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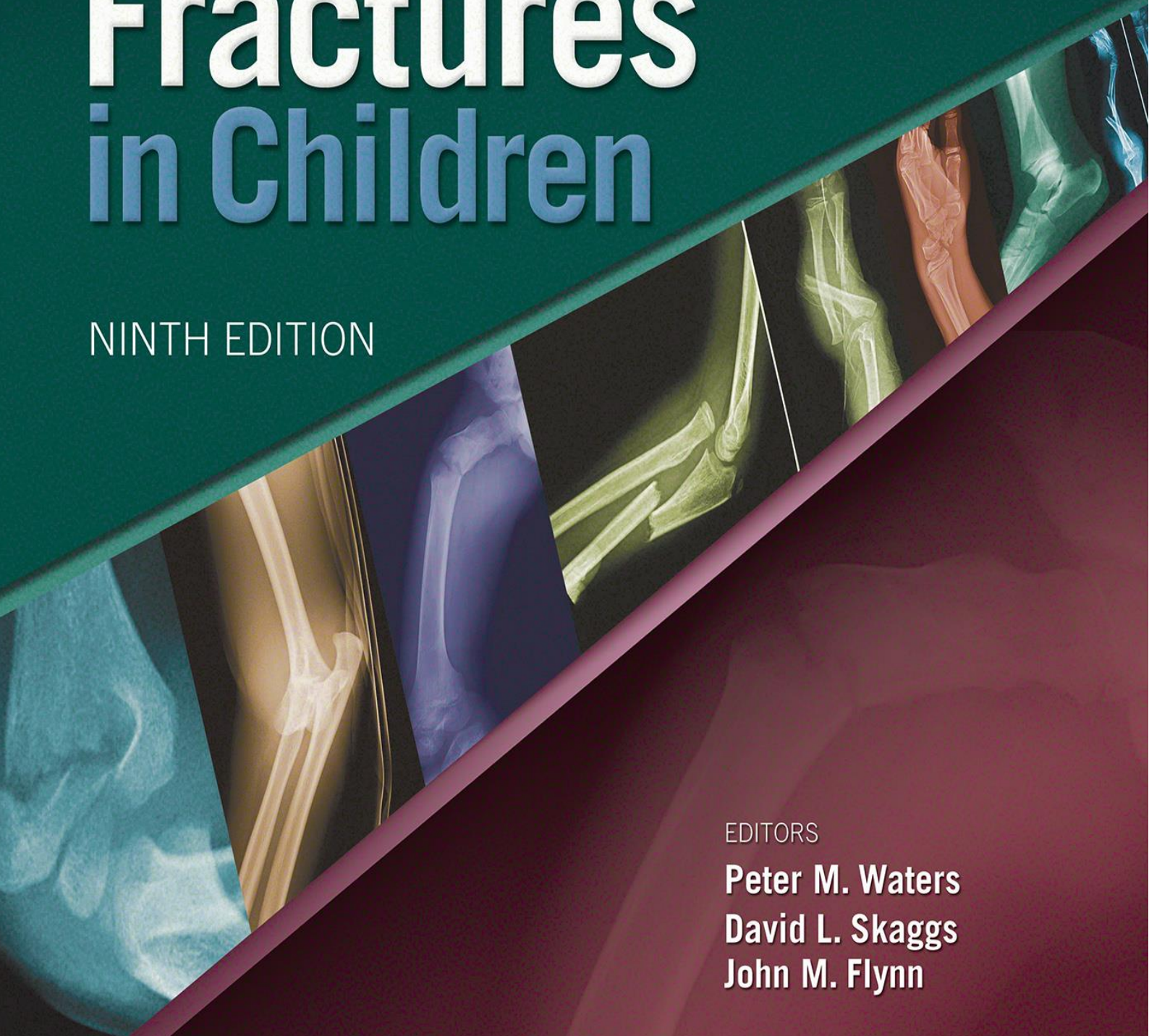
Rockwood and Wilkins' Fractures in Children

NINTH EDITION



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Dedication

I want to acknowledge my wife Mary and children Erin, Colleen, John, and Kelly for patience and understanding while I dedicated many hours to this project. They understand that Dad has homework too. Also, I would like to recognize the injured children, medical students, residents, and fellows who count on this textbook to be the trusted source of information for fracture care; their needs are the inspiration that drive us to create an excellent final product.

Jack Flynn

I wish to thank my wife Janet, expanding family of James, Rebecca, Billy, Izzy, and Elle. They, along with our unique collection of friends, supported me doing the binge work on this book during the hours before the real fun begins. I am indebted to my partners and patients who teach me about fracture care every day.

