

General Principles of Managing Orthopaedic Injuries

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Introduction

Skeletal injuries are common in children. In a review of over 8,000 children's fractures, Landin¹⁵² estimated that over 40 percent of boys and 25 percent of girls had sustained a fracture by age 16 years. Because of the unique properties of the immature skeleton, these injuries have different characteristics, complications, and management than similar injuries in adults.

A number of studies have examined the epidemiology of fractures in children.^{125,152,219,272,277} Most studies have shown a male predominance, particularly in adolescence. Fractures in children less than 18 months old are rare and should raise the question of nonaccidental trauma.²⁷⁷ Combining the data from five large epidemiologic studies reveals fractures of the distal forearm to be the most common fracture in children, accounting for nearly a quarter of 12,946 fractures. The clavicle is the next most commonly injured site, representing over 8 percent of all children's fractures (Table 39-1).^{125,152,219,272,277}

Unique Properties of the Immature Skeleton

The immature skeleton has several unique properties that affect the management of injuries in children. These properties include an increased resiliency to stress, a thicker periosteum, an increased potential to remodel, shorter healing times, and the presence of a physis.

PLASTIC DEFORMATION

A few studies have compared the mechanical properties of bone in children and bone in adults.^{7,61,115,139,260} Currey and Butler found immature bone to be weaker in bending

strength but to absorb more energy prior to fracture.⁶¹ This is a result of the ability of immature bone to undergo plastic (permanent) deformation (Fig. 39-1). Although plastic deformation has been described in adults,^{220,236} it is much more common in children. Borden is often credited with the first clinical description of plastic deformation in children.³³ In children, plastic deformation is most common in the forearm, particularly the ulna.^{32,77,168,190,228,241} Although bone in young children may remodel plastic deformation, most authors recommend reduction of plastic deformation of the forearm if there is more than 20 degrees of angulation or the child is more than 4 years old and has either a clinically evident deformity or limitation of pronation/supination. Sanders and Heckman were able to reduce an average of 85 percent of the angulation present prior to reduction. They used general anesthesia and a fulcrum to apply a steady force at the apex of the deformity for several minutes.²²⁸ Plastic deformation of the ulna has also been reported in a majority of isolated radial head dislocations.¹⁶²

BUCKLE (TORUS) FRACTURES

Buckle fractures, also called torus fractures because of their resemblance to the base of an architectural column, most commonly occur at the transition between metaphyseal woven bone and the lamellar bone of the diaphyseal cortex (Fig. 39-2).^{161,215} Buckle fractures represent a spectrum of injuries from mild plastic deformation of one area of the cortex to complete fractures with a buckled appearance.

It is not uncommon for torus fractures to be diagnosed several days or even weeks after injury, as the pain and swelling may be attributed to a sprain. Although most torus fractures can be managed successfully with minimal symptomatic treatment, it is important to identify minimally displaced complete fractures that have a buckled appearance. These complete fractures are potentially unstable and may

TABLE 39-1 Frequency of Fractures at Selected Sites in Children

	Epidemiological Study					Total Fx	%
	A ²⁷⁸	B ²⁷²	C ²¹⁹	D ¹²⁵	E ¹⁵²		
Total fractures in series	923	2,040	410	291	8,682	12,946	
<i>Anatomic site</i>							
Clavicle	58	222	55	45	703	1,083.0	8.4
Humerus (proximal end and shaft)	18	81	14	13		126.0	.9
Distal humerus	71	158		68	287	584.0	4.5
Radial neck	25	45		1	104	175.0	1.4
Radius/ulna (shafts)	60	108	23	39	295	525.0	4.1
Distal radius/ulna	330	755	81	80	1,971	3,217.0	24.8
Hand	136	494	88			718	5.4
Femur	18	87	27	13		145	1.1
Tibia/fibula (shafts)	40	256	19	10	434	759.0	5.9
Ankle	37	61	28	14	478	618	4.8
Foot	71	172	28			271	2.1

Note: Because not all fractures are listed, fractures do not total 100 percent.

Modified from Reed MH: Epidemiology of children's fractures. In Letts RM (ed): Management of Pediatric Fractures, p 2. New York, Churchill Livingstone, 1994.

displace if not managed with a well-molded cast (Fig. 39-3). Although such late displacement is usually mild and remodels with no sequelae, parents are often upset when the fracture is more displaced when the cast is removed than at the time of injury.

GREENSTICK FRACTURES

Greenstick fractures are unique to children because immature bone is more flexible and has a thicker periosteum than mature adult bone. In a greenstick fracture, the cortex in tension fractures completely while the cortex and periosteum in compression remain intact but frequently undergo plastic deformation. It has been said that it is necessary to "complete" the fracture on the intact compression side of greenstick fractures;^{74,196} however, this has not been our experience. We believe it is only necessary to achieve an anatomic reduction of a greenstick fracture. In order to reduce a greenstick fracture, it is usually necessary to "unlock" the impacted fragments on the tension side. This is accomplished by initially exaggerating the deformity and then applying traction and a reducing force. In our experience, whether or not the fracture is completed during the exaggeration of the deformity has not been important. Due to the intact cortex and periosteum, greenstick fractures are usually



FIGURE 39-1 Stress-strain curves for mature and immature bone. The increased strain of immature bone prior to failure represents plastic deformation. (From Rang M: Children's Fractures. Philadelphia, JB Lippincott Co, 1983. Originally from Currey JD, Butler G: The mechanical properties of bone tissue in children. *J Bone Joint Surg* 1975;57-A:810.)

quite stable following reduction (Fig. 39-4). Greenstick fractures have been reported to have an increased likelihood of refracture.²⁶¹

REMODELING/OVERGROWTH

Not only do children's fractures heal more rapidly than in adults but, once healed, they are more likely to remodel

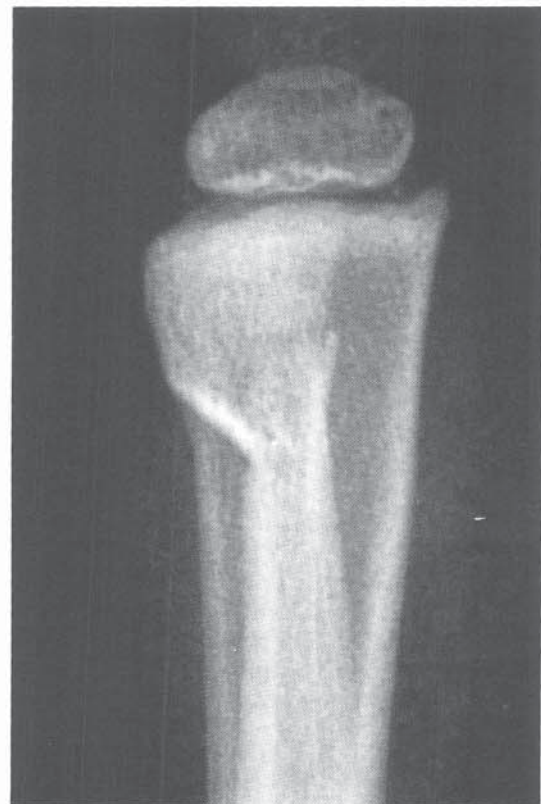


FIGURE 39-2 Lateral radiograph of the distal radius showing a buckle fracture of the dorsal cortex. The volar cortex is uninvolved and the dorsal cortex is not completely fractured.

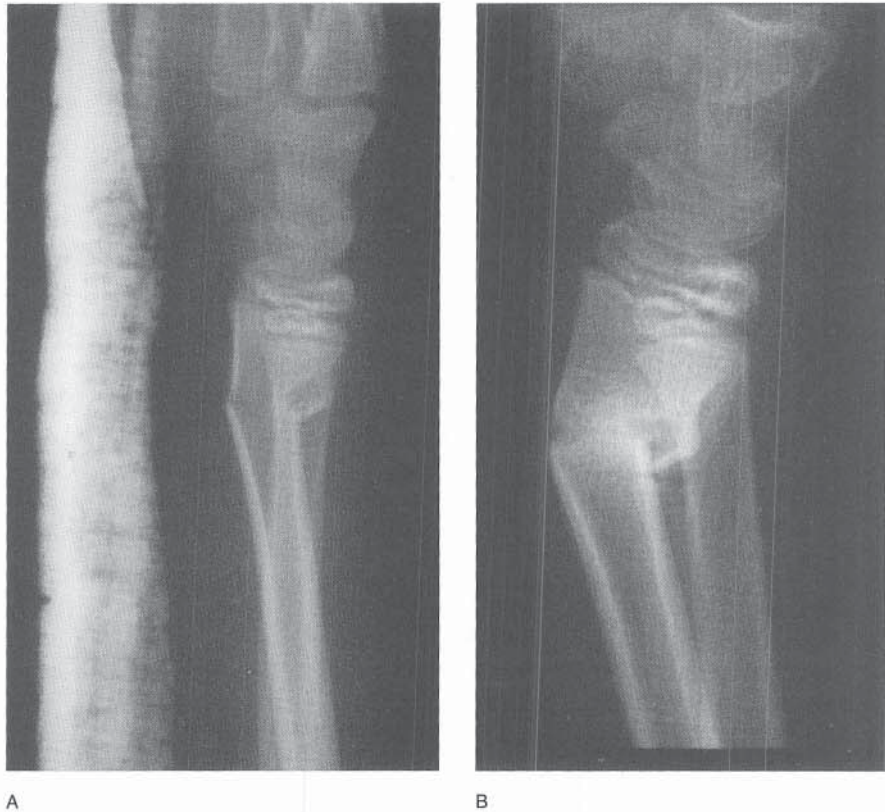


FIGURE 39-3 A, Lateral radiograph of a minimally displaced fracture of the distal radial metaphysis. Despite the buckled appearance, both cortices are completely fractured. This fracture was managed in a poorly molded volar splint. B, Radiograph obtained after removal of the volar splint, 4 weeks after the injury. Note the increased angulation.



FIGURE 39-4 A, Lateral radiograph of a greenstick both bone forearm fracture. The dorsal cortex angles without completely fracturing (plastic deformation). B, Lateral radiograph obtained following reduction.

residual deformity (Fig. 39-5). Factors that affect the remodeling potential of a deformity include the amount of growth remaining and the plane of the deformity in relation to adjacent joints.^{79,80,87,158,261,265} There are several factors to consider when determining how much growth will contribute to the remodeling potential of a fracture. Obviously, the patient's skeletal age is the single most important factor. Other factors include the deformity's proximity to the physis and the growth potential of the particular physis. For example, since 80 percent of the growth of the proximal humerus comes from the proximal physis, deformity associated with proximal humeral fractures is much more likely to remodel than deformity associated with distal humeral fractures.²¹⁴

Wolf's law states that bone remodels according to the stress placed across it.^{149,279} It follows that posttraumatic deformity in the plane of motion of a joint will have greater potential to remodel than deformity not in the plane of motion. This fact is demonstrated with fractures of the femoral shaft, which will remodel a large amount of sagittal plane deformity, a lesser amount of coronal deformity, and little or no rotational deformity.^{63,265}

Another consideration in the management of children's fractures is the potential for accelerated growth of an injured limb. Clinically, this is most frequently seen in diaphyseal femoral fractures. It has long been recognized that fractures of the femoral shaft will spontaneously correct shortening of up to 2 cm.^{1,55,87,110,117,128,129} It has been hypothesized that this "overgrowth" is a result of hyperemia associated with the fracture. However, recent evidence casts some doubt on this theory. First, fractures of the radius do not demonstrate this propensity for overgrowth.⁶⁵ Second, efforts to stimulate blood flow by periosteal stripping do not result in permanent

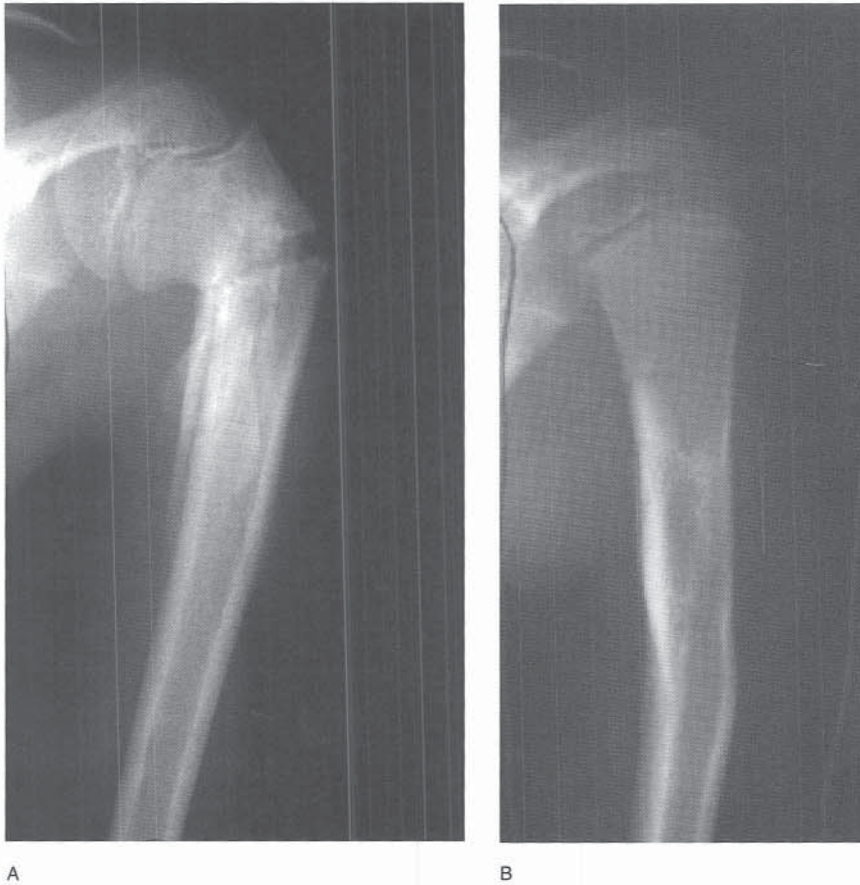


FIGURE 39-5 A, AP radiograph of a proximal humerus fracture in an 8-year-old boy. The fracture has healed with significant angulation. B, AP radiograph obtained 1½ years after injury demonstrates extensive remodeling of the proximal humerus.

growth increases.^{85,132,239,255} Finally, anatomic reduction of femoral shaft fractures treated operatively has not resulted in significant overgrowth.^{17,30,66,142,148} Thus, there may exist some other, yet to be determined, factor that predisposes an injured extremity to return to its normal, preinjury length.

PHYSEAL INJURIES

Physeal injuries represent 15 to 30 percent of all fractures in children.^{28,54,169,183,277} The incidence varies with age and has been reported to peak in adolescents.^{183,208,210} Physeal injuries involving the phalanges have been reported to account for over 30 percent of all physeal fractures.^{183,210} Fortunately, although physeal injuries are common, growth deformity is a rare occurrence, occurring in only 1 to 10 percent of all physeal injuries.^{183,208,226}

Although problems arising from physeal injury are rare, they are often predictable and, occasionally, preventable. A basic understanding of the anatomy and physiology of the physis and its response to injury is necessary to effectively manage injuries to the growth plate.

Physeal Anatomy. It is important to distinguish the physis (also referred to as the epiphyseal plate, epiphyseal growth plate, or epiphyseal cartilage) from the epiphysis, or secondary ossification center. The physis is connected to the epiphysis and metaphysis via the zone of Ranvier and the perichondral ring of LaCroix (Fig. 39-6). The zone of Ranvier is a wedge-shaped group of germinal cells that is continuous

with the physis and contributes to latitudinal, or circumferential, growth of the physis.¹²⁴ The zone of Ranvier consists of three cell types—osteoblasts, chondrocytes, and fibroblasts. Osteoblasts form the bony portion of the perichondral ring at the metaphysis. Chondrocytes contribute to latitudinal growth, and fibroblasts circumscribe the zone and anchor it to perichondrium above and below the growth plate.¹²⁴ The perichondral ring of LaCroix is a fibrous structure that is continuous with the fibroblasts of the zone of Ranvier and the periosteum of the metaphysis. It provides strong mechanical support for the bone-cartilage junction of the growth plate.¹³³

The physis consists of chondrocytes in an extracellular matrix. Both the chondrocytes and the matrix are preferentially oriented along the longitudinal axis of long bones. The physis has traditionally been divided into four zones: the resting or germinal zone, the proliferative zone, the zone of hypertrophy, and the zone of enchondral ossification, which is continuous with the metaphysis (Fig. 39-6). The first two zones have an abundant extracellular matrix and, subsequently, a great deal of mechanical integrity, particularly in response to shear forces. The third layer, the hypertrophic zone, contains scant extracellular matrix and is weaker. On the metaphyseal side of the hypertrophic zone there is an area of provisional calcification leading to the zone of enchondral ossification. The calcification in these areas provides additional resistance to shear. Thus, the area of the hypertrophic zone just above the area of provisional calcification is the weakest area of the physis, and it is here that

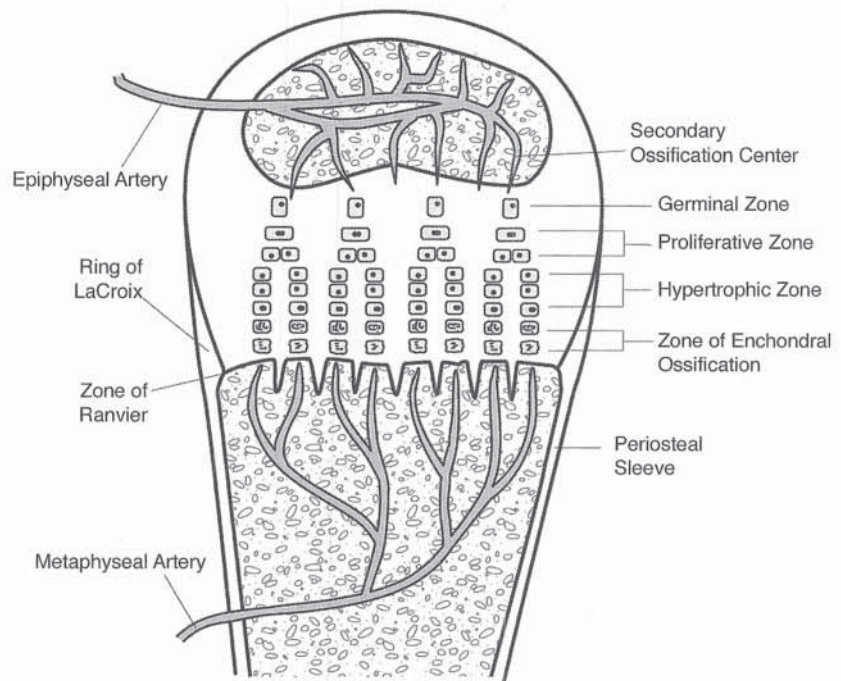


FIGURE 39–6 Anatomy of a physis. Most injuries occur just above the area of provisional calcification within the hypertrophic zone. Subsequently the germinal layer frequently remains intact and attached to the epiphysis.

most injuries to the physis occur.^{100,108,226} The fact that the cleavage plane through the physis is through the hypertrophic zone implies that after most injuries, the germinal layer of the physis remains intact and attached to the epiphysis. Thus, provided there is not an insult to the blood supply of the germinal layer or the development of a “bony bridge” across the injured physis, normal growth should resume after an injury.

