainder of the tibial physis. The fragment is then reduced in its bed, and sutures are brought out through the drill holes anteromedially and tied securely ([Fig. 23-52](#)). In skeletally mature patients, in whom the integrity of the tibial physis is not a concern, the anterior tibial spine can be fixed with a minifragment screw directed obliquely into the proximal tibial metaphysis ([Fig. 23-53](#)).



**FIGURE 23-51.** Techniques of fixation of tibial spine fractures. **A:** Peripheral sutures for large cartilaginous fragment in young child. **B:** Transepiphyseal pull-out suture for smaller fragment in an adolescent. **C through F:** An arthroscopic technique using suture to pull down on the tibial spine fragment through two drill holes, tied anteriorly. **C:** A threaded Kirschner wire passed into the reduced fracture. **D:** Nonthreaded Kirschner wire passed parallel to the threaded Kirschner wire. **E:** A Hewson passed through the medial drill hole retrieving a polyglactin (Vicryl) suture passed through the medial porthole. **F:** The suture knotted down with the fracture

reduced. (C through F redrawn from Mah JY, Otsuka NY, McLean J. an arthroscopic technique for the reduction and fixation of tibial-eminence fractures. *J Pediatr Orthop* 1996;16:119–121; with permission.)



**FIGURE 23-52.** A: Displaced type III tibial spine fracture. B: Three months after open reduction and internal fixation with pull-out suture technique.



**FIGURE 23-53.** Type III displaced tibial spine fracture in 17-year-old boy fixed with small fragment screw.

#### Open Reduction and Internal Fixation of Fractures of the Tibial Spine

The knee is exposed through the distal portion of an anteromedial parapatellar incision ( 196). The capsule is opened medially to expose the fracture fragments and the defect in the proximal tibia. After the medial meniscus is examined, retraction is used to allow examination of the anterior horn of the lateral meniscus to ensure the menisci are not impeding the reduction. The knee is placed in extension, and the fragment is reduced after any clots and cancellous bone have been removed from the defect. Two holes are drilled from distal to proximal through the tibial metaphysis and epiphysis, with care taken to drill the holes into the central area of the crater in the tibial epiphysis. A 1-0 nonabsorbable suture is passed through the distalmost portion of the ACL just proximal to the fracture fragment. With suture carriers, the ends of the suture are passed through the drill holes and tied on themselves after the reduction is satisfactory. The knee is flexed and extended to ensure the reduction is stable. The wound is irrigated and closed.

After reduction and fixation, a long leg cast is applied with the knee in neutral or slight (10 to 20 degrees) flexion. The cast is removed 6 weeks after surgery, and strengthening exercises of the quadriceps and hamstrings and active range-of-motion exercises are begun. Weight bearing progresses from touch-down to full weight bearing.

#### Arthroscopic Technique

After removal of any obstructions to reduction, the fracture is reduced by probe manipulation. Reduction is maintained by use of an ACL reconstruction guide. For fixation of noncomminuted fragments larger than 1 cm, a 20-mm cannulated screw is inserted through a proximal medial parapatellar portal ( 194,223). The screw should be placed so the proximal tibial physis is not violated. The screw head should be within the ACL substance to prevent impingement in extension. Countersinking the screw in an attempt to restore isometry is counterproductive because the ligament already is attenuated from the initial injury.

#### Prognosis and Complications

Prognosis of tibial spine injuries in children is good, with most patients regaining an excellent range of motion. Wiley and Baxter ( 224) found a measurable loss of extension in 45 patients with tibial spine fractures, 60% of whom had losses of more than 10 degrees; no patient, however, complained of any subjective feelings of knee instability. Willis et al. ( 225) found clinical signs of anterior instability in 64% of 50 patients at an average follow-up of 4 years and objective evidence of laxity in 74%, but again no patient complained of instability.

Poor results may occur after type III fractures of the tibial spine associated with unrecognized injuries of the collateral ligaments or complications from associated physeal fracture (211,220,221). Malunion of type III fractures may cause mechanical impingement of the knee during full extension ( 206,207 and 208) (Fig. 23-54). For symptomatic adolescents, this can be corrected by excision of the malunited fragment and anatomic reinsertion of the ACL. Lombardo ( 205) reported avulsion of a fibrous union of the tibial spine 3 years after original injury.



**FIGURE 23-54.** Lateral radiograph of a malunited displaced fracture of the intercondylar eminence of the tibia with an extension block.

Mylle et al. (212) reported anterior epiphysiodesis with hyperextension of the knee 2 years after transepiphyseal screw fixation of an ACL avulsion in an 11-year-old girl. They recommended early removal of transepiphyseal metalwork or the use of arthroscopically inserted intraepiphyseal fixation to avoid this complication. This technique probably should be reserved for skeletally mature patients or the screw should be angled to avoid the physis. Mah et al. ( 206,207) found that both of their

surgically treated type III patients had negative Lachman, anterior drawer, and pivot-shift tests at follow-up, with a mean anterior translation of 2.5 mm measured arthrometrically. They suggested that the anatomic reconstruction prevented laxity, findings not shown by other reports that acknowledged that the laxity after injury or surgery was due to the interstitial elongation of the ACL accompanying the avulsion fracture.

## LIGAMENT INJURIES

The increasing number of reports of acute disruption of the knee ligaments in children and adolescents ( [218,229,234,241,242,257,262,263,266,268,277,278,280](#)) refutes the long-held belief that complete disruption of knee ligaments can occur only after the physes have stopped growing ( [188](#)). This increased frequency of knee ligament injuries in children probably is related to the more frequent participation of children in vigorous sports, the increasing incidence of multiple trauma, and a heightened awareness among physicians concerning the frequent association of ligamentous injury with fractures about the knee.

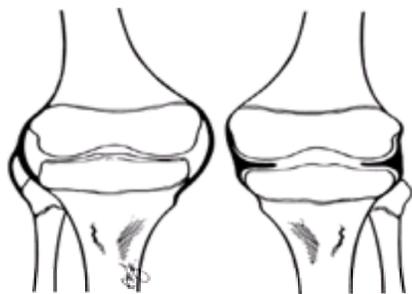
Fractures of the epiphyses or physes about the knee are more common than ligamentous injuries alone. Isolated knee ligament injury is rare in children younger than 14 years of age because the ligaments are stronger than the physes ( [237,243,261,267,288](#)). The inherent ligamentous laxity in children also may offer some protection against ligament injury, but this decreases as the adolescent approaches skeletal maturity. Bertin and Goble ( [232](#)), after reviewing 29 fractures, concluded that physeal fractures about the knee are associated with a higher incidence of ligamentous injury.

Before the 1990s, reports of ligamentous injuries in children were isolated case reports, and most recommendations were for conservative treatment. More recent reports have indicated an increased awareness of ligament injury in association with physeal fractures ( [238](#)), as well as isolated ligament injuries, and a more aggressive approach, especially in adolescents approaching skeletal maturity ( [231,242,244,246,272,279,287](#)).

Stanitski et al. ( [285](#)) reported 70 children and adolescents with acute traumatic knee hemarthroses; arthroscopic examination revealed ACL injuries in 47% of those 7 to 12 years of age and in 65% of those 13 to 18 years of age. They determined that boys 16 to 18 years of age engaged in organized sports and girls 13 to 15 years of age engaged in unorganized sports had the highest risk for complete ACL tears; 60% of these patients had isolated ACL tears.

### Surgical Anatomy

The MCL and LCL of the knee originate from the distal femoral epiphysis and insert into the proximal tibial and fibular epiphyses, except for the superficial portion of the MCL, which inserts into the proximal tibial metaphysis distal to the physis ( [Fig. 23-55](#)). In children, these ligaments are stronger than the physes, and significant tensile stresses usually produce epiphyseal or physeal fractures rather than ligamentous injury. The ACL originates from the posterolateral intercondylar notch and inserts into the tibia slightly anterior to the intercondylar eminence. The PCL originates from the posteromedial aspect of the intercondylar notch and attaches on the posterior aspect of the proximal tibial epiphysis. The ACL in children has collagen fibers continuous with the perichondrium of the tibial epiphyseal cartilage; in adults, the ligament inserts directly into the proximal tibia by way of Sharpey's fibers. This anatomic difference probably accounts for the fact that fracture of the anterior tibial spine occurs more frequently in children than does ACL injury.



**FIGURE 23-55.** Anatomy of medial and collateral ligaments of the knee in the adolescent. **A:** Superficial origins and insertions. **B:** Capsular and meniscal attachments.

### Mechanism of Injury

The mechanism of ligamentous injury varies with the child's age. In younger children, ligamentous injury typically is associated with significant polytrauma. Clanton et al. ( [237](#)) reported that five of nine children with acute knee ligament injuries were struck by automobiles. In contrast, adolescents are more likely to sustain ligamentous injury during contact sports or sports that require "cutting" maneuvers while running ( [219,268](#)). As exact a description as possible of the mechanism of injury should be obtained, including the position of the knee at the time of injury, the weight-supporting status of the injured knee, whether the force applied was direct or indirect (generated by the patient's own momentum), and the position of the extremity after injury. Older adolescents may describe the knee as buckling or "jumping out of place" and can usually relate the location and severity of their pain as well as the time between injury and onset of pain and swelling. Rapid intraarticular effusion within 2 hours of injury suggests hemarthrosis, usually from injury to the ACL.

Palmer ( [274](#)) described four mechanisms capable of producing disruption of the ligamentous structures about the knee: abduction, flexion, and internal rotation of the femur on the tibia; adduction, flexion, and external rotation of the femur on the tibia; hyperextension; and anterior–posterior displacement. The most common mechanism in adolescents is abduction, flexion, and internal rotation of the femur on the tibia occurring during athletic competition when the weight-bearing extremity is struck from the lateral side. The classic abduction, flexion, and internal rotation injury in the adolescent may cause the "unhappy triad" of O'Donoghue: tears of the MCL and ACL and injury to the medial meniscus.

Isolated injury of the LCL is rare in children, but a direct blow to the medial aspect of the knee may tear the LCL, usually with avulsion from the fibula or a physeal injury through the distal femur ( [258](#)). Isolated injuries of the ACL and PCL have been reported ( [256,267,278](#)). Disruption of the ACL with minimal injury to other supporting structures may be caused by hyperextension, marked internal rotation of the tibia on the femur, and pure deceleration. In contrast, isolated injury of the PCL most often is caused by a direct blow to the front of the tibia with the knee in flexion.

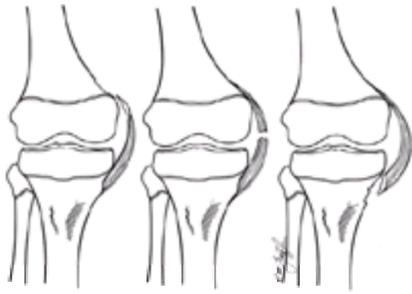
### Classification

Classification of knee ligament injuries is based on three considerations: the severity of the injury, the specific anatomic location of the injury, and the direction of the subsequent instability caused by an isolated ligament injury or combination of ligament injuries.

A first-degree ligament sprain is a tear of a minimal number of fibers of the ligament with localized tenderness but no instability. A second-degree sprain is disruption of more ligamentous fibers, causing loss of function and more joint reaction but no significant instability. A third-degree sprain is complete disruption of the ligament, resulting in instability. Although difficult to assess clinically, the degree of sprain also is determined during stress testing by the amount of separation of the joint surfaces: first-degree sprain, 5 mm or less; second-degree sprain, 5 to 10 mm; and third-degree sprain, more than 10 mm.

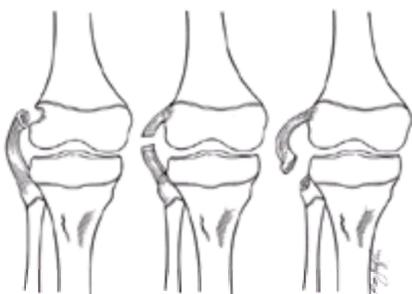
The anatomic classification of ligament injuries describes the exact location of the disruption ( [250](#)):

- A. MCL insufficiency ( [Fig. 23-56](#))



**FIGURE 23-56.** Medial collateral ligament injury. **A:** Femoral origin. **B:** Middle portion. **C:** Tibial insertion.

1. Superficial
  - a. Femoral origin
  - b. Middle portion
  - c. Tibial insertion
2. Deep
  - a. Menisiofemoral
  - b. Middle portion
  - c. Menisiofibular
- B. ACL insufficiency
  1. Femoral origin
  2. Interstitial
  3. Tibial insertion
    - a. Without avulsion of the intercondylar eminence
    - b. With avulsion of the intercondylar eminence
- C. LCL insufficiency ([Fig. 23-57](#))



**FIGURE 23-57.** Lateral collateral ligament injury. **A:** Femoral origin. **B:** Middle portion. **C:** Fibular insertion.

1. Femoral origin
2. Middle portion
3. Fibular insertion
- D. PCL insufficiency
  1. Femoral origin
  2. Interstitial
  3. Tibial insertion
    - a. Without avulsion of the intercondylar eminence
    - b. With avulsion of the intercondylar eminence

Finally, the instability of the knee joint caused by the ligament disruption is classified as follows ([255,281](#)):

- A. One-plane instability (simple or straight)
  1. One-plane medial
  2. One-plane lateral
  3. One-plane posterior
  4. One-plane anterior
- B. Rotary instability
  1. Anteromedial
  2. Anterolateral
    - a. In flexion
    - b. Approaching extension
  3. Posterolateral
  4. Posteromedial
  5. Combined instability
    1. Anterolateral–posterolateral
    2. Anterolateral–anteromedial
    3. Anteromedial–posteromedial

Using this classification, one-plane medial instability, for example, means that the tibia moves abnormally away from the femur on the medial side. In anteromedial rotary instability, the tibia rotates anteriorly and externally and moves away from the femur on the medial side. The classification becomes more complex as more significant ligamentous injuries involving more anatomic locations are included, but determining the type of instability resulting from the injury is helpful in planning treatment.

### Signs and Symptoms

Both lower extremities are examined for comparison. Large areas of ecchymosis and extensive effusion are easily identified, but smaller areas may require careful palpation. In general, acute hemarthrosis suggests rupture of a cruciate ligament, an osteochondral fracture, a peripheral tear in the vascular portion of a meniscus, or a tear in the deep portion of the joint capsule ([239,240,245](#)). The absence of hemarthrosis is not, however, an indication of a less severe ligament injury, because with complete disruption the blood in the knee joint may escape into the soft tissues rather than distend the joint. The range of motion of the injured knee, especially extension, is compared with that of the uninjured knee. If significant effusion prevents full extension, sterile aspiration can be performed. If complete knee extension is impossible after aspiration, the diagnosis of an entrapped meniscus should be considered.

Palpation of the collateral ligaments and their bony origins and insertions should locate tenderness at the site of the ligament injury. A defect in the collateral ligaments often can be felt if the MCL is avulsed from its insertion on the tibia or if the LCL is avulsed from the fibular head. If the neurovascular status is normal, stability should

be evaluated by stress testing.

Stress testing for ligament injury in children (248,249), with or without concomitant fracture, is very subjective, and its usefulness depends on the knowledge and experience of the examiner. Stress testing may be done immediately after injury in cooperative adolescents who do not have other significant injuries, but sedation or general anesthesia may be required for accurate diagnosis. In nonemergent situations, beginning the examination by testing the uninjured knee often calms patients and makes them more cooperative; it also establishes a baseline for assessing the ligamentous stability of the injured knee. In the standard stress tests of specific collateral and cruciate ligaments of the knee described in the following sections, the uninjured knee should be examined first.

#### **Valgus Stress Test of Medial Collateral Ligament**

The valgus or abduction stress test is done with the child supine on the examining table and the knee to be examined on the side of the table closest to the examiner. The extremity is abducted off the side of the table and the knee is flexed approximately 20 degrees (Fig. 23-58). With one hand about the lateral aspect of the knee and the other supporting the ankle, the examiner applies gentle abduction or valgus stress to the knee while the hand at the ankle gently externally rotates the leg. Stability of the knee is noted at 20 degrees of flexion and again at neutral.



**FIGURE 23-58.** Valgus stress test of medial collateral ligament. Extremity is abducted off table, knee is flexed to 20 degrees, and valgus stress is applied. **A:** Frontal view. **B:** Lateral view.

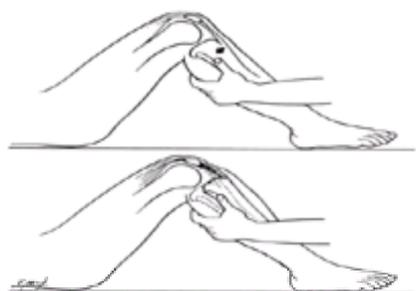
#### **Varus or Adduction Stress Test of Lateral Collateral Ligament**

The varus or adduction stress test is done in a manner similar to the valgus stress test. It should be done with the knee in full extension and in 20 degrees of flexion. The LCL may be palpable as a taut structure on the lateral aspect of the knee.

The stability of the collateral ligaments may be different when tested in extension and in flexion. If a collateral ligament is torn but the cruciate ligaments and posterior capsule are intact, little instability can be detected with the knee extended. Flexion of the knee relaxes the capsule and more instability is evident with the same degree of ligamentous injury. Significant instability with varus or valgus stress testing with the knee in full extension usually indicates a cruciate as well as a collateral ligament disruption.

#### **Stress Testing of Anterior Cruciate Ligament**

The anterior drawer test, as described by Slocum, is the classic maneuver for testing the stability of the ACL (Fig. 23-59). The Lachman and pivot-shift tests, however, are considered more sensitive for evaluating ACL injury when the examination can be done in a relaxed, cooperative adolescent (Fig. 23-60). To perform the Lachman test, the examiner firmly stabilizes the femur with one hand while using the other hand to grip the proximal tibia, with the thumb placed on the anteromedial joint margin. An anteriorly directed lifting force applied by the palm and fingers causes anterior translation of the tibia in relation to the femur that can be palpated by the thumb; a soft or mushy end point indicates a positive test. When the ACL is disrupted, the normal patellar ligament slope is obliterated.



**FIGURE 23-59.** Anterior drawer test of anterior cruciate ligament. Foot is positioned in internal, external, and neutral rotation during examination. With anterior cruciate insufficiency, an anterior force (**A**) displaces the tibia forward (**B**).



**FIGURE 23-60.** Lateral pivot-shift test of anterior cruciate ligament.

#### **Stress Testing of Posterior Cruciate Ligament**

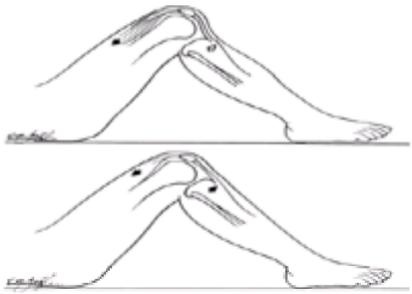
The posterior drawer test for evaluation of the PCL is done with the patient supine. With the patient's foot secured to the table and the hip flexed 90 degrees, a posterior force is applied to the proximal tibia. Posterior movement of the tibia on the femur greater than in the uninjured extremity indicates posterior instability (Fig. 23-61).



**FIGURE 23-61.** Posterior cruciate ligament injury. Note posterior sagging of the tibia with posterior cruciate injury.

#### **Active Quadriceps Contraction Test of Posterior Cruciate Ligament**

With the patient supine, the relaxed limb is supported with the knee flexed to 90 degrees. It is important to support the thigh adequately so that the child's muscles are completely relaxed. The examiner's other hand stabilizes the foot and the patient gently slides the foot down the table to contract the quadriceps muscle. Tibial displacement resulting from the quadriceps contraction is noted ( [Fig. 23-62](#)). When the PCL is ruptured, contraction of the quadriceps muscle results in an anterior shift of the tibia.



**FIGURE 23-62.** Active quadriceps contraction test of posterior cruciate ligament. **A:** Patient gently slides foot down the table to contract the quadriceps muscle. **B:** The tibia shifts anteriorly with quadriceps contraction when the posterior cruciate is torn.

#### **Radiographic Findings**

Anteroposterior and lateral radiographs are obtained when any ligament injury of the knee is suspected in children. The radiographs are carefully inspected for evidence of occult epiphyseal or physeal fractures or bony avulsions. The intercondylar notch, especially, is inspected to detect a tibial spine fracture, which is confirmed by anterior or posterior instability on physical examination. Occasionally, a small fragment of bone avulsed from the medial femur or proximal tibia indicates injury to the MCL. Similarly, avulsion of a small fragment of bone from the proximal fibular epiphysis or the lateral aspect of the distal femur may indicate LCL injury.

In children with open physes, stress radiographs are especially helpful to evaluate medial and lateral instability associated with physeal fractures ( [Fig. 23-63](#)). Gentle stress views may be obtained with sedation, but general anesthesia may be required if the diagnosis is unclear. There are no accepted guidelines correlating joint space widening medially or laterally with knee joint instability in children, and stress views of the opposite knee may be required for comparison. If plain radiographs show a fracture of the distal femoral or proximal tibial physis, stress views may be obtained only to evaluate suspected ligamentous instability. Conversely, if the initial radiographs appear normal but there is significant effusion about the knee, stress views should be obtained to rule out a fracture of the physis of the distal femur or proximal tibia.



**FIGURE 23-63.** Stress radiographs of suspected ligamentous injury. **A:** Valgus stress radiograph of 14-year-old boy with medial collateral ligament injury and tibial spine fracture. **B:** Varus stress radiograph of 10-year-old boy with lateral collateral ligament injury. ( **A** courtesy of John Roberts, MD, Bangor, Maine.)

Other radiographic findings include avulsion of the anterior or posterior tibial spine indicative of injury to the ACL or PCL, widening of the joint space, and posterior subluxation of the tibia on the femur. Clanton et al. ( [237](#)) consider a joint space of 8 mm or wider to be a definitive indication of ligament injury. Sanders et al. ( [278](#)) reported 1.8 cm of posterior subluxation of the tibia on the anteroposterior stress view in a 6-year-old child with complete PCL disruption. MRI should be used to evaluate ligamentous and meniscal lesions in confusing cases ( [284](#)).

#### **Methods of Treatment**

Clanton et al. ( [237](#)) and others have noted that the best treatment for knee ligament injuries in children has not been clearly defined. Poor results have been reported after conservative treatment and after primary repair ( [236,238,270](#)). Most authorities recommend conservative treatment of acute ligament injuries in young children, but give no clear indications for surgery. The numerous reconstructive techniques devised for the ACL are basically either extraarticular (avoiding the physes) or intraarticular ( [228,254,260,265,286](#)). The intraarticular procedures more accurately replace the ACL with an anatomic or biomechanical substitute that allows the isometric placement of the graft critical to prevent subsequent loosening.

McCarroll et al. ( [268](#)) compared 16 adolescents with ACL injuries treated conservatively with a similar group of 24 adolescents who had arthroscopic examination and extraarticular or intraarticular reconstruction of the ACL. Of the 16 treated nonoperatively, 6 required later surgery for meniscal injury and only 7 returned to sports activities; all experienced recurring episodes of giving way, effusion, and pain. Of the 24 treated surgically, torn menisci were found arthroscopically in 18 and 22 had

satisfactory results.

Lipscomb and Anderson (263) reported treatment of 24 ACL injuries in adolescents (12 to 15 years of age) with intraarticular augmentation using the semitendinosus or gracilis tendon. They used holes drilled in the appropriate position through the tibial physis and an over-the-top technique for placement of the augmentation over the femoral condyle. One patient had significant growth disturbance. Wester et al. (289) developed graphs to help predict the amount of shortening and angular deformity to expect after ACL repair in skeletally immature patients. According to their calculations, when done within 1 year of maturity, reconstruction should result in a maximum of 5 degrees of valgus angulation and 1 cm of shortening.

DeLee and Curtis (242) and Engebretsen et al. (247) reported moderate to severe instability in adolescents with ACL ruptures after primary suture with intraarticular or extraarticular augmentation. Kannus and Järvinen (262) reported successful closed treatment of grade II (moderate) ligament injuries in adolescents, but unacceptable results in grade III (complete) ligamentous tears; they recommended surgical repair of all grade III ligament injuries in adolescents. Parker et al. (275) reported good results after ligament reconstruction using hamstring tendons in six children with open physes (10 to 14 years of age). They placed a groove over the front of the tibia and another over the top of the femur without violating the physes, although this is not an isometric position of the ACL.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

Before determining treatment, we analyze each knee ligament injury, considering the child's skeletal age, the family's athletic expectations for the child, the presence of associated fractures, whether the injury is to an isolated ligament or to multiple ligaments, and whether the resulting instability is single-plane or multiplane.

### Medial Collateral Ligament

Isolated grade I or II sprains of the MCL are treated with crutches or a hinged knee brace for 1 to 3 weeks, depending on resolution of symptoms. Return to athletic activities is allowed when a full, painless range of motion is achieved and the patient can run and cut without pain. Isolated complete (grade III) disruption of the MCL can be treated with 6 weeks of immobilization in a hinged knee brace followed by rehabilitation of the quadriceps muscles and knee motion provided this is an isolated injury. The physician must ensure that there is no associated injury to the ACL before using nonoperative treatment for a grade III MCL injury.

Grade III disruptions of the MCL in adolescents often are associated with ACL injury or tibial spine fracture, as well as severe injury to the posteromedial knee capsule. If stress testing demonstrates gross instability, surgical repair is indicated. Midsubstance tears of the MCL and tears of the capsule can be sutured. An MCL avulsed from its distal insertion can be reattached with a staple if it can be placed well distal to the physis; if not, the ligament is repaired to the surrounding soft tissue.

### Anterior Cruciate Ligament

A torn ACL does not constitute a surgical emergency, despite the image projected by celebrity athletes and their urgent care. A frank discussion must be held with the parents and the patient concerning future vocation, sport demands, treatment options, outcomes, and risks involved with return to current sport activity. The orthopaedic surgeon must assume the role of a "knee counselor," particularly with patients with a history of chronic knee abuse. All treatment algorithms are based on an accurate and complete diagnosis, which is achieved by clinical, imaging, and, if necessary, arthroscopic means. The treatment goal is a functional knee without progressive intraarticular damage or predisposition to premature osteoarthritis.

Nonsurgical treatment does not indicate nontreatment (229,252,271). The goal of this program is defined from the outset: it is a temporizing measure until the patient becomes mature enough for an adult-type ACL reconstruction, or it is the definitive choice for a patient willing to accept the functional limitations. We use a three-phase approach. Phase one begins shortly after injury and lasts 7 to 10 days. Brief immobilization (3 to 5 days) with a knee immobilizer for comfort is followed by daily out-of-brace exercises with active knee flexion and passive knee extension. Ambulation is with crutch-protected partial weight bearing. During this time, we reinforce patient education on the consequences of imprudent return to high-level sports. Phase two focuses on rehabilitation of the lower extremity and lasts approximately 6 weeks. Emphasis is placed on restoration of full knee motion, flexibility, strength, and endurance with particular attention to regaining the normal quadriceps/hamstring strength ratio. As the ratio is normalized, crutch use is decreased and then eliminated. The role of functional bracing has not been defined in children and issues of fit, size, and cost need to be considered. Phase three continues the rehabilitation, incorporates the use of a functional brace for sports, and allows return to low- and moderate-demand sports when quadriceps and hamstring strength and endurance are equal to those of the opposite, noninjured side as determined by isokinetic testing at functional speeds (>260 degrees/second). In the final part of this phase, sports readiness tasks are done at less than full speed. Monthly follow-up evaluates program compliance and rules out any further knee changes in function. Compliance with a nonoperative program designed to be a temporizing measure often is quite difficult for an emerging athlete who is surrounded by peer, coaching, and, often, parental pressures to return to high-demand sports.

Surgical management of ACL tears in skeletally immature patients is highly controversial. In patients with what we term an *ACL plus knee* (one with combined ACL and meniscal injuries), surgical reconstruction usually is recommended because of the poor prognosis of meniscal repair alone with compromised ACL function (227,229,233,252,264,269,272,275,282,283). However, physeal injury and restoration of ligamentous isometry are concerns in the maturing femur and tibia. The safe percentage of physeal invasion, even in the central portion, that will be tolerated, is unknown. Experimental work in rodents by Garces et al. (251), lapin models by Guzzanti et al. (253) and Janarv et al. (259), and in canines by Stadelmeier et al. (282) suggests that central femoral physeal invasion by drilling that involves less than 7% to 9% of the total physeal area does not produce significant length or angular deformities in these experimental models. In animal studies that used tendon or fascial autografts, either free or in continuity, to fill the defects created in femoral and proximal tibial physes and epiphyses, Janarv et al. (259) found that thin bone cylinders developed around the tunnels filled with tendon grafts and questioned whether large tunnels would create enough cylinder bone to cause physeal bridges of consequential size, even when centrally placed. Whether the effects seen in these various quadruped models can be translated to the unique demands of human bipedal gait remains unanswered. Also in question are the effects of exposure time after physeal invasion because the animal models had short exposure times in contrast to the 2 to 4 years of growth remaining in adolescent humans, especially boys. An additional concern is the outcome of the grafted tissue that provides the biologic scaffold for neoligament formation in terms of the tissue's size, strength, isometry, and growth. None of the animal series used any type of transphyseal fixation.

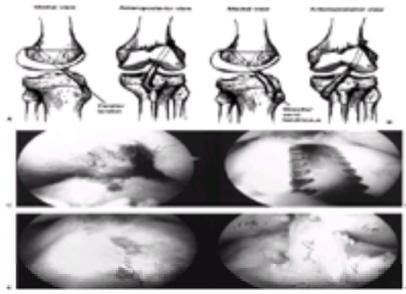
No data specifically address the timing of ACL reconstruction in skeletally immature patients after acute injury. We wait 14 to 21 days to allow abatement of the initial inflammatory phase, in keeping with the data that suggest in adults a reduced frequency of fibroarthrotic complications if surgery is delayed. During this time, crutch-protected gait is used and restoration of full passive extension is sought. We also use this time to reemphasize the 6-month time frame expected for recovery before return to sports. Even with contemporary accelerated rehabilitation programs, return to sport is a function of biology and not arthroscopic technology. The rare patient who presents with a locked knee from a concomitant displaced meniscal tear requires more urgent surgery for both injured structures.

Reconstruction of the ACL requires consideration of the three "Ts": tissues, tunnels, and technique. Autograft tissues include patellar and hamstring tendons, most commonly the semitendinosus alone or with gracilis augmentation. Allograft ACL substitution has been reported in eight children (227). Concerns of potential viral transmission and graft strength alterations secondary to sterilization effects remain (273). Synthetic ACL use as a backup to autograft or allograft causes concerns about longevity, foreign body synovitis from wear particles, and fixation techniques. Direct ACL repair is destined to failure unless a sufficient-sized bone fragment is avulsed to allow direct bone union.

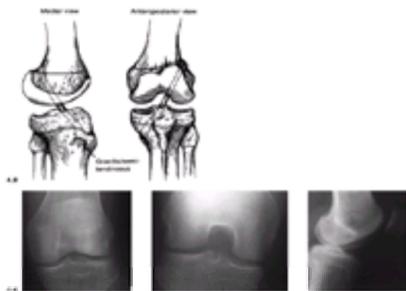
Extraarticular ACL reconstruction using fascia lata slings in a variety of configurations has had limited success in adults, and similar outcomes are predicted in skeletally immature patients who wish to return to high-demand sports. An additional concern is the potential tethering effect on the lateral knee produced by this method with secondary femoral or tibial physeal asymmetric growth.

Tunnels for ACL reconstruction are technique dependent and include grooves in the proximal tibial epiphysis, over-the-top femoral placement, and more vertical proximal tibial physeal and epiphyseal tunnel orientation to provide improved graft isometry. When selecting tunnels, we consider three types: physeal sparing, partial transphyseal, and complete transphyseal (283). Physeal-sparing methods avoid any physeal invasion with intraarticular passage of the graft. Partial transphyseal techniques use a central transphyseal and epiphyseal proximal tibial tunnel with intraarticular passage of a hamstring or patellar tendon graft to an over-the-top femoral position with femoral metaphyseal fixation. Complete transphyseal procedures involve central, isometrically oriented tibial and femoral tunnels and intraarticular graft passage and femoral and tibial epiphyseal or metaphyseal graft fixation sites. Physeal-sparing techniques are used with quite skeletally immature, Tanner stage 0 to 1 patients, usually with an associated meniscal injury that requires treatment (Fig. 23-64). In patients who are somewhat more mature (Tanner

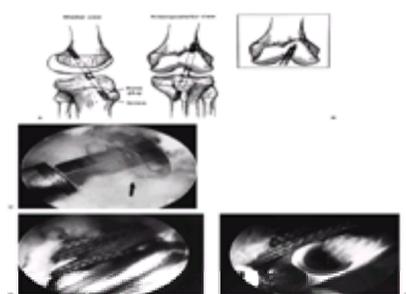
stage 2), partial transphyseal methods are suggested (Fig. 23-65). In patients who are Tanner stage 3 (boys with curly axillary hair and girls who are postmenarchal) and above, complete transphyseal, adult-type reconstructions are done because growth remaining at the distal femur and proximal tibia is limited ( Fig. 23-66).



**FIGURE 23-64.** Physeal-sparing anterior cruciate ligament reconstruction technique (A and B). Intraoperative arthroscopic views of tibial tunnel preparation of the epiphysis (C) and notchplasty (D). Epiphyseal “tunnel” groove under the anterior transverse meniscal ligament (E) with semitendinosus graft in place (F). (Courtesy of Scott Cameron, MD, Marshfield, Wisconsin.)



**FIGURE 23-65.** A and B: Schematic views of a partial transphyseal anterior cruciate ligament (ACL) reconstruction with medial hamstring graft. C, D, and E: Radiographs of a 16-year-old girl 5 years after partial transphyseal ACL reconstruction done at Tanner stage 2. Note the notchplasty effect. There is no evidence of femoral or tibial deformity.



**FIGURE 23-66.** A and B: Schematic views of one type of complete transphyseal anterior cruciate ligament reconstruction using a patellar tendon–bone construct. Inset shows an alternative intraarticular fixation method. C: An arthroscopic view of such fixation using a bioabsorbable screw. D and E: An alternative fixation technique using a multistranded hamstring graft with “button” femoral fixation.

The major limitation of the physeal-sparing procedure described by Parker et al. ( 275) is the lack of anatomic isometry of the neoligament. Each of their six patients who were 10 to 14 years of age had an associated meniscal tear that required treatment. At 2-year follow-up, outcomes were favorable. Although not optimal, this technique provides a reasonable option in very young patients with symptomatic functional instability, usually due to noncompliance in a nonoperative program and often associated with a subsequent meniscal tear.

Partial transphyseal techniques use 6- to 8-mm patellar or hamstring autografts in continuity that are passed through 6- to 8-mm tibial proximal tunnels more vertically oriented in an attempt to improve graft isometry. The over-the-top graft is attached to the femoral metaphysis, with care taken to not violate the lateral femoral physis. Lo et al. ( 264) reported 29 truly skeletally immature patients who had reconstruction of their torn ACL using this method, the largest series to date. All patients had excellent functional outcomes and objective test results at an average follow-up of 3 years. No clinical or radiographic evidence of limb deformity was seen.

Complete transphyseal ACL reconstruction methods are the same as adult techniques and are done in patients who are Tanner stage 3 or higher.

Graft choice depends on the surgeon's preference and may be a multistrand hamstring configuration or a patellar bone–patellar tendon–tibial tubercle construct. Graft fixation may be intraarticular or extraarticular. McCarroll et al. ( 269) reported good results from such reconstructions in mature adolescents with arthroscopically documented ACL complete tears that commonly were associated with meniscal tears that required repair. Even with the excellent results achieved by this experienced group of knee surgeons, they advocated delaying reconstruction in skeletally immature patients until sufficient maturity is reached to allow a complete transphyseal procedure.

Postoperative rehabilitation is based on physiologic principles that reduce deleterious effects of immobilization, protect the healing tissues, avoid negative misuse effects, and encourage graft maturation by providing physiologically tolerable stresses to it. Advancement to the next rehabilitative phase is predicated on the patient achieving previously determined functional criteria. The program is divided into four phases: phase 1, weight bearing and motion restoration; phase 2, muscle strength gains; phase 3, progressive improvement in speed, power, endurance, and agility; and phase 4, sport readiness and return to sport. Phase 1 begins immediately after surgery and continues for 6 weeks. The patient uses crutches for protected weight bearing and is encouraged to progress to full weight bearing without crutch support by 4 to 5 weeks. The postoperative knee immobilizer is replaced with a postoperative brace that limits extension to  $-30$  degrees and flexion to 90 degrees. Active flexion is encouraged and passive full extension out of the brace is done a minimum of four times daily. At the end of this phase, full weight bearing and passive extension are expected, as is knee flexion to 110 degrees. Resistive closed-chain hip flexor, quadriceps, hamstring, and gastrocnemius and anterior tibial and peroneal exercises are done three times daily. If concomitant meniscal repair was done, protected weight bearing and knee flexion limitation to 90 degrees are continued for 8 weeks. Phase 2 encompasses the next 6 weeks, during which time closed-chain resistive exercises are continued with quadriceps strength expected to be two thirds of the opposite normal side by isokinetic testing at 120 degrees per second with the knee protected at  $-30$  degrees of extension. Gait should be normal and passive knee motion full. Phase 3 lasts 6 weeks, with progressive emphasis on restoration of the quadriceps–hamstring strength ratio, which should be 75% to 80% of normal by isokinetic testing. A functional brace is fitted during this time and a running program is instituted. Phase 4 lasts for 6 weeks and involves normalization of lower extremity muscle strength, power, and endurance. Agility drills are advanced and sport-specific readiness tasks are done at progressively higher rates of speed. At the end of this phase, knee range of motion should be full and without pain, there should be no signs of functional instability, and the knee sagittal stability should be within 3 mm of the opposite normal side with normal muscle strength, power, and endurance. Functional brace use is continued for 3 to 6

months after return to sport, usually at the patient's request.

**Prognosis and Complications.** Limited data exist about postoperative complications after ACL reconstruction in skeletally immature patients, but the same type of complications seen in adults also might be expected in younger patients: loss of motion, donor and insertion site problems, and reflex sympathetic dystrophy. Although limited clinical and experimental data in skeletally immature models suggest that deformity and growth complications are uncommon after ACL reconstruction, these complications are possible, especially when judgmental or surgical technical errors occur.

**Authors' Preferred Treatment.** In immature patients with isolated ACL tears, we recommend nonoperative management with strong emphasis on patient counseling about the potential for additional intraarticular damage with return to high-demand sports. In patients with a concomitant meniscal injury that requires repair, the so-called "ACL plus" knee, we recommend ACL reconstruction, even in those who are relatively immature, because of the high potential for repeated intraarticular damage leading to premature degenerative arthrosis. Three types of ACL reconstruction are possible: physeal sparing, partial transphyseal, and complete transphyseal. The choice must be tailored to the patient's physiologic and anatomic assets and liabilities.

### **Grade III Sprains of Both Medial Collateral and Anterior Cruciate Ligaments**

The combination of grade III injuries of both the MCL and ACL is, unfortunately, fairly common, especially in older adolescents. In addition to complete disruption of the ACL, the MCL is torn either from its femoral origin, in its midsubstance, or from its tibial insertion. This combination of injuries results in multiplane instability. Treatment is based on the child's age and the specific injuries. In children with more than 2 years of skeletal growth remaining who have an avulsion of the MCL from its femoral origin, we recommend 6 weeks of immobilization in a hinged knee brace, followed by vigorous rehabilitation. If the MCL is torn in its midsubstance or avulsed from its tibial insertion with gross laxity, we recommend surgical repair with sutures in the midsubstance tear or a staple in the proximal tibia distal to the physis with no repair of the ACL, thus converting the multiplane instability to single-plane instability. If anterior or anterolateral instability persists during growth in a very young child, an extraarticular tenodesis can be done to protect the menisci until skeletal maturity. If symptoms warrant, intraarticular reconstruction of the ACL can be done as the adolescent nears skeletal maturity. For an adolescent within 12 to 24 months of skeletal maturity with grade III injuries of the MCL and ACL resulting in multiplane instability, we recommend surgical treatment of both ligaments. Careful repair of the posteromedial knee joint capsule often is required.

### **Lateral Collateral Ligament**

Grade III injuries of the LCL are rare in children. Occasionally, the lateral capsular sign is seen on radiographs obtained for evaluation of knee injury. Most often, the LCL is avulsed from the proximal fibular epiphysis; proximal and midsubstance tears are uncommon. This injury is treated in the same manner as injury to the MCL. For isolated grade III injuries, a 6-week period of immobilization in a hinged knee brace is recommended. If ACL injury is associated with LCL injury, treatment is as described for combined injuries of the MCL and ACL. In young children, the LCL is repaired and the ACL is left untreated. If instability persists as the adolescent nears skeletal maturity, intraarticular reconstruction can be done at that time. In adolescents near skeletal maturity at the time of injury, we prefer to repair both the ACL and LCL.

### **Posterior Cruciate Ligament**

In general, PCL injuries in children are associated with a bony avulsion. If nondisplaced, they should be treated with immobilization; if displaced, they should be treated with open reduction and internal fixation.

Isolated grade III injury of the PCL in children and adolescents can be treated with 6 weeks of immobilization in a cast-brace or hinged knee brace. If significant functional symptoms persist in an adolescent, the PCL can be reconstructed at or near skeletal maturity. In a young child with grade III MCL or LCL injury in addition to PCL injury, the collateral ligament can be surgically repaired to convert the multiplane instability to a single-plane instability. PCL injury may be associated with ACL injury, primarily in knee dislocations, and surgical intervention usually is appropriate for this combination of injuries, especially in patients nearing skeletal maturity (235).

### **Postoperative Care and Rehabilitation**

After cast-brace or hinged brace immobilization, aggressive rehabilitation is recommended, including strengthening of the quadriceps and hamstrings, progressive range-of-motion exercises, and progressive weight bearing. A vigorous physical therapy program is more difficult for younger children, but rehabilitation becomes progressively more important as the child becomes older, and it is critical in adolescents returning to high-demand competitive sports. Bracing is recommended during sports activities for adolescents with previous ACL injury.

### **Prognosis**

Isolated injuries of the collateral ligaments, with or without physeal fractures, have a generally good prognosis. With grade III isolated injuries of the ACL, however, long-term results are extremely variable, depending on the amount of late instability and the demands placed on the knee during athletic activities during adolescence or young adulthood. The worst prognosis is for combined grade III injuries of the cruciate and collateral ligaments that are treated conservatively.

### **Complications**

The three most serious potential complications of knee ligament injury are neurovascular complications, meniscal injuries, and late instability.

Careful assessment of the neurovascular status is mandatory at the initial examination. Any indication of neurovascular injury associated with severe knee ligament injury should prompt immediate investigation, including arteriography of the popliteal artery.

There is a high incidence of meniscal injury associated with ACL disruption, especially in adolescents. Even if surgical reconstruction of an isolated ACL rupture is not planned, arthroscopy may be done to identify any associated meniscal injury and to determine if primary repair or partial meniscectomy is appropriate.

Rarely, children with combined grade III injuries of the collateral and cruciate ligaments that have been treated conservatively acquire late instability. For young children with functional instability, reconstruction of the collateral ligament should be done to convert the multiplane instability to a single-plane instability. Then, as the child nears skeletal maturity, the cruciate ligament can be reconstructed if necessary. In an adolescent approaching skeletal maturity who has multiplane instability, both the cruciate and collateral ligaments should be reconstructed as in adults.

## **KNEE DISLOCATIONS**

Acute dislocations of the knee are uncommon in children because the forces required to produce dislocation are more likely to fracture the distal femoral or proximal tibial epiphysis (300). Acute knee dislocation usually involves major injuries of associated soft tissues and ligaments and often neurovascular injuries.

Adequate follow-up studies of acute knee dislocations in children younger than 10 years of age are few (296), and most information has been obtained from reports of knee dislocations in adults. Kennedy (305), in a classic 1963 study, analyzed 22 dislocations in young adults and recommended early ligament repair if the knee was unstable after reduction. Sisk and King (313) reviewed 62 patients treated for acute knee dislocations and recommended surgical treatment for patients younger than 40 years of age.

### **Surgical Anatomy**

#### **Soft Tissue Anatomy**

The pertinent surgical anatomy in acute dislocation of the knee involves the soft tissues and the vascular and ligamentous structures about the knee. Knee dislocation usually occurs with disruption of both cruciate ligaments. With direct anterior or posterior dislocation, the collateral ligaments and the soft tissues may be retained because the femoral condyles are stripped out of their capsular and collateral ligament attachments, and when reduced slip back inside them. Knee dislocations in

adolescents have been associated with tibial spine fractures, osteochondral fractures of the femur or tibia, meniscal injuries, and peroneal nerve injuries ( [295](#)).

### **Vascular Injury**

The anatomic structure of particular clinical significance is the popliteal artery. The collateral circulation about the knee is relatively poor, and if the popliteal artery is damaged, collateral circulation usually is insufficient to maintain viability of the extremity distal to the knee ( [295,302](#)). The popliteal artery basically is fixed to the femur at the adductor hiatus and to the tibia by a fibrous arch. During anterior dislocation of the knee, the artery often is stretched enough to cause intimal disruption and possibly subsequent vascular occlusion.

### **Mechanism of Injury**

Most knee dislocations in children occur with multiple trauma, especially in car, bicycle, or motorcycle accidents or pedestrian–vehicle accidents.

### **Classification**

There is no specific classification of knee dislocations in children, but the dislocation may occur anteriorly, posteriorly, medially, or laterally. The most common single-plane dislocation is anterior.

### **Signs and Symptoms**

Because of the potential for associated vascular injury, acute knee dislocations in children may be emergent situations. The dislocation causes obvious deformity about the knee. With anterior dislocation, the tibia is prominent in an abnormal anterior position. With posterior dislocation, the femoral condyles are abnormally prominent anteriorly.

After the dislocation is reduced, the stability of the knee should be evaluated with gentle stress testing. For isolated anterior or posterior dislocations, the integrity of the collateral ligaments should be carefully evaluated.

The neurovascular status of the extremity should be carefully evaluated both before and after reduction, especially the dorsalis pedis and posterior tibial pulses and peroneal nerve function. Any abnormal vascular findings, either before or after reduction, require arteriography and, if necessary, arterial exploration. Abnormalities in the sensory or motor function of the foot and distribution of the peroneal nerve function should be noted.

### **Radiographic Findings**

Anteroposterior and lateral radiographs at the initial evaluation confirm the clinically suggested direction of the dislocation, most commonly anterior ( [Fig. 23-67](#)). Radiographs in both views after reduction detect any occult fractures of the tibial spine, distal femoral physis, or proximal tibial physis. If collateral ligament injury is suspected, stress views also are obtained.



**FIGURE 23-67.** Dislocation of the knee. **A and B:** Anteromedial dislocation of the knee in a 14-year-old girl. **C:** Intimal tear and occlusion of popliteal artery after anterior knee dislocation in a male 19-year-old. (Courtesy of Richard E. King, MD, Atlanta, Georgia.)

Femoral arteriograms are obtained at the initial evaluation if dorsalis pedis or posterior tibial pulses are absent or diminished. The knee dislocation is reduced as quickly as possible and the vascular status reevaluated. Although distal pulses often are absent immediately after injury and return after reduction, an arteriogram can be done even after satisfactory pulses are restored to detect an intimal tear that may cause late thrombosis and occlusion. Arteriography is unnecessary when pulses are normal before and after reduction; however, the vascular status should be carefully monitored for 48 to 72 hours after reduction.

### **Methods of Treatment**

When treating a knee dislocation in a child, the physician must consider not only the acute knee injury with associated soft tissue and ligament injury and underlying fractures, but the possibility of neurovascular injury. No large series are available for comparison of the methods of treatment. In reports of knee dislocations in adults, most surgeons recommend repair of ligamentous injuries, especially in young patients. Closed reduction and splinting followed by 6 weeks of immobilization in a long leg cast usually is appropriate for children with acute knee dislocations without arterial injury. In adolescents approaching skeletal maturity, grade III injuries of the cruciate or collateral ligaments may be surgically repaired.

### **AUTHOR'S PREFERRED METHOD OF TREATMENT**

Our choice of treatment of knee dislocations in children is based on three factors: the child's age, associated ligamentous injury, and associated vascular injury.

In a child without vascular injury, the dislocation is reduced and the stability is tested by clinical examination and, if necessary, stress radiographs. Isolated cruciate ligament injury is treated nonoperatively, as described earlier.

### **Operative Treatment**

Injuries to both the cruciate and collateral ligaments are treated as outlined earlier. In an adolescent approaching skeletal maturity with cruciate and collateral ligament injuries but no vascular injury, we recommend repair or reconstruction of the ligaments as previously described.

In any child with a suspected arterial injury, arteriography is mandatory. If pulses are absent before reduction, even if they are satisfactory after reduction, we frequently obtain an arteriogram to detect any intimal tears that we believe should be repaired surgically. Obviously, the absence of the pulses after reduction is an emergent situation. If vascular injury has occurred, surgical correction must be done within 6 to 8 hours of injury to prevent subsequent ischemia and possible limb loss. In this critical situation, we obtain the arteriogram in the operating room, if at all. Arteriography is not mandatory in an isolated injury but is necessary to pinpoint the site of vascular injury if ipsilateral proximal injuries are present.

If primary arterial repair or grafting of the popliteal artery is required, we recommend fasciotomy of all four compartments of the leg at the same time to prevent the development of a compartment syndrome. Repair or reconstruction of ligaments should not be attempted at this time because this is too extensive a procedure to be

undertaken during emergent arterial repair.

If there are no postoperative complications after vascular repair, children may be immobilized in a cast for an additional 4 to 6 weeks and then started on a rehabilitation program. For adolescents near skeletal maturity who require vascular repair, two options are available for treatment of the ligamentous injuries. The ligaments can be repaired approximately 2 weeks after vascular repair; under ideal circumstances, with good wound healing and no postoperative complications, an experienced surgeon can undertake delayed primary repair of the ligament injuries. The other option is to wait and perform late reconstruction if required for cruciate or collateral ligament instability.

### Postoperative Care

For most knee dislocations treated with closed reduction, 3 to 6 weeks of cast immobilization is followed by quadriceps and hamstring strengthening, range-of-motion exercises, and progressive ambulation with crutches. If surgical repair is required, postoperative rehabilitation is the same as that described earlier for ligament injuries.

### Prognosis

The prognosis for acute knee dislocations in children is identical to that for isolated or combined ligamentous injuries. The worst prognosis is in children in whom late vascular repair results in ischemic changes in the extremity and in adolescents who require late ligament reconstruction because of knee instability.

### Complications

The most serious complications result from unrecognized and untreated vascular injury or a late intimal tear that causes thrombosis of the popliteal artery within 24 to 72 hours of injury. Careful evaluation and constant monitoring of the vascular status of the injured limb are mandatory, and aggressive use of arteriography is recommended, with immediate surgical exploration and repair or vein grafting when indicated.

## PATELLAR DISLOCATIONS

Patellar dislocation is relatively common in children if all subluxations and dislocations from varying causes are considered, but acute traumatic patellar dislocation caused by a direct blow is rare in children. McManus et al. (309) reviewed 55 acute patellar dislocations in children and concluded that acute dislocation occurs only in children with underlying patellofemoral dysplasia. Most acute patellar dislocations occur in patients between 16 and 20 years of age and are more common in female than in male patients.

### Surgical Anatomy

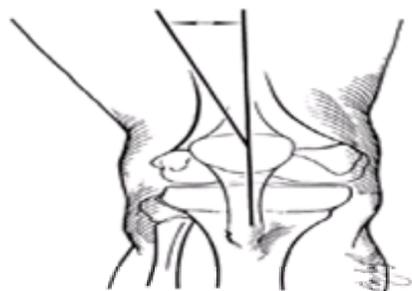
The patella is a sesamoid bone in the quadriceps mechanism. As the insertion site of all muscle components of the quadriceps complex, it serves biomechanically to provide an extension moment during range of motion of the knee joint. The trochlear shape of the distal femur stabilizes the patella as it tracks through a range of motion. The hyaline cartilage of the patella is the thickest in the body.

At 20 degrees of knee flexion, the inferior pole of the patella contacts a relatively small area of the femoral groove. With further flexion, the contact area moves superiorly and increases in size. The medial facet of the patella comes in contact with the femoral groove only when flexion reaches 90 to 130 degrees.

The average adult trochlear femoral groove height is 5.2 mm and lateral femoral condyle height is 3.4 mm. The patellar articular cartilage is 6 to 7 mm deep, the thickest articular cartilage in the body and a reflection of the joint's inherent incongruity. The usual normal lateral alignment of the patella is checked by the medial quadriceps expansion and focal thickening of the capsule in the areas of the medial patellofemoral and medial meniscopatellar ligaments (297). Dynamic stability depends on muscle forces, primarily the quadriceps and hamstrings acting through an elegant lower extremity articulated lever system that creates and modulates forces during gait. The quadriceps blends with the joint capsule to provide a combination of dynamic and static balance. Tightness or laxity of any of the factors involved with maintenance of the balance leads to varying levels of instability. Acute patellar dislocation almost always is in a lateral direction unless it is due to a medially oriented direct blow or follows overvigorous lateral retinacular release. Sallay et al. (312) demonstrated avulsions of the medial patellofemoral ligament from the femur in 94% (15 of 16) of patients during surgical exploration after acute patellar dislocation. Desio et al. (297), using a cadaveric serial cutting model, found that the medial patellofemoral ligament provided 60% of the resistance to lateral patellar translation at 20 degrees of knee flexion. The medial patellomeniscal ligament accounted for an additional 13% of the medial quadrant restraining force. If the deficit produced by attenuation of the medial vectors after acute dislocation is not eliminated, patellofemoral balance is lost, resulting in feelings of knee instability and recurrent dislocation.

The patella is under significant biomechanical compressive load during activity. It has been estimated that at 60 degrees of knee flexion, the forces across the patellofemoral articulation are three times the body weight and increase to over seven times the body weight during full knee flexion.

The quadriceps mechanism is aligned in a slightly valgus position in relation to the patellar tendon. This alignment can be approximated by a line drawn from the anterosuperior iliac spine to the center of the patella. The force of the patellar tendon is indicated by a line drawn from the center of the patella to the tibial tubercle. The angle formed by these two lines is called the *quadriceps angle* or *Q angle* (Fig. 23-68). As this angle increases, the pull of the extensor mechanism tends to subluxate the patella laterally. Recurrent patellar dislocation is most likely associated with some congenital or developmental deficiency of the extensor mechanism, such as patellofemoral dysplasia, deficiency of the vastus medialis obliquus, or an increased Q angle with malalignment of the quadriceps–patellar tendon complex.



**FIGURE 23-68.** The Q angle. Normal valgus alignment of the quadriceps mechanism: line drawn from the anterosuperior iliac spine to center of the patella, line drawn from center of the patella to tibial spine.

### Mechanism of Injury

Larsen and Lauridsen (306) found that a direct blow to the medial aspect of the patella accounted for only 10% of the acute knee dislocations in their series. Patellar dislocations are more likely to be caused by falls, gymnastics, dancing, cheerleading, and a wide variety of other activities. Acute patellar dislocation also should be considered in the evaluation of all athletic injuries in adolescents and young adults.

### Classification

Although there is no specific classification of patellar dislocations in children, acute dislocation should be distinguished clinically from chronic patellar subluxation or dislocation. Approximately 15% of children with acute patellar dislocations experience recurrent dislocations. Cash and Hughston (293) reported a 60% incidence of

redislocation in patients 11 to 14 years of age, 30% in patients 19 to 28 years of age, and in only one patient older than 28 years of age. Intraarticular dislocation of the patella also should be recognized ([292,298,299,301](#)). This injury is uncommon in children but occurs frequently in adults with chronic soft tissue laxity or severe degeneration of the quadriceps tendon.

### Signs and Symptoms

The orthopaedist rarely sees a child with an acute patellar dislocation in whom the patella remains in a dislocated position ([Fig. 23-69](#)). Usually, the patella has reduced spontaneously with active or passive extension of the knee. Symptoms include diffuse parapatellar tenderness and pain with any attempt passively to displace the patella. A defect may be palpable in the medial attachment of the vastus medialis obliquus to the patella if the medial retinaculum is completely avulsed. Tenderness on the lateral aspect of the knee usually is not as severe as on the medial side. Hemorrhage into the joint may cause hemarthrosis, and severe hemarthrosis should suggest the possibility of an osteochondral fracture ([311](#)). Nietosvaara et al. ([183](#)) reported that of 72 patients with acute patellar dislocations, 28 (39%) had associated osteochondral fractures. These fractures included 15 capsular avulsions of the medial patellar margin and 15 loose intraarticular fragments detached from the patella, the lateral femoral condyle, or both. All knee ligaments should be carefully evaluated because the mechanism of patellar dislocation may cause associated ligamentous injuries.



**FIGURE 23-69.** Acute dislocation of the left patella in a 6-year-old boy.

### Radiographic Findings

Radiographs after acute dislocation are obtained primarily to detect any associated osteochondral fracture. Occasionally, an osteochondral fragment from the medial aspect of the patella or the lateral femoral condyle is visible on the anteroposterior or lateral view. The classic “sunrise” view is difficult to obtain in a child after acute dislocation because the required positioning of the knee causes pain. Rarely, stress radiographs may be obtained for evaluation of suspected physeal fracture or ligamentous injury. CT or MRI is valuable to check for an osteochondral fracture.

### Methods of Treatment

Most acute patellar dislocations in children reduce spontaneously; if not, reduction usually can be easily obtained. After appropriate sedation, reduction is done by flexing the hip to relax the quadratus femoris, gradually extending the knee, and gently pushing the patella medially back into its normal position.

Surgery rarely is indicated for acute patellar dislocations in children ([170,294,306](#)). Surgical repair may be indicated if the vastus medialis obliquus is completely torn from the medial aspect of the patella, leaving a large, palpable soft tissue gap. If osteochondral fracture has occurred, arthroscopy may be indicated for removal or repair of an osteochondral loose body. Even more rarely, open reduction and fixation of a large fragment and repair of the medial retinaculum may be necessary.

Avulsion of the quadriceps tendon from the patella most often causes intraarticular dislocation. Closed treatment is unsuccessful, so open reduction is necessary to reduce the patella and repair the tear of the quadriceps tendon. Even more uncommon is the intraarticular dislocation, in which the patellar ligament is torn from the tibial tuberosity. Associated injury of the cruciate ligaments should be carefully evaluated at the time of surgery and surgically repaired as indicated.

### AUTHOR'S PREFERRED METHOD OF TREATMENT

Most acute patellar dislocations in children are easily treated by closed methods with satisfactory results. A cylinder cast is used for 2 to 4 weeks or, in children with compliant parents, a knee immobilizer is worn for the same duration. Surgical intervention is appropriate in the circumstances outlined previously, most commonly for an intraarticular osteochondral fracture. We generally prefer arthroscopic examination and removal of the fragment if possible, and perform arthrotomy only if necessary for removal or fixation of a large osteochondral fracture. Acute surgical repair of the vastus medialis obliquus rarely is indicated.

### Postoperative Care and Rehabilitation

After the 2- to 4-week immobilization period, a physical therapy program is begun for progressive strengthening of the quadriceps muscles and gentle range-of-motion knee exercises. After arthroscopic debridement and removal of an osteochondral fragment, immobilization is continued an additional 3 weeks, followed by the same rehabilitation program. Rarely, if surgical repair of the vastus medialis obliquus is required, cylinder cast immobilization is continued for 4 to 6 weeks during the postoperative period.

### Prognosis

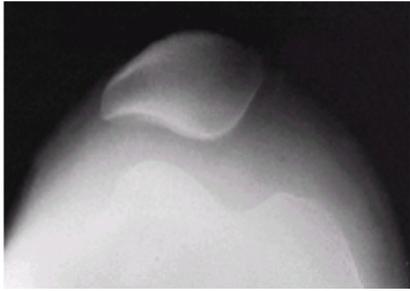
The prognosis of patellar dislocations in children is generally good. Approximately one in six children with acute patellar dislocations experiences recurrent dislocations. Cash and Hughston ([293](#)) noted 75% satisfactory results after nonoperative treatment in carefully selected patients.

### Complications

Complications frequently occur because of an unrecognized osteochondral fracture, which is diagnosed later by the presence of a loose body. Radiographs obtained after injury should be carefully scrutinized for any osteochondral injury. Occasionally, complications occur because of associated ligamentous injury, particularly of the medial collateral or cruciate ligaments, and especially in adolescent male athletes.

### Chronic Patellar Subluxation

Chronic patellar subluxation or dislocation is common in adolescents, especially girls. Several risk factors have been identified in children likely to have chronic subluxation or dislocation, including age younger than 16 years, radiographic evidence of dysplasia of the patella or lateral femoral condyle, significant atrophy of the vastus medialis obliquus, hypermobility of the patella, and multiple previous dislocations ([Fig. 23-70](#)).

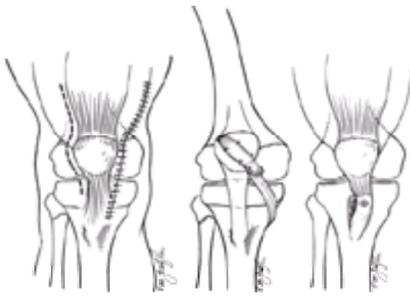


**FIGURE 23-70.** Chronic lateral patellar subluxation in 13-year-old girl.

Initial treatment of chronic patellar subluxation or dislocation in adolescents is immobilization followed by aggressive physical therapy for rehabilitation of the vastus medialis obliquus and quadriceps muscles. Surgical intervention is warranted in children with several risk factors who do not respond to this treatment regimen and continue to have subluxation or dislocation ( [291,304,307,308](#)). Micheli and Stanitski ( [310](#)) reviewed 33 skeletally immature patients with lateral retinacular releases and found that the procedure did not interfere with permanent alignment of the extensor mechanism. They recommended the technique for children who do not respond to an aggressive physical therapy program.

### Operative Treatment

If subluxation or dislocation persists after adequate lateral release, correction of the medial deficiency is indicated, usually by advancement of the medial retinaculum and muscle and plication of the vastus medialis muscle ( [Fig. 23-71A](#)). If deficiency of the vastus medialis obliquus is severe, this procedure can be done at the time of lateral retinacular release. Transfer of the semitendinosus through the inferior pole of the patella also has been reported in skeletally immature patients ( [303](#)) ( [Fig. 23-71B](#)). This may be indicated in adolescents with continued instability after lateral release and medial realignment, or in children with associated connective tissue disorders. In skeletally mature patients with a significantly abnormal Q angle, the Elmslie-Trillat procedure reportedly achieves good results in approximately 80%. This technique displaces the anterior tibial tubercle medially to decrease the Q angle ( [Fig. 23-71C](#)). The Elmslie-Trillat procedure is contraindicated in patients with open physes because of the possibility of growth disturbance of the anterior tibial tubercle, with resulting genu recurvatum.



**FIGURE 23-71.** Surgical technique for treatment of chronic patellar subluxation or dislocation. **A:** Lateral retinacular release and medial imbrication. **B:** Semitendinosus tenodesis. **C:** Elmslie-Trillat procedure.

## MENISCAL INJURIES

Although less common in children than in adults, meniscal injuries are becoming more frequent in children and adolescents, especially those involved in competitive athletics ( [314,319,343,364,377](#)). DeHaven and Lintner ( [330](#)), in a study of 3,431 athletes, found internal derangement of the knee to be one of the two most common injuries in young athletes, particularly boys. Most meniscal injuries occur in adolescents older than 12 years of age, but they have been reported in very young children ( [290,339,363,374](#)). King ( [348](#)) reported 52 patients younger than 15 years of age who had undergone arthrotomy because of suspected meniscal injuries, and Fowler ( [334](#)) reported 117 meniscectomies in patients 12 to 16 years of age.

### Surgical Anatomy

The menisci develop early in fetal life from the intermediate zone of mesenchyme between the distal femur and the proximal tibia and assume their adult semilunar form by the 10th fetal week ( [325,347](#)). During the remainder of growth, the menisci change in size but not in shape. Growth of the meniscus occurs in the peripheral part, where the avascular fibrocartilage becomes loose-textured vascular fibrous tissue. The peripheral edges of the menisci are convex, fixed, and attached to the inner surface of the knee joint capsule, except where the popliteus is interposed laterally, and are attached loosely to the borders of the tibial plateaus by the coronary ligaments. The inner edges are concave, thin, and unattached. The menisci are largely avascular, except near their peripheral attachment to the coronary ligaments.

Although the lateral meniscus shows more developmental variation, as demonstrated by Clark and Ogden ( [325](#)), it is never discoid in shape during normal development. The predominant collagen is type I arranged circumferentially with oblique, vertical, and radial fibers present to diminish hoop stresses with weight bearing. The menisci show constant ratios of meniscal to articular tibial plateau surface areas during development and demonstrate intimate concordant maturation of the menisci with the tibia ( [325](#)). In addition to lesser roles of synovial fluid distribution and contribution to knee stability, the major role of the menisci is to share weight-bearing loads by increasing articular contact area and reducing the load per unit area on articular cartilage, with 70% of the lateral compartment load borne by the lateral meniscus and 50% of the medial load by the medial meniscus. Recent *in vivo* MRI documentation of normal meniscal motion under load was reported by Vedi et al. ( [373](#)). With weight bearing, the anterior medial meniscus moved an average of 7.1 mm and the posterior medial horn showed an average excursion of 3.9 mm along with 3.6 mm average mediolateral radial displacement. The lateral anterior meniscus and the lateral posterior horn moved an average of 9.5 and 5.6 mm, respectively, with a radial mediolateral average displacement of 3.7 mm. These findings of dramatic dynamic changes of meniscal position reaffirm the preliminary work reported by Thompson et al. ( [371](#)).