Peter M. Waters

I want to most of all thank my wife Val for always being there for me. Thanks to my children Kira, Jamie, and Clay for being good friends in every sense of the word and assisting me with the many children who come to our home with injuries. I hope readers will welcome my daughter Kira to the medical profession; she is a first-year medical student at the time of this publication. And thank you to everyone at the Children's Orthopaedic Center at Los Angeles Children's Hospital that make it fun and rewarding to come to work every day.

David L. Skaggs

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Preface

With all of the information that is now available to physicians, it can be difficult to know what sources to trust. By asking top thought leaders in pediatric orthopedics to critically evaluate the medical literature, we hope the ninth edition of *Rockwood and Wilkins' Fractures in Children* eliminates this problem for our readers. In each chapter, subject matter experts offer their overviews and analyses of existing clinical research and provide the very best evidence-based recommendations possible. The "author's preferred method" at the end of each chapter serves as a concise and practical algorithm for treating children.

We've added a number of new features for the ninth edition, including checklists for preoperative planning and key surgical steps, tables of potential pitfalls and preventative measures, and short lists of key annotated references. We've retained the author's preferred treatment section, which has been popular since its introduction a few editions ago.

This ninth edition also inaugurates a new partnership with the Orthopaedic Trauma Association (OTA). The ninth edition is the official publication of the OTA, and is a foundational component in *OTAOnline*, an electronic knowledge portal that brings together *Rockwood, Green, and Wilkins Fractures in Adults and Children*, *Journal of Orthopaedic Trauma*, an extensive orthopaedic trauma video library, OTA International (an open access journal), and routine updates.

This project has been a labor of love for its editors. Our collaboration on the ninth edition has both satisfied our never-ending drive to improve patient care and strengthened our already close friendship. We hope our work will be of service to you, the reader, and your young patients whose lives you strive to make better.

Peter M. Waters, MD
David L. Skaggs, MD, MMM
John M. (Jack) Flynn, MD

Table of Contents

1: Epidemiology of Fractures in Children

Brian K. Brighton, Michael Vitale

Introduction

Epidemiology is defined as the study of the distribution and determinants of health and disease and the application of this science to the control of diseases and other health problems. As such, epidemiology is the cornerstone of an evidence-based approach to preventing disease and to optimizing treatment strategies. Various epidemiologic methods including surveillance and descriptive studies can be used to investigate the distribution of frequency, pattern, and burden of disease whereas analytical methods can be used to study the determinants of disease. An understanding of the epidemiology of pediatric trauma is a prerequisite for the timely evolution of optimal care strategies, and for the development of effective prevention strategies.

Injuries in children and adolescents represent a major public health challenge facing pediatric patients, families, and health care providers worldwide. Given the wide-reaching impact that pediatric musculoskeletal injury has on public health, an understanding of the epidemiology of pediatric fractures provides an opportunity to maximize efforts aimed at prevention and optimal treatment. Unintentional injuries are the leading cause of death for children in the United States. In 2015, the Centers for Disease Control and Prevention (CDC) reported over 10,000 deaths of children between the ages of 0 and 18 years caused by unintentional injuries (http://webappa.cdc.gov/sasweb/ncipc/mortrate.html). However, fatalities only represent a small portion of the impact unintentional injuries have on children. There were over 7.5 million nonfatal unintentional injuries to children of the same age group in 2015 (http://webappa.cdc.gov/sasweb/ncipc/nfirates.html). Pediatric trauma often results in temporary activity limitation, hospitalization, and sometimes in permanent disability.[1,40] The Center of Disease Control's Web-based Injury Statistics Query and Reporting System (CDC WISQARS™) estimates that nonfatal injuries requiring medical attention affected more than 8.5 million children and adolescents and resulted in $24 billion in medical care and work loss costs (https://wisqars.cdc.gov:8443/costT/). As the leading cause of death and disability in children, pediatric trauma presents one of the largest challenges to the health of children, as well as an important opportunity for positive impact.