The blood supply to the germinal zone of the physis was studied in a classic set of experiments in monkeys by Dale and Harris.⁶² They described two types of epiphyseal vascularization (Fig. 39–7). Type A epiphyses are nearly entirely covered by articular cartilage. In these epiphyses, the blood supply enters the periphery after traversing the perichondrium. Consequently, the blood supply is vulnerable to damage if the epiphysis is separated from the metaphysis. Type B epiphyses are only partially covered by articular cartilage. Their blood supply enters from the epiphyseal side and is protected from vascular injury during separation. The

proximal femur and proximal radius are the only two type A epiphyses. Dale and Harris confirmed their theory that type B epiphyses were protected from vascular injury by studying the histologic changes that occurred following separation of the distal radial epiphysis in rabbits. They noted that by 3 weeks after separation it was nearly impossible to distinguish the injured epiphysis from the control.⁶²

Harris Growth Arrest Lines. Harris is credited with the first radiographic observation of “bony striations” in the metaphysis of long bones.¹⁰⁶ These “Harris growth arrest lines” are transversely oriented condensations of normal bone and are thought to represent slowing or cessation of growth. They may be present in a single bone, following an isolated traumatic injury, or in all long bones, following a significant systemic illness.^{106,123,197,198} When present following a physal injury, they serve as an effective representation of the health of the physis.¹²³ If the growth arrest line is transverse and parallel to the physis, the physis can be assumed

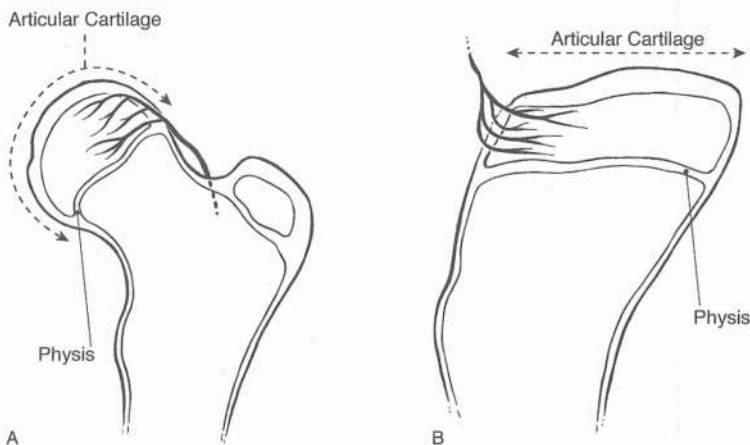


FIGURE 39–7 Two types of epiphyseal blood supply as defined by Dale and Harris. A, Type A. The epiphysis is nearly entirely covered by articular cartilage. Consequently the blood supply traverses the metaphysis and may be damaged on separation of the metaphysis and epiphysis. B, Type B. The epiphysis is only partially covered by articular cartilage. Because the blood supply enters through the epiphysis, separation of the metaphysis and epiphysis will not compromise the blood supply to the germinal layer. (From Dale GC, Harris WR: Prognosis of epiphyseal separation: an experimental study. *J Bone Joint Surg* 1958;40-B:116.)

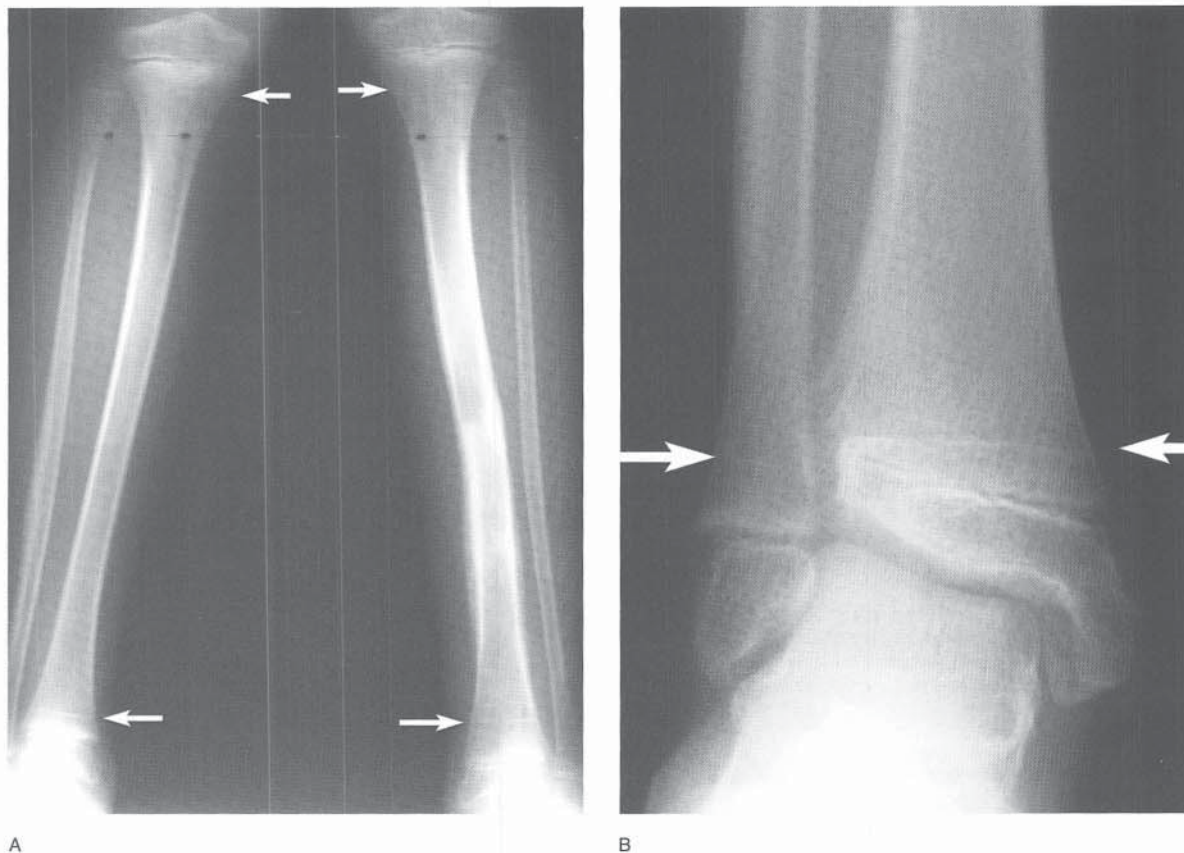


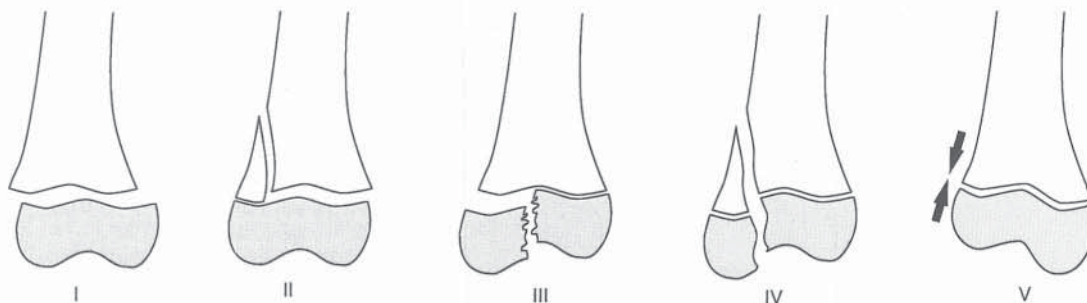
FIGURE 39-8 Harris growth arrest lines. **A**, Bilateral, symmetric, transverse growth arrest lines (*arrows*) in the proximal and distal tibiae of a 7-year-old boy 1 year after a vehicle-pedestrian accident in which the boy sustained multiple injuries. Note the healed left tibial fracture. On the left side, both the proximal and distal growth arrest lines have migrated farther from their physes, probably as a result of the fracture. **B**, Asymmetric growth arrest line in the distal tibia (*arrow*). Although the line appears perpendicular to the tibial shaft, it is not parallel to the physis. There has been medial growth, but no lateral growth; thus the growth arrest line appears “tethered” to the physis laterally. Note the normal growth arrest line in the fibula (*arrow*).

to be growing normally. If there has been a partial injury to the physis, the growth arrest line will be asymmetric. There will be no growth arrest line if there has been no growth due to a total physal injury (Fig. 39-8). Harris growth arrest lines may also be seen on magnetic resonance imaging (MRI).²⁸¹

Classification of Physal Injuries. Over the years a number of classification systems for physal injuries have been described, including those by Foucher, Poland, Aitken, and Ogden.^{2-4,78,201,202,211} However, the most widely utilized system is that of Salter and Harris (Fig. 39-9).²²⁷

A Salter-Harris type I injury is a separation of the epiphysis from the metaphysis occurring entirely through the physis. It is quite rare and seen most frequently in infants or in pathologic fractures, such as those secondary to rickets or scurvy. Because the germinal layer remains with the epiphysis, growth is not disturbed unless the blood supply is interrupted, as frequently occurs with traumatic separation of the proximal femoral epiphysis.

In a Salter-Harris type II injury the fracture extends along the hypertrophic zone of the physis and at some point exits through the metaphysis. The epiphyseal fragment contains the entire germinal layer as well as a metaphyseal fragment



Salter-Harris Classification

FIGURE 39-9 Salter-Harris classification of physal fractures.

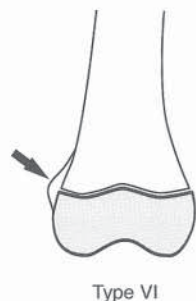


FIGURE 39-10 Rang's type VI physeal injury. This represents an injury to the perichondral ring.

of varying size. This fragment is known as Thurston Holland's sign. The periosteum on the side of the metaphyseal fragment will be intact and will provide stability once the fracture is reduced. Growth disturbance is rare, as the germinal layer remains intact.

In a Salter-Harris type III injury the fracture extends along the hypertrophic zone until it exits through the epiphysis. Thus, by definition, type III fractures cross the germinal layer and are usually intra-articular. Consequently, if displaced, they require an anatomic reduction, which may need to be achieved open.

Salter-Harris type IV injuries extend from the metaphysis across the physis and into the epiphysis. Thus, the fracture crosses the germinal layer of the physis and usually extends into the joint. As in type III injuries, it is important to achieve an anatomic reduction to prevent osseous bridging across the physis and to restore the articular surface.

A Salter-Harris type V injury is a crushing injury to the physis from a pure compression force. It is quite rare; in fact, Peterson and Burkhart have questioned whether such an injury can occur.²⁰⁹ Those authors who have reported Salter-Harris type V injuries have noted a poor prognosis, with almost universal growth disturbance.^{227,238}

Although the Salter-Harris classification of physeal fractures is by far the most widely utilized system, there are a few physeal injuries that do not fit into this classification scheme. The first is an injury to the perichondral ring. Salter's colleague Mercer Rang termed this a type VI physeal injury (Fig. 39-10).^{201,202} (This injury is also included in Ogden's classification.) Basing his system on a review of 951 fractures, Peterson purposed a new classification scheme (Fig. 39-11).²⁰⁸ Although this classification system has many

similarities to the Salter-Harris scheme, its important addition is the Peterson type I fracture—a transverse fracture of the metaphysis with extension longitudinally into the physis. Clinically, this fracture is seen quite commonly in the distal radius. Peterson also described a type VI injury, which is an open injury associated with loss of the physis.

Treatment of Physeal Injuries. In general, the principles involved in the treatment of physeal injuries are the same as those involved in the treatment of all fractures, although there are a few important caveats. As with all traumatic injuries, before an injury to the physis is treated, the patient must be thoroughly assessed using the ABC's of trauma (see subsequent discussion under Care of the Multiply Injured Child). Once the child has been stabilized and all life- and limb-threatening injuries identified, a treatment plan can be developed. It is important to remember that physeal fractures can and often do coexist with neurovascular or open injuries.¹¹² When this occurs the physeal fracture is treated after appropriate management of the soft tissue injuries. The goal in treating physeal fractures is to achieve and maintain an acceptable reduction without subjecting the germinal layer of the physis to any further damage. The most subjective of these goals, and perhaps the most important, is determining the limits of an acceptable reduction. A number of factors must be considered when assessing a "nonanatomic" reduction. These include the amount of residual deformity, the location of the injury, the age of the patient, and the amount of time that has elapsed since the injury. The location of the injury and the patient's age are determining factors in the bone's remodeling potential. Obviously, more deformity can be accepted if the potential to remodel is high. Both Rang and Salter have stressed the importance of avoiding damage to the germinal layer of the physis during reduction. Thus, they recommend accepting *any* displacement in type I or II injuries after 7 to 10 days, believing it is safer to perform an osteotomy later than to risk injuring the physis with a traumatic reduction of a physeal fracture that has begun to heal. Because of the intra-articular component, displaced type III and IV injuries must be reduced regardless of the time that has elapsed since the injury.^{217,226} Once a physeal fracture has been reduced, the reduction can be maintained with a cast, pins, internal fixation, or some combination of these three. Specific recommendations regarding the method and duration of immobilization are discussed later with each injury.

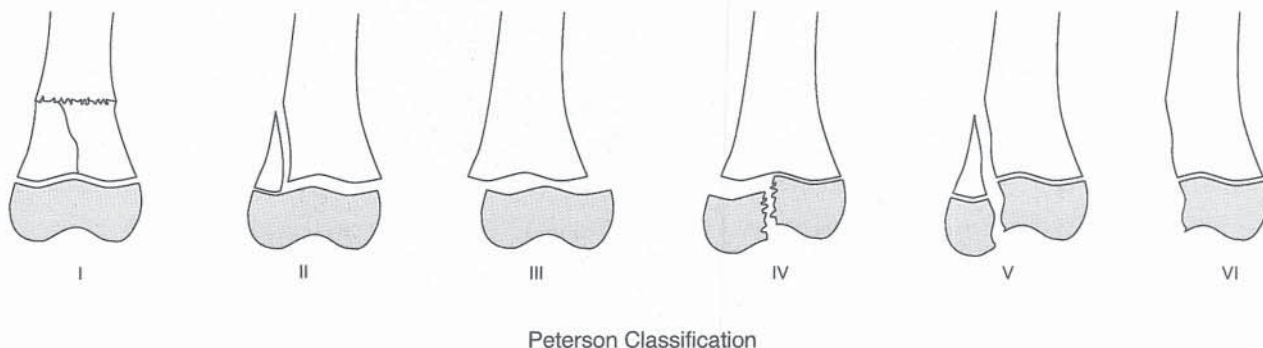


FIGURE 39-11 Peterson's classification of physeal fractures. Type I injuries are frequently seen in the distal radius. Type VI injuries are open and associated with loss of a portion of the physis.

Complications of Physeal Injuries. Like all fractures, physeal injuries may be complicated by malunion, infection, neurovascular problems, or osteonecrosis. The best treatment of these complications is avoidance, but even under the best of circumstances these problems can arise. The treatment of these complications is discussed later in the context of specific injuries.

A complication unique to physeal fractures is growth disturbance. Although trauma is the most common cause of growth disturbance, growth disturbance is also seen as a sequela of Blount's disease, infection, and irradiation.^{27,153,157} Although physeal injuries represent 15 to 30 percent of all fractures, growth arrest occurs following only 1 to 10 percent of physeal fractures. A number of factors affect the likelihood of developing a growth arrest. Most important is the severity of the injury to the physis. Comminuted fractures from high-energy injury are more likely to result in physeal arrest. Physeal injuries that cross the germinal layer (i.e., Salter-Harris type III and IV injuries) are also more likely to be associated with subsequent growth disturbance. Fortunately, not all patients who develop a physeal arrest will require treatment. This is because physeal injuries are most common in adolescents, who often have limited growth remaining.^{183,208,226}

Growth disturbance from a physeal fracture is usually evident 2 to 6 months after the injury, but it may not become evident for up to a year.²²⁶ Thus, it is important not only to warn parents about this potential problem, but also to

follow patients with physeal fractures long enough to identify growth arrest. Early identification of a traumatic growth disturbance can make its management considerably easier, as the treatment can be directed solely toward resolving the arrest, rather than addressing both the arrest and an acquired growth deformity. Growth disturbance is usually the result of the development of a bony bridge, or bar, across the physeal cartilage. However, growth disturbance may occur following traumatic injury without the development of a bony bridge. Presumably, this occurs because the injury slows growth of a portion of the physis rather than stopping it completely. The resulting asymmetric growth can produce clinically significant angular deformity (Fig. 39–12).

The development of a bony bar may create either a complete or partial growth disturbance. If the area of the bar is large, it may stop the growth of the entire physis (Fig. 39–13). More often a bar forms in a portion of the physis and stops growth at that point, while the rest of the physis continues to grow. This produces a tethering effect, which may result in shortening or progressive angular deformity or both (Figs. 39–14 and 39–15). In order to appropriately treat a physeal bar, both the extent and location of the bar and the amount of growth remaining from the physis must be determined. The anatomy of a physeal bar may be delineated using plain radiography, tomography, CT, or MRI.^{27,44,135,166,199} Of these, CT is the modality most commonly used today.^{135,166} Partial physeal arrests are usually classified as peripheral (type A) or central (type B or C), depending

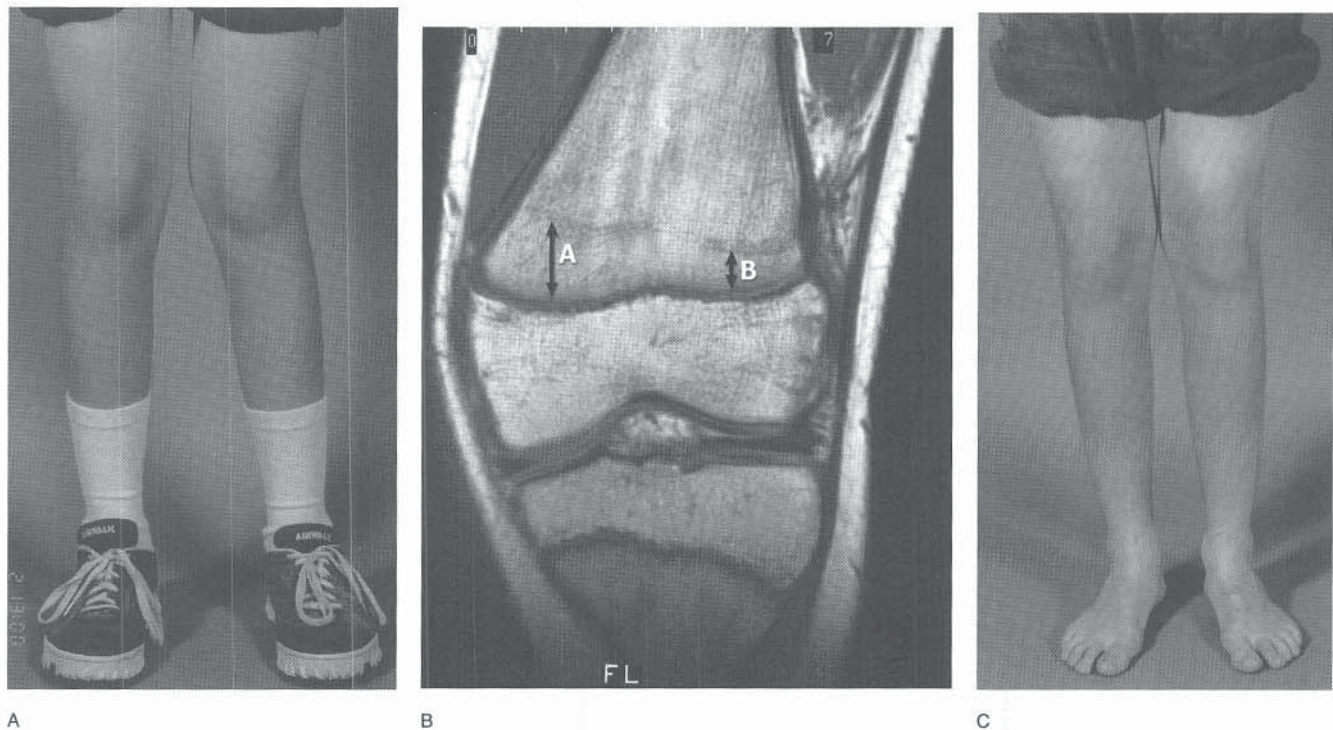
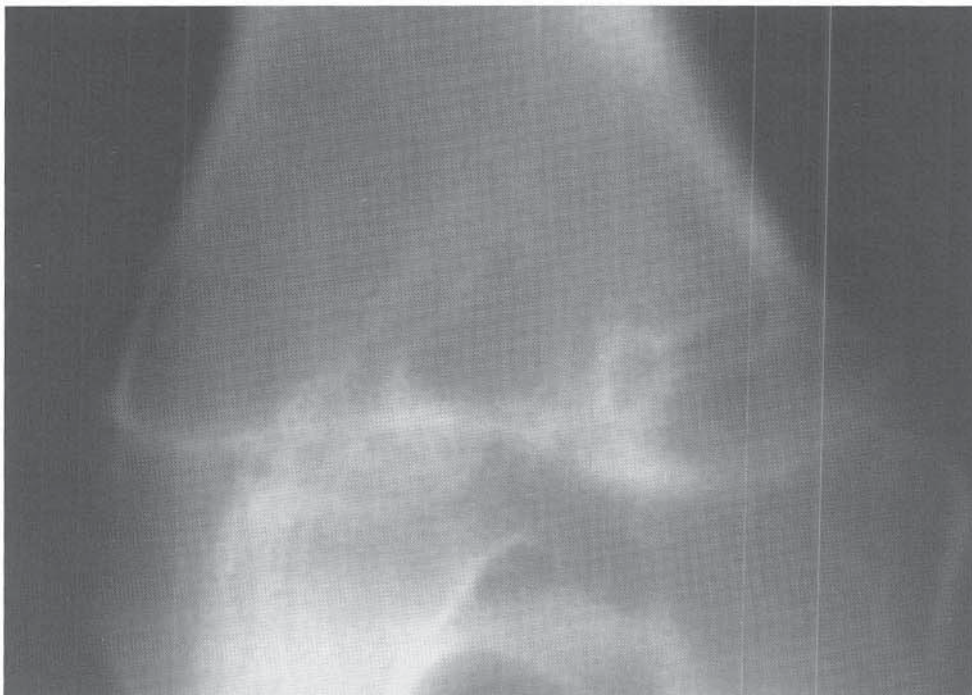