Biomechanically, the menisci act as a joint filler to compensate for the incongruity between the articulating surfaces of the femur and tibia and prevent capsular and synovial impingement during flexion and extension of the knee. The menisci are pushed outward by the compression forces of the tibia and femur, but the strong anterior and posterior attachments of the menisci generate circumferential tension forces (hoop forces) that counteract this outward or radial force ( [366](#)). The menisci transmit and distribute loads between the articular surfaces, serving as shock absorbers to spare the articular cartilage from compressive loads and to protect the joint from osteoarthritic changes.

The menisci also are believed to nourish the articular cartilage and lubricate the joint by the distribution of synovial fluid. They contribute to stability in all planes but are especially important rotary stabilizers and are essential for the smooth gliding or rotation motion as the knee moves from flexion into extension.

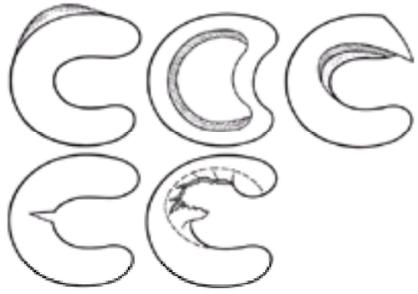
### Mechanism of Injury

Meniscal tears are most commonly produced by rotation as the flexed knee moves toward extension. This rotational force with the knee partially flexed changes the relation of the femoral condyles to the menisci, and forces the menisci toward the center of the joint, where they are likely to be injured. These twisting mechanisms occur primarily in sports and may cause associated ligamentous injuries. Meniscal injuries also may be associated with degenerative changes, cystic formation, or

congenital anomalies (334).

### Classification

The most commonly used classification is based on the type of meniscal tear found at surgery, either peripheral, bucket-handle, horizontal cleavage, transverse, or complex. Although previous studies reported that lateral lesions were more common in children, King (348) found more medial lesions in his patients, most commonly peripheral detachment of the posterior portion (Fig. 23-72). He also noted that the younger the patient, the more peripheral the tear; conversely, bucket-handle tears occurred most often in older children and adolescents.



**FIGURE 23-72.** Meniscal tears in adolescents. **A:** Peripheral. **B:** Bucket-handle. **C:** Horizontal cleavage. **D:** Transverse. **E:** Complex.

### Signs and Symptoms

The most difficult aspect of treatment may be making the correct diagnosis. Bergstrom et al. (231) and Juhl and Boe (345) reported diagnostic accuracy in approximately 20% of children believed to have meniscal injury. An accurate history may be difficult to obtain in a very young child. The older the patient, the more likely a history of specific injury. The patient usually relates feeling or hearing a “pop” at the time of injury, with frequent popping and giving way after injury. Pain is reported by approximately 85% of patients, with tenderness over the affected joint line. More than half report giving way and effusion of the knee joint. McMurray's and Apley's tests may be helpful in the diagnosis of a chronic lesion, but with acute injury the knee usually is too painful to allow these maneuvers (329).

In Vahvanen and Aalto's series of patients with documented meniscal tears (372), almost one third of the patients had no significant findings on physical examination. We find the classic McMurray test of little value in this age group whose tears are peripheral and not degenerative posterior horn lesions. The most accurate physical findings are joint line tenderness (especially middle to posterior) and exacerbation of the pain with varus (medial) and valgus (lateral) and rotation stress (internal, medial; external, lateral) at 30 to 40 degrees of knee flexion. These signs have value: 92.3% negative predictive value, 93.3% sensitivity, and 92.3% specificity (284).

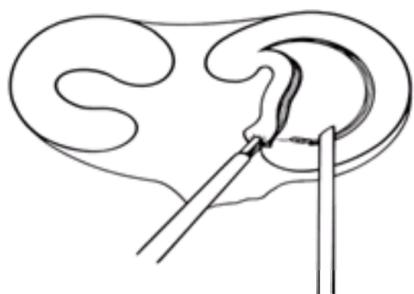
### Radiographic Findings

Routine radiographs are obtained primarily to eliminate other sources of knee pain. Arthrography (326) may help delineate meniscal tears, but has been used less frequently since the advent of arthroscopy and MRI (357,359). MRI's accuracy rates reportedly range from 45% to 90% in the diagnosis of meniscal tears (324,343,361,365), and it is the preferred imaging method for evaluating meniscal injuries in children. MRI should not be used as a screening procedure because of significant limitations of the technique in this age group (284,323,351,361,368). Takeda et al. (370) reviewed the MRI signal intensity and pattern in the menisci of 108 knees in 80 normal children 8 to 15 (average 12.2) years of age using the classification of Zobel et al. (379), which allows for equivocation for type III signals. Using tibial tubercle maturity as a definition of skeletal maturity, Takeda et al. (370) found signal intensity to be proportional to age, with high signal (grades III and III) evident in 80% of patients 10 years of age or younger, 65% by 13 years of age, and 33% at 15 years of age, similar to the false-positive rate of 29% reported in asymptomatic adults (336,351). Overall, two thirds of the patients had positive findings (grades II or III), often grade III-A, which is equivocal extension through the surface of the meniscus. Takeda et al. suggested that the decrease in signal intensity was proportional to diminution of peripheral vascularity, especially in the posterior horn of the meniscus. Schwartzberg et al. (368) used unilateral MRI to study 55 asymptomatic 9- to 15-year-olds with negative knee examinations and found that 51% had grade III and 27% grade II signal changes in their medial menisci. Grade III signals were found in 7% and grade II changes were seen in 7% of lateral menisci. All children with grade III changes in the lateral meniscus also had grade III signals in the medial side. These investigators cautioned against misinterpretation of pediatric knee MRIs and emphasized the necessity for correlation of the clinical findings with any imaging study results. In another series (284), poor correlation was seen between MRI reports and arthroscopic findings, with significant numbers of false-positive and false-negative findings reported on MRI interpretation by radiologists: 37.5% accuracy, 50% positive and 50% negative predictive indexes, 50% sensitivity, and 45% specificity. Current MRI techniques usually provide limited information about tear size and stability or predictability of healing after repair. As with any test, clinical correlation is mandatory before treatment decisions are made.

### Methods of Treatment

The traditional treatment of a torn meniscus has been meniscectomy, but numerous reports (317,322,333,342,350,355,369,372) indicating the poor long-term results of meniscectomy in children have made this less common. Up to 60% to 75% of patients have degenerative changes after meniscectomy. Manzione et al. (352) reported 60% poor results in 20 children and adolescents after meniscectomy. In cadaver studies, Baratz et al. (320) showed that the contact stresses on the tibiofemoral articulation increase in proportion to the amount of the meniscus removed and the degree of disruption of the meniscal structure. Clearly, as much of the meniscus should be preserved as possible.

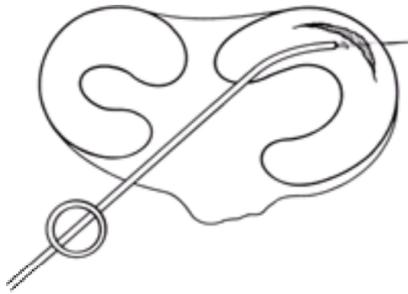
As King (348) pointed out, removal of a damaged meniscus to minimize irreversible damage to the joint is advisable, but every effort should be made to spare the meniscus. The exact meniscal injury and potential for repair can be determined arthroscopically to help formulate treatment plans. Zaman and Leonard (378) recommended observation of small peripheral tears, repair of larger peripheral tears, and, when necessary, partial meniscectomy, leaving as much of the meniscus as possible; they concluded that total meniscectomy is contraindicated in young patients (Fig. 23-73). In general, peripheral tears, which are most common in children, and longitudinal tears are good candidates for repair, with success rates of up to 90% reported (328,338,341,353,357). Sisk (366) recommended that repair be limited to the most peripheral 25% of the meniscus.



**FIGURE 23-73.** Arthroscopic partial medial meniscectomy for bucket-handle tear in an adolescent.

Open and arthroscopic techniques for meniscal repair share several important factors:

1. Anatomic reduction of the meniscus
2. Multiple, closely placed sutures that are not readily absorbable
3. Careful avoidance of neurovascular structures
4. Fixation of the meniscus directly on the knee capsule ( [Fig. 23-74](#) )



**FIGURE 23-74.** Arthroscopic repair of a peripheral tear of the posterior horn of the medial meniscus.

Although King (349) suggested over six decades ago that, based on experimental evidence in dogs, longitudinal meniscal tears could heal if communication with peripheral blood supply existed, it was not until the work of Arnoczky and Warren (318) in the 1980s that meniscal repairs were begun based on documentation of the meniscal blood supply. They believed that tears within 3 mm of the meniscosynovial junction were vascularized, and ones more than 5 mm away were avascular unless bleeding was seen at surgery. Tears in the 3- to 5-mm range had inconsistent vascularity. The zones of meniscal injury are 0 to 3: 0 represents the synovial-perimeniscal vascular plexus, 1 corresponds to the red-red junction of vascularity (0 to 3 mm), 2 is the red-white transitional area from vascular to avascular (3 to 5 mm), and 3 is the white-white avascular region (5 mm to the central border).

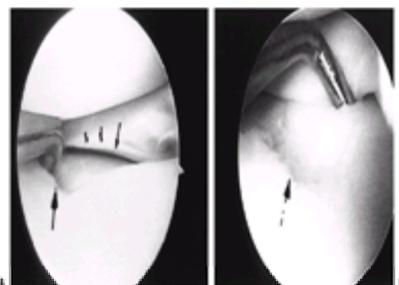
Some questions still remain about the ability of the repaired meniscus to transmit load because of the altered fiber arrangement.

### AUTHOR'S PREFERRED METHOD OF TREATMENT

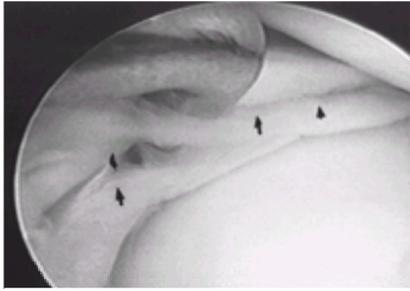
Treatment is based on a classification scheme we call SAKS, an acronym that represents size, site, shape and stability of the tear (S); acuity of the lesion (A); and knee stability (KS) or ACL and MCL integrity. In a stable knee with an acute, arthroscopically documented outer third peripheral tear that is less than 1 cm long and cannot be displaced more than 3 mm, we immobilize the knee in a cylinder cast for 3 weeks in the acute setting. For similar chronic injury (>3 months), we arthroscopically rasp the interface between the meniscal edges and immobilize the knee in a cylinder cast for 4 weeks. If the tear is in zones 1 or 2, and is longitudinal with a noncomminuted inner segment that can be reduced anatomically, repair is done ( [Fig. 23-75](#) and [Fig. 23-76](#) ). If a chronic situation exists, rasping of the fragment edges and use of a fibrin clot are techniques that have been reported to enhance healing. Mintzer et al. (356) had a 100% success rate in meniscal repairs in 26 adolescent athletes (29 repairs) with an average follow-up of 5 years. Four patients had open physes at time of the meniscal repair. Most (78.5%) were zone 0 to 1 tears with an average length of 2.3 cm. Thirteen patients had associated ACL tears that were reconstructed. All but two of the patients returned to their preinjury sport level. If the central fragment is macerated with multiplanar tears or ones that cannot be reduced, repair is not done ( [Fig. 23-77](#) ). The fragment is excised to the stable remaining meniscal rim. Results of repairs of tears that are unstable and located in the central third (zone 2 to 3) have not been reported to any extent in skeletally immature patients. Rubman et al. (362) reported good results in adults with central zone meniscal repairs, with 80% being asymptomatic at an average follow-up of under 4 years. Most (72%) had associated ACL injuries that were reconstructed. These investigators suggested that the repairs would seem to be more indicated in patients in their second and third decades than in older patients. Unstable tears in the central third, white-white zone are excised, taking care to preserve as much stable meniscal rim as possible. The rare radial tear or horizontal tear is debrided, leaving as much intact meniscus as is stable. With horizontal tears, the smaller of the two leaves is resected.



**FIGURE 23-75.** Arthroscopic view of a red-white junction tear in a 15-year-old girl. *Arrowheads* denote the vascular edge of the peripheral meniscus.



**FIGURE 23-76. A:** Arthroscopic view of a displaced fragment (*large arrow*) of a large peripheral longitudinal tear of the lateral meniscus. The *small arrows* indicate the stable peripheral meniscal margin. **B:** Full-thickness articular damage (*arrow*) of the lateral tibial compartment from the trauma that caused the meniscal injury.



**FIGURE 23-77.** Arthroscopic view of a comminuted, macerated white-zone free edge tear of the medial meniscus. This pattern of injury is best treated by tear debridement and not by repair.

In an adolescent patient with an unstable knee from an MCL injury and a peripheral meniscal tear, repair of the meniscus and ligament is indicated. If the meniscal tear is in a zone not amenable to repair, the fragment is debrided and the collateral ligament injury is managed nonoperatively.

Outcomes after meniscal repair in adults are negatively related to the length and chronicity of the meniscal injury. Failure rates of 10% to 15 % are seen in peripheral tears of less than 2 cm and in 60 % if the tears exceed 4 cm. In tears that have been present for less than 8 weeks, the results are good in approximately 85 %, compared with 65 % in more chronic tears (>8 weeks) (325). These data probably are applicable to patients older than 11 or 12 years of age because the perimeniscal blood supply pattern at that age is identical to the flow in adults.

In patients with what we call the “ACL plus” knee, one with concomitant meniscal and ACL tears, meniscal salvage, by repair if possible, and ACL reconstruction are indicated. To allow knee instability to persist invites repeat meniscal injury and additional articular compromise.

Multiple techniques exist for repair (309,316,323,325,356). Four main types of repair are used: outside-in, inside-out, all inside, and open, depending on the meniscal tear characteristics. Open repair usually is reserved for injuries associated with complete tears of the MCL or meniscal damage that cannot be approached arthroscopically. Inside-out repairs use various zone-specific cannulae that allow precise suture placement, usually a vertical mattress stitch, which has been shown to have suture strength twice that of a horizontal stitch. Outside-in repairs use spinal needles placed across the capsule and meniscus. A suture is passed through the needle and brought outside the knee, a knot is fashioned, the suture is reintroduced intraarticularly, and the knot is used to maintain meniscal reduction and fixation. The outsides of the sutures are then tied to the outer capsule. This technique is helpful in areas where more blind suture passage could cause neural and vascular compromise. The all-inside suture technique is technically demanding and currently an emerging method. A variety of bioabsorbable meniscal anchors are in use that obviate the need for capsular exposure and external sutures. These devices sometimes are too long or bulky to use in a child's knee.

Preparation of the edges of the meniscal tear is a matter of the tear's acuity and pattern. A meniscal rasp can be used to stimulate a vascular response at the perimeniscal vascular leash. Improved results in patients with chronic tears have been reported with use of a fibrin clot, which provides a collagen framework for vascular invasion and repair. The reader is referred to standard arthroscopic texts for details of meniscal repair (325).

#### Postoperative Care and Rehabilitation

Because meniscal healing is slow, careful rehabilitation after surgical repair is especially important. Knee motion should be restricted by a cast or brace for 4 to 6 weeks, with limited weight bearing. Full weight bearing is then allowed and a strengthening program is begun. Return to vigorous sports activities is not allowed for at least 6 months.

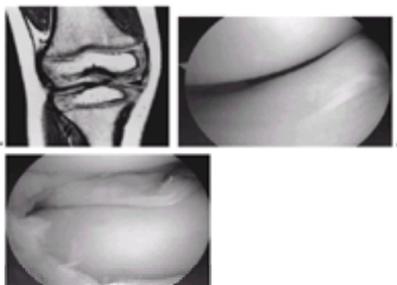
#### H4>Complications

Complications after either arthroscopic or open repair may include hemorrhage, infection, persistent effusion, stiffness, and neuropathy. Both the popliteal artery and inferior geniculate branches are close to the posterior capsule and are easily lacerated. Postoperative infection should be suspected if swelling or pain persists with an elevated temperature. Swelling is best treated with external compression dressings, and stiffness is best prevented by appropriate postoperative rehabilitation. Neuroma formation rarely causes significant symptoms, but occasionally persistent localized tenderness may warrant excision.

### DISCOID MENISCUS

Discoid meniscus (327,331,332,344,358) is a common cause of knee symptoms in children and adolescents. As noted by Woods and Whelan (376), discoid meniscus probably is a congenital deviation that usually occurs laterally. According to Hayashi et al. (340), the thickness of a discoid meniscus, its poor vascularization, and a flimsy attachment of the posterior area to the capsule make it more susceptible to mechanical stress than a normal meniscus. In their study of 53 symptomatic discoid lateral menisci, most of the patients were between 10 and 15 years of age.

The most frequent symptom is pain during ordinary activities, followed by giving way, locking, and snapping of the knee joint (346). Knee extension usually is limited and the quadriceps muscles often are atrophied. Occasionally, effusion, hemarthrosis, and instability also are present. Radiographs often are negative, but they may show widening of the lateral compartment, squaring of the femoral condyle, hypoplasia of the lateral tibial spine, tilting of the tibial articular surface, and apparent elevation of the fibular head. Arthrography or MRI may be helpful in establishing the diagnosis (330,333,337), but a horizontal tear in the midsubstance or a transverse cleavage in the middle segment may not be visible. Even with arthroscopy, identification of tears in the midsubstance of the meniscus may be difficult. Hamada et al (337) reported that intrameniscal regions of high signal intensity and flattening on MRI correspond to intrasubstance tears or degeneration of lateral discoid menisci not detectable by arthroscopy. Based on their findings, MRI seems to be more sensitive than arthroscopy in the detection of intrasubstance lesions of lateral discoid menisci (Fig. 23-78).



**FIGURE 23-78.** A: Magnetic resonance image of a 3-year-old girl who had symptoms of popping and catching in the knee demonstrates large discoid lateral meniscus. B: Arthroscopic view of discoid lateral meniscus. C: Arthroscopic subtotal meniscectomy. (Courtesy of Robert H. Miller III, MD, Memphis, Tennessee.)

#### Classification

Watanabe et al. (375) described three major types of discoid meniscus: type 1, completely disk-shaped semilunar type with a thinner center; type 2, incomplete semilunar type with a concave or convex free edge; and type 3, hypermobile or Wrisberg type without posterior tibial capsular attachment. The most common is the complete type. Neither Bellier et al. (321) nor Hayashi et al. (340) found any Wrisberg type 3 lesions in their combined total of 72 discoid menisci, and they believed it to be a rare lesion. The most common sites of meniscal tear are at the posterior segment and inside the middle segment.

### Methods of Treatment

When chronic locking of the knee, joint effusion, or pain warrants surgical treatment, arthroscopic subtotal or partial meniscectomy is preferred whenever possible (335,354). Hayashi et al. (340) advised reducing the thickness of complete-type discoid menisci to prevent new tears; they recommended leaving a rim of 6 mm in complete and 8 mm in incomplete lesions. Because the arrangement of collagen fibers of the discoid meniscus differs from that in the normal meniscus, and because the tear is most commonly close to the least vascular and most mobile area, partial meniscectomy may not be helpful for many tears in discoid menisci.

In a study of 47 knees with symptomatic discoid lateral menisci, Pellacci et al. (360) found that the results of partial meniscectomy were better than those of total meniscectomy. Aichroth et al. (315), however, recommended arthroscopic partial meniscectomy only when the posterior attachment of the discoid meniscus is stable; total meniscectomy is recommended for Wrisberg-type (type 3) discoid menisci with posterior instability.

Sugawara et al. (367) evaluated 139 partial or complete arthroscopic meniscectomies and found that 9 knees (6.5%) required 2 or more operations. Of this group, seven knees had partial meniscectomies of lateral discoid menisci. Because of their findings, they suggested that the most appropriate initial treatment for lateral discoid menisci without tears is observation of the clinical course, with minimal treatment using diagnostic arthroscopy. If clinical symptoms are serious or if an apparent meniscal tear is identified, they recommended subtotal or total meniscectomy rather than partial meniscectomy.

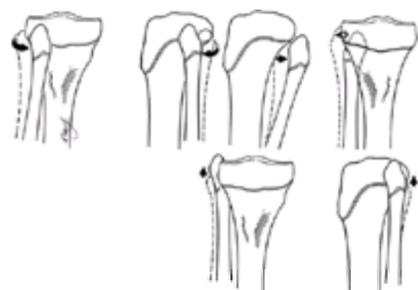
The need for total meniscectomy usually is dictated by the thickness of the discoid meniscus, the location of the tear, and the duration of symptoms preoperatively. Despite the generally poor prognosis after total meniscectomy, several investigators (321,331,340) report excellent results in children after total meniscectomy of discoid lateral menisci.

### AUTHOR'S PREFERRED METHOD OF TREATMENT

For types 1 and 2 discoid menisci, we recommend arthroscopic partial meniscectomy and shaving to a stable meniscus with an intact peripheral rim. For the rare type 3 discoid meniscus with no posterior capsular attachment, the options are total meniscectomy or posterior repair combined with partial meniscectomy.

### TIBIOFIBULAR DISLOCATIONS

Dislocations of the proximal tibiofibular joint are infrequent in children and adolescents, but may be more common than appreciated; the injury is estimated to go unrecognized initially in approximately one third of patients (378,381,382,386,387,388,390,395). Most of these dislocations occur during athletic activities, especially with a severe twisting movement. The dislocation or subluxation may be associated with other skeletal injuries, especially proximal tibial fractures (392). Disruption of the proximal tibiofibular joint is classified as subluxation, anterolateral dislocation, posteromedial dislocation, or superior dislocation (Fig. 23-79). The most common type is anterolateral dislocation. Pain along the lateral side of the knee and leg is the most common symptom of subluxation. Isolated anterolateral dislocation may be identified by a palpable mass lateral to the tibia and usually is visible on radiographic examination.



**FIGURE 23-79.** Tibiofibular subluxation–dislocation (Ogden). **A:** Anterolateral. **B:** Posteromedial. **C:** Superior.

Symptoms caused by subluxation usually subside with rest, but cylinder cast immobilization may be required. Most dislocations, especially acute dislocations, can be reduced closed by pushing the head of the fibula backward. Posteromedial dislocations often are associated with severe disruption of the joint capsule and rupture of the LCL, and these may require open reduction, capsulorrhaphy, and repair of the ligament. Superior dislocations are rare and usually are associated with fractures of the tibia, which may require open reduction and internal fixation. Recurrent subluxation or dislocation may cause progressive peroneal neuropathy and may require ligament reconstruction, resection of the proximal fibula, or arthrodesis of the tibiofibular joint (380,383,385,389,391,393,394).

### AUTHOR'S PREFERRED METHOD OF TREATMENT

We prefer ligament reconstruction to arthrodesis or proximal fibular resection. Shapiro et al. (391) described a technique of reconstruction using an iliotibial band fascial graft. They cited as advantages of this procedure the maintenance of normal motion of the knee and normal mechanics of the ankle. Arthrodesis results in loss of rotational motion of the fibula, which can cause problems at the knee and ankle joints. Resection of the fibular head alters the normal contour of the knee and may cause peroneal nerve palsy, chronic ankle pain, and lateral knee instability. These procedures should be reserved for patients with failed ligament reconstruction or those who are not candidates for reconstruction.

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## CHAPTER REFERENCES

### Distal Femoral Epiphysis Fractures

1. Abbott LC, Gill GG. Valgus deformity of the knee resulting from injury to the lower femoral epiphysis. *J Bone Joint Surg* 1942;24:97–113.
2. Aitken AP, Magill HK. Fractures involving the distal femoral epiphyseal cartilage. *J Bone Joint Surg Am* 1952;34:96–108.
3. Anderson M, Green WT, Messner MB. Growth and predictions of growth in the lower extremities. *J Bone Joint Surg Am* 1963;45:1–14.
4. Bassett FH III, Goldner JL. Fractures involving the distal femoral epiphyseal growth line. *South Med J* 1962;55:545–557.
5. Bellin H. Traumatic separation of epiphysis of lower end of femur. *Am J Surg* 1937;37:306–311.
6. Beaty JH, Kumar A. Current concepts review: fractures about the knee in children. *J Bone Joint Surg Am* 1994;76:1870–1880.
7. Bertin KC, Goble EM. Ligament injuries associated with physeal fractures about the knee. *Clin Orthop* 1983;177:188–195.
8. Blount WP. *Fractures in children* [Reprint]. Huntington, NY: Robert E. Krieger, 1977.
9. Böhler L. *The treatment of fractures*, 5th ed, vol 2. New York: Grune & Stratton, 1957.
10. Bowen JR, Leahy JL, Zhang Z, et al. Partial epiphysiodesis at the knee to correct angular deformity. *Clin Orthop* 1985;198:184–190.

11. Brash JC, ed. *Cunningham's textbook of anatomy*, 9th ed. New York: Oxford University Press, 1951.
12. Brashear HR Jr. Epiphyseal fractures. *J Bone Joint Surg Am* 1959;41:1055–1064.
13. Brashear HR Jr. Epiphyseal fractures of the lower extremity. *South Med J* 1958;51:845–851.
14. Brashear HR Jr. In Discussion of Bassett FH, Goldner JL. Fractures involving the distal femoral epiphyseal growth line. *South Med J* 1962;55:545–557.
15. Brone LA, Wroble RR. Salter-Harris type III fracture of the medial femoral condyle associated with an anterior cruciate ligament tear. *Am J Sports Med* 1998;26:581–586.
16. Burman MS, Langsam MJ. Posterior dislocation of lower femoral epiphysis in breech delivery. *Arch Surg* 1974;38:250–260.
17. Caffey J, Madell SH, Royer C, et al. Ossification of the distal femoral epiphysis. *J Bone Joint Surg Am* 1958;40:647–654.
18. Caffey J. *Pediatric x-ray diagnosis*, 2nd ed. Chicago: Year Book Medical Publishers, 1950.
19. Carey J, Spence L, Blickman H, et al. MRI of pediatric growth plate injury: correlation with plain film radiographs and clinical outcome. *Skeletal Radio*. 1998;27:250–255.
20. Carlson WO, Wenger DR. A mapping method to prepare for surgical excision of a partial physeal growth arrest. *J Pediatr Orthop* 1984;4:232–238.
21. Cole PA, Ehrlich MG. Management of the completely stiff pediatric knee. *J Pediatr Orthop* 1997;17: 67–73.
22. Coleman SS. Lower limb-length discrepancy. In: Lovell WW, Winter RB, eds. *Pediatric orthopaedics*. Philadelphia: JB Lippincott, 1986:781–863.
23. Connelly JF. Epiphyseal traction to correct acquired growth deformities. An animal and clinical investigation. *Clin Orthop* 1986;215:258–268.
24. Cuxart A, Iborra J, Melendez M, et al. Physeal injury in myelomeningocele patients. *Paraplegia* 1992;30:791–794.
25. Caterini R, Farsetti P, L'Arrigo C, et al. Unusual physeal lesions of the lower limb: a report of 16 cases with very long-term follow-up observation. *J Orthop Trauma* 1991;5:38–46.
26. Diamond LS, Alegado R. Perinatal fractures in arthrogryposis multiplex congenita. *J Pediatr Orthop* 1981;1:189–192.
27. Edvardsen P. Physioepiphyseal injuries of lower extremities in myelomeningocele. *Acta Orthop Scand* 1972;43:550–557.
28. Gomes LSM, Volpon JB. Experimental physeal fracture separations treated with rigid internal fixation. *J Bone Joint Surg Am* 1993;75:1756–1764.
29. Goss CM, ed. *Gray's anatomy of the human body*, 29th ed. Philadelphia: Lea & Febiger, 1973:664.
30. Grant JCB. *An atlas of anatomy*. Baltimore: Williams & Wilkins, 1947.
31. Green WT, Anderson M. Epiphyseal arrest for the correction of discrepancies in length of the lower extremities. *J Bone Joint Surg Am* 1957;39:853–872.
32. Griswold AS. Early motion in the treatment of separation of the lower femoral epiphysis. *J Bone Joint Surg* 1928;10:75–77.
33. Heller EP. Fracture separation "slipping" of the lower femoral epiphysis. *J Bone Joint Surg* 1933;15:474–476.
34. Hresko MT, Kasser JR. Physeal arrest about the knee associated with nonphyseal fractures in the lower extremity. *J Bone Joint Surg Am* 1989;71:698–703.
35. Hutchinson J Jr. Lectures on injuries to the epiphyses and their results. *BMJ* 1894;1:669–673.
36. Hutchinson J Jr, Barnard HL. An improved method of treatment of separation of the lower epiphysis of the femur. *Lancet* 1898;2:1630.
37. Ilizarov GA, Deviatov AA. Operative elongation of the leg with simultaneous correction of the deformities. *Ortop Traumatol Prot* 1969;30:32–37.
38. Kendall RW, Taylor DC, Salvan AJ, et al. Selective arteriography in assessing vascular injury associated with dislocation of the knee. *J Trauma* 1993;35:875–878.
39. Kumar SJ, Cowell HR, Townsend P. Physeal, metaphyseal, and diaphyseal injuries of the lower extremities in children with myelomeningocele. *J Pediatr Orthop* 1984;4:25–27.
40. Kurlander JJ. Slipping of the lower femoral epiphysis. *JAMA* 1931;96:513–517.
41. Kwolek C, Sundaram S, Schwarcz TH, et al. Popliteal artery thrombosis associated with trampoline injuries and anterior knee dislocations in children. *Am Surg* 1998;64:1183–1187.
42. Langenskiold A. An operation for partial closure of an epiphyseal plate in children and its experimental basis. *J Bone Joint Surg Br* 1975;57:325–330.
43. Langenskiold A, Osterman K. Surgical Elimination of post-traumatic partial fusion of the growth plate. In: Houghton GR, Thompson GH, eds. *Problematic musculoskeletal injuries in children*. London: Butterworths, 1983.
44. Loder RT, Swinford AE, Kuhns LR. The use of helical computed tomographic scan to assess bony physeal bridges. *J Pediatr Orthop* 1997;17:356–359.
45. Lombardo SJ, Harvey JP Jr. Fractures of the distal femoral epiphyses: factors influencing prognosis: a review of 34 cases. *J Bone Joint Surg Am* 1977;59:742–751.
46. Mann DC, Rajmaira S. Distribution of physeal and nonphyseal fractures in 2650 long-bone fractures in children age 0–16 years. *J Pediatr Orthop* 1990;10:713–716.
47. Menelaus MB. Correction of leg-length discrepancy by epiphyseal arrest. *J Bone Joint Surg Br* 1966;48:336–339.
48. Monticelli G, Spinelli R. Distraction epiphysiolysis as a method of limb lengthening: three clinical applications. *Clin Orthop* 1981;154:274–285.
49. Moseley CF. A straight-line graph for leg-length discrepancies. *J Bone Joint Surg Am* 1977;59:174–179.
50. Mubarak SJ, Hargens AR, Owen CA, et al. The Wick catheter technique for measurement of intramuscular pressure. *J Bone Joint Surg Am* 1976;58:1016–1020.
51. Neer CS II. Separation of the lower femoral epiphysis. *Am J Surg* 1960;99:756–761.
52. Neer CS II, Horwitz BS. Fractures of the proximal humeral epiphysal plate. *Clin Orthop* 1965;41:24–31.
53. Ogden JA. *Skeletal injury in the child*, 2nd ed. Philadelphia: WB Saunders, 1990.
54. Ozonoff MB. *Pediatric orthopaedic radiology*. Philadelphia: WB Saunders, 1979.
55. Paley D. Review article: current techniques of limb lengthening. *J Pediatr Orthop* 1988;8:73–92.
56. Partio EK, Tuompo P, Hirvensalo E, et al. Totally absorbable fixation in the treatment of fractures of the distal femoral epiphyses. *Arch Orthop Trauma Surg* 1997;116:213–216.
57. Patterson WJ. Separation of the lower femoral epiphysis. *CMAJ* 1929;21:301–303.
58. Pauwels F. *Biomechanics of the locomotor apparatus*. New York: Springer-Verlag, 1980.
59. Peltonen JI. Bone formation and remodeling after symmetric and asymmetric physeal distraction. *J Pediatr Orthop* 1989;9:191–196.
60. Peterson HA. Review: partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246–258.
61. Poland J. *Traumatic separation of the epiphyses*. London: Smith, Elder, 1898.
62. Rang M. *Children's fractures*, 2nd ed. Philadelphia: JB Lippincott, 1983.
63. Riseborough EJ, Barrett IR, Shapiro F. Growth disturbances following distal femoral physeal fracture–separations. *J Bone Joint Surg Am* 1983;65:885–893.
64. Roberts JM. Fracture–separation of the distal femoral epiphysis. *J Bone Joint Surg Am* 1973;55:1434.
65. Roberts JM. Operative treatment of fractures about the knee. *Orthop Clin North Am* 1990;21:365–379.
66. Rodgers WB, Schwend RM, Jaramillo D, et al. Chronic physeal fractures in myelodysplasia. *J Pediatr Orthop* 1997;17:615–621.
67. Salter RB, Czitrom A, Willis RB. Fractures involving the distal femoral epiphyseal plate. In: Kennedy JC, ed. *Injury to the adolescent knee*. Baltimore: Williams & Wilkins, 1979.
68. Salter RB, Harris WR. Injuries involving the epiphyseal plate. *J Bone Joint Surg Am* 1963;45:587–622.
69. Scheffer MA, Peterson HA. Opening-wedge osteotomy for angular deformities of the long bones in children. *J Bone Joint Surg Am* 1994;76:325–334.
70. Sharrard WJW. *Paediatric orthopaedics and fractures*, 2nd ed. Oxford: Blackwell Scientific Publications, 1979.
71. Simoninan PT, Saheli LT. Periarticular fractures after manipulation for knee contractures in children. *J Pediatr Orthop* 2000;15: 288–291.
72. Smith LA. Concealed injury to the knee. *J Bone Joint Surg Am* 1962;44:1659–1660.
73. Snedecor ST, Knapp RE, Wilson HB. Traumatic ossifying periostitis of the newborn. *Surg Gynecol Obstet* 1935;61:385–387.
74. Snedecor ST, Wilson HB. Some obstetrical injuries to the long bones. *J Bone Joint Surg Am* 1949;31:378–384.
75. Soutter FE. Spina bifida and epiphyseal displacement. *J Bone Joint Surg Br* 1962;44:106–109.
76. Stephens DC, Louis DS, Louis E. Traumatic separation of the distal femoral epiphyseal cartilage plate. *J Bone Joint Surg Am* 1974;56:1383–1390.
77. Tachdjian MO. *Pediatric orthopaedics*, 2nd ed, vol 4. Philadelphia: WB Saunders, 1990.
78. Thomson JD, Stricker SJ, Williams MW. Fractures of the distal femoral epiphyseal plate. *J Pediatr Orthop* 1995;15:474–478.
79. Tolo VT. External skeletal fixation in children's fractures. *J Pediatr Orthop* 1983;3:435–442.
80. Torg JS, Pavlov, H, Morris VB. Salter-Harris type III fracture of the medial femoral condyle occurring in the adolescent male. *J Bone Joint Surg Am* 1981;63:586–591.
81. Treiman GS, Yellin AE, Weaver FA, et al. Examination of the patient with a knee dislocation: the case for selective arteriography. *Arch Surg* 1992;127:1056–1062.
82. Watson-Jones R. *Fractures and joint injuries*, 4th ed. Edinburgh: E. & S. Livingstone, 1955–56.
83. Weber BG, Brunner C, Freuler F. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980.
84. Wenger DR, Jeffcoat BT, Herring JA. The guarded prognosis of physeal injury in paraplegic children. *J Bone Joint Surg Am* 1980;62:241–246.
85. Zuege RC. Epiphyseal stapling for angular deformity at the knee. *J Bone Joint Surg Am* 1979;61:420–329.

#### Separation of the Proximal Tibial Epiphysis

86. Blanks RH, Lester DK, Shaw BA. Flexion-type Salter II fracture of the proximal tibia. *Clin Orthop* 1994;301:256–259.
87. Burkhart SS, Peterson HA. Fractures of the proximal tibial epiphysis. *J Bone Joint Surg Am* 1979;61:996–1002.
88. Bylander B, Hagglund G, Selvik G. Stapling for tibial growth deformity. *Acta Orthop Scand* 1989;60:487–490.
89. Gautier E, Ziran BH, Egger B, et al. Growth disturbances after injuries of the proximal tibial epiphysis. *Arch Orthop Trauma Surg* 1998;118:37–41.
90. Nicholson JT. Epiphyseal fractures about the knee. *Instr Course Lect* 1967;18:74–83.
91. Poulsen TD, Skak SV, Toftgaard-Jensen T. Epiphyseal fractures of the proximal tibia. *Injury* 1989;20:111–113.
92. Resnick D, Niwayama G. *Diagnosis of bone and joint disorders, with emphasis on articular abnormalities*. Philadelphia: WB Saunders, 1981.
93. Shelton WR, Canale ST. Fractures of the tibia through the proximal tibial epiphyseal cartilage. *J Bone Joint Surg Am* 1979;61:167–173.
94. Welch PH, Wynne GF Jr. Proximal tibial epiphyseal fracture–separation: case report. *J Bone Joint Surg Am* 1963;45:782–784.
95. Wood KB, Bradley JP, Ward WT. Pes anserinus interposition in a proximal tibial physeal fracture. *Clin Orthop* 1991;264:239–242.
96. Wozasek GF, Moser KD, Haller H, et al. Trauma involving the proximal tibial epiphysis. *Arch Orthop Trauma Surg* 1991;110:301–306.

#### Avulsion of the Tibial Tubercle

97. Borch-Madsen P. On symmetrical bilateral fracture of the tuberositas tibiae and eminentia intercondyloidea. *Acta Orthop Scand* 1954;24:44–49.
98. Blackburne JS, Peel TE. A new method of measuring patellar height. *J Bone Joint Surg Br* 1977;59:241–242.
99. Blumensaat C. Die Lageabweichungen und Verrenkungen der Kniescheibe. *Ergeb Chir Orthop* 1938;31:149–223.
100. Breathnach AS, ed. *Frazer's anatomy of the human skeleton*, 6th ed. Boston: Little, Brown, 1965.
101. Bruijn JD, Sanders RJ, Hansen BRH. Ossification in the patellar tendon and patella alta following sports injuries in children. *Arch Orthop Trauma Surg* 1993;112:157–158.
102. Chrisman OD, Snook GA, Wilson TC. A long-term prospective study of the Hauser and Roux-Goldthwait procedures for recurrent patellar dislocation. *Clin Orthop* 1979;144:27–30.
103. Christie MJ, Dvonch VM. Tibial tuberosity avulsion fracture in adolescents. *J Pediatr Orthop* 1981;1:391–394.
104. Clark HO. Discussion on fracture of the tibia involving the knee joint. *Proc R Soc Med* 1935;28:1035–1050.
105. Crosby EB, Insall J. Recurrent dislocation of the patella. relation of treatment to osteoarthritis. *J Bone Joint Surg Am* 1976;58:9–13.
106. Deliyannis SN. Avulsion of the tibial tuberosity. *Injury* 1973;4:341–344.
107. Ehrenborg G. The Osgood-Schlatter lesion: a clinical and experimental study. *Acta Chir Scand Suppl* 1962;288:1–36.
108. Fairbank HAT. Contribution to discussion. *Proc R Soc Med* 1935;28:1049–1050.
109. Hand WL, Hand CR, Dunn AW. Avulsion fractures of the tibial tubercle. *J Bone Joint Surg Am* 1971;53:1579–1583.
110. Helfet AJ. *Disorders of the knee*. Philadelphia: JB Lippincott, 1974.
111. Insall J, Salvati E. Patella position in the normal knee joint. *Radiology* 1971;101:101–104.
112. Kaplan EB. Avulsion fracture of proximal tibial epiphysis: case report. *Bull Hosp Joint Dis* 1963;24:119–122.
113. Kaufer H. Mechanical function of the patella. *J Bone Joint Surg Am* 1971;53:1551–1560.
114. Lancourt JE, Cristini JA. Patella alta and patella infera. *J Bone Joint Surg Am* 1975;57:1112–1115.
115. Levi JH, Coleman CR. Fracture of the tibial tubercle. *Am J Sports Med* 1976;4:254–263.
116. Maquet P. Mechanics and osteoarthritis of the patellofemoral joint. *Clin Orthop* 1979;144:70–73.
117. Ogden JA, Tross RB, Murphy MJ. Fractures of the tibial tuberosity in adolescents. *J Bone Joint Surg Am* 1980;62:205–215.
118. Pape JM, Goulet JM, Hensinger RN. Compartment syndrome complicating tibial tubercle avulsion. *Clin Orthop* 1993;295:201–204.
119. Roberts JM. Avulsion of the proximal tibial epiphysis. In: Kennedy JC, ed. *The injured adolescent knee*. Baltimore: Williams & Wilkins, 1979.
120. Sobotta J, Figge FHJ. *Sobotta-Figge atlas of human anatomy*, 9th ed. New York: Hafner Press, 1974.
121. Trueta J. *Studies of the development and decay of the human frame*. London: Heinemann, 1968.
122. Wiss DA, Schilz JL. Frontal type III fracture of the tibial tubercle in adolescents. *J Orthop Trauma* 1991;5:475–479.

#### Osgood-Schlatter Lesion

123. Aparicio G, Abril JC, Calvo E, et al. Radiologic study of patellar height in Osgood-Schlatter disease. *J Pediatr Orthop* 1997;17:63–66.
124. Cole JP. A study of Osgood-Schlatter disease. *Surg Gynecol Obstet* 1937;65:55–67.
125. Ehrenborg G. The Osgood-Schlatter lesion: a clinical study of 170 cases. *Acta Chir Scand* 1962;124:89–105.
126. Ehrlich JG, Strain RE Jr. Epiphyseal injuries about the knee. *Orthop Clin North Am* 1979;10:91–103.
127. Holstein A, Lewis GB, Schulze ER. Heterotropic ossification of patellar tendon. *J Bone Joint Surg Am* 1963;45:656.
128. Jakob RP, von Gumpfenberg S, Engelhardt P. Does Osgood-Schlatter disease influence the position of the patella? *J Bone Joint Surg Br* 1981;63:579–582.
129. Larson RL. Epiphyseal injuries in the adolescent athlete. *Orthop Clin North Am* 1973;4:839–851.
130. Mital MA, Matza RA, Cohen J. The so-called unresolved Osgood-Schlatter lesion. *J Bone Joint Surg Am* 1980;62:732–739.
131. Ogden JA, Southwick WO. Osgood-Schlatter's disease and tibial tuberosity development. *Clin Orthop* 1976;116:180–189.
132. Osgood RB. Lesions of the tibial tubercle occurring during adolescence. *Boston Med Surg J* 1903;148:114–117.
133. Rosenberg ZS, Kawelblum M, Cheung YY, et al. Osgood-Schlatter lesion: fracture or tendonitis. Scintigraphic, CT and MR imaging features. *Radiology* 1992;185:853–858.
134. Rosenthal RK, Levine DB. Fragmentation of the distal pole of the patella in spastic cerebral palsy. *J Bone Joint Surg Am* 1977;59:934–939.
135. Rostron PKM, Calver RF. Subcutaneous atrophy following methylprednisolone injection in Osgood-Schlatter epiphysitis. *J Bone Joint Surg Am* 1979;61:627–628.
136. Schlatter C. Verletzungen des schnabelformigen Fortsatzes der oberen Tibiaepiphyse. *Beitr Klin Chir* 1903;38:874–887.
137. Stirling RI. Complications of Osgood-Schlatter's disease. *J Bone Joint Surg Br* 1952;34:149–150.
138. Turner MS, Smillie IS. The effect of tibial torsion on the pathology of the knee. *J Bone Joint Surg Br* 1981;63:396–398.
139. Willner P. Osgood-Schlatter's disease: etiology and treatment. *Clin Orthop* 1969;62:178–179.
140. Woolfry BF, Chandler EF. Manifestations of Osgood-Schlatter's disease in late teenage and early adulthood. *J Bone Joint Surg Am* 1960;42:327–332.

#### Fractures of the Patella

141. Andersen PT. Congenital deformities of the knee joint in dislocation of the patella and achondroplasia. *Acta Orthop Scand* 1958;28:27–50.
142. Beddow FH, Corkery PH, Shatwell GL. Avulsion of the ligamentum patellae from the lower pole of the patella. *J R Coll Surg Edinb* 1963;64–65:66–69.
143. Belman DAJ, Neviasser RJ. Transverse fracture of the patella in a child. *J Trauma* 1973;13:917–918.
144. Bernhang AM, Levine SA. Familial absence of the patella. *J Bone Joint Surg Am* 1973;55:1088–1090.
145. Bostrom A. Fracture of the patella. *Acta Orthop Scand Suppl* 1972;43:1–80.
146. Crock HV. The arterial supply and venous drainage of the bones of the human knee joint. *Anat Rec* 1962;144:199–217.
147. Devas MB. Stress fractures of the patella. *J Bone Joint Surg Br* 1960;42:71–74.
148. Diebold O. Uber Kniescheibenbrüche im Kindesalter. *Arch Klin Chir* 1927;147:664–681.
149. George R. Bilateral bipartite patellae. *Br J Surg* 1935;22:555–560.
150. Green WT Jr. Painful bipartite patellae. *Clin Orthop* 1975;110:197–200.
151. Griswold AS. Fractures of the patella. *Clin Orthop* 1954;4:44–56.
152. Grogan DP, Carey TP, Leffers D, et al. Avulsion fractures of the patella. *J Pediatr Orthop* 1990;10:721–730.
153. Houghton GR, Ackroyd CE. Sleeve fractures of the patella in children. *J Bone Joint Surg Br* 1979;61:165–168.
154. Kaye JJ, Freiburger RH. Fragmentation of the lower pole of the patella in spastic lower extremities. *Radiology* 1971;101:97–100.
155. Kohler A, Zimmer EA. *Borderlands of the normal and early pathologic in skeletal roentgenology*, 3rd ed. New York: Grune & Stratton, 1968.
156. Lieb FJ, Perry J. Quadriceps function: an anatomical and mechanical study using amputated limbs. *J Bone Joint Surg Am* 1968;50:1535–1548.
157. Lieb FJ, Perry J. Quadriceps function: an electromyographic study under isometric conditions. *J Bone Joint Surg Am* 1971;53:749–758.
158. Peterson, L, Stener B. Distal disinsertion of the patellar ligament combined with avulsion fractures at the medial and lateral margins of the patella. *Acta Orthop Scand* 1976;47:680–685.
159. Ray JM, Hendrix T. Incidence, mechanism of injury and treatment of fractures of the patella in children. *J Trauma* 1992;32:464–457.
160. Rorabeck CH, Bobeck WP. Acute dislocation of the patella with osteochondral fracture. a review of 18 cases. *J Bone Joint Surg Br* 1976;58:237–240.
161. Saue E. Beitrag zur Patella Bipartita. *Fortschr Geb Rontgenstr* 1921;28:37–41.
162. Scapinelli R. Blood supply of the human patella: its relation to ischaemic necrosis after fracture. *J Bone Joint Surg Br* 1967;49:563–570.
163. Schoenbauer HR. Brüche der Kniescheibe. *Ergeb Chir Orthop* 1959;42:56–79.
164. Sinding-Larsen MF. A hitherto unknown affection of the patella in children. *Acta Radiol* 1922;1:171–173.
165. Weber MJ, Janecki CJ, McLeod P, et al. Efficacy of various forms of fixation of transverse fractures of the patella. *J Bone Joint Surg Am* 1980;62:215–220.
166. Wu CD, Huang SC, Liu TK. Sleeve fracture of the patella in children. *Am J Sports Med* 1992;19:525–528.

#### Osteochondral Fractures

167. Aichroth PM. Osteochondral fractures and osteochondritis dissecans in sportsmen's knee injuries. *J Bone Joint Surg Br* 1977;59:108(abstr).
168. Ahstrom JP. Osteochondral fracture in the knee joint associated with hypermobility and dislocation of the patella: report of 18 cases. *J Bone Joint Surg Am* 1965;47:1491–1502.
169. Bailey WH, Blundell GE. An unusual abnormality affecting both knee joints in a child. *J Bone Joint Surg Am* 1974;56:814–816.
170. Bassett FH. III. Acute dislocation of the patella, osteochondral fractures, and injuries to the extensor mechanism of the knee. *Instr Course Lect* 1976;25:40–49.
171. Benz G, Roth H, Zachariou Z. Fractures and cartilage injuries of the knee joint in children. *Z Kinderchir* 1986;41:219–226.
172. Cofield RH, Bryan RS. Acute dislocations of the patella: results of conservative treatment. *J Trauma* 1977;17:526–531.
173. Coleman HM. Recurrent osteochondral fracture of the patella. *J Bone Joint Surg Br* 1948;30:153–157.
174. Edwards DH, Bentley G. Osteochondritis dissecans patellae. *J Bone Joint Surg Br* 1977;59:58–63.
175. Goodfellow J, Hungerford DS, Zindel M. Patellofemoral joint mechanics and pathology: I. Functional anatomy of the patellofemoral joint. *J Bone Joint Surg Br* 1976;58:287–290.
176. Henderson NJ, Houghton GR. Osteochondral fractures of the knee in children. In: Houghton GR, Thompson GH, eds. *Problematic musculoskeletal injuries in children*. London: Butterworths, 1983.
177. Hughston JC, Hergenroeder PT, Courtenay BG. Osteochondritis dissecans of the femoral condyles. *J Bone Joint Surg Am* 1984;66:1340–1348.
178. Hungerford DS, Barry M. Biomechanics of the patellofemoral joint. *Clin Orthop* 1979;144:9–15.

179. Johnson EW, McLeod TL. Osteochondral fragments of the distal end of the femur fixed with bone pegs: report of two cases. *J Bone Joint Surg Am* 1977;59:677–679.
180. Kennedy JC. *The injured adolescent knee*. Baltimore: Williams & Wilkins, 1979.
181. Lewis PC, Foster BK. Herbert screw fixation of osteochondral fractures about the knee. *Aust N Z J Surg* 1990;60:511–513.
182. McManus F, Rang M, Heslin DJ. Acute dislocation of the patella in children: the natural history. *Clin Orthop* 1979;139:88–91.
183. Nietosvaara Y, Aalto K, Kallio PE. Acute patellar dislocation in children: incidence and associated osteochondral fractures. *J Pediatr Orthop* 1994;14:513–515.
184. Ogden JA. *Skeletal injury in the child*, 2nd ed. Philadelphia: Lea & Febiger, 1989.
185. Rorabeck CH, Bobechko WP. Acute dislocation of the patella with osteochondral fracture: review of 18 cases. *J Bone Joint Surg Br* 1976;58:237–240.
186. Rosenberg NJ. Osteochondral fractures of the lateral femoral condyle. *J Bone Joint Surg Am* 1964;46:1013–1026.
187. Seitz WH Jr, Bibliani LU, Andrews DL, et al. Osteochondritis dissecans of the knee: a surgical approach. *Orthop Rev* 1985;14(2):56–63.
188. Smillie IS. *Injuries of the knee joint*, 5th ed. Edinburgh: Churchill-Livingstone, 1978.
189. Stanitski C, Cannon WD, eds. Patellar instability in the school-age athlete. Instr Course Lect 1998;47:345–350.
190. Wombwell JH, Nunley JA. Compressive fixation of osteochondritis dissecans fragments with Herbert screws. *J Orthop Trauma* 1987;1:74–77.
191. Woo R, Busch M. Management of patellar instability in children. *Oper Tech Sports Med* 1998;6:247–258.

#### Fracture of the Tibial Spine (Intercondylar Eminence)

192. Bakalim G, Wilpulla E. Closed treatment of fracture of the tibial spines. *Injury* 1974;5:210–212.
193. Baxter MP, Wiley JJ. Fractures of the tibial spine in children: an evaluation of knee stability. *J Bone Joint Surg Br* 1988;70:228–230.
194. Berg E. Pediatric tibial eminence fractures: arthroscopic cannulated screw fixation. *Arthroscopy* 1995;11:328–331.
195. Burstein DB, Viola A, Fulkerson JP. Entrapment of the medial meniscus in a fracture of the tibial eminence. *Arthroscopy* 1988;4:47–50.
196. Canale ST. Fractures and dislocations. In: Canale ST, Beaty JH, eds. *Operative pediatric orthopaedics*, 2nd ed. St. Louis: Mosby-Year Book, 1995:947–948.
197. Falstie-Jensen S, Søndergard-Petersen PE. Incarceration of the meniscus in fractures of the intercondylar eminence of the tibia in children. *Injury* 1984;15:236–238.
198. Fyfe IS, Jackson JP. Tibial intercondylar fractures in children: a review of the classification and the treatment of malunion. *Injury* 1981;13:165–169.
199. Garcia A, Neer CS II. Isolated fractures of the intercondylar eminence of the tibia. *Am J Surg* 1958;95:593–598.
200. Goodrich A, Ballard A. Posterior cruciate ligament avulsion associated with ipsilateral femur fracture in a 10-year-old child. *J Trauma* 1988;28:1393–1396.
201. Gronkvist H, Hirsch G, Johansson L. Fracture of the anterior tibial spine in children. *J Pediatr Orthop* 1984;4:465–468.
202. Hayes JM, Masear VR. Avulsion fracture of the tibial eminence associated with severe medial ligamentous injury in an adolescent: a case report and review of the literature. *Am J Sports Med* 1984;12:330–333.
203. Janarv P-M, Westblad P, Johansson C, et al. Long-term follow-up of anterior tibial spine fractures in children. *J Pediatr Orthop* 1995;15:63–68.
204. Keys GW, Walters J. Nonunion of intercondylar eminence fracture of the tibia. *J Trauma* 1988;28:870–871.
205. Lombardo SJ. Avulsion of a fibrous union of the intercondylar eminence of the tibia: a case report. *J Bone Joint Surg Am* 1994;76:1565–1567.
206. Mah JY, Adili A, Otsuka NY, et al. Follow-up study of arthroscopic reduction and fixation of type III tibial eminence fractures. *J Pediatr Orthop* 1998;18:475–477.
207. Mah JY, Otsuka NY, McLean J. An arthroscopic technique for the reduction and fixation of tibial eminence fractures. *J Pediatr Orthop* 1996;16:119–121.
208. McLennan JG. Lessons learned after second-look arthroscopy in type III fractures of the tibial spine. *J Pediatr Orthop* 1995;15:59–62.
209. Meyers MH, McKeever FM. Follow-up notes. fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg Am* 1970;52:1677–1684.
210. Meyers MH, McKeever FM. Fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg Am* 1959;41:209–222.
211. Meyers MH. Isolated avulsion of the tibial attachment of the posterior cruciate ligament of the knee. *J Bone Joint Surg Am* 1975;57:669–672.
212. Mylle J, Reynders R, Broos P. Transepiphyseal fixation of anterior cruciate avulsion in a child: report of a complication and review of the literature. *Arch Orthop Trauma Surg* 1993;112:101–103.
213. Oostvogel HJ, Klasen HJ, Reddingius RE. Fractures of the intercondylar eminence in children and adolescents. *Arch Orthop Trauma Surg* 1988;107:242–247.
214. Pellacci F, Mignani G, Valdiserri L. Fractures of the intercondylar eminence of the tibia in children. *Ital J Orthop Traumatol* 1986;12:441–446.
215. Roberts JM. Fractures of the condyles of the tibia: an anatomical and clinical end-result study of 100 cases. *J Bone Joint Surg Am* 1968;50:1505–1521.
216. Roberts JM, Lovell WW. Fractures of the intercondylar eminence of the tibia. *J Bone Joint Surg Am* 1970;52:827.
217. Robinson SC, Driscoll SE. Simultaneous osteochondral avulsion of the femoral and tibial insertion of the anterior cruciate ligament: report of a case in a 13-year-old boy. *J Bone Joint Surg Am* 1981;63:1342–1343.
218. Ross AC, Chesterman PJ. Isolated avulsion of the tibial attachment of the posterior cruciate ligament in childhood. *J Bone Joint Surg Br* 1986;68:747.
219. Skak SV, Jensen TT, Poulsen TD, et al. Epidemiology of knee injuries in children. *Acta Orthop Scand* 1987;58:78–81.
220. Smith JB. Knee instability after fractures of the intercondylar eminence of the tibia. *J Pediatr Orthop* 1984;4:462–464.
221. Sullivan DJ, Dines DM, Hershon SJ, et al. Natural history of a type III fracture of the intercondylar eminence of the tibia in an adult: a case report. *Am J Sports Med* 1989;17:132–133.
222. Torisu T. Isolated avulsion fracture of the tibial attachment of the posterior cruciate ligament. *J Bone Joint Surg Am* 1977;59:68–72.
223. Wall E. Tibial eminence fractures in children. *Oper Tech Sports Mech* 1998;6:206–212.
224. Wiley JJ, Baxter MP. Tibial spine fractures in children. *Clin Orthop* 1990;255:54–60.
225. Willis RB, Blokker C, Stoll TM, et al. Long-term follow-up of anterior tibial eminence fractures. *J Pediatr Orthop* 1993;13:361–364.
226. Zaricznyj B. Avulsion fracture of the tibial eminence: treatment by open reduction and pinning. *J Bone Joint Surg Am* 1977;59:1111–1114.

#### Ligament Injuries

227. Andrews M, Noyes FR, Barber-Westin SD. Anterior cruciate ligament allograft reconstruction in the skeletally immature athlete. *Am J Sports Med* 1994;22:48–54.
228. Andrews JR, Sanders RA. “Mini-reconstruction” technique in treating anterior lateral rotary instability. *Clin Orthop* 1983;72:93–96.
229. Angel KR, Hall DJ. Anterior cruciate ligament injury in children and adolescents. *Arthroscopy* 1989;5:197–200.
230. Baker RH, Carroll N, Dewar FP, et al. The semitendinosus tenodesis for recurrent dislocation of the patella. *J Bone Joint Surg Br* 1972;54:103–109.
231. Bergstrom R, Gillquist J, Lysholm J, et al. Arthroscopy of the knee in children. *J Pediatr Orthop* 1984;4:542–545.
232. Bertin KC, Goble EM. Ligament injuries associated with physeal fractures about the knee. *Clin Orthop* 1983;177:188–195.
233. Bisson L, Wickiewicz T, Lennson M, et al. ACL reconstruction in children with open physes. *Orthopaedics* 1998;21:659–663.
234. Bradley GW, Shives TC, Samuelson KM. Ligament injuries in the knees of children. *J Bone Joint Surg Am* 1979;61:588–591.
235. Clancy WG, Shelbourne KD, Zoellner GB, et al. Treatment of knee joint instability secondary to rupture of the posterior cruciate ligament: report of a new procedure. *J Bone Joint Surg Am* 1983;65:310–322.
236. Clancy WG, Ray JM, Zoltan DJ. Acute third-degree anterior cruciate ligament injury: a prospective study of conservative nonoperative treatment and operative treatment with repair and patellar tendon augmentation. *Am J Sports Med* 1985;13:435–436(abstr).
237. Clanton TO, DeLee JC, Sanders B, et al. Knee ligament injuries in children. *J Bone Joint Surg Am* 1979;61:1195–1201.
238. Crawford AH. Fractures about the knee in children. *Orthop Clin North Am* 1976;7:639–656.
239. DeHaven KE. Diagnosis of acute knee injuries with hemarthrosis. *Am J Sports Med* 1980;8:9.
240. DeHaven KE, Collins HR. Diagnosis of internal derangement of the knee. *J Bone Joint Surg Am* 1975;57:802–810.
241. DeHaven KE, Linter DM. Athletic injuries: comparison by age, sport and gender. *Am J Sports Med* 1986;14:218–224.
242. DeLee JC, Curtis R. Anterior cruciate ligament insufficiency in children. *Clin Orthop* 1983;172:112–118.
243. Eady JL, Cardenas CD, Sopa D. Avulsion of the femoral attachment of the anterior cruciate ligament in a 7-year-old child. *J Bone Joint Surg Am* 1982;64:1376–1378.
244. Eilert R. Arthroscopy of the knee joint in children. *Orthop Rev* 1976;5(9):61–65.
245. Eiskjaer S, Larsen ST, Schmidt MB. The significance of hemarthrosis of the knee in children. *Arch Orthop Trauma Surg* 1988;107:96–98.
246. Eiskjaer S, Larsen ST. Arthroscopy of the knee in children. *Acta Orthop Scand* 1987;58:273–276.
247. Engebretsen L, Svenningsen S, Benum P. Poor results of anterior cruciate ligament repair in adolescence. *Acta Orthop Scand* 1988;59:684–686.
248. Fetto JF, Marshall JL. The natural history and diagnosis of anterior cruciate ligament insufficiency. *Clin Orthop* 1980;147:29–38.
249. Fetto JF, Marshall JL. Injury to the anterior cruciate ligament producing the pivot-shift sign. *J Bone Joint Surg Am* 1979;61:710–714.
250. Fowler PJ. The classification and early diagnosis of knee joint instability. *Clin Orthop* 1980;147:15–21.
251. Garces GL, Mugica-Garay I, Lopez-Gonzales Coviella N, et al. Growth-plate modifications after drilling. *J Pediatr Orthop* 1994;14:225–228.
252. Graf BK, Lange RH, Fujisaki K, et al. Anterior cruciate ligament tears in skeletally immature patients: meniscal pathology at presentation and after attempted conservative treatment. *Arthroscopy* 1992;8:229–233.
253. Guzzanti V, Falciglia F, Gigante A, et al. The effect of intra-articular ACL reconstruction on the growth plates of rabbits. *J Bone Joint Surg Br* 1994;76:960–963.
254. Holden DL, Jackson DW. Treatment selection in anterior cruciate ligament tears. *Orthop Clin North Am* 1985;16:99–109.
255. Hughston JC, Andrews JR, Cross MJ, et al. Classification of knee ligament instabilities: part I. The medial compartment and cruciate ligaments. *J Bone Joint Surg Am* 1976;58:159–172.
256. Hughston JC, Bowden JA, Andrews JR, et al. Acute tears of the posterior cruciate ligament. *J Bone Joint Surg Am* 1980;62:438–450.
257. Hyndman JC, Brown JC. Major ligamentous injuries of the knee in children. *J Bone Joint Surg Br* 1979;61:245(abstr).
258. Indelicato PA. Nonoperative treatment of complete tears of the medial collateral ligaments of the knee. *J Bone Joint Surg Am* 1983;65:323–329.
259. Janarv PM, Nystrom A, Werner S, et al. Anterior cruciate ligament injuries in skeletally immature patients. *J Pediatr Orthop* 16:673.
260. Jones KG. Reconstruction of the anterior cruciate ligament: a technique using the central one-third of the patellar ligament. *J Bone Joint Surg Am* 1978;45:925–932.

261. Joseph KN, Poggrund H. Traumatic rupture of the medial ligament of the knee in a 4-year-old boy: a case report and review of the literature. *J Bone Joint Surg Am* 1978;60:402–403.
262. Kannus P, Järvinen M. Knee ligament injuries in adolescents: eight-year follow-up of conservative management. *J Bone Joint Surg Br* 1988;70:772–776.
263. Lipscomb AB, Anderson AF. Tears of the anterior cruciate ligament in adolescents. *J Bone Joint Surg Am* 1986;68:19–28.
264. Lo IKY, Fowler PJ, Miniaci A. The outcome of operative treated anterior cruciate disruptions in the skeletally immature child. *Arthroscopy* 1997;13:627–634.
265. Marshall JL, Warren RF, Wickiewicz TL, et al. The anterior cruciate ligament: a technique of repair and reconstruction. *Clin Orthop* 1979;143:97–106.
266. Matz SO, Jackson DW. Anterior cruciate ligament injury in children. *Am J Knee Surg* 1988;1:59–63.
267. Mayer PJ, Micheli LJ. Avulsion of the femoral attachment of the posterior cruciate ligament in an 11-year-old boy: a case report. *J Bone Joint Surg Am* 1979;61:431–432.
268. McCarroll JR, Rettig AC, Shelbourne KD. Anterior cruciate ligament injuries in the young athlete with open physes. *Am J Sports Med* 1988;16:44–47.
269. McCarroll JR, Shelbourne KB, Rettig AC, et al. Patellar tendon graft reconstruction for midsubstance anterior cruciate ligament rupture in junior high school athletes: an algorithm for management. *Am J Sports Med* 1994;22:478–484.
270. McDaniel WJ, Dameron TB. Untreated ruptures of the anterior cruciate ligament. *J Bone Joint Surg Am* 1980;62:696–705.
271. Mizuta H, Kubota K, Shiraishi M, et al. The conservative treatment of complete tears of the anterior cruciate ligament in skeletally immature patients. *J Bone Joint Surg Br* 1995;77:890–894.
272. Morrissy RT, Eubanks RG, Park JP, et al. Arthroscopy of the knee in children. *Clin Orthop* 1982;162:103–107.
273. Nemzek JA, Arnoczky S, Swenson CL. Retroviral transmission by the transplantation of connective tissue allografts. *J Bone Joint Surg Am* 1984;76:1036–1041.
274. Palmer L. On the injuries to the ligaments of the knee: a clinical study. *Acta Chir Scand Suppl* 1938;81:53.
275. Parker AW, Drez D Jr, Cooper JL. Anterior cruciate ligament injuries in patients with open physes. *Am J Sports Med* 1994;22:44–47.
276. Pressman AE, Letts RM, Jarvis JG. Anterior cruciate ligament tears in children: an analysis of operative versus nonoperative treatment. *J Pediatr Orthop* 1997;17:505–511.
277. Ritter G, Neugebauer H. Ligament lesions of the knee in childhood. *Z Kinderchir* 1989;44:94–96.
278. Sanders WE, Wilkins KE, Neidre A. Acute insufficiency of the posterior cruciate ligament in children. *J Bone Joint Surg Am* 1980;62:129–130.
279. Sigge W, Ellebrecht T. Arthroscopy of the injured knee in children. *Z Kinderchir* 1988;43[Suppl 1]:68–70.
280. Singer KM, Henry J. Knee problems in children and adolescents. *Clin Sports Med* 1985;4:385–397.
281. Sisk TD. Knee injuries. In: Crenshaw AH, ed. *Campbell's operative orthopaedics*, 7th ed, vol 3. St. Louis: CV Mosby, 1987:2336–2338.
282. Stadelmanler DM, Arnoczky SP, Dodds J, et al. The effect of drilling and soft tissue grafting across open growth plates: a histologic study. *Am J Sports Med* 1995;23:431–435.
283. Stanitski CL. Anterior cruciate ligament injury in skeletally immature patient: diagnosis and treatment. *J Am Acad Orthop Surg* 1995;3:145–158.
284. Stantiski CL. Correlation of arthroscopic and clinical examinations with magnetic resonance imaging findings of injured knees in children. *Am J Sports Med* 1998;26:2–6.
285. Stanitski CL, Harvell JC, Fu F. Observations on acute knee hemarthrosis in children and adolescents. *J Pediatr Orthop* 1993;13:506–510.
286. Sydnor RW, Andrews JR. Combined arthroscopy and “mini-reconstruction” techniques in the acutely torn anterior cruciate ligament. *Orthop Clin North Am* 1985;16:171–179.
287. Suman RK, Stother IG, Illingworth G. Diagnostic arthroscopy of the knee in children. *J Bone Joint Surg Br* 1984;66:535–537.
288. Waldrop JI, Broussard TS. Disruption of the anterior cruciate ligament in a 3-year-old child. *J Bone Joint Surg Am* 1984;66:1113–1114.
289. Wester W, Canale ST, Dutkowsky JP, et al. Prediction of angular deformity and leg-length discrepancy after anterior cruciate ligament reconstruction in skeletally immature patients. *J Pediatr Orthop* 1994;14:516–521.