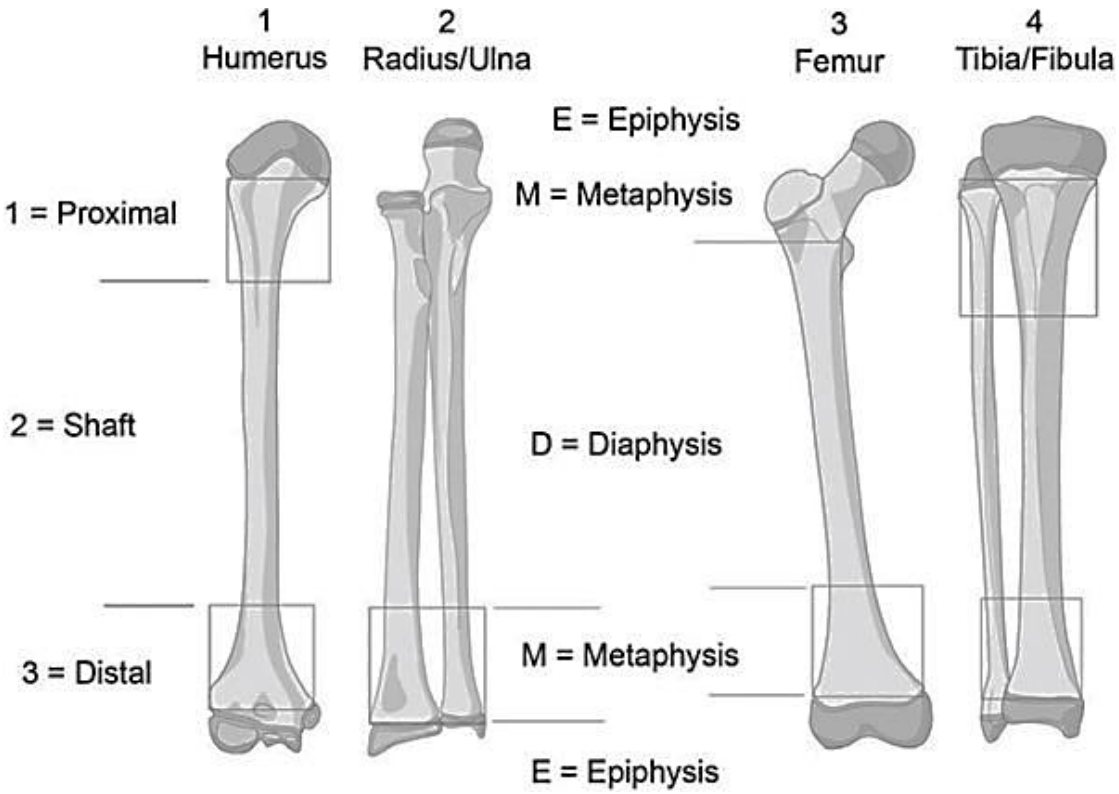
Incidence of Fractures in Children

"Classification Bias": Difficulties Defining Disease

Descriptive epidemiologic studies demand consistent information about how we define and classify a given disease state. This is a challenge in pediatric trauma, making it difficult to compare studies. An international study group has developed and performed early validation of a standardized classification system of pediatric fractures.[96,97,98,99] The authors of an agreement study found that with appropriate training, the AO Pediatric Comprehensive Classification of Long Bone Fractures (PCCF) system could be used by experienced surgeons as a reliable classification system for pediatric fractures for future prospective studies (Fig. 1-1).[96,99] In addition, follow-up studies have provided useful epidemiologic reporting of pediatric long-bone fractures using the AO PCCF.[5,33,34,35]

Figure 1-1 The AO PCCF for fracture classification with bone, segment, and subsegment nomenclature.

(From Slongo TF, Audige L. Fracture and dislocation classification compendium for children: the AO Pediatric Comprehensive Classification of Long Bone Fractures (PCCF). *J Orthop Trauma*. 2007;21(10 Suppl):S135–S160 .)



The incidence of pediatric fractures differs among published series because of geographical, environmental, gender, and age differences. Early studies on the incidence of fractures in children formed a knowledge base about fracture healing in children. Landin's 1983 report on 8,682 fractures remains a landmark study on the incidence of fractures in children.[45] He reviewed the data on all fractures in children that occurred in Malmö, Sweden, over 30 years and examined the factors affecting the incidence of children's fractures. By studying two populations, 30 years apart, he determined that fracture patterns were changing and suggested reasons for such changes. His initial goal was to establish data for preventive programs, so he focused on fractures that produced clean, concise, concrete data. Lempesis provided the most recent update from Malmö, Sweden over the years 2005 to 2006 and noted the previously reported declines in overall fracture rate remained unchanged and may have been related to a change in the region's demographics. There was however a decrease in incidence among girls. The pediatric fracture incidence during the period 2005 to 2006 was 1,832 per 10,000 person-years (2,359 in boys and 1,276 in girls), with an age-adjusted boy-to-girl ratio of 1.8 (1.6% to 2.1%).[48]