FIGURE 39–12 Asymmetric growth following a Salter-Harris type II distal femoral fracture. **A**, Valgus deformity 15 months after fracture. **B**, MR image demonstrating asymmetric growth of the distal femoral physis. The distance from the physis to the Harris growth arrest line is greater medially (*A*) than laterally (*B*). The fact that the growth arrest line has migrated proximally on the lateral aspect reflects a “slowing” of growth rather than a complete “arrest.” **C**, Clinical appearance 8 months after a medial distal femoral epiphysiodesis was performed. Lateral growth continued until the deformity was corrected. At this point, a lateral hemi-epiphysiodesis and a contralateral epiphysiodesis were performed.



A



B

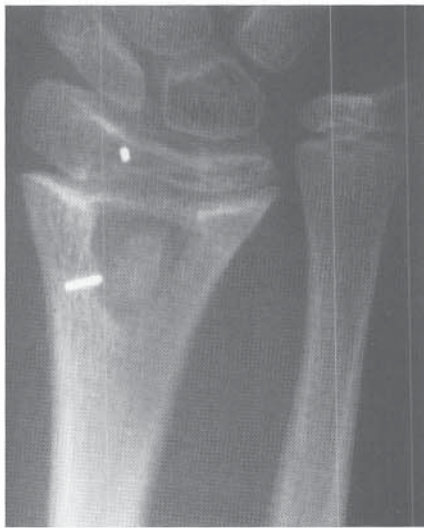
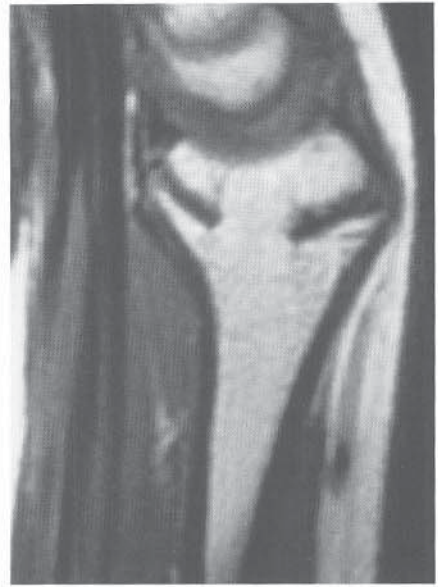
FIGURE 39-13 Salter-Harris type II fracture of the right distal femur complicated by pin tract sepsis and complete physeal arrest. **A**, AP radiographs of the right and left knee. The uninvolved left knee has a healthy-appearing distal femoral physis. On the right side there is no radiolucency corresponding to the physis. **B**, Tomograph revealing a small amount of physis on the far medial aspect of the right distal femur. Most of the physis has been replaced by radio-dense scar. (Radiographic evidence of the cross-pins is present on both the plain radiograph and the tomograph.)



A



B



C



D

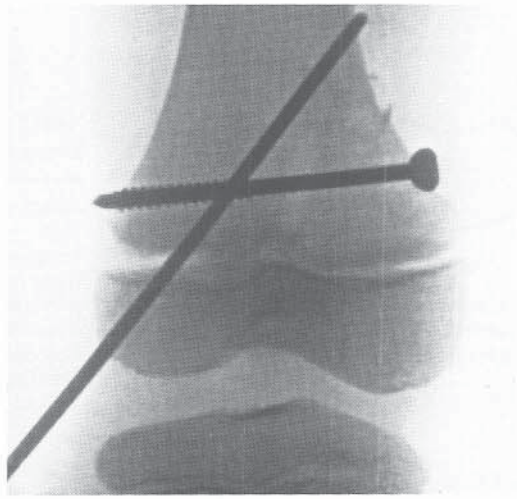


E

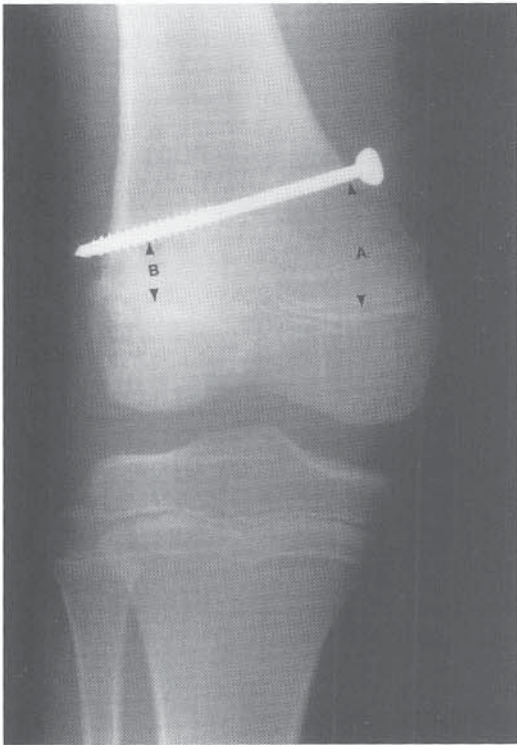
FIGURE 39-14 Partial physeal arrest (type B) producing primarily shortening. **A**, AP radiograph of the wrist of a 12-year-old girl who had sustained a Salter-Harris type II fracture of the distal radius 6 years earlier. Note the ulnarly positive variance as well as the physeal bar in the center of the distal radius. **B**, Coronal and sagittal MR images show the extent of the bar. **C**, The bar has been resected and metallic markers placed in the epiphysis and metaphysis. **D**, AP and lateral radiographs showing resumption of growth, as evidenced by an increased distance between metallic markers. The ulnarly positive variance persists. **E**, Lateral radiograph following ulnar shortening to treat symptomatic ulnarly positive variance.



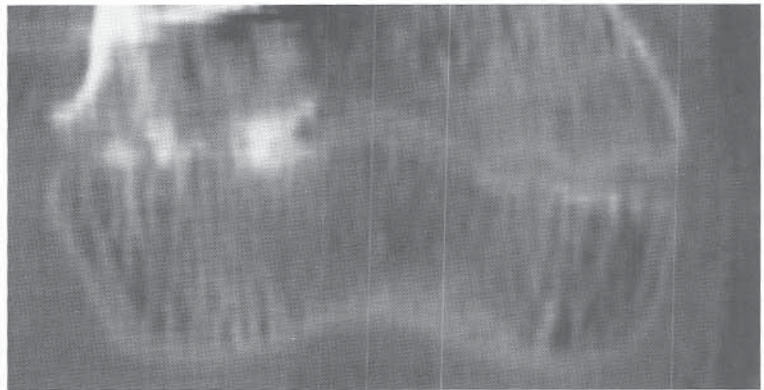
A



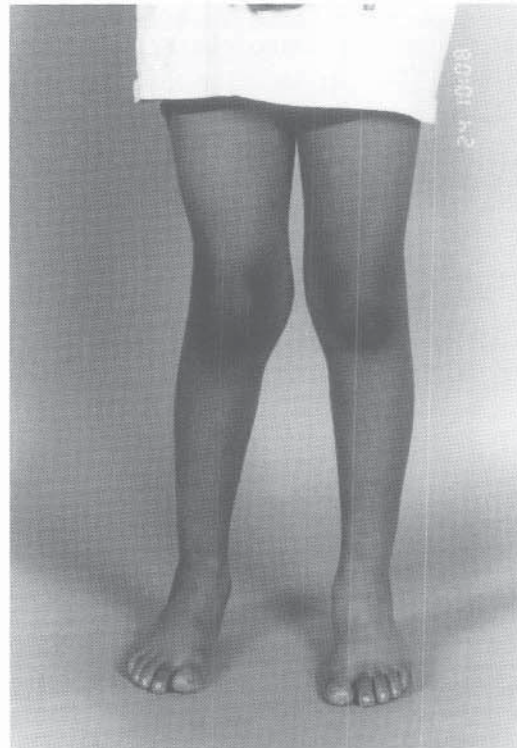
B



C



D



E

FIGURE 39-15 Physeal arrest producing angular deformity. **A**, Salter-Harris type II fracture of the distal femur. **B**, Immediate postreduction film. **C**, AP radiograph 9 months after injury. The distance between the physis and the screw medially (*A*) is substantially greater than it was immediately postoperatively. However, the distance laterally (*B*) is relatively unchanged. Note the radiodense appearance of the physis laterally. **D**, CT scan demonstrating lateral bar formation. **E**, The asymmetric growth has produced a valgus clinical appearance.

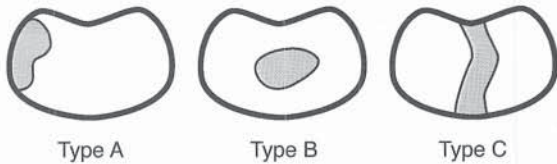


FIGURE 39-16 Classification of partial physal arrest. Type A—peripheral; type B—central, surrounded by normal physis; type C—central, traversing the physis completely.

on their location within the physis (Fig. 39-16). There are two types of central bars. The first, type B, is surrounded by a perimeter of healthy physis. This type of bar may produce a tethering effect that “tents” the epiphysis and produces a joint deformity. In the second type of central bar, type C, the bar traverses the entire physis from front to back (or side to side). The physis on either side of the bar is normal. This pattern is commonly seen with injuries to the medial malleolus.^{27,200}

Once the extent and location of the bar have been defined, the amount of growth remaining from the physis must be determined. This can be accomplished by determining the skeletal age of the patient and using information on growth patterns assembled by Green and Anderson.^{11-13,89,90} Skeletal age can be determined by comparing a radiograph of the left hand and wrist with standards in an atlas of skeletal age.⁹² It is generally assumed that girls grow until a bone

TABLE 39-2 Yearly Growth of Various Long Bone Physes

Location	Yearly Growth (mm/yr)
Proximal femur	2
Distal femur	9
Proximal tibia	6
Distal tibia	4
Proximal humerus	12
Distal radius	8

age of 14 and boys until a bone age of 16.^{21,180,213,269} Future growth for the distal femur and proximal tibia can be estimated using the graphs initially published by Anderson and colleagues (Fig. 39-17) or by using approximations of yearly physal growth (Table 39-2).^{11,13,89,90,135,180}

Treatment options for physal arrests include observation, completion of a partial arrest, or physal bar resection. If the bar appears to involve the entire physis and there is an acceptable existing limb length inequality or angular deformity and little contralateral growth remaining, observation may be the best option. Completion of a physal bar may be indicated if there is an acceptable existing angular deformity that might become clinically unacceptable if untreated. With completion of an arrest, the surgeon must evaluate the likelihood of a subsequent limb length inequality

GROWTH REMAINING IN NORMAL DISTAL FEMUR AND PROXIMAL TIBIA FOLLOWING CONSECUTIVE SKELETAL AGE LEVELS

MEANS AND STANDARD DEVIATIONS DERIVED FROM LONGITUDINAL SERIES 50 GIRLS AND 50 BOYS

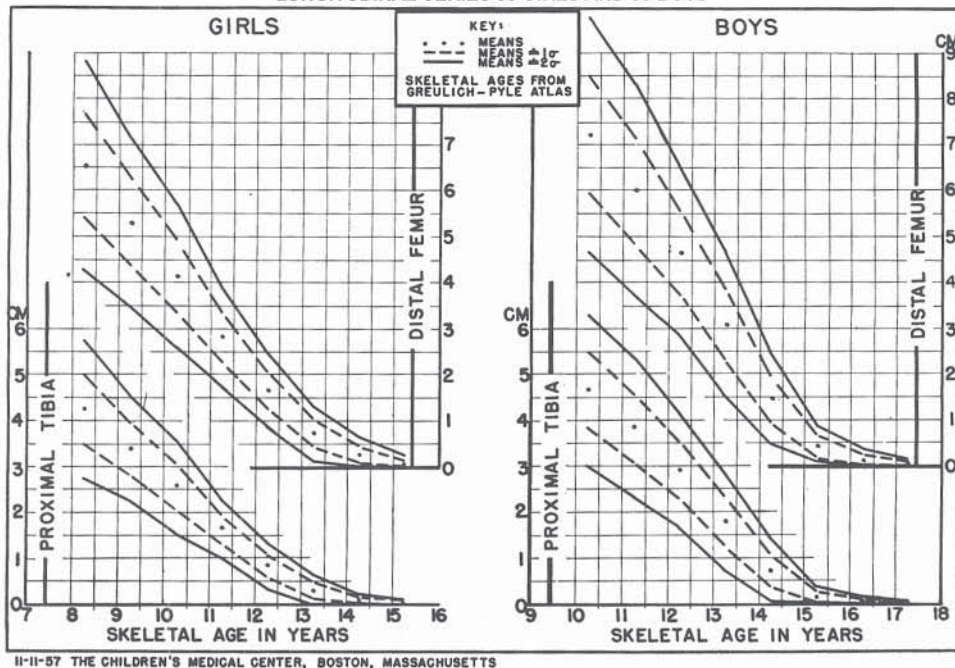


FIGURE 39-17 The Green-Anderson growth remaining chart. This chart can be used to estimate the growth remaining at the normal distal femur and proximal tibia at the skeletal ages indicated. (From Anderson M, Green WT, Messner MB: Growth and predictions of growth in the lower extremities. J Bone Joint Surg 1963;45-A:10.)

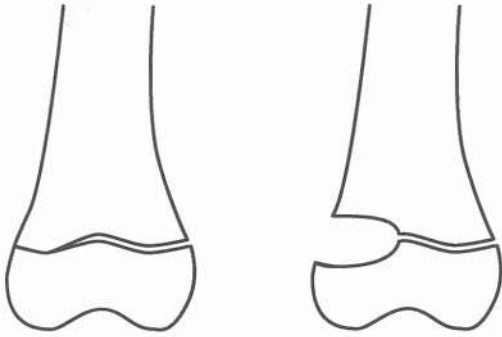


FIGURE 39–18 Schematic representation of peripheral bar resection. The bar should be excised until the cavity is rimmed with normal physis.

ity. If the likelihood of significant limb length inequality (more than 20 to 25 mm) is high, a contralateral epiphysiodesis should be performed at the time of completion of the partial arrest. Resection of a physal bar is indicated for partial arrests with substantial growth remaining.

Physal bar resection was first introduced by Langenskiöld and has been studied in both human and animal models.^{27,36,135,153–156,166} The technique of bar excision involves removing the bone bridging the metaphysis and epiphysis

at least 1 year of growth remaining, while Kasser has stated that successful bar resection requires at least 2.5 cm of growth and Birch has recommended at least 2 years of growth. Clearly, the younger the patient and the more the potential growth from the physis, the greater the benefit of a successful resection.^{26,135,154}

The surgeon must decide whether an osteotomy is necessary to correct existing angular deformity. Angular deformities of less than 20 degrees may correct spontaneously following bar resection and can be observed. However, if a deformity of greater than 20 degrees is present, an osteotomy to achieve angular correction should be performed at the time of bar resection.^{26,135,200,207}

When a peripheral (type A) bar is resected, the bar should be approached directly and removed under direct vision with a wide margin of periosteum (Fig. 39–18). The bar should be resected until the cavity is rimmed completely with normal physis. Type B and C bars are approached through a window in the metaphysis or through an osteotomy. Although some have advocated magnification with loupes or a microscope, we have not routinely used these aids. However, resection of central bars can be facilitated by the use of fluoroscopy, fiberoptic lighting, and dental mirrors (Fig. 39–19).

Once the bar has been completely resected, the cavity created can be filled with fat or Cranioplast. Silicone (Silas-

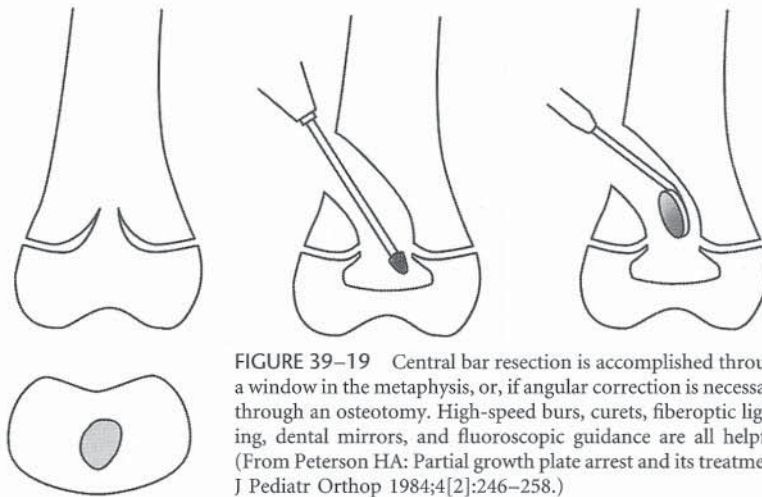


FIGURE 39–19 Central bar resection is accomplished through a window in the metaphysis, or, if angular correction is necessary, through an osteotomy. High-speed burs, curets, fiberoptic lighting, dental mirrors, and fluoroscopic guidance are all helpful. (From Peterson HA: Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4[2]:246–258.)

and filling the void created with an interposition material that will prevent reformation of the bony bar. The remaining physis must be undamaged and large enough that growth is likely to continue. In addition, there should be a significant amount of growth remaining before skeletal maturity and physiologic physal closure. Numerous studies have documented that bars involving more than 50 percent of the physis are unlikely to respond to bar resection.* Recommendations regarding requirements of amount of growth remaining are less uniform. Langenskiöld has recommended

tic) has been utilized experimentally in both humans and animals, but is currently unavailable for use.³⁶ Each of these interposition materials has advantages and advocates.^{26,135,202,207} Fat is commonly used because it is readily available and autogenous. Its only drawback is that a separate incision in the gluteal area is often required to harvest a graft of adequate size. Methylmethacrylate, available commercially as Cranioplast, is radiolucent and thermally nonconductive. Its solid structure may help support an epiphysis if a large metaphyseal defect has been created. Regardless of which interposition material is selected, the goal is to pack the defect with the material so that bar formation is

*See references 26, 135, 154, 156, 157, 202, 207.