#### Knee and Patellar Dislocations and Subluxations

290. Abrams RC. Meniscus lesions in the knee in young children. *J Bone Joint Surg Am* 1957;39:194–195.
291. Betz RR, Longergan R, Patterson R, et al. The percutaneous lateral retinacular release. *Orthopaedics* 1982;5:57–62.
292. Brady TA, Russell D. Interarticular horizontal dislocation of the patella: a case report. *J Bone Joint Surg Am* 1965;47:1393–1396.
293. Cash JD, Hughston JC. Treatment of acute patellar dislocation. *Am J Sports Med* 1988;16:244–249.
294. Cofield RH, Bryan RS. Acute dislocation of the patella: results of conservative treatment. *J Trauma* 1977;17:526–531.
295. Dart CH, Jr, Braitman HE. Popliteal artery injury following fracture or dislocation at the knee: diagnosis and management. *Arch Surg* 1977;112:969–973.
296. DeLee JC. Complete dislocation of the knee in a 9-year-old. *Contemp Orthop* 1979;1:29–32.
297. Desio S, Burks R, Bachus K. Soft tissue restraints to lateral patellar translation in the human knee. *Am J Sports Med* 1998;26: 59–65.
298. Donelson RG, Tomaioli M. Intra-articular dislocation of the patella. *J Bone Joint Surg Am* 1979;61:615–616.
299. Frangakis EK. Intra-articular dislocation of the patella: a case report. *J Bone Joint Surg Am* 1974;56:423–424.
300. Gartland JJ, Brenner JH. Traumatic dislocations in the lower extremity in children. *Orthop Clin North Am* 1976;7:687–700.
301. Goletz TH, Brodhead WT. Intra-articular dislocation of the patella: a case report. *Orthopaedics* 1981;4:1022–1024.
302. Green NE, Allen BL. Vascular injuries associated with dislocation of the knee. *J Bone Joint Surg Am* 1977;59:236–239.
303. Hall JE, Micheli LJ, McManana GB. Semitendinosus tenodesis for recurrent subluxation or dislocation of the patella. *Clin Orthop* 1979;144:31–35.
304. Hejgaard N, Skive L, Perrild C. Recurrent dislocation of the patella. *Acta Orthop Scand* 1980;51:673–678.
305. Kennedy JC. Complete dislocation of the knee joint. *J Bone Joint Surg Am* 1963;45:889–904.
306. Larsen E, Lauridsen F. Conservative treatment of patellar dislocations. *Clin Orthop* 1982;171:131–136.
307. Larson RL. The unstable patella in the adolescent and preadolescent. *Orthop Rev* 1985;14:156–162.
308. Madigan R, Wissinger AH, Donaldson WF. Preliminary experience with a method of quadricepsplasty in recurrent subluxation of the patella. *J Bone Joint Surg Am* 1975;57:600–607.
309. McManus F, Rang M, Heslin DJ. Acute dislocation of the patella in children: a natural history. *Clin Orthop* 1979;139:88–91.
310. Micheli LJ, Stanitski CL. Lateral patellar retinacular release. *Am J Sports Med* 1981;9:330–336.
311. Rorabeck CH, Bobechko WP. Acute dislocation of the patella with osteochondral fracture. *J Bone Joint Surg Br* 1976;58:237–240.
312. Sallay P, Poggi J, Speer K, et al. Acute dislocation of the patella. *Am J Sports Med* 1996;24:52–60.
313. Sisk TD, King LM. Cited by Freeman BL III. Acute dislocations. In: Crenshaw AH, ed. *Campbell's operative orthopaedics*, 7th ed, vol 3. St. Louis: CV Mosby, 1987:2126–2127.

#### Meniscal Injuries

314. Abdou P, Bauer M. Incidence of meniscal lesions in children. *Acta Orthop Scand* 1989;60:710–711.
315. Aichroth PM, Patel DV, Marx CL. Congenital discoid lateral meniscus in children: a follow-up study and evolution of management. *J Bone Joint Surg Br* 1991;73:932–936.
316. Andrich J. The diagnosis and management of meniscus injuries in skeletally immature athlete. *Oper Tech Sports Med* 1998;6:186–196.
317. Appel H. Late results after meniscectomy in the knee joint: a clinical and roentgenologic follow-up investigation. *Acta Orthop Scand Suppl* 1970;133.
318. Arnoczky SP, Warren RF, Spivak JM. Meniscal repair using an exogenous fibrin clot: an experimental study in dogs. *J Bone Joint Surg Am* 1988;70:1209–1217.
319. Baker BE, Peckham AC, Puppato FL, et al. Review of meniscal injury and associated sports. *Am J Sports Med* 1985;13:1–4.
320. Baratz ME, Fu FH, Mengato R. Meniscal tears: the effect of meniscectomy and of repair on intra-articular contact areas and stress in the human knee. *Am J Sports Med* 1986;14:270–275.
321. Bellier G, Dupont J-Y, Larrain M, et al. Lateral discoid menisci in children. *Arthroscopy* 1989;5:52–56.
322. Bhaduri T, Glass A. Meniscectomy in children. *Injury* 1972;3:176–178.
323. Boden S, Davis D, Dina T, et al. A prospective and blinded investigation of magnetic resonance imaging of the knee: abnormal findings in asymptomatic subjects. *Clin Orthop* 1992;282:177–185.
324. Boger DC, Kingston S. MRI of the normal knee. *Am J Knee Surg* 1988;1:99–103.
325. Clark CR, Ogden JA. Development of the menisci of the human knee joint. *J Bone Joint Surg Am* 1983;65:538–547.
326. Dalinka MK, Brennan RE, Canino C. Double-contrast knee arthrography in children. *Clin Orthop* 1977;125:88–93.
327. Dashefsky JH. Discoid lateral meniscus in three members of a family. *J Bone Joint Surg Am* 1971;53:1208–1210.
328. DeHaven KE. Meniscus repair in the athlete. *Clin Orthop* 1985;98:31–35.
329. DeHaven KE. Diagnosis of acute knee injuries with hemarthrosis. *Am J Sports Med* 1980;8:9–14.
330. DeHaven KE, Linter DM. Athletic injuries: comparison by age, sport and gender. *Am J Sports Med* 1986;14:218–224.
331. Dickhaut SC, DeLee JC. The discoid lateral meniscus syndrome. *J Bone Joint Surg Am* 1982;64:1068–1073.
332. Dickason JM, del Pizzo W, Blazina ME, et al. A series of 10 discoid medial menisci. *Clin Orthop* 1982;168:75–79.
333. Fairbank TJ. Knee joint changes after meniscectomy. *J Bone Joint Surg Br* 1948;30:664–670.
334. Fowler PJ. Meniscal lesions in the adolescent: the role of arthroscopy in the management of adolescent knee problems. In: Kennedy JC, ed. *The injured adolescent knee*. Baltimore: Williams & Wilkins, 1979:43–76.
335. Fujikawa K, Iseki F, Mikura Y. Partial resection of the discoid meniscus in the child's knee. *J Bone Joint Surg Br* 1981;63:390–395.
336. Gelb H, Glasgow S, Sapega A, et al. Magnetic resonance imaging of knee disorders: clinical value and cost effectiveness in a sports medicine practice. *Am J Sports Med* 1996;24:99–103.
337. Hamada M, Shino K, Kawano K, et al. Usefulness of MRI for detecting intrasubstance tear and/or degeneration of lateral discoid meniscus. *Arthroscopy* 1994;10:645–653.
338. Hamberg P, Gillquist J, Lysholm J. Suture of new and old peripheral meniscus tears. *J Bone Joint Surg Am* 1983;65:193–197.
339. Harway RA, Handler S. Internal derangement of the knee in an infant. *Contemp Orthop* 1988;17:49–51.
340. Hayashi LK, Yamaga H, Ida K, et al. Arthroscopic meniscectomy for discoid lateral meniscus in children. *J Bone Joint Surg Am* 1988;70:1495–1500.
341. Henning CE, Lynch MA, Clark JR. Vascularity for healing of meniscus repairs. *Arthroscopy* 1987;3:13–18.
342. Huckell JR. Is meniscectomy a benign procedure? A long-term follow-up study. *Can J Surg* 1965;8:254–260.
343. Jackson DW, Jennings LD, Maywood RM, et al. MRI of the knee. *Am J Sports Med* 1988;16:29–38.
344. Johnson RG, Simmons EH. Discoid medial meniscus. *Clin Orthop* 1982;167:176–179.

345. Juhl M, Boe S. Arthroscopy in children, with special emphasis on meniscal lesions. *Injury* 1986;17:171–173.
346. Kaplan EB. Discoid lateral meniscus of the knee joint. nature, mechanism, and operative treatment. *J Bone Joint Surg Am* 1957;39:77–87.
347. Kaplan EB. The embryology of the menisci of the knee joint. *Bull Hosp Joint Dis* 1955;16:111–124.
348. King AG. Meniscal lesions in children and adolescents: a review of the pathology and clinical presentation. *Injury* 1983;15:105–108.
349. King D. The healing of semi-lunar cartilages. *J Bone Joint Surg* 1936;18:333.
350. Krause WR, Pope MH, Johnson RJ, et al. Mechanical changes in the knee after meniscectomy. *J Bone Joint Surg Am* 1976;58:599–604.
351. LaPrade R, Burnett Q, Veenstra M, et al. The prevalence of abnormal magnetic resonance imaging findings in asymptomatic knees: with correlation of magnetic resonance imaging arthroscopic findings in symptomatic knees. *Am J Sports Med* 1994;22:739–745.
352. Manzione M, Pizzutillo PD, Peoples AB, et al. Meniscectomy in children: a long-term follow-up study. *Am J Sports Med* 1983;11:111–115.
353. Marshall SC. Combined arthroscopic/open repair of meniscal injuries. *Contemp Orthop* 1987;14(6):15–24.
354. McGinty JB, Geuss LF, Marvin RA. Partial or total meniscectomy: a comparative analysis. *J Bone Joint Surg Am* 1977;59:763–766.
355. Medlar RC, Mandiberg JJ, Lyne ED. Meniscectomies in children: report of long-term results (mean, 8.3 years) of 26 children. *Am J Sports Med* 1980;8:87–92.
356. Mintzer C, Richmond J, Taylor J. Meniscal repair in the young athlete. *Am J Sports Med* 1998;26:630–633.
357. Morrissy RT, Eubanks RG, Park JP, et al. Arthroscopy of the knee in children. *Clin Orthop* 1982;162:103–107.
358. Nathan PA, Cole SC. Discoid meniscus: a clinical and pathologic study. *Clin Orthop* 1969;64:107–113.
359. Noyes FR, Bassett RW, Grood ES, et al. Arthroscopy in acute traumatic hemarthrosis of the knee: incidence of anterior cruciate tears and other injuries. *J Bone Joint Surg Am* 1980;62:687–695.
360. Pellacci F, Montanari G, Proserpi P, et al. Lateral discoid meniscus: treatment and results. *Arthroscopy* 1992;8:526–530.
361. Polly DW, Callaghan JJ, Sikes RA, et al. The accuracy of selective MRI compared to the findings of arthroscopy of the knee. *J Bone Joint Surg Am* 1988;70:192–198.
362. Rubman M, Noyes F, Barber-Westin S. Arthroscopic repair of meniscal tears that extend into the avascular zone. *Am J Sports Med* 1998;26:87–95.
363. Saddawi ND, Hoffman BK. Tear of the attachment of a normal meniscus of the knee in a 4-year-old child. *J Bone Joint Surg Am* 1970;52:809–811.
364. Schlonsky J, Eyring EJ. Lateral meniscus tears in young children. *Clin Orthop* 1973;97:117–118.
365. Silva I, Silver DM. Tears of the meniscus as revealed by MRI. *J Bone Joint Surg Am* 1988;70:199–202.
366. Sisk TD. Knee injuries. In: Crenshaw AH, ed. *Campbell's operative orthopaedics*, 8th ed, vol 3. St. Louis: Mosby-Year Book, 1992:1504–1532.
367. Sugawara O, Miyatsu M, Yamashita I, et al. Problems with repeated arthroscopic surgery in the discoid meniscus. *Arthroscopy* 7:68–71, 1991.
368. Schwartzberg R, Lemak LJ, Schwartz ML. MRI of the asymptomatic pediatric knee. Personal communication. Presented at the American Academy of Orthopaedic Surgeons annual meeting, New Orleans, 1997.
369. Tapper EM, Hoover NW. Late results after meniscectomy. *J Bone Joint Surg Am* 1969;51:517–526.
370. Takeda Y, Ikata T, Yoshida S, et al. MRI high signal intensity in the menisci of asymptomatic children. *J Bone Joint Surg Br* 1998;80:463–467.
371. Thompson WO, Thaete FL, Fu FH, et al. Tibial meniscal dynamics using three-dimensional reconstruction of magnetic resonance images. *Am J Sports Med* 1991;19:210–216.
372. Vahvanen V, Aalto K. Meniscectomy in children. *Acta Orthop Scand* 1979;50:791–795.
373. Vedi V, Williams A, Tennant SJ, et al. Meniscal movement: an in-vivo study using dynamic MRI. *J Bone Joint Surg Br* 1999;81:37–41.
374. Volk H, Smith FM. "Bucket-handle" tear of the medial meniscus in a 5-year-old boy. *J Bone Joint Surg Am* 1953;35:234–236.
375. Watanabe M, Takeda S, Kieuchi H. *Atlas of arthroscopy*, 3rd ed. Tokyo: Igaku-Shoin, 1979.
376. Woods GW, Whelan JM. Discoid meniscus. *Clin Sports Med* 1990;9:695–706.
377. Yawn BP, Amadio P, Harmsen WS, et al. Isolated acute knee injuries in the general population. *J Trauma* 2000;48:716–723.
378. Zaman M, Leonard MA. Meniscectomy in children: a study of 59 knees. *J Bone Joint Surg Br* 1978;60:436–437.
379. Zobel MS, Borrello JA, Siegel ML, et al. Pediatric knee MR imaging: pattern of injuries in the immature skeleton. *Radiology* 1994;190:397–401.

#### Tibiofibular Dislocations

380. Baci CC, Olaru I, Tudor A. Idiopathic recurrent proximal tibiofibular dislocation in adults: 3 cases treated by an original surgical technique. *Rev Chir Orthop* 1983;69:75–79.
381. Christensen S. Dislocation of the upper end of the fibula. *Acta Orthop Scand* 1966;37:107–109.
382. Crothers OD, Johnson JTH. Isolated acute dislocation of the proximal tibiofibular joint. *J Bone Joint Surg Am* 1973;55:181–183.
383. Dennis JB, Rutledge BA. Bilateral recurrent dislocations of the superior tibiofibular joint with peroneal nerve palsy. *J Bone Joint Surg Am* 1958;40:1146–1148.
384. Falkenberg P, Nygaard H. Isolated anterior dislocation of the proximal tibiofibular joint. *J Bone Joint Surg Br* 1983;65:310–311.
385. Giachino AA. Recurrent dislocations of the proximal tibiofibular joint. *J Bone Joint Surg Am* 1986;68:1104–1106.
386. Ginnerup P, Sorensen VK. Isolated traumatic luxation of the head of the fibula. *Acta Orthop Scand* 1978;49:618–620.
387. Odgen JA. Subluxation and dislocation of the proximal tibiofibular joint. *J Bone Joint Surg Am* 1974;56:145–154.
388. Odgen JA. Subluxation of the proximal tibiofibular joint. *Clin Orthop* 1974;101:192–197.
389. Owen R. Recurrent dislocation of the superior tibiofibular joint: a diagnostic pitfall in knee joint derangement. *J Bone Joint Surg Br* 1968;50:342–345.
390. Parkes JC II, Zelko RR. Isolated acute dislocation of the proximal tibiofibular joint: case report. *J Bone Joint Surg Am* 1973;55:177–180.
391. Shapiro GS, Fanton GS, Dillingham MF. Reconstruction for recurrent dislocation of the proximal tibiofibular joint: a new technique. *Orthop Rev* 1993;22:1229–1232.
392. Shelbourne KD, Pierce RO, Ritter MA. Superior dislocation of the fibular head associated with a tibia fracture. *Clin Orthop* 1982;160:172–174.
393. Sijbrandij S. Instability of the proximal tibiofibular joint. *Acta Orthop Scand* 1978;49:621–626.
394. Weinert CR, Raczka R. Recurrent dislocation of the superior tibiofibular joint. *J Bone Joint Surg Am* 1986;68:126–128.
395. Wong K, Weiner DS. Proximal tibiofibular synostosis. *Clin Orthop* 1978;135:45–47.

## FRACTURES OF THE SHAFT OF THE TIBIA AND FIBULA

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### EPIDEMIOLOGY

Tibial and fibular fractures are the third most common pediatric long bone injuries (15%) after femoral and radial/ulnar fractures (11,12). The overall prevalence of tibial fractures in both boys and girls has increased since 1950 (Table 24-1) (7). The average age of occurrence is 8 years, and the frequency of occurrence does not change significantly with age (6). Seventy percent of pediatric tibial fractures are isolated injuries; ipsilateral fibular fractures occur with 30% (4,12,13). Fifty percent to 70% of tibial fractures occur in the distal third, and 19% to 39% in the middle third. The least commonly affected portion of the tibia is the proximal third, yet these may be most problematic. Thirty-five percent of pediatric tibial fractures are oblique, 32% comminuted, 20% transverse, and 13% spiral (11). Tibial fractures in children under 4 years of age usually are isolated spiral, sharply oblique fractures of the distal and middle thirds. Most tibial fractures in older children and adolescents are at the ankle. Rotational forces produce an oblique or a spiral fracture and are responsible for approximately 81% of all tibial fractures without fibular fractures (2,4,5,10,11). Bicycle spoke injuries are common among children 1 to 4 years of age, whereas most tibial fractures in children 4 to 14 years of age occur in sporting or traffic accidents (2,4,6,7,10,11). Over 50% of combined tibial and fibular fractures result from vehicular trauma. In contrast to the rotational etiology of most tibial fractures, most isolated fibular fractures result from a direct blow (6,11).

	Years	
	1950-1955	1980-1983
Female		
0-9 years old	6.6	7.2
10-19 years old	3.8	4.9
Male		
0-9 years old	10.1	14.3
10-19 years old	7.1	10.7

<sup>a</sup> Per 10,000 population.  
 Reprinted from Bengner V, Ekholm T, Johnell O, et al. Incidence of femoral and tibial shaft fractures. *Acta Orthop Scand* 1990;51:251; with permission

TABLE 24-1. CHANGE IN THE PREVALENCE OF PEDIATRIC TIBIAL FRACTURES<sup>a</sup>

The tibia is the second most commonly fractured bone in abused children. Approximately 26% of all abused children with a fracture have an injured tibia (9).

Approximately 9% of pediatric tibial fractures are open. Concomitant fractures of the ankle and foot are the most common injuries associated with fractures of the tibia and fibula, followed by humeral, femoral, and radial/ulnar fractures (3). The average index severity score of a child with a tibial fracture is 10 (range 0-45). The average hospital stay is 6½ days (range 1-50). The average hospital cost for a pediatric tibial fracture is \$7,156.00 (range \$488.00 to \$88,706.00) (3). The cost variation depends on (a) associated injuries, (b) open fracture, and (c) need for surgical management.

### Surgical Anatomy

#### Bony Structure

The tibia ("flute") is the second largest bone in the body. At the proximal aspect of the tibia are two concave condyles: the medial condyle is larger, deeper, and narrower than the lateral condyle. An elevated process, the tibial tubercle, located between the two condyles is the site of attachment of the patellar tendon. The shaft of the tibia is prismoid, with a broad proximal extent that decreases in size until the distal third, where it gradually increases in size. The tibial crest is prominent medially from the tibial tubercle to the tibial plafond and is subcutaneous without any overlying muscles (5).

The tibia develops from three ossification centers: one in the shaft and one in each epiphysis. The tibial diaphysis ossifies at 7 weeks of gestation and expands both proximally and distally. The proximal epiphyseal center appears shortly after birth and unites with the shaft at approximately 16 years of age. The distal epiphyseal ossification center appears in the second year of life, and the distal tibial physis closes at approximately 15 years of age. Two other centers may occur occasionally: in the medial malleolus and in the tibial tubercle (5).

The tibia articulates with the condyles of the femur proximally, with the fibula at the knee and the ankle, and with the talus distally (5). Twelve muscles have either their origin or insertion on the tibia (Table 24-2).

Semimembranosus	Inserts on inner tuberosity of the proximal tibia
Tibialis anterior, extensor digitorum longus,iceps femoris	Attaches to the lateral condyle of the tibia
Sartorius, gracilis, semitendinosus	Inserts on the proximal medial surface of the tibial metaphysis
Tibialis anterior	Arises on the lateral surface of the tibial diaphysis
Popliteus, soleus, flexor digitorum longus, tibialis posterior	Attach to the posterior diaphysis of the tibia
Patellar tendon	Inserts into the tibial tubercle
Secondary slip of fascia lata	Occasionally inserts into the tibial tubercle
Tensor fascia lata	Attaches to Gerdy's tubercle the lateral aspect of the proximal tibial metaphysis

Reprinted from Gray H. *Anatomy: descriptive and surgical*. In: Fick TE, Hoadley R, eds. *Anatomy: descriptive and surgical*. New York: Bourne Books, 1977:112; with permission.

**TABLE 24-2. MUSCLE ORIGINS AND INSERTIONS ON THE TIBIA**

The fibula articulates with the tibia and the talus. The fibular diaphysis ossifies at about 8 weeks of gestation. The distal epiphysis is visible at 2 years of age, and the proximal secondary ossification center at 4 years. The distal fibular physis closes at about 16 years; the proximal physis closes later, at age 16 to 18 years ( 5). Nine muscles have either their origin or insertion on the fibula ( Table 24-3) (5).

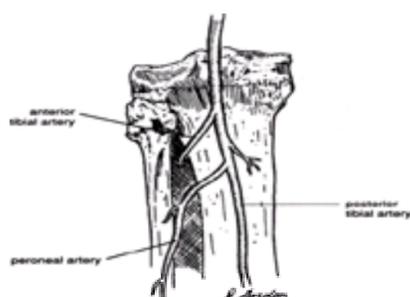
Soleus, flexor hallucis longus	Arise from the posterior aspect of the diaphysis
Peroneus longus, peroneus brevis	Arise from the lateral aspect of the fibular diaphysis
Biceps femoris, soleus peroneus longus	Attach to the head of the fibula
Extensor digitorum longus, peroneus tertius	Attach to the anterior surface of the fibular shaft
Extensor hallucis longus	
Tibialis posterior	Arises from the medial aspect of the fibular diaphysis

Reprinted from Buckley SL, Gotschall C, Robertson W, et al. The relationship of skeletal injuries with trauma score, injury severity score, length of hospital stay, hospital charges and mortality in children admitted to a regional pediatric trauma center. *J Pediatr Orthop* 1994;14:449; with permission.

**TABLE 24-3. MUSCLE ORIGINS AND INSERTIONS ON THE FIBULA**

### Vascular Anatomy

The popliteal artery descends vertically between the condyles of the femur and passes between the two heads of the gastrocnemius muscle. It ends at the distal border of the popliteus muscle, where it divides into the anterior and posterior tibial arteries. The anterior tibial artery passes between the tibia and the fibula over the proximal aspect of the intraosseous membrane. The posterior tibial artery divides several centimeters distal to this point, leading to the peroneal artery ( Fig. 24-1) (5).



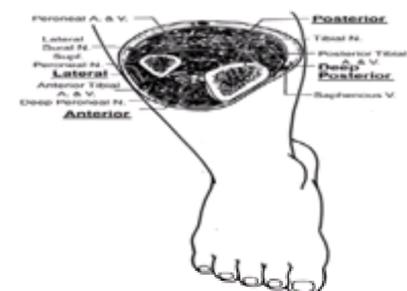
**FIGURE 24-1.** Vascular anatomy of the proximal tibia.

### Nerves

The posterior tibial nerve runs adjacent and posterior to the popliteal artery through the popliteal fossa. The common peroneal nerve passes around the proximal neck of the fibula. It divides into the deep and superficial branches, passing into the anterior and the lateral compartments of the lower leg ( 5), respectively. Each branch innervates the muscles within its compartment. The deep peroneal nerve provides sensation to the first web space, whereas the superficial branch has a sensor representation across the dorsal surface of the foot.

### Fascial Compartments

The lower leg has four fascial compartments (Fig. 24-2). The anterior compartment contains the extensor digitorum longus, the extensor hallucis longus, and the tibialis anterior muscles; the anterior tibial artery and deep peroneal nerve run in this compartment. The lateral compartment contains the peroneus longus and brevis muscles. The superficial posterior compartment contains the soleus and gastrocnemius muscles. The deep posterior compartment contains the flexor digitorum longus, the flexor hallucis longus, and the tibialis posterior muscles. The posterior tibial artery, peroneal artery, and tibial nerve run in this compartment ( 5).



**FIGURE 24-2.** Fibrous compartments of the leg. (Reprinted from Sharps CH, Cardea JA. Fractures in the shaft of the tibia and fibula. In: MacEwen GD, Kasser JR, Heinrich SD, eds. *Pediatric fractures: a practical approach to assessment and treatment*. Baltimore: Williams & Wilkins, 1993:318; with permission.)

## CLASSIFICATION

Nonphyseal injuries of the tibia and the fibula can be classified into three major categories based on the combination of bones fractured and the location of the injuries ([Table 24-4](#)).

**TABLE 24-4. CLASSIFICATION: NONPHYSEAL FRACTURES OF THE PEDIATRIC TIBIA/FIBULA**

### Fractures of the Proximal Tibial Metaphysis

The peak incidence for proximal tibial metaphyseal fractures is between the ages of 3 and 6 years. The most common mechanism of injury is a force applied to the lateral aspect of the extended knee. The cortex of the medial tibial metaphysis fails in tension, often resulting in an incomplete fracture. The fibula ordinarily escapes injury, although plastic deformation may occur ([14,15,16,17,21,22,23,24,27,32,33,34,35,36,37](#) and [38](#)).

Children with proximal tibial metaphyseal fractures present with pain, swelling, and tenderness in the region of the fracture. Motion of the knee causes moderate pain, and the child will not walk. Crepitance is seldom identified on physical examination, especially if the fracture is incomplete ([14,15,16,17,21,22,23,24,27,32,33,35,36,37](#) and [38](#)).

Radiographs usually show a complete or incomplete fracture of the proximal tibial metaphysis. The medial aspect of the fracture often is open, producing a valgus deformity ([14,15,16,17,21,22,23,32,33,35,36,37](#) and [38](#)).

### Valgus Deformity

The most common sequelae resulting of proximal tibial metaphyseal fractures is valgus deformity ([Fig. 24-3](#)). In 1953, Cozen ([17](#)) reported four patients with valgus deformities after fractures of the proximal tibial metaphysis. Since that time, many other investigators ([12,15,16,21,22,23,24,25,27,35,36](#)) have reported tibia valga after proximal tibial metaphyseal fractures.



**FIGURE 24-3. A:** Anteroposterior and lateral radiographs of the proximal tibia metaphyseal fracture with an intact fibula in a 3-year-old child. **B:** Anteroposterior and lateral radiographs in the initial long leg cast demonstrate an acceptable alignment. **C:** Posttraumatic tibia valga is present 1 year after fracture union. (Reprinted from Sharps CH, Cardea JA. Fractures in the shaft of the tibia and fibula. In: MacEwen GD, Kasser JR, Heinrich SD, eds. *Pediatric fractures: a practical approach to assessment and treatment*. Baltimore: Williams & Wilkins, 1993:321; with permission.)

Many theories have been proposed to explain the development of a valgus deformity after a proximal metaphyseal fracture ([Table 24-5](#)). Proximal tibia valga can be caused by a poor reduction, particularly if the knee is immobilized in flexion, because the flexed position of the knee makes it difficult to determine the mechanical axis of the leg on radiographs ([33,37](#)). Lehner and Dabas suggested that an expanding medial callus produced a valgus deformity ([25](#)), whereas Goff ([20](#)) and Keret et al. ([24](#)) believed that the lateral aspect of the proximal tibial physis was injured (Salter Harris type V fracture), resulting in asymmetric growth of the proximal tibial physis. Taylor ([23](#)) believed that the valgus deformity was secondary to postfracture stimulation of the tibial physis without a corresponding stimulation of the fibular physis. Pellen ([28](#)) suggested that premature weight bearing produced an angular deformity of the fracture before union. Rooker and Salter ([31](#)) believed that the periosteum was trapped in the medial aspect of the fracture, producing an increase in medial physeal growth and a developmental valgus deformity.

Asymmetric activity of medial portion of proximal tibial physis (overgrowth) ([14,21,23,25,27,30,35,37](#))  
Tethering effect, fibula ([8,22,35](#))  
Inadequate reduction ([29](#))  
Interposed soft tissue (pes anserinus) ([36](#)); medial collateral ligament ([16](#))  
Loss of tethering effect of the pes anserinus ([23](#))  
Early weight bearing producing developmental valgus ([28](#))  
Physeal arrest of the lateral aspect of the proximal tibial physis

**TABLE 24-5. PROPOSED ETIOLOGIES OF TRAUMA INDUCED TIBIA VALGUS**

A recently suggested etiology for the valgus deformity that develops after a proximal tibial fracture in a child relates the deformity to a concomitant injury to the pes anserinus tendon plate. The pes anserinus tethers the medial aspect of the physis just as the fibula tethers the lateral aspect of the proximal tibial physis. The proximal tibial fracture disrupts the pes anserinus tendon plate, producing a loss of the tethering effect and leading to medial physeal overgrowth and a hemichondrodiastasis ([15,18,19,36,38](#)). Removal from the fracture and repair of the folded periosteum that forms the foundation of the plate has been suggested to decrease the risk of recurrence of the valgus deformity.

Another theory postulates that the progressive valgus deformity occurs because vascular flow to the proximal tibial physis increases after fracture, producing an asymmetric physeal response that causes increased medial growth ([23](#)). Support for this theory includes quantitative bone scans performed months after proximal tibial metaphyseal fractures that have shown increased tracer uptake in the medial aspect of the physis compared with the lateral aspect ([37](#)). Developmental tibia valga has been reported to occur after simple excision of a bone graft from the proximal tibial metaphysis ([35](#)), tibial osteotomy ([14,22](#)), and osteomyelitis of the proximal tibial metaphysis ([14,35](#)). Ogden identified an increase in the collateral geniculate vascularity to the medial proximal tibia in a cadaver angiography study of a

5-year-old child. This also supports the theory that medial overgrowth occurs secondary to an increase in the blood flow supplying the medial aspect of the proximal tibia following injury (26). The overgrowth of the tibia that occurs in many children with tibial valgus deformity after fracture is further evidence of an abnormality in the response of the proximal tibial physis to fracture (22,35). Tibia valga deformity can occur after nondisplaced fracture and can recur after corrective tibial osteotomy (Fig. 24-4), further supporting the premise that asymmetric physeal growth is the cause of most posttraumatic tibia valga deformities (37). Robert et al. (30), in an analysis of 25 patients with proximal tibial fractures, identified three groups of patients. Twelve children with greenstick or complete fractures developed valgus deformities, which were progressive in 11. No child with a torus fracture developed a valgus deformity. In 3 children, altered growth at the distal tibial physis compensated for the compromised tibia valga; in 4 children, corrective osteotomies were performed. The valgus deformity recurred in 2 of these 4 children, and two had iatrogenic compartment syndromes. This study supports the recommendation that developmental tibia valga should not be corrected until the child reaches puberty. At that point, a proximal tibial medial epiphysiodesis can be performed to allow the tibia to correct slowly (2,27,30,34,35).



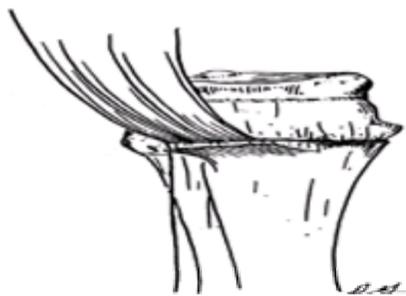
**FIGURE 24-4.** Developmental valgus after a proximal tibial metaphyseal fracture and a subsequent corrective osteotomy. **A:** Radiograph taken 6 months after a fracture of the proximal and distal tibia in a 5-year-old girl. The proximal tibial injury was nondisplaced. The scar from the initial proximal metaphyseal fracture is still seen (*arrow*). This child developed a moderate valgus deformity of the tibia within 6 months of fracture. **B:** A proximal tibial corrective osteotomy was performed. **C:** Two months postoperatively the osteotomy was healed and the deformity corrected. **D:** Five months later there was a recurrent valgus deformity of 13 degrees. (Courtesy of John J.J. Gugenheim, M.D.).

Support for a “wait and see” attitude also was provided by Zionts and MacEwen (38), who followed for an average of 39 months seven children with progressive valgus deformities of the tibia after metaphyseal fractures. Most of the deformity occurred during the first year after injury. The tibia continued to deform at a slower rate for up to 17 months after injury. Six of their seven patients had spontaneous clinical corrections. At follow-up, all children had less than a 10 degree deformity.

### Treatment

Nondisplaced proximal tibial metaphyseal fractures are stabilized with a long leg cast with the knee in nearly full extension and a varus mold.

Displaced proximal tibial fractures require closed reduction with general anesthesia in the operating room, with fluoroscopy to verify an anatomic reduction. If closed reduction to an anatomic position or slight varus cannot be obtained, an open reduction is indicated. Interposed soft tissue is removed from the fracture ( Fig. 24-5), and the pes anserinus plate is repaired if ruptured. The child is placed into a long leg, straight knee cast after reduction, and the fracture is checked once again. Frequent follow-up visits are required to verify maintenance of the reduction. The cast is removed approximately 6 weeks after injury. The child may return to normal activities after recovery of normal knee and ankle range of motion. Long-term follow up with a warning to the family of possible growth abnormality is mandatory.

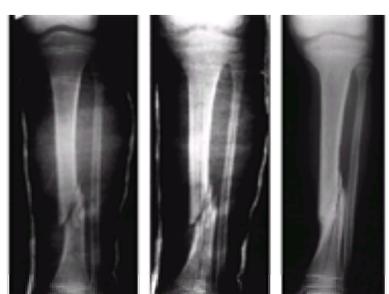


**FIGURE 24-5.** Proximal tibial metaphyseal fracture with interposition of both the pes anserinus fibers and the periosteum into the fracture medially.

A child with a posttraumatic valgus deformity is followed until spontaneous correction occurs or until the child is old enough for surgical intervention. Tibial osteotomies rarely are indicated and generally should be delayed until adolescence. A well-timed proximal tibial medial epiphysiodesis can provide comparable results without many of the risks of osteotomy (34). Orthotic devices do not alter the natural history of posttraumatic tibia valga and are not recommended. However, because the valgus deformity usually is associated with overgrowth, a contralateral shoe lift of appropriate size may make the deformity less apparent.

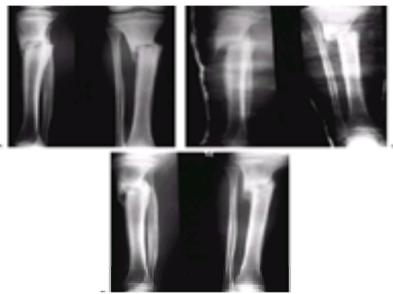
### DIAPHYSEAL FRACTURES OF THE TIBIA AND FIBULA

Seventy percent of pediatric tibial fractures are isolated injuries ( 11,13,101). The fractures can be incomplete (torus, greenstick) or complete. Most tibial fractures in children under 11 years of age are caused by a torsional force and are located in the distal third of the tibia. These oblique or spiral fractures occur when the body rotates with the foot in a fixed position on the ground. The fracture line starts in the distal anteromedial aspect of the bone and propagates proximally in a posterolateral direction. Although the intact fibula prevents significant shortening of the tibia, varus angulation develops in approximately 60% of isolated tibial fractures within the first 2 weeks after injury ( 101) (Fig. 24-6) because the force of contraction of the long flexor muscles of the lower leg are converted into an angular moment by the intact fibula (Fig. 24-7). Isolated transverse and comminuted fractures of the tibia are caused by direct trauma. Transverse fractures of the tibia with an intact fibula seldom displace (8,46). Comminuted fractures drift into varus alignment like oblique and spiral fractures ( 8,46,101).



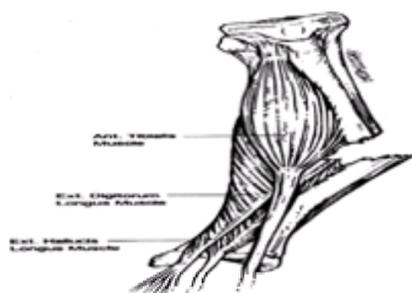
**FIGURE 24-6.** Anteroposterior radiograph of a distal one-third tibia fracture without a concomitant fibula fracture in a 10-year-old child. **A:** The alignment in the coronal plane is acceptable (note that the proximal and distal tibial growth plates are parallel). **B:** A varus angulation developed within the first 2 weeks after injury. **C:**

At radiographic union, a 10 degree varus angulation is present.



**FIGURE 24-7.** **A:** Fracture of the proximal third of the tibia without a concomitant fibular fracture. Note the valgus angulation of the tibia fracture. **B:** The valgus malalignment spontaneously corrected into neutral alignment. **C:** The fracture united with a mild posterior and medial translation without an angular deformity.

Approximately 30% of pediatric tibial diaphyseal fractures have associated fibular fractures ( 11,13,101). Plastic deformation of the fibula may produce valgus malalignment of the tibia. The fibular injury must be identified to prevent recurrence of the malalignment after reduction. An associated complete fracture of the fibula usually results in valgus malalignment because of the action of the muscles in the anterolateral aspect of the leg ( Fig. 24-8).



**FIGURE 24-8.** The muscles in the anterior and the lateral compartments of the lower leg produce a valgus deformity in complete ipsilateral tibia and fibula fractures.

An isolated fracture of the fibular shaft is rare in children and normally results from a direct blow to the lateral aspect of the leg ( Fig. 24-9). Most isolated fractures of the fibular shaft are nondisplaced and heal quickly with symptomatic care. Compartment syndrome may accompany this injury, and observation for this associated problem is mandatory.



**FIGURE 24-9.** **A:** Plastic deformation of fibula secondary to a direct blow to the lateral aspect of the leg ( *closed arrows*). Note medial nondisplaced fracture of the tibia. **B:** The tibia fracture displaced in the cast a week later from the force exerted by the plastically deformed fibula.

### Signs and Symptoms

The signs and symptoms associated with tibial and fibular diaphyseal fractures vary with the severity of the injury and the mechanism by which it was produced. Pain is the most common symptom. An isolated fibular fracture normally produces mild pain, whereas tibial fractures produce more severe pain. Children with stress fractures of the tibia or fibula complain of pain on weight bearing, but no pain at rest.

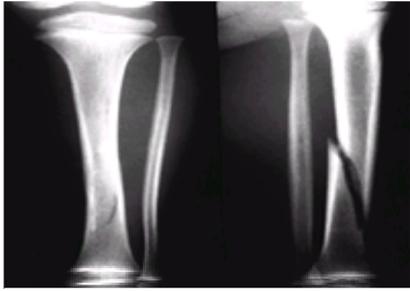
Children with fractures of the tibia or fibula have edema at the fracture site, and the area is tender to palpation. Young children with nondisplaced fractures may refuse to walk. If there is significant injury to the periosteum, a bony defect may be present to palpation in patients with a complete fracture. Neurologic impairment secondary to the force that created the fracture is rare except with fibular neck fractures caused by direct trauma.

Although arterial disruption is uncommon in pediatric tibial and fibular diaphyseal fractures, both the dorsalis pedis and the posterior tibial pulses should be checked, and a Doppler examination should be performed if they are not palpable. Capillary filling, sensation, and pain response patterns also should be monitored.

Concomitant soft tissue injuries must be evaluated carefully. Open fractures must be treated aggressively to reduce the risk of late complications.

### Radiographic Evaluation

Anteroposterior and lateral radiographs that include the knee and ankle joints ( Fig. 24-10) should be taken whenever a tibial or fibular shaft fracture is suspected. Comparison views of the uninvolved leg normally are not necessary. Children with suspected fractures not apparent on the initial radiographs may need to be treated with supportive casting to control the symptoms associated with the injuries. Technetium radionuclide scans obtained at least 3 days after injury are useful to identify fractures that are unapparent radiographically.



**FIGURE 24-10. A:** Spiral fracture of the distal tibia. The fracture is difficult to identify on the anteroposterior radiograph. **B:** The fracture is easily identified on the lateral radiograph.

## Treatment

### Cast Immobilization

Pediatric tibial and fibular shaft fractures usually are uncomplicated and can be treated by a simple manipulation and cast application ( 67). Isolated fractures of the tibial shaft often develop a varus malalignment. Valgus angulation and shortening can present a significant problem in children who have complete fractures of both the tibia and the fibula (Fig. 24-11). A recurvatum deformity also can develop.



**FIGURE 24-11. A:** Complete fracture of the tibia and the fibula with a valgus deformity and shortening. **B:** The fracture was closed reduced. **C:** The valgus deformity increased after 2 weeks. **D:** The cast was wedged. **E:** Radiograph 6 weeks after injury shows a good alignment and callus formation.

Displaced fractures are reduced in the operating room to allow painless manipulation with muscle relaxation and the use of fluoroscopy to guide the reduction. The child's ankle initially may be left in some plantar flexion (20 degrees for fractures of the middle and distal thirds, 10 degrees for fractures of the proximal third) to prevent posterior angulation at the fracture.

The fracture is evaluated under fluoroscopy before cast application to determine the most appropriate way to manipulate the bone to achieve an acceptable reduction. A short leg cast is applied with the foot in appropriate position and with either a varus or valgus mold. The cast material is taken to the inferior aspect of the patella anteriorly and to a point 2 cm distal to the popliteal flexion crease posteriorly. Plaster is used for the initial cast because of its ability to mold to the contour of the leg and the ease with which it can be manipulated while setting. The alignment of the fracture is rechecked after the short leg cast has been applied. The cast is then extended to the mid-thigh with the knee flexed. Most children with complete unstable diaphyseal tibial fractures are placed into a bent knee (45 degrees) long leg cast to control rotation at the fracture site and assist in keeping the child non-weight bearing during the initial healing phase.

The alignment of the fracture must be checked closely during the first 3 weeks after the cast has been applied, when muscle atrophy and a reduction in tissue edema may lead to a loss of fracture alignment. Some children require a second cast application with remanipulation of the fracture under general anesthesia several weeks after injury. Acceptable position is somewhat controversial. Remodeling of angular deformity is limited. No absolute number can be given, but the following general principles may be beneficial in decision making.

1. Varus and valgus deformity in the upper and mid-shaft tibia remodel slowly if at all. Up to 5 degrees of deformity can be accepted, but not more than 10 degrees.
2. Translation of the entire shaft of the tibia in a young child is satisfactory, whereas in an adolescent, 50% apposition should be obtained.
3. Up to 10 degrees of posterior angulation may be tolerated, although remodeling is slow
4. No more than 1 cm is acceptable, because overgrowth is minimal.

### Wedging of a Cast

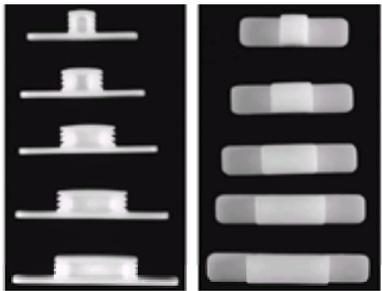
Occasionally, loss of fracture reduction requires the cast to be "wedged." The fracture alignment in the cast can be changed by creating a closing wedge, an opening wedge, or a combination of wedges. The location for the wedge manipulation is determined by evaluating the child's leg under fluoroscopy and marking the mid-point of the tibial fracture on the outside of the cast. If fluoroscopy is not available, a series of paper clips are placed at 2-cm intervals on the cast. Anteroposterior and lateral radiographs are then taken. The paper clips define the location of the fracture and the location of the cast manipulation.

### Closing Wedge Technique

A wedge of cast material encompassing 90% of the circumference of the leg with its base over the apex of the fracture is removed. The cast is left intact opposite the apex of the fracture. The edges of the cast are brought together to correct the angulation at the fracture. This wedging technique produces mild fracture shortening and may pinch the skin at the location the wedge is removed. It also may increase compartment pressures because the volume of the cast is reduced.

### Opening Wedge Technique

The side of the cast opposite the apex of the fracture is cut perpendicular to the long axis of the bone. A small segment of the cast is left intact directly over the apex of the malaligned tibia (approximately 25%). A cast spreader is used to "jack" the cast open. A plastic block ( Fig. 24-12) of the appropriate size is placed into the open segment and the cast is wrapped with new casting material after the alignment has been checked radiographically. This wedging technique lengthens the tibia while correcting the malalignment (Fig. 24-13).



**FIGURE 24-12. A and B:** Blocks used to hold casts open after wedge corrections of malaligned fractures. The wings on the blocks prevent the blocks from migrating toward the skin.



**FIGURE 24-13. A:** Anteroposterior and lateral tibial radiographs of an 11-year-old boy who was struck by an automobile sustaining a markedly comminuted tibia fracture without a concomitant fibula fracture. **B:** Despite the comminution, length and alignment were maintained in a cast. **C:** The patient's fracture shifted into a varus malalignment that measured 10 degrees (**right**). The cast was wedged, resulting in the reestablishment of an acceptable coronal alignment (**left**). **D:** The patient's fracture healed without malunion.

### Combination Technique

Approximately 45% of the cast opposite the apex of the malaligned fracture is cut perpendicular to the shaft of the tibia. Two vertical cuts separated by approximately 0.5 cm are made 90 degrees from the first cut in both directions directly over the fracture. A wedge of casting material is removed from the apex side of the malaligned fracture, and the cast opposite the apex of the fracture is opened. This closes the defect in the cast over the apex of the fracture and produces a change in the angular alignment of the bone without a significant change in the length of the bone.

### Operative Treatment

Unstable fractures of the tibia and fibula may require operative reduction and surgical stabilization. Common methods of fixation include percutaneous pins, bioabsorbable pins (42), external fixation, and plates with screws. Intramedullary nails are seldom indicated.

Operative treatment is rarely needed for tibial fractures in children. Weber et al. (13) reported that only 29 (4.5%) of 638 pediatric tibial fractures required surgical intervention. The indications for operative treatment include open fractures, fractures in children with spasticity (head injury or cerebral palsy), and fractures in which open treatment facilitates nursing care (floating knee, multiple long bone fractures, multiple system injuries) (40,44,52,54,57,60,64,70,72,77,80,81,87,92,100).

Children with stable fractures or minimally displaced fractures should be treated conservatively whenever possible. Comminuted tibial fractures, unreducible fractures, fractures that cannot be maintained in a reduced position, fractures associated with a compartment syndrome, grade II or III open injuries (Fig. 24-13), or fractures associated with multiple system injuries should be treated with operative stabilization (40,44,52,54,57,60,64,70,72,77,80,81,87,92,100).

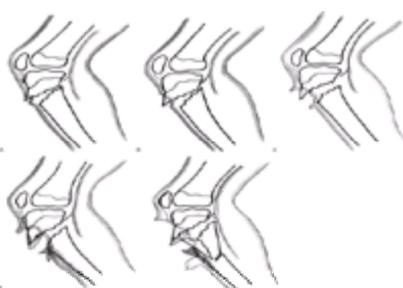
### Open Fractures

Open tibial fractures in children are treated in the same manner as comparable injuries in adults, and they are classified by the Gustillo and Anderson System (61) (Table 24-6 and Fig. 24-14). Most open fractures of the tibia result from high-velocity injuries.

Grade I
Low-energy wound, < 1 cm long
Bone piercing skin from inside/out
Minimal muscle damage
Grade II
Wound > 1 cm in length
Moderate soft tissue injury
Grade III
High-energy wound
Usually > 10 cm
Extensive muscle devitalization
Bone widely displaced or comminuted
Special cases
Shotgun wound
High-velocity gunshot (> 2,000 ft/s)
Segmental fracture
Segmental diaphyseal loss
Farmyard environment
Associated vascular injury

Reprinted from Gustillo RB, Anderson JT. Prevention of infection in the treatment of one-thousand and twenty-five fractures of long bones. J Bone Joint Surg [Am] 1976;58:453, with permission.

**TABLE 24-6. CLASSIFICATION OF OPEN FRACTURES**



**FIGURE 24-14.** Gustillo and Anderson classification of open fractures. **A:** Grade I. The skin wound measures less than 1 cm long, usually from within, with little or no skin contusion. **B:** Grade II. The skin wound measures more than 1 cm long, with skin and soft tissue contusion but no loss of muscle or bone. **C:** Grade IIIA. There is

a large severe skin wound with extensive soft tissue contusion, muscle crushing or loss, and severe periosteal stripping. **D:** Grade IIIB. Like grade IIIA but with bone loss and nerve or tendon injury. **E:** Grade IIIC. Like grade IIIA or B with associated vascular injury. (Reprinted from Alonso JE. The initial management of the injured child: musculoskeletal injuries. In: MacEwen GD, Kasser J, Heinrich SD, eds. *Pediatric fractures: a practical approach to assessment and treatment*. Baltimore: Williams & Wilkins, 1993:32; with permission.)

### Treatment Principles

Edwards outlined management principles for high-grade open fractures in 1983 (56):

- Rapid debridement, irrigation, and antibiotic therapy
- Fracture reduction followed by application a rigid fixation device
- Intraoperative angiography and compartment pressure studies when sufficiency of the vascular profusion is unclear or compartments tight
- Open wound treatment with loose gauze packing
- Postoperative suspension of the leg
- Staged debridement of necrotic soft tissue and bone in the operating room every 24 to 72 hours until a good granulation base develops
- Delayed closure or application of a split-thickness skin graft when necessary; delayed myocutaneous flap as needed
- Closed cancellous bone grafting for bone defects or delayed union
- Removal of external fixation device followed by application of a weight-bearing cast in compressible fractures after successful soft tissue coverage
- Continued external fixation and grafting for noncompressible fractures

The principles of treatment for open tibial fractures in adults have been modified by the unique characteristics of the pediatric skeleton. These differences include the following (40,52,60,95):

- Soft tissue injuries heal better in children than in adults.
- Devitalized uncontaminated bone that can be covered with soft tissue will incorporate into the fracture callus.
- External fixation can be maintained, when necessary, until fracture consolidation.
- Periosteum can reform bone even with segmental bone loss in younger children.
- Some uncontaminated grade I open wounds can be primarily closed.

Buckley et al. (47) reported on 41 children with 42 open fractures of the tibia (18 grade II, 6 grade IIIA, 4 grade IIIB, and 2 grade IIIC). Twenty-two (52%) of the fractures were comminuted. All wounds were irrigated and debrided, and antibiotics were administered for at least 48 hours. Twenty-two fractures were treated with reduction and cast application, and 20 with external fixation. Three children had early infections, and one of them developed late osteomyelitis. All infections had resolved at last follow-up. The average time to union was 5 months (range 2–21 months). The time to union was directly proportional to the severity of the soft tissue injury. Fracture pattern also had an effect on time to union. Segmental bone loss, infection, and the use of an external fixation device were associated with delayed union. Four angular malunions of more than 10 degrees occurred, three of which spontaneously corrected. Four children had more than 1 cm of overgrowth (47).

In a series of 40 open lower extremity diaphyseal fractures in 35 children, Kramer et al. (76) reported 22 tibial fractures (1 grade I, 10 grade II, and 11 grade III). External fixation was used for 15 fractures, casting for 5, and internal fixation for 2. Two children required early amputation, 4 required soft tissue flaps, and 13 children had skin grafts. Two additional children required fasciotomy for compartment syndrome and were included in the group of open tibial fractures. Ten of the 24 injuries healed within 24 weeks. Five children required bone grafting before healing.

Hope and Cole (69) reported the results of open tibial fractures in 92 children (22 grade I, 51 grade II, and 19 grade III). Debridement and irrigation were performed on admission, antibiotics were given for 48 hours, and tetanus prophylaxis was administered as needed. Primary closure was performed in 51 children, and 41 fractures were left open. Eighteen soft tissue injuries healed secondarily, and 23 required either a split-thickness skin graft or a tissue flap. Sixty-five (71%) of the 92 fractures were stable and were reduced and immobilized in an above-the-knee plaster cast. External fixation was used for unstable fractures, injuries with significant soft tissue loss, and fractures in patients with multiple system injuries. Early complications of open tibial fractures in these children were comparable with those in adults (Table 24-7) (44,47,49,51,53,62,66,69,78,84,86,89). Primary closure did not increase the risk of infection if the wound was small and uncontaminated (40,52,69). At reevaluation 1.5 to 9.8 years after injury, Hope and Cole found that 50% of the patients complained of pain at the fracture site; 23% reported decreased abilities to participate in sports, joint stiffness, and cosmetic defects; and 64% had leg length inequalities (Table 24-8). Levy et al. (81) found comparable late sequelae after open tibial fractures in children, including a 25% prevalence of nightmares surrounding the events of the accident. Blasier and Barnes (43) and Song et al. (95) found that most late complications associated with pediatric open tibial fractures occurred in children over the age 12 and 11, respectively.

Complication	Grade			Combined
	I	II	III	
Delayed union	18%	12%	26%	16%
Nonunion	5%	6%	16%	7.5%
Malunion	9%	4%	11%	6.5%
Infection	—	12%	21%	11%
Compartment syndrome	9%	2%	5%	4%
Physcal arrest	—	2%	5%	—
Weeks to union	13	12	17	13.5

Reprinted from Hope PG, Cole WG. Open fractures of the tibia in children. *J Bone Joint Surg [Br]* 1992;74:546; with permission.

TABLE 24-7. EARLY COMPLICATIONS ASSOCIATED WITH OPEN PEDIATRIC TIBIAL FRACTURES

Complication	Grade			Combined
	I	II	III	
Pain	38%	48%	65%	—
Decreased athletic activity	19%	15%	47%	—
Decreased mobility (Stiffness)	19%	20%	35%	—
Cosmetic complaints	6%	17%	53%	—
Leg length				
Short (0.5–2 cm)	22%	23%	40%	27%
Equal	14%	46%	33%	36%
Long (5.0–2 cm)	64%	31%	27%	37%

Reprinted from Hope PG, Cole WG. Open fractures of the tibia in children. *J Bone Joint Surg [Br]* 1992;74:546; with permission.

TABLE 24-8. LATE COMPLICATIONS ASSOCIATED WITH OPEN PEDIATRIC TIBIA FRACTURES

### Soft-Tissue Closure

The timing of soft tissue closure after an open tibial fracture that cannot be primarily closed is important in decreasing the morbidity associated with this injury (73,83).

In a series of 168 open tibial fractures with late secondary wound closure, Small and Mollan ( 93) found increased complications with early fasciocutaneous flaps and late free flaps and no complications with fasciocutaneous flaps created more than 1 month after injury. Complications associated with free flaps were decreased if the procedure was performed within 7 days of injury.

Ostermann, Henry, and Seligson (85) reported 115 grade II and 239 grade III tibial fractures in a series of 1,085 open fractures. All patients were treated with early broad-spectrum antibiotics, serial debridements, and the application of an external fixation device. Tobramycin-impregnated polymethylmethacrylate was placed into the wounds, and dressings were changed every 48 to 72 hours until the wounds spontaneously closed, or had delayed primary closure or a flap. No infections occurred in grade I fractures; approximately 3% of grade II fractures and 8% of grade III fractures became infected. No infections occurred in patients who had the wound closed by 8 days after injury. On the basis of this and other analyses, it now is recommended that wounds associated with open tibial fractures be closed within 7 days of injury (7,48,49,50,75,85,88,98). A delayed primary closure can be performed if the wound is clean and does not involve significant muscle loss. Tension at the suture line must be avoided.

### Vascular Injuries

Vascular injuries have been reported in approximately 5% of children with open tibial fractures. Arterial injuries associated with open tibial fractures include those to the popliteal artery, the posterior tibial artery, the anterior tibial artery, and the peroneal artery. Complications are common in patients with open tibial fractures and associated vascular injuries. Amputation rates as high as 21% have been reported with grade IIIB fractures and as high as 79% with grade IIIC fractures. Isolated anterior tibial and peroneal artery injuries have a good prognosis, whereas injuries of the posterior tibial and popliteal arteries have a poor prognosis ( 39,63,68). Patients with open tibial fractures and vascular disruption may benefit from an arterial and possibly venous shunt before the bony reconstruction is performed. This allows a meticulous repair of fracture and keeps the limb perfused until the primary vascular repair is performed ( 58).

### Compartment Syndrome

The prevalence of compartment syndromes in adults with open tibial fractures ranges from 6% to 9% ( 44,53,78). Compartment syndromes occur in approximately 5% of children with open tibial fractures. Normal compartment tissue pressure in the lower leg is approximately 0 mm Hg. Compartment blood inflow is decreased at 20 mm Hg, and prolonged pressures of 30 to 40 mm Hg or within 30 mm Hg of diastolic blood pressure may cause severe nonreversible injury to the muscles within a fascial compartment.

Symptoms and signs associated with compartment syndrome include pain out of proportion to the injury, burning, throbbing, pain increased by passive stretch, and nerve dysfunction. The compartment is tense to palpation. Because tissue pressures can be elevated enough to produce an ischemic injury but not high enough to occlude arterial inflow, the pressure of peripheral pulses is an unreliable sign of elevated tissue pressures.

Compartment pressure measurements are mandatory in any patient suspected of having a compartment syndrome. This can be performed by needle manometry, saline infusion, or a wick catheter. A fasciotomy is indicated for any patient with a significant elevation of compartment pressures or, more importantly, symptoms suggestive of a compartment syndrome. When measuring compartment pressure in the leg after a tibial fracture, placement of the needle is a problem. Buchholz showed that there was a pressure gradient vertically through a compartment.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

### Diaphyseal Fractures

Simple pediatric diaphyseal tibial fractures unite quickly, and cast immobilization can be used without affecting the long-term range of motion in the knee and the ankle. A bent knee, long leg cast provides maximal comfort to the patient and controls rotation of the fractured fragments. Children with nondisplaced fractures generally do not need to be admitted to the hospital. Children with more extensive injuries should be admitted for observation and teaching of wheelchair, crutch, or walker use.

Displaced fractures may significantly disrupt the surrounding soft tissues and produce a large hematoma in the fascial compartments of the lower leg. Circulation, sensation, and movement of the toes should be monitored carefully after injury. The child should be admitted to the hospital, and reduction should be performed with general anesthesia and fluoroscopy. Stable fractures are casted after reduction. The fracture must be checked within a week of manipulation to verify maintenance of the reduction. The cast can be wedged to correct minor alignment problems. Significant loss of reduction requires repeat reduction with general anesthesia ( Table 24-9). The long leg cast is changed to a short leg cast at 4 to 6 weeks after injury. Children over 11 years of age are placed into a patellar tendon-bearing cast after removal of the long leg cast (89).

	Patient Age	
	<8 Years	≥8 Years
Valgus	5°	5°
Varus	10°	5°
Anterior angulation	10°	5°
Posterior angulation	5°	0°
Shortening	10 mm	5 mm
Rotation	5°	5°

TABLE 24-9. ACCEPTABLE ALIGNMENT OF A PEDIATRIC DIAPHYSEAL TIBIAL FRACTURE

Fractures in patients with spasticity, a floating knee, multiple long bone fractures, extensive soft tissue damage, or multiple system injuries are stabilized with an external fixation device, percutaneous Kirschner wires, or flexible intramedullary nails.

### Open Fracture Treatment

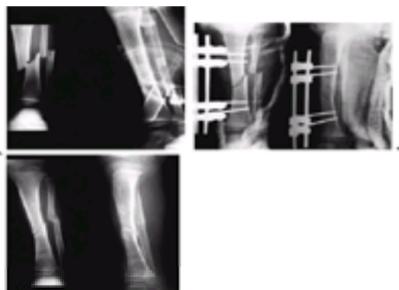
Grade I open fractures can be pinned after irrigation and debridement of the wound ( Fig. 24-15). A long leg splint is applied and is changed to a long leg cast after wound closure. External fixation generally is used for grade II or III open tibial fractures.



**FIGURE 24-15. A:** Anteroposterior radiograph of a grade I open distal one-third tibia fracture in a 7-year-old child. **B:** Two percutaneous pins were used to stabilize this fracture after irrigation and debridement. **C:** Good fracture callus was present and the pins were removed 4 weeks after injury.

Open tibial fractures of any grade should have thorough irrigation and debridement of the wound as soon as possible. The patient's tetanus status is determined, and prophylaxis is administered as needed. The soft tissue wounds can be extended to be certain that the area is cleansed and debrided of all foreign material. Devitalized bone can be left in place if it is clean. The operative wound extension can be closed with the open segment in clean grade I injuries. The wound is allowed to heal by secondary intention if it has moderate contamination at the time of the irrigation and debridement. Grade II and III wounds also are debrided of devitalized tissue and foreign material. Patients with uncomplicated grade I fractures can be placed in a cast. Smooth pin fixation will prevent displacement of unstable fractures. Most children with grade II and all children with grade III wounds require more rigid fracture stabilization, usually with external fixation.

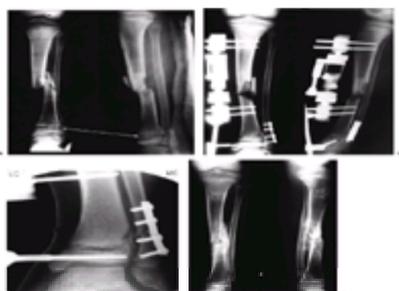
The most versatile external fixation device for grade II or III open pediatric tibial fractures is a unilateral frame ( Fig. 24-16). The unilateral frame is easy to apply and allows minor corrections in angular alignment and length. Secondary pins can be used for added support ( Fig. 24-17). These are connected to the standard pins or the body of the external fixation device. Fracture reduction clamps can be applied to the pin clamps to assist in manipulating the fracture. A small-pin circular frame can be used for complicated fractures adjacent to the joint ( Fig. 24-18).



**FIGURE 24-16. A and B:** Type II open fracture of the tibia in a 5-year-old boy treated with debridement, unilateral external fixation, and split-thickness skin graft. **C:** Four months after removal of the external fixation.



**FIGURE 24-17. A:** Anteroposterior and lateral radiographs of a grade IIIB open fracture of the distal tibia and fibula. **B:** Anteroposterior and lateral radiographs after fracture reduction and stabilization with an Ilizarov circular fixation frame. (Reprinted from Sharps CH, Cardea JA. Fractures in the shaft of the tibia and fibula. In: MacEwen GD, Kasser J, Heinrich SD, eds. Pediatric fractures: a practical approach to assessment and treatment. Baltimore: William & Wilkins, 1993:325; with permission.)



**FIGURE 24-18. A:** Anteroposterior and lateral radiograph of the tibia of a 12-year-old boy who was struck by a car. This child sustained a grade IIIB open middle one-third tibia fracture, a Salter-Harris type II fracture of the distal tibial physis with associated distal fibular fracture ( *closed arrows*), and a tibial eminence fracture ( *open arrow*). **B:** Irrigation and debridement, and application of an external fixation device, were performed. **C:** The fracture of the distal tibial physis was stabilized with a supplemental pin attached to the external fixation device. Open reduction and internal fixation of the fibula were performed to enhance the stability of the external fixator in the distal tibia. **D:** Anteroposterior and lateral radiographs of the tibia approximately 9 months after injury demonstrate healing of the tibial eminence fracture, the comminuted middle one-third tibia fracture, and the distal tibial physeal fracture. The distal tibial physis remains open at this time.