More recently, studies on the incidence of fractures in Edinburgh, Scotland in 2000, as reviewed by Rennie et al.,[84] was 20.2 per 1,000 children annually. A similar fracture incidence of 201/10,000 among children and adolescents was reported in northern Sweden between 1993 and 2007 with a 13% increase during the years between 1998 and 2007. The authors also reported the accumulated risk of sustaining a fracture before the age of 17 being 34%.[29] In Landin's series from Malmö, Sweden, the chance of a child sustaining a fracture during childhood (birth to age 16) was 42% for boys and 27% for girls.[45] When considered on an annual basis, 2.1% of all the children (2.6% for boys; 1.7% for girls) sustained at least one fracture each year. These figures were for all fracture types and included those treated on an inpatient basis and an outpatient basis. The overall chance of fracture per year was 1.6% for both girls and boys in a study from England of both outpatients and inpatients by Worlock and Stower.[114] The chance of a child sustaining a fracture severe enough to require inpatient treatment during the first 16 years of life is 6.8%.[10] Thus, on an annual basis, 0.43% of the children in an average community will be admitted for a fracture-related problem during the year. The overall incidence and lifetime risk of children's fractures are summarized in Table 1-1.

Overall Frequency of Fractures[16,30,36,46,57,68]

Percentage of children sustaining at least one fracture from 0–16 yrs of age:
Boys, 42–60%
Girls, 27–40%
Percentage of children sustaining a fracture in 1 yr: 1.6–2.1%
Annual rate of fracture in childhood: 12–36/1,000 persons

Early reports of children's fractures grouped the areas fractured together, and fractures were reported only as to the long bone involved (e.g., radius, humerus, femur). More recent reports have split fractures into the more specific areas of the long bone involved (e.g., the distal radius or the distal humerus). In children, fractures in the upper extremity are much more common than those in the lower extremity.[115] Overall, the radius is the most commonly fractured long bone, followed by the humerus. In the lower extremity, the tibia is more commonly fractured than the femur (Table 1-2).[35]

Incidence of Fractures in Long Bones

Bone	*%*
Radius/ulna	59
Humerus	21
Tibia/fibula	15
Femur	5

From Joeris A, Lutz N, Wicki B, et al. An epidemiological evaluation of pediatric long bone fractures: a retrospective cohort study of 2716 patients from two Swiss tertiary pediatric hospitals. *BMC Pediatr*. 2014;14:314 © Joeris et al; licensee BioMed Central. 2014.

The individual reports agree that the most common area fractured in children is the distal radius. The next most common area involves the hand (phalanges and metacarpals), clavicle and distal humerus.[46,71,83,84]

Physeal Fractures

The incidence of physeal injuries overall varied from 14.8% to as high as 30% in the literature across various series.[37,60,63,77,84,106]

Open Fractures

The overall reported incidence of open fractures in children has changed over time ranging 1.5% to 2.6% in older series[10,60,114] to 0.7% to 1% in recent reports.[35,84]Regional trauma centers often see patients exposed to more severe trauma, so there may be a higher incidence of open fractures in these patients. The incidence of open fractures was 9% in a report of patients admitted to an urban trauma center.[7]

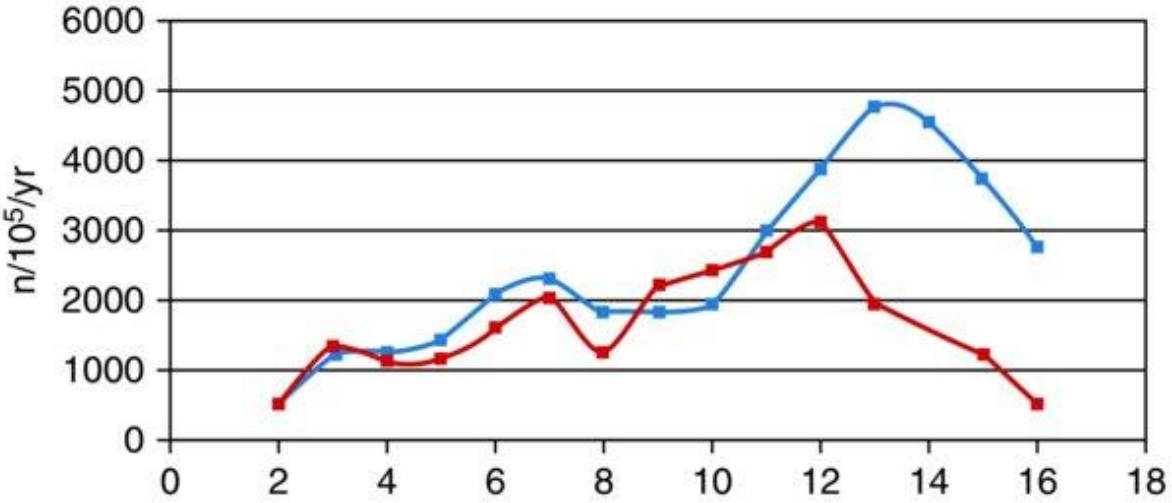
Despite the importance of understanding the epidemiology of pediatric fractures, there are still significant gaps in our knowledge base, and there is much work to be done. There are several challenges to gathering appropriate data in this area: risk factors for pediatric injury are diverse and heterogeneous, practice patterns vary across countries and even within countries, and the available infrastructure to support data collection for pediatric trauma is far from ideal.