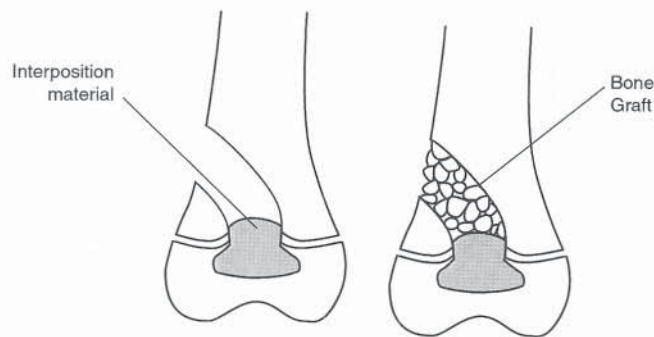


FIGURE 39–20 Once a bar has been successfully resected, the void in the epiphysis and metaphysis should be filled with fat or Cranioplast. It is helpful to contour and anchor the material into the epiphysis so that it will migrate distally with the physis with growth. The metaphyseal defect can be backed with local bone graft. (From Peterson HA: Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4[2]:246–258.)

prevented. Ideally, the interposition material should migrate with the epiphysis. The remaining metaphyseal defect can be packed with the removed bone. Peterson has advocated contouring the epiphyseal defect or creating drill holes or “pods” in the epiphysis to anchor the interposition material in the epiphysis so that the interposition material will migrate distally with the epiphysis as growth resumes (Fig. 39–20).²⁰⁷ Once the bar has been resected and the interposition material has been placed, radiographic markers should be placed on either side of the physis to aid in evaluating resumption of growth (Fig. 39–14).

Results following bar resection are variable. Nearly all authors report poor results with bars involving more than 50 percent of the physis.^{26,135,202,207} Peterson reported results as a percentage of growth of the normal (contralateral) physis ranging from 0 to 200 percent, with a mean of 84 percent.²⁰⁷ Our experience has been less dramatic, with clinically significant growth resuming in approximately 40 percent of cases.²⁶ The surgeon must remember that premature closure of the physis is to be expected, even if above-normal growth has resumed.^{26,135,200,207} Thus, while bar resection is a viable option for the young patient with a physeal arrest, close clinical follow-up to maturity is imperative.

Care of the Multiply Injured Child

Blunt trauma is the leading cause of death in children over 1 year old. Although a number of these deaths are from such massive injuries that there is no chance of resuscitation, there are deaths that could be prevented with proper trauma care.^{45,68,70,91} Although most preventable deaths are the result of pulmonary, intracranial, or intra-abdominal pathology, it is important for all physicians, including orthopaedists, caring for victims of acute trauma to be thoroughly familiar with the systematic, multidisciplinary approach to the assessment and resuscitation of the polytraumatized child. The principles of assessment and resuscitation are outlined and well presented in the Advanced Trauma Life Support course provided by the American College of Surgeons. This comprehensive course provides specific training for the management of the pediatric trauma patient.²⁴⁶

Children possess a number of anatomic and physiologic characteristics that make their injuries and their injury response different from adults'. Head and visceral injuries are more common in children, while chest and thorax injuries are less frequent. Several factors contribute to the fact that head injuries occur in over 80 percent of polytraumatized children. First, because a child's head is relatively large compared to the trunk, the head is usually the point of first contact during high-energy injuries. Second, the cortical bone of the cranial vault is thinner in children. Finally, a child's brain is less myelinated than an adult's and more easily injured. Fortunately, there are also several characteristics that make recovery from head injury more favorable in children. These include a larger subarachnoid space, greater extracellular space, and open cranial sutures.* Visceral injuries are also more common in children than in adults, in part because there is less abdominal musculature and less subcutaneous fat. Conversely, the elasticity of the thoracic cage makes fractures of the ribs and sternum uncommon in children.^{102,143,159,163,192,246}

A child's response to injury is also different from an adult's. It is unusual for children to have preexisting disease, and they usually have large cardiopulmonary reserves. Consequently they can often maintain a normal systolic blood pressure in the face of significant hypovolemia, although they will develop tachycardia. Children also become hypothermic rapidly because their surface area is large relative to their body mass. This hypothermia can compound the lactic acidosis associated with hypovolemic shock.

Evaluation and resuscitation of the polytraumatized child begins with the ABC's (Airway, Breathing, Circulation) of trauma. Management of the airway should begin with the assumption that cervical spine pathology exists, and cervical spine precautions should be used until the cervical spine is cleared clinically and radiographically. It is important to remember that the relatively large head of a child forces the cervical spine into flexion. Thus, appropriate immobilization includes a collar or sand bags, as well as a backboard that has a cutout for the head. If these special backboards are unavailable, children may be safely transported by placing a roll under the shoulders to elevate the torso relative to the head (Fig. 39–21).† With these cervical spine precautions, an adequate airway must be maintained. The jaw thrust or lift will often open the airway. It is also important to remember that the nostrils must be kept clear in infants. All obvious foreign materials (food, mucus, blood, vomit) must be removed from the mouth and oropharynx. Placement of a nasogastric tube will decompress the stomach and help prevent aspiration. In the unconscious or obtunded child, endotracheal intubation ensures a secure airway.¹⁹¹

Once an adequate airway has been obtained, breathing and circulation should be assessed. Ventilation should be confirmed by auscultating breath sounds in both lung fields. Absence or decreased breath sounds should alert the surgeon to the possibility of an improperly placed airway or a potential pneumothorax. Assessment of blood volume status in children can be deceptive, owing to their large physiologic reserves. Although children often maintain a normal blood pressure despite significant volume loss, tachycardia will

*See references 37, 102, 137, 143, 159, 163, 230, 246, 247, 264.

†See references 60, 107, 114, 120, 121, 130, 140, 151, 163, 246, 252.

develop early in hypovolemic shock. Life-threatening hemorrhage in children is usually the result of solid visceral injury, as children are less likely than adults to sustain massive blood loss from pelvic or extremity trauma.^{49,127} As assessment and management of the airway, breathing, and circulation is being undertaken, attempts should be made to obtain venous access. Once venous access has been established, fluid resuscitation can begin. A child's circulating blood volume can be estimated as 80 mL/kg. A child's weight in kilograms can be estimated as:

$$\text{weight (kg)} = [\text{Age (yr)} \times 2] + 8.$$

As in adults, fluid resuscitation begins with a crystalloid bolus equal to one-fourth of the circulating blood volume (20 mL/kg). If tachycardia or other signs of hypovolemia persist after two crystalloid boluses, consideration should be given to transfusion of packed red blood cells. Once fluid resuscitation has begun, the bladder should be decompressed with a Foley catheter. Urine output can then be monitored. Normal urine output in an infant is 1 to 2 mL/kg/hr and in a child or adolescent 0.5 mL/kg/hr.^{163,246}

The primary survey is completed with a quick history, which should include assessment for medical allergies, current medications, significant past medical history, and the details of the accident and management to date. As the primary survey is completed, the secondary survey begins. The secondary survey includes calculation of the Glasgow Coma Scale (GCS) score (Table 39–3) and radiographs of the chest (AP), cervical spine (lateral), and pelvis (AP).^{246,247} Additional studies (CT of the head and abdomen, radiography of the extremities and thoracolumbar spine) should be

TABLE 39–3 Glasgow Coma Scale²⁴⁷

Variable	Score
Opening of the eyes	
Spontaneously	4
To speech	3
To pain	2
None	1
Best verbal response	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Children's best verbal response ¹⁰²	
Smiles, orients to sound, follow objects, interacts	5
Consolable when crying, interacts inappropriately	4
Inconsistently consolable, moans inconsolably, irritable, restless	3
No response	2
None	1
Best motor response	
Spontaneous (obedience to commands)	6
Localization of pain	5
Withdrawal	4
Abnormal flexion to pain	3
Abnormal extension to pain	2
None	1

Modified from Armstrong PF, Smith JT: Initial management of the multiply injured child. In Letts RM (ed): Management of Pediatric Fractures, p 32. New York, Churchill Livingstone, 1994.

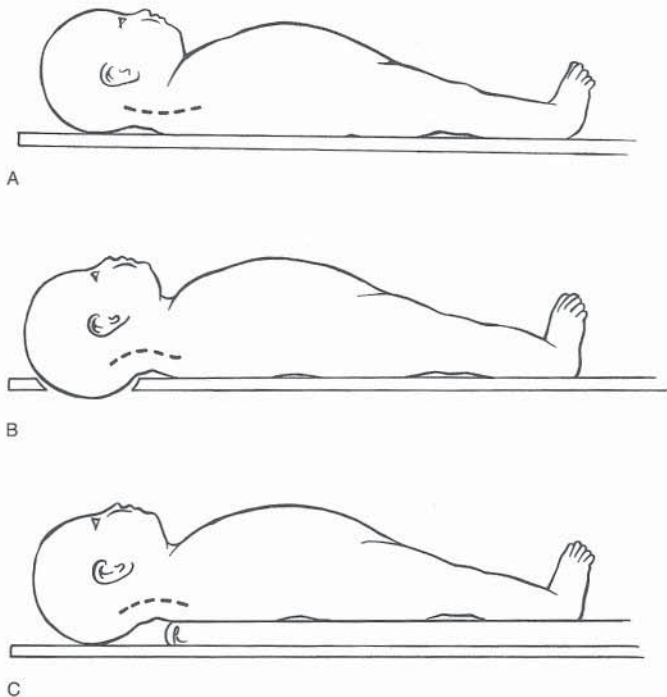


FIGURE 39–21 A, When placed on an adult backboard, the relatively large head of a child forces the cervical spine into flexion. B and C, Cervical flexion may be avoided either by using a backboard with a cutout for the head (B) or by elevating the torso (C).

performed as indicated. We obtain AP and lateral radiographs of any extremity that is painful, swollen, ecchymotic, or abraded. Routine blood work should include a complete blood cell count as well as a typing and crossmatching. It is prudent to draw ample extra blood at the time venous access is established so that appropriate tests may be added as indicated. The secondary assessment also provides an opportunity to gather information that will allow the computation of an injury score that can be used to classify injury severity and to predict morbidity and mortality. A number of scoring systems are available, including the Injury Severity Scale (ISS) the Abbreviated Injury Scale, the pediatric trauma score, the trauma score, and the revised trauma score.* The revised trauma score is not specific for children; however, it has the advantage of being universally applicable and has been shown to correlate with survival and with the ISS score as well as with the more specific pediatric trauma score (Table 39–4).^{73,83} The ISS score is used primarily for injury classification and outcomes research, but also as a measure of quality assurance. It has not been shown to have a direct correlation with mortality.†

It is important to stress that management of the traumatized child is a multidisciplinary process. As the secondary survey begins, continuous monitoring of airway, breathing, and circulation must continue. Deterioration of vital signs or GCS score may warrant emergency consultation with a neurosurgeon or a trauma surgeon. CT of the head is perhaps the single most important study in the management of intracranial trauma. Often, an abdominal CT may be

*See references 15, 19, 73, 83, 136, 194, 225, 249.

†See references 10, 15, 18, 19, 51, 73, 136, 194, 249.

TABLE 39-4 Revised Trauma Score

Revised Trauma Score	Glasgow Coma Scale	Systolic Blood Pressure (mm Hg)	Respiratory Rate (breaths/min)
4	13-15	>89	10-29
3	9-12	76-89	>29
2	6-8	50-75	6-9
1	4-5	1-49	1-5
0	3	0	0

Each of the three variables is scored (GCS, BP, RR). The scores are totaled (range: 0-12). A total score ≥ 11 indicates potentially important trauma.

From Armstrong PF, Smith JT: Initial management of the multiply injured child. In Letts RM (ed): Management of Pediatric Fractures, p 34. New York, Churchill Livingstone, 1994. Originally from American Hospital and Prehospital Resources for Optimal Care of the Injured Patient and Appendices F and J. Chicago, American College of Surgeons, 1986.

performed at the same time with little or no delay. In today's increasingly specialized environment, the orthopaedist may become involved in the care of a multiply injured child after the initial assessment has begun. One of the advantages of a multidisciplinary approach is that it has a built-in system of checks and balances. Thus, the prudent orthopaedist will *never assume* that the initial assessment has been completed accurately and thoroughly and will begin the assessment with the ABC's and progress to the assessment of any orthopaedic injuries.

Perhaps the two greatest mistakes an orthopaedic surgeon can make in managing a traumatically injured child is to assume that a long bone fracture is an isolated injury and to assume that a patient has an unsurvivable injury. At our acute care institution, we routinely consult a trauma surgeon for patients with isolated long bone fractures and a high-energy mechanism of injury (such as pedestrians or bicyclists hit by automobiles). Additionally, we aggressively treat all children with the expectation that they will recover from even the most severe head injuries. It is important to remember that the secondary survey continues 24 and 48 hours after the injury. Continuous reassessment will help identify the "missed injuries" that are noted in 2 to 12 percent of polytraumatized patients.^{34,150,182} Unlike in adults, early mobilization is not as important in the management of orthopaedic injuries in a polytraumatized child.^{31,82,148,212,282} Nevertheless, orthopaedic injuries should be managed in a fashion that accommodates the needs of all members of the trauma team.

Open Fractures

Although the incidence and mechanism of open fractures differ somewhat between children and adults, their management in these two populations is similar, requiring an aggressive, thorough, and systematic approach (Table 39-5). The most common open fractures in children involve the hand and upper extremity. The majority of these injuries are the result of falls.^{101,229} Open fractures of the lower extremities, particularly the tibia, are usually the result of

TABLE 39-5 Open Fracture Management

1. Thorough assessment for life-threatening injuries
2. Immediate IV antibiotics, continue for 48 hours:
 - Grade I—first-generation cephalosporin
 - Grade II and III—first-generation cephalosporin + aminoglycoside
 - "Barnyard" injuries—add anaerobic coverage (penicillin or Flagyl)
3. Tetanus prophylaxis
4. Thorough operative debridement
5. Adequate fracture stabilization
6. Second operative debridement in 48-72 hours *if indicated*
7. Early definitive soft tissue coverage
8. Early bone grafting *if indicated*

higher-energy trauma, most commonly trauma sustained in auto-pedestrian or auto-bicycle accidents.^{39,59,93,126,221,240} Although recent reports have highlighted the problems of inter- and intraobserver reliability, the classification system of Gustilo and Anderson is still the one most widely used for classifying open fractures in both children and adults (Table 39-6).^{38,95,96,98,99,119}

The treatment of open fractures begins in the emergency room with a complete and thorough assessment in order to identify any life-threatening injuries (see previous discussion under Care of the Multiply Injured Child). Once an open fracture has been identified, intravenous (IV) antibiotics should be administered. In a review of over 1,100 open fractures, Patzakis and Wilkins found the timely administration of IV antibiotics to be the single most important factor in reducing the infection rate.²⁰⁶ We currently use a first-generation cephalosporin for all open fractures. For grade II and III open fractures we generally add gram-negative

TABLE 39-6 Open Fracture Classification

Type I	Wound <1 cm long Moderately clean puncture wound Usually "inside-out" injury Little soft tissue damage, no crushing Little comminution
Type II	Wound >1 cm long No extensive tissue damage Slight or moderate crush injury Moderate comminution and/or contamination
Type III	Extensive soft tissue damage to muscles, skin, and neurovascular structures and a high degree of contamination Three subtypes: <ul style="list-style-type: none"> A Adequate soft tissue coverage (includes high-energy segmental, comminuted fractures, regardless of normal size) B Local or free flap required for coverage C Arterial injury requiring repair

Modified from the following: Gustilo RB, Anderson JT: Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. *J Bone Joint Surg* 1976;58-A:453. 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. Gustilo RB, Merkow RL, Templeman D: The management of open fractures. *J Bone Joint Surg* 1990;72-A:299.

coverage with the addition of an aminoglycoside. For “barnyard” injuries we add anaerobic coverage with penicillin or Flagyl.⁹⁹ Additionally, the status of the patient’s tetanus immunization should be reviewed. The American College of Surgeons recommends a booster of tetanus toxoid to all patients with wounds unless they have completed immunization or received a “booster” in the past 5 years. Patients with “tetanus-prone” wounds (severe, neglected, or more than 24 hours old) should be given a booster unless it can be confirmed that they have received one in the past year. The decision to provide passive immunization with human tetanus immune globulin must be made on an individual basis. Passive immunization with human immune globulin should be considered in all patients with “tetanus prone” wounds who have not been immunized or whose immunization status cannot be confirmed.⁹

All open fractures are an operative emergency and require operative debridement as soon as the patient can be assessed and stabilized. At the time of debridement, all open wounds should be extended proximally and distally and all loose debris and nonviable tissue, including devascularized bone, should be removed. Both ends of the fracture should be visualized and debrided. After a systematic, circumferential, superficial to deep debridement, the wound should be thoroughly irrigated with 5 to 10 liters of saline.^{95,96,98,99,205,248,254}

Once the wound has been thoroughly debrided the fracture should be stabilized. Fracture stabilization reduces the rate of infection by protecting the integrity of the soft tissue envelope. In children, fracture stabilization can frequently be accomplished with cast immobilization, often supplemented with percutaneous pin fixation. If, however, there are large soft tissue wounds, internal or external fixation may be indicated.* Recommendations regarding fracture stabilization are discussed with each injury.

The necessity of a second debridement for all open fractures is controversial. Although routine re-debridement has been recommended,^{95,96,99} there are several large series in which open fractures in children were managed successfully with a single debridement and loose wound closure over a drain.^{59,93,126} We make the decision to perform a second debridement on an individual basis, based on the amount of contamination, soft tissue devitalization, and bony comminution present at the initial debridement. However, we would rather perform a few “unnecessary” second debridements than treat infected, delayed unions; consequently, we have a low threshold for recommending re-debridement.

If primary wound closure is not possible either initially or on a delayed basis, wound closure with skin grafts or soft tissue transfer should be accomplished as soon as a clean, stable wound can be achieved, preferably within 5 to 10 days.^{40,50,95,99} The use of rotational (gastrocnemius or soleus) flaps or free microvascular tissue transfer in children is well established and similar to the principles in adults.^{16,48,52,76,204,232} Once soft tissue closure has been achieved, attention can be directed at bony reconstruction. Fortunately, open fractures in children rarely go on to delayed or nonunion; consequently, such procedures are rarely indicated.† The management of bone loss is discussed with specific injuries.