External fixation pins are applied no closer than 1 cm to the physis. The external fixation device is applied, and a reduction maneuver is performed. All of the connections in the external fixation device are tightened after reduction has been obtained. Secondary pins to improve fracture stability are placed at this time. Limited internal fixation of the fracture can be used to aid in controlling fracture alignment ( Fig. 24-19). A posterior splint can be applied to prevent the foot from dropping into plantar flexion. This splint is easy to remove during subsequent pin care and dressing changes of the open injury.



**FIGURE 24-19.** Safe drilling zone. **A:** The area occupied by the growth plate is approximately 1 cm in height because of its undulations. **B:** The safe drilling zone is at least 1 cm from the physis. (Reprinted from Alonso JE. The initial management of the injured child: musculoskeletal injuries. In: MacEwen GD, Kasser J, Heinrich SDH, eds. *Pediatric fractures: a practical approach to assessment and treatment*. Baltimore: Williams & Wilkins, 1993:36; with permission.)

## POSTFRACTURE IMMOBILIZATION

The length of immobilization varies with the child's age and the type of fracture. The duration of immobilization was 8 to 10 weeks in the Steinert and Bennek series (96). Hanson et al. (6) found that healing time ranged from 5 to 8 weeks for "fissures and infractions" and from 5 to 13 weeks for oblique, transverse, and comminuted fractures. Hoaglund and States (66) reported that in 43 closed fractures in children, the average time in a cast was 2.5 months (range 1.5–5.5 months), whereas the five children with open fractures were immobilized for 3 months.

Kreder and Armstrong (77) found an average time to union of 5.4 months (range 1.5–24.8 months) in a series of 56 open tibial fractures in 55 children. The factor with the most effect on union time was the age of the patient. Grimard et al. (60) reported that the age of the patient and the grade of the fracture were significantly associated with union time. Blasier and Barnes (43) found that children under 12 years of age required less aggressive surgical treatment and healed faster than older children. They also found that younger children were more resistant to infection and had fewer complications than older children.

## POSTFRACTURE REHABILITATION

Most children with a tibial fracture do not require extensive rehabilitation. Children's normal walking and running activities serve as therapy. Most children limp with an out-toeing rotation gait on the involved extremity for several weeks after the cast is removed. This usually is due to muscle weakness rather than a malalignment of the fracture. As the muscle atrophy and weakness resolve, so does the limp.

Active physical therapy may be beneficial in treating children with a tibial fracture. In an older child progressing from a bent knee cast to weight bearing on a short leg cast, knee range of motion exercises and quadriceps strengthening are useful. Progressive weight bearing on a short leg cast requires the patient to wean off crutches or a walker. In some children, this requires supervision. After removal of the cast, toe rises for strength should be performed. The child may return to sports with a healed fracture and the ability to hop equal to the uninjured side.

## FRACTURES OF THE DISTAL TIBIAL METAPHYSIS

Fractures of the distal tibial metaphysis often are greenstick injuries. The anterior cortex is impacted while the posterior cortex is displaced, with a tear of the overlying periosteum. A recurvatum deformity often occurs (Fig. 24-20). Reduction of these injuries should be performed with general anesthesia and maintained with a long leg cast. The foot should be in plantar flexion to prevent recurvatum at the fracture site. The foot is brought up after 3 to 4 weeks, and a short leg walking cast is applied. Unstable injuries can be treated with percutaneous pins (Fig. 24-21) or with open reduction and internal fixation (Fig. 24-22).



**FIGURE 24-20.** **A:** The lateral radiographs demonstrate a mild recurvatum deformity. **B:** The ankle was initially immobilized in an ankle neutral position, producing an increased recurvatum deformity. The cast was removed and the ankle remanipulated into plantar flexion to reduce the deformity. **C:** The ankle was then immobilized in plantar flexion, which is the proper position for this type of fracture.



**FIGURE 24-21.** **A and B:** Unstable distal metadiaphyseal fracture of the tibia and fibula in a 15-year-old girl. **C:** This fracture was stabilized with percutaneous pins because of marked swelling and fracture instability.



**FIGURE 24-22. A:** Anteroposterior radiograph of a distal one-third tibia/fibula fracture in a 9-year-old girl with a closed head injury and severe spasticity. The initial reduction in a cast could not be maintained. **B:** An open reduction and internal fixation with a medial buttress plate was performed to achieve and maintain the alignment.

## COMPLICATIONS ASSOCIATED WITH DIAPHYSEAL TIBIA/FIBULA FRACTURES

### Compartment Syndrome

The anterior compartment of the leg includes the extensor hallucis, the extensor digitorum longus, and the tibialis anterior muscles. It is surrounded by the tibia, the fibula, the interosseous membrane, and the anterior fascia. A compartment syndrome may occur after either a minor closed fracture or a severe injury in which the interosseous membrane is disrupted (79). Schrock (90) also described compartment syndromes after derotational osteotomies of the tibia in children.

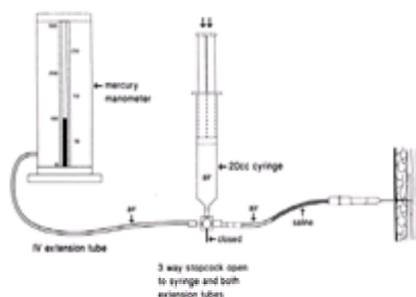
Compartment syndromes also have been reported in the other three compartments of the lower leg after trauma. Hemorrhage and soft tissue edema produce an elevation in the pressure within the myofascial compartment that impairs venous outflow. The small arterioles leading into the compartment become less efficient in delivering blood as venous outflow becomes occluded. The arterioles and capillaries close when the pressure in the compartment exceeds the pressure in the vessels. Ischemia soon follows.

Patients with a compartment syndrome complain of pain out of proportion to the severity of the injury. The compartment is firm to palpation. The patient may have a sensory defect in the distribution of the nerves that run through the compartment. Weakness of the muscles within the involved compartment and pain on passive motion of the toes also are common. Paralysis of the muscles in the involved compartment is a late finding.

Patients with a compartment syndrome of the deep posterior compartment have severe pain that increases with passive extension of the toes, plantar hyperesthesias, weakness of toe flexion, and tenseness of the fascia between the tibia and the triceps area in the distal medial part of the leg (82). Late complications include clawed toes and limited subtalar motion secondary to a fibrous contracture of the muscles in the deep posterior compartment (71).

### Diagnosis

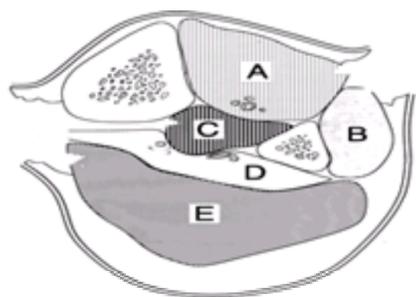
Direct intracompartmental pressure measurements at the level of the fracture provide an accurate assessment of compartment conditions and allow early fasciotomy to reduce the pressure. Whitesides et al. (99) designed an inexpensive apparatus that permits accurate measurement of compartment tissue pressure (Fig. 24-23). Other commercial devices also are available to measure compartment pressures. Normal tissue pressures are about 0 mm Hg. Vascular flow ceases in the microcirculation of an extremity by the time tissue pressures within a closed compartment reach the diastolic pressure. Compartment release should be performed when the pressures are within 20 to 30 mm Hg of the diastolic pressure. This is especially critical in a hypotensive polytrauma patient.



**FIGURE 24-23.** Whitesides' technique for measuring intercompartmental pressure. (Reprinted from Whitesides TE, Hanley TC, Morinotok K, et al. Tissue pressure measurement as a determinant for the need of fasciotomy. *Clin Orthop* 1975;113:43; with permission.)

### Treatment

First the cast should be bivalved and the padding divided. If after removal of circular wraps there is no relief, fasciotomy should be considered. Any child who has evidence of a compartment syndrome should undergo an emergent fasciotomy. The two-incision technique is preferred (Fig. 24-24). One incision is anterolateral and the second posteromedial. The fascia surrounding each of the four compartments should be opened from proximal to distal. If the wound can be closed primarily, it should be, but usually the wound is left open with retention sutures and a delayed closure is performed. Fibulectomy should not be performed for decompression of a compartment syndrome in a skeletally immature patient because subsequent fibular shortening can produce a valgus deformity at the ankle and may result in external tibial torsion and a severe gait impairment (17,55).



**FIGURE 24-24.** Preferred decompression technique for the lower leg, using medial and lateral incisions. **A:** Anterior compartment. **B:** Lateral compartment. **C:** Tibialis posterior. **D:** Posterior compartment. **E:** Superficial posterior compartment. (Reprinted from Alonso JE. The initial of evaluation of the injured child: musculoskeletal injuries. In: MacEwen G, Kasser J, Heinrich SD, eds. *Pediatric fracture: the practical approach to assessment and treatment*. Baltimore: Williams & Wilkins, 1993; with permission.)

Irreversible injury to muscles and nerves begins after approximately 5 hours of ischemia (91). Ninety percent of muscle fibers show evidence of injury after 8 hours of ischemia (65). Hyperesthesia, motor defects, and decreased pulses are late changes and denote significant tissue injury. They occur only after the ischemia has been well established and the injury is permanent (71,99).

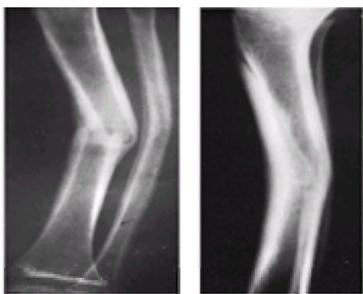
### Vascular Injuries

Vascular injuries associated with tibial fractures are uncommon in children; however, when they do occur, the sequelae can be devastating. In an evaluation of 14 patients with lower extremity fractures and concomitant vascular injuries, Allen et al. (39) noted that only 3 children returned to normal function. One factor leading to a poor outcome was a delay in diagnosis. Evaluation for vascular compromise is imperative (during the primary and secondary trauma surveys) in all children with tibial fractures.

The tibial fracture most frequently associated with vascular injury is in the proximal metaphysis. The anterior tibial artery is in close proximity to the proximal tibia as it passes over the interosseous membrane into the anterior compartment (63,68). Distal tibial fractures also are associated with injuries to the anterior tibial artery. The vessels are injured when the distal fragment is translated posteriorly. Posterior tibial artery injury is rare.

### Angular Deformity

Spontaneous correction of axial malalignment after a diaphyseal fracture of a child's forearm or femur is common. Remodeling of a malaligned tibial fracture, however, often is incomplete (Fig. 24-25) (45).



**FIGURE 24-25.** Four-year, two-month old child with a middle one-third transverse tibia fracture and a plastically deformed fibula fracture. **A:** Lateral view shows 20 degrees posterior angulation. **B:** The deformity is still 15 degrees 4 years after the injury.

Swaan and Oppers (97) evaluated 86 children treated for fractures of the tibia. The original angulation of the fracture was measured on radiographs in the sagittal and frontal projections. Girls 1 to 8 years of age and boys 1 to 10 years of age demonstrated moderate spontaneous correction of residual angulation after union. In girls 9 to 12 years of age and boys 11 to 12 years of age, approximately 50% of the angulation was corrected. No more than 25% of the deformity was corrected in children over 13 years of age. Bennek and Steinert (41) found that a recurvatum malunion of more than 10 degrees did not completely correct. Twenty-six of 28 children with varus or valgus deformities at union had significant residual angular deformities at follow-up. Valgus deformities had a worse outcome because the tibiotalar joint was left in a deficient position.

Weber et al. (13) demonstrated that a fracture with varus malalignment of 5 to 13 degrees completely corrected at the level of the physis. Most children with valgus deformities of 5 to 7 degrees did not have a full correction.

Hanson et al. (6) reported 102 pediatric tibial fractures, 25 of which had malunions of 4 to 19 degrees. Angular malunions ranged from 3 to 19 degrees at follow-up, without a single patient having a complete correction. The spontaneous correction was approximately 13.5% of the total deformity. Shannak (11) reviewed the results of treatment of 117 children with tibial shaft fractures treated in above-the-knee casts. Deformities in two planes did not remodel as completely as those in one plane. The smallest correction occurred in posteriorly angulated fractures, followed by fractures with a valgus malalignment (Fig. 24-26). Spontaneous remodeling of malunited tibial fractures in children has been reported to stop 18 months after fracture (6).



**FIGURE 24-26. A:** Anteroposterior and lateral radiographs 2 months after injury in a 6-year-old boy reveal a valgus and an anterior malunion at the fracture. **B:** One year later, the child still has a moderate valgus and anterior malalignment of the distal fractured segment. This malalignment produced painful hyperextension of the knee at heel strike during ambulation.

### Malrotation

Because rotational malalignment of the tibia does not spontaneously correct with remodeling (6), all malrotation should be avoided. A rotational computerized tomographic evaluation can be performed if there is any question about the rotational alignment of the fracture.

Rotational malalignment of more than 10 degrees may produce significant functional impairment and necessitate a derotational osteotomy of the tibia. The derotational osteotomy should be performed in the supramalleolar aspect of the distal tibia. The tibia is osteotomized and internally fixed. The fibula is left intact.

### Leg Length Discrepancy

Hyperemia associated with fracture repair stimulates the physes in the involved leg, producing growth acceleration. Tibial growth acceleration after fracture is less than that seen after femoral fractures in children of comparable ages. Shannak (10) showed that the average growth acceleration of a child's tibia after fracture is approximately 4.5 mm. Comminuted fractures have the greatest risk of accelerated growth and overgrowth.

Swaan and Oppers (97) reported that young children have a greater chance for overgrowth than older children. Accelerated growth after tibial fracture occurs in children under 10 years of age, whereas older children may have a mild growth inhibition associated with the fracture (6). The amount of fracture shortening also has an effect on growth stimulation. Fractures with significant shortening have more physal growth than fractures without shortening at union (10). The presence of angulation at union does not affect the amount of overgrowth (59).

### Upper Tibial Physeal Closure

Morton and Starr (83) reported closure of the upper tibial physis after fracture in two children. Both patients sustained a comminuted fracture of the tibial diaphysis without a concomitant injury of the knee. Both fractures were reduced and stabilized with Kirschner wires placed distal to the tibial tubercle. A genu recurvatum deformity developed after the anterior physis closed.

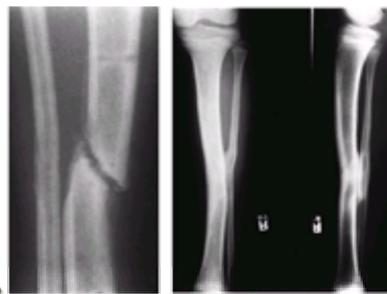
Smillie (94) reported one child who had an open tibial fracture complicated by a second fracture involving the supracondylar aspect of the femur. This patient also developed a recurvatum deformity secondary to closure of the anterior proximal tibial physis. At present, no universally acceptable explanation can be given for this phenomenon. Some patients have iatrogenic closure after placement of a proximal tibial traction pin, the application of pins and plaster, or the application of an external fixation device. Other children have an undiagnosed injury of the tibial physis at the time of the ipsilateral tibial diaphyseal fracture (74). Regardless of etiology, closure of the physis produces a progressive recurvatum deformity as the child grows.

### Delayed Union and Nonunion

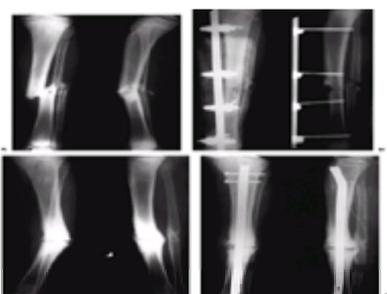
Delayed union and nonunion are uncommon after tibial fractures in children. The use of an external fixation device or infection after operative intervention may lengthen the time to union in some patients. Inadequate immobilization that allows patterned motion also can slow the rate of healing. A fibulectomy approximately 4 cm from a tibial nonunion allows compression at the nonunion site and induces healing (Fig. 24-27). A posterolateral bone graft also is an excellent technique to produce union in younger children (Fig. 24-28). Adolescents near skeletal maturity can be treated with a reamed intramedullary nail, with a concomitant fibular osteotomy and correction of angulation at the nonunion site performed as necessary (Fig. 24-29).



**FIGURE 24-27.** **A:** Anteroposterior radiograph of the distal tibia and fibula in a 5-year-old boy with an open fracture. **B:** Early callus formation is seen 1 month after injury. **C:** The tibia has failed to unite 10 months after injury. **D:** The patient underwent a fibulectomy 4 cm proximal to the tibial nonunion. The tibial fracture united 8 weeks after surgery.



**FIGURE 24-28.** **A and B:** Posterolateral tibial bone graft for a nonunion of an open tibia fracture.

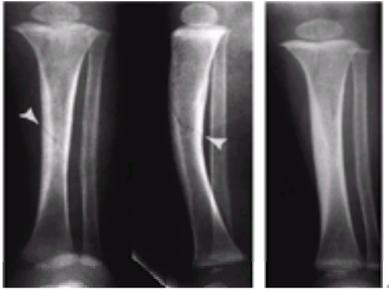


**FIGURE 24-29.** **A:** Anteroposterior and lateral radiographs of a 14-year-old adolescent who was struck by a car, sustaining a grade IIIB open fracture of the tibia. **B:** Anteroposterior and lateral radiographs of the tibia after irrigation and debridement, and application of an external fixation device. **C:** The patient developed a nonunion at the tibia, which progressively deformed into an unacceptable varus alignment. **D:** The nonunion was treated with a fibular osteotomy followed by a closed angular correction of the deformity and internal fixation with a reamed intramedullary nail.

## SPECIAL FRACTURES

### Toddler's Fractures

External rotation of the foot with the knee fixed in an infant or toddler can produce a spiral fracture of the tibia without a concomitant fibular fracture (Fig. 24-30). This fracture pattern was first reported by Dunbar et al. (108) in 1964. The traumatic episode often is unwitnessed by the adult caretaker. Most children with this injury are under 6 years of age. Sixty-three of 76 such fractures reported by Dunbar et al. were in children under 2½ years of age. Spiral tibial fractures occur in boys more often than in girls and in the right leg more often than in the left. The average age at injury in one study was 27 months. Most children report tripping or twisting their ankles. Occasionally, a child sustains a toddler's fracture in a fall from a height (104,140).

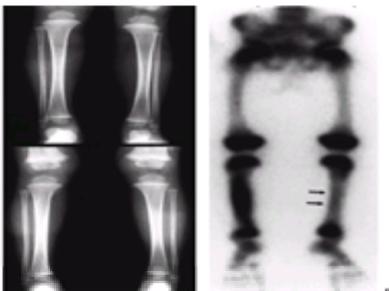


**FIGURE 24-30. A:** Anteroposterior and lateral radiographs of an 18-month-old child who presented with refusal to bear weight on her leg. Note the spiral middle one-third “toddler’s” fracture ( *arrows*). **B:** This fracture healed uneventfully after 4 weeks of immobilization in a cast.

Oujhane et al. (129) analyzed the radiographs of 500 acutely limping toddlers and identified 100 in whom a fracture was the etiology of the gait disturbance. The most common site of fracture was the distal metaphysis of the tibia. The fibula was fractured with the tibia in 12 of the 56 tibial fractures. Only one physal injury was noted.

The examination of a child with an acute limp starts on the uninvolved side. This serves as a control for the symptomatic extremity. The examination begins at the hip and proceeds to the thigh, knee, lower leg, ankle, and foot. It is important to note the areas of point tenderness, an increase in local temperature, and any swelling or bruising of the leg (140).

Radiographs of the tibia and fibula should be obtained in both anteroposterior and lateral projections. An internal oblique view can be helpful in identifying a nondisplaced toddler’s fracture. Fluoroscopy also may assist in the identification of subtle fractures. Occasionally, a fracture line cannot be identified, and the first evidence of fracture becomes apparent radiographically when periosteal new bone forms 1 week to 10 days after the injury ( Fig. 24-31). Technetium radionuclide imaging can assist in the diagnosis of radiographically unapparent fractures. A patient with a spiral fracture of the tibia has diffuse increased uptake of tracer throughout the affected bone (black tibia). This can be differentiated from infection because infection tends to produce a local area of increased tracer uptake ( 108).

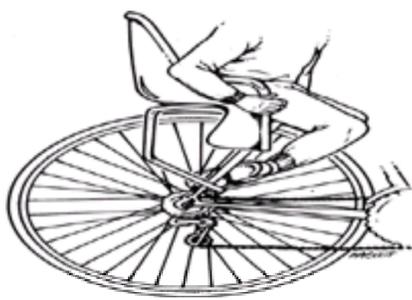


**FIGURE 24-31. A:** An anterior posterior radiograph of the tibia is in a 3-year-old child who refused to bear weight on the right leg 3 weeks before presentation. The history of obvious trauma was absent. His parents brought him into the hospital also when he refused to weight bear on his left leg. The radiographs revealed periosteal new bone formation in the mid-shaft of the right tibia. There was also tenderness to palpation in the mid-left tibia as well despite normal radiographs. **B:** A bone scan was obtained which showed increased uptake in both the left and right tibia. There was significantly less uptake on the left side, the more recent injury.

A child with a toddler’s fracture should be immobilized in a bent knee, long leg cast for approximately 3 weeks. Most children require an additional 2 weeks of immobilization in a below-the-knee walking cast once the above-the-knee cast is removed.

### Bicycle Spoke Injuries

Bicycle spoke injuries normally occur when a bicycle overturns and the child’s foot is thrust forcibly between the spokes of the turning wheel ( Fig. 24-32). This produces a severe compression or crushing injury to the soft tissues of the foot and ankle. An oblique or spiral fracture of the tibia also can occur ( Fig. 24-33). The injury to the foot, ankle, and lower leg can be compounded when the child’s foot is extracted forcibly from the spokes of the bicycle. This scenario occurs most commonly when two children are riding a bicycle designed for one, with the injured passenger riding on the handlebars or the rear fender ( 111,126).



**FIGURE 24-32.** The mechanism of a bicycle spoke injury. The foot is impinged between the spokes of the wheel and the frame of the bike.



**FIGURE 24-33.** Oblique radiograph of a 4-year-old child who sustained an oblique fracture of the distal tibia and transverse fracture of the fibula when his foot was caught in the spokes of a bicycle. Note the proximal fibular fracture ( *arrow*).

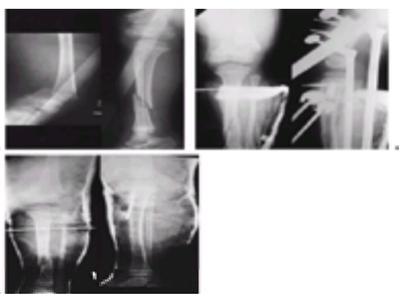
Izant et al. (120) reviewed 60 bicycle spoke injuries in children under 14 years of age. The most common age range of injury was 2 to 8 years.

The initial appearance of the extremity in a child with a bicycle spoke injury may be deceiving. The foot often appears normal or may show only minor skin abrasions. The patient often presents 24 to 48 hours after the accident complaining of a painful swollen foot and leg. This injury is similar to a “wringer” injury of the arm because the initial examination may not reveal the true extent of the injury. Izant et al. (120) identified three components to this trauma: (a) a laceration of the tissue from the knifelike action of the spoke, (b) a crushing from the impingement between the wheel and the frame of the bicycle, and (c) a shearing injury from the coefficient of these two forces. The laceration created by this injury often involves the malleoli, the Achilles tendon area of the heel, and the dorsum of the foot. A child with a bicycle spoke injury may require multiple debridements, and definitive treatment must await delineation of the necrotic area.

A child with a bicycle spoke injury should be admitted to the hospital because the extent of the damage may not be identified initially. Initial therapy consists of a mild compression dressing with a multilayered cotton bandage. The extremity is elevated and the child is kept at bedrest during the first 24 hours. A long leg splint is applied if a tibial fracture is present. The child may ambulate with crutches non-weight bearing after that. Frequent inspection of the extremity must be made during the subsequent 48 hours. Debridement of devitalized tissue is performed as necrosis becomes apparent. Large areas of hematoma formation are treated with aspiration to prevent further elevation of the overlying skin. Wound closure with a split-thickness skin graft is performed if full-thickness skin loss occurs. Occasionally, a free flap may be required, but this is rare. Most patients regain normal function of the foot and ankle. The average time for complete healing may be 5 to 6 weeks (111,120,126). A child with a concomitant tibial fracture is placed in an above-the-knee non-weight-bearing cast at the completion of care for the foot injury (111). The tibial fracture may need to be stabilized with an external fixation device if the soft tissue loss on the foot and ankle is severe.

### Floating Knee

Significant trauma can cause fractures involving both the femur and the tibia. In the past, these injuries often were treated with traction and casting (Fig. 24-34). The extent of the injuries often left permanent functional deficits when not aggressively managed (45,80).



**FIGURE 24-34.** A: Ipsilateral fracture of the distal femur and tibia without an ipsilateral fibula fracture in a 5-year-old. B: The child was treated with tibial pin traction for the femoral injury (pin applied below the tibial tubercle) and a short leg splint for the tibia fracture initially. C: The child was placed into a spica cast after 2 weeks of traction. The tibial traction pin was used to help stabilize both fractures.

Most children over 2 years of age with ipsilateral femoral and tibial fractures are treated with operative stabilization of the femur and cast immobilization of the tibia after reduction. In children under 6 years of age, the femoral fracture can be stabilized with a unilateral external fixator or a plate with screws; in children 6 years of age and older, flexible intramedullary nails can be used. Open reduction and plate fixation are used for fractures in the subtrochanteric or supracondylar area of the femur in adolescents. The tibial fracture is reduced closed and stabilized with a long leg cast after the femoral fracture has been reduced and stabilized. Open tibial fractures and those in which a reduction cannot be maintained after casting are reduced (open if necessary) and stabilized with a unilateral external fixator (Fig. 24-35). If closed reduction is not possible, open reduction with pin fixation or external fixation is indicated.



**FIGURE 24-35.** A: Anteroposterior radiograph of a grade IIIA segmental tibia fracture with an ipsilateral fibula and femur fracture in a 10-year-old boy with aplastic anemia. B: The tibia fracture was irrigated, debrided, reduced, and stabilized with an external fixation device. The femur fracture was stabilized with antegrade Ender nail fixation. C: Lateral radiograph of the tibia demonstrates the segmental nature of this fracture (arrows) and the excellent alignment achieved with the external fixation device.

### Tibial Fracture in Paraplegic Children

Motor paralysis from poliomyelitis was once the most common cause of lower extremity weakness in children. Because these patients have sensation, fractures are identified early. Disease trends have changed, however. As late as 1958, 90% to 95% of children with myelomeningocele died in the first year of life, usually from a neurosurgical complication. Recent advances in neurosurgery and urology have significantly increased the life span of children with myelomeningocele. The mortality rate for these children is now 3% to 5%. Orthopaedic surgeons, therefore, are seeing an increasing number of paraplegic children who have sensory deficits (107,112,113).

Tibial fractures in children without sensation require special attention. Gillies and Hartung (113) are the first to report children with myelomeningocele who sustained pathologic fractures of the proximal tibia. These two children had tense hyperemic skin and radiographic evidence of exuberant new bone formation, suggesting a malignant tumor (Fig. 24-36). Both children had a biopsy because of this radiographic finding.



**FIGURE 24-36.** An undisplaced fracture of the proximal tibial metaphysis in a child with myelodysplasia. Note the exuberant new bone formation.

Fractures around the knee are common in children with paraplegia because of the length of the lower extremity and the association with joint contractures ( [116](#)). These fractures are more common in children with flaccid paralysis than those with spastic paralysis ( [121,133](#)).

Soutter ([98](#)) stressed that clinical findings such as swelling, warmth, and erythema are common in paraplegic children with a fracture. He stated that “fractures to the growth plate in paraplegic children often resemble osteomyelitis.”

James ([121](#)) reported 44 fractures in 22 children in a population of 122 children with myelomeningocele. The most common age range at fracture was 3 to 6 years. These fractures were more common in a flail limb. Only 6.6% of the patients with quadriceps activity had fractures, whereas 19% of those with no active muscle contraction (flail limb) had fractures. This incidence decreased to 12.5% in a group of children with spastic paralysis. The tibia and femur were the most common bones affected. The most common locations for tibial fractures were the distal diaphysis and distal metaphysis.

Parsch and Rossak ([130](#)) reported 31 fractures in 120 patients with myelomeningocele. They emphasized treatment with a wrap and splint until early fracture consolidation. To prevent “fracture disease,” they stressed early standing and walking to help reduce the degree of osteopenia these children develop.

Drummon and Freehafer ([107](#)) reported 58 fractures in 25 patients among 84 children with myelomeningocele. Ten fractures were in the tibia. Eleven of the 58 fractures occurred after the removal of a spica cast, suggesting that immobilization worsened osteopenia and increased the risk of subsequent fracture.

Complications in patients with paralysis and a tibial fracture are rare and usually are related to pressure sores produced by ill-padded casts. Early physeal arrest can occur, producing a leg length discrepancy, and physeal involvement is common in paralytic children with fractures ( [137](#)). Golding ([114](#)) reported one physeal injury to the distal tibia in a child with myelomeningocele. Gyepes et al. ( [116](#)) reported seven metaphyseal and physeal injuries in patients with myelomeningocele. Radiographs were characterized by an irregular, dense, widened physis and adjacent subperiosteal new bone formation. Stern et al. ( [138](#)) reported bilateral distal tibial and fibular physeal injuries in a child with myelomeningocele.

Trauma probably is the primary factor producing the complex radiographic findings associated with a tibial fracture in a paralytic child ( [116](#)). Repeated microtrauma can lead to a metaphyseal infraction, subperiosteal hemorrhage, and, perhaps, physeal hemorrhage. Subsequent healing produces endosteal and periosteal callus formation and some generalized osteopenia secondary to the immobilization ( [Fig. 24-36](#)). The widening of the physis most likely is secondary to a disturbance of the normal reabsorption mechanism during bone development at the metaphysis. This results from frequent subclinical metaphyseal infractions. It also could be produced by hemorrhage into the physis itself.

Fracture of the tibia in a paralytic child can occur with minor trauma. Many fractures are nondisplaced, and nonoperative therapy is indicated. Displaced fractures are reduced closed and immobilized for 3 to 4 weeks in a bulky dressing or a posterior, molded, well-padded splint. The child should be fitted with an orthosis or placed back into a reciprocal gait orthosis (if the child already had one) after the immobilization is discontinued.

Lower extremity fractures are common after a child has been immobilized in a cast, especially after hip surgery. This finding has led to the term *fracture disease*, which describes multiple fractures secondary to osteopenia after immobilization. Because of this, a child predisposed to fractures after immobilization may benefit from a short course of in-hospital split Russell's traction with frequent general physical therapy for range of motion. The child should not be allowed to walk or crawl for approximately 7 days after the traction is discontinued. This allows the stiffness that accompanies the immobilization to improve.

Physeal injuries may require prolonged periods of immobilization. Healing is determined by the absence of local warmth and swelling and the reconstitution of the width of the physis radiographically ( [124](#)).

### Stress Fractures of the Tibia and Fibula

Roberts and Vogt ([132](#)) reported an “unusual type of lesion” in the tibia of 12 children in 1939. All of these were determined to be stress fractures involving the upper third of the tibial shaft. Since then, numerous reports of stress fractures involving the tibia and the fibula have been published ( [Fig. 24-34](#)) ([56,102,106,110,117,119,123,125,127,128,131,134,135,139](#)).

The pattern of stress fractures in children differs from that in adults ( [105,106,136](#)). In adults, the fibula is involved in stress fractures twice as often as the tibia; in children the tibia is affected more often than the fibula. The prevalence of stress fractures in boys and girls is equal.

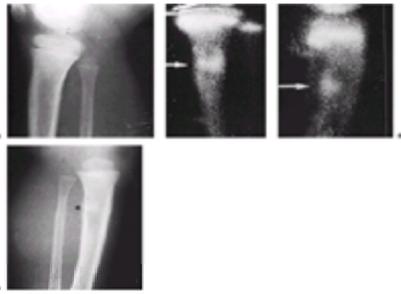
A child with a stress fracture usually has an insidious onset of symptoms ( [105,106](#)). There is evidence of local tenderness that worsens with activity. The child may have a painful limp. A toddler with a stress fracture will not bear weight on the involved extremity. The pain tends to be worse in the day and to improve at night and with rest. The knee and the ankle have full ranges of motion. Usually, there is no swelling at the fracture site ( [75,103,105,106,110,118](#)).

Radiographs reveal changes consistent with a stress fracture approximately 2 weeks after the onset of symptoms ( [106](#)). Radiographic evidence of fracture repair can manifest in one of three ways: localized periosteal new bone formation, endosteal thickening, or, rarely, a radiolucent cortical fracture line ( [Fig. 24-37](#)) ([75,105,106,117,135](#)).

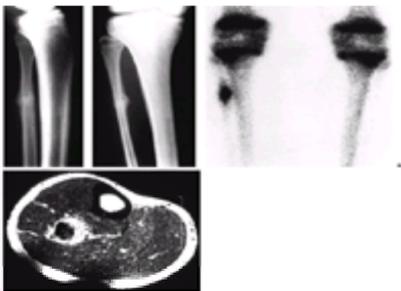


**FIGURE 24-37.** Bilateral mid-tibial stress fractures in an adolescent with genu varus.

Technetium radionuclide bone imaging reveals a local area of increased tracer uptake at the site of the fracture ( [Fig. 24-38](#)). Computerized tomography rarely demonstrates the fracture line, but delineates increased marrow density and endosteal and periosteal new bone formation. It also may show soft tissue edema. Magnetic resonance imaging (75,118) shows a localized band of very low signal intensity continuous with the cortex. MR signaling can be diagnostic of stress fracture and differentiate it nicely from malignancy obviating the need for biopsy ( [Fig. 24-39](#)).



**FIGURE 24-38. A:** Lateral radiograph of the proximal tibia in a 9-year-old who complained of pain in the right leg. There was no history of trauma, and the radiographs were unremarkable. **B:** A bone scan 2 days after the onset of symptoms demonstrates increased tracer uptake in the proximal one third of the tibia in both the anteroposterior and the lateral projections ( *arrows*). **C:** Increased bone density and subtle periosteal new bone formation was identified in the proximal tibia 3 weeks later (*arrows*). (Courtesy of James Conway, M.D.)



**FIGURE 24-39. A:** Stress fracture of the fibula in a 14-year-old girl with mild genu varus. **B:** Bone scan of stress fracture showing marked increased tracer uptake. **C:** Magnetic resonance image demonstrates new central bone formation and an inflammatory zone around the fibular cortex. (Reprinted from Sharps CA, Cardea JA. Fractures of the shaft of the tibia and fibula. In: MacEwen GD, Kassir JR, Heinrich SD, eds. Pediatric fractures: a practical approach to assessment and treatment. 1993:324; with permission.)

Stress fractures occur when the force applied to a bone is exceeded by the bone's capacity to withstand it. Initially, osteoclastic tunnel formation increases. These tunnels normally fill with mature bone. With continued force, cortical reabsorption accelerates. New bone is produced to splint the weakened cortex. This bone is immature, however, and does not have the strength of the bone it replaces. A fracture occurs when bone reabsorption outstrips bone production. When the offending force is reduced or eliminated, bone production exceeds bone reabsorption. This produces cortical and endosteal widening with dense repair bone that later remodels to mature bone ([109,122,129](#)).

### Tibia

The most common location for a tibial stress fracture is in the upper third. The child normally has a painful limp of gradual onset with no history of a specific injury. The peak incidence of tibial stress fractures in children is between 10 and 15 years of age. Pain is relieved with rest. The pain is described as dull, occurring in the calf near the upper end of the tibia on its medial aspect, and occasionally is bilateral. Physical findings include local tenderness on one or both sides of the tibial crest with a varying degree of swelling.

The treatment of a child with a stress fracture of the tibia (or fibula) begins with activity modification. An active child can rest in a long leg walking cast for 4 to 6 weeks followed by gradual increase in activity. Nonunions of stress fractures of the tibia have been described. Green ( [114](#)) reported six nonunions, three of which were in children. Two required excision of the nonunion site with iliac crest bone grafting. The third was treated by electromagnetic stimulation. In all three, the stress fractures occurred in the middle third of the tibia.

### Fibula

Pediatric fibular stress fractures normally occur between the ages of 2 and 8 years ( [115](#)). The fractures are normally localized to the distal third of the fibula.

The child presents with a limp and may complain of pain. Tenderness is localized to the distal half of the fibular shaft. Swelling normally is not present. The obvious bony mass commonly seen in a stress fracture of the fibula in an adult is normally not seen in a comparable fracture in a child.

No radiographic abnormalities are identified in the first 10 days to 2 weeks after the symptoms begin. The earliest sign of a stress fracture of the fibula is the presence of "eggshell" callus along the shaft of the fibula ( [Fig. 24-37](#)) (103). Fracture itself cannot always be seen because the periosteal callus may obscure the changes in the narrow canal. Radionuclide bone imaging can help to identify stress fractures before the presence of radiographic changes.

The differential diagnosis includes sarcoma of bone, osteomyelitis, and a soft tissue injury without accompanying bony injury. Treatment consists of rest or, in a very active child, a short leg walking cast for 4 to 6 weeks.

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## CHAPTER REFERENCES

### Introduction, Epidemiology, Surgical Anatomy

1. Bengner V, Ekholm T, Johnell O, et al. Incidence of femoral and tibial shaft fractures. *Acta Orthop Scand* 1990;61:251.
2. Blount WP. *Fractures in children*. Baltimore: Williams & Wilkins, 1955.
3. Buckley SL, Gotschall C, Robertson W, et al. The relationship of skeletal injuries with trauma score, injury severity score, length of hospital stay, hospital charges and mortality in children admitted to a regional pediatric trauma center. *J Pediatr Orthop* 1994;14:449.
4. Cheng JCY, Shen WY. Limb fracture pattern in different pediatric age groups: a study of 3,350 children. *J Orthop Trauma* 1993;7:15–22.
5. Gray H. Anatomy: descriptive and surgical. In: Pick TP, Howden R, eds. *Anatomy: descriptive and surgical*. New York: Bounty Books, 1977:182.
6. Hansen BA, Greiff S, Bergmann F. Fractures of the tibia in children. *Acta Orthop Scand* 1976;47:448.

7. Karrholm J, Hansson LI, Svensson K. Incidence of tibio-fibular shaft and ankle fractures in children. *J Pediatr Orthop* 1982;2:386.
8. Karlsson MK, Nilsson BE, Obrant KJ. Fracture incidence after tibial shaft fractures: a 30-year follow-up study. *Clin Orthop* 1993;287:87.
9. King J, Defendorf D, Apthorp J, et al. Analysis of 429 fractures in 1889 battered children. *J Pediatr Orthop* 1988;8:585.
10. Mellick LB, Reesor K, Demers D, et al. Tibial fractures of young children. *Pediatr Emerg Care* 1988;4:97.
11. Shannak AO. Tibial fractures in children: follow-up study. *J Pediatr Orthop* 1988;8:306.
12. Steinert VV, Bennek J. Unterschenkelfrakturen in Kindesalter. *Zentralbl Chir* 1966;91:1387.
13. Weber BG, Brunner C, Freuner F, eds. *Treatment of fractures in children and adolescents*. Berlin: Springer-Verlag, 1980.

#### Proximal Metaphyseal Fractures

14. Balthazar DA, Pappas AM. Acquired valgus deformity of the tibia in children. *J Pediatr Orthop* 1984;4:538.
15. Basse LO. Valgus deformity following proximal metaphyseal fractures in children: experiences in the African tropics. *J Trauma* 1990;30:102.
16. Coates R. Knock-knee deformity following upper tibial "greenstick" fractures. *J Bone Joint Surg [Br]* 1977;59:516.
17. Cozen L. Fracture of the proximal portion of the tibia in children followed by valgus deformity. *Surg Gynecol Obstet* 1953;97:183.
18. DeBastiani G, Aldegheiri R, Renzi-Brivio L, et al. Limb lengthening by distraction of the epiphyseal plate. *J Bone Joint Surg [Br]* 1986;68:545.
19. DeBastiani G, Aldegheiri R, Renzi-Brivio LR, et al. Chondrodiastasis-controlled symmetrical distraction of the epiphyseal plate. *J Bone Joint Surg [Br]* 1986;68:550.
20. Goff CW. *Surgical treatment of unequal extremities*. Springfield, IL: Charles C Thomas, 1960:135–136.
21. Green NE. Tibia valga caused by asymmetrical overgrowth following a nondisplaced fracture of the proximal tibia metaphysis. *J Pediatr Orthop* 1983;3:235.
22. Jackson DW, Cozen L. Genu valgum as a complication of proximal tibial metaphyseal fractures in children. *J Bone Joint Surg [Am]* 1971;53:1571.
23. Jordan SE, Alonso JE, Cook FF. The etiology of valgus angulation after metaphyseal fractures of the tibia in children. *J Pediatr Orthop* 1987;7:450.
24. Keret D, Harcke HT, Bowen JR. Tibia valga after fracture: documentation of mechanism. *Arch Orthop Trauma Surg* 1991;110:216–219.
25. Lehner A, Dubas J. Sekundäre Deformierungen nach Epiphysenlosungen und Epiphysenliniennahen Frakturen. *Helv Chir Acta* 1954;21:388.
26. Ogden JA. Tibia and fibula. In: *Skeletal injury in the child*. Philadelphia: Lea & Febiger, 1982:587.
27. Ogden JA, Ogden DA, Pugh L, et al. Tibia valga after proximal metaphyseal fracture in childhood: a normal biologic response. *J Pediatr Orthop* 1995;15:489–494.
28. Pollen AG. *Fractures and dislocations in children*. Baltimore: Williams & Wilkins, 1973:179.
29. Rang M. Tibia. In: *Children's fractures*, 2nd ed. Philadelphia: JB Lippincott, 1983:189.
30. Robert M, Khouri N, Carlizo H, et al. Fractures of the proximal tibial metaphysis in children: review of a series of 25 cases. *J Pediatr Orthop* 1987;7:444.
31. Rooker G, Salter R. Presentation of valgus deformity following fracture of the proximal metaphysis of the tibia in children. *J Bone Joint Surg [Br]* 1980;62:527.
32. Salter RB, Best T. The pathogenesis and prevention of valgus deformity following fractures of the proximal metaphyseal region in the tibia in children. *J Bone Joint Surg [Am]* 1973;55:1324.
33. Skak S. Valgus deformity following proximal tibial metaphyseal fracture in children. *Acta Orthop Scand* 1982;53:141.
34. Steel HH, Sandrow RE, Sullivan PD. Complications of tibial osteotomy in children for genu varum or valgum. *J Bone Joint Surg [Am]* 1971;53:1629.
35. Taylor SL. Tibial overgrowth: a cause of genu valgum. *J Bone Joint Surg [Am]* 1963;45:659.
36. Weber BG. Fibrous interposition causing valgus deformity after fracture of the upper tibial metaphysis in children. *J Bone Joint Surg [Br]* 1977;59:290.
37. Zions LE, Harcke HT, Brooks KM, et al. Post traumatic tibia valga: a case demonstrating asymmetric activity at the proximal growth plate on technetium bone scan. *J Pediatr Orthop* 1977;7:458.
38. Zions LE, MacEwen GD. Spontaneous improvement of post traumatic tibia valga. *J Bone Joint Surg [Am]* 1986;68:680.

#### Diaphyseal Fractures

39. Allen MJ, Nash JR, Ioannides TT, et al. Major vascular surgeries associated with orthopaedic injuries to the lower limb. *Ann R Coll Surg Engl* 1984;66:101.
40. Bartlett GS III, Weiver LS, Yang EC. Treatment of type II and type III open tibia fractures in children. *J Orthop Trauma* 1997;11:357–362.
41. Bennek J, Steinert V. Knochenwachstum kindern. *Zentralbl Chir* 1966;91:633.
42. Benz G, Kallieris D, Seebock T, et al. Bio-reabsorbable pins and screws in pediatric traumatology. *Eur J Pediatr Surg* 1994;4:103–107.
43. Blasier RD, Barnes CL. Age as a prognostic factor in open tibial fractures in children. *Clin Orthop* 1996;331:261–264.
44. Blick SS, Brumback RJ, Poka A, et al. Compartment syndrome in open tibial fractures. *J Bone Joint Surg [Am]* 1986;68:1348.
45. Bohn WW, Durbin RA. Ipsilateral fractures of the femur and tibia in children and adolescents. *J Bone Joint Surg [Am]* 1991;73:429.
46. Briggs TWR, Orr MM, Lightowler CDR. Isolated tibial fractures in children. *Injury* 1992;23:308.
47. Buckley SL, Smith G, Sponseller PD, et al. Open fractures of the tibia in children. *J Bone Joint Surg [Am]* 1990;72:1462.
48. Byrd HS, Spicer PJ, Cierney G. Management of open tibial fractures. *Plast Reconstr Surg* 1985;76:719.
49. Caudle RJ, Stern PJ. Severe open fractures of the tibia. *J Bone Joint Surg [Am]* 1987;69:801.
50. Cierny G, Byrd HS, Jones RE. Primary versus delayed tissue coverage for severe open tibial fractures: a comparison of results. *Clin Orthop* 1983;178:54.
51. Clancey GJ, Hansen ST Jr. Open fractures of the tibia: a review of one hundred and two cases. *J Bone Joint Surg [Am]* 1978;60:118.
52. Cullen MC, Roy DR, Crawford AH, et al. Open fractures of the tibia in children. *J Bone Joint Surg [Am]* 1996;78:1039–1047.
53. DeLee JC, Strehl JB. Open tibia fracture with compartment syndrome. *Clin Orthop* 1981;160:175.
54. Demetriades D, Nikolaider N, Filiopoulos K, et al. The use of methylmethacrylate as an external fixator in children and adolescents. *J Pediatr Orthop* 1995;15:499–503.
55. Dias LS. Ankle valgus in children with myelomeningocele. *Dev Med Child Neuro*. 1978;20:627.
56. Edwards CC. Staged reconstruction of complex open tibial fractures using Hoffmann external fixation. *Clin Orthop* 1983;178:130.
57. Evanoff M, Strong ML, MacIntosh R. External fixation maintained until fracture consolidation in the skeletally immature. *J Pediatr Orthop* 1983;13:98.
58. Gates JD. The management of combined skeletal and arterial injuries of the lower extremity. *Am J Orthop* 1995;24:674–680.
59. Greiff J, Bergmann F. Growth disturbance following fracture of the tibia in children. *Acta Orthop Scand* 1980;15:315.
60. Grimard G, Navdie D, Laberge LC, et al. Open fractures of the tibia in children. *Clin Orthop* 1996;332:62–70.
61. Gustillo RB, Anderson JT. Prevention of infection in the treatment of one-thousand and twenty-five fractures of long bones. *J Bone Joint Surg [Am]* 1976;58:453.
62. Gustillo RB, Mendoza RM, Williams PN. Problems in the management of type III (severe) open fractures: a new classification of type III open fractures. *J Trauma* 1984;24:742.
63. Haas LM, Staple TW. Arterial injuries associated with fractures of the proximal tibia following blunt trauma. *South Med J* 1969;62:1439.
64. Hansen ST. Internal fixation of children's fractures of the lower extremity. *Orthop Clin North Am* 1990;21:353.
65. Harman JW, Guinn RP. The recovery of skeletal muscle fibers from acute ischemia as determined by histologic and chemical methods. *Am J Pathol* 1948;25:751.
66. Hoaglund FT, States JD. Factors influencing the rate of healing in tibial shaft fractures. *Surg Gynecol Obstet* 1967;124:71.
67. Holderman WD. Results following conservative treatment of fractures of the tibial shaft. *Am J Surg* 1959;98:593.
68. Hoover NW. Injuries of the popliteal artery associated with fractures and dislocations. *Surg Clin North Am* 1961;41:1099.
69. Hope PG, Cole WG. Open fractures of the tibia in children. *J Bone Joint Surg [Br]* 1992;74:546.
70. Hull JB, Sanderson PL, Rickman M, et al. External fixation of children's fractures: use of the Orthofix Fynamic Axial Fixator. *J Pediatr Orthop* 1997;6:203–206.
71. Karlstrom G, Lonnerholm T, Olerud S. Cavus deformity of the foot after fracture of the tibial shaft. *J Bone Joint Surg [Am]* 1975;57:893.
72. Katzman SS, Dickson K. Determining the prognosis for limb salvage in major vascular injuries with associated open tibial fractures. *Orthop Rev* 1992;21:195.
73. Klein DM, Caligiuri, DA, Katzman BM. Local-advancement soft-tissue coverage in a child with ipsilateral grade IIIB open tibial and ankle fractures. *J Orthop Trauma* 1996;10:577–580.
74. Knight JL. Genu recurvatum deformity secondary to partial proximal tibial epiphyseal arrest. *Am J Knee Surg* 1998;11:111–115.
75. Kozlowski K, Azouz M, Barrett IR, et al. Midshaft tibial stress fractures in children. *Aust Radiol* 1992;36:131–134.
76. Kramer KE, Limbird TJ, Green NE. Open fractures of the diaphysis of the lower extremity in children. *J Bone Joint Surg [Am]* 1992;74:218.
77. Kreder HJ, Armstrong P. A review of open tibia fractures in children. *J Pediatr Orthop* 1995;15:482–488.
78. Larsson K, van der Linden W. Open tibial shaft fractures. *Clin Orthop* 1983;180:63.
79. Leach RE, Hammond G, Stryker WS. Anterior tibial compartment syndrome. *J Bone Joint Surg [Am]* 1967;49:451.
80. Letts M, Vincent M. The "floating knee" in children. *J Bone Joint Surg [Br]* 1986;68:442.
81. Levy AS, Wetzlan M, Lewars M, et al. The orthopaedic and social outcome of open tibia fractures in children. *Orthopaedics* 1997;20:593–598.
82. Matsen FA III, Clawson DK. The deep posterior compartmental syndrome of the leg. *J Bone Joint Surg [Am]* 1975;57:34.
83. Morton KS, Starr DE. Closure of the anterior portion of the upper tibial epiphysis as a complication of tibial-shaft fracture. *J Bone Joint Surg [Am]* 1964;46:570.
84. Nicoll EA. Fractures of the tibial shaft, a survey of 705 cases. *J Bone Joint Surg [Br]* 1964;46:373.
85. Ostermann PAW, Henry SL, Seligson D. Timing of wound closure in severe compound fractures. *Orthopedics* 1994;17:397.
86. Patzakis MJ, Wilkins J, Moore TM. Used antibiotics in open tibial fractures. *Clin Orthop* 1983;118:31.
87. Robertson P, Karol CA, Rab GT. Open fractures of the tibia and femur in children. *J Pediatr Orthop* 1996;16:621–626.
88. Russel GG, Henderson R, Arnett G. Primary or delayed closure for open tibial fractures. *J Bone Joint Surg [Br]* 1990;72:125.
89. Sarmiento A. A functional below-the-knee cast for tibial fractures. *J Bone Joint Surg [Am]* 1967;49:855.
90. Schrock RD. Peroneal nerve palsy following derotation osteotomies for tibial torsion. *Clin Orthop* 1969;62:172.
91. Scully RE, Shannon JM, Dickerson JR. Factors involved in recovery from experimental skeletal ischemia in dogs. *Am J Pathol* 1961;39:721.
92. Siegmeth A, Wruhs O, Vecsei V. External fixation of lower limb fractures in children. *Eur J Pediatr Surg* 1998;8:35–41.

93. Small JO, Mollan RAB. Management of the soft tissues in open tibial fractures. *Br J Plast Surg* 1992;45:571.
94. Smillie IS. *Injuries of the knee joint*, 2nd ed. Baltimore: Williams & Wilkins, 1951.
95. Song KM, Sangeorzan B, Benirschke S, et al. Open fractures of the tibia in children. *J Pediatr Orthop* 1996;16:635–638.
96. Steinert VV, Bennek J. Unterschenkelfrakturen im Kindesalter. *Zentralbl Chir* 1966;91:1387.
97. Swaan JW, Oppers VM. Crural fractures in children. *Arch Chir Neen* 1971;23:259.
98. Van der Werkon C, Meeuwis JD, Oostuogel HJM. The simple fix: external fixation of displaced isolated tibial fractures. *Injury* 1993;24:46.
99. Whitesides TE Jr, Haney TC, Morimoto K, et al. Tissue pressure measurements as a determinant for the need of fasciotomy. *Clin Orthop* 1975;113:43.
100. Wood D, Hoffer MH. Tibial fractures in head injured children. *J Trauma* 1987;27:65.
101. Yang J, Letts M. Isolated fractures of the tibia with intact fibula in children: a review of 95 patients. *J Pediatr Orthop* 1997;17:347–351.

#### Special Fractures

102. Berkebile RD. Stress fracture of the tibia in children. *AJR* 1964;91:588.
103. Burrows HJ. Fatigue fractures of the fibula. *J Bone Joint Surg [Br]* 1948;30:266.
104. DeBoeck K, van Eldere, DeVos P, et al. Radionuclide bone imaging in toddler's fracture. *Eur J Pediatr* 1991;150:166.
105. Devas MB. Stress fractures in children. *J Bone Joint Surg [Br]* 1963;45:528.
106. Devas MB, Sweetman R. Stress fracture of the fibula. *J Bone Joint Surg [Br]* 1956;38:818.
107. Drennan JC, Freehafer AA. Fractures of the lower extremities in paraplegic children. *Clin Orthop* 1971;77:211.
108. Dunbar JS, Owen HF, Nogrady MB, et al. Obscure tibial fracture of infants—the toddler's fracture. *J Can Assoc Radio* 1964;25:136.
109. Elton RL. Stress reaction of bone in army trainees. *JAMA* 1968;204:314.
110. Engh CA, Robinson RA, Milgram J. Stress fractures in children. *J Trauma* 1970;10:532.
111. Felman AH. Bicycle spoke fractures. *J Pediatr* 1973;82:302.
112. Freehafer AA, Mast WA. Lower extremity fractures in patients with spinal-cord injury. *J Bone Joint Surg [Am]* 1965;47:683.
113. Gillies CL, Hartung W. Fracture of the tibia in spina bifida vera. *Radiology* 1938;31:621.
114. Golding C. Museum pages. III: spina bifida and epiphyseal displacement. *J Bone Joint Surg [Br]* 1960;42:387.
115. Griffiths AL. Fatigue fracture of the fibula in childhood. *Arch Dis Child* 1952;27:552.
116. Gyepes MT, Newbern DH, Neuhauser EBD. Metaphyseal and physeal injuries in children with spina bifida and meningocele. *AJR* 1965;95:168.
117. Hartley JB. Fatigue fracture of the tibia. *Br J Surg* 1942;30:9.
118. Horev G, Korenreich L, Ziv N, et al. The enigma of stress fractures in the pediatric age: clarification or confusion through the new imaging modalities. *Pediatr Radio*. 1990;20:469.
119. Ingersoll CF. Ice skater's fracture. A form of fatigue fracture. *AJR* 1943;50:469.
120. Izant RJ, Rothman BF, Frankel V. Bicycle spoke injuries of the foot and ankle in children: an underestimated "minor" injury. *J Pediatr Surg* 1969;4:654.
121. James CCM. Fractures of the lower limbs in spina bifida cystica: a survey of 44 fractures in 122 children. *Dev Med Child Neurol Suppl* 1970;22:88.
122. Johnson LC. Morphologic analysis. In: Frost HM, ed. *Pathology in bone biodynamics*. Boston: Little, Brown, 1963.
123. Kozlowski K, Urbonaviciene A. Stress fracture of the fibula in the first few years of life (report of six cases). *Aust Radio*. 1996;40:261–263.
124. Kumar SJ, Lowell HR, Townsend P. Physeal, metaphyseal and diaphyseal injuries of the lower extremities in children with myelomeningocele. *J Pediatr Orthop* 1984;4:25.
125. Matin P. The appearance of bone scans following fractures, including immediate and long-term studies. *J Nucl Med* 1979;20:1227.
126. Mellick LB, Reesor K. Spiral tibial fractures of children: a commonly accidental spiral long bone fracture. *Am J Emerg Med* 1990;8:234.
127. Meurman KOA, Elfving S. Stress fracture in soldiers: a multifocal bone disorder. *Radiology* 1980;134:483.
128. Micheli LJ, Gerbino PG. Etiologic assessment of stress fractures of the lower extremity in young athletes. *Orthop Trans* 1980;4:1.
129. Oujhane K, Newman B, Oh KS, et al. Occult fractures in pre-school children. *Trauma* 1988;28:858.
130. Parsch K, Rossak K. Die Pathologischen Frakturen Bei Spina Bifida. *Arch DeVecchi Anat Pat* 1968;53:165.
131. Prather JL, Nusynowitz ML, Snowdy HA, et al. Scintigraphic findings in stress fractures. *J Bone Joint Surg [Am]* 1977;59:869.
132. Roberts SM, Vogt EC. Pseudofracture of the tibia. *J Bone Joint Surg* 1939;21:891.
133. Robin G. Fracture in childhood paraplegia. *Paraplegia* 1966;3:165.
134. Roub LW, Gumerman LW, Hanley EN, et al. Bone stress: a radionuclide imaging perspective. *Radiology* 1979;132:431.
135. Savoca CJ. Stress fractures. A classification of the earliest radiographic signs. *Radiology* 1971;100:519.
136. Sawmiller S, Michener WM, Hartman JT. Stress fracture in childhood. *Cleveland Clin Q* 1965;32:119.
137. Soutter FE. Spina bifida and epiphyseal displacement. *J Bone Joint Surg [Br]* 1962;44:106.
138. Stern MB, Grant SS, Isaacson AS. Bilateral distal tibial and fibular epiphyseal separation associated with spina bifida. *Clin Orthop* 1967;50:191.
139. Taunton JE, Clement DB, Webber D. Lower extremity stress fractures in athletes. *J Sports Med Phys Fitness* 1981;9:77.
140. Tenebein M, Reed MH. The Fodder's fracture revisited. *Am J Emerg Med* 1990;8:208.

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Injuries to the distal tibial and fibular physes are generally reported to account for 25% to 38% of all physeal fractures ( 57), second in frequency only to distal radial physeal fractures (93); however, Peterson et al. (94) reported that phalangeal physeal fractures were most common, followed by physeal injuries of the radius and ankle. In skeletally immature individuals, physeal ankle fractures are slightly more common than fractures of the tibial or fibular diaphysis ( 80). Up to 58% of physeal ankle fractures occur during sports activities ( 45,127) and account for 10% to 40% of all injuries to skeletally immature athletes ( 87,91,115). Physeal ankle fractures are more common in males than in females (112). Tibial physeal fractures most commonly occur between the ages of 8 and 15 years, and fibular fractures between the ages of 8 and 14 years (112).

Although Foucher (39) reported the first pathologic study of these injuries in 1863, Poland's 1898 monograph ( 97) is generally recognized as the most extensive early study of physeal fractures. He pointed out that in children ligaments are stronger than physeal cartilage; forces that result in ligament damage in adults cause fractures of the physes in children. In 1922, Ashhurst and Bromer ( 4) published a thorough review of the literature and the results of their own extensive investigations and described a classification of ankle injuries based on the mechanism of injury. This classification did not differentiate between ankle injuries in adults and those in children. Bishop (8) in 1932 classified 300 ankle fractures according to Ashhurst and Bromer's system; 33 fractures were physeal injuries, and the grouping of these injuries according to mechanism of injury represents one of the first attempts to classify physeal ankle injuries.

Aitken's (2) study of 21 physeal ankle injuries in 1936 is one of the first to attempt to determine the results of treatment of these injuries; he also outlined an anatomic classification. Only one of his patients (5%) had a deformity after fracture, in contrast to McFarland ( 82), who in 1932 reported deformities in 40% of a larger series of patients. In 1955, Caruthers and Crenshaw (21) reported 54 physeal ankle fractures, which were classified according to their modification of Ashhurst and Bromer's system. They confirmed that growth-related deformities were frequent after adduction (Salter-Harris type III and IV injuries) fractures and infrequent after fractures caused by external rotation, abduction, and plantarflexion (Salter-Harris type II injuries). Spiegel and colleagues ( 112), in a 1978 review of 237 physeal ankle fractures, reported a high incidence of growth abnormalities after Salter-Harris type III and IV injuries but also found complications in 11 (16.7%) of 66 patients with Salter-Harris type II fractures. Most of these patients had only mild shortening, but 6 had angular deformities that did not correct with growth. Based on the results of 65 physeal ankle fractures, Kling and co-workers (69) concluded that the frequency of growth-related deformities could be reduced by open reduction and internal fixation of Salter-Harris III and IV fractures.

A separate group of fractures, occurring in adolescents and known as transitional fractures, has been identified. Such fractures, which include juvenile Tillaux and triplane fractures with two to four fracture fragments, have been described by Kleiger and Mankin ( 67), Marmor (81), Cooperman and co-workers (27), Karrholm (61), and Denton and Fischer (31).

## DIAGNOSIS

Patients with significantly displaced fractures have severe pain and obvious deformity. The position of the foot relative to the leg may provide important information about the mechanism of injury (Fig. 25-1) and should be considered in planning reduction. The status of the skin, pulses, and sensory and motor function should be determined and recorded. Tenderness, swelling, and deformity in the ipsilateral leg and foot should be noted. In patients with tibial shaft fractures, the ankle should be carefully evaluated clinically and radiographically.



**FIGURE 25-1.** Severe clinical deformity in a 14-year-old boy with an ankle fracture. It is obvious without radiographs that internal rotation will be needed to reduce this fracture.

Patients with nondisplaced or minimally displaced ankle fractures often have no deformity, minimal swelling, and moderate pain. Because of their benign clinical appearance, such fractures may be easily missed if radiographs are not obtained. The fact that all physicians have an obligation to try to minimize unnecessary radiographs, both to avoid unnecessary irradiation of patients and for cost containment, creates a dilemma for physicians evaluating pediatric ankle injuries. Guidelines known as the Ottawa Ankle Rules have been established for adults to try to determine which injuries require radiographs ( 114). The indications for radiography according to the guidelines are complaints of pain near a malleolus with either inability to bear weight or tenderness to palpation at the malleolus. Chande prospectively studied 71 children with acute ankle injuries to determine if these guidelines could be applied to pediatric patients with ankle injuries ( 25). It was determined that if radiographs were obtained only in children who had tenderness over the malleoli and an inability to bear weight, a 25% reduction in radiographic

examinations could be achieved without missing any fractures. Therefore, a systematic search for localized tenderness, keeping in mind the physal anatomy of the ankle, is helpful in determining when to order radiographs as well as how to interpret them.

For patients with obvious deformities, anteroposterior and lateral radiographs centered over the ankle may provide sufficient information to plan treatment. Although obtaining views of the joint above and below is recommended for most fractures, obtaining a film centered over the mid-tibia to include the knee and ankle joints on the radiograph significantly decreases the quality of ankle views and is not recommended.

For patients without obvious deformities, a high-quality mortise view of the ankle is essential in addition to anteroposterior and lateral views. On a standard anteroposterior view, the lateral portion of the distal tibial physis is usually partially obscured by the distal fibula. The vertical component of a triplane or Tillaux fracture can be hidden behind the overlying fibular cortical shadow ( 73). A study by Vangsness and co-workers (121) found that diagnostic accuracy was essentially equal when using anteroposterior, lateral, and mortise views compared with using only mortise and lateral views. Therefore, if only two views are to be obtained, the anteroposterior view may be omitted and lateral and mortise views obtained.

Haraguchi et al. described two special views designed to detect avulsion fractures from the lateral malleolus that are not visible on routine views and to distinguish whether they represent avulsions of the anterior tibiofibular ligament or the calcaneofibular ligament attachments ( 49). The anterior tibiofibular ligament view is made by positioning the foot in 45 degrees of plantar flexion and elevating the medial border of the foot 15 degrees. The calcaneofibular ligament view is obtained by rotating the leg 45 degrees inward.

Stress views may occasionally be needed to rule out ligamentous instability ( Fig. 25-2), although ligamentous injury at the ankle is infrequent in skeletally immature patients. Stress views may be considered to document a Salter-Harris type I fracture, but a patient with clinical signs of this fracture should be treated appropriately, regardless of stress view findings. Stress views may help document lack of motion at the fracture in an apparent nonunion ( Fig. 25-3).



**FIGURE 25-2. A:** Stress radiograph showing abnormal varus tilt in a 5-year-old who had sustained a left ankle injury. Note the small avulsion fracture from the talus (arrow) and soft tissue swelling laterally. **B:** Comparative stress radiograph of the right ankle.



**FIGURE 25-3. A:** Anteroposterior radiograph of a 10-year-girl after 12 weeks of immobilization for an initially nondisplaced Salter-Harris type III fracture of the distal tibia. **B:** Stress view showing no motion at the fracture site.

Bozic et al. studied the age at which the radiographic appearance of the incisura fibularis, tibiofibular clear space, and tibiofibular overlap develop in children ( 15). The purpose of their study was to facilitate the diagnosis of distal tibiofibular syndesmotc injury in children. They found that the incisura became detectable at a mean age of 8.2 for girls and 11.2 years for boys. The mean age at which tibiofibular overlap appeared on the anteroposterior view was 5 years for both sexes; on the mortise view, it was 10 years for girls and 16 years for boys. The range of clear space measurements in normal children was 2 to 8 mm, with 23% of children having a clear space of greater than 6 mm, a distance considered abnormal in adults.