Patient Factors That Influence Fracture Incidence and Fracture Patterns

Age

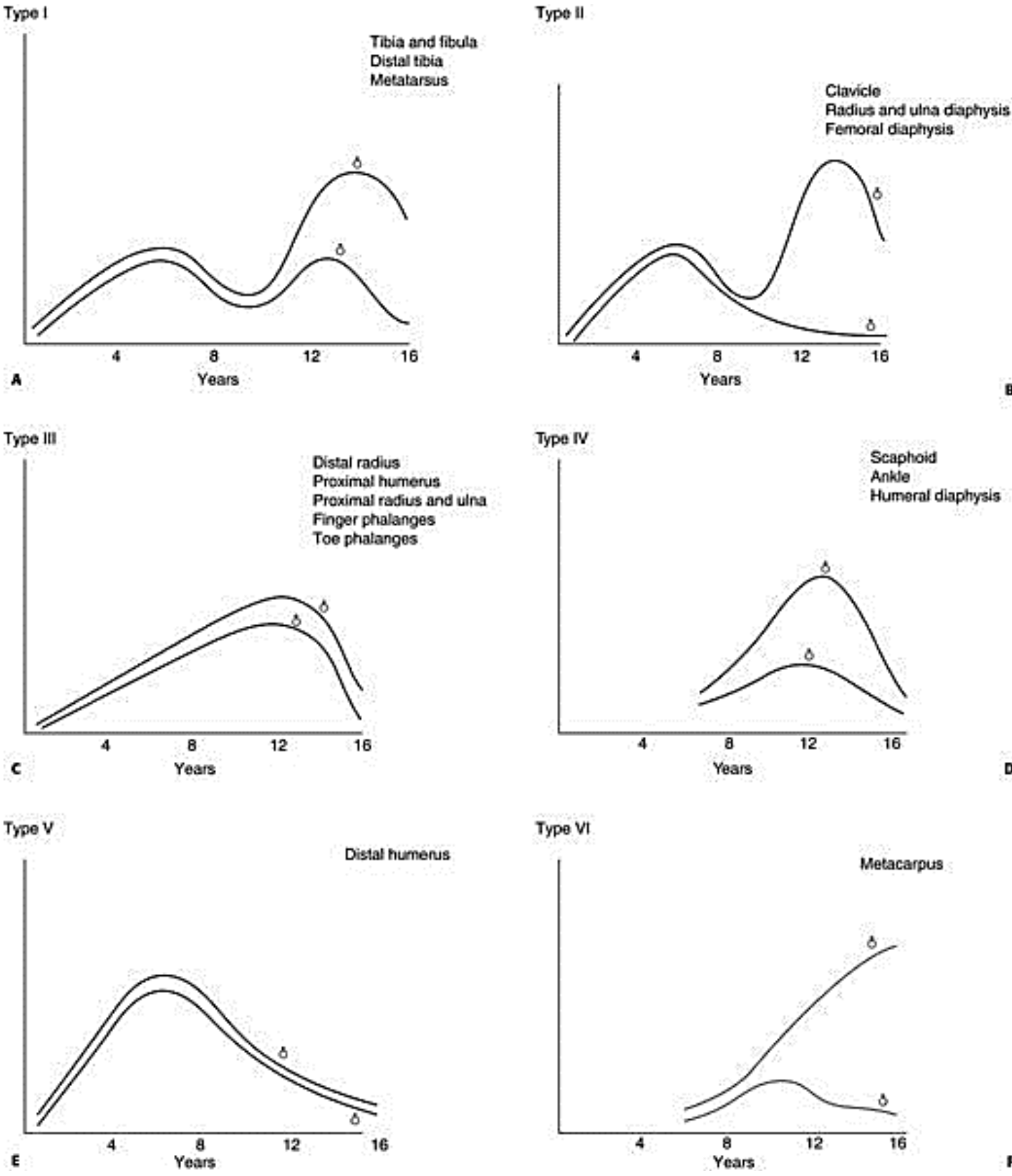
Fracture incidence in children increases with age. Age-specific fracture patterns and locations are influenced by many factors including age-dependent activities and changing intrinsic bone properties. Starting with birth and extending to age 12, all the major series that segregated patients by age have demonstrated a linear increase in the annual incidence of fractures with age (Fig. 1-2). The peak age for fracture occurrence in girls is age 11 to 12 and for boys it is age 13 to 14.[16,28,36,83,84]