GUNSHOT WOUNDS

In large urban settings in the United States, gunshot injuries in children are becoming increasingly common. Gunshot wounds may be classified as either high or low velocity. High-velocity gunshots usually produce extensive soft tissue damage, gross contamination, and comminuted fractures. These injuries should be treated as type III open fractures. Low-velocity gunshot wounds have little soft tissue injury or fracture comminution. Recently, several authors have reported the successful treatment of these injuries with local wound debridement and short-term IV or oral antibiotic therapy. It is important to realize that most of these studies have been performed in adults. However, the few reports specifically discussing gunshot wounds in children suggest that, as is often the case, children have a better prognosis than adults.*

LAWNMOWER INJURY

Lawnmower injuries are another unique subcategory of open fractures. Not surprisingly, most children injured by lawnmowers are bystanders rather than operators or even riders. Most reports note that 30 to 50 percent of patients require some level of amputation. The vortex of air that is created by the lawnmower and the inherently dirty setting produce massively contaminated wounds. Acute management of lawnmower injuries involves multiple thorough debridements. We routinely debride these wounds multiple times at 48-hour intervals, until there is no evidence of debris and there is a healthy granulation bed. In addition to thorough debridement, initial management should include broad-spectrum antibiotics, including coverage for potential anaerobic infection. If amputation is required, every effort is made to keep the level as distal as possible. Consideration should be given to using the amputated parts to provide cartilaginous caps over any exposed residual bone in the hope of preventing appositional overgrowth of the residual limb. Like most traumatic injuries, many, if not all, lawnmower injuries are easily preventable. There are currently many educational efforts under way to ensure that operators of lawnmowers have adequate knowledge of the potential danger these machines represent not only to operators but to bystanders as well.†

Compartment Syndrome

Compartment syndrome is a potentially devastating entity that may develop when injury induces increased pressure within a closed space. Because the earliest signs of compartment syndrome are often subtle and the patients are frequently obtunded or difficult to assess for other reasons, the diagnosis may be delayed or altogether missed, resulting in devastating complications that may be avoided with prompt surgical decompression.‡

Eaton and associates have outlined the pathophysiology of compartment syndrome.⁷² Initially, ischemia produces

*See references 39, 101, 126, 148, 221, 240, 251, 279.

†See references 39, 59, 93, 101, 126, 148, 221, 240.

*See references 67, 75, 116, 122, 144, 160, 185, 195, 243, 245, 259, 267.

†See references 8, 14, 23, 24, 69, 146, 165, 167, 189, 263.

‡See references 105, 111, 171, 176, 186–188, 250, 253, 262.

anoxia in muscles, which in turn causes release of histamine-like substances, which increases capillary permeability and leads to intramuscular edema. The increasing intramuscular edema produces a progressive increase in the intrinsic tissue pressure of the muscles. A taut fascial envelope creates venous compression, which further increases the intramuscular intrinsic pressure. Unyielding circular dressings on the limb can also contribute to increases in the intramuscular pressure. Pressor receptors within the muscle produce vaso-spasm, which aggravates the initial vascular compromise, creating a destructive ischemia-edema cycle. The only treatment for this potentially devastating cycle is prompt wide surgical decompression of the fascial compartment.* Compartment syndrome can be recognized by the so-called six P's: pain out of proportion to physical examination findings, increased pressure, pink skin color, pulse present, paresthesias, and paresis. However, it is important to remember that the only early sign may be pain, particularly pain on passive stretching.^{134,171,274} In fact, paresis and paresthesias are late findings, often present only after permanent damage has occurred.

The best treatment of a compartment syndrome is avoidance. However, once it has developed it must be promptly recognized and treated. Although compartment syndrome can occur within any compartment after accidental injury, following elective surgical procedures, or with infections, in children it is most commonly seen following fractures of the supracondylar humerus or tibia.† Appropriate management of "at-risk" extremities may help prevent compartment syndrome. Elevation of affected extremities is recommended immediately after an injury to decrease soft tissue swelling. Although elevation decreases edema, it also decreases arterial blood flow and reduces oxygen perfusion by reducing the arteriovenous gradient.^{105,171,172,175,177,280} Thus, if an involving compartment syndrome is suspected, the limb should be kept at the level of the heart rather than elevated. It is important to remember that circumferential dressings can cause an elevation in compartment pressures, which can accelerate the development of ischemia and the spiraling increase in edema and pressure.^{64,111,187,268} Removal of circumferential dressings has been shown to reduce compartment pressure by as much as 85 percent. Therefore, once a compartment syndrome is suspected, all circumferential dressings should be removed *to the skin*.^{25,86}

Diagnosis of an acute compartment syndrome can be aided by measurement of the pressure within the compartment. Numerous techniques have been described to measure intracompartmental pressure. One of the earliest was the Whitesides needle technique. This technique employs an 18-gauge needle, a syringe, IV tubing, sterile saline, a three-way stopcock, and a mercury manometer (Fig. 39–22). The needle is placed into the compartment and the plunger is advanced until the fluid column begins to enter the compartment. The pressure reading on the manometer at this point represents the compartment pressure. Other techniques have been developed to allow continuous monitoring of compartment pressures or to simplify pressure measurement. These include the wick or slit catheter technique, the infusion

technique, commercially available gauges, and an IV catheter with an infusion pump or arterial line pressure monitor.^{186,188,257} Wick and slit catheters were developed because of theoretical concerns that the injection technique created nonequilibrium conditions at the tip of the catheter and overestimated the compartment pressure.^{186,188,224} Recently, Wilson and colleagues have shown slit catheters and 16-gauge IV catheters produce similar compartment pressure measurements.²⁷⁵ Uppal and colleagues described a technique utilizing an 18-gauge needle and an IV alarm control (IVAC) pump. After zeroing the IVAC pump and adjusting the unit to read in mm Hg rather than mm H₂O, the fluid flow rate is set at 25 mm/hr. An 18-gauge needle is then introduced into the compartment and the "read pressure" button is depressed. The compartmental pressure is displayed on the IVAC pump.²⁵⁷ Similarly, a needle or angiocath (with or without a side port) can be connected to an arterial line monitor. After zeroing the monitor (which requires a small fluid bolus), the pressure is displayed on the arterial line monitor (Fig. 39–22). This technique can be used with a slit indwelling catheter to provide continuous pressure monitoring.

The intracompartmental pressure at which a compartment syndrome exists is unknown, and the pressure may vary with the technique of measurement.^{184,231,275} Whitesides and colleagues recommended surgical decompression when compartment pressure rose to within 10 to 30 mm Hg of the diastolic pressure using the needle technique.^{270,271} Matsen has recommended decompression at pressures of 45 mm Hg using the infusion technique, while Mubarak and Rorabeck have recommended decompression at 30 to 35 mm Hg using the wick or slit catheter.^{35,173,174,186,224} These thresholds for pressure measurements are only guidelines, however, and decisions regarding fasciotomy must be made in the context of the entire clinical setting, taking into account the patient's blood pressure, local perfusion, trends of intracompartmental pressures, and symptoms, as well as the patient's ability to cooperate with repeated examinations. It is also important to remember that compartment syndrome is a dynamic entity and that the at-risk extremity must be continuously reassessed.

Although the specific surgical technique for fasciotomy depends on the anatomic location, a few general points merit discussion. When treating compartment syndrome of the leg, it is important that all four compartments be widely released. We prefer to do this with a two-incision technique. The anterior and lateral compartments are released through a lateral incision that extends proximally to the origin of these muscles in the leg. The superficial and deep posterior compartments are addressed through a medial incision that extends distally to allow release of the entire deep posterior compartment (Fig. 39–23). In treating compartment syndrome of the forearm, we utilize the volar approach of Henry (Fig. 39–24). It is important to remember that there is both a superficial and deep compartment to the forearm and that the deep compartment, consisting of the flexor profundi, the flexor pollicis longus, and the pronator quadratus, is more susceptible to developing compartment syndrome.²²³ Once fasciotomy has been performed, Eaton and Green recommend careful assessment of each individual muscle. If the epimysium is a constricting, compressive structure, they recommend episiot-

*See references 105, 134, 171, 172, 175–177, 186.

†See references 20, 39, 58, 59, 101, 104, 111–113, 126, 131, 170, 176, 187, 221, 222, 234, 242.

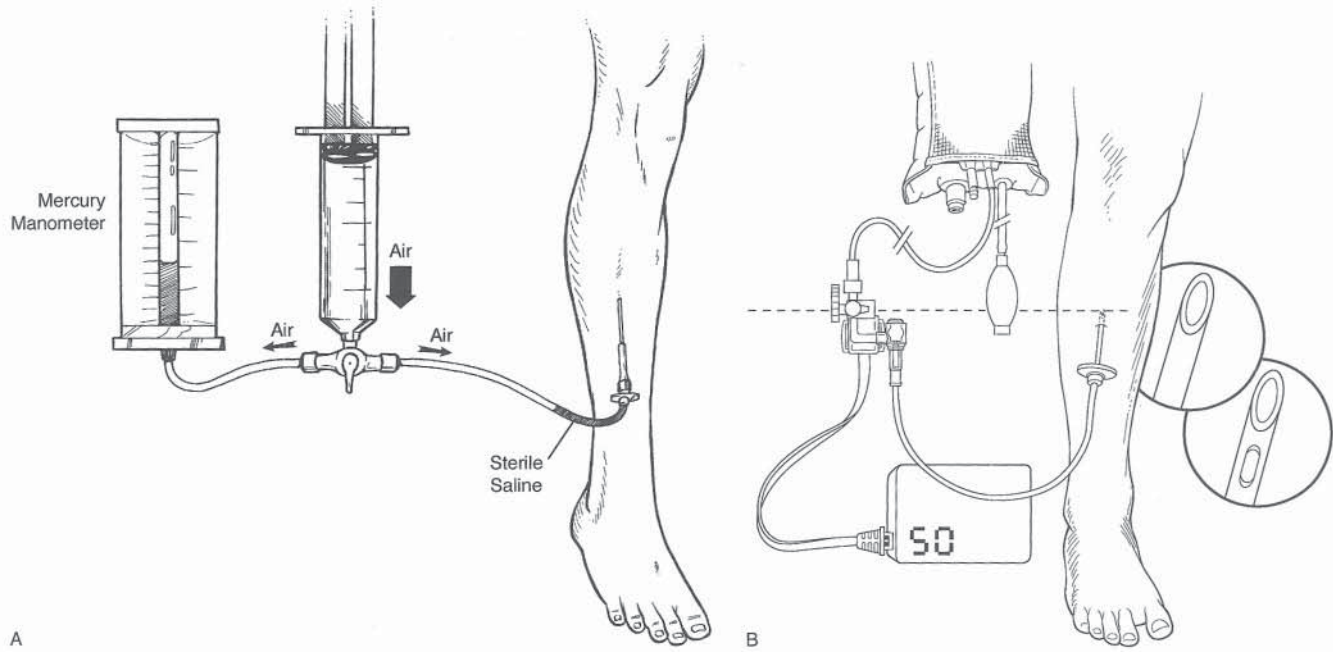


FIGURE 39-22 A, Whiteside's needle technique for measuring compartment pressures (see text for technique). B, Arterial line technique for compartment pressure measurement (see text for technique).

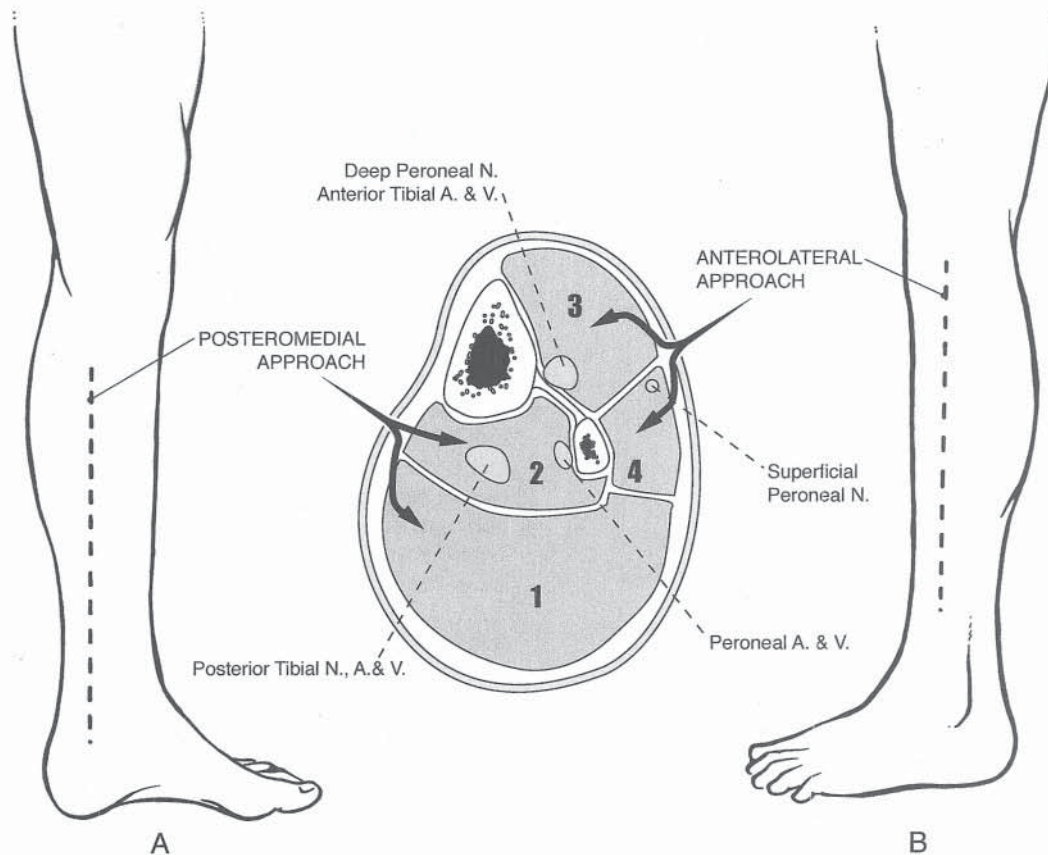


FIGURE 39-23 Two-incision technique for four-compartment fasciotomy of the leg. A, Posterior medial incision for decompression of the superficial and deep posterior compartments. This incision must extend far enough distally to allow complete decompression of the entire deep posterior compartment. B, Anterolateral incision for release of the anterior and lateral compartments. Care should be taken to identify and protect the superficial peroneal nerve. This incision must extend far enough proximally to ensure complete decompression of the muscles near their origin.

omy.⁷¹ Similarly, Rorabeck recommends external neurolysis if indicated.²²³ Following decompression, the initial findings may be quite mild. However, massive swelling is the rule following fascial release; thus it is wise to utilize generous incisions that allow full and complete release of the fascia.

Once wide surgical decompression has been achieved, all untreated fractures should be stabilized in a fashion that will allow appropriate treatment of the soft tissue wounds. The condition of the underlying muscle is then assessed. Initially ischemic muscle may respond favorably to decompression; thus, all nonviable tissue should be removed but any questionable tissue left alone. A sterile bulky dressing is applied to the extremity and the patient should be returned to the operating room at 48- to 72-hour intervals for continued debridement of nonviable tissue. Once a stable, healthy wound has been achieved, soft tissue closure can be performed either primarily or with split-thickness skin grafting, if necessary. After primary healing has occurred, reconstruction of any permanent deficits can be undertaken.

Although prompt recognition and early appropriate treatment of compartment syndrome can limit or avoid some of the potentially devastating problems, one should remember that as Sir Robert Jones noted, "It cannot be too emphatically stated that despite every precaution ischemic contractures may occur."²² Littler later echoed these comments, stating, "occasionally, despite professional awareness and all preventive effort, this distressing complication develops."²²

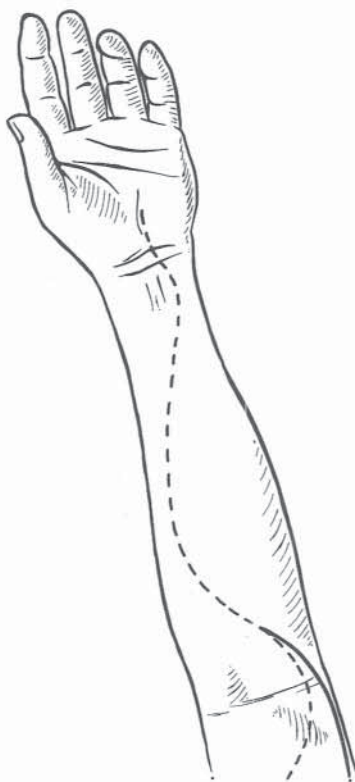


FIGURE 39-24 Skin incision for volar fasciotomy of the forearm. Distally, a skin flap is preserved to cover the median nerve. Proximally, the incision may be extended either medially or anterior laterally.

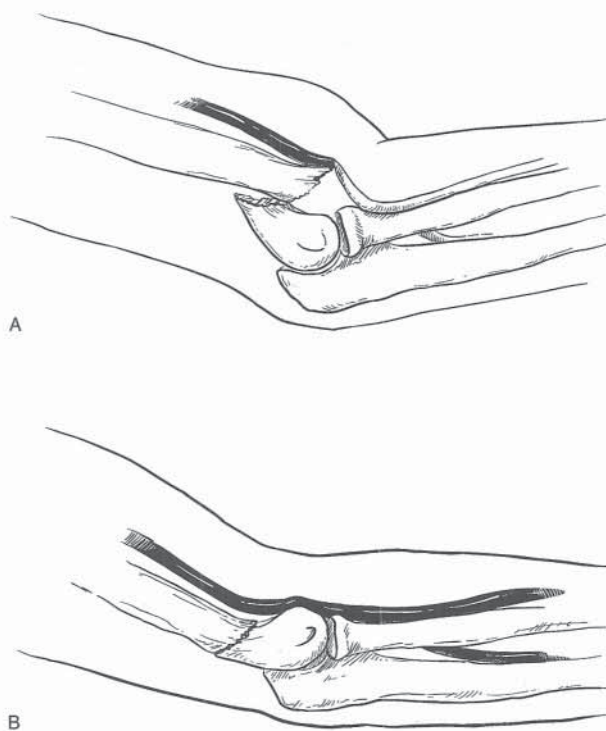


FIGURE 39-25 Emergency realignment of an ischemic extremity may reduce the tension on a vessel and restore the circulation.

Vascular Injuries

Vascular injury may result from severely displaced fractures. This is most commonly seen with extension supracondylar humerus fractures or with fractures of the distal femur or proximal tibia.* Patients who present with an ischemic limb and a fracture should undergo immediate "closed reduction" of the fracture in the emergency room. This closed reduction is actually a simple realignment of the limb, performed with gentle traction to restore the limb to a more anatomic position, thus removing any tension on the neurovascular structures (Fig. 39-25). If, as it frequently the case, realignment of the limb restores circulation to the extremity, fracture management can usually proceed in the normal fashion. However, if a nonviable limb persists after realignment, the patient should be taken immediately to the operating room for fracture stabilization and vascular exploration and, if indicated, repair. We believe that "preoperative" arteriography in an ischemic/nonviable extremity only prolongs the ischemic time and should not routinely be performed. We proceed immediately to the operating room and stabilize the fracture. Once the fracture has been stabilized vascular exploration can be accomplished. If necessary, fluoroscopy can be utilized to obtain an intraoperative arteriogram, although we find this is seldom necessary as the anatomic location of the vascular injury is usually obvious. Ideally, revascularization should be achieved within 6 to 8 hours. Prolonged ischemia and subsequent revascularization may be associated with the development of compartment syndrome. Subsequently, we have a low threshold for performing fasciotomies at the time of revascularization.†

*See references 39, 42, 53, 56, 81, 147, 203, 244, 258, 266, 283.

†See references 39, 42, 53, 56, 81, 147, 203, 244, 258, 266, 283.