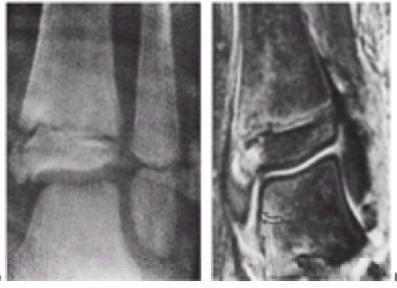
Computed tomography (CT) is useful in the evaluation of intraarticular fractures, especially juvenile Tillaux and triplane fractures ( Fig. 25-4) (54). In the past, some physicians preferred plain tomograms ( 126). A lower cost appears to be their only advantage over CT scans. CT evaluations involve less radiation to the patient and in many institutions are now easier to obtain than are plain tomograms. Cuts are generally made in the transverse plane. With thin cuts localized to the joint, it is possible to generate high-quality reconstructions that allow evaluation in the coronal and sagittal planes without repositioning the ankle. With plain tomography the transverse anatomy can only be deduced from the anteroposterior and lateral tomograms. Three-dimensional CT reconstructions rarely add further useful information. Other indications for CT scanning are discussed in the section on Treatment.



**FIGURE 25-4. A:** Anteroposterior view of a 13-year-old boy with a tibial fracture extending into the ankle joint. It is unclear on this view whether the metaphyseal fracture is a separate injury or part of the intraarticular fracture. **B:** Transverse CT scan a few millimeters above the ankle joint confirmed the triplane nature of this injury. **C:** Anteroposterior reconstruction from the CT scan shows 3 mm of displacement at the fracture site in the tibial epiphysis.

Magnetic resonance imaging (MRI) has been reported to be occasionally helpful in the identification of osteochondral injuries to the joint surfaces in children with ankle fractures (66). Smith and associates (111) found that of four patients with acute (3–10 days) physal injuries, MRI showed that three had more severe fractures

than indicated on plain films ([Fig. 25-5](#)). Early MRI studies (3–17 weeks after injury) not only added information about the pattern of physal disruption but also supplied early information about the possibility of growth abnormality.



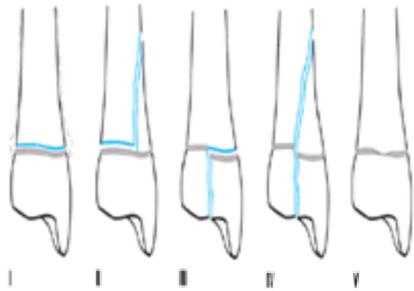
**FIGURE 25-5.** **A:** Follow-up radiograph of a 7-year-old boy 1 week after an initially nondisplaced Salter-Harris type III fracture from a supination–inversion injury of the distal tibia. **B:** Because of the incomplete ossification of this area and concern that the fracture might have displaced, magnetic resonance imaging was performed. Note that the distance between the medial malleolus and the talus is greater than the distance between the talus and the distal tibia or lateral malleolus, confirming displacement of the fracture.

Carey et al. obtained MRI studies on 14 patients with known or suspected growth plate injury ([20](#)). The MRI detected five radiographically occult fractures in the 14 patients, changed the Salter-Harris classification in 2 patients, and resulted in a change in treatment plan in 5 of the 14 patients studied. These studies would seem to contradict an earlier study by Petit et al. that showed only 1 patient in a series of 29 patients in whom MRI revealed a diagnosis different from that made on plain films ([96](#)). Later (>6 months after injury) scans have been reported useful for mapping physal bars ([42,52](#)).

### MECHANISM OF INJURY AND CLASSIFICATION

Classifications of ankle fractures are of two broad types: anatomic ([2,89,95,106,112](#)) and mechanism-of-injury ([4,8,71](#)). Anatomic classifications divide fractures into groups based on the parts of the epiphyses or metaphyses that make up the fracture fragments. Mechanism-of-injury classifications are based on the nature of the force that creates the fractures and often include the position of the foot at the time the force is applied. Most mechanism-of-injury classifications include the anatomic type of injury produced by a particular mechanism.

Anatomic classifications ([Fig. 25-6](#)) are effective for rapid communication because most have few groups without multiword titles that require visualization of movements that sometimes seem incompatible with the position of the foot on the ground. Because they are applicable to all physes, anatomic classifications are used more frequently and are easier to recall than mechanism-of-injury classifications. Mechanism-of-injury classifications are considered by some to be more precise because they reflect not only the anatomic fracture pattern but also the position of the fragments in relation to each other. This increased precision, however, may result in less rapid and possibly more confusing communication.



**FIGURE 25-6.** Salter-Harris anatomic classification as applied to injuries of the distal tibial epiphysis.

Both anatomic and mechanism-of-injury classifications can provide useful information for determining appropriate treatment. The prognoses for growth and deformity have been predicted on the basis of both types of classification ([62,63,112](#)). A theoretical advantage of mechanism-of-injury classifications is that identification of the force producing the injury might give even more information about the possible development of growth arrest than anatomic classifications. For example, a Salter-Harris type III or IV fracture of the tibia produced by a shearing or crushing force might be more likely to result in growth arrest than is a similar injury produced by an avulsion force ([Fig. 25-7](#)). However, it is difficult to establish that one type of classification is superior to the other in this regard because of the relatively small numbers of patients reported, the varying ages of patients in most series, and questions about the reproducibility of various classifications.



**FIGURE 25-7.** Comminuted Salter-Harris type IV fracture of the distal tibia and displaced Salter-Harris type I fracture of the distal fibula produced by an inversion (shearing) mechanism in a 10-year-old girl.

An important benefit of mechanism-of-injury classifications ([Table 25-1](#)) is that they provide clues that may be helpful in planning closed reduction: the mechanism of injury is simply reversed to reduce the fracture. Surgeons who rely on anatomic classifications usually attempt to obtain reduction by returning the displaced fragments directly to their proper anatomic locations. In reality, surgeons who use either classification probably reduce most fractures in similar manners.

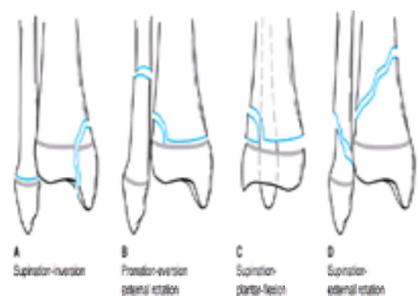
		Applied Force				
<b>Alford-Bower (Adult)</b>	Supination	Abduction	Abduction	—	Compression	—
<b>Greifens-Cookley (Adult)</b>	Supination	Abduction	Abduction	Plantarflexion	Compression	—
<b>Lauge-Hansen (Adult)</b>	Supination	Pronation	Supination	—	—	Pronation
	Inversion	Abduction	Adduction	—	Compression	Distraction
	External rotation					Internal rotation
<b>Dias-Tachdjian (Child)</b>	Supination	Pronation	Supination	Supination	Plantarflexion	—
	Internal rotation	Inversion	Inversion	Plantarflexion	—	—
	External rotation	Abduction	Adduction	—	—	—

Lauge-Hansen: supination, external rotation of foot, abduction of foot/heel, and eversion of foot/heel. Lauge-Hansen: supination, external rotation of foot, abduction of foot/heel, and eversion of foot/heel.

**TABLE 25-1. REPRESENTATIVE MECHANISM OF INJURY CLASSIFICATIONS**

Any classification system that cannot be easily remembered and reproducibly applied will ultimately prove to be less than useful. Thomsen and co-workers (118) studied the reproducibility of the Lauge-Hansen (mechanism-of-injury) and Weber (anatomic) classifications in a series of adult ankle fractures. After all investigators in the study had received a tutorial on both systems and their application, they were asked to classify 94 fractures. On the first attempt, only the Weber classification produced an acceptable level of interobserver agreement. On a second attempt, the Weber classification and most of the Lauge-Hansen classification achieved an acceptable level of interobserver agreement. These investigators concluded that all fracture classification systems should have demonstrably acceptable interobserver agreement rates before they are adopted, an argument made even more forcefully in an editorial by Burstein (18). Vahvanen and Aalto (119) compared their ability to classify 310 ankle fractures in children with the Weber, Lauge-Hansen, and Salter-Harris classifications. They found that they were “largely unsuccessful” using the Weber and Lauge-Hansen classifications but could easily classify the fractures using the Salter-Harris system.

The most widely accepted mechanism-of-injury classification of ankle fractures in children is that described by Dias and Tachdjian (34) (Fig. 25-8), who modified the Lauge-Hansen classification based on their review of 71 fractures. Their original classification (1978) consisted of four types in which the first word refers to the position of the foot at the time of injury and the second word refers to the force that produces the injury. Four other types were subsequently added (116) (in 1985). Although these are designated differently, the first three have identifiable mechanisms of injury. *Axial compression injury* describes the mechanism of injury but not the position of the foot. *Juvenile Tillaux and triplane fractures* are believed to be caused by external rotation. The final category, *other physeal injuries*, includes diverse injuries, many of which have no specific mechanism of injury.



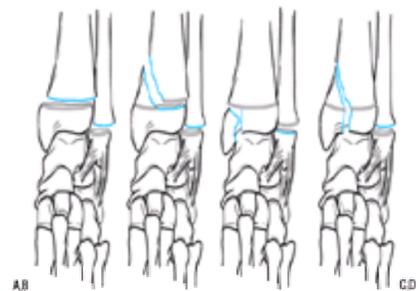
**FIGURE 25-8.** Dias-Tachdjian classification of physeal injuries of the distal tibia and fibula.

### Classification of Ankle Fracture in Children (Dias-Tachdjian)

#### Supination–Inversion

Grade I: The adduction or inversion force avulses the distal fibular epiphysis (Salter-Harris type I or II fracture). Occasionally, the fracture is transepiphyseal; rarely, the lateral ligaments fail.

Grade II (Fig. 25-9): Further inversion produces a tibial fracture, usually a Salter-Harris type III or IV and rarely a Salter-Harris type I or II injury, or the fracture passes through the medial malleolus below the physis (Fig. 25-10).



**FIGURE 25-9.** Variants of grade II supination–inversion injuries (Dias-Tachdjian classification). **A:** Salter-Harris I fracture of the distal tibia and fibula. **B:** Salter-Harris I fibula, Salter-Harris II tibia fractures. **C:** Salter-Harris I fibula, Salter-Harris III tibia fractures. **D:** Salter-Harris I fibula, Salter-Harris IV tibia fractures.



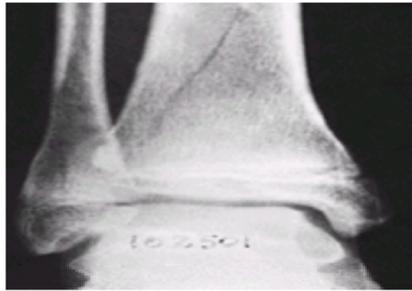
**FIGURE 25-10.** Severe supination–inversion injury with displaced fracture of the medial malleolus distal to the physis of the tibia.

### **Supination–Plantarflexion**

The plantarflexion force displaces the epiphysis directly posteriorly, resulting in a Salter-Harris type I or II fracture. Fibular fractures were not reported with this mechanism. The tibial fracture usually is difficult to see on anteroposterior radiographs.

### **Supination–External Rotation**

Grade I: The external rotation force results in a Salter-Harris type II fracture of the distal tibia ( [Fig. 25-11](#)). The distal fragment is displaced posteriorly, as in a supination–plantarflexion injury, but the Thurston-Holland fragment is visible on the anteroposterior radiograph, with the fracture line extending proximally and medially. Occasionally, the distal tibial epiphysis is rotated but not displaced.



**FIGURE 25-11.** Stage I supination–external rotation injury in a 10-year-old child; the Salter-Harris type II fracture begins laterally.

Grade II: With further external rotation, a spiral fracture of the fibula is produced, running from anteroinferior to posterosuperior ( [Fig. 25-12](#)).



**FIGURE 25-12.** Stage II supination–external rotation injury. **A:** Oblique fibular fracture is also visible on anteroposterior view. **B:** Lateral view shows the posterior metaphyseal fragment and posterior displacement.

### **Pronation–Eversion–External Rotation**

A Salter-Harris type I or II fracture of the distal tibia occurs simultaneously with a transverse fibular fracture. The distal tibial fragment is displaced laterally and the Thurston-Holland fragment, when present, is lateral or posterolateral ( [Fig. 25-13](#)). Less frequently, a transepiphyseal fracture occurs through the medial malleolus (Salter-Harris type II). Such injuries may be associated with diastasis of the ankle joint, which is uncommon in children.



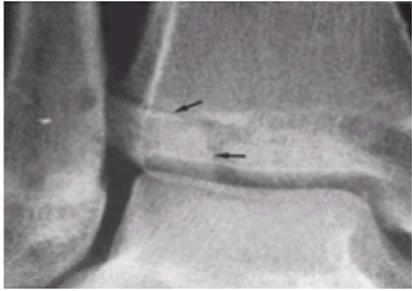
**FIGURE 25-13. A:** According to the Dias-Tachdjian classification, this injury in a 12-year-old boy would be considered a pronation–eversion–external rotation injury resulting in a Salter-Harris type II fracture of the distal tibia and a transverse fibular fracture. **B:** The anterior displacement of the epiphysis, visible on the lateral view, however, makes external rotation an unlikely component of the mechanism of injury; the mechanism is more likely pronation–dorsiflexion.

### **Axial Compression**

This is a Salter-Harris type V injury of the distal tibial physis. Initial radiographs usually show no abnormality, and the diagnosis is established when growth arrest is demonstrated on follow-up radiographs.

### **Juvenile Tillaux Fracture**

This is a Salter-Harris type III fracture involving the anterolateral distal tibia. The portion of the physis not involved in the fracture is closed ( [Fig. 25-14](#)).



**FIGURE 25-14.** Salter-Harris type III fracture (juvenile Tillaux) ( *arrows*) of the anterolateral portion of the distal tibial epiphysis in a 13-year-old girl.

### ***Triplane Fracture***

This group of fractures have the appearance of a Salter-Harris type III fracture on the anteroposterior radiograph and of a Salter-Harris type II fracture on the lateral radiograph.

### ***Other Physeal Injuries***

These are the fractures that do not fit into any of the other seven types (such as injuries to the perichondral ring and stress fractures) ( [Fig. 25-15](#)).



**FIGURE 25-15.** Stress fracture of the distal tibia in normal 13-year-old child who complained of pain in his ankle after running. **A:** Initial radiographs were interpreted as being normal, although abnormal widening of the tibial physis was present. The ankle was immobilized for 10 days. **B:** Six weeks later, pain persisted and radiographs showed further widening and irregularity of the physis, with a faint periosteal new bone formation ( *arrows*) around the distal tibial metaphysis.

### ***Unusual Fracture Patterns***

A number of accessory ossification centers and normal anatomic variations may cause confusion in the interpretation of plain films of the ankle ( [Fig. 25-16](#)). In a group of 100 children between the ages of 6 and 12 years, Powell (99) found accessory ossification centers on the medial side (os subtibiale) in 20% and on the lateral side (os subfibulare) in 1%. If they are asymptomatic on clinical examination, these ossification centers are of little concern, but tenderness localized to them may indicate an injury. Stress views to determine motion of the fragments or bone scanning may occasionally be considered if an injury to an accessory ossification center is suspected.



**FIGURE 25-16.** Secondary ossification center in the lateral malleolus ( *arrows*) of a 10-year-old girl. Note the smooth border of the fibula and the ossification center. She also has a secondary ossification center in the medial malleolus.

Clefts in the lateral side of the tibial epiphysis may simulate juvenile Tillaux fractures, and clefts in the medial side may simulate Salter-Harris type III fractures ( [65](#)). The presence of these clefts on a radiograph of a child with an ankle injury may result in overtreatment if they are misdiagnosed as a fracture. Conversely, attributing a painful irregularity in these areas to anatomic variation may lead to undertreatment ( [Fig. 25-17](#)). Other anatomic variations include a bump on the distal fibula that simulates a torus fracture and an apparent offset of the distal fibular epiphysis that simulates a fracture.



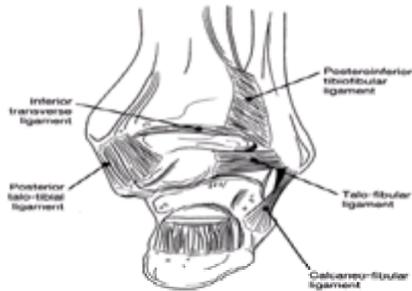
**FIGURE 25-17. A:** Mortise view of the ankle of a 10-year-old girl who had slight swelling and tenderness at the medial malleolus after an “ankle sprain.” The ossicle at the tip of the medial malleolus was correctly identified as an os subtibiale. A subtle line extending from the medial physis to just distal to the medial tibial plafond (*arrow*) was believed to also be an anatomic variant. **B:** Four weeks after injury, soreness persisted and radiography clearly demonstrated a Salter-Harris type III

fracture.

## ANATOMY

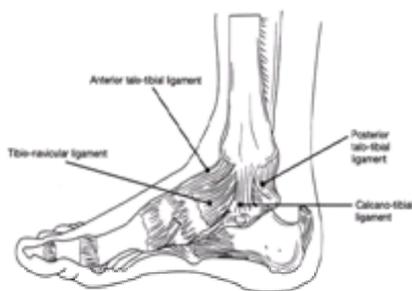
The ankle is the joint that most closely approximates a hinge joint. It is the articulation between the talus and the ankle mortise, which is a syndesmosis consisting of the distal tibial articular surface, the medial malleolus, and the distal fibula or lateral malleolus.

Ligamentous structures bind the distal tibia and fibula into the ankle mortise ( [Fig. 25-18](#)). The anterior and posterior inferior tibiofibular ligaments course inferiorly from the anterior and posterior surfaces of the distal lateral tibia to the anterior and posterior surfaces of the lateral malleolus. The anterior ligament is important in the pathomechanics of transitional ankle fractures. Just anterior to the posteroinferior tibiofibular ligament is the broad, thick inferior transverse ligament, which extends down from the lateral malleolus along the posterior border of the articular surface of the tibia, almost to the medial malleolus. This ligament serves as a part of the articular surface for the talus. Between the anterior and posterior inferior tibiofibular ligaments, the tibia and fibula are bound by the interosseous ligament, which is continuous with the interosseous membrane above. This ligament may be important in the pathomechanics of what we have termed incisural fractures.

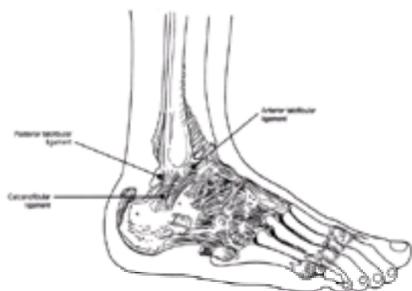


**FIGURE 25-18.** Posterior view of the distal tibia and fibula and the ligaments making up the ankle mortise.

On the medial side of the ankle, the talus is bound to the ankle mortise by the deltoid ligament ( [Fig. 25-19](#)). This ligament arises from the medial malleolus and divides into superficial and deep layers. Three parts of the superficial layer are identified by their attachments: tibionavicular, calcaneotibial, and posterior talotibial ligaments. The deep layer is known as the anterior talotibial ligament, again reflecting its insertion and origin. On the lateral side, the anterior and posterior talofibular ligament, with the calcaneofibular ligaments, make up the lateral collateral ligament ( [Fig. 25-20](#)).



**FIGURE 25-19.** Medial view of the ankle demonstrating the components of the deltoid ligament.



**FIGURE 25-20.** Lateral view of the ankle demonstrating the anterior and posterior talofibular ligaments and the calcaneofibular ligament.

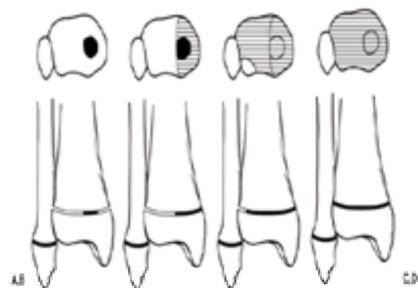
In children, all of the ligamentous structures that bind the medial and lateral malleoli to the talus and the distal tibial epiphysis to the distal fibular epiphysis are attached to the malleoli distal to the physes. Because the ligaments are stronger than the physes, physeal fractures are more common than ligamentous injuries in children. When they accompany distal tibial physeal injuries, displaced diaphyseal fibular fractures are usually associated with injuries to and displacement of the entire distal tibial epiphysis rather than with injuries to the ligaments, making diastasis of the ankle uncommon in children ( [Fig. 25-21](#)).



**FIGURE 25-21.** **A:** Pronation–external rotation injury resulting in a Salter-Harris type I fracture of the distal tibial physis. **B and C:** Anteroposterior and lateral

radiographs demonstrate satisfactory closed reduction.

The distal tibial ossification center generally appears at 6 to 24 months of age. Its malleolar extension begins to form around the age of 7 years and is mature or complete at the age of 10 years. The physis usually closes around the age of 15 years in girls and 17 years in boys. This process takes approximately 18 months and occurs first in the central part of the physis, extending next to the medial side, and finally ending laterally. This asymmetric closure sequence is an important anatomic feature of the growing ankle and is responsible for certain fracture patterns in adolescents ( [Fig. 25-22](#)).



**FIGURE 25-22.** Closure of the distal tibial physis begins centrally (**A**), and extends medially (**B**) and then laterally (**C**) before final closure (**D**).

The distal fibular ossification center appears around the age of 9 to 24 months. Its physis is located at the level of the ankle joint. Closure of this physis generally follows closure of the distal tibial physis by 12 to 24 months.

## TREATMENT

Appropriate treatment of ankle fractures in children depends on the location of the fracture, the degree of displacement, and the age of the child. Nondisplaced fractures may be simply immobilized. Closed reduction and cast immobilization may be appropriate for displaced fractures; if the closed reduction cannot be maintained with casting, skeletal fixation is necessary. If closed reduction is not possible, open reduction is indicated, provided there is significant physeal or articular displacement, followed by internal fixation or cast immobilization.

The anatomic type of the fracture (usually defined by the Salter-Harris classification), the mechanism of injury, and the amount of displacement of the fragments are important considerations. When the articular surface is disrupted, the amount of articular step-off or separation must be measured. The neurovascular status of the limb or the status of the skin may require emergency treatment of the fracture and associated problems. The general health of the patient and the time since injury also must be considered.

Other considerations in treatment decision making include the following:

Is a below-knee or above-knee cast appropriate?

How long should immobilization be continued?

When can weight bearing be allowed?

How important is immediate reduction of the ankle fracture, especially if other injuries or logistical problems exist?

Is a general anesthetic required or can some form of local anesthesia or sedation be as effective and safer for closed reduction?

If closed reduction is incomplete, how much residual displacement in each plane is acceptable?

If open reduction is necessary, what surgical approach is appropriate?

How can the most anatomic reduction be ensured at the time of surgery?

What type of internal fixation device is most appropriate?

## Distal Tibial Physeal Fractures

### Salter-Harris Type I Fractures

According to Dias and Tachdjian ([34,116](#)), Salter-Harris type I fractures of the distal tibia can be caused by any of four mechanisms: supination–inversion, supination–plantarflexion, supination–external rotation, or pronation–eversion–external rotation. Spiegel and associates ([112](#)) reported that these fractures accounted for 15.2% of 237 ankle injuries in their series and occurred in children significantly younger (average age, 10.5 years) than those with other Salter-Harris types of fractures.

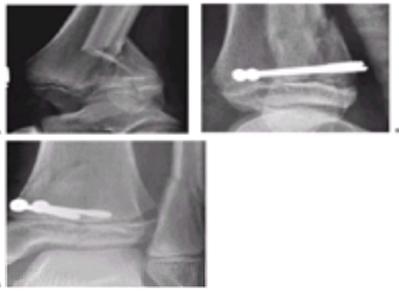
The mechanism of injury is deduced primarily by the direction of displacement of the distal tibial epiphysis; for example, straight posterior displacement indicates a supination–plantarflexion mechanism. The type of associated fibular fracture is also indicative of the mechanism of injury; for example, a high, oblique, or transverse fibular fracture indicates a pronation–eversion–external injury, whereas a lower spiral fibular fracture indicates a supination–external rotation injury. Lovell ([77](#)), Broock and Greer ([16](#)), and Nevelos and Colton ([86](#)) reported unusual Salter-Harris type I fractures in which the distal tibial epiphysis was externally rotated 90 degrees without fracture of the fibula or displacement of the tibial epiphysis in any direction in the transverse plane.

Cast immobilization is generally sufficient treatment for nondisplaced Salter-Harris type I fractures of the distal tibia. Recommended treatment ranges from a below-knee cast worn for 4 weeks to a non–weight-bearing long cast worn for 3 weeks, followed by 3 weeks of immobilization in a short leg walking cast. Most displaced fractures can be treated with closed reduction and cast immobilization. An above-knee non–weight-bearing cast is preferable initially but can be changed to a short leg walking cast at 3 to 4 weeks.

### Salter-Harris Type II Fractures

Salter-Harris type II fractures also can be caused by any of the four mechanisms of injury described by Dias and Tachdjian ([34](#)). In the series of Spiegel and associates ([112](#)), Salter-Harris type II fractures were the most common injuries (44.8%). In addition to the direction of displacement of the distal tibial epiphysis and the nature of any associated fibular fracture, the location of the Thurston-Holland fragment is helpful in determining the mechanism of injury; for example, a lateral fragment indicates a pronation–eversion–external rotation injury; a posteromedial fragment, a supination–external rotation injury; and a posterior fragment, a

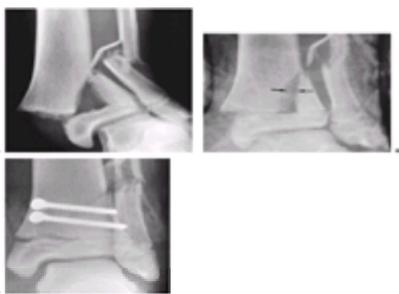
supination–plantarflexion injury ([Fig. 25-23](#)).



**FIGURE 25-23. A:** Severe plantarflexion injury with severe swelling of the ankle and foot; the reduction obtained was unstable. **B:** The reduction was stabilized by two transmetaphyseal screws placed percutaneously. **C:** Anteroposterior view confirms an anatomic reduction.

Nondisplaced fractures can be treated with cast immobilization usually with an above-knee cast for 3 to 4 weeks, followed by a below-knee walking cast for another 3 to 4 weeks.

Although most researchers agree that closed reduction of significantly displaced Salter-Harris type II ankle fracture should be attempted, opinions differ as to what degree of residual displacement or angulation is unacceptable and requires open reduction. Based on follow-up of 33 Salter-Harris type II ankle fractures, Caruthers and Crenshaw ([21](#)) concluded that “accurate reposition of the displaced epiphysis at the expense of forced or repeated manipulation or operative intervention is not indicated since spontaneous realignment of the ankle occurs even late in the growing period.” They found no residual angulation at follow-up in patients who had up to 12 degrees of tilt after reduction, even in patients as old as 13 years at the time of injury. Spiegel and associates ([112](#)), however, reported complications at follow-up in 11 of 16 patients with Salter-Harris type II ankle fractures. Because 6 of these 11 patients had angular deformities that were attributed to lack of adequate reduction of the fracture, Spiegel and associates recommended “precise anatomic reduction.” Incomplete reduction is usually caused by interposition of soft tissue between the fracture fragments. Grace ([46](#)) reported three patients in whom the interposed soft tissue included the neurovascular bundle, resulting in circulatory embarrassment when closed reduction was attempted. In this situation, open reduction and extraction of the soft tissue obviously is required. A less definitive indication for open reduction is interposition of the periosteum, which causes physeal widening with no angulation or with minimal angulation. Good results have been reported after open reduction and extraction of the periosteal flap ([Fig. 25-24](#)) ([69](#)). It is not clear that failure to extract the periosteum in such cases results in problems sufficient to warrant operative treatment.



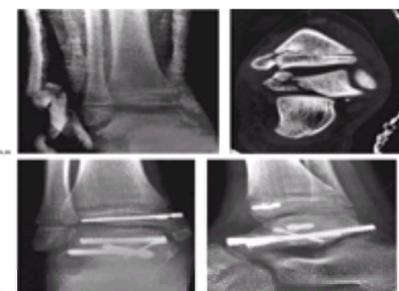
**FIGURE 25-24. A:** Severely displaced pronation–eversion–external rotation injury. **B:** Closed reduction was unsuccessful and a valgus tilt of the ankle mortise was noted. At surgery, soft tissue was interposed laterally (*arrows*). **C:** Reduction completed and stabilized with two cancellous screws placed above the physis.

Because of fears of iatrogenic damage to the distal tibial physis during closed reduction, many researchers recommend the use of general anesthesia with adequate muscle relaxation for all patients with Salter-Harris type II distal tibial fractures. However, no study has compared the frequency of growth abnormalities in patients with these fractures reduced under sedation and local analgesia to those with fractures reduced with the use of general anesthesia. I compared nine patients who underwent closed reduction in the emergency department with the use of sedation and hematoma block to nine patients who had closed reduction in the operating room with the use of general anesthesia. All fractures were reduced with a single manipulation, except for one in the emergency department group that required repeat manipulation. One patient in each group had a growth alteration.

When closed reductions are not performed under general anesthesia, they are usually performed under intravenous sedation. Furia et al. demonstrated significantly improved pain relief with hematoma block for ankle fractures in a study comparing patients treated with intravenous sedation to patients receiving hematoma block ([41](#)). Intravenous regional anesthesia or Bier block also has been reported to be effective for pain relief in lower extremity injuries ([72](#)).

### **Salter-Harris Type III and IV Fractures**

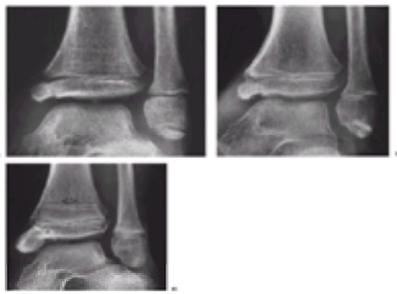
Salter-Harris type III and IV fractures are discussed together because their mechanism of injury is the same (supination–inversion) and their treatment and prognosis are similar. Juvenile Tillaux and triplane fractures are considered separately. In the series of Spiegel and associates ([112](#)), 24.1% of the fractures were Salter-Harris type III injuries and 1.4% were type IV. These injuries are usually produced by the medial corner of the talus being driven into the junction of the distal tibial articular surface and the medial malleolus. As the talus shears off the medial malleolus, the physis also may be damaged ([Fig. 25-25](#)).



**FIGURE 25-25. A:** Severe ankle injury sustained by an 8-year-old involved in a car accident. The anteroposterior view in the splint does not clearly show the Salter-Harris type IV fracture of the tibia. The dome of the talus appears abnormal. **B:** Computed tomography scan shows the displaced Salter-Harris type IV fracture of the medial malleolus and a severe displaced intraarticular fracture of the body of the talus. **C and D:** Open reduction of both fractures was performed and Herbert screws were used for internal fixation. (Courtesy of Armen Kelikian, M.D.)

Nondisplaced Salter-Harris type III and IV fractures can be treated with above-knee cast immobilization, but care must be taken to be sure no displacement is present, which may require CT evaluation, and that no displacement occurs after casting, which requires weekly radiographic evaluation for the first 2 weeks after casting.

Displaced fractures require as anatomic a reduction as possible. Failure to obtain anatomic reduction frequently results in articular incongruity and posttraumatic arthritis, which often becomes symptomatic 5 to 8 years after skeletal maturity ( [Fig. 25-26](#)) (23). The risk of growth arrest also has been linked to the accuracy of reduction (70). Closed reduction may be attempted but is likely to succeed only in minimally displaced fractures. If closed reduction is obtained, it can be maintained with a cast or with percutaneous pins or screws supplemented by a cast.



**FIGURE 25-26. A:** Displaced Salter-Harris type III supination–inversion fracture in an 8-year-old girl who presented for treatment 2 weeks after injury. **B:** After cast removal 6 weeks after injury. **C:** Two years after injury. Although the injury does not seem to have interfered with growth, as evidenced by the Park-Harris growth arrest line parallel to the physis ( *black arrow*), her articular incongruity ( *white arrow*) is associated with occasional mild ankle pain.

If anatomic reduction cannot be obtained by closed methods, open reduction and internal fixation should be performed. Lintecum and Blasier described a technique of open reduction achieved through a limited exposure of the fracture with the incision centered over the fracture site combined with percutaneous cannulated screw fixation (74). This technique was performed on 13 patients: 8 Salter-Harris IV fractures, 4 Salter-Harris III fractures, and 1 triplane fracture. The investigators reported one growth arrest at follow-up averaging 12 months. Beaty and Linton (6) reported a Salter-Harris type III fracture with an intraarticular fragment ( [Fig. 25-27](#)); these fractures require open reduction for inspection of the joint to ensure that no osteochondral fragments are impeding reduction. Internal fixation devices should be inserted within the epiphysis, parallel to the physis, and should avoid the physis and ankle joint if possible ( [Fig. 25-28](#)).

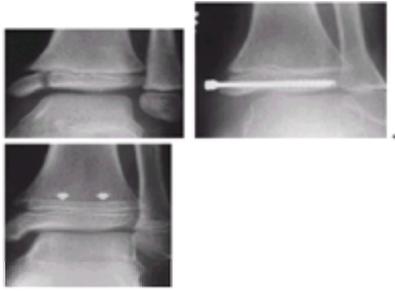


**FIGURE 25-27. A:** Salter-Harris type III fracture of the medial malleolus and Salter-Harris type I fracture of the fibula in a 9-year-old girl. An intraarticular fragment was visible only on a mortise view radiograph. **B:** Computed tomography scan outlined the Salter-Harris type III fracture of the medial malleolus and the fragment of bone. **C:** Two years after excision of the osteochondral fragment, open reduction of the malleolar fracture, and internal fixation. ( **A and B** reprinted from Beaty JH, Linton RC. Medial malleolar fracture in a child: a case report. *J Bone Joint Surg [Am]* 1988;70:1254–1255; with permission.)



**FIGURE 25-28. A:** Grade II supination–inversion injury in a 12-year-old girl, resulting in a displaced Salter-Harris type IV fracture of the distal tibia and a nondisplaced Salter-Harris type I fracture of the distal fibula. **B:** After anatomic open reduction and stable internal fixation.

Options for internal fixation include smooth Kirschner wires, small fragment cortical and cancellous screws, and 4-mm cannulated screws ( [Fig. 25-29](#)). Several reports (7,11,17) have advocated the use of absorbable pins for internal fixation of ankle fractures. Benz and colleagues (7) reported no complications or growth abnormalities after the use of absorbable pins with metal screw supplementation for fixation of five ankle fractures in patients between the ages of 5 and 13 years. In reports of the use of absorbable pins without supplemental metal fixation in adults (9,10,40,55), complications have included displacement (14.5%), sterile fluid accumulation requiring incision and drainage (8.1%), pseudarthrosis (8%), distal tibiofibular synostosis (3.8%), and infection (1.6%). Bucholz and co-workers (17) reported few complications in a series of fractures in adults fixed with absorbable screws made of polylactide and suggested that complications in earlier series might be related to the fact that those pins were made of polyglycolide. A report in 1993 by Bostman and associates (11), however, included few complications in a series of fractures in children fixed with polyglycolide pins. A follow-up report by Rokkanen et al. in 1996 reported a 3.6% rate of infection and a 3.7% rate of failure of fixation (103).

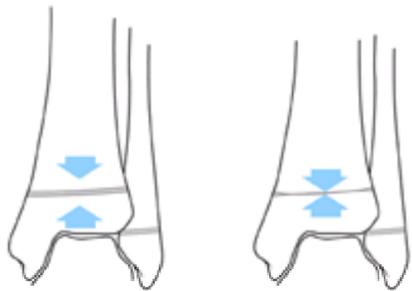


**FIGURE 25-29.** A: Supination–inversion injury with a Salter-Harris type III fracture of the medial malleolus. B: Six months after open reduction and internal fixation with two transepiphyseal cannulated screws. C: Eighteen months after injury, the fracture has healed with no evidence of growth arrest or angular deformity. Arrows note normal, symmetrical Park-Harris growth arrest line.

The main advantage of absorbable pins and screws is that hardware removal is avoided. Bostman compared the cost effectiveness of absorbable implants in 994 patients treated with absorbable implants to 1,173 patients treated with metallic implants. To be cost effective, the hardware removal rates required were calculated to range from 19% for metacarpal fractures to 54% for trimalleolar fractures (12). At this time, the use of absorbable pins remains investigational.

### **Salter-Harris Type V Fractures**

Salter-Harris type V fractures of the ankle are believed to be caused by severe axial compression and crushing of the physis (Fig. 25-30). As originally described, these injuries are not associated with displacement of the epiphysis relative to the metaphysis, which make diagnosis of acute injury impossible from plain radiographs; the diagnosis can only be made on follow-up radiographs when premature physeal closure is evident. Spiegel and associates (112) have designated comminuted fractures that are otherwise unclassifiable as Salter-Harris type V injuries.



**FIGURE 25-30.** Compression-type injury of the tibial physis. Early physeal arrest can cause leg length discrepancy.

The incidence of Salter-Harris type V ankle fractures is difficult to establish because of the difficulty of diagnosing acute injuries. Spiegel and associates (112) included two type V fractures in their series, but both were comminuted fractures rather than the classic crush injury.

Because of the uncertain nature of this injury, no specific treatment recommendations have been formulated. Treatment is usually directed primarily toward the sequelae of growth arrest that invariably follows Salter-Harris type V fractures. Perhaps more sophisticated scanning techniques will eventually allow identification and localization of areas of physeal injury so that irreparable damaged cells can be removed and replaced with interposition materials to prevent growth problems, but at present this diagnosis is made only several months after injury.

### **Other Fractures of the Distal Tibia**

Accessory ossification centers of the distal tibia (os subtibiale) and distal fibula (os fibulare) are common and may be injured. Treatment usually consists of cast immobilization for 3 to 4 weeks. Ogden and Lee (90) reported good results after cast immobilization in 26 of 27 patients with injuries involving the medial side of the tibia; only 1 patient required surgery. In contrast, 5 to 11 patients with injuries involving the lateral side had persistent symptoms that required excision.

Injuries to the perichondral ring of the distal tibial and fibular physes, with physeal disruption, have been described. Most of these injuries are caused by skiving of the bone by machinery such as lawn mowers. They may result in growth arrest or retardation and in angular deformities (see section on [Open Fractures and Lawn Mower Injuries](#)).

## **AUTHOR'S PREFERRED METHOD OF TREATMENT**

### **Salter-Harris Type I and II Fractures of the Distal Tibia**

I prefer to treat nondisplaced Salter-Harris type I and II fractures initially with above-knee cast immobilization. Non–weight bearing is continued until 3 to 4 weeks postinjury, when the cast is changed to a below-knee walking cast that is worn for an additional 3 to 4 weeks. Follow-up radiographs are obtained every 6 months for 2 years or until a Park-Harris growth arrest line parallel to the physis is visible and there is no evidence of physeal deformity.

For displaced fractures in children with at least 2 years of growth remaining, my objective is to obtain no more than 15 degrees of plantar tilt for posteriorly displaced fractures, 10 degrees of valgus for laterally displaced fractures, and 0 degrees of varus for medially displaced fractures (Fig. 25-31). For children with 2 years or less of growth remaining, the amount of acceptable angulation is reduced to less than 5 degrees. I prefer to attempt reduction of markedly displaced fractures with the use of general anesthesia with good muscle relaxation and image intensifier control. In children with mildly displaced fractures, especially if anesthesia is not going to be available for many hours, I occasionally make one attempt at gentle closed reduction under a hematoma block supplemented as needed by well-monitored intravenous sedation. Once adequately reduced, the fractures are usually stable and a long-leg cast can be used for immobilization. Rarely, for markedly unstable fractures or severe soft tissue injuries that require multiple debridements, percutaneous screws are used when the Thurston-Holland fragment is large enough to accept screw fixation. When the fragment is too small, smooth wire fixation across the physis is the only alternative. Repeated attempts at closed manipulation of these fractures may increase the risk of growth abnormality and should be avoided. In patients with fractures that are not seen until 7 to 10 days after injury, residual displacement is probably best accepted. If growth does not sufficiently correct malunion, corrective osteotomy can be performed later.



**FIGURE 25-31. A:** Displaced pronation–eversion–external rotation fracture of the distal tibia in a 12-year-old boy was treated with closed reduction and cast immobilization. **B:** After cast removal a 10 degree valgus tilt was present. **C:** At maturity, the deformity has completely resolved.

I have found that open reduction of these fractures is rarely indicated. The exception usually has been pronation–eversion–external rotation fractures with interposed soft tissue. For fractures with lateral and posterior displacement, an anteromedial incision is made and any interposed soft tissues, such as periosteum or tendons, are extracted before the fracture is reduced. Even though reduction is usually stable, I generally use internal fixation through the metaphyseal fragment, avoiding fixation across the physis if possible.

### Salter-Harris Types III and IV Fractures of the Distal Tibia

Treatment of nondisplaced Salter-Harris type III and IV fractures is the same as for nondisplaced type I and II fractures with three modifications. First, after cast application I confirm the reduction of the fracture fragments with CT scanning. Second, these patients are examined more frequently (once a week) for the first 3 weeks after cast application to ensure that the fragments do not become displaced. Third, these patients are examined every 6 to 12 months after cast removal for a minimum of 24 to 36 months to detect any growth abnormality.

Generally, only truly nondisplaced Salter-Harris types III and IV fractures, or those with 1 mm or less of displacement, can be treated closed. Fractures with 2 mm or more of displacement require open reduction and internal fixation with anatomic alignment of the physis and fracture fragments.

For fractures with 2 mm or less of displacement, closed reduction is attempted in the operating room with the use of general anesthesia. After the extremity is prepared and draped, gentle longitudinal traction is applied to the foot, followed by eversion of the foot and direct digital pressure over the medial malleolus. If image intensification confirms anatomic reduction, the fracture may be fixed with two percutaneous smooth wires placed in the epiphysis parallel to the physis. Reduction is confirmed by a short, continuous fluoroscopic examination. Cannulated screws can be inserted if the epiphysis is large enough. For fractures that are seen more than 7 days after injury, I accept up to 2 mm of displacement without attempting closed or open reduction ([Fig. 25-32](#)). Reliable patients whose fractures are fixed with screws can be immobilized in below-knee casts. Above-knee casts are used for all other patients.



**FIGURE 25-32. A:** Salter-Harris type III fracture after supination–inversion injury of the distal tibia in a 10-year-old girl demonstrates 2 mm of displacement 4 weeks after injury. **B:** After 8 weeks of cast immobilization, healing is apparent but there is a suggestion of early physeal bar formation. **C:** Nine months later, growth is normal, as evidenced by the Park-Harris growth arrest line parallel to the physis.

Fractures with more than 2 mm of displacement should be reduced, regardless of whether the fracture is acute or not. Closed reduction can be attempted, but these fractures usually require open reduction. Occasionally, primary debridement of callus and soft tissue back to normal-appearing physis and fat grafting have been successful for fractures that are more than 7 days old ([Fig. 25-33](#)).



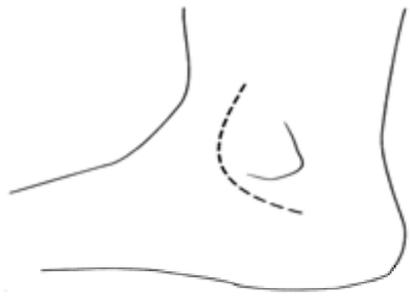
**FIGURE 25-33. A:** Eight-year-old girl who presented 10 days after a displaced Salter-Harris type IV fracture of the distal tibia. **B:** At open reduction, comminution of the physis was noted. The physis was debrided and a fat graft was inserted before reduction and internal fixation of the fracture. **C:** Three years after injury there is no evidence of physeal bar formation or growth abnormality.

## OPERATIVE TREATMENT

### Open Reduction and Internal Fixation of Salter-Harris Type III or IV Fracture of the Distal Tibia

The patient is placed supine on an operating table that is radiolucent at the lower extremity. After exsanguination of the extremity and inflation of the tourniquet, a hockey stick incision is made, extending from approximately 4 cm above the ankle joint to 1 cm posterior to the tip of the medial malleolus ([Fig. 25-34](#)). Alternatively, if more anterior exposure is required, a 4- to 6-cm transverse incision is made from the posterior aspect of the medial malleolus to the anterior aspect of the ankle. The saphenous vein is identified, dissected free, and retracted. The fracture site is identified, and an anteromedial capsulotomy of the ankle joint is performed. The arthrotomy is continuous with the fracture line and the traumatic disruption of periosteum and perichondral ring. The fracture surfaces are exposed and gently cleaned

with irrigation and forceps (curettage is not used).



**FIGURE 25-34.** Anteromedial surgical approach for reduction of a Salter-Harris type IV fracture of the medial malleolus.

For Salter-Harris type IV fractures, the periosteum may be elevated several millimeters from the metaphyseal fracture edges. I prefer not to excise the metaphyseal portion of a Salter-Harris type IV fragment. The epiphyseal edges and joint surfaces are examined through the arthrotomy. The perichondral ring should not be elevated from the physis. For Salter-Harris type III fractures, the reduction is evaluated by checking the joint surface and epiphyseal fracture edges through the arthrotomy. The epiphyseal fragment is grasped with a small towel clip or reduction forceps, and the fracture is reduced ([Fig. 25-35](#)). Internal fixation is performed under direct vision and fluoroscopic control. It is important to view both the lateral and anteroposterior projections because of the curved shape of the distal tibial articular surface. If the fragment is large enough, 4-mm cannulated lag screws are inserted through the epiphyseal fragment ([Fig. 25-28](#)); if the fragment is too small for screws, smooth Kirschner wires are used. The reduction and the position of the internal fixation are checked through the arthrotomy. In fractures with a significant Thurston-Holland fragment, a metaphyseal screw may be used if a gap exists after the epiphyseal screws are inserted. After reduction of the tibial fracture, the associated Salter-Harris type I or II fibular fracture usually reduces and is stable. If it is not, closed reduction and fixation with percutaneous oblique smooth Kirschner wires are performed. The wound is closed using a subcuticular suture for the skin. A well-padded above-knee cast is applied.



**FIGURE 25-35.** Technique for reduction of a Salter-Harris type IV fracture of the distal tibia.

The patient is kept non-weight bearing for 3 weeks, and then the cast is changed to a below-knee walking cast, which is worn for an additional 3 weeks. Frequent follow-up evaluations (every 3 months for the first year and yearly thereafter) are necessary to detect growth abnormalities until skeletal maturity.

### Fractures Involving the Distal Fibula

Fractures involving the fibular physis are most commonly Salter-Harris type I or II fractures that are caused by a supination-inversion injury. Isolated fibular fractures are usually minimally displaced and can be treated with immobilization in a below-knee cast for 4 to 6 weeks. Significantly displaced fibular fractures accompany Salter-Harris types III and IV tibial fractures and usually reduce when the tibial fracture is reduced. Internal fixation of the tibial fracture generally results in stability of the fibular fracture such that cast immobilization is sufficient. If the fibular fracture is unstable after reduction and fixation of the tibial fracture, fixation with a smooth intramedullary or obliquely inserted Kirschner wire is recommended. In older adolescents in whom growth is not a consideration, an intramedullary rod or plate-and-screw device may be used as in adults ([Fig. 25-36](#)).



**FIGURE 25-36.** **A:** Salter-Harris type II fracture of the distal fibula in a 15-year-old. **B:** Lateral view shows the fibular metaphyseal fragment (*arrow*). Considerable soft tissue swelling was noted in the medial aspect of the ankle. **C:** Stress films showed complete disruption of the deltoid ligament. **D:** The fibular fracture was fixed with a cannulated screw; the deltoid ligament was not repaired.

Avulsion fractures from the lateral malleolus are seen in children with inversion sprain-type injuries to the ankle. These may fail to unite with cast immobilization. Patients with such nonunions may have pain without associated instability. In such patients simple excision of the ununited fragment usually relieves their pain ([30,50](#)). When the nonunions are associated with instability, reconstruction of one or more of the lateral ankle ligaments is required (see section on [Lateral Ankle Sprains](#)).

### AUTHOR'S PREFERRED METHOD OF TREATMENT

I usually treat nondisplaced fibular physeal fractures with immobilization in a below-knee walking cast for 3 to 4 weeks. I have attempted closed reduction of displaced Salter-Harris types I and II fibular fractures, but when reduction was unsuccessful, I have accepted up to 50% displacement without problems at long-term follow-up ([Fig. 25-37](#)). Dias ([32](#)), however, reported a patient with a symptomatic spike that required excision after inadequate reduction. I have not found open reduction of isolated physeal fibular fractures necessary but would not hesitate to perform open reduction of a displaced Salter-Harris type III or IV fracture if necessary.



**FIGURE 25-37. A:** Lateral radiograph of a 13-year-old girl who was seen 7 days after an inversion sprain of the ankle; the Salter-Harris type I fracture of the distal fibula is displaced 50%. Closed reduction was unsuccessful. **B:** Six months after injury, remodeling is complete at the fracture site and the patient is asymptomatic.

### Juvenile Tillaux and Triplane Fractures

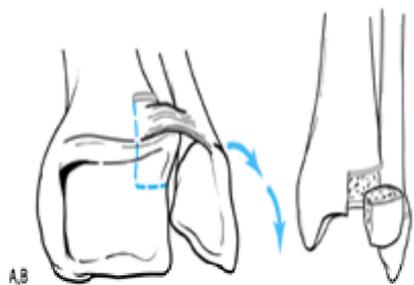
Because the distal tibial physis closes in an asymmetric pattern over a period of about 18 months, injuries sustained during this period can produce fracture patterns that are not seen in younger children with completely open physes (78). This group of fractures has been labeled transitional fractures because they occur during the transition from a skeletally immature ankle to a skeletally mature ankle.

Classification of these fractures is even more confusing than that of other distal tibial fractures. Advocates of mechanism-of-injury systems agree that most juvenile Tillaux and triplane fractures are caused by external rotation, but they disagree as to the position of the foot at the time of the injury (32,33,98). Some researchers (33) classify juvenile Tillaux fractures as stage I injuries, with further external rotation causing triplane fractures, and still further external rotation causing stage II injuries with fibular fracture. Others emphasize the extent of physal closure as the only determinant of fracture pattern (26).

Advocates of anatomic classifications are handicapped by the different anatomic configurations triplane fractures may exhibit on different radiographic projections, making tomography, CT scanning, or examination at open reduction necessary to determine fracture anatomy and number of fragments. Because these fractures occur near the end of growth, growth disturbance is rarely significant. Therefore, anatomic classification is more useful for descriptive purposes than for prognosis.

### Juvenile Tillaux Fractures

This fracture is the adolescent counterpart of the fracture described in adults by the French surgeon Tillaux. It occurs when with external rotation of the foot, the anteroinferior tibiofibular ligament through its attachments to the anterolateral tibia avulses a fragment of bone corresponding to the portion of the distal tibial physis that is still open (Fig. 25-38). In the series of Spiegel and associates (112), these fractures occurred in 2.9% of patients.



**FIGURE 25-38.** Juvenile Tillaux fracture. Mechanism of injury, the anteroinferior tibiofibular ligament avulses a fragment of the lateral epiphysis (A) corresponding to the portion of the physis that is still open (B).

Tillaux fractures may be isolated injuries or may be associated with ipsilateral tibial shaft fractures (28). The fibula usually prevents marked displacement of the fracture, and clinical deformity is generally absent. Swelling is usually slight, and local tenderness is at the anterior lateral joint line, in contrast to ankle sprains, where the tenderness tends to be below the level of the ankle joint.

A mortise view is essential to obtain a view of the distal tibial epiphysis that is unobstructed by the fibula (Fig. 25-39). Steinlauf et al. reported a patient in whom the Tillaux fragment became entrapped between the distal tibia and fibula, producing apparent diastasis of the ankle joint (113). To allow measurement of displacement from plain films, the x-ray beam would have to be directly in line with the fracture site, which makes CT confirmation of reduction mandatory after all closed reductions of these fractures.



**FIGURE 25-39.** Anteroposterior mortise view of a 14-year-old who sustained a juvenile Tillaux fracture.

### Treatment

Both below-knee and above-knee casts have been used for immobilization of nondisplaced juvenile Tillaux and triplane fractures. Fractures with more than 2 mm of displacement require closed or open reduction. Closed reduction is attempted by internally rotating the foot and applying direct pressure over the anterolateral tibia. If necessary, percutaneous pins can be used for stabilization of the reduction. If closed reduction is not successful, open reduction is required. Occasionally, percutaneously inserted pins can be used to manipulate the displaced fragment into anatomic position and then advanced to fix the fragment in place (108).

## AUTHOR'S PREFERRED METHOD OF TREATMENT

For nondisplaced fractures and fractures displaced less than 2 mm, I prefer immobilization in an above-knee cast with the knee flexed 30 degrees and the foot internally rotated. If the position appears acceptable on plain films, CT scanning in the transverse plane with coronal and sagittal reconstructions is used to confirm acceptable reduction. For fractures with more than 2 mm of initial displacement, manipulation may be attempted by internal rotation of the foot and application of direct pressure over the anterolateral joint line. If reduction is not obtained with this maneuver, reduction can be attempted by dorsiflexing the pronated foot and then internally rotating the foot (79). Again, if the reduction appears adequate on plain radiographs, CT scanning is used for confirmation. I prefer to immobilize patients with these injuries in a long-leg cast with the knee flexed and the foot internally rotated.

## OPERATIVE TREATMENT

### Fixation of Juvenile Tillaux Fracture

If radiographs or CT scan after casting show an unacceptable reduction, the patient is taken to the operating room, where closed reduction is again attempted. If reduction is confirmed by C-arm fluoroscopy in all projections, a percutaneous cannulated screw is inserted for fixation. Because preservation of growth is not a concern, the screw does not have to be intraepiphyseal. If closed reduction is unsuccessful but residual displacement is not severe, I occasionally insert one or two smooth pins into the Tillaux fragment under fluoroscopic control (Fig. 25-40) and use them to attempt to guide the fragment into proper position (Fig. 25-41). Cannulated screw guide pins may not be stiff enough for this purpose, and a larger pin may be required. If successful, the pins are advanced and one or two screws are inserted (Fig. 25-42). If reduction is not successful, open reduction is performed through an anterolateral approach and a cannulated or cancellous screw is inserted for fixation (Fig. 25-43). In the anterolateral approach, be sure to identify the superficial perineal nerve to prevent injury. A short leg, non-weight-bearing cast is worn for 3 weeks, followed by a weight-bearing cast for another 3 weeks.

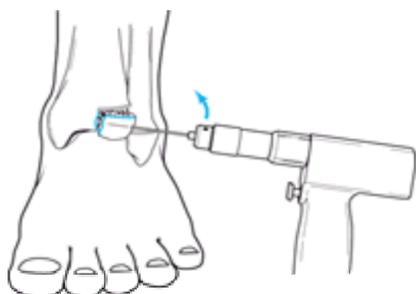


FIGURE 25-40. Technique of placement of percutaneous pins for manipulation of a juvenile Tillaux fracture.

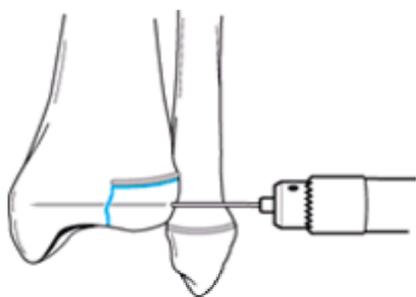


FIGURE 25-41. Advancement of pin after reduction of juvenile Tillaux fracture.

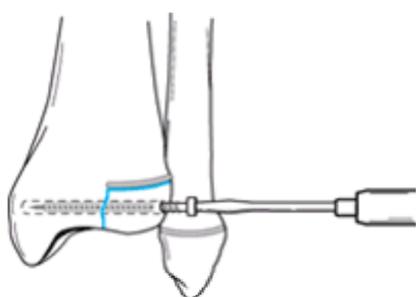


FIGURE 25-42. Percutaneous insertion of 4.0-mm cannulated screw over pin that has been advanced into the medial distal tibia after reduction of the juvenile Tillaux fracture fragment.

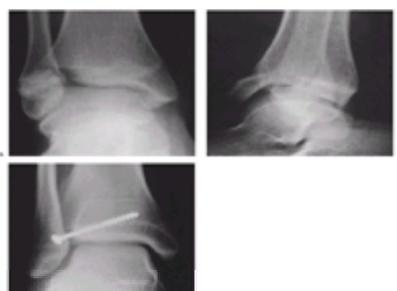


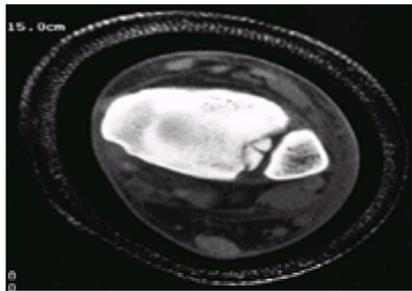
FIGURE 25-43. **A and B:** Displaced juvenile Tillaux fracture. Closed reduction was not successful. **C:** After open reduction and internal fixation with a small fragment screw.

## Fractures of the Incisura

At my institution we have seen two patients with what we have termed incisural fractures. On anteroposterior and mortise views, these patients appeared to have Tillaux fractures with the fractured piece appearing smaller than usual and essentially nondisplaced ( Fig. 25-44). CT scans revealed that the fracture line did not extend through the anterior cortex, as is seen in a typical Tillaux fracture ( Fig. 25-45). It would therefore appear that the fragment could not have been avulsed by the anterior tibiofibular ligament, the accepted mechanism of injury for a Tillaux fracture. It is my hypothesis that the fragment is avulsed by the intraosseous ligament and represents the pediatric equivalent of an adult diastasis injury.



**FIGURE 25-44.** Anteroposterior (A), lateral (B), and oblique (C) views of the ankle demonstrating an apparent small juvenile Tillaux fracture in a 14-year-old girl.

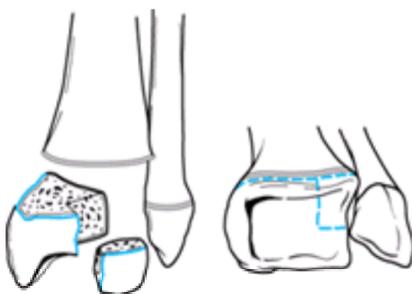


**FIGURE 25-45.** Computed tomography scan at the level of the tibiotalar joint demonstrates that the fracture fragment does not include the attachment of the anterior inferior tibiofibular ligament.

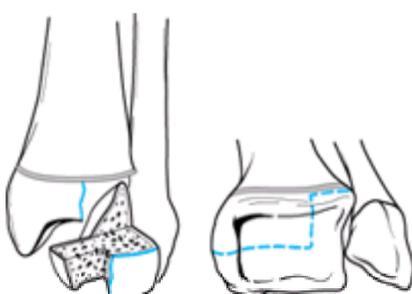
Both of these patients were treated elsewhere with cast immobilization initially. Casting was continued for 12 weeks. At that time, the patients demonstrated no bony healing. Cast immobilization was discontinued and rehabilitation begun. The patients were symptom free 2 years postinjury despite lack of bony union. It is therefore my feeling at this time that nonoperative treatment of this injury is indicated if the mortise is not widened.

## Triplane Fractures

Karrholm attributes the original description of this injury to Bartl ( 5 ), in 1954, and notes that Gerner-Smidt ( 44 ), in 1963, described triplane and Tillaux fractures as different stages of the same injury. In 1957, Johnson and Fahl ( 58 ) described a triplane fracture in their report of 27 physeal ankle injuries and reported that they had seen 10 such fractures. Despite these earlier reports, the nature of triplane fractures was not appreciated until Marmor's ( 81 ) report in 1970 of an irreducible ankle fracture that at surgery was found to consist of three parts ( Fig. 25-46). Two years after Marmor's report, Lynn ( 78 ) reported two additional such fractures and coined the term *triplane fracture*. He described the fracture as consisting of three major fragments: (a) the anterolateral quadrant of the distal tibial epiphysis, (b) the medial and posterior portions of the epiphysis in addition to a posterior metaphyseal spike, and (c) the tibial metaphysis. However, in their 1978 report of 15 such fractures, Cooperman and associates ( 27 ) concluded that, based on tomographic studies, most were two-part fractures produced by external rotation ( Fig. 25-47). Variations in fracture patterns were attributed to the extent of physeal closure at the time of injury. Karrholm and colleagues reported that CT evaluation of four adolescents with triplane fractures confirmed the existence of two-part and three-part fractures and also revealed four-part fractures ( 62 ) ( Fig. 25-48). Denton and Fischer ( 31 ) described a two-part medial triplane fracture that they believed was caused by adduction and axial loading, and Peiro and associates ( 92 ) reported a three-part medial triplane fracture. In the series of Spiegel and associates, 7.3% were triplane fractures ( 112 ).

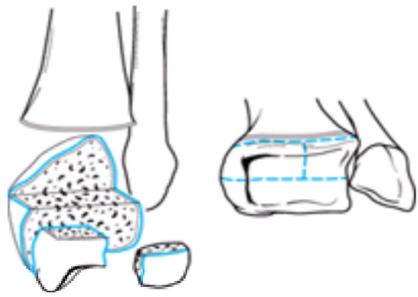


**FIGURE 25-46.** Anatomy of a three-part lateral triplane fracture (left ankle). Note the large epiphyseal fragment with its metaphyseal component and the smaller anterolateral epiphyseal fragment.



**FIGURE 25-47.** Anatomy of a two-part lateral triplane fracture (left ankle). Note the large posterolateral epiphyseal fragment with its posterior metaphyseal fragment.

The anterior portion of the medial malleolus remains intact.

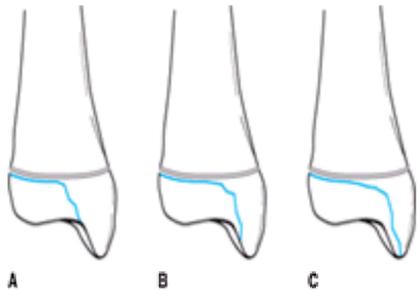


**FIGURE 25-48.** Anatomy of a four-part lateral triplane fracture (left ankle). The anterior epiphysis has split into two fragments and the posterior epiphysis is the larger fragment with its metaphyseal component.

Von Laer (122) described a subgroup of two-part and three-part triplane fractures in which the fracture line on the anteroposterior radiograph did not extend into the ankle joint but into the medial malleolus instead (Fig. 25-49). Feldman and co-workers also reported a case of an extraarticular triplane fracture in a skeletally immature patient (38). Shin et al. reported five patients with intramalleolar triplane variants (109). They divided these into three types: type I, an intramalleolar intraarticular fracture: type II, an intramalleolar, intraarticular fracture outside the weight-bearing surface; and type III, an intramalleolar, extraarticular fracture (Fig. 25-50). These researchers found that CT scans with three-dimensional reconstruction were helpful in determining displacement and deciding if surgery is indicated.



**FIGURE 25-49.** **A and B:** Anteroposterior and lateral radiographs of an "intramalleolar" variant triplane fracture in a 14-year-old boy. **C and D:** CT scans demonstrate extraarticular nature of the fracture.



**FIGURE 25-50.** Schematic drawing of the immature distal tibial physis demonstrating types I, II, and III intramalleolar triplane fractures. **A:** Type I intramalleolar, intraarticular fracture at the junction of the tibial plafond and the medial malleolus. **B:** Type II intramalleolar, intraarticular fracture outside the weight-bearing zone of the tibial plafond. **C:** Type III intramalleolar, extraarticular fracture. (Adapted from Shin A, Moran ME, Wenger DR. Intramalleolar triplane fractures. *J Pediatr Orthop* 1997;17:352–355; with permission.)

Karrholm reviewed 209 triplane fracture patients and found that the mean age at the time of injury was 14.8 years for boys and 12.8 years for girls (60). This type of injury did not occur in children under 10 or over 16.7 years. Patients with triplane fractures may have completely open physes. Swelling is usually more severe than with Tillaux fractures, and deformity may be more severe, especially if the fibula is also fractured. Radiographic views should include anteroposterior, lateral, and mortise views. Rapariz et al. found that 48% of triplane fractures were associated with fibular fracture and 8.5% were associated with ipsilateral tibial shaft fracture (101). Healy et al. reported a triplane fracture associated with a proximal fibula fracture and syndesmotic injury (Masionneuve equivalent) (53). Failure to detect such injury may lead to chronic instability. Therefore, tenderness proximal to the ankle should be sought; if found, it is certainly an indication for radiography of the proximal leg. CT has largely replaced plain tomography for evaluation of the articular surface and the fracture anatomy and should be routinely performed (Fig. 25-51).



**FIGURE 25-51.** Computed tomography scanning of a three-part triplane fracture. **A:** Coronal cut shows lateral epiphyseal fragment. **B:** Sagittal cut shows posterior displacement of the epiphyseal-metaphyseal fragment. **C:** Horizontal cut through the epiphysis shows displacement of the lateral epiphyseal fragment (arrows). **D:** Horizontal cut through the metaphysis shows the fibular fracture and the displaced metaphyseal fragment (arrows).

## Treatment

Nondisplaced triplane fractures (those with <2 mm of displacement) as well as extraarticular fractures can be treated with long leg cast immobilization with the foot in internal rotation for lateral fractures and in eversion for medial fractures. Fractures with more than 2 mm of displacement (65% of the injuries in Karrholm's series) require reduction; this may be attempted in the emergency department or in the operating room with the use of general anesthesia. Closed reduction of lateral triplane fractures is attempted by internally rotating the foot. Based on the mechanism of injury, the most logical maneuver for reduction of medial triplane fractures is abduction. If closed reduction is shown to be adequate by image intensification as is the case about half the time, a long leg cast is applied or percutaneous screws are inserted for fixation if necessary. If closed reduction is unsuccessful, open reduction is required. This can be accomplished through an anterolateral approach for lateral triplane fractures or through an anteromedial approach for medial triplane fractures. Additional incisions are frequently necessary for adequate exposure.