Figure 1-2 Incidence of fractures by age.
Boys (*blue*) peak at 13 years whereas girls (*red*) peak earlier, at 12 years, and then decline. (Reprinted from Rennie L, Court-Brown CM, Mok JY, et al. The epidemiology of fractures in children. *Injury*. 2007;38(8):913–922 . Copyright © 2007 Elsevier Ltd. With permission.)



Although there is a high incidence of injuries in children of ages 1 to 2, the incidence of fractures is low with most fractures being related to accidental or nonaccidental trauma from others.[14,42] The anatomic areas most often fractured seem to be the same in the major series, but these rates change with age. Rennie et al.[84] demonstrated in their 2000 study from Edinburgh that the incidence of fractures increased and fracture patterns changed as children aged. Fracture incidence curves for each of the most common fractures separated by gender were shown on six basic incidence curves similar to Landin's initial work (Fig. 1-3).[45] When Landin compared these variability patterns with the common etiologies, he found some correlation. For example, late-peak fractures (distal forearm, phalanges, proximal humerus) were closely correlated with sports and equipment etiologies. Bimodal pattern fractures (clavicle, femur, radioulnar, diaphyses) showed an early increase from lower-energy trauma, then a late peak in incidence caused by injury from high- or moderate-energy trauma likely caused by motor vehicle accidents (MVAs), recreational activities, and contact sports in the adolescent population. Early-peak fractures (supracondylar humeral fractures are a classic example) were mainly caused by falls from high levels.

Figure 1-3 Variations of fracture patterns with age.
The peak ages for the various fracture types occur in one of six patterns. (Reprinted from Rennie L, Court-Brown CM, Mok JY, et al. The epidemiology of fractures in children. *Injury*. 2007;38(8):913–922 . Copyright © 2007 Elsevier Ltd. With permission.)



Gender

Gender differences can be seen across the incidence of injures, location of injuries, and etiology of injuries across all age groups. For all age groups, the overall ratio across a number of series of boys to girls which sustains a single fracture is about 1.5:1.[16,29,30,36,46,84] In some areas, there is little difference in the incidence of fractures between boys and girls. For example, during the first 2 years of life, the overall incidence of injuries and fractures in both genders is nearly equal. During these first 2 years, the injury rates for foreign-body ingestion, poisons, and burns have no significant gender differences. With activities in which there is a male difference in participation, such as with sports equipment and bicycles, there is a marked increase in the incidence of injuries in boys.[9,85]The injury incidence may not be

caused by the rate of exposure alone; behavior may be a major factor.[107] For example, one study found that the incidence of auto/pedestrian childhood injuries peaks in both sexes at ages 5 to 8.[86] When the total number of street crossings per day was studied, both sexes did so equally. Despite this equal exposure, boys had a higher number of injuries. Thus, the difference in the rate between the sexes begins to develop a male predominance when behaviors change.

Socioeconomic and Cultural Differences

The incidence of pediatric fracture varies in different geographic settings, socioeconomic climates, and differing ethnicities. Two studies from the United Kingdom looked at the relationship of affluence to the incidence of fractures in children and had differing conclusions. Lyons et al.[56] found no difference in the fracture rates of children in affluent population groups compared to those of children in nonaffluent families. On the other hand, Stark et al.[103] in Scotland found that the fracture rates in children from nonaffluent social groups were significantly higher than those in affluent families. There are also contradictory results in the literature with regard to fracture risk associated with living urban versus rural settings.[21,30] In the United States, the increased rate of pediatric femur fractures was influenced by adverse socioeconomic and sociodemographic fractures.[32]Wren et al.[115] in a large prospective cohort studied the association of race and ethnicity as a risk factor for fracture in children and adolescents. They found that fracture rates were higher, regardless of sex, for white children compared with all other racial and ethnic groups.