The management of a viable limb with an absent pulse is controversial. This is often the case with a limb that was initially ischemic but improved with realignment. Some authors have advocated arteriography and/or exploration with appropriate vascular repair. Others have documented that a viable but pulseless extremity may be safely observed. We manage these patients in consultation with a trauma, vascular, or microsurgeon, with decisions made on an individual basis.^{56,81} However, we usually recommend a conservative course with close observation.^{47,233,273,283} The importance of an adequate period of close observation must be emphasized, as propagation of a thrombus can turn a pulseless, viable hand to an ischemic, nonviable hand. Pulse oximetry has been reported to be an effective continuous monitoring device in such situations.^{88,218} Although the ability of pulse oximetry to accurately reflect tissue oxygenation (a function of oxygen saturation *and* blood flow) has been questioned,²³⁷ we believe it is an effective adjunctant in monitoring an extremity for viability.

Casts

No discussion of the general principles of traumatic injuries in children would be complete without a discussion of the principles involved in good casting. With advances in orthopaedics, cast immobilization is increasingly less common. However, for a number of reasons, casts remain the mainstay of treatment for children's fractures and reconstructive pediatric orthopaedic surgery. Thus, the ability to apply a well-molded cast or splint is an important skill for the pediatric orthopaedic surgeon. Unfortunately, too often the task of reducing and splinting a fracture is delegated to the most junior member of the team, often with little instruction and no supervision. This may result in less than desirable outcomes, as even an undisplaced buckle fracture can angulate in a poorly applied splint or cast (see Fig. 39-3). It is increasingly common to hear comments regarding the "lost art of casting." Applying a well-molded cast or splint, particularly on a small, moving child with a chubby arm, is indeed an acquired skill.

A well-applied cast has only two layers of cast padding on all areas except bony prominences, which will require a third or fourth. A cast with too much padding will fail to hold a reduction, while one with too little may result in pressure sores. Once a cast has been applied it should be molded to provide three-point fixation of the fracture (Fig. 39-26).⁴⁶ When applying a cast, one should remember that the length on the convex side of an angle is significantly more than that on the concave side. Failure to account for this difference will result in too much material on the concave side or insufficient material on the convex side. Technically this problem may be addressed with the use of splints or by fanning the cast material out over the convex side. During cast application, attention must be given to the position of the entire extremity. Moving a joint once the padding and plaster have been applied will result in a crease, which can lead to soft tissue problems (Fig. 39-27). Similarly, applying a short-leg cast with the ankle in equinus or a long-arm cast with the elbow extended may allow the cast to shift distally, which can also lead to pressure sores (Fig. 39-28). There are clinical situations in which a cast in extension (e.g., supracondylar humerus fractures after pinning) or

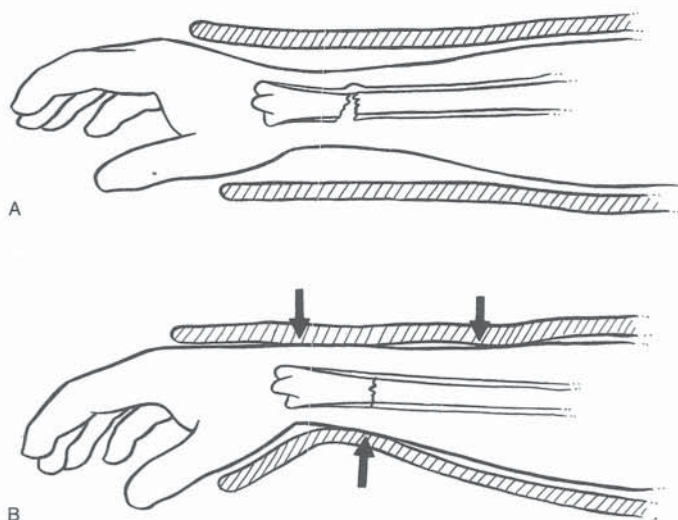


FIGURE 39-26 A, A cast with too much padding and an inadequate mold will not maintain a reduction. B, Proper casting technique provides three-point fixation of the fracture.

equinus (distal tibia fractures) is required. In these instances, careful molding of the cast around bony prominences will help prevent migration of the cast distally.

The Dutch military surgeon Antonius Mathijssen began impregnating open meshed bandages with plaster-of-Paris powder in 1852, and for over a century there were few fundamental changes in casting materials.⁹⁴ Recently, several new casting materials were introduced. These include fiberglass casting tape and Gore-Tex spica liners. Despite the considerable debate over the efficacy of these new materials, there has been little scientific experimentation with them, and the choice of materials remains primarily a subjective one. Proponents of fiberglass casts note that they are lighter and more durable. Others have argued that fiberglass is more difficult to mold and less forgiving when swelling is expected. In one of the few studies comparing casting material, Davids and co-workers demonstrated that a properly applied fiberglass cast produces less skin pressure than a plaster-of-Paris cast.⁶⁴ 3M has developed a fiberglass casting material (SCOTCHCAST) that is removed with simple unrolling. The ease of removal of this material has led to its widespread use in the treatment of clubfeet.⁵⁷

Child Abuse

One of the earliest descriptions of the orthopaedic manifestations of child abuse was by Caffey in 1946. He described six infants with femur fractures and chronic subdural hematomas.⁴¹ In 1953, Silverman²³⁵ implicated the parents and guardians in these traumatic lesions. In 1962, Kempe and colleagues introduced the phrase "battered child syndrome."¹³⁸ This paper brought multidisciplinary medical attention to the problem of child abuse and led to mandatory reporting laws, which now exist in all 50 states. Originally, child abuse was defined as physical injury inflicted on children by persons caring for them.¹⁰⁹ Since this early definition in 1968, the definition of abuse has expanded to include physical neglect and endangerment as well as emotional and sexual abuse.

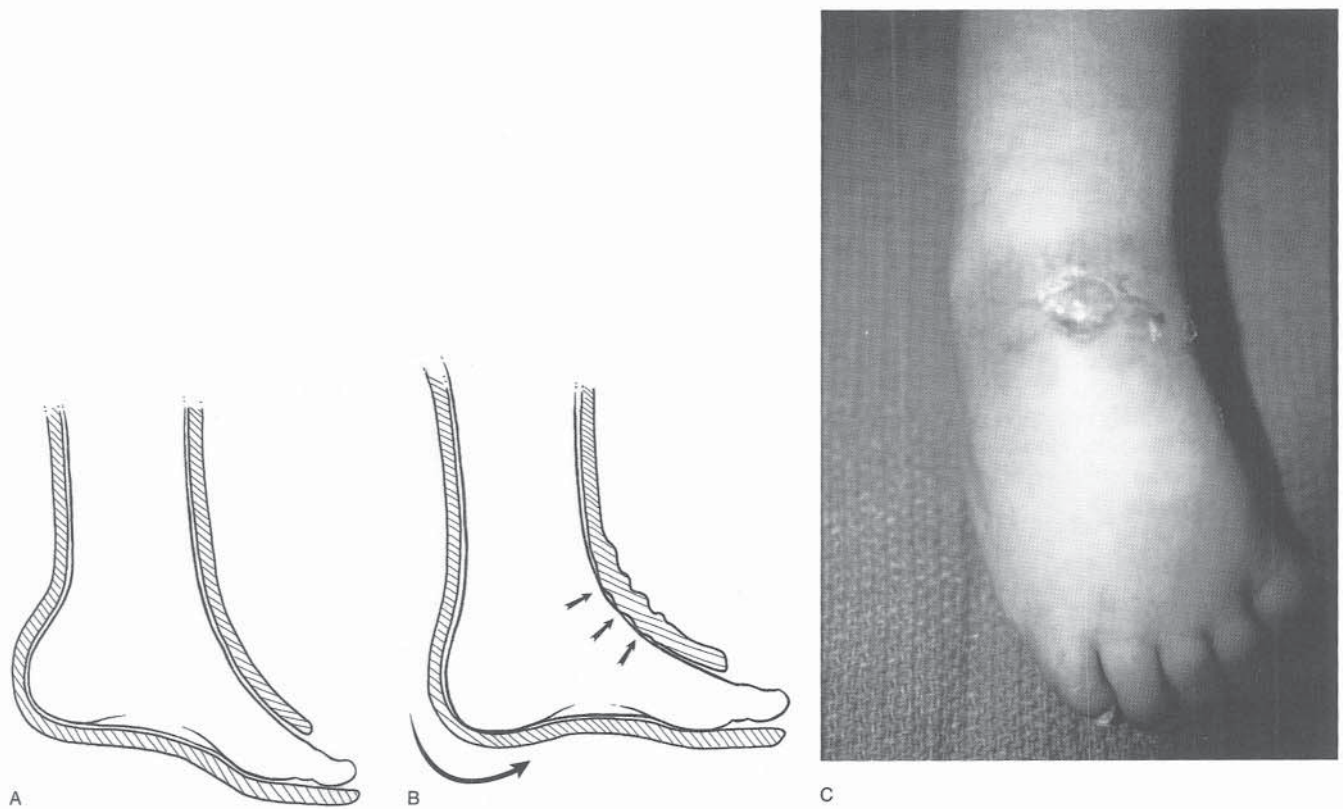


FIGURE 39-27 Once casting materials have been applied, a joint must not be moved. Moving the foot out of equinus creates creases in the cast, which can lead to skin breakdown.

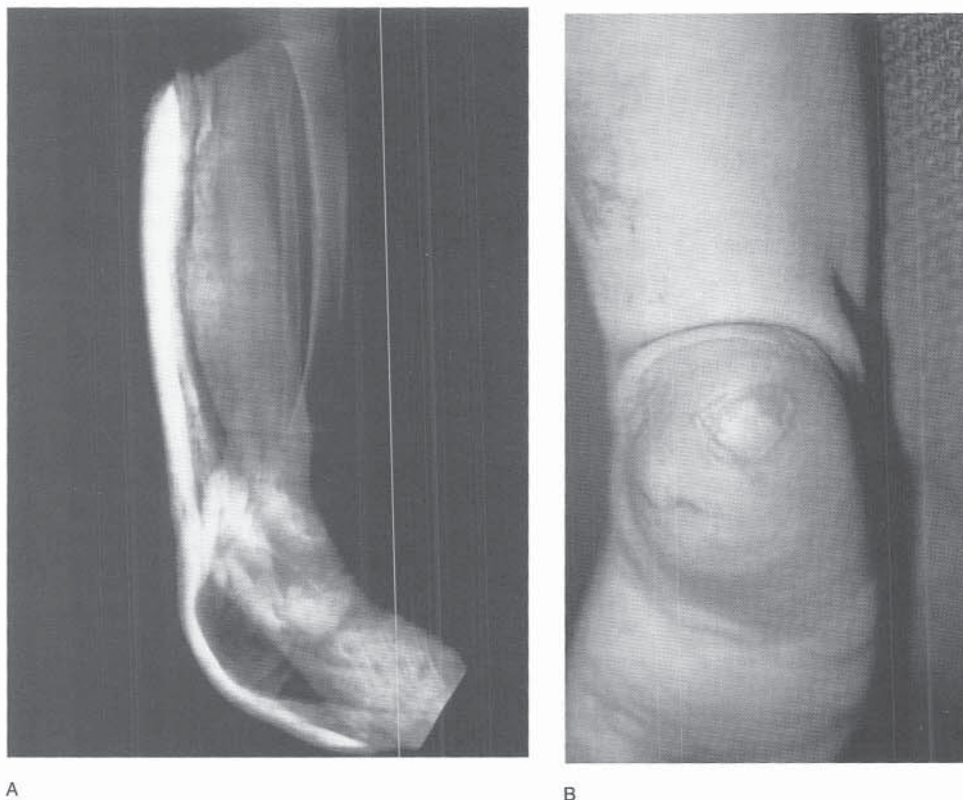


FIGURE 39-28 Pressure sores following distal migration of a splint. **A**, Lateral radiograph of a poorly molded posterior splint. The splint has slid distally and is impinging on the heel. **B**, When the splint is removed there is blistering on the heel.

The incidence of child abuse is difficult to determine. It has been estimated that 1 to 1.5 percent of all children are abused each year.²⁵⁶ In the United States in 1991, there were more than two million reports alleging maltreatment of more than three million children. These reports were substantiated in approximately one million children. Although this represents a nearly 20 percent increase from 1990, it is difficult to determine whether the increase represents an improvement in reporting or an actual increase in the number of abused children.²⁵⁶ Statistical analysis of reported cases shows that children are more likely to be abused by caregivers who are young, poor, and of minority status. However, abuse in affluent families may be underreported because of medical practitioners' desire to protect their social peers from the stigma of investigation by public agencies. Abuse is also less likely to be reported if it is emotional rather than physical and if the mother is the perpetrator. There is no doubt that child abuse is a problem that crosses all age, sex, ethnic, and socioeconomic groups.^{43,103}

Although children of any age can be abused, younger children are more frequently victims.²⁷⁸ Akbarnia and associates reported that 50 percent of 243 abused children were less than 1 year old and 78 percent were less than 3.⁵ Younger children are also more likely to die from abuse.^{43,193} In the United States, in 1996, 76 percent of the 1,077 fatalities from abuse were in children less than 4 years old.²⁵⁷

The diagnosis of abuse can be straightforward and obvious or frustratingly difficult. Regardless of the ease with which the diagnosis can be made, a high degree of suspicion is required in order to make the diagnosis. Child abuse has been found in up to half of all children with fractures in the first year of life and in one-third of children less than 3 years old with a fracture.^{118,145,178,278} A number of the "pathognomonic" signs of abuse are actually quite rare. The classic finding of "multiple fractures in different stages of healing" has been reported to be present in only 10 to 15 percent of documented cases of abuse.^{84,164} Similarly, corner fractures or bucket-handle metaphyseal fractures, are not as frequent as diaphyseal fractures. The importance of soft tissue injuries should not be overlooked. In fact, a number of reports have stressed the fact that fractures rarely exist without other signs of abuse and that abused children are more likely to have soft tissue injuries than fractures.^{84,179,181} It is important to consider, identify, and report neglect and endangerment. There may be no question of intentional injury when a toddler is brought in by paramedics after falling out of a three-story window; however, such a scenario suggests neglect or endangerment. Allowing a child to return to such an environment may be as dangerous as failing to report physical injury.

As in all areas of orthopaedics, there are few absolutes in child abuse. The best approach to child abuse is to maintain a high degree of vigilance by considering the diagnosis in all children with traumatic injuries. Certain factors, such as a changing history or a history not consistent with the injury, a delay in seeking treatment, long bone fractures in children less than 1 year old, multiple fractures in different stages of healing, corner fractures, rib fractures, skull fractures, thermal injuries, and unexplained soft tissue injuries should raise concern and trigger a report to the appropriate child protective agencies. Perhaps the most frequently overlooked part of the assessment of the abused child is the interview

with the child. Children, when time is taken to place them in a comfortable, secure, nonthreatening environment (characteristics that are difficult, if not impossible, to find in most busy emergency rooms!), will display remarkable candor. Despite mandatory reporting laws, physicians are often reluctant to report suspected abuse because of concern over upsetting the parents or caregivers. It has been our experience that when approached in a nonaccusational fashion with a simple explanation of the legal and ethical duty to report suspected abuse, parents are usually quite understanding of the physician's role. In fact, our suspicions are often heightened when a caregiver so counseled becomes indignant or threatening when informed of the necessity to report.

The consequences of failing to identify and report abuse are high. The reinjury rate of battered children is between 30 and 50 percent and the risk of death between 5 and 10 percent.^{6,29,84} If a reinjury occurs, it is likely that the caregivers will seek medical attention at a different medical facility. Because the risk of death increases with each subsequent emergency room visit,^{5,29} it is of paramount importance to report all cases of *suspected* abuse. However, simply reporting the incident may not ensure adequate safety for the child; hospitalization may be necessary to allow adequate assessment.

Most large urban children's hospitals have developed an interdisciplinary approach to the treatment of abused children. The "child abuse team" includes pediatricians, social workers, chaplains, and, when indicated, specialists such as orthopaedists. This approach streamlines what can be a cumbersome process as the parties involved develop an understanding of the legal issues and a repertoire with representatives from the legal system. At our acute care institution, this multidisciplinary approach, which utilizes mandatory parenting classes and other community resources, allows approximately 80 percent of abused children to remain safely in their home. Using a similar system, Galleno and Oppenheim demonstrated a decrease in the reinjury rate from 50 percent to 9 percent.⁸⁴

Summary

While managing skeletal injuries in children is generally straightforward, yielding excellent clinical results, there are times when even the simple can become difficult. In his book, *Children's Fractures*, Mercer Rang likens fracture management to a game of chess.²¹⁶ This classic discussion is full of tips and pearls of wisdom and is well worth the brief amount of time it takes to read. He outlines six principles of fracture care (which apply to all areas of pediatric orthopaedics) that are worth repeating:

1. Use your working knowledge of the various complications to look deliberately for them.
2. Children are uncooperative only when something is wrong.
3. Ensure your system of follow-up does not permit patients to be lost.
4. Recognize a loose cast.
5. Recognize the earliest signs of a displacing fracture.
6. Talk to the parents ("If parents are a nuisance, it is always your fault").