Whipple and associates (124) described arthroscopic reduction of two-part triplane fractures in two patients. With the arthroscope in an anterolateral portal and an anteromedial portal used for inflow, two pins were inserted laterally into the epiphyseal fragment and used to maneuver it into proper position under direct arthroscopic vision. The pins were then advanced for fixation of the fragment.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

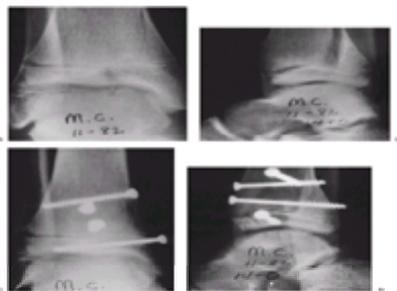
For nondisplaced or minimally displaced (less than 2 mm) fractures, I prefer immobilization in a long-leg cast with the knee flexed 30 to 40 degrees. The position of the foot is determined by whether the fracture is lateral (internal rotation) or medial (eversion). A CT scan is obtained immediately after casting to document adequate reduction. Plain films or CT scans are obtained approximately 7 days after cast application to verify that displacement has not recurred. At 3 to 4 weeks, the cast is changed to a below-knee walking cast, which is worn another 3 to 4 weeks.

For fractures with more than 2 mm of displacement, I usually attempt closed reduction with sedation in the emergency department. An above-knee cast is applied. If plain radiographs show satisfactory reduction, a CT scan is obtained. If reduction is acceptable, treatment is the same as for nondisplaced fractures. If the reduction is unacceptable, closed reduction is attempted in the operating room with the use of general anesthesia. If fluoroscopy shows an acceptable reduction, percutaneous screws are inserted, avoiding the physis, and a short leg cast is applied. If closed reduction is unacceptable, open reduction is performed. Preoperative CT scanning may be helpful for evaluating the position of the fracture fragments in the anteroposterior and lateral planes and for determining the appropriate skin incisions.

## OPERATIVE TREATMENT

### Open Reduction of Triplane Fracture

The patient is placed supine on a radiolucent operating table with padded elevation behind the hip on the affected side. The surgical approach depends on the fracture anatomy as determined by the preoperative CT scan. I prefer to approach a two-part medial triplane fracture through a hockey stick anteromedial incision. The fracture fragments are irrigated to remove debris, and any interposed periosteum is removed. The fracture is reduced, and reduction is confirmed by direct observation through an anteromedial arthrotomy and by image intensification. Two 4-mm cancellous screws are inserted from medial to lateral or from anterior to posterior or both, depending on the fracture pattern (Fig. 25-52). Anterior-to-posterior screw placement may require an additional anterolateral incision, or the screws may be inserted percutaneously.



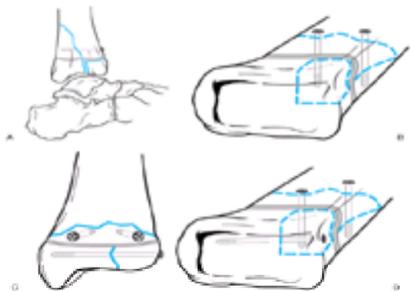
**FIGURE 25-52.** A and B: Irreducible three-part triplane fracture in a 13-year-old girl. C and D: After open reduction and internal fixation. Note anterior-to-posterior and medial-to-lateral screw placement that avoids the physis.

For two-part lateral triplane fractures, I prefer a hockey stick anterolateral approach. The fracture is reduced and stabilized with two screws placed from lateral to medial or from anterior to posterior or both, and reduction is confirmed through direct observation and by image intensification.

Fractures with three or more parts may occasionally require more exposure for reduction and internal fixation. If the fibula is fractured, posterior exposure of the tibial fracture can be readily obtained by detaching the anterior and posterior inferior tibiofibular ligaments and turning down the distal fibula on the lateral collateral ligament (Fig. 25-53). If the fibula is not fractured, a fibular osteotomy may be performed. Careful dissection is necessary to avoid iatrogenic fractures through the physis of the fibula. Medial exposure is obtained through an anteromedial or posteromedial incision. Reduction and internal fixation are performed in a stepwise fashion. For typical three-part fractures, I prefer to reduce the Salter-Harris type II fracture first and provisionally fix it to the distal tibia through the metaphyseal fragment. Usually, the Salter-Harris type III fragment can then be reduced and provisionally fixed to the stabilized type II fragment (Fig. 25-54). Occasionally, the order of reduction and fixation should be reversed. Fractures with four or more fragments require additional steps, but I have found it easier to fix the Salter-Harris type II or IV fragment through the metaphysis to the distal tibia before attaching the Salter-Harris type III fragment or fragments (Fig. 25-55). After reduction, reliable patients may be treated with immobilization in a short-leg cast for 6 to 8 weeks, with weight bearing allowed at 3 to 4 weeks.



**FIGURE 25-53.** Transfibular approach to a complex lateral triplane fracture.



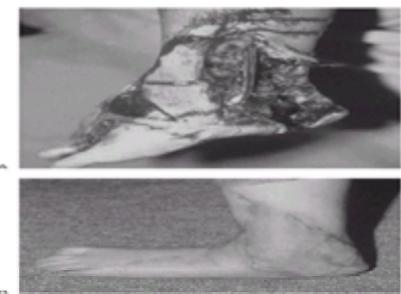
**FIGURE 25-54.** Open reduction and internal fixation of a three-part lateral triplane fracture. **A and B:** Reduction and fixation of the Salter-Harris type II fragment to the metaphysis. **C and D:** Reduction and internal fixation of the Salter-Harris type III fragment to the Salter-Harris type II fragment.



**FIGURE 25-55.** **A and B:** Irreducible three-part lateral triplane fracture in a 14-year-old boy. **C and D:** After open reduction through a transfibular approach and internal fixation with anterior-to-posterior and lateral-to-medial screws.

### Open Fractures and Lawn Mower Injuries

Severe open ankle fractures are often produced by high-velocity motor vehicular accidents or lawn mower injuries ([Fig. 25-56](#)) ([37,47](#)). Approximately 25,000 lawn mower injuries occur each year, 20% of which are in children. Riding lawn mowers are associated with the most severe injuries, requiring more surgical procedures and resulting in more functional limitations ([1,3,35,39,105,123](#)). Loder et al. reviewed 144 children injured by lawn mowers ([75](#)). The average age at the time of injury was 7 years. The child was a bystander in 84 cases. Sixty-seven children required amputation. Soft-tissue infection occurred in 8 of 118 and osteomyelitis in 6 of 117.



**FIGURE 25-56.** **A:** Severe lawn mower injury in a 5-year-old boy. **B:** One year after initial treatment with debridement, free flap, and skin graft coverage.

Principles of treatment are the same as in adults: copious irrigation and debridement, tetanus toxoid, and intravenous antibiotics. Gaglani et al. reported the bacteriologic findings in three children with infections secondary to lawn mower injuries ([43](#)). They found that organisms infecting the wounds were frequently different from those found on initial debridement. Gram-negative organisms were common, and all three patients were infected with fungi as well. In children with lawn mower injuries, grass, dirt, and debris are blown into the wound under pressure, and removal of these embedded foreign objects requires meticulous mechanical debridement.

In most patients, the articular surface and physis should be aligned and fixed with smooth pins that do not cross the physis at the time of initial treatment. Exposed physeal surfaces should be covered with local fat to help prevent union of the metaphysis to the epiphysis. An external fixator may be used if necessary, but small pins should be used through the metaphysis and epiphysis, avoiding the physis ([56,59,76,102,104](#)). Wound closure may be a problem in cases with significant soft tissue injury and exposed bone. Skin coverage with local tissue is ideal, but if local coverage is not possible, split-thickness skin grafting is generally the next choice. Free vascular flaps and rotational flaps may be required for adequate coverage. Klein et al. reported on two patients who had associated vascular injury precluding such flaps. The injury was covered successfully with local advancement flaps made possible by multiple relaxing incisions ([68](#)) Mooney et al. reported the use of cross-extremity flaps for such cases ([85](#)). They found external fixation for linkage of the lower extremities during the procedure to be valuable. After fixation removal, range of motion returned.

Vosburgh and associates ([123](#)) reported 33 patients with lawn mower injuries to the foot and ankle. They found that the most severe injuries were to the posterior-plantar aspect of the foot and ankle. Of their patients, five required split-thickness skin grafts and one required a vascularized flap for soft tissue coverage. Two ultimately required Syme amputation. Four of the patients had complete disruption of the Achilles tendon. Three had no repair or reconstruction of the triceps surae tendon, and one had delayed reconstruction 3 months after injury. Vosburgh and associates ([123](#)) speculated that dense scarring in the posterior ankle results in a “physiologic tendon” and that extensive reconstructive surgery is not always necessary for satisfactory function. Boyer et al. reported on a patient with deltoid ligament loss due to a severe grinding injury that required a free plantaris tendon graft to reconstruct the ligament ([14](#)). Soft tissue coverage was achieved using a free muscle transfer.

### Lateral Ankle Sprains

In 1984, Vahvanen published a prospective study of 559 children who presented with severe supination injuries or sprains of the ankle ([120](#)). Forty patients, 28 boys and 12 girls, with an average age of 12 years (range 5–14) were surgically explored. The indications for surgery included swelling, pain over the anterior talofibular ligament, limp, clinical instability, and, when visible, a displaced avulsion fracture. Such fractures were visible radiographically in only 8 patients but were found at surgery in 19. Thirty-six ankles were found to have injury of the anterior talofibular ligament at surgery. Only 16 of these had either a positive lateral or anterior drawer stress test. At follow-up all patients were pain free and none complained of instability. Based on the incidence of residual disability after such injuries in adults reported in the literature (21%–58%), these researchers suggested primary surgical repair.

Busconi and Pappas reported 60 skeletally immature children with chronic ankle pain and instability ([19](#)). Fifty of these children responded to rehabilitation, but 10

had persistent symptoms. Although 3 of these patients' initial radiographs were within normal limits, all patients with persistent symptoms eventually were found to have ununited osteochondral fractures of the fibular epiphysis. All 10 patients with persistent symptoms were treated with excision of the ununited osteochondral fracture and a Brostrom reconstruction of the lateral collateral ligament. All were able to return to activities, and none reported further pain or instability.

It is my opinion that the diagnosis of an acute ankle sprain in a child with an open fibular physis is reasonable when the child has tenderness well localized to the tip of the fibula or over the anterior talofibular ligament with little or no tenderness over the open fibular physis. The presence on the otherwise normal radiograph of an avulsion fracture of either the tip of the fibula or the lateral aspect of the talus is also consistent with this diagnosis. When routine views fail to demonstrate such avulsion fractures, the views described by Haraguchi et al. may be helpful ( 49).

Because chronic pain and instability do not always complicate such injuries, I treat these injuries nonoperatively when they are seen acutely. Patients who have persistent complaints after adequate rehabilitation following such treatment are treated with excision of any ununited fragments and Brostrom reconstruction as necessary.

### Ankle Dislocations

Nusem et al. reported on a 12-year-old girl who was seen with a posterior dislocation of the ankle without associated fracture ( 88). This was a closed injury and resulted from forced inversion of a maximally plantar flexed foot. The dislocation was reduced under intravenous sedation and the ankle immobilized in a short leg cast for 5 weeks. The patient was asymptomatic at follow-up 4 years postinjury. The inversion stress views at that time revealed only a 3 degree increase in laxity compared with the uninjured side. The anterior drawer sign was negative. There was no evidence of avascular necrosis of the talus on follow-up radiographs.

## REHABILITATION

For patients treated with cast immobilization, quadriceps, hamstring, and abductor exercises are begun as soon as pain and swelling allow. Usually a below-knee cast is worn during the last 2 to 3 weeks of immobilization, and weight bearing to tolerance is allowed during this time. After immobilization is discontinued, ankle range-of-motion exercises and strengthening exercises are begun. Protective splinting or bracing is usually not required after cast removal. Running is restricted until the patient demonstrates an essentially full, painless range of ankle and foot motion and can walk without a limp. Running progresses from jogging to more strenuous running and jumping as soreness and endurance dictate. For athletes, unrestricted running and jumping ability should be achieved before return to sports. Patients should be able to hop on the injured side equal to the noninjured side. Protective measures such as taping or bracing are recommended initially for return to most sports.

Most patients with physeal ankle fractures recover quickly and require little or no formal physical therapy. For this reason, and because of compliance considerations, fractures treated with internal fixation are usually protected with below-knee casting instead of starting an early range-of-motion program in a removable splint.

Reflex sympathetic dystrophy occasionally develops after these injuries and is treated initially with an intensive formal physical therapy regimen that encourages range of motion and weight bearing ( 125). For patients who do not respond quickly to such a program, I have had good success with a brief hospital admission for physical therapy in association with continuous epidural analgesia.

## PROGNOSIS AND COMPLICATIONS

### Delayed Union and Nonunion

Delayed union and nonunion are rare after distal tibial physeal fractures ( Fig. 25-57). Dias (32) reported on one patient with a delayed union and one patient with a previous physeal bar excision who had a nonunion that healed after open reduction, internal fixation, and bone grafting. Siffert ( 110) reported nonunion in a patient with avascular necrosis of the distal tibial epiphysis. I have seen two younger patients with Salter-Harris type III fractures that appeared to be progressing to nonunion. Because neither patient had any complaints of pain or any evidence of progressive displacement of the fracture and stress views showed no instability, no treatment was undertaken. Both fractures eventually united (Fig. 25-58). I have seen one patient with a nonunion after open reduction and internal fixation in whom pin fixation and cast immobilization were discontinued prematurely. The fracture healed after repeat open reduction and internal fixation.



**FIGURE 25-57.** Complex nonunion of a Salter-Harris type III fracture of the medial malleolus in an 8-year-old boy. Note that the distal tibial epiphysis is in valgus, whereas the talus is in varus. (Courtesy of Brent Broztman, M.D.)



**FIGURE 25-58.** Nonunion and delayed union. **A:** Ten-year-old girl with incomplete healing of a supination-inversion Salter-Harris type III fracture of the distal tibia after 12 weeks of cast immobilization. **B:** Sixteen months after injury, the fracture united without further immobilization; no physeal bar formation or growth abnormality has occurred.

### Deformity Secondary to Malunion

Rotational malunion usually occurs after triplane fractures that are either incompletely reduced or are initially immobilized in below-knee casts. It also has been reported after Salter-Harris type I and II injuries. Derotational osteotomy may be performed for extraarticular fractures if discomfort and stiffness occur. Guille et al. reported a rotational malunion of lateral malleolar fracture that led to a stress fracture of the distal fibula that went on to delayed union ( 48). Their patient required

correction of the malrotated distal fibula and bone grafting of the delayed union site.

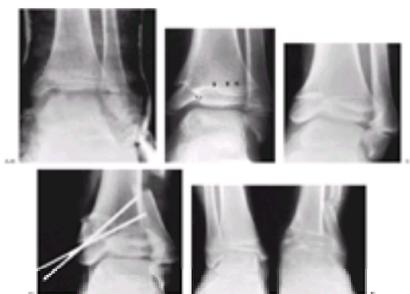
Anterior angulation or plantarflexion deformity usually occurs after supination–plantarflexion Salter-Harris type II fractures. Theoretically, an equinus deformity might occur if the angulation exceeds the range of ankle dorsiflexion before fracture, but this is rare, probably because the deformity is in the plane of joint motion and tends to remodel with growth.

Valgus deformity is most common after external rotation Salter-Harris type II fractures. The degree to which the deformity may spontaneously resolve or remodel with growth is controversial. Caruthers and Crenshaw (21) reported resolution of a 12 degree valgus deformity in a 13½-year-old boy, but Spiegel and associates (112) reported persistent residual deformity in a significant number of their patients (Fig. 25-59). Varus deformity most often results from growth abnormality and infrequently is the result of simple malunion.



**FIGURE 25-59.** Radiograph of a 14-year-old boy, 4 months after pronation–eversion–external rotation injury, reveals 16 degrees of valgus angulation.

If significant angular deformity persists at the completion of growth, supramalleolar osteotomy should be performed. Moon et al. followed nine children with posttraumatic varus deformities of the ankle secondary to supination inversion injuries (84). These patients went on to develop medial subluxation of their ankles with associated internal rotational deformity. Takakura et al. described successful open wedge osteotomy for varus deformity in nine patients (117). Scheffer and Peterson recommend opening wedge osteotomy when the angular deformity is 25 degrees or less and the limb length discrepancy is or will be 25 mm or less at maturity (107). Preoperative planning should include templating the various types of osteotomies to determine which technique will maintain the proper mechanical alignment of the tibia and ankle joint and will not make the malleoli unduly prominent. Osteotomy is not recommended for malunion of intraarticular fractures because it cannot correct the joint incongruity that results from malunion (Fig. 25-60).



**FIGURE 25-60.** **A:** This apparently nondisplaced medial malleolus fracture in an 11-year-old boy was treated with immobilization in a long leg cast. **B:** Fourteen months after injury, there is a clear medial osseous bridge and asymmetric growth of the Park-Harris growth arrest lines (black arrows). Note the early inhibition of growth on the subchondral surface of the fracture (open arrow). **C:** Five years after injury, the varus deformity has increased significantly and fibular overgrowth is apparent. **D:** The deformity was treated with a medial opening-wedge osteotomy of the tibia, an osteotomy of the fibula, and epiphysiodesis of the most lateral portion of the tibial physis and fibula. **E:** Three months after surgery, the osteotomies are healed and the varus deformity is corrected; the joint surface remains irregular. (Courtesy of Earl A. Stanley, Jr., M.D.)

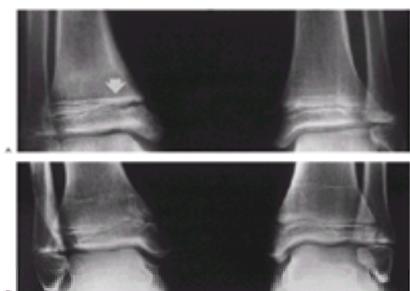
### Growth Arrest

Deformity caused by growth arrest usually occurs after Salter-Harris types III and IV fractures in which a physeal bar develops at the fracture site, leading to varus deformity that progresses with continued growth. Spiegel and associates (112) reported growth problems in 9 of 66 patients with Salter-Harris type II fractures.

Earlier reports (21,29) attributed the development of physeal bars to crushing of the physis at the time of injury, but more recent reports (58,70) discount this explanation and claim that with anatomic reduction (open reduction and internal fixation if needed), the incidence of physeal bar formation can be decreased. The validity of this claim is difficult to determine from published reports. One problem is the small numbers of patients in all series and the even smaller numbers within each group in each series. Another problem is the age of the patients in operative and nonoperative groups in the various series; for example, many children reported to do well with a particular treatment method have so little growth remaining that treatment may have had little or no effect on growth.

Kling and co-workers (70) reported physeal bars in two of five patients treated nonoperatively and in none of three patients treated operatively in children 10 years of age and younger. However, in my experience with eight patients, two of five treated operatively developed physeal bars, whereas none of the three patients treated nonoperatively had physeal bars. This supports the conclusion of Cass and Peterson (22), Ogden (89), and others that growth problems after these injuries may not always be prevented by open reduction and internal fixation. Open reduction of displaced Salter-Harris type III and IV ankle fractures would seem advisable to restore joint congruity, regardless of whether growth potential can be preserved.

Harris growth lines have been reported to be reliable predictors of growth abnormality (57), but I have found that although lines parallel to the physis are reliable, lines that appear to diverge from the physis may be misleading (Fig. 25-61). Harcke and colleagues reported early detection of growth arrest with bone scanning techniques (51).



**FIGURE 25-61. A:** Six months after cast immobilization of a nondisplaced supination–inversion Salter-Harris type III fracture of the right distal tibia in an 8-year-old boy. The Park-Harris growth arrest line (*arrow*) appears to end in the physis medially and diverge from the physis laterally. **B:** Two years later, no physeal bar is present and growth is normal.



**FIGURE 25-62. A:** One year after open reduction and internal fixation of a Salter-Harris type III fracture of the distal tibia in a 7-year-old boy, varus deformity has been caused by a physeal bar. **B:** Two years after excision of the physeal bar and insertion of cranioplast, satisfactory growth has resumed and the deformity has resolved.

Karrholm and co-workers (64) reported progressive ankle deformity caused by complete growth arrest of the fibula with normal growth of the tibia ( Fig. 25-63). They found that continued fibular growth with complete arrest of tibial growth was usually compensated by proximal migration of the fibula so that varus deformity did not occur.



**FIGURE 25-63.** Valgus deformity of the ankle, lateral displacement of the talus with widening of the joint medially, and severe shortening of the fibula after early physeal arrest in a child who sustained an ankle injury at 6 years of age. (Courtesy of James Roach, M.D.)

Because the amount of growth remaining in the distal tibial physis is small (~0.25 inch per year) in most older patients with these injuries, the amount of leg length discrepancy resulting from complete growth arrest tends to be relatively small. Treatment may be required if the anticipated discrepancy is projected to be clinically significant ( Fig. 25-64).



**FIGURE 25-64. A:** Stage II supination–inversion injury in an 11-year-old child. Salter-Harris III fracture of the medial malleolus and a Salter-Harris I fracture of the fibula. **B:** After open reduction and internal fixation. **C:** At 14 months postinjury, the distal fibular physis is completely closed. The Park-Harris growth arrest lines (*arrows*) in the tibia are asymmetric, indicating a possible arrest of the medial physis. **D:** Three years later, the lateral malleolus is short, mild varus deformity is present, and the medial physis is partially closed. The child had a leg length discrepancy of 2.4 cm. (Courtesy of Douglas Hyde, M.D.)

### Arthritis

Epiphyseal ankle fractures that do not extend into the joint have a low risk of posttraumatic arthritis, but injuries that extend into the joint may produce this complication. Caterini and co-workers (23) found that 8 of 68 (12%) patients had pain and stiffness that began 5 to 8 years after skeletal maturity. Ertl and associates found that 18 to 36 months after injury 20 patients with triplane fractures were asymptomatic, but at 36 months to 13 years after injury only 8 of 15 patients evaluated were asymptomatic (36).

Ramsey and Hamilton (100) demonstrated in a cadaver study that 1 mm of lateral talar displacement decreases tibiotalar contact area by 42%, which greatly increases the stress on this weight-bearing joint. More recently, Michelson and colleagues (83) reported that a cadaver study using unconstrained specimens suggested that some lateral talar displacement occurs with normal weight bearing. Because of their findings, they questioned the current criterion of 2 mm of displacement for unstable ankle fractures. However, the results of Ramsey and Hamilton's study correlate well with other studies that have shown increased symptoms in patients in whom more than 2 mm of displacement was accepted (23,36).

## Avascular Necrosis of the Distal Tibial Epiphysis

Siffert and Arkin (110), in 1950, were the first to call attention to this complication of distal tibial fractures. In their patient, the combination of nonunion of a medial malleolar fracture and avascular necrosis caused pain that required an arthrodesis 14 months after injury. Dias (32) reported a patient with this complication who did not require arthrodesis but who had a significant leg length discrepancy that required epiphysiodesis of the contralateral tibia. I have seen one patient with this complication. The patient had significant joint stiffness and developed a valgus deformity secondary to collapse. After revascularization of the epiphysis, the ankle was realigned with a supramalleolar osteotomy, and 5 years later the patient had satisfactory function without pain ( Fig. 25-65).



**FIGURE 25-65. A:** A 12-year-old girl with ankle pain 6 months after open reduction of severe open comminuted fractures of the distal tibia and fibula. Note the sclerosis and collapse of the distal lateral tibial epiphysis. **B:** Magnetic resonance imaging demonstrates lack of marrow signal from the tibial epiphysis. **C:** Five years after osteotomy for correction of the valgus deformity the patient's pain was relieved but joint motion remains limited.

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## CHAPTER REFERENCES

1. Adler P. *Ride on mower hazard analysis 1987–1990*. Washington, DC: Directorate for Epidemiology, USA Consumer Product Safety Commission, 1993:1–65.
2. Aitken AP. The end results of the fractured distal tibial epiphysis. *J Bone Joint Surg* 1936;18:685–691.
3. Alonso JE, Sanchey FL. Lawn mower injuries in children: a preventable impairment. *J Pediatr Orthop* 1995;15:83–89.
4. Ashhurst APC, Bromer RS. Classification and mechanism of fractures of the leg bones involving the ankle. *Arch Surg* 1922;4:51–129.
5. Bartl R. Die Traumatische Epiphysenlösung am Distalen Ende des Schienbeines und des Wadenbeines. *Hefte Unfallheilkd* 1957;54:228.
6. Beaty JH, Linton RC. Medial malleolar fracture in a child: a case report. *J Bone Joint Surg [Am]* 1988;70:1254–1255.
7. Benz G, Kallieris D, Seebeck T, et al. Bioresorbable pins and screws in paediatric traumatology. *Eur J Pediatr Surg* 1994;4:103–107.
8. Bishop PA. Fractures and epiphyseal separation fractures of the ankle. *Am J Roentgenol* 1932;28:49–67.
9. Bostman OM. Distal tibiofibular synostosis after malleolar fractures treated using absorbable implants. *Foot Ankle* 1993;14:38–43.
10. Bostman OM, Hirvensalo E, Vainionpää S, et al. Degradable polyglycolide rods for the internal fixation of displaced bimalleolar fractures. *Int Orthop* 1990;14:1–8.
11. Bostman OM, Makela EA, Sodergard J, et al. Absorbable polyglycolide pins in internal fixation of fractures in children. *J Pediatr Orthop* 1993;13:242–245.
12. Bostman O. Metallic or absorbable fracture fixation devices. *Clin Orthop Rel Res* 1996;329:233–239.
13. Bostock SH, Peach GS. Spontaneous resolution of an osseous bridge affecting the distal tibial epiphysis. *J Bone Joint Surg [Br]* 1996;78:662–663.
14. Boyer MI, Bowen U, Weiler P. Reconstruction of a severe grinding injury to the medial malleolus and the deltoid ligament of the ankle using a free plantaris tendon graft and vascularized gracilis free muscle transfer: case report. *J Trauma* 1994;36:454–457.
15. Bozic KJ, Jaramillo D, DiCanzio J, et al. Radiographic appearance of the normal distal tibiofibular syndesmosis in children. *J Pediatr Orthop* 1999;19:14–21.
16. Brook GJ, Greer RB. Traumatic rotational displacements of the distal growth plate. *J Bone Joint Surg [Am]* 1970;52:1666–1668.
17. Bucholz RW, Henry S, Henley MB. Fixation with bioabsorbable screws for the treatment of fractures of the ankle. *J Bone Joint Surg [Am]* 1994;76:319–324.
18. Burstein AH. Editorial: Fracture classification systems: do they work and are they useful? *J Bone Joint Surg [Am]* 1993;75:1743–1744.
19. Busconi BD, Pappas AM. Chronic, painful ankle instability in skeletally immature athletes. Ununited osteochondral fractures of the distal fibula. *Am J Sports Med* 1996;24:647–651.
20. Carey J, Spence L, Blickman H, et al. MRI of pediatric growth plate injury: correlation with plain film radiographs and clinical outcome. *Skel Radio*. 1998;27:250–255.
21. Caruthers CO, Crenshaw AH. Clinical significance of a classification of epiphyseal injuries at the ankle. *Am J Surg* 1955;89:879–889.
22. Cass JR, Peterson HA. Salter-Harris type IV injuries of the distal tibial epiphyseal growth plate, with emphasis on those involving the medial malleolus. *J Bone Joint Surg [Am]* 1983;65:1059–1070.
23. Caterini R, Fursetti P, Ippolito E. Long-term follow-up of physeal injury to the ankle. *Foot Ankle* 1991;11:372–383.
24. Chadwick L. Spontaneous resolution of varus deformity at the ankle following adduction injury of the distal tibial epiphysis. *J Bone Joint Surg [Am]* 1982;64:774–776.
25. Chande VT. Decision rules for roentgenography of children with acute ankle injuries. *Arch Pediatr Adolesc Med* 1995;149:255–258.
26. Clement DA, Worlock PH. Triplane fracture of the distal tibia: a variant in cases with an open growth plate. *J Bone Joint Surg [Br]* 1987;69:412–415.
27. Cooperman DR, Spiegel PG, Laros GS. Tibial fractures involving the ankle in children: the so-called triplane epiphyseal fracture. *J Bone Joint Surg [Am]* 1978;60:1040–1046.
28. Cox PJ, Clark NJ. Juvenile Tillaux fracture of the ankle associated with a tibial shaft fracture: a unique combination. *Injury* 1996;27:221–222.
29. Crenshaw AH. Injuries of the distal tibial epiphysis. *Clin Orthop* 1965;41:98–107.
30. Danielsson LG. Avulsion fracture of the lateral malleolus in children. *Injury* 1980;12:165–167.
31. Denton JR, Fischer SJ. The medial triplane fracture: report of an unusual injury. *J Trauma* 1981;21:991–995.
32. Dias L. Fractures of the tibia and fibula. In: Rockwood CA, Wilkins KE, King RE, eds. *Fractures in children*, 3rd ed. Philadelphia: JB Lippincott, 1991:1271–1381.
33. Dias L, Giegerich C. Fractures of the distal tibial epiphysis in adolescence. *J Bone Joint Surg [Am]* 1983;65:438–444.
34. Dias LS, Tachdjian MO. Physeal injuries of the ankle in children. *Clin Orthop* 1978;136:230–233.
35. Donmans JP, Azzoni M, Davidson RS, et al. Major lower extremity lawn mower injuries in children. *J Pediatr Orthop* 1995;15:78–82.
36. Ertl J, Barrack R, Alexander A, et al. Triplane fracture of the distal tibial epiphysis: long-term follow-up. *J Bone Joint Surg [Am]* 1988;70:967–976.
37. Farley FA, Senunas L, Greenfield ML, et al. Lower extremity lawn-mower injuries in children. *J Pediatr Orthop* 1996;16:669–672.
38. Feldman DS, Otsuka NY, Hedden DM. Extra-articular triplane fracture of the distal tibial epiphysis. *J Pediatr Orthop* 1996;16:479–481.
39. Foucher J. De la divulsion des epiphyses. *Cong Med France* 1867;1:63 (reprinted in English in *Clin Orthop* 1984;188:3–9).
40. Frokjaer J, Moller BN. Biodegradable fixation of ankle fractures: complications in a prospective study of 25 cases. *Acta Orthop Scand* 1992;63:434–436.
41. Furia JP, Alioto RJ, Marquardt JD. The efficacy and safety of the hematoma block for fracture reduction in closed, isolated fractures. *Orthopedics* 1997;20:423–426.
42. Gabel GT, Peterson HA, Berquist TH. Premature partial physeal arrest: diagnosis by magnetic resonance imaging in two cases. *Clin Orthop* 1991;272:242–247.
43. Gaglani MJ, Friedman J, Hawking EP, et al. Infections complicating lawn mower injuries in children. *Pediatr Infect Dis J* 1996;15:452–455.
44. Gerner-Smidt M. *Ankelbrud Hos Born*. Copenhagen: Nytt Nordiskt Forlag, 1963.
45. Goldberg VM, Aadalén R. Distal tibial epiphyseal injuries: the role of athletics in fifty-three cases. *Am J Sports Med* 1978;6:263–268.
46. Grace DL. Irreducible fracture-separations of the distal tibial epiphysis. *J Bone Joint Surg [Br]* 1983;65:160–162.
47. Grosfeld JL, Muse TS, Eyring EJ. Lawn mower injuries in children. *Arch Surg* 1970;100:582–583.
48. Guille JT, Lipton GE, Bowen JR, et al. Delayed union following stress fracture of the distal fibula secondary to rotational malunion of lateral malleolar fracture. *Am J Orthop* 1997;26:442–445.
49. Haraguchi N, Kato F, Hayashi H. New radiographic projections for avulsion fractures of the lateral malleolus. *J Bone Joint Surg [Br]* 1998;80:684–688.
50. Haramati N, Roye DP, Adler PA, et al. Non-union of pediatric fibula fractures: easy to overlook, painful to ignore. *Pediatr Radio* 1994;24:248–250.
51. Harcke HT, Macy NJ, Mandell GA, et al. Quantitative assessment of growth plate activity [Abstract]. *J Nucl Med* 1984;25:P115.
52. Havranek P, Lizler J. Magnetic resonance imaging in the evaluation of partial growth arrest after physeal injuries in children. *J Bone Joint Surg [Am]* 1991;73:1234–1241.
53. Healy WA III, Starkweather KD, Meyer J, et al. Triplane fracture associated with a proximal third fibula fracture. *Am J Orthop* 1996;25:449–451.
54. Herzenberg J. Computed tomography of pediatric distal tibial growth plate fractures: a practical guide. *Tech Orthop* 1989;4:53–64.
55. Hirvensalo E. Fracture fixation with biodegradable rods. *Acta Orthop Scand* 1989;60:601–606.
56. Horowitz JH, Nichter LS, Kenny JG, et al. Lawn-mower injuries in children: lower extremity reconstruction. *J Trauma* 1985;25:1138–1146.
57. Hynes D, O'Brien T. Growth disturbance lines after injury of the distal tibial physis. *J Bone Joint Surg [Br]* 1988;70:231–233.
58. Johnson EW Jr, Fahl JC. Fractures involving the distal epiphysis of the tibia and fibula in children. *Am J Surg* 1957;93:778–781.
59. Johnstone BR, Bennett CS. Lawn-mower injuries in children. *Aust N Z J Surg* 1989;59:713–718.

60. Karrholm J. The triplane fracture: four years of follow-up of 21 cases and review of the literature. *J Pediatr Orthop* 1997;6:91–102.
61. Karrholm J, Hansson LI, Laurin S. Computed tomography of intraarticular supination-eversion fractures of the ankle in adolescents. *J Pediatr Orthop* 1981;1:181–187.
62. Karrholm J, Hansson LI, Laurin S. Supination-eversion injuries of the ankle in children: a retrospective study of radiographic classification and treatment. *J Pediatr Orthop* 1982;2:147–159.
63. Karrholm J, Hansson LI, Laurin S. Pronation injuries of the ankle in children. *Acta Orthop Scand* 1983;54:1–17.
64. Karrholm J, Hansson L, Selvik G. Changes in tibiofibular relationships due to growth disturbances after ankle fractures in children. *J Bone Joint Surg [Am]* 1984;66:1198–1210.
65. Keats T. *Atlas of normal roentgen variants that may simulate disease*, 5th ed. St. Louis: Mosby Year Book, 1992.
66. Kerr R, Forrester DM, Kingston S. Magnetic resonance imaging of foot and ankle trauma. *Orthop Clin North Am* 1990;21:591–601.
67. Kleiger B, Mankin HJ. Fractures of the lateral portion of the distal tibial epiphysis. *J Bone Joint Surg [Am]* 1964;46:25–32.
68. Klein DM, Caligiuri DA, Katzman BB. Local-advancement soft-tissue coverage in a child with ipsilateral grade IIIB open tibial and ankle fractures. *J Orthop Trauma* 1996;10:577–580.
69. Kling T. Fractures of the ankle and foot. In: Drennan J, ed. *The child's foot and ankle*. New York: Raven, 1992.
70. Kling T, Bright R, Hensinger R. Distal tibial physeal fractures in children that may require open reduction. *J Bone Joint Surg [Am]* 1984;66:647–657.
71. Lauge-Hansen N. Fractures of the ankle II. *Arch Surg* 1950;60:957–985.
72. Lehman W, Jones W. Intravenous lidocaine for anesthesia in the lower extremity. *J Bone Joint Surg [Am]* 1984;66:1056–1060.
73. Letts RM. The hidden adolescent ankle fracture. *J Pediatr Orthop* 1982;2:161–164.
74. Lintecum N, Blasier RD. Direct reduction with indirect fixation of distal tibial physeal fractures: a report of a technique. *J Pediatr Orthop* 1996;16:107–112.
75. Loder RT, Brown KL, Zaleske DJ, et al. Extremity lawn-mower injuries in children: report by the Research Committee of the Pediatric Orthopaedic Society of North America. *J Pediatr Orthop* 1997;17:360–364.
76. Love SM, Grogan DP, Ogden JA. Lawn-mower injuries in children. *J Orthop Trauma* 1988;2:94–101.
77. Lovell E. An unusual rotating injury of the ankle. *J Bone Joint Surg [Am]* 1968;50:163–165.
78. Lynn MD. The triplane distal tibial epiphyseal fracture. *COOR* 1972;86:187–190.
79. Manderson EL, Ollivierre CO. Closed anatomic reduction of a juvenile Tillaux fracture by dorsiflexion of the ankle. *Clin Orthop* 1992;276:262–266.
80. Mann DC, Rajmaira S. Distribution of physeal and nonphyseal fractures in 2,650 long-bone fractures in children aged 0–16 years. *J Pediatr Orthop* 1990;10:713–716.
81. Marmor L. An unusual fracture of the tibial epiphysis. *COOR* 1970;73:132–135.
82. McFarland B. Traumatic arrest of epiphyseal growth at the lower end of the tibia. *Br J Surg* 1931;19:78–82.
83. Michelson JD, Clarke HJ, Jinnah RH. The effect of loading on tibiotalar alignment in cadaver ankles. *Foot Ankle* 1990;10:280–284.
84. Moon MS, Kim I, Rhee SK, et al. Varus and internal rotational deformity of the ankle secondary to distal tibial physeal injury. *Bull Hosp Joint Dis* 1997;56:145–148.
85. Mooney JR III, DeFranzo A, Marks MW. Use of cross-extremity flaps stabilized with external fixation in severe pediatric foot and ankle trauma: an alternative to free tissue transfer. *J Pediatr Orthop* 1998;18:26–30.
86. Nevelos A, Colton C. Rotational displacement of the lower tibial epiphysis due to trauma. *J Bone Joint Surg [Br]* 1977;59:331–332.
87. Nilsson S, Roaas A. Soccer injuries in adolescents. *Am J Sports Med* 1978;6:358–361.
88. Nusem I, Ezra E, Wientroub S. Closed posterior dislocation of the ankle without associated fracture in a child. *J Trauma* 1999;46:350–351.
89. Ogden JA. *Skeletal injury in the child*. Philadelphia: Lea & Febiger, 1982.
90. Ogden JA, Lee J. Accessory ossification patterns and injuries of the malleoli. *J Pediatr Orthop* 1990;10:306–316.
91. Orava S, Saarela J. Exertion injuries to young athletes: a follow-up research of orthopaedic problems of young track and field athletes. *Am J Sports Med* 1978;6:68–74.
92. Peiro A, Aracil J, Martos F, et al. Triplane distal tibial epiphyseal fracture. *Clin Orthop* 1981;160:194–200.
93. Peterson CA, Peterson HA. Analysis of the incidence of injuries to the epiphyseal growth plate. *J Trauma* 1972;12:275–281.
94. Peterson H, Modhok R, Benson J, et al. Physeal fractures: I. Epidemiology in Olmsted County, Minnesota, 1979–1988. *J Pediatr Orthop* 1994;14:423–430.
95. Peterson HA. Physeal fractures: III. Classification. *J Pediatr Orthop* 1994;14:439–448.
96. Petit P, Panuel M, Faure F, et al. Acute fracture of the distal tibial physis: role of gradient-echo mr imaging versus plain film examination. *Am J Radiol* 1996;166:1203–1206.
97. Poland J. *Traumatic separation of the epiphysis*. London: Smith, Elder & Co, 1898.
98. Pollen AG. Fractures involving the epiphyseal plate. *Reconstr Surg Traumatol* 1979;17:25–39.
99. Powell H. Extra centre of ossification for the medial malleolus in children: incidence and significance. *J Bone Joint Surg [Br]* 1961;43:107–113.
100. Ramsey P, Hamilton W. Changes in tibiotalar area of contact caused by lateral talar shift. *J Bone Joint Surg [Am]* 1976;58:356–357.
101. Rapariz JM, Ocete G, Gonzalez-Herranz P, et al. Distal tibial triplane fractures: long term follow up. *J Pediatr Orthop* 1996;16:113–118.
102. Reff RB. The use of external fixation devices in the management of severe lower extremity trauma and pelvic injuries in children. *Clin Orthop Rel Res* 1984;188:21–23.
103. Rokkanen P, Bostman O, Vainionpaa S, et al. Absorbable devices in the fixation of fractures. *J Trauma* 1996;40(suppl):123–127.
104. Ross PM, Schwentker EP, Bryan H. Mutilating lawn mower injuries in children. *JAMA* 1976;236:480–481.
105. Rougraff BT, Kernek CB. Lawn mower injury resulting in chopart amputation in a young child. *Orthopedics* 1996;19:689–691.
106. Salter RB. Injuries of the ankle in children. *Orthop Clin North Am* 1974;5:147–152.
107. Scheffer MM, Peterson HA. Opening-wedge osteotomy for angular deformities of long bones in children. *J Bone Joint Surg [Am]* 1994;76:325–334.
108. Schlesinger I, Wedge JH. Percutaneous reduction and fixation of displaced juvenile Tillaux fractures: a new surgical technique. *J Pediatr Orthop* 1993;13:389–391.
109. Shin AY, Moran ME, Wenger DR. Intramalleolar triplane fractures of the distal tibial epiphysis. *J Pediatr Orthop* 1997;17:352–355.
110. Siffert R, Arkin A. Post-traumatic aseptic neurosis of the distal tibial epiphysis. *J Bone Joint Surg [Am]* 1950;32:691–697.
111. Smith BG, Rand F, Jaramillo D, et al. Early MR imaging of lower-extremity physeal fracture-separations: a preliminary report. *J Pediatr Orthop* 1994;14:526–533.
112. Spiegel P, Cooperman D, Laros G. Epiphyseal fractures of the distal ends of the tibia and fibula. *J Bone Joint Surg [Am]* 1978;60:1046–1050.
113. Steinlauf SD, Stricker SJ, Halen CA. Juvenile Tillaux fracture simulating syndesmosis separation: a case report. *Foot Ankle Int* 1998;19:332–335.
114. Stiell IG, Greenberg GH, McKnight RD, et al. A study to develop clinical decision rules for the use of radiography in acute ankle injuries. *Ann Emerg Med* 1992;21:384–390.
115. Sullivan JA, Gross RH, Grana WA, et al. Evaluation of injuries in youth soccer. *Am J Sports Med* 1980;8:325–327.
116. Tachdjian MO. *The child's foot*. Philadelphia: WB Saunders, 1985.
117. Takakura Y, Takaoka T, Tanaka Y, et al. Results of opening-wedge osteotomy for the treatment of a post-traumatic varus deformity of the ankle. *J Bone Joint Surg [Am]* 1998;80:213–218.
118. Thomsen NO, Overgaard S, Olsen LH, et al. Observer variation in the radiographic classification of ankle fractures. *J Bone Joint Surg [Br]* 1991;73:676–678.
119. Vahvanen V, Aalto K. Classification of ankle fractures in children. *Arch Orthop Trauma Surg* 1980;97:1–5.
120. Vahvanen V, Westerlund M, Nikku R. Lateral ligament injury of the ankle in children. Follow-up results of primary surgical treatment. *Acta Orthop Scand* 1984;55:21–25.
121. Vangsnæs C, Carter V, Hunt T, et al. Radiographic diagnosis of ankle fractures: are three views necessary. *Foot Ankle Int* 1994;15:172–174.
122. Von Laer L. Classification, diagnosis, and treatment of transitional fractures of the distal part of the tibia. *J Bone Joint Surg [Am]* 1985;67:687–698.
123. Vosburgh CL, Gruel CR, Herndon WA, et al. Lawn mower injuries of the pediatric foot and ankle: observations on prevention and management. *J Pediatr Orthop* 1995;15:504–509.
124. Whipple TL, Martin DR, McIntyre LF, et al. Arthroscopic treatment of triplane fractures of the ankle. *Arthroscopy* 1993;9:456–463.
125. Wilder RT, Berde CB, Wolohon MA, et al. Reflex sympathetic dystrophy in children. *J Bone Joint Surg [Am]* 1992;74:910–919.
126. Yao J, Huerman WW. Tomography in a juvenile Tillaux fracture. *J Pediatr Orthop* 1986;6:349–351.
127. Zaricznyj B, Shattuck LJM, Mast TA, et al. Sports-related injuries in school age children. *Am J Sports Med* 1980;8:318–324.

## FRACTURES AND DISLOCATIONS OF THE FOOT

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#### Chapter References

Fractures of the foot in children are relatively unusual injuries that have generated little interest because they usually present little difficulty. As Rang ( 132) stated in *Fractures in Children*, "Injuries to children's feet, despite all the little bones and joints, are remarkably uninteresting. Very few fractures will be encountered that display any subtleties or tricks; few are even displaced" ( 26). Occasionally, a severe injury challenges the orthopaedist's ingenuity when deciding whether to attempt to salvage a functional foot or proceed to amputation. On the other end of the clinical spectrum, it is sometimes difficult to differentiate between relatively minor injuries and the multitude of sesamoids and minor growth variants in the growing foot.

At present there are more reports of children's foot injuries, but they are still rare when compared with forearm or femoral fractures. Because they are infrequent, guidance from the experience of others is helpful, but perplexing fractures, such as a badly displaced calcaneal fracture in a young child, must still be managed without a firm data base.

Computed tomography (CT) and magnetic resonance imaging (MRI) allow us a much greater understanding of the nature of injuries to the foot. The development of sophisticated techniques such as Ilizarov fixation and ankle arthroscopy allows more options for treatment, but expertise in all relevant techniques is difficult for any of us to attain.

## ANATOMY

As in any injury, knowledge of the regional anatomy is essential in evaluating the injury and formulating an intelligent treatment plan. Although the foot lacks the intricacies of fine motor control evident in the hand, it is a remarkable structure in its own right. During the walking cycle, it is both flexible and rigid—flexible on foot strike and rigid at lift-off ( 102,187). Any structural aberration that deprives the foot of its flexibility (such as talar arthrodesis) alters the normal distribution of forces through the foot during stance phase. A combination of normal muscular action and the architecture of the foot provides rigidity at toe-off, a situation obviously affected by injuries involving muscle–tendon units in the hindfoot ( 104).

The anatomic factors pertinent to the management of foot injuries in children are reviewed briefly, because an exhaustive knowledge of the ligaments of the mid-foot, for instance, is not often clinically applicable. Standard anatomy references can supplement the material in this chapter if further details are needed ( 74,146).

## Ossification

The calcaneus is the first major bone to ossify, usually in the fifth month of gestation; the talus may ossify as early as the eighth month or as late as after birth. The timing of the appearance and fusion of ossification centers in the foot has been well documented ( Fig. 26-1). However, wide individual variations occur, for instance, in the appearance of the ossific nucleus for the tarsal navicular bone. Accessory bones in the foot have been noted in the fetus as early as the fourth postovulatory month.



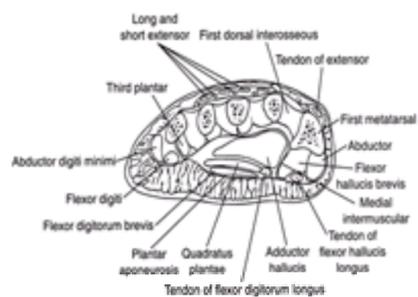
**FIGURE 26-1.** Time of appearance and fusion of ossification centers of the foot. Figures in parentheses indicate the time of fusion of primary and secondary ossification centers (y., years; m.i.u., months *in utero*). (Redrawn from Aitken JT, Joseph J, Causey G, et al. *A manual of human anatomy*, 2nd ed. Vol. IV. London: E & S Livingstone, 1966:80; with permission.)

Ogden (123) and others have emphasized that the shape of the ossific nuclei of the bones does not resemble the shape of the adult structure, or even the shape of the chondro-osseous bones. Talar ossification begins in the head and neck in the infant, extending in a retrograde direction into the body with growth. The calcaneal ossification center in the young child has a protective cartilaginous covering that alters fracture patterns.

## Soft Tissue Structures

The skin on the plantar surface of the foot is the body's interface with the ground, and it is especially suited for that task. The superficial fascia is heavy and continuous with that of the leg. On the plantar surface, at the distal metatarsal level, it blends with the deeper tissue, surrounding the plantar metatarsal and common digital nerves. The fascial layer on the dorsum of the foot is thin and filmy. The deep fascia is analogous to the deep palmar fascia of the hand, with a strong central aponeurosis and thinner medial and lateral parts covering the intrinsic muscles of the big and little toes. Serrafian (153) described the plantar fascia as acting as a tie-rod; the bony columns and the plantar aponeurosis form a truss, a triangular structure with two struts or beams connected at the base by a tie-rod. When the foot is loaded, tensile stresses are taken up by the plantar aponeurosis, thus sparing the bony structures from tension.

Septae from the plantar aponeurosis descend to attach to the first and fifth metatarsals, dividing the plantar surface of the foot into three compartments, in addition to the four dorsal interosseous compartments. Shereff (154) and Myerson (117) noted that the three muscles of the central compartment—the abductor hallucis, the quadratus plantae, and the flexor digitorum brevis—all have separate compartments; thus, there are nine fascial compartments in the foot (the four interosseous, the three central, one lateral, and one medial; Fig. 26-2). Therefore, when a fasciotomy is performed for ischemia of the intrinsic muscles, all compartments must be released to avoid subsequent contracture.



**FIGURE 26-2.** Cross section of the foot at the level of the proximal metatarsals to demonstrate the fascial compartments. (Modified from Hollinshead WH. *Anatomy for surgeons*, 2nd ed. Vol. 3. New York: Hoeber-Harper, 1969:857; with permission.)

## Vascular Anatomy

The blood supply to the foot is almost exclusively derived from the terminal branches of the anterior and posterior tibial arteries. Occasionally, the perforating branch of the peroneal artery supplies much of the dorsum, but ordinarily the contribution of the peroneal artery is several lateral calcaneal branches and an anastomotic branch of the perforating peroneal artery with the lateral malleolar branch of the anterior tibial artery.

The dorsalis pedis artery, beginning at the ankle joint, is the continuation of the anterior tibial artery on the dorsum of the foot. It usually extends over the superior talus, navicular bone, and second cuneiform bone to branch at the second metatarsal into the deep plantar artery and the arcuate artery. The dorsal metatarsal arteries are branches of the arcuate artery. Medial and lateral tarsal arteries supply the dorsum of the mid-foot.

The posterior tibial artery divides into medial and lateral plantar arteries under cover of the flexor retinaculum. The medial artery is much smaller, passing under the abductor hallucis with the medial plantar nerve, and proceeding distally along the medial side of the flexor hallucis longus tendon to terminate at the great toe, accepting an anastomosis from the deep plantar artery. The larger lateral branch proceeds laterally and distally with its accompanying nerve between the flexor digitorum brevis and quadratus plantae. It is protected by the plantar aponeurosis as well. Between the flexor digitorum brevis and the abductor digiti minimi, it turns deeply and medially to lie on the plantar surface of the interossei, and forms the plantar arch. The plantar metatarsal arteries arise from the arch to form the common digital arteries and anastomose with smaller branches from the dorsal metatarsal arteries to supply the toes.

Injury to the posterior tibial artery at the ankle can severely compromise the blood supply to the heel pad. Injuries of the forefoot generally are less serious because of the more generous anastomosis in this area of the blood supply to the soft tissues.

The anatomic arrangements described here are not invariably present, and variants can be present.

## Neurologic Anatomy

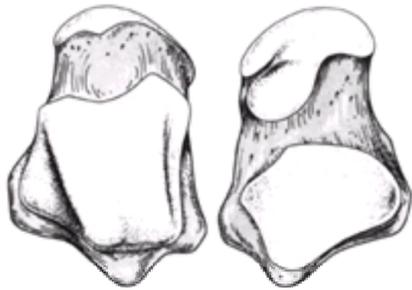
The nerve supply to the important plantar aspect of the foot is from the posterior tibial nerve. The medial and lateral plantar nerves supply all the musculature of the sole of the foot. In contrast to the medial plantar artery, though, the medial plantar nerve supplies most of the terminal sensory branches, including the proper digital

nerves to the great toe and the common digital nerves to the next three interspaces. The three and a half toes supplied by the medial plantar nerve are analogous to the median nerve innervation in the hand. The lateral one and a half toes are supplied by superficial branches of the lateral plantar nerve. This nerve also supplies all the interossei and the adductor hallucis, analogous to the distribution of the ulnar nerve in the hand.

### Bony Anatomy

The bony architecture of the foot is well adapted for weight bearing, providing stability with sufficient flexibility to accommodate the complex rotational factors essential for a smooth gait. By convention, the talus and calcaneus are known as the hindfoot, the navicular cuboid and cuneiform bones as the mid-foot, and the metatarsals and phalanges as the forefoot.

The talus is the intermediary between the foot and leg, with the ankle joint above and the subtalar joint below. Its blood supply is rather precarious, because so much of its surface is covered with articular cartilage, leaving only the constricted neck to accept most of its nutrient vessels. Because a basic knowledge of the blood supply to the talus is helpful in determining the prognosis for an injury to the talus, it is discussed with the description of that fracture. The body of the talus is quadrilateral when viewed superiorly, being slightly wider anteriorly (Fig. 26-3). The tendon of the flexor hallucis longus passes between the tubercles; the lateral tubercle is sometimes separate and known as the os trigonum (62). The lateral process projects between the two articular surfaces, and to it attaches the talocalcaneal ligament. The inferior surface of the talus contains the articular surfaces of the subtalar joint. The anterior and middle facets often are joined and separated from the large posterior facet by the sulcus. The anastomotic ring passes through the sulcus, and the strong interosseous talocalcaneal ligament also attaches here.



**FIGURE 26-3.** The superior (**left**) and inferior (**right**) surfaces of the talus. The divergence of the medial and lateral articular facets is evident on the superior view. The constricted neck is the major recipient of the vascular supply to the head and body; both are covered with articular cartilage. The confluence of the anterior and middle articular facets on the inferior surface is common. (Reprinted from Weber BG, Brunner C, Freuler F. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980:373; with permission.)

The superior surface of the calcaneus is adapted to conform to the inferior talus. In addition, the prominent sustentaculum tali projects medially and articulates with the medial facet of the talus. The tendon of the flexor hallucis longus passes beneath the sustentaculum, and the strong calcaneonavicular “spring” ligament arises from it. The tuberosity is the posterior extremity of the calcaneus and is represented by an apophysis in childhood. The tuberosity has medial and lateral processes, with the medial tuber giving origin to the abductor hallucis and flexor digitorum brevis, the lateral to the plantar aponeurosis. The Achilles tendon inserts into the inferior half of the tuberosity; the superior half of the tuberosity is separated from the tendon by a bursa.

The navicular bone is interposed between the talus and the three cuneiform bones and articulates laterally with the cuboid. The medial tuberosity affords an insertion for the posterior tibial tendon and often remains as an “accessory” navicular bone, sometimes confused with a fracture.

The three cuneiform bones and cuboid form the distal row of tarsals. The first or medial cuneiform bone is the largest, the second the smallest. When viewed in cross section, the bones are wedge shaped, being blunt dorsally, to accommodate the arch of the foot. The relationship of the middle cuneiform bone with the second metatarsal (the longest metatarsal) is of particular clinical importance. The base of the second metatarsal is recessed between the medial and lateral cuneiform bones and is enclosed by strong ligaments to all three cuneiform bones. Thus, a tarsometatarsal dislocation must fracture the base of the second metatarsal to dislocate the remaining four joints. There is extensive ligamentous support of the mid-foot, and the ligaments are much more substantial on the plantar surface than the dorsal surface.

The metatarsals taper from their bases to their necks, then enlarge to form the heads. The secondary ossification center of the first metatarsal is proximal; those of the other four metatarsals are distal. The bases of the first and fifth metatarsals bear tuberosities for the insertion of the peroneus longus on the first and the peroneus brevis on the fifth. The tuberosity of the fifth often forms a separate ossification center that can remain ununited and is known as os vesalianum.

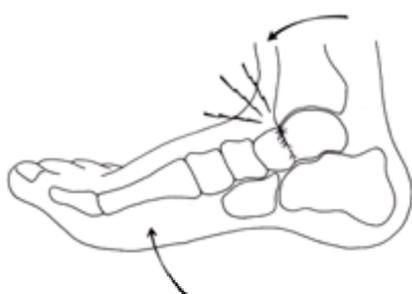
The proximal phalanges of the toes are much longer than the middle and distal phalanges. The middle phalanges can have little form and, except for the big toe, the distal phalanges can be tiny.

Knowledge of surface anatomy often is helpful in assessing a clinical injury. Landmarks in the small child are more difficult to delineate, but localized tenderness is always helpful in evaluating a clinical injury and can often point one to areas of the x-ray deserving special attention.

### FRACTURES OF THE TALUS

Fractures of the talus are rare in children and appear to differ from those in adults. For some time it was thought that these fractures did not occur in children with any regularity. Canale and Kelly (23), in a study of 71 fractures of the neck of the talus, included 12 children who were 16 years of age or younger. Six were 15 or 16 at the time of injury, and four were less than 10 years old. Letts and Gibeault (95) reported 12 fractures of the neck of the talus in children, four of whom were under 3 years of age.

Forced dorsiflexion appears to be the common mechanism of injury in all series (Fig. 26-4) (31,108). However, the reported association of talar fractures with fractures of the medial malleolus implicates a supination component to at least some injuries; the disability reported seemed more attributable to the ankle fracture than the talar fracture (74). A supination mechanism has been associated with more severe injuries. With excessive forced supination, the talar neck impinges against the medial malleolus, which has been postulated as the cause of medial neck comminution and subluxation of the subtalar joint commonly associated with these fractures (37).



**FIGURE 26-4.** The mechanism of fracture of the neck of the talus by forced dorsiflexion of the foot. The talar neck is impinged on the anterior lip of the tibia.

Most talar fractures reported in children involve the talar neck. Fractures of the neck of the talus do not involve the articular surface of the ankle. The inferior portion of the neck is in the region of the interosseous ligament, so talar neck fractures are extraarticular. Although extraarticular, the inferior aspect of the talus articulates with the three facets of the calcaneus. Significant malalignment of talar neck fractures may alter the biomechanical relationships of the talocalcaneal articulations and could lead to early degenerative changes. Sangeorzan et al. emphasized the importance of avoiding varus malalignment at the time of fracture reduction ( 144).

Isolated fractures of the talar dome, or body, have been described. In adults, fractures of the body account for fewer than 20% of talar fractures; the incidence may be even less in children (159). Fractures of the body of the talus in adults have a worse prognosis than neck fractures. From an anatomic standpoint, the same principle should apply in children, but there is no published series to substantiate this opinion.

### Anatomy

The talus generally is divided into three parts—the body, neck, and head. The body articulates with the ankle and calcaneus and its surface is almost entirely cartilaginous. The more constricted neck is perforated about its entire circumference to receive nutrient vessels, with the body then dependent on interosseous vessels from the neck. The head again flares and is cartilaginous for its articulation with the navicular bone. Inferiorly, the posterior facet articulates with the calcaneus. The interosseous ligament and sulcus lie under the neck, and the anterior and variable middle facet lie under the head.

A general knowledge of the vascular supply to the talus is basic to understanding the risk of avascular necrosis. Mulfinger and Trueta ( 116) described the vascular anatomy well, acknowledging the previous contributions by Wildenauer and Haliburton and associates ( 64,180).

The tarsal canal, formed by the sulcus of the talus and the sulcus of the calcaneus, is funnel-shaped from a posteromedial to anterolateral direction. The artery of the tarsal canal, arising from the posterior tibial artery about 1 cm proximal to the origin of the medial and lateral plantar arteries, passes posterolaterally through the canal, much closer to the talus than the calcaneus. It gives off a deltoid branch soon after its origin that anastomoses with branches from the dorsalis pedis over the neck of the talus.

The other main arterial source supplying the talus is the artery of the tarsal sinus, usually starting from an anastomotic loop between the perforating peroneal and the lateral tarsal branches of the dorsalis pedis artery. It usually is a little larger than the artery of the tarsal canal, with which it forms an anastomosis in the canal. This anastomotic artery supplies most of the talar body; the deltoid branches nourish the medial quarter.

### Signs and Symptoms

Severe fractures with obvious local signs of injury present little problem in diagnosis, but nondisplaced fractures can be elusive. Swelling and pain in the region of the talus, especially with a history of a fall or forced dorsiflexion, should alert the examiner to the possibility of a talar fracture. Localized tenderness accompanies all fractures. Even nondisplaced talar fractures are characterized by tenderness just distal to the anterior ankle joint and pain with dorsiflexion. In their series of talar fractures in children, Letts and Gibeault ( 95) noted that occasionally the fracture was not suspected initially from the physical examination. This should not be interpreted as indicating that there were no physical findings; physical findings are noted only when the examiner has an index of suspicion for a particular injury. With nondisplaced fractures, minimal swelling may be present, but localized tenderness is always present. The presence or absence of tenderness posteriorly helps differentiate a talar fracture from an os trigonum.

### Radiographic Findings

Anterior, posterior, lateral, and oblique radiographs should be made with the beam centered on the hindfoot. The technique described by Canale and Kelly ( 23) of pronating the foot 15 degrees with the tube angled at 75 degrees to the tabletop produces an excellent shadow of the talus and is recommended for evaluating the talus in the anteroposterior view (Fig. 26-5). Hawkins, in 1971, detailed his experience with 57 talar neck fractures in adults, and his classification of such fractures has become widely accepted (76,177): type I, nondisplaced; type II, displaced with a subtalar dislocation or subluxation; and type III, subluxation or dislocation of both subtalar and ankle joints.



**FIGURE 26-5.** The technique of Canale and Kelly to determine the position of fracture of the talar neck in the anteroposterior plane. This view is obtained with the ankle plantarflexed, 15 degrees internal rotation of the foot, and the x-ray beam directed 75 degrees cephalad from the table. (Reprinted from Canale ST, Kelly EB Jr. Fractures of the neck of the talus. Long-term evaluation of 71 cases. *J Bone Joint Surg [Am]* 1978;60A:151; with permission.)

## AUTHOR'S PREFERRED METHOD OF TREATMENT OF TALAR NECK FRACTURES

Treatment of fractures of the neck of the talus in children is comparable with that in adults. Nondisplaced fractures are treated with simple immobilization in a long leg cast with the knee flexed to prevent weight bearing. When the fracture is united, usually in 6 to 8 weeks, weight bearing is allowed; the application of a weight-bearing short leg cast for 2 to 3 weeks smooths the transition from non-weight-bearing to weight-bearing status.

The amount of displacement that can be safely accepted in children is not definitely established. Canale and Kelly ( 23) considered a reduction of less than 5 mm displacement and less than 5 degrees of malalignment on the anteroposterior view to be adequate. If this position cannot be attained, open reduction is indicated. Adelaar recommended open reduction when closed reduction fails to achieve anatomic reduction and when there is 3 mm or more of dorsal displacement and 5 degrees of varus rotation (1).

## OPERATIVE TREATMENT OF TALAR NECK FRACTURE

**Patients with displaced fractures should be taken to the operating room for immediate reduction.** Minimally displaced fractures of the talar neck (type II) often can be successfully treated with closed reduction by plantarflexion of the foot ( Fig. 26-6). The hindfoot may be inverted or everted, depending on the direction of the subtalar instability. If the reduction is stable, the foot can be dorsiflexed for immobilization. If not, immobilization in plantarflexion for 6 to 8 weeks is indicated, carefully monitoring the fracture position when dorsiflexion is attempted. If reduction cannot be maintained by simple positioning, internal fixation is indicated. Fluoroscopy is of great help and often obviates the need for open reduction. Type III injuries usually require open reduction. Indirect reduction with the use of the calcaneal traction pin

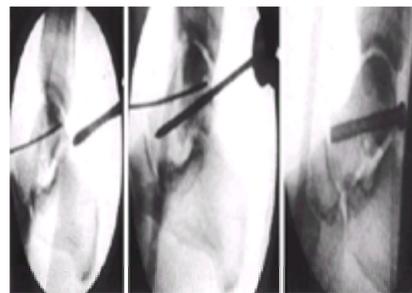
connected to a small traction bow may aid in the reduction maneuver to reduce the dislocated or subluxed talar body back into the subtalar joint ( Fig. 26-7). If satisfactory reduction of the talar neck fracture cannot be obtained, a limited anterior exposure can be made through either an anteromedial or anterolateral approach to allow reduction under direct vision. Through this exposure the fracture can be provisionally stabilized with Kirschner wires from anterior to posterior. A posterolateral approach is then made and two cannulated screws are placed from posterior to anterior for superior biomechanical fixation ( Fig. 26-8). The provisional wires are then removed.



**FIGURE 26-6.** A: Displaced fracture of the talar neck in a 7.2-year-old boy. B: Position after closed reduction by plantarflexion of the foot. C: Avascular necrosis is evident 10 weeks after injury. The fracture is healed and the boy is fully weight bearing despite instructions. D: Thirty months after injury, secondary changes are evident. The boy is asymptomatic and has not restricted activities. He returned for follow-up only as a result of a newly fractured forearm.

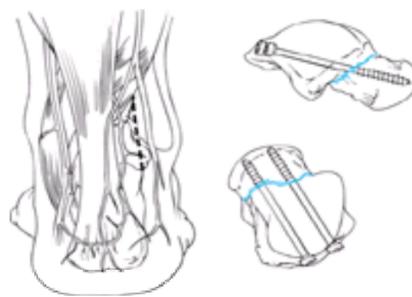


**FIGURE 26-7.** A calcaneal traction pin may at times aid in the reduction of the dislocated talar body within the subtalar joint in a Hawkins type III injury. The pin is attached to a small traction bow, then with combined traction plantarflexion and rotation of the foot the reduction is achieved. (Reprinted from Adelaar RS. Complex fractures of the talus. *Instr Course Lect* 1997;46:330; with permission.)