Clinical Factors

In recent years there has been an attention to a number of clinically related factors in determining children's fractures, such as obesity, low bone mineral density (BMD), and low calcium and vitamin D intake. Obesity is an increasing health problem in children and adolescents representing a complex interaction of host factors, and is the most prevalent nutritional problem for children in the United States. In a retrospective chart review, Taylor et al.[105] noted that overweight children had a higher-reported incidence of fractures and musculoskeletal complaints. Although Leonard et al.[50] found increased BMD in obese adolescents, the lack of physical activity often seen in obesity may in fact lead to reduced muscle mass, strength, and coordination resulted in impaired proprioception, balance and increased risk of falling and fracture. In a recent study, Valerio et al.[108]confirmed a greater prevalence of overweight/obesity in children and adolescents with a recent fracture when compared to age- and gender-matched fracture-free children, and found obesity rate was increased in girls with upper limb fractures and girls and boys with lower limb fractures. Low BMD and decreased bone mass are linked to increased fracture risk in the adult population; however, in children, the relationship is less clear with a meta-analysis showing some association between fracture risk and low BMD.[13] In 2006, Clark examined in a prospective fashion the association between bone mass and fracture risk in childhood. Over 6,000 children at 9.9 years of age were followed-up for 2 years and the study showed an 89% increased risk of fracture per standard deviation (SD) decrease in size-adjusted BMD.[11] In a follow-up study of this same cohort, the risk of fracture following slight or moderate to severe trauma was inversely related to bone size relative to body size perhaps reflecting the determinants of volumetric BMD such as cortical thickness on skeletal fragility.[12] Nutritional factors may also play a role in the incidence of fractures in children.

The Impact of Environmental Factors on Fractures in Children

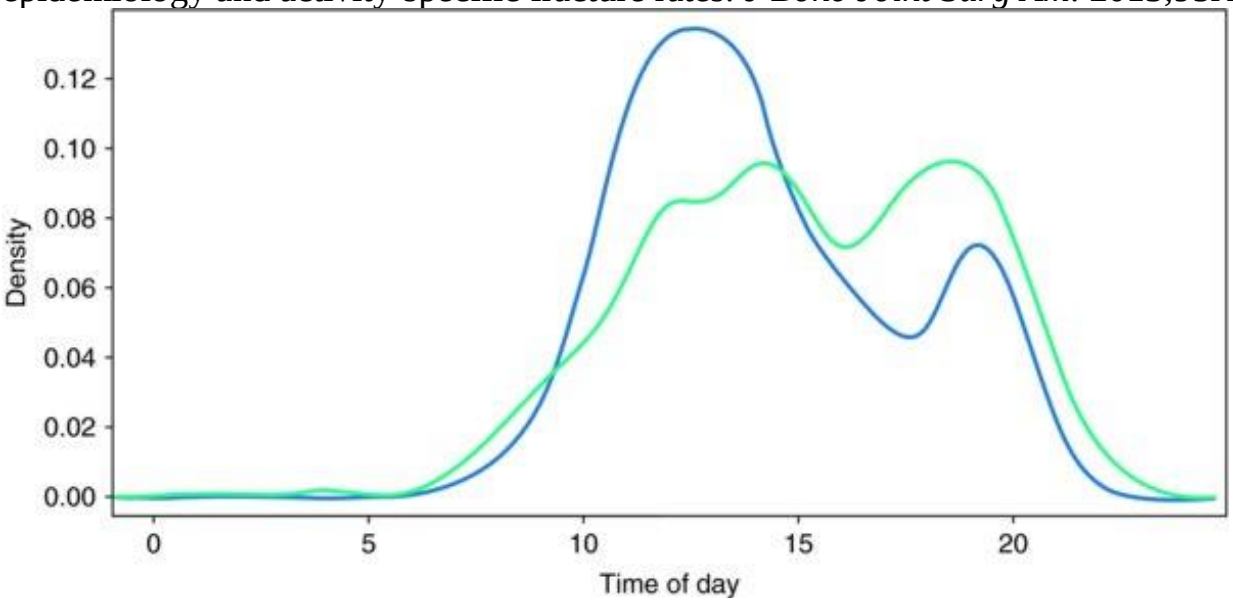
Seasonal and Climatic Differences

Fractures are more common during the summer, when children are out of school and exposed to more vigorous physical activities. An analysis of seasonal variation in many studies shows an increase in fractures in the warmer months of the year.[9,10,29,45,83,84,95,111,114]
Children in colder climates, with ice and snow, are exposed to risks different from those of children living in warmer climates. The exposure time to outdoor activities may be greater for children who live in dry and warm weather climates.[94] The most consistent climatic factor appears to be the number of hours of sunshine. Masterson et al.,[61] in a study from Ireland, found a strong positive correlation between monthly sunshine hours and monthly fracture admissions. There was also a weak negative correlation with monthly rainfall. Overall, the average number of fractures in the summer was 2.5 times than that in the winter. In days with more sunshine hours than average, the average fracture admission rate was 2.31/day; on days with fewer sunshine hours than average, the admission rate was 1.07/day. Pediatric trauma should be viewed as a disease where there are direct and predictable relationships between exposure and incidence.