REFERENCES

1. Aitken A, Blackett CW, Ciaccotti JJ: Overgrowth of the shaft following fractures in childhood. *J Bone Joint Surg* 1939;21-A:334.
2. Aitken AP: The end results of the fractured distal radial epiphysis. *J Bone Joint Surg* 1935;17:302.
3. Aitken AP: The end results of the fractured distal tibial epiphysis. *J Bone Joint Surg* 1936;18:685.
4. Aitken AP: End results of fractures of the proximal humeral epiphysis. *J Bone Joint Surg* 1936;18:1036.
5. Akbarnia B, Torg JS, Kirkpatrick J, et al: Manifestations of the battered-child syndrome. *J Bone Joint Surg* 1974;56-A:1159.
6. Akbarnia BA, Akbarnia NO: The role of orthopedist in child abuse and neglect. *Orthop Clin North Am* 1976;7:733.
7. Albright JA, Brand RA: *The Scientific Basis of Orthopaedics*. New York, Appleton-Century-Crofts, 1979.
8. Alonso JE, Sanchez FL: Lawn mower injuries in children: a preventable impairment. *J Pediatr Orthop* 1995;15:83.
9. American College of Surgeons: A guide to prophylaxis against tetanus in wound management. *Bull Am Coll Surg* 1972;57:32.
10. American College of Surgeons: Hospital and Prehospital Resources for Optimal Care of the Injured Patient and Appendices F and J. Chicago, American College of Surgeons, 1986.
11. Anderson M, Green W, Messner M: Growth and predictions of growth in the lower extremities. *J Bone Joint Surg* 1963;45-A:1.
12. Anderson M, Green WT: Lengths of the femur and tibia: norms derived from orthoroentgenograms of children from five years of age until epiphyseal closure. *Am J Dis Child* 1948;75:279.
13. Anderson M, Messner M, Green W: Distribution of lengths of the normal femur and tibia in children from one to eighteen years of age. *J Bone Joint Surg* 1964;46-A:1197.
14. Anger DM, Ledbetter BR, Stasikelis PJ, et al: Injuries of the foot related to the use of lawn mowers. *J Bone Joint Surg* 1995;77-A:719.
15. Aprahamian C, Catey RP, Walker AP, et al: Pediatric trauma score: predictor of hospital resource use? *Arch Surg* 1990;125:1128.
16. Arnez ZM, Hanel DP: Free tissue transfer for reconstruction of traumatic limb injuries in children. *Microsurgery* 1991;12:207.
17. Aronson J, Tursky EA: External fixation of femur fractures in children. *J Pediatr Orthop* 1992;12:157.
18. Association for the Advancement of Automotive Medicine: The Abbreviated Injury Scale, 1990 revision. Des Plaines, IL, Association for the Advancement of Automotive Medicine, 1990.
19. Baker SP, O'Neill B, Haddon W Jr, et al: The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care. *J Trauma* 1974;14:187.
20. Baumann E: [Mutilation of hand and arm with Volkmann's ischemic contracture following a compound Monteggia fracture treated by circular plaster cast]. *Ther Umsch* 1973;30:877.
21. Bayley N: Individual patterns of development. *Child Dev* 1956;27:45.
22. Beasley RW, Cooley SGE, Flatt AE, et al: The hand and upper extremity. In Little JW (ed): *Reconstructive Plastic Surgery: Principles and Procedures in Correction, Reconstruction and Transplantation*, vol 6, p 3131. Philadelphia, WB Saunders Co, 1977.
23. Benevenia J, Makley JT, Leeson MC, et al: Primary epiphyseal transplants and bone overgrowth in childhood amputations. *J Pediatr Orthop* 1992;12:746.
24. Bernardo LM, Gardner MJ: Lawn mower injuries to children in Pennsylvania, 1989 to 1993. *Int J Trauma Nurs* 1996;2:36.
25. Bingold AC: On splitting plasters: a useful analogy. *J Bone Joint Surg* 1979;61-B:294.
26. Birch JG: Surgical technique of physeal bar resection. *Instr Course Lect* 1992;41:445.
27. Birch JG, Herring JA, Wenger DR: Surgical anatomy of selected physes. *J Pediatr Orthop* 1984;4:224.
28. Bisgard JD, Martenson L: Fractures in children. *Surg Gynecol Obstet* 1937;65:464.
29. Bittner S, Newberger EH: Pediatric understanding of child abuse and neglect. *Pediatr Rev* 1981;2:197.
30. Blasler RD, Aronson J, Tursky EA: External fixation of pediatric femur fractures. *J Pediatr Orthop* 1997;17:342.
31. Bone LB, Johnson KD, Weigelt J, et al: Early versus delayed stabilization of femoral fractures: a prospective randomized study. *J Bone Joint Surg* 1989;71-A:336.
32. Borden S: Roentgen recognition of acute plastic bowing of the forearm in children. *AJR Am J Roentgenol* 1975;125:524.
33. Borden S: Traumatic bowing of the forearm in children. *J Bone Joint Surg* 1974;56-A:611.
34. Born CT, Ross SE, Iannacone WM, et al: Delayed identification of skeletal injury in multisystem trauma: the "missed" fracture. *J Trauma* 1989;29:1643.
35. Bourne RB, Rorabeck CH: Compartment syndromes of the lower leg. *Clin Orthop* 1989;240:97.
36. Bright RW: Operative correction of partial epiphyseal plate closure by osseous-bridge resection and silicone-rubber implant: an experimental study in dogs. *J Bone Joint Surg* 1974;56-A:655.
37. Bruce DA, Schut L, Bruno LA, et al: Outcome following severe head injuries in children. *J Neurosurg* 1989;48:679.
38. Brumback RJ, Jones AL: Interobserver agreement in the classification of open fractures of the tibia: the results of a survey of two hundred and forty-five orthopaedic surgeons. *J Bone Joint Surg* 1994;76-A:1162.
39. Buckley SL, Smith G, Sponseller PD, et al: Open fractures of the tibia in children. *J Bone Joint Surg* 1990;72-A:1462.
40. Byrd HS, Cierny GD, Tebbets JB: The management of open tibial fractures with associated soft-tissue loss: external pin fixation with early flap coverage. *Plast Reconstr Surg* 1981;68:73.
41. Caffey J: Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. *AJR Am J Roentgenol* 1946;56:163.
42. Campbell CC, Waters PM, Emans JB, et al: Neurovascular injury and displacement in type III supracondylar humerus fractures. *J Pediatr Orthop* 1995;15:47.
43. Cappelleri JC, Eckenrode J, Powers JL: The epidemiology of child abuse: findings from the Second National Incidence and Prevalence Study of Child Abuse and Neglect. *Am J Public Health* 1993;83:1622.
44. Carlson WO, Wenger DR: A mapping method to prepare for surgical excision of a partial physeal arrest. *J Pediatr Orthop* 1984;4:232.
45. Chan B, Walker P, Cass D: Urban trauma: an analysis of 1,116 paediatric cases. *J Trauma* 1989;29:1540.
46. Charnley J: *The Closed Treatment of Common Fractures*. Edinburgh, Livingstone, 1980.
47. Cheng JC, Lam TP, Shen WY: Closed reduction and percutaneous pinning for type III displaced supracondylar fractures of the humerus in children. *J Orthop Trauma* 1995;9:511.
48. Chiang YC, Jeng SF, Yeh MC, et al: Free tissue transfer for leg reconstruction in children. *Br J Plast Surg* 1997;50:335.
49. Ciarallo L, Fleisher G: Femoral fractures: are children at risk for significant blood loss? *Pediatr Emerg Care* 1996;12:343.
50. Cierny GD, Byrd HS, Jones RE: Primary versus delayed soft tissue coverage for severe open tibial fractures: a comparison of results. *Clin Orthop* 1983;178:54.
51. Civil ID, Schwab CW: The Abbreviated Injury Scale, 1985 revision: a condensed chart for clinical use. *J Trauma* 1988;28:87.
52. Clarke HM, Upton J, Zuker RM, et al: Pediatric free tissue transfer: an evaluation of 99 cases. *Can J Surg* 1993;36:525.
53. Cole WG: Arterial injuries associated with fractures of the lower limbs in childhood. *Injury* 1981;12:460.
54. Compere EL: Growth arrest in long bones as a result of fractures that include the epiphysis. *JAMA* 1935;105:2140.
55. Compere E, Adams C: Studies of the longitudinal growth of long bones: the influence of trauma to the diaphysis. *J Bone Joint Surg* 1937;19:922.
56. Copley LA, Dormans JP, Davidson RS: Vascular injuries and their sequelae in pediatric supracondylar humeral fractures: toward a goal of prevention. *J Pediatr Orthop* 1996;16:99.
57. Coss HS, Hennrikus WL: Parent satisfaction comparing two bandage materials used during serial casting in infants. *Foot Ankle Int* 1996;17:483.
58. Costecalde M, Gaubert J, Durand J, et al: [Subtotal traumatic amputation of the limb in young children: analysis of 2 successful repairs]. *Chir Pediatr* 1988;29:184.
59. Cullen MC, Roy DR, Crawford AH, et al: Open fracture of the tibia in children. *J Bone Joint Surg* 1996;78-A:1039.
60. Curran C, Dietrich AM, Bowman MJ, et al: Pediatric cervical-spine immobilization: achieving neutral position? *J Trauma* 1995;39:729.
61. Currey JD, Butler G: The mechanical properties of bone tissue in children. *J Bone Joint Surg* 1975;57-A:810.
62. Dale GG, Harris WR: Prognosis of epiphyseal separation: an experimental study. *J Bone Joint Surg* 1958;40-B:116.
63. Davids JR: Rotational deformity and remodeling after fracture of the femur in children. *Clin Orthop* 1994;302:27.
64. Davids JR, Frick SL, Skewes E, et al: Skin surface pressure beneath

- an above-the-knee cast: plaster casts compared with fiberglass casts. *J Bone Joint Surg* 1997;79-A:565.
65. de Pablos J, Franzreb M, Barrios C: Longitudinal growth pattern of the radius after forearm fractures conservatively treated in children. *J Pediatr Orthop* 1994;14:492.
 66. 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.
 67. Dickey RL, Barnes BC, Kearns RJ, et al: Efficacy of antibiotics in low-velocity gunshot fractures. *J Orthop Trauma* 1989;3:6.
 68. Division of Injury Control, Centers for Disease Control: Childhood injuries in the United States. *Am J Dis Child* 1990;144:627.
 69. Dormans JP, Azzoni M, Davidson RS, et al: Major lower extremity lawn mower injuries in children. *J Pediatr Orthop* 1995;15:78.
 70. Dykes E, Spence L, Bohn D, et al: Evaluation of pediatric trauma care in Ontario. *J Trauma* 1989;29:724.
 71. Eaton RG, Green WT: Epimysiotomy and fasciotomy in the treatment of Volkmann's ischemic contracture. *Orthop Clin North Am* 1972;3:175.
 72. Eaton RG, Green WT: Volkmann's ischemia: a volar compartment syndrome of the forearm. *Clin Orthop* 1975;113:58.
 73. Eichelberger MR, Gotschall CS, Sacco WJ, et al: A comparison of the trauma score, the revised trauma score, and the pediatric trauma score. *Ann Emerg Med* 1989;18:1053.
 74. Evans EM: Fractures of the radius and ulna. *J Bone Joint Surg* 1951;33-B:548.
 75. Ferraro SP Jr, Zinar DM: Management of gunshot fractures of the tibia. *Orthop Clin North Am* 1995;26:181.
 76. Fleischmann W, Suger G, Kinzl L: Treatment of bone and soft tissue defects in infected nonunion. *Acta Orthop Belg* 1992;58 (suppl 1):227.
 77. Ford LT, Gilula LA: Plastic bones of the forearm [Roentgen Rounds #42]. *Orthop Rev* 1978;7:101.
 78. Foucher M: De la divulsion des epiphyses. *Cong Med Fr (Paris)* 1863;1:63.
 79. Friberg KS: Remodelling after distal forearm fractures in children. I. The effect of residual angulation on the spatial orientation of the epiphyseal plates. *Acta Orthop Scand* 1979;50:537.
 80. Friberg KS: Remodelling after distal forearm fractures in children. III. Correction of residual angulation in fractures of the radius. *Acta Orthop Scand* 1979;50:741.
 81. Friedman RJ, Jupiter JB: Vascular injuries and closed extremity fractures in children. *Clin Orthop* 1984;188:112.
 82. Fry K, Hoffer MM, Brink J: Femoral shaft fractures in brain-injured children. *J Trauma* 1976;16:371.
 83. Furnival RA, Schunk JE: ABCs of scoring systems for pediatric trauma. *Pediatr Emerg Care* 1999;15:215.
 84. Galleno H, Oppenheim WL: The battered child syndrome revisited. *Clin Orthop* 1982;162:11.
 85. Garces GL, Hernandez Hermoso JA: Bone growth after periosteal stripping in rats. *Int Orthop* 1991;15:49.
 86. Garfin SR, Mubarak SJ, Evans KL, et al: Quantification of intracompartmental pressure and volume under plaster casts. *J Bone Joint Surg* 1981;63-A:449.
 87. Gasco J, de Pablos J: Bone remodeling in malunited fractures in children: is it reliable? *J Pediatr Orthop B* 1997;6:126.
 88. Graham B, Paulus DA, Caffee HH: Pulse oximetry for vascular monitoring in upper extremity replantation surgery. *J Hand Surg* 1986;11-A:687.
 89. Green W, Anderson M: Experiences with epiphyseal arrest in correcting discrepancies in length of the lower extremities in infantile paralysis. *J Bone Joint Surg* 1947;29:659.
 90. Green W, Anderson M: Skeletal age and the control of bone growth. *Instr Course Lect* 1960;17:199.
 91. Greensher J: Recent advances in injury prevention. *Pediatr Rev* 1988;10:171.
 92. Greulich W, Pyle S: *Radiographic Atlas of the Skeletal Development of the Hand and Wrist*. Stanford, CA, Stanford University Press, 1959.
 93. Grimard G, Naudie D, Laberge LC, et al: Open fractures of the tibia in children. *Clin Orthop* 1996;332:62.
 94. Guerra JJ, Bednar JM: Equipment malfunction in common hand surgical procedures: complications associated with the pneumatic tourniquet and with the application of casts and splints. *Hand Clin* 1994;10:45.
 95. Gustilo RB: Current concepts in the management of open fractures. *Instr Course Lect* 1987;36:359.
 96. Gustilo RB, Anderson JT: Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. *J Bone Joint Surg* 1976;58-A:453.
 97. Gustilo RB, Corpuz V, Sherman RE: Epidemiology, mortality and morbidity in multiple trauma patients. *Orthopedics* 1985;8:1523.
 98. 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.
 99. Gustilo RB, Merkow RL, Templeman D: The management of open fractures. *J Bone Joint Surg* 1990;72-A:299.
 100. Haas SL: The localization of the growing point in the epiphyseal cartilage plate of bones. *Am J Orthop Surg* 1917;15:563.
 101. Haasbeek JF, Cole WG: Open fractures of the arm in children. *J Bone Joint Surg* 1995;77-B:576.
 102. Hahn YS, Chyung C, Barthel MJ, et al: Head injuries in children under 36 months of age: demography and outcome. *Childs Nerv Syst* 1988;4:34.
 103. Hampton RL, Newberger EH: Child abuse incidence and reporting by hospitals: significance of severity, class, and race. *Am J Public Health* 1985;75:56.
 104. Hanlon M, Barnes M, Lamb G, et al: Central compartment pressure monitoring following clubfoot release. *J Pediatr Orthop* 1996;16:63.
 105. Hargens AR, Mubarak SJ: Current concepts in the pathophysiology, evaluation, and diagnosis of compartment syndrome. *Hand Clin* 1998;14:371.
 106. Harris HA: Lines of arrested growth in the long bones in childhood: the correlation of histological and radiographic appearances in clinical and experimental conditions. *Br J Radiol* 1931;4:561.
 107. Harris MB, Waguespack AM, Kronlage S: "Clearing" cervical spine injuries in polytrauma patients: is it really safe to remove the collar? *Orthopedics* 1997;20:903.
 108. Harris WR: The endocrine basis for slipping of the upper femoral epiphysis. *J Bone Joint Surg* 1950;32-B:5.
 109. Helfer RE, Kempe CH (eds): *The Battered Child*. Chicago, University of Chicago Press, 1968.
 110. Henderson OL, Morrissy RT, Gerdes MH, et al: Early casting of femoral shaft fractures in children. *J Pediatr Orthop* 1984;4:16.
 111. Henssge J, Linka F: [Volkmann's contracture and constricting bandage]. *Beitr Orthop Traumatol* 1968;15:27.
 112. 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.
 113. Herring JA, Mubarak SJ: Complications of a tibial osteotomy. *J Pediatr Orthop* 1983;3:625.
 114. 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* 1989;71-A:15.
 115. Hirsch C, Evans FG: Studies on some physical properties of infant compact bone. *Acta Orthop Scand* 1965;35:300.
 116. Hoffer MM, Johnson B: Shrapnel wounds in children. *J Bone Joint Surg* 1992;74-A:766.
 117. Holschneider A, Vogl D, Dietz H: Differences in leg length following femoral shaft fractures in childhood. *Z Kinderchir* 1985;40:341.
 118. Holter JC, Friedman SB: Child abuse: early case finding in the emergency department. *Pediatrics* 1968;42:128.
 119. Horn BD, Rettig ME: Interobserver reliability in the Gustilo and Anderson classification of open fractures. *J Orthop Trauma* 1993;7:357.
 120. Hubbard DD: Injuries of the spine in children and adolescents. *Clin Orthop* 1974;100:56.
 121. Huerta C, Griffith R, Joyce SM: Cervical spine stabilization in pediatric patients: evaluation of current techniques [see comments]. *Ann Emerg Med* 1987;16:1121.
 122. Hull JB: Management of gunshot fractures of the extremities. *J Trauma* 1996;40:S193.
 123. Hynes D, O'Brien T: Growth disturbance lines after injury of the distal tibial physis: their significance in prognosis. *J Bone Joint Surg* 1988;70-B:231.
 124. Iannotti JP, Goldstein S, Kuhn J, et al: Growth plate and bone development. In Simon SR (ed): *Orthopaedic Basic Science*, p 191. Rosemont, IL, American Academy of Orthopaedic Surgeons, 1994.
 125. Iqbal QM: Long bone fractures among children in Malaysia. *Int Surg* 1974;59:410.
 126. Irwin A, Gibson P, Ashcroft P: Open fractures of the tibia in children. *Injury* 1995;26:21.