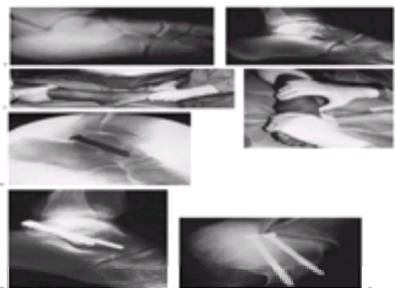
**FIGURE 26-8.** Anatomic reduction may need to be performed under direct visualization through an open anterior approach. Once reduced, the fracture is provisionally stabilized anteriorly with pin fixation. Posterior-to-anterior-directed screws are then placed for improved biomechanical stability, and the provisional Kirschner wire is then removed. (Reprinted from Adelaar RS. Complex fractures of the talus. *Instr Course Lect* 1997;46:329; with permission.)

Since the publication of the previous edition of this chapter, evidence for the mechanical superiority of screw fixation from posterior to anterior has been documented (163). The posterolateral approach allows extraarticular introduction of cannulated or noncannulated screws, which is not feasible through the talar head or neck ( Fig. 26-9). Lemaire and Bustin (94), crediting Trillat and colleagues (169) for the idea, first recommended the posterior approach in 1980 for displaced fractures of the talus. If the ankle joint is intact, closed manipulation is performed; if the ankle joint is also disrupted by displacement of the talar body, open reduction is necessary. The posterior approach is simple anatomically: through a direct approach just lateral to the Achilles tendon. The posterior capsule is divided and the posterior talus exposed. Either two 4.0-mm or 4.5-mm cannulated screws or a single larger screw can be used with similar mechanical properties if the talus is large enough. The present assortment of cannulated screws allows greater application of this technique. Titanium screws have been used because their nonmagnetic properties allow better MRI scans should the presence of osteonecrosis later be suspected (1,166).



**FIGURE 26-9.** Left: Posterior lateral approach. Posterior-to-anterior screws may be placed percutaneously or through a formal posterior lateral approach. The incision is made lateral to the Achilles tendon, and the flexor hallucis longus is reflected medially, with the starting point of the guide pin at the posterior lateral tubercle. Right: The screws are directed slightly plantar medial in line with the long axis of the talar neck. Careful attention is focused on staying as extraarticular as possible with the starting point of the guide pin. The screw's threads should end beyond the fracture line of the talar neck for optimal compression at the fracture site. In general, two parallel screws are placed; however, a larger screw and a pin may be placed as an alternative. (Modified from Adelaar RS. Complex fractures of the talus. *Instr Course Lect* 1997;46:328; with permission.)

This approach is most useful if a manipulative reduction of the fracture can be attained, then held with posteriorly inserted fixation ( Fig. 26-10). In this way, no surgical dissection around the vascularly vulnerable neck of the talus is necessary. Adelaar ( 1 ) noted that he does not hesitate to add a minimal anterior exposure if necessary to obtain a satisfactory reduction.



**FIGURE 26-10.** Anteroposterior (A) and lateral views (B) of a displaced talar neck fracture in a 15-year-old girl. Closed reduction was accomplished with longitudinal traction (C) and valgus movement (D) applied to the talus. This position was held while two guide pins were placed for 4.5-mm cannulated screw fixation. Intraoperative images (E). Solid union 5 months postoperatively (F and G).

Traditionally, open reduction of the talus has been performed through a dorsomedial approach, keeping the incision to the medial side of the extensor hallucis longus to avoid further damage to the anterior tibial vessels. Obviously, dissection in this region should be kept to the minimum necessary for reduction. Kirschner wire fixation is used, generally through the navicular and the head of the talus, or the head of the talus only if possible.

Monthly follow-up is indicated for the first 6 months after injury to monitor healing and the vascular status of the talus. Virtually all reported cases of avascular necrosis of the talus in children have been noted earlier than 6 months after injury. In the absence of complications, a nondisplaced fracture should be followed for 1 to 2 years after injury. Fractures healing with residual displacement require longer follow-up.

## Complications

### Avascular Necrosis

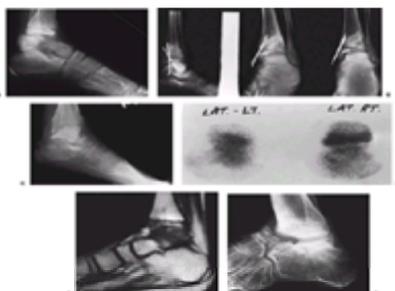
The most significant complication of fractures of the talus in children is avascular necrosis. The prognosis of any particular fracture depends on the location of the fracture line and the degree of displacement. The illustration of Marti ( 106 ) indicates the prognosis of any particular fracture of the talus in childhood. Nondisplaced fractures appear to have a better prognosis. However, Letts and Gibeault ( 95 ) noted the development of avascular necrosis in 3 of 12 nondisplaced talar fractures in children, a disconcerting proportion. On the other hand, Jensen et al. ( 78 ) reported no avascular necrosis in their 11 patients with nondisplaced talar fractures. They quoted a report in German by Linhart and Höllwarth ( 96 ) that noted a 27% incidence of avascular necrosis after nondisplaced fractures.

Hawkins ( 69,179 ) reported that no avascular necrosis resulted from nondisplaced fractures (type I) in his series of 57 talar neck fractures in adults. All type II fractures united, but the rate of avascular necrosis was 42%. Type III fractures, with subluxation or dislocation of both the subtalar and ankle joints, had a 91% rate of avascular necrosis following these severe injuries.

Mazel et al. ( 109 ) also noted a worse outcome in their younger patients with talar fractures associated with severe multiple trauma, such as falls from a considerable height. The severe fractures resulting from lawn mower injuries also have a worse prognosis.

The subchondral lucency in the dome of the talus, the so-called Hawkins sign, is an excellent indicator of viability of the talar body after fracture. Canale and Kelly ( 23 ) found that some patients, mostly those with nondisplaced fractures and relatively short periods of immobilization, did not develop this sign, presumably from the lack of disuse hyperemia. Thus, absence of this radiographic finding of subchondral lucency in a child does not necessarily indicate avascular necrosis, although it should raise suspicion that it may be developing.

Ogden ( 123 ) pointed out that the subchondral area in younger children is cartilaginous, which obviously interferes with the ability to see this area radiographically. MRI is now regarded as the noninvasive method of choice for the diagnosis of osteonecrosis; it may demonstrate findings on technetium scanning to be misleading ( Fig. 26-11 ). MRI is clearly the optimal study at this time for the early diagnosis of avascular necrosis, and is useful for evaluating the extent or percentage of talar body involvement and potential for segmental collapse ( 166 ). Segmental collapse occurs in approximately one third of patients with total talar body involvement; however it is uncommon in patients with only partial avascular necrosis ( 40 ).



**FIGURE 26-11.** A: Injury film of a boy 11 years and 11 months of age who had a crush injury of the foot, along with other severe injuries (including head injury), sustained as a passenger in a motor vehicle accident. The talar neck is displaced and the tibiotalar joint subluxed. An open reduction was performed with the medial malleolar fracture fixed with Kirschner wires. The talus was not fixed. B: One month later, avascular necrosis of the talus is evident. A split-thickness skin graft was applied after further wound debridement, and coverage was obtained. Weight relief was afforded with a patellar tendon-bearing ankle-foot orthosis. C: Six months after injury, spontaneous drainage was noted from the posterior part of the wound. Film demonstrates avascular necrosis of the body. D: Technetium bone scan showed uptake in the body of the talus. E: Magnetic resonance imaging scan definitively demonstrated avascular necrosis and collapse of the talar body, despite the increased uptake on bone scan. F: Talectomy was performed, and the subsequent functional result has been gratifying. (Courtesy of J. Banta, M.D.)

The necessity of non-weight bearing in the management of avascular necrosis after fracture of the talus has not been determined. Hawkins recommended restricting weight bearing only until union was established, even in the presence of avascular necrosis ( 69,70 ). He stated that collapse of the talus was well tolerated by most patients, and that non-weight bearing did not necessarily protect against collapse. Furthermore, a period of several years may be required before radiographic evidence of revascularization is complete, and this period of non-weight bearing is difficult to impose. However, in the series reported by Letts and Gibeault ( 95 ), two of three children with avascular necrosis developed flattening of the talus and stiff ankles. Both of these children were allowed to bear weight, because the fracture was not recognized in one and the developing avascular necrosis was not recognized in the other. In another child who was not allowed to bear weight, the talus revascularized in 6 months with no residual deformity.

Canale and Kelly (23) recommended non-weight bearing for avascular necrosis, noting that the results in patients treated with non-weight bearing were superior to those allowed to bear weight. None of their 22 patients with avascular necrosis had undergone surgical treatment after an average of 15 years' follow-up. Stephens' (162) patients, both with displaced fractures, apparently developed satisfactory final results with early weight bearing, but radiographic deformity was present.

Avascular necrosis can run a protracted course (Fig. 26-12), and protected weight bearing until reossification is complete can be a test of patience for the child, family, and physician. In circumstances such as these, the conservative approach generally is recommended. Either approach, allowing weight bearing or prolonged protection from weight bearing, has undesirable side effects, and these decisions should be made with the family. Whether weight bearing can be sufficiently limited in children by a patellar tendon-bearing (PTB) orthosis is problematic; my impression is that a long leg cast or orthosis with a flexed knee is the only way to ensure non-weight bearing early, with conversion to a PTB orthosis until reossification is complete. Parents should be made aware that late segmental collapse may occur despite this treatment and may be more related to the natural history of avascular necrosis than to the choice of treatment or weight-bearing status.



**FIGURE 26-12.** A: Nondisplaced fracture of the talar neck in a 5-year-old boy at injury. B: Three months after injury. C: Eighteen months after injury, irregularities of the subchondral bone are evident. D: Nine years after injury. Clinically he is asymptomatic, and radiographically the subchondral radiolucencies have reossified. (Courtesy of S. Terrance Canale, M.D.)

### Varus Malalignment

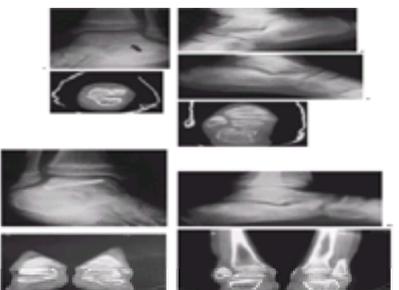
Varus malalignment is the most common posttraumatic deformity. Its presence leads to a varus hindfoot combined with a supinated forefoot as a result of medial column shortening (38,83,98). In a biomechanical study, 17 degrees of varus malalignment restricted 30% of subtalar motion (38). The varus malalignment should be corrected when possible, although its long-term significance in a child is undetermined. The potential for remodeling of a child's talus is illustrated with a 10-year follow-up of a 1-year-old boy with extensive open injury to the talus after his foot was crushed by an automobile (Fig. 26-13). He regenerated a peculiar-appearing but functional talus and can carry out all activities of early teenage years without difficulty. Figure 26-13 depicts the foot position of a talar neck fracture that healed or remodeled into varus. This example suggests the merits of a conservative approach before recommending talectomy for the residual effects of a fractured talus, unless infection is present or function is severely impaired.



**FIGURE 26-13.** An open crushing injury to the talus and distal tibia in a 2.3-year-old boy whose foot was run over by an automobile. A: At the time of injury. B: Avascular changes are present 6 months after injury. C: Eleven years after injury, he is clinically asymptomatic and has 10 degrees dorsiflexion and 30 degrees plantarflexion of the involved ankle. D: Clinical appearance of the feet.

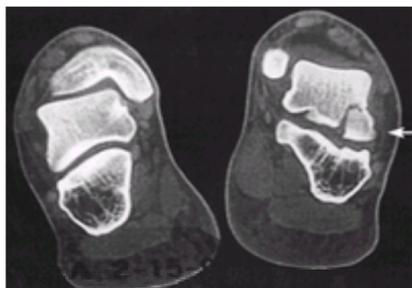
### Other Fractures of the Talus

Fractures of the body (dome) of the talus appear to be unusual in children. Spak (160) reported two compression fractures of the talus in children; both were treated conservatively and showed radiographic deformity at 1 year of follow-up. Clinically, no deformity or loss of function was noted, but a 1-year follow-up for such an injury is very short. Marti (106) has recommended anatomic reduction of displaced fractures of the talar body (Fig. 26-14). Because the ankle joint does not tolerate derangement of articular surfaces well, it would seem prudent to attempt accurate reduction of any intraarticular fracture, regardless of the patient's age. The relationship between residual displacement and subsequent osteoarthritis has been documented in adults. Complications related to displacement can be controlled by the treating surgeon, but the frequent disruption of vascularity is a result of the injury. Whether early accurate reduction affects the rate of vascular complications is conjectural.



**FIGURE 26-14.** A 10-year-old boy sustained injuries to both feet from a fall. A and B: Displaced fracture of the right talar body. The arrow on the lateral view points to the talar fracture; the calcaneus is normal on this side. C: CT scan documents actual displacement of fracture. An open reduction was performed. D: Lateral view of left foot, demonstrating flattening of Böhler's angle secondary to an intraarticular fracture of the calcaneus. E: Widening and depression of calcaneal joint surface secondary to fracture. The fracture was manipulated in an effort to decrease hindfoot widening and was treated nonoperatively. F: Right talus 6 months after injury. The talus is viable, and the joint surface is congruous. G: Left calcaneus 6 months after injury. The joint surface is well maintained; the lateral process of the talus has hypertrophied, but Böhler's angle is still flattened. H: Computed tomography scan of talus 6 months after injury. The ankle joint surface is well maintained. I: Computed tomography scan of calcaneus 6 months after injury. The joint surface of the calcaneus has remodeled. Hindfoot posture is symmetric, with residual increased width of the calcaneus. The patient is asymptomatic. (Courtesy of Keith Merrill, M.D.)

The lesser fractures of the talus involving the lateral or posterior process appear to be rare. However, with the increasing popularity of snowboarding as a recreational activity for the younger population, fractures of the lateral talar process are being recognized with increasing frequency. Winter sports data have indicated that 80% of children who participate in snow sports have ridden snowboards by age 12 (111). Kirkpatrick et al. (89) reported that lateral talar process fractures represented 34% of fractures sustained about the ankle in snowboarders. The fracture is often missed on initial presentation and presents late as a "persistently painful sprained ankle." The mechanism of injury is believed to be dorsiflexion of the ankle combined with inversion of the hindfoot (69). Internal rotation views of 20 to 25 degrees better demonstrate this fracture than traditional anteroposterior, lateral, and mortise views (115). CT scanning has been recommended for better delineation of the fracture (Fig. 26-15). Treatment is based on the size and displacement of the fracture fragment. For fragments larger than 1 cm and with more than 2 mm of displacement, open reduction and internal fixation has been recommended (83,106). Nondisplaced or minimally displaced fractures (<2 mm) can be treated in a short leg non-weight-bearing cast. For small displaced fragments or those with significant comminution, simple exclusion of the fragment can be performed if painful symptoms persist after conservative treatment.



**FIGURE 26-15.** Coronal images of a computed tomography scan best demonstrate this rather large displaced fracture of the lateral talar process (arrow). The contralateral foot displays normal anatomy. (Reprinted from Kirkpatrick DP, Hunter RE, Janes PC, et al. The snowboarder's foot and ankle. *Am J Sports Med* 1998;26:275; with permission.)

A bipartite talus simulating a fracture has been reported (180) but appears to be so rare that treatment must be individualized. The os trigonum, a normal variant, can sometimes be confused on the radiographs with a fracture of the posterior process of the talus. This accessory center of ossification generally appears at 8 to 10 years of age in girls and 11 to 13 years of age in boys (62). It usually fuses to the body about a year after it appears radiographically, but it can remain separate and be a source of confusion. Careful inspection of the os trigonum reveals that it has smooth, rounded edges, not the sharp, jagged edges of a fracture.

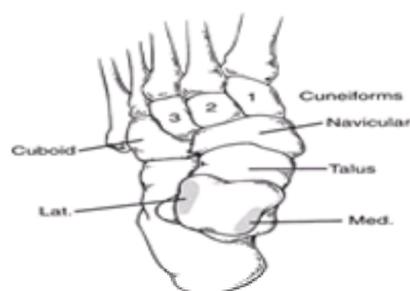
## OSTEOCHONDRAL FRACTURES OF THE TALUS

Several terms are commonly used to describe this entity, which previously could be diagnosed only by radiography or tomography (3,8,12,22,61,109,128,157). The use of modern imaging and arthroscopic surgical techniques has challenged some of our traditional tenets regarding this condition. In the literature, it may be called "osteochondral defect or lesion," "osteochondritis dissecans," "transchondral fracture," or "osteochondral fracture." Newer imaging techniques have demonstrated that some lesions may not involve the cartilage and may not in fact be "transchondral" (6).

Although this entity is most common in early adult life, it does occur in teenagers. Most of the patients in the series reported by Canale and Belding (22) were in the second decade of life at the onset of symptoms.

### Mechanism of Injury

Berndt and Harty (12) coined the term *torsional impaction*, which is useful in understanding the mechanism of injury of these lesions. They experimentally reproduced the posteromedial lesion by inversion and plantarflexion of the foot combined with external rotation of the tibia (Fig. 26-16). Inversion and dorsiflexion produced the anterolateral lesion that could be accompanied by rupture of the fibular collateral ligaments. The posteromedial lesion was slightly more common, but in Canale and Belding's series (22) the lateral lesion predominated. Unless the fractured fragment is nondisplaced, healing is unlikely. If displacement is present, or if immobilization is incomplete, ingrowing capillaries from the body of the talus cannot invade the fractured fragment, and a layer of dense fibrous connective tissue becomes interposed between the fragment and the body. The articular cartilage (106), dependent only on the available synovial fluid, continues to thrive. The fracture fragment becomes avascular, "a dead prisoner in a sterile cell."



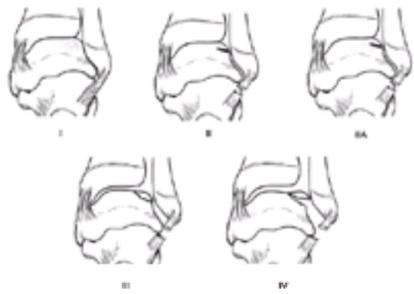
**FIGURE 26-16.** The usual sites of transchondral fractures of the talus in 201 cases reported by Berndt and Harty. The medial side was involved in 56%, the lateral in 44%. (Reprinted from Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg [Am]* 1959;41:997; with permission.)

Canale and Belding (22) modified the concepts of Berndt and Harty (12) in that they could not elicit a history of injury as reliably for medial injuries as for lateral ones. They speculated that not all medial lesions were traumatic in origin. They also noted that the medial lesions were more often cup shaped, the lateral lesion wafer shaped (Fig. 26-17).



**FIGURE 26-17.** The medial lesion is deeper and cup shaped; the lateral lesion is thin and wafer shaped. (Reprinted from Canale ST, Belding RH. Osteochondral lesions of the talus. *J Bone Joint Surg [Am]* 1980;62:100; with permission.)

Anderson et al. (6) described a modified staging system for the classification of osteochondral fractures based on their examinations with CT and MRI ( Fig. 26-18). Stage I, recognizable only with MRI scanning, consists of a subchondral fracture without collapse. Stage II denotes incomplete separation of the fragment, with the subcategory of IIA including a subchondral cyst. Stage III describes an unattached, undisplaced fragment, and stage IV an unattached, displaced fragment.



**FIGURE 26-18.** Modified staging system of Berndt and Harty proposed by Anderson and co-workers. Stage I is confined to subchondral trabecular compression, only identifiable by magnetic resonance imaging. Stage II denotes incomplete separation of an osteochondral fragment; if a subchondral cyst is present, the lesion is classified as IIA. Stage III designates the fragment as unattached but undisplaced. A displaced fragment would be classified as stage IV. (Reprinted from Alexander IF, Chrichton KI, Grattan-Smith Y, et al. Osteochondral fractures of the dome of the talus. *J Bone Joint Surg [Am]* 1989;71:1143; with permission.)

### Signs and Symptoms

Osteochondral fractures have characteristically been diagnosed after a sprained ankle that does not resolve symptomatically.

### Radiographic Findings

Smith and co-workers (157) recommended anteroposterior, lateral, and oblique radiographs for any persistently painful ankle sprain. Anderson and co-workers (6) incorporated newer imaging techniques for a diagnostic approach after study of 30 patients undergoing examination for persistent posttraumatic instability of the ankle.

The earliest stage of osteochondral fracture, microfracture of the subchondral bone, is not evident on conventional radiography. Small nondisplaced fractures also may not be visible unless the foot is positioned in such a way as to demonstrate the fracture. If symptoms persist after an ankle sprain, the previously recommended approach was to obtain bone scintigraphy (6). This approach is falling out of favor, with MRI as the alternative. Even when radiographs are negative, MRI can detect an early osteochondral defect as well as other soft tissue pathology without exposing the child to the radiation involved with bone scanning. MRI is superior for assessing the cancellous bone, especially with early lesions, but CT scanning is generally more helpful for staging and preoperative planning to locate the exact position of the fracture. Therefore, MRI is unnecessary for patients with lesions evident on conventional radiographs, but is helpful in those with positive bone scans and apparently normal radiographs. CT scanning is helpful for surgical planning.

## AUTHOR'S PREFERRED METHOD OF TREATMENT OF OSTEOCHONDRAL FRACTURES

Treatment principles for osteochondral fractures of the talus are becoming fairly well established. For stage I and II lesions, a trial of 6 weeks of non-weight-bearing immobilization is warranted—the theory is that eliminating shear forces will allow reestablishment of vascular continuity. For persistently symptomatic lesions, surgical treatment is warranted. Arthroscopic treatment has become the preferred approach, because it is accompanied by less postoperative morbidity and can eliminate the need for malleolar osteotomy when treating posteromedial lesions. Arthroscopic techniques for moderate-sized lesions have focused on penetration of the subchondral bone surface (10,16,20,51,61,81,97,124,128,171). These techniques include subchondral drilling, abrasion arthroplasty, and the microfracture technique. The goal of these procedures is recruitment of stem cells brought via formation of a fibrin clot with subsequent differentiation into fibrocartilaginous repair tissue (21). Recent technological advances in the repair of cartilaginous lesions has led to alternative techniques with the goal of providing hyaline cartilage repair tissue. These include osteochondral autografts (mosaicplasty), periosteal and periosteal grafting, and allograft and autogenous chondrocyte implantation. The follow-up studies of these techniques have been short term and primarily in adults. Longer follow-up will determine whether these results offer improved results compared with previous methods. A medial malleolar osteotomy usually is required for open treatment of medial lesions (8,18,101). This technique should be restricted to older adolescents whose physis had closed.

Postoperative management of arthroscopically treated defects varies with the stage of the lesion and the procedure performed. If, for instance, a stage II lesion is drilled, cast immobilization is indicated in a young patient in an attempt to gain healing. Conversely, if an unattached fragment is debrided and a crater created, early motion is mandatory to create a favorably shaped fibrocartilaginous repair. The point at which weight bearing can be safely resumed is conjectural, but obviously depends in some degree on the site and extent of the lesion.

An intriguing alternative to arthroscopic management of medial osteochondral fractures in children was described by Greenspoon and Rosman (61). They bone grafted the defect after curettage and improved their percentage of favorable results when compared with curettage alone. However, an osteotomy of the medial malleolus is required.

### SUBTALAR DISLOCATION

Subtalar dislocations are extremely rare in young children. Dimentberg and Rosman (44) reported five subtalar dislocations in children, two of which had delayed diagnosis due to the presence of other injuries. Zimmer and Johnson (190) reported this injury in a 15-year-old girl, but she was skeletally mature. Miller and Lehman (112) reported a 17-year-old patient who fractured a calcaneonavicular coalition accompanying a subtalar dislocation. Subtalar dislocations usually are caused by a plantarflexion mechanism (40,41,63), but, as in adults, complex injury patterns can cause associated injuries that complicate the subtalar dislocation and worsen the prognosis (28,110). Treatment of subtalar dislocation is closed reduction, with pin stabilization if the reduction is unstable.

### CALCANEUS

Before 1982, fractures of the calcaneus in children were rarely reported (26,107,165). Of the 241 fractures of the calcaneus reported by Essex-Lopresti (49), only 12 were in the youngest age group, 9 to 20 years. Thomas (165) reported on five boys with fractures of the calcaneus in 1969, the largest reported series of that era. He found comminution and joint depression to be common. Marti's clinic in Amsterdam (106) treated three fractures of the calcaneus in children over a 10-year period; the youngest was 12 years old. Matteri and Frymoyer (103) described three calcaneal fractures in young children.

Schmidt and Weiner (150) reported that they had discovered 59 fractures of the calcaneus in patients under 20 years of age, of which 46 were in skeletally immature children. This was by far the largest series reported to date (1982). One third of the children had associated injuries, including three lumbar vertebral fractures. In 16

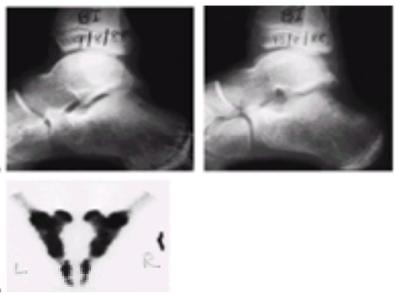
patients, the fracture was initially unrecognized. Soon thereafter, Wiley and Profitt ( 184) reported an additional 34 fractures of the calcaneus in children, and in 1986 Rasmussen and Schantz (133) reviewed 143 calcaneal fractures in patients under the age of 19.

The increased frequency of reports of calcaneal fractures in children since the early 1980s appears to be a result of improved diagnosis of nondisplaced or minimally displaced fractures. Children have fewer intraarticular fractures than adults, presumably because of less exposure to trauma, and have less severe injuries when fractures do occur (147). All studies reporting large numbers of calcaneal fractures in children mention difficulty in diagnosis, delay in diagnosis, and the benign nature of calcaneal fractures in children.

Younger children generally have fewer displaced fractures or fractures of the tuberosity or apophysis; fractures of the anterior process and inferolateral process are more common in older children. Displaced intraarticular fractures are much more common in older children.

None of the children studied by Wiley and Profitt ( 184) underwent surgery, but 3 of 20 patients examined at short-term follow-up did have mild symptoms. De Beer et al. (34) reported the outcome of nine children with calcaneal fractures, six of which were intraarticular. None were treated operatively, and all the patients were pain free at short-term follow-up. Schmidt and Weiner (150) did not report follow-up, nor did Rasmussen and Schantz (133), but both suggested that a good result usually can be expected (excepting obvious complicating factors such as lawn mower injuries).

Although operative treatment of intraarticular calcaneal fractures in adults has become standard, younger children may not require the same approach. Sandermann et al. (142) reported two patients treated nonoperatively, both with good results. Rasmussen and Schantz ( 133) studied 15 children with displaced intraarticular fractures an average of 12 years after injury and reported only "slight inconvenience" ( 148). When treated conservatively these fractures may heal, often with minimal symptoms, but they can result in significant restriction of subtalar motion and lead to problems with shoe wear. In adolescents, as the fracture patterns begin to resemble those of adults and less remodeling is anticipated, operative management should be considered if significant joint involvement or displacement occurs. Stress fractures of the calcaneus also have been reported in children ( Fig. 26-19).



**FIGURE 26-19.** Stress fracture of the calcaneus in a boy 13 years and 10 months of age, whose symptoms began 2 weeks after starting football practice. **A:** Normal radiograph at onset of symptoms. **B:** Normal bone scan at onset of symptoms. **C:** Radiograph 4 weeks later, showing characteristic findings of stress fracture. (Courtesy of H.L. Brilliant, M.D.)

## Anatomy

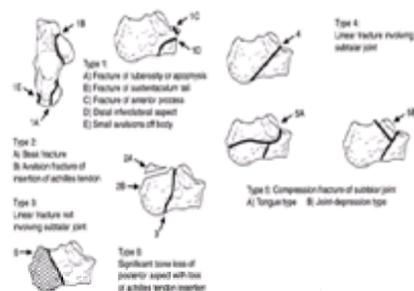
The calcaneus is the largest bone of the foot and the earliest to ossify. The posterior projection is called the tuberosity and has two plantar processes, which serve as sites of origin of the plantar fascia and the short muscles of the foot. The bone generally is rectangular when viewed laterally, with the posterior facet inclining superiorly to become the highest point of the calcaneus at the posterior margin of its articular surface ( 68). Flattening of this facet is of considerable clinical importance. The superior articular surfaces of the calcaneus are complex and adapted for articulation with the talus. The sustentaculum projects medially, supporting the end of the talus, and the anterior surface articulates with the cuboid.

The calcaneal apophysis covers the calcaneal physis, essentially occupies the tuberosity during the growing years, and demonstrates many irregularities in ossification ( 175). The heel cord inserts into the inferior portion of the tuberosity.

The differences in fracture patterns in children and adults are to some degree results of the anatomic differences between the immature and mature talus and calcaneus. The lateral process, the wedge that impacts on the calcaneus in joint depression injuries, is tiny in the immature talus, and the posterior facet is parallel to the ground rather than inclined. Add the cartilaginous covering of the bony nucleus, and it is easy to see how injuring forces from the lateral process in an adult are dissipated over a wider area in a child, with less severe injuries as a result.

## Classification of Calcaneal Fractures

Most series have used a classification based on the original work of Essex-Lopresti ( 49,133,150,184). Generally, involvement of the subtalar joint is first determined, with subpatterns for each large group. The classification ( Fig. 26-20) is difficult to memorize, but it may be helpful in analyzing data.



**FIGURE 26-20.** Classification used to evaluate calcaneal fracture pattern in children. **A:** Extraarticular fractures. **B:** Intraarticular fractures. **C:** Type VI injury with significant bone loss, soft tissue injury, and loss of insertion of Achilles tendon. (From Schmidt TL, Weiner DS. Calcaneus fractures in children: an evaluation of the nature of injury in 56 children. *Clin Orthop* 1982;171:150; with permission.)

The classification of calcaneal fractures is outlined as follows:

### Type 1

- A. Fracture of the tuberosity or apophysis
- B. Fracture of the sustentaculum
- C. Fracture of anterior process
- D. Fracture of anterior inferolateral process

E. Avulsion fracture of body

Type 2. Fracture of posterior and/or superior parts of tuberosity

Type 3. Fracture of body not involving subtalar joint

Type 4. Fracture through the subtalar joint without displacement

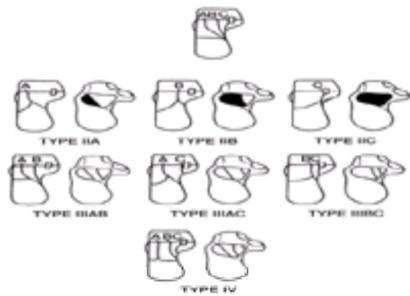
Type 5. Displaced fracture through subtalar joint

A. Tongue type

B. Joint depression type

Type 6. Either unclassified (Rasmussen and Schantz) or serious soft tissue injury, bone loss, and loss of the insertions of the Achilles tendon

Intraarticular fractures are more accurately analyzed by CT scanning, with treatment individualized to age and degree of displacement in addition to fracture classification (57,126). Intraarticular fractures in adolescents may resemble adult fracture patterns. The Sanders CT classification scheme is commonly used to describe these patterns when the posterior facet is involved (Fig. 26-21).



**FIGURE 26-21.** Sanders computed tomography classification of intraarticular calcaneal fractures. (Reprinted from Sanders R. Intra-articular fractures of the calcaneus: present state of the art. *J Orthop Trauma* 1992;6:254; with permission.)

### Mechanism of Injury

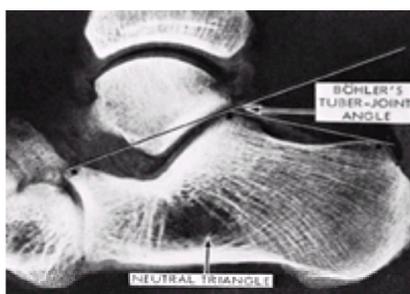
Closed fractures of the calcaneus in children almost always occur as the result of a fall; however, the height of the fall appears to be less in children, possibly accounting for the lesser severity of injury in children's calcaneal fractures (148). Open fractures resulting from lawn mowers also are relatively common and are discussed later in the chapter.

### Signs and Symptoms

Pain, localized swelling, tenderness, a history of a fall, and inability to walk as a result of this injury are clear indications of diagnosis. Many minimal fractures of the calcaneus are unrecognized, and careful palpation of this region in a child refusing to walk may provide the key to diagnosis. Localized tenderness in the region of the calcaneus in a toddler should raise suspicion of a calcaneal fracture, although the diagnosis may be difficult to establish without extensive imaging. Whether it is necessary to use such extensive technical assistance is subject to clinical judgment.

### Radiographic Findings

Traditionally, three views (dorsoplantar, lateral, and axial) have been recommended for the diagnosis of calcaneal fractures, but many nondisplaced or minimally displaced fractures are not recognized at the time of injury. Rasmussen and Schantz (133) added the lateral oblique view, taken with the knee tilted medially with the foot on the cassette. They believed that all four projections should be routinely obtained. A lateral view of the spine should be made in the presence of more obvious fractures, because Schmidt and Weiner (150) reported that compression fractures of the spine can relate to fractures of the calcaneus in children as well as in adults. The lateral view, often not helpful in diagnosing minimally displaced fractures, is quite helpful in assessing joint depression. The tuber-joint angle (Böhler's angle, salient angle) is formed by a line parallel to the articular surfaces of the calcaneus, with a line drawn from the posterior lip of the posterior facet to the superior margin of the calcaneal tuberosity (Fig. 26-22) (100). An angle less than the uninjured side indicates depression of the joint surface. The tuber-joint angle is normally smaller in young children than the 25 degrees to 40 degrees that is normal in adults.

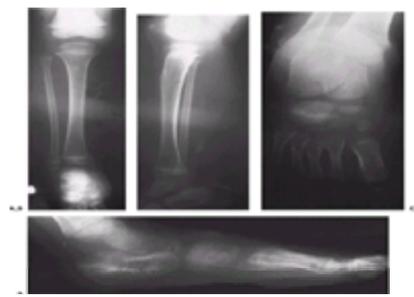


**FIGURE 26-22.** The landmarks for measuring Böhler's angle are the anterior and posterior facets of the calcaneus and the superior border of the tuberosity. The neutral triangle, largely occupied by blood vessels, offers few supporting trabeculae directly beneath the lateral process of the talus. (Reprinted from Harty MJ. Anatomic considerations in injuries of the calcaneus. *Orthop Clin North Am* 1973;4:180; with permission.)

### Bone Scan

Starshak, Simons, and Sty (161) evaluated 10 children between the ages of 19 and 41 months with the so-called toddler's fracture to determine the role of scintigraphy in the diagnosis of unrecognized calcaneal fractures. Only 4 of the 10 patients with positive scans had radiographically demonstrable fractures. In 2 others, a fracture was evident on follow-up films 12 days later. The others had no follow-up films because symptoms had subsided. A similar series more recently reported by Laliotis and co-workers (90) also recommended bone scintigraphy. Schindler et al. (149) reported five occult fractures of the calcaneus in children under 36 months of age. Initial radiographs were normal, and treatment in a long leg cast was elected based on clinical examination results. Radiographs at 2- and 4-week intervals confirmed the diagnosis with notable sclerosis. They found no need for bone scanning in these individuals. I often apply a short leg walking cast for 2 weeks when confronted with a toddler who refuses to walk and has foot tenderness and normal radiographs. If after 2 weeks the child still cannot walk, further imaging can

be undertaken. If the child can walk, treatment is finished at considerably less cost than if scintigraphy were used ( [Fig. 26-23](#)).



**FIGURE 26-23. A and B:** Radiographs of the leg and foot of a 2-year-old girl who refused to bear weight on the foot. No fracture was identified. A careful physical examination could have guided the treating physician toward a more localized radiographic examination. **C:** Two weeks later, tenderness of the calcaneus elicited on follow-up examination. Repeat radiography revealed a nondisplaced fracture of the lateral calcaneus. **D:** Obvious callus formation in the calcaneus 4 weeks after injury. (Courtesy of Paul Griffin, M.D.)

### Computed Tomographic Scanning

Several reports of the value of CT scanning have been documented since the last edition of this book was published, and the role of CT scanning in the evaluation of calcaneal fractures is now well established ([4,32,48,143](#)). CT scanning has probably been the primary factor in the reassessment of treatment modalities for adults with calcaneal fractures, and anatomic reconstruction is emphasized much more than before the advent of CT scanning. For intraarticular fractures, CT scanning more reliably demonstrates the degree of anatomic alteration of the subtalar joint; for process fractures, CT scanning may be the only way to demonstrate a small fracture. The role of CT scanning for children's calcaneal fractures has been studied less extensively, but CT is warranted in fractures with significant comminution and in older adolescents.

Pablot et al ([126](#)) reported that CT scanning in adolescents with calcaneal fractures revealed fracture patterns similar to those in adults. Although this severity of injury in younger children is rare, Abel and Wenger reported open reduction of a calcaneal fracture in a 3-year-old on the basis of CT findings ( [Fig. 26-24](#)).



**FIGURE 26-24.** A boy 3 years and 9 months of age was struck by a car and sustained fractures of the talus and calcaneus. **A:** X-rays on the day of the injury demonstrate joint depression fracture of the calcaneus and minimally displaced fracture of the talar neck. **B:** Computed tomography scans demonstrate comminution and lateral spread of the calcaneus. **C:** Both fractures underwent open reduction and Kirschner wire fixation. **D:** Fractures healed in good position 8 weeks after injury. The child was lost to follow-up after this time. (Courtesy of D.R. Wenger, M.D., and M.E. Abel, M.D.)

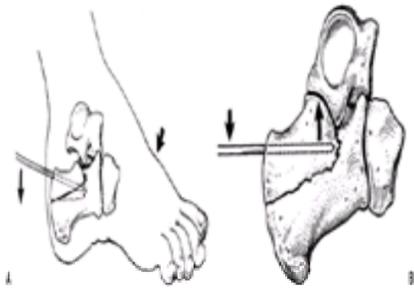
### AUTHOR'S PREFERRED METHOD OF TREATMENT OF CALCANEUS FRACTURES

Most calcaneal fractures in very young children heal uneventfully, even when not promptly diagnosed. Treatment is relatively simple in this age group and consists of immobilization until healing is present in a few weeks. Restriction of weight bearing is not necessary for a nondisplaced fracture in a young child. In older children or in any displaced intraarticular fracture for which nonoperative treatment is selected, non-weight bearing is mandatory to prevent further displacement.

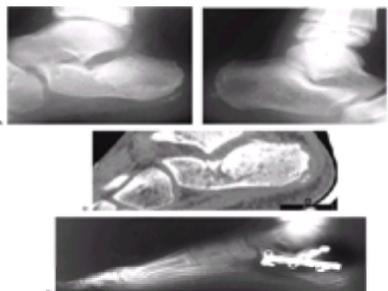
Thomas ([165](#)) gave a complete anatomic description of the five fractures in his series. One child sustained a tongue-type fracture; the other four demonstrated a similar fracture line commencing immediately behind the articular margin of the posterior facet to a delta-shaped comminuted fracture on the anterior or lateral cortex. There also was a second fracture, much less distinct, running from about the mid-portion of the first fracture toward the anterior margin of the posterior facet. This fragment appeared to involve the entire facet, because the axial view did not demonstrate the lateral compression usually seen in adults. All children were initially treated by immobilization with soft dressing and elevation. Active and passive motion of the foot was permitted. Weight bearing was forbidden for 6 weeks, after which all the patients regained a full range of painless motion and walked without a limp within 1 week. Follow-up examinations were performed 9 to 22 months after injury, and the results were satisfactory in all patients. Two patients had "trivial" limitation of motion in the subtalar joint, but range of motion was full in the other three. The clinical appearance was identical to that of the noninjured foot.

In four of the patients reported by Thomas, the Bohler's tuber-joint angle was 6 to 12 degrees less on the injured side, indicating some degree of joint depression. However, the inferior articular facet of the talus appeared to overgrow to such a degree as to accommodate the joint depression of the calcaneus precisely. The same researcher described a "talar facet" angle, measuring the overgrowth of the inferior articular facet of the talus; the sum of this angle, and the tuber-joint angle equaled the sum of the two angles on the normal side. He concluded that this remodeling potential of the talus minimized the effect of the depression of the calcaneal facet, and that a satisfactory clinical result was thus obtained. I have observed the same phenomenon.

Marti ([106](#)), however, stated, "A fracture of the calcaneus that has led to compression or simple discontinuity of the joint requires perfect reduction and reconstitution of the articular surface." He treated calcaneal fractures in three boys, ages 12 to 15, in a 10-year period. All fractures were intraarticular, although one was very minimally displaced. All were managed surgically with lag screw fixation. Operative treatment of adults with intraarticular calcaneal fractures has become standard, and in older adolescents with minimal growth remaining the same approach is appropriate. In a report by Biert et al. ( [15](#)), the researchers advocated the use of combined indirect reduction technique via ligamentotaxis through distraction, percutaneous reduction, and internal screw fixation as an alternative to conservative treatment of depressed intraarticular calcaneus fractures. They described a case of a 10-year-old with bilateral calcaneus fractures treated in this manner. An article on adults revisited the use of a percutaneous reduction technique originally described by Essex-Lopresti ( [167](#)). This technique offered an alternative to either conservative treatment or open reduction and internal fixation for certain displaced calcaneus fractures. In particular, this technique may have merit in selected displaced pediatric fractures with more of a tongue-type pattern ( [Fig. 26-25](#)). Other researchers advocate that displaced intraarticular fractures with significant joint incongruity in children ought to be treated with open reduction and internal fixation using similar methods described in adults ( [Fig. 26-26](#)). The largest series of operative fixation of intraarticular fractures of the os calcis in children was recently presented ( [164](#)). Thirty-four fractures with significant intraarticular involvement underwent open reduction and internal fixation. The researchers reported excellent results in 95% of their patients regaining near full subtalar range of motion and function at average 4.2 year follow-up ( [164](#)). The only complication noted was subsequent claw toe deformity in 4 of the 31 patients.



**FIGURE 26-25.** Essex-Lopresti reduction technique. **A:** Pin is placed into the tongue fragment. The fragment is then disengaged using the pin as a joystick. A downward force is placed on both the pin and the mid-foot as depicted. **B:** The thumbs are used as a fulcrum to elevate and reduce the posterior facet, then the pin is driven across the fracture site to stabilize the fracture. Pins or screws may be placed for definitive fixation. (Reprinted from Tornetta P III. The Essex-Lopresti reduction for calcaneal fractures revisited. *J Orthop Trauma* 1998;12:471; with permission.)



**FIGURE 26-26.** A 10-year-old boy sustained a calcaneus fracture following a trampoline injury. **A:** Radiographs displayed a severely depressed intraarticular fracture involving the posterior facet. Bohler's angle measured 15 degrees. **B:** Comparison lateral of the noninvolved side demonstrates significant difference in Bohler's angle measuring 40 degrees. **C:** Computed tomography scan further delineates amount of depression as the posterior facet has been driven down within the cancellous bone of the calcaneus. **D:** Open reduction internal fixation was performed through a lateral approach using an L-shaped incision. Anatomic reduction was achieved and maintained with a lateral calcaneal plate. No bone graft was used.

With the remodeling potential of the talus and calcaneus in a growing child, and the reported good results after conservative treatment, most intraarticular fractures in younger children can be managed nonoperatively. Certainly, with falls from unusual heights, severe joint depression is possible, and operative treatment may be best in individual instances. My approach for most children with intraarticular fractures would be non-weight-bearing immobilization for 6 weeks, because mild amounts of joint incongruity remodel very well ([Fig. 26-22](#)). In adolescents with significant joint involvement or displacement, operative management is considered. To date, there are no reports of impairment resulting from failure to surgically reduce a child's calcaneal fracture.

Extraarticular fractures seem to do well regardless of treatment, with the possible exception of fractures of the anterior process. The anterior process is not seen radiographically until about age 10 and varies greatly in shape. The mechanism of injury is most commonly plantarflexion and inversion, with the bifurcate ligament responsible for avulsing the anterior process via its calcaneal attachment ([40](#)). The distal portion of the fracture fragment is intraarticular and involves the calcaneocuboid joint ([Fig. 26-27](#)).



**FIGURE 26-27.** Fracture of anterior process of calcaneus in an 11-year-old girl. The fracture was evident only on the oblique view. (Courtesy of Kaye Wilkins, M.D.)

Degan et al. ([39](#)) reported 25 patients with fractures of the anterior process of the calcaneus. Eighteen healed with nonoperative management and became asymptomatic. Seven patients had excision of the fracture fragment because of nonunion and pain, and three of these seven were 13 to 17 years of age. Drvaric and Schmitt ([47](#)) reported an irreducible fracture of the calcaneus in a 4-year-old boy that resulted from displacement of the peroneal tendons into the fracture site. Degan et al. ([35](#)) thought this injury was often missed because the injuring force is usually that of an ankle sprain. However, the point of maximal tenderness indicated the diagnosis if pertinent radiographs were made. In addition to conventional views, an oblique radiograph made with the beam directed 10 to 15 degrees superior and posterior to the middle of the foot, as recommended by Bachman and Johnson ([9](#)), was considered helpful. A protracted period of disability was common with this injury, which often was not initially diagnosed.

There is some controversy about the need for open reduction of larger displaced anterior calcaneal fractures ([40](#)). With the problems reported by Degan et al. ([39](#)), a good case can be made for open reduction if severe articular displacement and joint depression are obvious.

Cole et al. ([29](#)) reported four patients with avulsion fractures of the tuberosity of the calcaneus, three of whom had taut overlying skin secondary to fracture displacement. Excellent results were obtained with open reduction of the three displaced fractures.

## FRACTURES OF THE LESSER TARSAL BONES

Isolated fractures of the mid-foot are extremely unusual, although direct trauma, such as an object falling from a height, can produce such fractures. More often, they are accompanied by other injuries, and usually indicate a severe injury to the foot. Irregularities of ossification of the navicular bone can be confused with fracture, but lack of localized tenderness or the rounded bony margin should be adequate to differentiate between ossification irregularities and fracture. Treatment of these injuries is generally uncomplicated but may be determined by the nature of associated injuries. Open reduction and internal fixation is indicated for displaced intraarticular injuries.

## TARSOMETATARSAL INJURIES

Tarsometatarsal injuries, although well documented in adults (185), have been described rarely in children. In fact, Trillat et al. (170), in a review of 81 fracture–dislocations of the tarsometatarsal joint, stated that “the lesion is not seen in children.” Wiley (181), however, reported on a series of 18 injuries to this region in children under age 16, and Cehner (24) described the management of an open fracture–dislocation of the tarsometatarsal joint in a 10-year-old.

### Anatomy

Wiley (182) concisely described the anatomy and mechanism of tarsometatarsal injuries. The metatarsal bases are joined to each other and the tarsals by tough ligaments, with one exception: the ligamentous connection between the base of the first and second metatarsals is rather flimsy in comparison (74). In fact, Serrafian's (153) text noted that “there is no ligament between the first and second metatarsal on the dorsal or plantar aspect. The interosseous connection is through poorly individualized weak fibers.” Its presence is therefore not acknowledged on illustrations that are simplified to demonstrate the functional anatomy (Fig. 26-28). These ligamentous connections, much tougher on the plantar surface than dorsally, tether the base of the second metatarsal to all the cuneiform bones. The other metatarsal joints are all in the same plane, distal to the recessed second metatarsal base. The second metatarsal base, therefore, serves to lock the entire complex into place (2).



**FIGURE 26-28.** The ligamentous attachments at the tarsometatarsal joints. There is only a flimsy connection between the bases of the first and second metatarsals (not illustrated). The second metatarsal is recessed and firmly anchored. (Reprinted from Wiley JJ. The mechanism of tarsometatarsal joint injuries. *J Bone Joint Surg [Br]* 1971;53:474; with permission.)

### Mechanism of Injury

The tarsometatarsal joints can be injured directly or indirectly; the indirect method is by far the more common. The direct injury, sustained by a falling object, results in no specific pattern and obviously depends on the character of the object and the distance of the fall. The metatarsals generally are displaced in a plantar direction, rupturing the tough plantar ligaments. The amount of force imposed on the foot to produce this injury is considerable, and extensive soft-tissue injury can be expected.

Forces producing an indirect injury are violent abduction or forced plantarflexion of the forefoot, either alone or in combination. With violent abduction, the metatarsals are impacted laterally, fracturing the recessed base of the second and crushing the cuboid. This combination of injuries is pathognomonic of a tarsometatarsal dislocation by this maneuver. The first and fifth metatarsals rarely are fractured.

The other mechanism of tarsometatarsal dislocation is violent plantarflexion of the forefoot. Occasionally, this is a result of an object falling on the heel with the patient in a kneeling position, but is much more commonly produced when the foot is forcibly loaded in the “tip-toe” position with the ankle in marked plantarflexion (Fig. 26-29). The metatarsals are then in line with the tibia, and the weak point in the link is the feeble dorsal ligamentous support at the tarsometatarsal joint. This can occur when the patient is a passenger in a car or uses the foot to break a fall from a bicycle, motorcycle, or toboggan. Fractures of the metatarsal shaft or neck often accompany this injury, and the metatarsal joint dislocation can be unrecognized if only minimally displaced. In Wiley's (180) series of 18 tarsometatarsal injuries in children, only four were a result of direct injuries; the rest were the result of indirect injuries. Ten of these occurred after jumping in the “tip-toe” position, and two were a result of a toboggan accident.



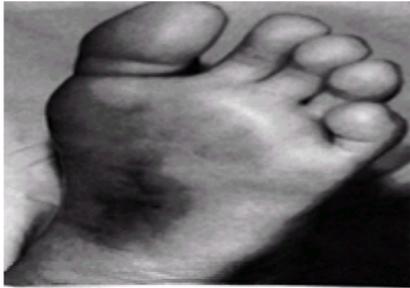
**FIGURE 26-29.** Vertical loading in the “tip-toe” position progresses to tarso/metatarsal dislocation as shown. (Reprinted from Wiley JJ. The mechanism of tarsometatarsal joint injuries. *J Bone Joint Surg [Br]* 1971;53:479; with permission.)

### Classification of Tarsometatarsal Injuries

Hardcastle et al. (65) proposed an anatomic classification of tarsometatarsal injuries modified from a 1909 publication by Quénu and Küss (131). The merits of this classification are its simplicity and practicality. Type A is incongruity of the entire joint; type B denotes partial instability (either medial or lateral); and type C, which is quite rare, indicates divergent partial or total instability.

### Signs and Symptoms

Typically swelling is present on the dorsum of the foot overlying the tarsometatarsal joints. There may not be an obvious deformity, however, because spontaneous reduction of the injury to a near-anatomic position very often occurs. Marked local pain and tenderness, accompanied by an inability to bear any weight on the foot, should increase suspicion that injury has occurred in this region. With subtle injuries, swelling may be absent, but localized tenderness persists (134). Faciszewski et al. (50) found the weight-bearing lateral radiograph view to be valuable in the evaluation of subtle injuries, demonstrating plantar displacement of the medial cuneiform bone in relation to the fifth metatarsal. Two or three millimeters of diastasis between the first and second metatarsal bases were found in every patient with a subtle tarsometatarsal injury. A diagnostic clue to the injury is ecchymosis of the plantar aspect of the mid-foot. Its presence has been named the plantar ecchymosis sign (137) (Fig. 26-30).



**FIGURE 26-30.** Plantar ecchymosis sign. Ecchymosis along the plantar aspect of the mid-foot is an important clinical finding in subtle Lisfranc tarsometatarsal injuries. (Reprinted from Ross G, Cronin R, Hauzenblas J, et al. Plantar ecchymosis sign: a clinical aid to diagnosis of occult lisfranc tarsometatarsal injuries. *J Orthop Trauma* 1996;10:120; with permission.)

### Radiographic Findings

Anteroposterior, lateral, and oblique radiographs of the foot are essential (80). A fracture of the base of the second metatarsal should alert the examiner to the likelihood of a tarsometatarsal dislocation, because often a dislocation can spontaneously reduce (Fig. 26-31). As discussed above, the combination of a fracture of the cuboid with a fracture of the base of the second metatarsal indicates the presence of a tarsometatarsal dislocation. Many researchers advocate weight-bearing radiographs or pronation/abduction stress views if a Lisfranc joint injury is suspected yet routine radiographs are normal. Stress views, however, are almost impossible to obtain in an unanesthetized pediatric patient; an ankle block or general anesthesia usually is required, limiting the usefulness of these views.

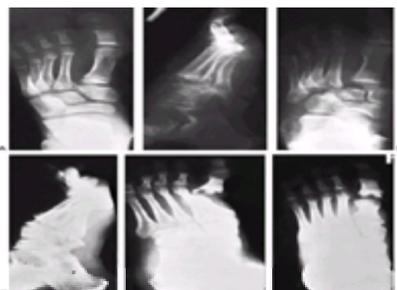


**FIGURE 26-31.** Fracture of the second metatarsal base and impaction of the cuboid are pathognomonic of disruption of the tarsometatarsal joints. (Reprinted from Wiley JJ. The mechanism of tarsometatarsal joint injuries. *J Bone Joint Surg [Br]* 1971;53:476; with permission.)

### Treatment

Treatment is relatively straightforward. The occasional minimally displaced tarsometatarsal dislocation (less than 2 or 3 mm) can be treated with elevation and a compression dressing initially (with care not to wrap the compression dressing too tightly) and application of a short leg cast when swelling has decreased. Casting is continued for 2 to 4 weeks.

For any injury with more than minimal displacement, reduction is required. Closed reduction often is successful, but supplemental Kirschner wire fixation is essential if the reduction is unstable. If closed reduction is unsuccessful, open reduction and internal fixation are indicated. The key to reduction is stabilizing the fracture of the second metatarsal base. The first and the fifth metatarsal bases may be pinned to enhance stability (Fig. 26-32) (135).



**FIGURE 26-32.** **A:** Avulsion fracture of the base of the second metatarsal in a 7.9-year-old girl. Although this injury resulted from direct trauma, this pattern of fracture of the base of the second metatarsal should alert the observer to the possibility of a tarsometatarsal disruption. **B:** A 13-year-old boy was injured when a hay wagon rolled over his foot and produced violent plantarflexion of the forefoot. With the suitable injury force, metatarsal dislocations are produced in skeletally immature patients. This injury was treated with closed reduction with pin fixation. Two years after the injury, he had minor discomfort with strenuous running or climbing in the region of the tarsometatarsal joints. **C:** A 14-year-old boy who fell 18 feet to the ground, landing in a tip-toe position, sustained a tarsometatarsal and metatarsal phalangeal dislocation of the first ray. Note that there is an impaction fracture of the cuboid, indicating an injury to the tarsometatarsal joint. Reduction was easily accomplished with closed manipulation under anesthesia. Casting was continued for 4 weeks; he was asymptomatic at follow-up. (Courtesy of James J. Wiley, M.D.)

With the patient prone, Chinese finger traps on the toes with about a 10-lb weight for traction can be used to accomplish reduction. A short leg cast, well molded over the dorsum of the foot, maintains reduction. The cast is removed at 4 weeks.

### AUTHOR'S PREFERRED METHOD OF TREATMENT

I prefer to apply finger traps with the patient supine, confirming the reduction fluoroscopically. Percutaneous pinning then is performed to stabilize the base of the first, second, or fifth metatarsals. Conventional radiographs are taken before completion of the procedure, because fluoroscopy is occasionally misleading. Hardcastle et al. (65) noted that redisplacement was most likely when the first metatarsal was inadequately fixed. Type A injuries require at least medial and lateral pins; laterally displaced type B injuries require a lateral pin; medially displaced type B injuries require one or two medial pins.

In Wiley's series (181), seven fractures required operative reduction, which can be accomplished through a dorsal incision; four were internally fixed. After reduction, I prefer to leave Kirschner wires protruding through the skin and to bend the wires at a right angle to prevent migration. A bulky dressing is applied, with padding

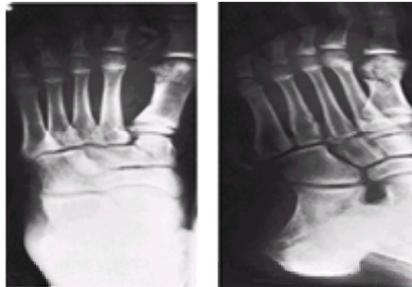
between the pins and the skin to prevent erosion of the skin by the pins. At 4 weeks, the Kirschner wires and cast can be removed.

## Results and Complications

The only series confined to pediatric tarsometatarsal injuries is that reported by Wiley (181). Fourteen of 18 patients were asymptomatic at follow-up. Of the four with symptoms, two had residual angular deformity. One patient developed Freiberg's infarction 4 months after this injury, but no definite connection between these two conditions has been established. This injury appears to be more benign in children than in adults: in a series of adults reported by Wiss et al. (186), no patient had a normal gait after a displaced Lisfranc fracture–dislocation. More recent reports have confirmed that in adults and in young adult athletes, delay in diagnosis and failure to attain an anatomic reduction have an ominous effect on the prognosis (33). Due to the recognition of poor functional outcome in adult patients when more than 2 mm of displacement was accepted, there is a trend toward a more aggressive treatment of adult transmetatarsal injuries (119). Rigid internal fixation with screws along the medial column combined with pin fixation of the lateral column has been advocated in adults.

## METATARSALS

Fractures of the metatarsals are relatively common in children. They may be a result of direct trauma from a falling object or may occur indirectly from the mechanism of injury described for injuries of the tarsometatarsal joints or from torsional forces (Fig. 26-33). A particular type of injury to the first metatarsal, “bunk-bed fracture,” was described by Johnson (80). These injuries were caused by a fall on the foot, often from a bunk bed. The proximal first metatarsal, the lateral cuneiform bone, and less often the medial cuneiform bone were impacted. In short-term follow-up, healing was uneventful and uncomplicated.



**FIGURE 26-33.** Minimally displaced fracture of the first metatarsal shaft sustained as a result of a brick falling on the foot of a 13-year-old boy. Treatment was uncomplicated, and a short-leg walking cast was used for 4 weeks.

### Fractures of the Metatarsal Shafts and Necks

Because of their relatively small diameter, the metatarsal necks fail first when torque is applied to the forefoot, accounting for the frequency of fractures at this site. Fractures of the shafts usually result from a direct crushing blow. Only one study was found that focused on metatarsal fractures in children (125): a report of 62 such fractures. Age was found to be a factor in the distribution of injury. Overall, fifth metatarsal fractures were the most common, followed by first metatarsal fractures. However, there was a noticeably higher proportion of first metatarsal fractures in children under 5 years of age (73%). The most common mechanism of injury for first metatarsal fractures was a fall; for fifth metatarsal fractures the usual mechanism was inversion; and fractures of the second, third, and fourth metatarsals were crush injuries. No fractures in this study were treated operatively.

### Signs and Symptoms

Severe metatarsal injuries produce obvious swelling, pain, and ecchymosis, but minimal swelling can occur with only slightly displaced fractures. The presence of localized tenderness, as usual, is an indispensable sign of such fractures.

### Radiographic Findings

As is true for all injuries of the foot (with the exception of the phalanges), radiographic evaluation in three planes is essential to determine the site of the fracture and the amount of displacement. The anteroposterior and oblique views generally are more revealing, but the lateral view is necessary to assess displacement toward the plantar surface of the foot. Anderson et al. (6) noted that when radiographs of the foot are made, the exposure generally is set to provide penetration of the larger tarsal bones. This results in overexposure of the smaller metatarsal and phalanges. Therefore, when injuries to the forefoot are suspected, the technician should be requested to provide optimal exposure to this area of the foot rather than to the hindfoot.

### Treatment of Metatarsal Fractures

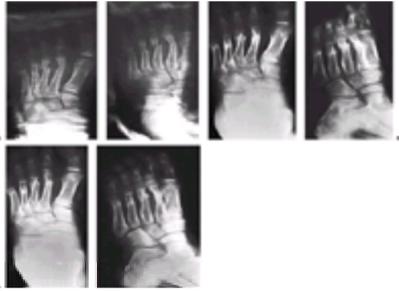
Initial management depends on the severity of injury. Admission to the hospital is indicated in the presence of marked swelling and soft-tissue injury. The interossei and short plantar muscles are contained in closed fascial compartments, so fasciotomy is indicated if swelling is severe—just as in the hand. The clinical indications for fasciotomy are marked pain and swelling with the skin stretched and taut, sometimes accompanied by venous congestion in the toes. In patients with multiple injuries, this can easily be overlooked. The long-term results of failure to perform fasciotomy of the interossei when increased tissue pressure is present are fibrosis of the interossei and an intrinsic minus foot with claw toes. All nine compartments should be released (Fig. 26-2).

Treatment of most fractures of the metatarsals in children is simple and requires only immobilization in a short leg walking cast for comfort until healing is complete. This occurs rapidly, especially in young children, usually taking no longer than 3 to 6 weeks; the shorter time is for a minimally displaced fracture in a younger child.

Despite the relative ease of management of most of these injuries, there are pitfalls, many related to plaster technique. Application of a circular cast should never be the initial measure for anything but a nondisplaced fracture with minimal or no soft tissue swelling. A circular cast should not be applied in the presence of swelling; instead, a bulky dressing (which does immobilize) with the foot in equinus position should be used for a few days. When the swelling has diminished, the cast can be safely applied. No unacceptable contractures in children will result from a few days of immobilizing the foot in equinus.

If a cast is applied and the child is sent home with the parents, written instructions should explain cast care and the danger signals of impending circulatory embarrassment.

For fractures requiring reduction, the method described by Giannestras (56) and Sammarco (141), using Chinese finger traps, is recommended (69,70). It consists simply of placing the respective toes of the injured metatarsals in finger traps with counterpressure on the ankle, a manipulative reduction, and application of a well-molded cast. When molding the cast, it is relatively safe to mold dorsally and plantarly over the metatarsals, flattening the cast, but care should be taken not to extend the mold proximal to the ankle joint. If the reduction is unstable, percutaneous Kirschner wire fixation can be helpful for the first and fifth metatarsals. Rarely is open reduction indicated. A considerable degree of lateral displacement can be accepted, and moderate amounts of dorsal angulation of the metatarsal necks that would be unacceptable in an adult will remodel in a child (Fig. 26-34).



**FIGURE 26-34.** **A:** Closed fractures of the distal shafts of the second through fifth metatarsals in a 10-year-old boy. Fracture of the third metatarsal was very unstable and motion could be felt at that fracture site 4 weeks after injury. **B:** Seven weeks after injury, the fractures are solidly healed and remodeling. **C:** Ten weeks after injury, the boy has returned to full activity. Open reduction of this type of fracture in a child is not necessary.

## OPERATIVE TREATMENT

When open reduction is required, the standard technique of dorsal exposure, Kirschner wire placement in the distal fragment exiting through the plantar skin, reduction of the fracture, and retrograde intramedullary fixation of the fracture can be used. Postoperatively, a non-weight-bearing cast (to protect the protruding pins from the plantar skin) is applied after the initial swelling has subsided. The pins are removed at about 3 weeks, and a weight-bearing cast is applied ( [Fig. 26-35](#)).



**FIGURE 26-35.** Open fractures of the necks of the second through fifth metatarsals sustained when a circular saw fell on this boy's foot. **A:** At injury. **B:** Five weeks after open reduction. Extensor tendons were lacerated, but not repaired; 0.045-inch Kirschner wires were used. No extensor lag has resulted.

## Complications

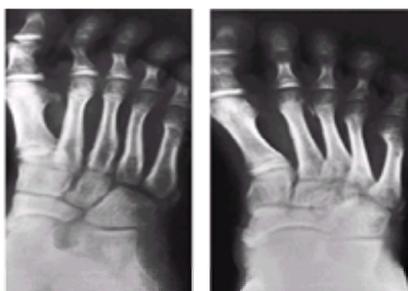
Albanese reported a 7-year-old girl with malunion of the metatarsals that interfered with ambulation. The foot injuries were not reduced initially because of a concomitant head injury. Function was improved after osteotomy ([Fig. 26-36](#)). The necessity of managing fractures expectantly in children with head injuries is underscored by this experience. The metatarsals' capability for remodeling is extensive but not infinite, and reasonable alignment must be obtained.



**FIGURE 26-36.** Malunion of metatarsal shaft fractures in a 7-year-old girl with multiple severe injuries, including head injury. **A:** Films on the day of the injury. **B:** After healing. Function was impaired by irregular weight-bearing surface of foot. **C:** After corrective osteotomy. (Courtesy of S.A. Albanese, M.D.)

## FRACTURES OF THE BASE OF THE FIFTH METATARSAL

Avulsion fractures of the base of the fifth metatarsal are fairly common in children. Differentiating such a fracture from an apophyseal growth center or os vesalianum, a sesamoid proximal to the insertion of the peroneus brevis, is the primary diagnostic difficulty. The apophysis can be recognized because its long axis is parallel with the shaft ([Fig. 26-37](#)) (14). The apophysis is not present before age 8 and usually is united to the shaft by age 12 in girls and age 15 in boys ( 35).



**FIGURE 26-37.** The apophysis at the base of the fifth metatarsal is parallel to the shaft, and is a normal variant. The fracture of the distal metatarsal shaft is not.

Historically, the mechanism of injury has been thought to be an avulsion of the metatarsal base by the action of the peroneus brevis at its insertion, but this has been

questioned. A biomechanical study implicated the tough lateral cord of the plantar aponeurosis inserting into the fifth metatarsal base ( 136). This would explain the minimal displacement and the direction of the fracture line, which is perpendicular to the longitudinal axis of the shaft ( Fig. 26-38). Dameron (35) dissected this muscle in 21 feet and found an insertion on the base of the fifth metatarsal in 18.



**FIGURE 26-38.** “Avulsion” fracture of the base of the fifth metatarsal. The fracture line appears to extend through the os vesalianum. The fibrous band of the abductor digiti minimi quinti, inserting into the base of the fifth metatarsal, probably tethers this portion of the bone as the foot is inverted and adducted. The peroneus brevis inserts into the dorsum of the fifth metatarsal, distal to the fracture line (illustrated here), and is not a likely culprit in the mechanism of injury. (Courtesy of Teresa M. Stacy, M.D.)

### Treatment

A short leg weight-bearing cast worn for 3 to 6 weeks, depending on the child's age, generally is sufficient ( Fig. 26-39). The swelling and local reaction to injury are much less than with metatarsal shaft fractures, so an initial period of elevation generally is unnecessary. Radiographic evidence of union may be hard to determine. I discontinue the external immobilization when swelling and tenderness have subsided and there is no pain with range of motion of the foot. Dameron ( 35) found that many of these fractures were asymptomatic after 3 weeks, and none had prolonged disability. Although the healing potential for this fracture is excellent, nonunion can occur. Nevertheless, these are rarely symptomatic, with many likely representing a fibrous union. Small, symptomatic nonunions can be excised. For larger symptomatic nonunions, fixation with a small lag screw may be considered. Operative fixation of acute tuberosity avulsion fractures is rarely indicated, but may be considered for significant displacement (>3 mm) in a young active patient with an unusually long tuberosity ( 36).

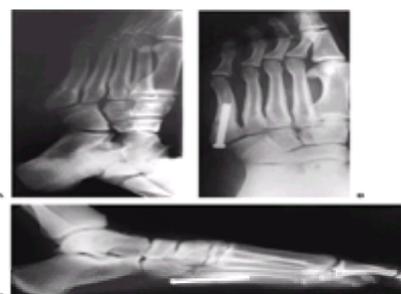


**FIGURE 26-39.** **A:** Nondisplaced incomplete fracture of the base of the fifth metatarsal in a boy 12 years and 10 months of age. **B:** Five weeks later, there is further resorption of the fracture line, but the fracture site is nontender and immobilization was discontinued. Radiographic union may be delayed for weeks to months.

Fractures of the base of the fifth metatarsal have been the subject of much more attention in the past two decades. Several classification systems have been presented to assist in differentiating between acute fractures and stress fractures, in addition to localizing the fracture to the metaphysis, diaphysis, or metaphyseal–diaphyseal junction (92). The literature concerning fractures of the base of the fifth metatarsal can be confusing, and much of it relates only to adult patients.

The main area of contention is the so-called Jones fracture: as with many eponyms, this term has been applied differently by different researchers. To summarize recent reports, a fracture of the diaphyseal–metaphyseal junction should arouse suspicion that a stress reaction may have predated the fracture ( 92,141,157,158). If such is the case, healing will be retarded, especially in athletes, and bone grafting or internal fixation usually is indicated.

Kavanaugh, Brower, and Mann (85) emphasized that the Jones fracture is not an avulsion fracture; rather, it appears to result from vertical or mediolateral ground forces on the weight-bearing foot. Because the foot was weight bearing, inversion was impossible, and these researchers concluded that inversion was not at fault. The strong ligamentous and capsular attachments firmly bind the base of the fifth metatarsal to the adjacent cuboid and fourth metatarsal, subjecting the proximal shaft to excess strain when loaded as described above. This injury is most common in patients 15 to 21 years of age and is most often seen in athletes. Conservative treatment with a non-weight-bearing cast was described as ineffective by Kavanaugh et al. ( 85), but effective by Torg et al. ( 167) and Josefsson et al. ( 82), who speculated that some of the injuries treated by Kavanaugh et al. might not have been acute, and nonacute injuries are less likely to heal with immobilization. With non-weight-bearing immobilization, 14 of the 15 acute fractures treated by Torg et al. ( 167) healed. DeLee, Evans, and Julian ( 42) reported on 10 young adult athletes they treated with immediate intramedullary screw fixation, with healing in all patients. Mindrebo et al. ( 113) and Portland et al. ( 130) demonstrated that the time to union was shorter after operative treatment, with earlier return to weight bearing and full activities. The average time to healing was 6 to 7 weeks compared with 2 to 3 months in series with nonoperative treatment. Acute surgical management can be considered as a treatment option, particularly in high-level athletes who desire to return promptly to their sporting activities ( Fig. 26-40).



**FIGURE 26-40.** A 15-year-old high-level high school basketball player sustained a proximal fifth metatarsal fracture at the metaphyseal–diaphyseal junction. The patient opted for intramedullary screw fixation with desire to return to sport as promptly as possible, lessen his time in immobilization, and lessen the risk of the possibility of delayed versus nonunion. **A:** Injury film. **B and C:** Following intramedullary screw fixation. (Courtesy of Keith S. Hechtman, M.D.)

A Jones fracture must be differentiated from a fracture of the tuberosity, because the two differ considerably in prognosis and management. Smith et al. ( [157,158](#)) found that the blood supply of the proximal diaphysis is much sparser than that of the tuberosity, which probably is a factor in the slower healing evident in proximal diaphyseal fractures.

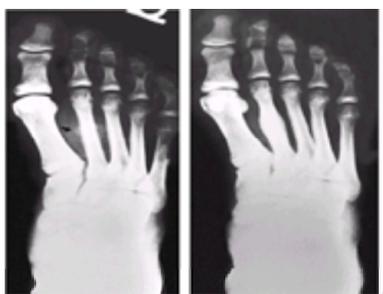
The results obtained by Torg et al. ( [167](#)) with non-weight-bearing immobilization of acute fractures support this method of treatment for Jones fractures. They emphasized that to be classified as an acute injury, there must be no previous history of pain or trauma, there must be a sharp, well-differentiated fracture line, and most importantly, there must be no intramedullary sclerosis. With repetitive symptoms or intramedullary sclerosis, the prognosis for healing with immobilization is dismal.

## OPERATIVE TREATMENT

Guidelines for operative treatment of Jones fractures are not well established. Most of these injuries occur in athletes, and in this group, regardless of age, there is a trend toward immediate intramedullary screw fixation of acute fractures, as well as for delayed unions and nonunions (without bone grafting). An inlay bone grafting technique has been described by Torg et al. ( [167](#)) with the proximal tibia as the donor site. This area is unsuitable as a donor site in skeletally immature children; if bone grafting is elected, the iliac crest is preferable. Intramedullary fixation avoids this problem, although there are technical traps. The surgical technique described by Lehman et al. ( [93](#)) is recommended when this method is selected.

## STRESS FRACTURES

Stress fractures have been reported in children in most of the bones of the foot. Yngve ( [189](#)) reviewed reports of stress fractures in children and found 94 fractures in which the involved bone was identified. However, if the number of reported cases indicates the actual incidence, these injuries are fewer in children than in adults. The metatarsals were involved in 14% of stress fractures in adults, the tarsals in 13%; in children, metatarsals and tarsals were each involved in only 2%. This could be a result of underreporting in children ( [Fig. 26-41](#)).



**FIGURE 26-41.** Painful second metatarsal of about 2 weeks' duration in a 16-year-old boy. **A:** A subtle fracture line can be seen underlying a callus beginning to ossify. **B:** Residual of callus 3 months later. (Courtesy of Teresa M. Stacy, M.D.)

The most common sites of stress fractures in children are the tibia and fibula, but 4 of the 35 stress fractures in children reported by Devas ( [43](#)) occurred in the metatarsals. Childress ( [30](#)) reported a typical stress fracture of the metatarsal in a 7-year-old. Symptoms are pain on weight bearing, but with much less discomfort at rest than with a fracture resulting from trauma. Swelling is minimal or nonexistent, but localized tenderness is marked. With most stress fractures, awareness of the possibility and an index of suspicion aid greatly in diagnosis. Bone scans can be fallible in excluding the diagnosis ( [Fig. 26-19](#)). Stress fractures seem to be particularly common in adults and children who undertake a sudden increase in physical activity. The pediatric population at risk are skeletally maturing adolescents starting to participate in intensive training for sports.

### Mechanism of Injury

The mechanism of injury appears to be reabsorption and demineralization at the site of stress, followed by a reparative process evidenced by periosteal layering of new bone. The relationship of stress fractures to sudden increases in activity is well documented and is supported experimentally. Rubin and Lanyon ( [139](#)) used rooster ulnae to demonstrate that bone mineral does not increase until after the second week of increased loading. This correlates with the appearance of symptoms resulting from stress fractures at this time during a training regimen ( [151](#)). In a study of Israeli army recruits, modifying the activity level during the third week resulted in a decrease in the stress fracture rate from 4.8% to 1.6% ( [140](#)).

A short first metatarsal (Morton's toe) is cited in many publications as a factor in the production of stress fractures in runners commonly located in the second and third metatarsals. However, Drez et al. ( [46](#)) could not document any difference in the relative lengths of the first and second metatarsals between patients with stress fractures and a control population. The factors responsible for the development of a stress fracture have not been precisely elucidated, but it does not appear that a short first metatarsal is one of them. Hormonal factors may play a role. An increased incidence of stress fractures was reported in ballet dancers 18 to 36 years of age, who were also older than average at the time of menarche ( [176](#)).

### Radiographic Findings

If the patient presents early, radiographs are normal. Anteroposterior, lateral, and oblique radiographs should be taken, but the lateral view usually is the least helpful, because the metatarsals are stacked one upon the other. Periosteal callus formation about 2 weeks after the process commences establishes the diagnosis ( [Fig. 26-41](#)). Bone scanning has been advocated for making the diagnosis if conventional radiographs demonstrate no abnormality ( [144](#)), but I prefer a 2-week trial of immobilization in a short-leg walking cast if the history, symptoms, and signs indicate a stress fracture. When the cast is removed, radiographs usually reveal periosteal callus. Scanning might be useful at this point if the diagnosis is still in question, but this would be quite unusual.

## AUTHOR'S PREFERRED METHOD OF TREATMENT

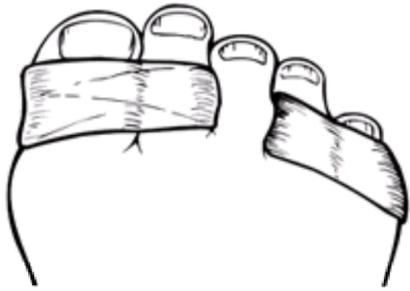
Patients with stress fractures differ from patients with traumatic injuries in that there is a definite temporal starting point with an acute fracture, and the period required for immobilization can be calculated from that point. There is no comparable starting point for stress fractures, although the onset of symptoms is helpful. The patient may be nearly healed the first time he or she presents for evaluation; in this case, immobilization is unnecessary. I try to adapt the treatment to the symptoms. If the foot is painful only during sports, restricting such activities until pain allows is all I recommend. If walking is painful, a 2-week period of immobilization in a short-leg cast is recommended, after which time it is discontinued if the tenderness has subsided and walking is painless. Bernstein and Stone ( [14](#)) reported that pain associated with stress fractures of the metatarsals was relieved by eliminating metatarsophalangeal motion, and this could be accomplished by the use of a metatarsal bar countersunk into the sole of the shoe. Especially with the casual shoes worn by today's youth, I prefer to apply a cast if the symptoms warrant immobilization. If there is only a clinical suspicion with negative radiographs, the same approach is used. Repeat radiographs in 2 weeks usually reveal an area of stress reaction, combined with less tenderness on clinical examination.

## FRACTURES OF THE PHALANGES

Fractures of the phalanges of the foot in children are uncommon and usually are caused by direct trauma from a falling object or kicking a structure less yielding than the toe. Radiographs can reveal the fracture site, but management is based entirely on clinical assessment. Reduction usually is unnecessary, although more care would be taken with the proximal phalanx of the great toe as the child matures. Rarely in an older child, reduction of a fracture of the proximal phalanx of the great toe

may be difficult to maintain with external immobilization, and percutaneous Kirschner wire fixation or open reduction is then indicated.

In most patients, however, the adjacent noninjured toes serve as splints and are simply taped to the injured toe, with gauze placed between the toes to prevent maceration (Fig. 26-42). Care should be taken to prevent rotational malunion; the nail bed of the injured toe should be in the same plane as the others. Healing usually is complete in 3 weeks, and immobilization generally is discontinued at this time if the fracture site is not tender. If the child is engaged in a running or kicking sport, an additional 2 to 3 weeks of prophylactic immobilization probably is worthwhile. Open reduction and internal fixation is indicated for fractures with displacement of more than 2 to 3 mm.



**FIGURE 26-42.** Method of taping to adjacent toe(s) for fractures or dislocations of the phalanges. Gauze is placed between the toes to prevent maceration. The nail beds are exposed to ascertain that malrotation of the injured toe is not present. (Reprinted from Weber BG, Brunner C, Freuler F. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980:392; with permission.)

### Dislocations of the Metatarsophalangeal or Interphalangeal Joint

Dislocations of the metatarsophalangeal or interphalangeal joint also are unusual in children, and again usually result from kicking. The toe is forced into hyperextension, the collateral ligaments rupture, and the proximal or middle phalanx comes to rest on the dorsal surface of the metatarsal head (or proximal phalanx; Fig. 26-43). In general, traction and manipulative reduction are effective. The toe is taped to the adjacent toes for 3 weeks to allow capsular healing, after which time full activity is resumed.



**FIGURE 26-43.** Dislocation of the proximal interphalangeal joint of the fourth toe in a 13-year-old boy. Reduction was easily accomplished with traction and manipulation of the dorsally displaced middle phalanx.

### OPEN FRACTURES OF THE FOOT

The management of open fractures of the foot is no different from that of any other anatomic site. Parenteral antibiotics are instituted before the patient is taken to the operating room for debridement. A broad-spectrum antibiotic is chosen, depending on the patient's allergies and the surgeon's experience. I prefer a cephalosporin for 48 hours, repeated with further surgical treatment. Tetanus booster is given if 5 years has elapsed since the initial series of immunizations or the last booster.

However, the choice of antibiotic or the method of immobilization is less important than the quality of the initial debridement. All contaminated tissue is meticulously removed; optical magnification can be helpful. Wound cultures are obtained. Despite the functional importance of a particular structure (tendon, bone, physis, or articular cartilage), if it is contaminated or devitalized, it must be debrided. If it is allowed to remain, bacteria or spores may be harbored in devitalized tissue, which by definition is not accessible to parenteral antibiotics. Debriding such tissue is the first step toward avoiding chronic osteomyelitis: the time and energy devoted to avoiding such a situation pays long-term dividends to the child.

The wound is dressed open, and the patient is returned to the operating room 2 to 5 days after the injury for inspection of the wound. If it is clean, delayed primary closure or another form of wound coverage can be performed at this time. If tissue of questionable viability remains, further serial debridements are performed until a clean wound is attained.

There is considerable latitude as to the optimal time for the application of internal fixation (when necessary). If the foot is so unstable that the neurovascular bundles are in jeopardy of twisting, kinking, or traction, internal fixation is indicated at the time of initial debridement after a thorough examination for any questionable nonviable tissue. Fractures crossing the physis can be most accurately approximated at the time of initial debridement, so this is a relative indication for primary internal fixation. However, unless I can be satisfied that the bone involved is clean, I still prefer to wait until the time of delayed closure. I would prefer to have the problem of malunion or even possible early physeal closure rather than chronic osteomyelitis involving this region, which would not only destroy bone but would also undoubtedly require a more destructive procedure at a later date to eradicate the infection.

### LAWN MOWER INJURIES AND OTHER CRUSH INJURIES

The most destructive injuries to the child's foot in northern latitudes result from being crushed by an automobile tire, a power lawn mower, or a snowmobile ( 129). These injuries require considerable judgment on the part of the surgeon, because the extent of tissue damage may be difficult to assess at the time of injury. Although more rigid manufacturing standards have resulted in more safeguards against injury, foot injuries sustained from power mowers are common during the spring and summer: over 10,000 injuries due to lawn mower accidents are reported annually. According to statistics from the Consumer Product Safety Commission, children under 14 years of age (along with older adults) are most susceptible to injury, and children under 6 have the greatest risk of death ( 122). Of the 18 children reported by Ross et al. ( 138), 11 were riding with a parent on the mower at the time of injury. Love, Grogan, and Ogden ( 99) reported 27 children injured by power lawn mowers, with acute amputations necessary in 19. Thirteen of these 19 amputations were through the mid-foot or toes. Fifteen of the 18 children reported by Ross et al. ( 138) had residual deformities. Older children are more often injured when using rotary lawn mowers, with the foot being the anatomic part most vulnerable to injury. In either case, the spinning blades carry tremendous destructive forces, cutting through soft tissue and bone alike ( 127). The kinetic energy generated from the blade at a typical speed of 3,000 rpm has been reported as 2,100 ft/lb. This amount of energy has been stated as the comparable to "dropping a 211 pound object from a height of 100 feet or three times the power of a .357 magnum gun" ( 5). Graham et al. ( 58) found that most rotary injuries involved the toes or foot, with avulsion or degloving of the injured part.

Fragments of dirt, debris, and grass are blown into the wound under pressure, and debridement of this type of wound requires especially meticulous and thorough

cleansing. The force with which the contaminants are blown into the tissue renders jet water lavage ineffective in removing debris, and it must be removed mechanically, bit by bit. Wound cultures from these injuries usually are polymicrobial. A mean of three organisms per intraoperative culture were identified when anaerobic and aerobic cultures were performed (7).

### Treatment

Considerable judgment is required to determine whether to amputate or save a questionable part. My approach is to perform a thorough debridement at the time of injury, preserving all tissue of questionable viability and leaving the wound open. The ability of children to maintain viability in injured fingertips is well recognized, and a very conservative approach at the time of the initial debridement is justified. The wound is examined with the child under anesthesia at intervals of 2 to 3 days until it is clean and ready for definitive coverage. Plastic surgery consultation is advisable at the time of initial injury to allow optimal preparation for a definitive procedure to provide adequate skin coverage (144).

Experience with the management of lawn mower injuries unfortunately is being compiled at a steady rate. Alonso and Sanchez emphasized the severity of these injuries secondary to the massive forces incurred and the need for triple antibiotic coverage (cephalosporin, aminoglycosides, penicillin), because they are in the same class as farm injuries (5). Formulas for determining the feasibility of salvaging the foot in adults are less meaningful for children because of the effects of subsequent growth. Split-thickness skin grafts can function surprisingly well on weight-bearing surfaces in children (45,173). Dormans et al. (45) found shredding types of injury tend to do poorly even if the limb is salvaged; paucilaceration types of injuries have a much better outlook. Amputation rates of about 70% are common in series of children with lawn mower injuries (173).

It is important for the surgeon to appraise the degree of injury to the foot realistically and not pursue limb salvage at any cost. There seldom is any need for an immediate decision, and the degree of tissue damage noted at the time of subsequent debridements often makes the best decision obvious. With the advent of stump-lengthening procedures, better function is possible with a short amputation than in the past. Available tendons can be transplanted to produce a functional mid-foot amputation, which has often been regarded as an unattractive option.

### Soft Tissue Treatment

Probably the most dramatic recent changes have been in the choice and timing of skin coverage. In the past, split-thickness skin grafts often were used, because the alternative of a pedicle flap was associated with considerable morbidity. However, in the past decade, several innovative free flaps have made the cumbersome cross-leg pedicle flap largely obsolete (11,25,66,67,192). The choices for coverage are numerous. Muscle flaps can be used for an improved biologic environment when the wound is not optimal (144). Cutaneous, fasciocutaneous, or composite flaps may be useful (Fig. 26-44). Local flaps are ideal when feasible but often are not large enough. Free flaps are described from various sites, including the forearm, scapula, upper arm, and scalp. The choice depends on the site and size of the wound, coupled with the expertise and experience of the consulting plastic surgeon. Advances in bone transport with ring or other external fixators allow more potential for reconstructing a severely injured child's foot.



**FIGURE 26-44.** A boy 7 years and 6 months of age whose foot was run over by a car. **A:** Wound on dorsum of the foot on the day of injury. **B:** Appearance of the foot following primary thigh fasciocutaneous flap applied on the day of injury. Although the foot did not sustain bony injury, the child subsequently developed premature growth arrest of the distal fibular physis. (Courtesy of W. Gould, M.D., and C. Crawford, M.D.)

The closure of large wounds by secondary intention, although often considered an outdated concept, may still be an attractive option for the treatment of wounds that do not involve weight-bearing surfaces (Fig. 26-45).

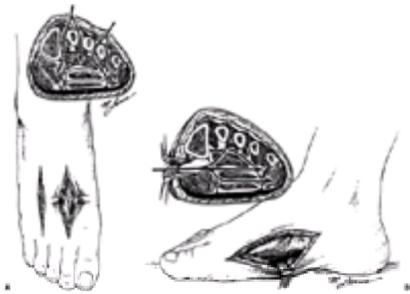


**FIGURE 26-45.** A boy 9 years and 8 months of age, whose foot was run over by a car. He sustained necrosis of the dorsal skin, which healed by secondary intention under a weight-bearing cast. **A:** After necrosis of dorsal skin. **B:** Three weeks later. **C:** Four months later.

Modern microvascular techniques make it possible to salvage some partial foot amputations (27), although the prevalence of untidy and avulsive wounds involving the foot make successful reimplantation unlikely. If there is any question about such a possibility, the amputated portion should be thoroughly cleansed, placed in a sterile plastic bag, and immersed in ice. The proximal stump should be cleansed and the patient should be referred to a reimplantation team as quickly as possible.

### Compartment Syndrome

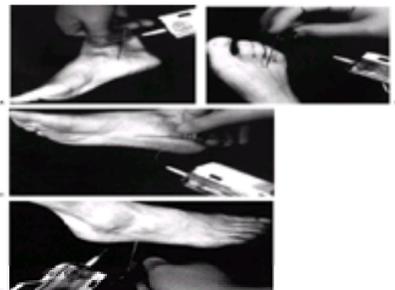
Crush injuries bring with them several difficult decisions for the attending orthopaedist. Compartment syndromes have been described in the foot (19,71,72,105,114,117,154,155,178). Ziv et al. (191) found elevated compartment pressures in the foot accompanying open fractures. Closed crush injuries, already mentioned in the section on metatarsal fractures, can demand rapid action. The number of publications on compartment syndromes of the foot since the last edition of this volume continues to expand, although the patients studied are mostly adults. Silas et al. (155) reported seven children treated for compartment syndrome of the foot. All but one had crush injuries, all had swelling and pain with passive motion, but none had any neurovascular deficit. Two had no bony injury, and in none of the others was the bony injury dramatic. Compartment pressures were measured under general anesthesia. A dorsal or a medial approach to the compartments is satisfactory for decompression; if a hindfoot injury is present, a medial approach is recommended to allow decompression of the tarsal tunnel (Fig. 26-46).



**FIGURE 26-46.** Surgical approaches for fasciotomy of the foot. **A:** The dorsal approach is via two incisions over the second and fourth metatarsal shafts and is more suitable for injuries of the forefoot or mid-foot. **B:** The medial approach is more suitable for injuries of the hindfoot, with the incision extending from the base of the first metatarsal to the medial malleolus. A tarsal tunnel release may be performed through this incision. (Reprinted from Myerson MS. Experimental decompression of the fascial compartments of the foot—the basis for fasciotomy in acute compartment syndromes. *Foot Ankle* 1988;8:308–314; with permission.)

Compartment syndrome should be suspected with crush injuries. Although uncommon in children, if the diagnosis of compartment syndrome is missed, the late sequelae can lead to significant disability in a young patient. Problems associated with missed compartment syndrome of the foot include claw toe deformities, paresthesias, cavus deformity, stiffness, and residual pain.

Often the diagnosis is indicated clinically by severe pain in association with tense swelling of the soft tissues or intense pain on passive stretching of the toes. The diagnosis can be made objectively with the measurement of compartment pressures ([Fig. 26-47](#)). There is no consensus in the literature regarding the threshold pressure level that mandates fasciotomy. In general, pressure readings of more than 40 mm Hg require surgical release of the compartments ([118](#)). Pressure readings of 30 to 40 mm Hg should prompt either fasciotomy or close monitoring. Less than 30 mm Hg can be observed clinically with strict ice and elevation. New devices such as foot pumps aimed at decreasing compartment pressures by actively eliminating edema have recently been advocated ([118](#)).



**FIGURE 26-47.** Compartment pressure measurement technique. **A:** The medial and calcaneal compartments are measured with the needle introduced within the medial hindfoot several centimeters below the medial malleolus. **B:** Forefoot pressures measured with the needle in the first or second interspace measuring the respective interosseous space pressure, advancement of the needle deeper measures the adductor compartment pressure. **C:** Superficial compartment is measured in the central plantar surface of the foot. **D:** Lateral compartment measurement is just below the base of the fifth metatarsal. (Reprinted from Manoli A II, Fakhouri AJ, Weber TG. Compartmental catheterization and fasciotomy of the foot. *Operative Techniques Orthop* 1992;2:205; with permission.)

## LACERATIONS OF THE FOOT

The common problem of managing lacerations of the foot has received little attention in the literature. In 1977, Yancey ([188](#)) sent a questionnaire to the members of the American Orthopaedic Foot Society and the Association of Bone and Joint Surgeons, asking for methods of dealing with deep lacerations on the plantar aspect of the foot. He also asked for any reference in this area of which the respondent might be aware. None was cited. There was no uniformity of opinion about the management of lacerated tendons and nerves, although most surgeons would attempt repair.

Wicks et al. ([183](#)), in a report on tendon injuries about the foot and ankle in children, found that injuries to the heel cord or the anterior tibial or posterior tibial tendons resulted in severe foot deformity when not promptly recognized and repaired. They did not advocate repair of the extensor hallucis longus tendon, because no impairment was noted when this tendon was left unrepaired. Conversely, two patients had stiffness of the metatarsophalangeal joint after attempted repair. They thought that walking would passively maintain extension, and that there was no indication for repair of the long flexors or extensors to the toes. I would modify this recommendation slightly: I have seen a cock-up deformity of the second toe in a child who had an unrepaired laceration of the flexor tendon. If the tendons are not repaired, immobilization of the foot and toes by taping to the adjacent toes should be imposed for 3 weeks to allow restoration of continuity of the lacerated tendon. Lacerations to the nerves in the distal part of the foot in children appear to do very well, even when not repaired.

## PUNCTURE WOUNDS

Since the 1960s, several reports ([17,52,53,58,59,77,79,91,120](#)) have documented the development of locally destructive osteomyelitis of the foot, usually near the metatarsophalangeal joints, after puncture wounds of the foot. Systemic symptoms, findings, and abnormal laboratory values do not accompany this entity.

Ninety-eight percent of all puncture wounds result from stepping on a nail ([172](#)). The incidence of cellulitis after initially treated puncture wounds of the foot is estimated at 8% to 15%; osteomyelitis develops in 0.6% to 1.8% ([172](#)). The prevalence of *Pseudomonas* as the offending organism in osteomyelitis appears to be due at least in part to the incidence of this organism in the soles of tennis shoes ([52](#)). However, the organism also has been found in work boots, and it appears that a warm, moist environment is the key factor; most footwear can provide that ([58](#)). The penetrating nail apparently acquires this organism as a rider on its passage into the sole of the foot.

No scientific basis for the initial management of puncture wounds is available, but the recommendations of Verdile, Freed, and Gerard seem reasonable ([172](#)). They use high-pressure wound irrigation with a 19-gauge needle, avoid hexachlorophene soaps, and perform debridement with a posterior tibial nerve block for especially dirty or deep wounds. Tetanus immunization is given when appropriate. They thought there was no basis for the use of routine prophylactic antibiotics, and that routine administration might even enhance the chances of subsequently developing gram-negative infection. Cellulitis generally is secondary to *Staphylococcus aureus* and is managed with wound culture, debridement, and appropriate antibiotic therapy.

The orthopaedist usually is consulted when there is a question of osteomyelitis. This entity is now well known among primary-care physicians and pediatricians, and delays in referral are less common ([76](#)). The instigating factor is a contaminating puncture wound, usually involving the cartilaginous metatarsal head or the proximal phalanx. Initially, the wound shows little evidence of infection because of the paucity of the inflammatory reaction (due to the relative avascularity of cartilage). Typically, after 5 to 10 days, the local reaction becomes more apparent. According to Green and Bruno ([60](#)), residual deformity depends on delay in diagnosis and treatment. Wounds managed by early surgical debridement and decompression of the joint, if involved, healed with minimal complications. If there was a 10- to 14-day delay in treatment, there sometimes was residual deformity. A 3-week delay in treatment essentially ensured residual deformity.

## Treatment

The symptoms and pain involved with a puncture wound of the foot should subside in 2 or 3 days. If pain persists after this time, surgical exploration is indicated. Bone scintigraphy or MRI has been used for evaluation of possible osteochondritis secondary to puncture wounds and may be helpful, but if the physical findings are obvious for a deep inflammatory process, there is no need to delay surgery to obtain bone scintigraphy. If there is delay until the need for exploration is obvious, permanent deformity may be inevitable.

The choice of antibiotic and the necessary duration of therapy have not been established. One of the first patients with this problem, reported by Johanson (79) was treated with only surgical debridement. The aminoglycosides, the previous antibiotics of choice, are now recommended only for life-threatening *Pseudomonas* infections (121). Many of the newer beta-lactam antibiotics have variable degrees of activity against *Pseudomonas*; of these, ceftazidime appears to be the most effective, both *in vitro* and *in vivo*. A course of 10 to 14 days of antibiotic therapy after debridement has been recommended, although the authors of this recommendation acknowledge that shorter periods may suffice (76). Jacobs et al. (76) recommended aggressive surgical debridement and a 7-day course of intravenous antibiotics; even shorter durations are sufficient if debridement is complete. I administer parenteral antibiotics for 5 to 7 days after debridement if a good clinical response is attained, but like others I have no scientific basis for recommending this duration.

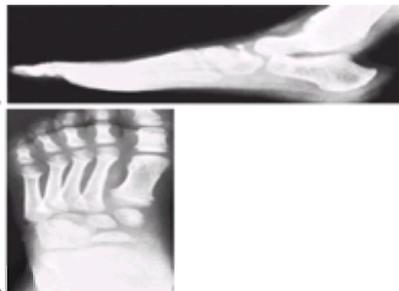
Two retrospective reviews of children with documented culture-positive *Pseudomonas* infections of the foot secondary to puncture wounds (77,120) emphasized the relative paucity of clinical signs of infection. Mild erythema and moderate tenderness at the puncture site were universal findings; however, relatively minimal inflammation and warmth were noted. In these patients, laboratory studies were often not suggestive of infection, and a high clinical suspicion was imperative. Adequate surgical debridement combined with anti-pseudomonal antibiotic coverage was stressed if rapid clinical improvement was not seen. In both of these studies the mean duration of intravenous antibiotic treatment approached 3 weeks.

## LESIONS THAT MAY RESEMBLE FRACTURE

More confusing anatomic variants exist in the foot than anywhere else in the body. The more common variants are described, but it is impractical to attempt to be all inclusive and even more impractical for the clinician to try to learn all the variants.

### Köhler's Disease

Waugh (177) studied the ossification and vascularization of the tarsal navicular bone and its relationship to Köhler's disease. He obtained radiographs of the feet of 52 boys and 52 girls at 6-month intervals from 2 to 5 years of age. Of these, 16 boys and 10 girls displayed abnormal ossification. Karp (84) found an even greater sex difference, with boys predominating by a ratio of 6.5 to 1. All the children in Waugh's series eventually developed a normal navicular bone due to the radial penetration of the arterial supply from the periphery of the ossifying bony nucleus. There was also no anatomic barrier (such as the physis in Legg-Perthes syndrome) to the ingrowing vascular supply, so revascularization was rapid and complete. Fragmentation of the ossification center of the navicular bone is a normal variant; irregularities and ossification at this site have no consistent relationship to symptoms (Fig. 26-48).



**FIGURE 26-48.** Köhler's disease in a 7-year-old boy. **A:** Collapse and condensation. **B:** Another boy with irregularity of ossification of medial portions of the navicular and medial cuneiform bones, a normal variant. Close inspection will reveal a fracture of the talar neck, shown in more detail in Fig. 26-6.

### Freiberg's Infraction

Freiberg's infraction, an osteochondrosis of the second metatarsal head, can be confused with fracture—in fact, of Freiberg's (43) six original cases, three had a history of trauma, and subsequent reports by Smillie and Braddock (17,156) supported a traumatic etiology. Braddock (17) experimentally reproduced a comminuted fracture of the second metatarsal physis in anatomic specimens by loading the plantarflexed foot in skeletally maturing bone. A similar injury force produced fractures of the phalanges in younger children or adults, and he postulated that the physis was especially vulnerable at a certain stage of physeal maturation. This correlates with the age distribution of Freiberg's infarction, which is most common in the second decade of life. The second metatarsal also is the one most often injured, as it is the most rigidly fixed metatarsal. Wiley (184) discovered that one of his patients being followed after a tarsometatarsal dislocation developed Freiberg's infarction.

Pain and localized tenderness at the second metatarsal head are characteristic of Freiberg's infarction. On radiographs, there is a dense sequestrum in the metatarsal head when the process is active, with collapse and shortening of the residual effects (Fig. 26-49).



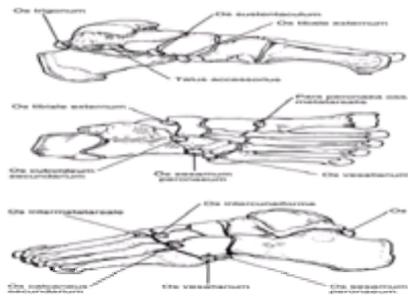
**FIGURE 26-49.** **A:** Freiberg's infarction of the second metatarsal head (arrow) in a 15.2-year-old girl. The metatarsal head is flattened and shortened. **B:** After curettage and bone grafting, with the base of the second metatarsal serving as a donor site. Symptoms were completely relieved.

Smillie (156) recommended immobilization as early treatment, with elevation of the depressed metatarsal head and bone grafting for a deforming head. He reported good results unless the fragment had completely separated. Wiley also had a favorable experience with this approach (J.J. Wiley, personal communication; Fig. 26-49), and my limited experience has also been favorable. Of course, many other procedures also have been recommended (73,88).

### Other Variants

It usually is not difficult to differentiate a fracture from an anatomic variant with physical examination after trauma and the determination of localized tenderness; usually, the questionable radiographic finding is elsewhere. If not, the gentle, rounded edges of anatomic variants generally pronounce their long-standing presence,

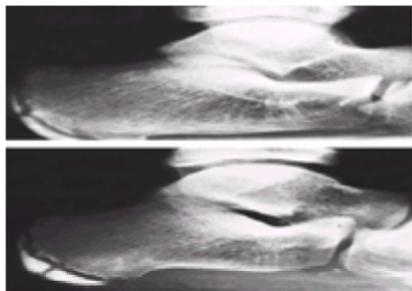
in contrast to the sharp, jagged edges of a recent fracture ( [Fig. 26-50](#)).



**FIGURE 26-50.** The supernumerary tarsal bones. **Top:** Medial view. **Center:** Plantar view. **Bottom:** Lateral view. (Redrawn from Schaeffer JP. *Morris' human anatomy*, 11th ed. New York: Blakiston Division, McGraw-Hill, 1953:279–280.)

The multiple accessory bones of the foot can be a source of confusion. Many ossification centers in the foot normally assume a peculiar configuration—for instance, the ossification center of the proximal phalanx of the first toe is often bipartite. Keats ( [86](#)) has beautifully illustrated all the common and many of the uncommon potentially confusing radiographic variants that may be encountered in the foot.

The normal calcaneal apophysis can assume various shapes and densities; some of them in the past were designated as Sever's disease. The existence of such an entity is questionable ( [Fig. 26-51](#)). A stress reaction at the physis, not distinguishable on routine radiographs, could be the source of pain. This would be analogous to Osgood-Schlatter disease at the tibial tubercle, where considerable force is generated by the quadriceps insertion on a relatively small maturing apophysis (recognizing that the radiographic picture of Osgood-Schlatter disease usually is unrelated to the clinical status). “Sever's disease” may be a comparable situation at the site of insertion of the Achilles tendon. Symptoms would then be present accompanying a variety of radiographic configurations of the calcaneal apophysis.



**FIGURE 26-51.** Two normal variants in configuration of the calcaneal apophysis. (Courtesy of Teresa M. Stacy, M.D.)

The clinician must attempt to correlate the symptoms, physical findings, and radiographic findings with his or her knowledge of anatomy and potential patterns of pathophysiology in the affected area. Solving a clinical puzzle often involves a higher level of analysis than merely trying to decide whether radiographic shadows are pathologic or normal.

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## CHAPTER REFERENCES

- Adelaar RS. Complex fractures of the talus. *Instr Course Lect* 1997;46:323–338.
- Aitken JT, Causey G, Joseph J, et al. *A manual of human anatomy*, 2nd ed. Edinburgh, UK: E & S Livingstone, 1966.
- Alexander AH, Lichtman DM. Surgical treatment of transchondral dome fractures (osteochondritis dissecans), long-term follow-up. *J Bone Joint Surg [Am]* 1980;62:646–652.
- Allon SM, Mears DC. Three-dimensional analysis of calcaneal fractures. *Foot Ankle* 1991;11:254–263.
- Alonso JE, Sanchez FL. Lawn mower injuries in children: a preventable impairment. *J Pediatr Orthop* 1995;15:83–89.
- Anderson IF, Crichton KJ, Grattan-Smith T, et al. Osteochondral fractures of the dome of the talus. *J Bone Joint Surg [Am]* 1989;71:1143–1152.
- Anger DM, Ledbetter BR, Stasikelis PJ, et al. Injuries to the foot related to the use of lawn mowers. *J Bone Joint Surg [Am]* 1995;77:719–725.
- Angermann P, Jensen P. Osteochondritis dissecans of the talus: long-term results of surgical treatment. *Foot Ankle* 1989;10:161–163.
- Bachman S, Johnson SR. Torsion of the foot causing fracture of the anterior calcaneal process. *Acta Chir Scand* 1953;105:460–466.
- Baker CL, Andrews JR, Ryan JB. Arthroscopic treatment of transchondral talar dome fractures. *Arthroscopy* 1986;2:82–87.
- Barwick WJ, Goodkind DJ, Serafin D. The free scapular flap. *Plast Reconstr Surg* 1982;69:779–785.
- Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg [Am]* 1959;41:988–1020.
- Bernstein A, Stone JR. Manual fracture: a report of 307 cases and a new method of treatment. *J Bone Joint Surg* 1994;26:743.
- Berquist TH. *Radiology of the foot and ankle*. New York: Raven, 1989.
- Biert J, Boll APM, Schoots FJ, et al. Bilateral calcaneal fractures in a child treated by percutaneous reduction and screw fixation. *J Trauma* 1998;44:1098–1100.
- Blevins FT, Steadman JR, Rodrigo JJ, et al. Coned treatment of articular cartilage defects in athletes: an analysis of functional outcome and lesion appearance. *Orthopedics* 1998;21:761.
- Braddock GTF. Experimental epiphyseal injury and Freiberg's disease. *J Bone Joint Surg [Br]* 1959;41:154–159.
- Bruns J, Rosenbach B. Osteochondritis dissecans of the talus. Comparison of results of surgical treatment in adolescents and adults. *Arch Orthop Trauma Surg* 1992;112:23–27.
- Bonutti PM, Bell GR. Compartment syndrome of the foot. A case report. *J Bone Joint Surg [Am]* 1986;68:1449–1451.
- Bryant DD, Siegel MG. Osteochondritis dissecans of the talus: a new technique for arthroscopic drilling. *Arthroscopy* 1993;9:238–241.
- Buckwalter JA, Rosenberg LC, Hunziker EB. Articular cartilage: composition, response to injury and methods of facilitating repair. In: Ewing JW, ed. *Articular cartilage and knee joint function: basic science and arthroscopy*. New York: Raven, 1990:19.
- Canale ST, Belding RH. Osteochondral lesions of the talus. *J Bone Joint Surg [Am]* 1980;62:97–102.
- Canale ST, Kelly FB Jr. Fractures of the neck of the talus. Long-term evaluation of 71 cases. *J Bone Joint Surg [Am]* 1978;60:143–156.
- Cehner J. Fractures of the tarsal bones, metatarsals, and toes. In: Weber BG, Brunner C, Freuler F, eds. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980.
- Chang KN, De Armond SJ, Buncke HJ. Sensory reinnervation in microsurgical reconstruction of the heel. *Plast Reconstr Surg* 1986;78:652–664.
- Chapman HG, Galway HR. Os calcis fractures in childhood. *J Bone Joint Surg [Br]* 1977;59:510.
- Chavoin JP, Dupin B, Gordow JF, et al. La microchirurgie, II. Reimplantation d'un pied apres amputation traumatique. *Rev Med Toulouse* 1978;14:31–36.
- Cheng MS, Ferkel RD, Applegate GR. Osteochondral lesions of the talus: a radiographic and surgical comparison. Presented at the annual meeting of the American Academy of Orthopedic Surgeons, New Orleans, February 1995.
- Cole RJ, Brown HP, Stein RE, Pearcey RG. Avulsion fracture of the tuberosity of the calcaneus in children. *J Bone Joint Surg [Am]* 1995;77:1568–1571.
- Childress HM. March fracture in a seven year old boy. *J Bone Joint Surg* 1946;28:877.
- Coltart WD. Aviator's astragalus. *J Bone Joint Surg [Br]* 1952;34:545–566.
- Crosby LA, Fitzgibbons T. Computerized tomography scanning of acute intra-articular fractures of the calcaneus. A new classification system. *J Bone Joint Surg [Am]* 1990;72:852–859.
- Curtis MJ, Myerson M, Szura B. Tarsometatarsal joint injuries in the athlete. *Am J Sports Med* 1993;21:497–502.
- de Beer JD, Maloon S, Hudson DA. Calcaneal fractures in children. *S Afr Med J* 1989;76:53–54.

35. Dameron TB. Fractures and anatomical variations of the proximal portion of the fifth metatarsal. *J Bone Joint Surg [Am]* 1975;57:788–792.
36. Dameron TB. Fractures of the proximal fifth metatarsal: selecting the best treatment option. *JAAOS* 1995;3:110–114.
37. Daniels TR, Smith JW. Talar neck fractures. *Foot Ankle* 1993;14:225–234.
38. Daniels TR, Smith JW, Ross TI. Varus malalignment of the talar neck: its effect on the position of the foot and on subtalar motion. *J Bone Joint Surg [Am]* 1996;78:1559–1967.
39. Degan TJ, Morrey BF, Braun DP. Surgical excision for anterior process fractures of the talus. *J Bone Joint Surg [Am]* 1982;64:519–524.
40. DeLee JC. Fractures and dislocations of the foot. In: Mann RA, Coughlin MJ, eds. *Surgery of the foot and ankle*. St. Louis: CV Mosby, 1993.
41. DeLee JC, Curtis R. Subtalar dislocation of the foot. *J Bone Joint Surg [Am]* 1982;64:433–437.
42. DeLee JC, Evans JP, Julian J. Stress fracture of the fifth metatarsal. *Am J Sports Med* 1983;11:349–353.
43. Devas MB. Stress fractures in children. *J Bone Joint Surg* 1963;45:528–541.
44. Dimentberg R, Rosman M. Peritalar dislocations in children. *J Pediatr Orthop* 1993;13:89–93.
45. Dormans JP, Azzoni M, Davidson RS, et al. Major lower extremity lawn mower injuries in children. *J Pediatr Orthop* 1995;15:78–82.
46. Drez D, Young JC, Johnston RD, et al. Metatarsal stress fractures. *Am J Sports Med* 1980;8:123–125.
47. Drvaric DM, Schmitt EW. Irreducible fracture of the calcaneus in a child. *J Orthop Trauma* 1988;2:154–157.
48. Eastwood DM, Gregg PJ, Atkins RM. Intra-articular fractures of the calcaneus. *J Bone Joint Surg [Br]* 1993;75:183–188.
49. Essex-Lopresti P. The mechanism, reduction technique, and results in fractures of the os calcis. *Br J Surg* 1952;39:395–419.
50. Faciszewski T, Burks RT, Manaster BJ. Subtle injuries of the Lisfranc joint. *J Bone Joint Surg [Am]* 1990;72:1519–1522.
51. Ferkel RD, Fasulo GJ. Arthroscopic treatment of ankle injuries. *Orthop Clin North Am* 1994;25:17–32.
52. Fisher MC, Goldsmith JF, Gilligan PH. Sneakers as a source of *Pseudomonas aeruginosa* in children with osteomyelitis following puncture wounds. *J Pediatr* 1985;106:607–609.
53. Fitzgerald RH, Cowan JDE. Puncture wounds of the foot. *Orthop Clin North Am* 1975;6:965–972.
54. Frank A, Cohen P, Beaufile P, et al. Arthroscopic treatment of osteochondral lesions of the talar dome. *Arthroscopy* 1989;5:57–61.
55. Freiberg AH. Infraction of the second metatarsal. A typical injury. *Surg Gynecol Obstet* 1914;19:191–193.
56. Giannestras NJ. Foot disorders. *Medical and surgical management*, 2nd ed. Philadelphia: Lea & Febiger, 1973:558.
57. Gilmer PW, Herzenberg J, Frank JL, et al. Computerized tomographic analysis of acute calcaneal fractures. *Foot Ankle* 1986;6:184–193.
58. Graham WP, Miller SH, De Muth WE, et al. Injuries from rotary power lawnmowers. *Am Fam Physician* 1976;13:75–79.
59. Graham BS, Gregory DW. *Pseudomonas aeruginosa* causing osteomyelitis after puncture wounds of the foot. *South Med J* 1984;77:1228–1230.
60. Green NE, Bruno J. *Pseudomonas* infection of the foot after puncture wounds. *South Med J* 1980;73:146–149.
61. Greenspoon J, Rosman M. Medial osteochondritis of the talus in children: review and new surgical management. *J Pediatr Orthop* 1987;7:705–708.
62. Grogan DP, Walling AK, Ogden JA. Anatomy of the os trigonum. *J Pediatr Orthop* 1990;10:618–622.
63. Gross RH. Medial peritalar dislocation-associated foot injuries and mechanism of injury. *J Trauma* 1973;15:682–688.
64. Haliburton RA, Sullivan CR, Kelly PJ, et al. The extraosseous and intraosseous blood supply of the talus. *J Bone Joint Surg [Am]* 1958;40:1115–1120.
65. Hardcastle PH, Reschauer R, Kitsha-Lissberg E, et al. Injuries to the tarsometatarsal joint. Incidence, classification, and treatment. *J Bone Joint Surg [Br]* 1982;64:349–356.
66. Hallock GG. Severe lower-extremity injury. the rationale for microsurgical reconstruction. *Orthop Rev* 1986;15:465–470.
67. Hallock GG, Rice DC, Keblish PA, et al. Restoration of the foot using the radial forearm flap. *Ann Plast Surg* 1988;20:14–25.
68. Harty M. Anatomic considerations in injuries of the calcaneus. *Orthop Clin North Am* 1973;4:179–183.
69. Hawkins LG. Fractures of the lateral process of the talus. *J Bone Joint Surg [Am]* 1965;47:1170–1175.
70. Hawkins LG. Fractures of the neck of the talus. *J Bone Joint Surg [Am]* 1970;52:991–1002.
71. Heckman J. Fractures and dislocations of the foot. In: Rockwood C, Green D, eds. *Fractures in adults*, 3rd ed. Philadelphia: JB Lippincott, 1995.
72. Heckman JD, McLean MR. Fractures of the lateral process of the talus. *Clin Orthop* 1985;199:108–113.
73. Helal B, Gibb P. Freiberg's disease: a suggested pattern of management. *Foot Ankle* 1987;8:94–102.
74. Hollinshead WH. *Anatomy for surgeons*, 2nd ed. Baltimore: Williams & Wilkins, 1968.
75. Inokuchi S, Usami N, Hiraishi E, et al. Calcaneal fractures in children. *J Pediatr Orthop* 1998;18:469–474.
76. Jacobs RF, McCarthy RE, Elser JM. *Pseudomonas* osteochondritis complicating puncture wounds of the foot in children: a 10-year evaluation. *J Infect Dis* 1989;160:657–661.
77. Jarvis JG, Skipper J. *Pseudomonas* osteochondritis complicating puncture wounds in children. *J Pediatr Orthop* 1994;14:755–759.
78. Jensen I, Wester JU, Rasmussen F, et al. Prognosis of fracture of the talus in children. *Acta Orthop Scand* 1994;65:398–400.
79. Johanson PH. *Pseudomonas* infections of the foot following puncture wounds. *JAMA* 1968;204:170–172.
80. Johnson EE, Gebhardt JS. Surgical management of calcaneal fractures using bilateral incisions and minimal internal fixation. *Clin Orthop* 1993;290:117–124.
81. Johnson LL. Arthroscopic abrasion arthroplasty: histologic and pathologic perspective: present status. *Arthroscopy* 1986;2:54.
82. Josefsson PO, Karlsson M, Redlund-Johnell I, et al. Closed treatment of Jones fracture. Good results in 40 cases after 11–26 years. *Acta Orthop Scand* 1994;65:545–547.
83. Juliano PJ, Myerson MS. Fractures of the hindfoot. In: Myerson MS, ed. *Foot and ankle disorders*. Philadelphia: WB Saunders, 2000.
84. Karp MG. Kohler's disease of the tarsal scaphoid. *J Bone Joint Surg* 1937;19:84–96.
85. Kavanaugh JHY, Brower TD, Mann RV. The Jones fracture revisited. *J Bone Joint Surg* 1978;60:776–782.
86. Keats TE. *An atlas of normal roentgen variants that may simulate disease*. Chicago: Year Book Medical, 1984.
87. Kenwright J, Taylor RG. Major injuries of the talus. *J Bone Joint Surg [Br]* 1970;52:36–48.
88. Kinnard P, Lirette R. Dorsiflexion osteotomy in Freiberg's disease. *Foot Ankle* 1989;9:226–231.
89. Kirkpatrick DP, Hunter RE, Janes PC, et al. The snowboarder's foot and ankle. *Am J Sports Med* 1998;26:271–277.
90. Laliotis N, Pennie BH, Carty H, et al. Toddler's fracture of the calcaneum. *Injury* 1993;24:169–170.
91. Lang AG, Peterson HA. Osteomyelitis following puncture wounds of the foot in children. *J Trauma* 1976;16:993–999.
92. Lawrence SJ, Botte MJ. Jones' fractures and related fractures of the proximal fifth metatarsal. *Foot Ankle* 1993;14:358–365.
93. Lehman RC, Torg JS, Pavlov H, et al. Fractures of the base of the fifth metatarsal distal to the tuberosity: a review. *Foot Ankle* 1987;7:245–252.
94. Lemaire RG, Bustin W. Screw fixation of fractures of the neck of the talus using a posterior approach. *J Trauma* 1980;20:669–673.
95. Letts RM, Gibeault D. Fractures of the neck of the talus in children. *Foot Ankle* 1980;1:74–77.
96. Linhart WE, Höllwarth ON. Talusfrakturen bei Kindern. *Unfallchirurgie* 1985;88:168–174.
97. Loomer R, Fisher C, Lloyd-Smith R, et al. Osteochondral lesions of the talus. *Am J Sports Med* 1993;21:13–19.
98. Lorentzen JE, Christensen SB, Krogsoe GW, et al. Fractures of the neck of the talus. *Acta Orthop Scand* 1977;48:115–120.
99. Love SM, Grogan DP, Ogden JA. Lawn-mower injuries in children. *J Orthop Trauma* 1988;2:94–101.
100. Lusted LB, Keats TE. *Atlas of roentgenographic measurements*. Chicago: Year Book Medical, 1978.
101. Ly PN, Fallat LM. Transchondral fractures of the talus: a review of 64 surgical cases. *J Foot Ankle Surg* 1993;32:352–374.
102. Mann RA. Biomechanics of the foot and ankle. *Orthop Rev* 1978;7:43–48.
103. Mann RA, Coughlin MJ. *Surgery of the foot and ankle*. St. Louis: CV Mosby, 1993.
104. Mann R, Inman VT. Biomechanics of the foot and ankle. *J Bone Joint Surg [Am]* 1964;46:469–481.
105. Manoli A II, Fakhouri AJ, Weber TG. Compartmental catheterization and fasciotomy of the foot. *Operative Tech Orthop* 1992;2:203–210.
106. Marti R. Fractures of the talus and calcaneus. In: Weber BG, Brunner C, Freuler F, eds. *Treatment of fractures in children and adolescents*. New York: Springer-Verlag, 1980:373–384.
107. Matteri RE, Frymoyer JW. Fracture of the calcaneus in young children. *J Bone Joint Surg [Am]* 1973;55:1091–1094.
108. Mazel C, Rigault P, Padovani JP, et al. Fractures of the talus in children. Apropos of 23 cases. *Rev Chir Orthop* 1986;72:183–195.
109. McCullough CL, Venugopal V. Osteochondritis dissecans of the talus; the natural history. *Clin Orthop* 1979;144:264–268.
110. Merchan EC. Subtalar dislocations: long-term follow-up of 39 cases. *Injury* 1992;23:97–100.
111. Meyers C. On the edge: new riders on the Olympic stage. *Ski Mag* 1996;25:25.
112. Miller AR, Lehman WB. Subtalar dislocation associated with calcaneonavicular coalition: a case report. *Bull Hosp Joint Dis Orthop Inst* 1990;50:84–87.
113. Mindrebo N, Shelbourne KD, Van Meter CD, et al. Outpatient percutaneous screw fixation of the acute Jones fracture. *Am J Sports Med* 1993;21:720–723.
114. Mubarak SJ, Hargens AR. *Compartment syndromes and Volkman's contracture*. Philadelphia: WB Saunders, 1981.
115. Mukherjee SK, Pringle RM, Baxter AD. Fractures of the lateral process of the talus: a report of 13 cases. *J Bone Joint Surg [Br]* 1974;56:263–273.
116. Mulfinger GL, Trueta J. The blood supply to the talus. *J Bone Joint Surg [Br]* 1970;52:160–167.
117. Myerson MS. Management of compartment syndromes of the foot. *Clin Orthop* 1991;271:239–248.
118. Myerson MS. Management of crush injuries and compartment syndromes of the foot. In: Myerson MS, ed. *Foot and ankle disorders*. Philadelphia: WB Saunders, 2000.
119. Myerson MS, Fisher RT, Burgess AR, et al. Fracture dislocations of the tarsometatarsal joints: end results correlated with pathology and treatment. *Foot Ankle* 1986;6:225–242.
120. Niall DM, Murphy PG, Fogarty EE, et al. Puncture wound related *Pseudomonas* infection of the foot in children. *Ir J Med Sci* 1997;166:98–101.
121. Neu HC, Labthavikul P. Antibacterial activity and B-lactamase stability of ceftazidime, and aminothiazolyl cephalosporin potentially active against *Pseudomonas aeruginosa*. *Antimicrob Agent Chemother* 1982;21:11–18.
122. Newman R, Miles R. *Hazard analysis: injuries associated with riding type mowers*. Washington, DC: U.S. Consumer Products Safety Commission, 1981.
123. Ogden JA. *Skeletal injury in the child*. Philadelphia: WB Saunders, 1990.
124. Ove PN, Bosse MJ, Reinert CM. Excision of posterolateral talar dome lesions through a medial transmalleolar approach. *Foot Ankle* 1989;9:171–175.
125. Owen RJT, Hickey FG, Finlay DB. A study of metatarsal fractures in children. *Injury* 1995;6:537–538.
126. Pablot SM, Daneman A, Stringer DA, et al. The value of computed tomography in the early assessment of comminuted fractures of the calcaneus. *J Pediatr Orthop* 1985;5:435–438.
127. Park WH, DeMuth WE. Wounding capacity of rotary lawnmowers. *J Trauma* 1975;15:36–38.

128. Parisien JS. Arthroscopic treatment of osteochondral lesions of the talus. *Am J Sports Med* 1986;14:211–217.
129. Peterson HA, Fitzgibbons TC, Arata MA. Snowmobile injuries in children. *Minn Med* 1979;62:193–196.
130. Portland GH, Kodros S, Kelikian A. *Acute surgical management of the Jones fracture*. Presented at the Annual meeting of the AAOS, American Orthopedic Foot and Ankle Society Specialty Day Meeting, Orlando, FL, March 18, 2000.
131. Quénu E, Küss G. étude sur les luxations du metatarsae (luxations métatarsariennes) du diastasis entre le 1er et la 2e metatarsien. *Rev Chir* 1909;39:281–336.
132. Rang M. Adverse events. In: Epps CH, Bowen JR. *Fractures in children*, 3rd ed. Philadelphia, J.B. Lippincott, 1995:249.
133. Rasmussen F, Schantz K. Radiologic aspects of calcaneal fractures in childhood and adolescence. *Acta Radiol Diagn (Stockh)* 1986;27:575–580.
134. Rosen PR, Micheli LJ, Treves S. Early scintigraphic diagnosis of bone stress and fractures in athletic adolescents. *Pediatrics* 1982;70:11–15.
135. Resch S, Stenstrom A. The treatment of tarsometatarsal injuries. *Foot Ankle* 1990;11:117–123.
136. Richli WR, Rosenthal DI. Avulsion fracture of the fifth metatarsal: experimental study of pathomechanics. *AJR* 1984;143:889–891.
137. Ross G, Cronin R, Hauzenblas J, et al. Plantar ecchymosis sign: a clinical aid to diagnosis of occult Lisfranc tarsometatarsal injuries. *J Orthop Trauma* 1996;10:119–122.
138. Ross PM, Schwentker EP, Bryan H. Mutilating lawn mower injuries in children. *JAMA* 1976;236:480–481.
139. Rubin CT, Lanyon LE. Osteoregulatory nature of mechanical stimuli: function as a determinant for adaptive remodeling in bone. *J Orthop Res* 1987;5:300–310.
140. Saunders AJS, El Sayed TF, Hilson AJW, et al. Stress fractures of the lower leg and foot. *Clin Radio* 1979;30:649–651.
141. Sammarco GJ. The Jones fracture. *Instr Course Lect* 1993;42:201–205.
142. Sandermann J, Torp FT, Thomsen PB. Intraarticular calcaneal fractures in children. *Arch Orthop Trauma Surg* 1987;106:129–131.
143. Sanders R. Intra-articular fractures of the calcaneus: present state of the art. *J Orthop Trauma* 1992;6:254.
144. Sangeorzan BJ, Hansen ST. Early and late posttraumatic foot reconstruction. *Clin Orthop* 1989;243:86–91.
145. Sangeorzan BJ, Wagner UA, Harrington RM, et al. Contact characteristics of the subtalar joint: the effect of talar neck malalignment. *J Orthop Res* 1992;10:544–551.
146. Schaffer JB. *Morriss' human anatomy*, 11th ed. New York: Blakiston, 1953.
147. Schantz K, Rasmussen F. Calcaneus fracture in the child. *Acta Orthop Scand* 1987;58:507–509.
148. Schantz K, Rasmussen F. Good prognosis after calcaneal fracture in childhood. *Acta Orthop Scand* 1988;59:560–563.
149. Schindler A, Mason DE, Allington NJ. Occult fracture of the calcaneus in toddlers. *J Pediatr Orthop* 1996;16:201–205.
150. Schmidt TL, Weiner DS. Calcaneal fractures in children. An evaluation of the nature of the injury in 56 children. *Clin Orthop* 1982;171:150–155.
151. Scully TJ, Besterman G. Stress fracture? A preventable training injury. *Milit Med* 1982;147:285–287.
152. Serrafian S. Functional characteristics of the foot and plantar aponeurosis under tibiotalar loading. *Foot Ankle* 1987;8:4–18.
153. Serrafian SK. Syndesmology. In: Serrafian SK, ed. *Anatomy of the foot and ankle: descriptive, topographic, functional*, 2nd ed. Philadelphia: JB Lippincott, 1993:207.
154. Shereff M. Compartment syndromes of the foot. *Instr Course Lect* 1990;39:127–132.
155. Silas SI, Herzenberg JE, Myerson MS, et al. Compartment syndrome of the foot in children. *J Bone Joint Surg [Am]* 1995;77:356–361.
156. Smillie IS. Freiberg's infraction (Kohler's second disease). *J Bone Joint Surg [Br]* 1957;39:580.
157. Smith GR, Winquist RA, Allan NK, et al. Subtle transchondral fractures of the talar dome: a radiological perspective. *Radiology* 1977;124:667–673.
158. Smith JW, Arnoczky SP, Hersh A. The intraosseous blood supply of the fifth metatarsal: implications for proximal fracture healing. *Foot Ankle* 1992;13:143–152.
159. Sneppen O, Christensen S, Krogsoe O. Fracture of the body of the talus. *Acta Orthop Scand* 1977;48:317–324.
160. Spak L. Fractures of the talus in children. *Acta Chir Scand* 1954;107:553–566.
161. Starshak RJ, Simons GW, Sty JR. Occult fracture of the calcaneus? Another toddler's fracture. *Pediatr Radio* 1984;14:37–40.
162. Stephens NA. Fracture? Dislocations of the talus in childhood. *Br J Surg* 1956;43:600–604.
163. Swanson TV, Bray TJ, Holmes GB Jr. Fractures of the talar neck. A mechanical study of fixation. *J Bone Joint Surg [Am]* 1992;74:544–551.
164. Tejwani N, DiGiovanni C, Kuo R, et al. *Fractures of the os calcis in children: a study of 43 fractures*. Presented at AAOS annual meeting, American Orthopedic Foot and Ankle Society Specialty Day, Orlando, FL, March 2000.
165. Thomas HM. Calcaneal fracture in childhood. *Br J Surg* 1969;56:664–666.
166. Thordarson DB, Triffon M, Terk M. Magnetic resonance imaging to detect avascular necrosis after open reduction and internal fixation of talar neck fractures. *Foot Ankle Int* 1996;17:742–747.
167. Torg JS, Balduini FC, Zelko RR, et al. Fractures of the base of the fifth metatarsal distal to the tuberosity. Classification and guidelines for non-surgical and surgical management. *J Bone Joint Surg* 1984;66:209–214.
168. Tornetta P III. The Essex-Lopresti reduction for calcaneal fractures revisited. *J Orthop Trauma* 1998;12:469–473.
169. Trillat A, Bousquet G, Lapeyre B. Les fractures-Séparation du col ou du corps de l'astragale: intérêt du visage par voie postérieure. *Rev Chir Orthop* 1970;56:529–536.
170. Trillat A, Lerat JL, Leclaire P, et al. Tarsometatarsal fracture dislocations. *Rev Chir Orthop* 1976;62(suppl):685–702.
171. Van Buecken K, Barrack RL, Alexander AH, et al. Arthroscopic treatment of transchondral talar dome fractures. *Am J Sports Med* 1989;17:350–355.
172. Verdile VP, Freed H, Gerard J. Puncture wounds to the foot. *J Emerg Med* 1989;7:193–199.
173. Vosburgh CL, Gruel CG, Herndon WA, et al. Lawn mower injuries of the pediatric foot and ankle: observations on prevention and management. *J Pediatr Orthop* 1995;15:504–509.
174. Vuori JP, Aro HT. Lisfranc joint injuries: trauma mechanisms and associated injuries. *J Trauma* 1993;35:40–45.
175. Walling AK, Grogan DP, Carty CT, et al. Fractures of the calcaneal apophysis. *J Orthop Trauma* 1990;4:349–355.
176. Warren MP, Brooks-Gunn J, Hamilton LH, et al. Scoliosis and fractures in young ballet dancers. *N Engl J Med* 1986;314:1348–1353.
177. Waugh W. The ossification and vascularization of the tarsal navicular and their relation to Kohler's disease. *J Bone Joint Surg [Br]* 1958;40:765–777.
178. Weber TG, Manoli A. II: Compartment syndromes of the foot. *Foot Ankle Clin* 1999;4:473–486.
179. Weinstien SL, Bonfiglio M. Unusual accessory (bipartite) talus simulating fracture. *J Bone Joint Surg [Am]* 1975;57:1161–1163.
180. Wildenauer E. Die blutversorgung des talus. *Z Anatomid Entwick Lungsgeschichte* 1950;115:32–36.
181. Wiley JJ. Tarso-metatarsal injuries in children. *J Pediatr Orthop* 1981;1:255–260.
182. Wiley JJ. The mechanism of tarso-metatarsal injuries. *J Bone Joint Surg [Br]* 1971;53:474–482.
183. Wicks MH, Harbison JS, Paterson DC. Tendon injuries about the foot and ankle in children. *Aust N Z J Surg* 1980;50:158–161.
184. Wiley JJ, Profitt A. Fractures of the os calcis in children. *Clin Orthop* 1984;188:131–138.
185. Wilson DW. Injuries of the tarso-metatarsal joints. *J Bone Joint Surg [Br]* 1972;54:677–686.
186. Wiss DA, Kull DM, Perry J. Lisfranc fracture-dislocations of the foot: a clinical-kinesiologic study. *J Orthop Trauma* 1987;1:267–274.
187. Wright DG, Desai SM, Henderson WH. Action of the subtalar and ankle joint complex during the stance phase of walking. *J Bone Joint Surg [Am]* 1964;46:361–382.
188. Yancey HA. Lacerations of the plantar aspect of the foot. *Clin Orthop* 1977;122:46–52.
189. Yngve DA. Stress fractures in the pediatric athlete. In: Sullivan JA, Grana WA, eds. *The pediatric athlete*. Park Ridge, IL: American Academy of Orthopaedic Surgeons, 1990:235–240.
190. Zimmer TJ, Johnson KA. Subtalar dislocations. *Clin Orthop* 1989;238:190–194.
191. Ziv I, Mosheiff R, Zeligowski A, et al. Crush injuries of the foot with compartment syndrome: immediate one-stage management. *Foot Ankle* 1989;9:185–189.
192. Zook EG, Russell RC, Asaadi M. A comparative study of free and pedicle flaps for lower extremity wounds. *Ann Plast Surg* 1986;17:21–33.