127. Ismail N, Bellemare JF, Mollitt DL, et al: Death from pelvic fracture: children are different. *J Pediatr Surg* 1996;31:82.
128. Izhar U, Munkonge L: Femoral fracture in children (a prospective study of two hundred and four fractures). *Med J Zambia* 1982;16:51.
129. Jacobsen FS: Periosteum: its relation to pediatric fractures. *J Pediatr Orthop B* 1997;6:84.
130. Jaffe DM, Binns H, Radkowski MA, et al: Developing a clinical algorithm for early management of cervical spine injury in child trauma victims. *Ann Emerg Med* 1987;16:270.
131. Janzing H, Broos P, Rommens P: Compartment syndrome as complication of skin traction, in children with femoral fractures. *Acta Chir Belg* 1996;96:135.
132. Jenkins D, Cheng D, Hodgson A: Stimulation of growth by periosteal stripping: a clinical study. *J Bone Joint Surg* 1975;57-B:482.
133. Juster M, Moscofian A, Balmain-Oligo N: [Formation of the skeleton. VIII. Growth of a long bone: periostealization of the metaphyseal bone]. *Bull Assoc Anat (Nancy)* 1975;59:437.
134. Kadiyala RK, Waters PM: Upper extremity pediatric compartment syndromes. *Hand Clin* 1998;14:467.
135. Kasser JR: Physeal bar resections after growth arrest about the knee. *Clin Orthop* 1990;255:68.
136. Kaufmann CR, Maier RV, Rivara FP, et al: Evaluation of the pediatric trauma score [see comments]. *JAMA* 1990;263:69.
137. Keen TP: Nursing care of the pediatric multitrauma patient. *Nurs Clin North Am* 1990;25:131.
138. Kempe CH, Silverman FN, Steele BF, et al: The battered child syndrome. *JAMA* 1962;181:17.
139. Kerley ER: The microscopic determinants of age in human bone. *Am J Phys Anthropol* 1965;23:149.
140. Kewalramani LS, Kraus JF, Sterling HM: Acute spinal-cord lesions in a pediatric population: epidemiological and clinical features. *Paraplegia* 1980;18:206.
141. King J, Diefendorf D, Aphorpe J, et al: Analysis of 429 fractures in 189 battered children. *J Pediatr Orthop* 1988;8:585.
142. Kirschenbaum D, Albert MC, Robertson WW Jr, et al: Complex femur fractures in children: treatment with external fixation. *J Pediatr Orthop* 1990;10:588.
143. Kissoon N, Dreyer J, Walia M: Pediatric trauma: differences in pathophysiology, injury patterns and treatment compared with adult trauma. *CMAJ (Canada)* 1990;142:27.
144. Knapp TP, Patzakis MJ, Lee J, et al: Comparison of intravenous and oral antibiotic therapy in the treatment of fractures caused by low-velocity gunshots: a prospective, randomized study of infection rates. *J Bone Joint Surg* 1996;78-A:1167.
145. Kowal-Vern A, Paxton TP, Ros SP, et al: Fractures in the under-3-year-old age cohort. *Clin Pediatr (Phila)* 1992;31:653.
146. Krajbich JI: Lower-limb deficiencies and amputations in children. *J Am Acad Orthop Surg* 1998;6:358.
147. Kreder HJ, Armstrong P: A review of open tibia fractures in children. *J Pediatr Orthop* 1995;15:482.
148. Kregor PJ, Song KM, Rott ML Jr, et al: Plate fixation of femoral shaft fractures in multiply injured children. *J Bone Joint Surg* 1993;75-A:1774.
149. Kummer B, Lohscheidt K: [Mathematical model of the longitudinal growth of long bones]. *Anat Anz* 1985;158:377.
150. Laasonen EM, Kivioja A: Delayed diagnosis of extremity injuries in patients with multiple injuries. *J Trauma* 1991;31:257.
151. Lally KP, Senac M, Hardin WD Jr, et al: Utility of the cervical spine radiograph in pediatric trauma. *Am J Surg* 1989;158:540.
152. 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;202:1.
153. Langenskiöld A: Growth disturbance after osteomyelitis of femoral condyles in infants. *Acta Orthop Scand* 1984;55:1.
154. Langenskiöld A: An operation for partial closure of an epiphysal plate in children, and its experimental basis. *J Bone Joint Surg* 1975;57-B:325.
155. Langenskiöld A: Partial closure of the epiphysal plate: principles of treatment. 1978 [Classical Article]. *Clin Orthop* 1993;297:4.
156. Langenskiöld A: Surgical treatment of partial closure of the growth plate. *J Pediatr Orthop* 1981;1:3.
157. Langenskiöld A, Osterman K: Surgical treatment of partial closure of the epiphysal plate. *Reconstr Surg Traumatol* 1979;17:48.
158. Larsen E, Vittas D, Torp-Pedersen S: Remodeling of angulated distal forearm fractures in children. *Clin Orthop* 1988;237:190.
159. Leape LL: Progress in pediatric trauma: anatomy and patterns of injury. In Harris BH (ed): *The First National Conference on Pediatric Trauma*. Boston, Nob Hill Press, 1985.
160. Letts RM, Miller D: Gunshot wounds of the extremities in children. *J Trauma* 1976;16:807.
161. Light TR, Ogden DA, Ogden JA: The anatomy of metaphyseal torus fractures. *Clin Orthop* 1984;188:103.
162. Lincoln TL, Mubarak SJ: "Isolated" traumatic radial-head dislocation. *J Pediatr Orthop* 1994;14:454.
163. Lloyd-Thomas AR, Anderson I: ABC of major trauma. Paediatric trauma: secondary survey. *BMJ* 1990;301:433.
164. Loder RT, Bookout C: Fracture patterns in battered children. *J Orthop Trauma* 1991;5:428.
165. 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.
166. Loder RT, Swinford AE, Kuhns LR: The use of helical computed tomographic scan to assess bony physeal bridges. *J Pediatr Orthop* 1997;17:356.
167. Logar M, Smrkolj V, Veselko M: An unusual lawn mower injury. *Unfallchirurg* 1996;99:152.
168. Mabrey JD, Fitch RD: Plastic deformation in pediatric fractures: mechanism and treatment. *J Pediatr Orthop* 1989;9:310.
169. 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.
170. Matsen FA 3rd, Staheli LT: Neurovascular complications following tibial osteotomy in children: a case report. *Clin Orthop* 1975;110:210.
171. Matsen FA 3rd, Winquist RA, Krugmire RB Jr: Diagnosis and management of compartmental syndromes. *J Bone Joint Surg* 1980;62-A:286.
172. Matsen FA, Krugmire RB Jr, King RV: Nicolas Andry Award. Increased tissue pressure and its effects on muscle oxygenation in level and elevated human limbs. *Clin Orthop* 1979;144:311.
173. Matsen FA 3rd, Mayo KA, Sheridan GW, et al: Continuous monitoring of intramuscular pressure and its application to clinical compartmental syndromes. *Bibl Anat* 1977;15(pt 1):112.
174. Matsen FA 3rd, Mayo KA, Sheridan GW, et al: Monitoring of intramuscular pressure. *Surgery* 1976;79:702.
175. Matsen FA 3rd, Rorabeck CH: Compartment syndromes. *Instr Course Lect* 1989;38:463.
176. Matsen FA 3rd, Veith RG: Compartmental syndromes in children. *J Pediatr Orthop* 1981;1:33.
177. Matsen FA 3rd, Wyss CR, Krugmire RB Jr, et al: The effects of limb elevation and dependency on local arteriovenous gradients in normal human limbs with particular reference to limbs with increased tissue pressure. *Clin Orthop* 1980;150:187.
178. McClelland CQ, Heiple KG: Fractures in the first year of life: a diagnostic dilemma. *Am J Dis Child* 1982;136:26.
179. McMahon P, Grossman W, Gaffney M, et al: Soft-tissue injury as an indication of child abuse. *J Bone Joint Surg* 1995;77-A:1179.
180. Menelaus M: Correction of leg length discrepancy by epiphysal arrest. *J Bone Joint Surg* 1966;48-B:336.
181. Merten DF, Radkowski MA, Leonidas JC: The abused child: a radiological reappraisal. *Radiology* 1983;146:377.
182. Metak G, Scherer MA, Dannohl C: [Missed injuries of the musculoskeletal system in multiple trauma—a retrospective study]. *Zentralbl Chir* 1994;119:88.
183. Mizuta T, Benson WM, Foster BK, et al: Statistical analysis of the incidence of physeal injuries. *J Pediatr Orthop* 1987;7:518.
184. Moed BR, Thorderson PK: Measurement of intracompartmental pressure: a comparison of the slit catheter, side-port needle, and simple needle. *J Bone Joint Surg* 1993;75-A:231.
185. Moyikoua A, Dolama F, Pena-Pitra B, et al: [Open fractures caused by gunshot in civilian practice: apropos of 31 cases]. *Ann Chir* 1994;48:1020.
186. Mubarak SJ: A practical approach to compartmental syndromes. Part II. Diagnosis. *Instr Course Lect* 1983;32:92.
187. Mubarak SJ, Carroll NC: Volkmann's contracture in children: aetiology and prevention. *J Bone Joint Surg* 1979;61-B:285.
188. Mubarak SJ, Owen CA, Hargens AR, et al: Acute compartment syndromes: diagnosis and treatment with the aid of the wick catheter. *J Bone Joint Surg* 1978;60-A:1091.
189. Muñoz-Juarez M, Drugas GT, Hallett JW, et al: Vena caval im-

- palement: an unusual lawn mower injury in a child. *Mayo Clin Proc* 1998;73:537.
190. Naga AH, Broadrick GL: Traumatic bowing of the radius and ulna in children. *NC Med J* 1977;38:452.
 191. Nakayama DK, Gardner MJ, Rowe MI: Emergency endotracheal intubation in pediatric trauma. *Ann Surg* 1990;211:218.
 192. Nakayama DK, Ramenofsky ML, Rowe MI: Chest injuries in childhood. *Ann Surg* 1989;210:770.
 193. National Child Abuse and Neglect Data Systems; Working Paper 2-1991. Summary data component. Washington, DC, Government Printing Office, 1993.
 194. Nayduch DA, Moylan J, Rutledge R, et al: Comparison of the ability of adult and pediatric trauma scores to predict pediatric outcome following major trauma. *J Trauma* 1991;31:452.
 195. Nicholas RM, McCoy GF: Immediate intramedullary nailing of femoral shaft fractures due to gunshots. *Injury* 1995;26:257.
 196. O'Brien ET: Fractures of the hand and wrist region. In Rockwood CA, Wilkins KE, King RE (eds): *Fractures in Children*, vol 3. Philadelphia, JB Lippincott Co, 1991.
 197. O'Brien T: Growth-disturbance lines in congenital dislocation of the hip. *J Bone Joint Surg* 1985;67-A:626.
 198. O'Brien T, Millis MB, Griffin PP: The early identification and classification of growth disturbances of the proximal end of the femur. *J Bone Joint Surg* 1986;68-A:970.
 199. Oestreich AE: Imaging of the skeleton and soft tissues in children. *Curr Opin Radiol* 1992;4:55.
 200. Ogden JA: The evaluation and treatment of partial physal arrest. *J Bone Joint Surg* 1987;69-A:1297.
 201. Ogden JA: Injury to the growth mechanisms of the immature skeleton. *Skeletal Radiol* 1981;6:237.
 202. Ogden JA: *Skeletal Injury in the Child*. Philadelphia, Lea & Febiger, 1981.
 203. Padovani JP, Rigault P, Mouterde P: [Vascular traumatic injuries of the limbs in children]. *Chir Pediatr* 1978;19:69.
 204. Parry SW, Toth BA, Elliott LF: Microvascular free-tissue transfer in children. *Plast Reconstr Surg* 1988;81:838.
 205. Patzakis MJ: Clostridial myonecrosis. *Instr Course Lect* 1990;39:491.
 206. Patzakis MJ, Wilkins J: Factors influencing infection rate in open fracture wounds. *Clin Orthop* 1989;243:36.
 207. Peterson HA: Partial growth plate arrest and its treatment. *J Pediatr Orthop* 1984;4:246.
 208. Peterson HA: Physal fractures. Part 3. Classification. *J Pediatr Orthop* 1994;14:439.
 209. Peterson HA, Burkhart SS: Compression injury of the epiphyseal growth plate: fact or fiction? *J Pediatr Orthop* 1981;1:377.
 210. Peterson HA, Madhok R, Benson JT, et al: Physal fractures. Part 1. Epidemiology in Olmsted County, Minnesota, 1979–1988. *J Pediatr Orthop* 1994;14:423.
 211. Poland J: *Traumatic Separation of the Epiphyses*. London, Smith, Elder, & Co, 1898.
 212. Porat S, Milgrom C, Nyska M, et al: Femoral fracture treatment in head-injured children: use of external fixation. *J Trauma* 1986;26:81.
 213. Prader A: [Normal growth and disorders of growth in children and adolescents]. *Klin Wochenschr* 1981;59:977.
 214. Pritchett JW: Growth plate activity in the upper extremity. *Clin Orthop* 1991;268:235.
 215. Rang M: Biomechanic differences. In *Children's Fractures*, p 2. Philadelphia, JB Lippincott Co, 1983.
 216. Rang M: *Children's Fractures*. Philadelphia, JB Lippincott Co, 1983.
 217. Rang M: Injuries of the perichondrial ring. In *Children's Fractures*, p 24. Philadelphia, JB Lippincott Co, 1983.
 218. Ray SA, Ivory JP, Beavis JP: Use of pulse oximetry during manipulation of supracondylar fractures of the humerus. *Injury* 1991;21:103.
 219. Reed MH: Fractures and dislocations of the extremities in children. *J Trauma* 1977;17:351.
 220. Reich RB: Traumatic plastic bowing deformity of the radius and ulna in a skeletally mature adult. *J Orthop Trauma* 1994;8:258.
 221. Robertson P, Karol LA, Rab GT: Open fractures of the tibia and femur in children. *J Pediatr Orthop* 1996;16:621.
 222. Rodgers WB, Waters PM, Hall JE: Chronic Monteggia lesions in children: complications and results of reconstruction. *J Bone Joint Surg* 1996;78-A:1322.
 223. Rorabeck CH: A practical approach to compartmental syndromes. Part III. Management. *Instr Course Lect* 1983;32:102.
 224. Rorabeck CH, Castle GS, Hardie R, et al: Compartmental pressure measurements: an experimental investigation using the slit catheter. *J Trauma* 1981;21:446.
 225. Sacco WJ, MacKenzie EJ, Champion HR, et al: Comparison of alternative methods for assessing injury severity based on anatomic descriptors. *J Trauma* 1999;47:441.
 226. Salter RB: Epiphyseal plate injuries. In Letts RM (ed): *Management of Pediatric Fractures*, p 11. New York, Churchill Livingstone, 1994.
 227. Salter RB, Harris WR: injuries involving the epiphyseal plate. *J Bone Joint Surg* 1963;45:587.
 228. Sanders WE, Heckman JD: Traumatic plastic deformation of the radius and ulna: a closed method of correction of deformity. *Clin Orthop* 1984;188:58.
 229. Schwarz N: [Incidence of open fractures in children]. *Aktuelle Traumatol* 1981;11:133.
 230. Seekamp A, Ziegler M, Biank J, et al: [The significance of hypothermia in polytrauma patients]. *Unfallchirurg* 1996;99:100.
 231. Shakespeare DT, Henderson NJ, Clough G: The slit catheter: a comparison with the wick catheter in the measurement of compartment pressure. *Injury* 1982;13:404.
 232. Shapiro J, Akbarnia BA, Hanel DP: Free tissue transfer in children. *J Pediatr Orthop* 1989;9:590.
 233. 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.
 234. Shelton WR, Canale ST: Fractures of the tibia through the proximal tibial epiphyseal cartilage. *J Bone Joint Surg* 1979;61-A:167.
 235. Silverman FN: The roentgen manifestations of unrecognized skeletal trauma in infants. *AJR Am J Roentgenol* 1953;69:413.
 236. Simonian PT, Hanel DP: Traumatic plastic deformity of an adult forearm: case report and literature review. *J Orthop Trauma* 1996;10:213.
 237. Singh D: Pulse oximetry and fracture manipulation. *Injury* 1992;23:70.
 238. Skak SV: A case of partial physal closure following compression injury. *Arch Orthop Trauma Surg* 1989;108:185.
 239. Sola C, Silberman F, Cabrini R: Stimulation of the longitudinal growth of long bones by periosteal stripping. *J Bone Joint Surg* 1963;45-A:1679.
 240. Song KM, Sangeorzan B, Benirschke S, et al: Open fractures of the tibia in children. *J Pediatr Orthop* 1996;16:635.
 241. Stenstrom R, Gripenberg L, Bergius AR: Traumatic bowing of forearm and lower leg in children. *Acta Radiol [Diagn]* (Stockh) 1978;19:243.
 242. Stott NS, Zions LE, Holtom PD, et al: Acute hematogenous osteomyelitis: an unusual cause of compartment syndrome in a child. *Clin Orthop* 1995;317:219.
 243. Stricker SJ, Volgas DA: Extremity handgun injuries in children and adolescents. *Orthopedics* 1998;21:1095.
 244. Stromqvist B, Lidgren L, Norgren L, et al: Neurovascular injury complicating displaced proximal fractures of the humerus. *Injury* 1987;18:423.
 245. Stucky W, Loder RT: Extremity gunshot wounds in children. *J Pediatr Orthop* 1991;11:64.
 246. Subcommittee of Advanced Trauma Life Support of the American College of Surgeons Committee on Trauma: *Advanced Trauma Life Support: Student Manual*, p. 11. Chicago, American College of Surgeons, 1989.
 247. Teasdale G, Jennett B: Assessment of coma and impaired consciousness: a practical scale. *Lancet* 1974;2:81.
 248. Templeman DC, Gulli B, Tsukayama DT, et al: Update on the management of open fractures of the tibial shaft. *Clin Orthop* 1998;350:18.
 249. Tepas JJ, Mollitt DL, Talbert JL, et al: The pediatric trauma score as a predictor of injury severity in the injured child. *J Pediatr Surg* 1987;22:14.
 250. Tollens T, Janzing H, Broos P: The pathophysiology of the acute compartment syndrome. *Acta Chir Belg* 1998;98:171.
 251. Tolo VT: External skeletal fixation in children's fractures. *J Pediatr Orthop* 1983;3:435.
 252. Treloar DJ, Nypaver M: Angulation of the pediatric cervical spine with and without cervical collar. *Pediatr Emerg Care* 1997;13:5.
 253. Trice M, Colwell CW: A historical review of compartment syndrome and Volkmann's ischemic contracture. *Hand Clin* 1998;14:335.
 254. Tsukayama DT, Gustilo RB: Antibiotic management of open fractures. *Instr Course Lect* 1990;39:487.
 255. Tupman G: Treatment of inequality of the lower limbs: the results of operations for stimulation of growth. *J Bone Joint Surg* 1960;42-B:489.
 256. Uppal GS, Smith RC, Sherk HH, et al: Accurate compartment pressure

- measurement using the Intravenous Alarm Control (IVAC) pump: report of a technique. *J Orthop Trauma* 1992;6:87.
257. US Department of Health and Human Services: Child Maltreatment 1996: Reports from the States to the National Child Abuse and Neglect Data System. Washington, DC; US Government Printing Office, 1998.
 258. Vasli LR: Diagnosis of vascular injury in children with supracondylar fractures of the humerus. *Injury* 1988;19:11.
 259. Victoroff BN, Robertson WW Jr, Eichelberger MR, et al: Extremity gunshot injuries treated in an urban children's hospital. *Pediatr Emerg Care* 1994;10:1.
 260. Vinz H: Die Änderung der Festigkeitseigenschaften des kompakten Knochengewebes im Laufe der Altersentwicklung. *Gegenbaurs Morphol Jahrb* 1970;115:257.
 261. Vittas D, Larsen E, Torp-Pedersen S: Angular remodeling of midshaft forearm fractures in children. *Clin Orthop* 1991;265:261.
 262. von Schroeder HP, Botte MJ: Definitions and terminology of compartment syndrome and Volkmann's ischemic contracture of the upper extremity. *Hand Clin* 1998;14:331.
 263. 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.
 264. Walker M, Storrs B, Mayer T: Head injuries. In Mayers TA (ed): *Emergency Management of Pediatric Trauma*, p 272. Philadelphia, WB Saunders, Co, 1985.
 265. Wallace ME, Hoffman EB: Remodelling of angular deformity after femoral shaft fractures in children. *J Bone Joint Surg* 1992;74-B:765.
 266. Walloe A, 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.
 267. Washington ER, Lee WA, Ross WA Jr: Gunshot wounds to the extremities in children and adolescents. *Orthop Clin North Am* 1995;26:19.
 268. Weiner G, Styf J, Nakhostine M, et al: Effect of ankle position and a plaster cast on intramuscular pressure in the human leg. *J Bone Joint Surg* 1994;76-A:1476.
 269. Westh R, Menelaus M: A simple calculation for the timing of epiphyseal arrest: a further report. *J Bone Joint Surg* 1981;63-B:117.
 270. Whitesides TE Jr, Haney TC, Harada H, et al: A simple method for tissue pressure determination. *Arch Surg* 1975;110:1311.
 271. Whitesides TE, Haney TC, Morimoto K, et al: Tissue pressure measurements as a determinant for the need of fasciotomy. *Clin Orthop* 1975;113:43.
 272. Wiley JJ, McIntyre WM: Fracture patterns in children. In *Current Concepts of Bone Fragility*, p 159. Berlin, Springer-Verlag, 1986.
 273. Wilkins KE: Supracondylar fractures: what's new? *J Pediatr Orthop B* 1997;6:110.
 274. Willis RB, Rorabeck CH: Treatment of compartment syndrome in children. *Orthop Clin North Am* 1990;21:401.
 275. Wilson SC, Vrahas MS, Berson L, et al: A simple method to measure compartment pressures using an intravenous catheter. *Orthopedics* 1997;20:403.
 276. Wolff J: *Das Gesetz der Transformation der Knochen*. Berlin: Verlag von August-Hirschwald, 1982.
 277. Worlock P, Stower M: Fracture patterns in Nottingham children. *J Pediatr Orthop* 1986;6:656.
 278. Worlock P, Stower M, Barbor P: Patterns of fractures in accidental and non-accidental injury in children: a comparative study. *BMJ (Clin Res Ed)* 1986;293:100.
 279. Wyrsch B, Mencia GA, Green NE: Open reduction and internal fixation of pediatric forearm fractures. *J Pediatr Orthop* 1996;16:644.
 280. Wyss CR, Matsen FA III, King RV, et al: Dependence of transcutaneous oxygen tension on local arteriovenous pressure gradient in normal subjects. *Clin Sci* 1981;60:499.
 281. Yao L, Seeger LL: Epiphyseal growth arrest lines: MR findings. *Clin Imaging* 1997;21:237.
 282. Ziv I, Rang M: Treatment of femoral fracture in the child with head injury. *J Bone Joint Surg* 1983;65-B:276.
 283. Zonis Z, Weisz G, Ramon Y, et al: Salvage of the severely injured limb in children: a multidisciplinary approach. *Pediatr Emerg Care* 1995;11:176.