Time of Day

The time of day in which children are most active seems to correlate with the peak time for fracture occurrence. Seasonal variation and geographic location seem to play a role as to which time during the day injury occurs (Fig. 1-4).[61] In a Swedish study, the incidence peaked between 2 PM and 3 PM,[83] whereas in a study out of Texas by Shank et al.,[73]the hourly incidence of fractures formed a well-defined bell curve peaking at about 6 PM.

Figure 1-4 Distribution of fractures during time of day by summertime (green) and wintertime (blue).
Density estimates are computed using kernel-smoothing method with normal kernel function and suitable bandwidth. The *x* axis represents the hours in 5-hour intervals throughout the day (i.e.,*0*, midnight; *5*, 5 AM; *10*, 10 AM; *15*, 3 PM; and *20*, 8 PM), and the *y* axis represents the probability density that a fracture would occur at any given time of day.
(Redrawn from Randsborg PH, Gulbrandsen P, Saltyte Benth J, et al. Fractures in children: epidemiology and activity-specific fracture rates. *J Bone Joint Surg Am*. 2013;95A:e42.)



Home Environment

Fractures sustained in the home environment are defined as those that occur in the house and surrounding vicinity. These generally occur in a fairly supervised environment and are mainly caused by falls from furniture, stairs, fences, and trees as well as from injuries sustained from recreational equipment (trampolines and home jungle gyms). Falls can vary in severity from a simple fall while running, to a fall of great magnitude, such as from a third

story window. In falling from heights, adults often land on their lower extremities, accounting for the high number of lower-extremity fractures, especially the calcaneus. Children tend to fall head first, using the upper extremities to break the fall. This accounts for the larger number of skull and radial fractures in children. Femoral fractures also are common in children falling from great heights. In contrast to adults, spinal fractures are rare in children who fall from great heights.[90] In one study, children falling three stories or less all survived. Falls from the fifth or sixth floor resulted in a 50% mortality rate.[6,62,93,102] Interestingly, a Swedish study showed that an increased incidence of fractures in a home environment did not necessarily correlate with the physical attributes or poor safety precautions of the house.[6] Rather, it appears that a disruption of the family structure and presence of social handicaps (alcoholism, welfare recipients, etc.) are important risk factors for pediatric fracture.

School Environment

The supervised environments at school are generally safe, and the overall annual rate of injury (total percentage of children injured in a single year) in the school environment ranges from 2.8% to 16.5%.[73] Most injuries occur as a result of use of playground or recreational equipment or participation in athletic activity. True rates may be higher because of inaccurate reporting, especially of mild injuries. The annual fracture rate of school injuries is thought to be low. Of all injuries sustained by children at school in a year, only 5% to 10% involved fractures.[52] In Worlock and Stower's series of children's fractures from England,[24,49,91] only 20% occurred at school. Most injuries (53%) occurring in school are related to athletics and sporting events,[114] and injuries are highest in the middle school children, with one study citing a 20% fracture rate in school-aged children of those injured during physical education class.[49]

Etiology of Fractures in Children

Three Broad Causes

Broadly, fractures have three main causes: accidental trauma, nonaccidental trauma (child abuse), and pathologic conditions. Accidental trauma forms the largest etiologic group and can occur in a variety of settings, some often overlapping others. Nonaccidental trauma and fractures resulting from pathologic conditions are discussed in later chapters of this book.

Sports-Related Activities

The last two decades have seen an increase in youth participation in organized athletic participation, especially among younger children. Wood et al. studied at the annual incidence of sports-related fractures in children 10 to 19 years presenting to hospitals in Edinburgh. The overall incidence was 5.63/1,000/yr with males accounting for 87% of fractures. Soccer, rugby, and skiing were responsible for nearly two-thirds of the fractures among the 30 sporting activities that adolescents participated in. Upper-extremity fractures were by far the most common injury accounting for 84% of all fractures with most being low-energy injuries and few requiring operative intervention.[74] A retrospective study over a 16-year time period from an emergency department at a level 1 trauma center in the Netherlands examined risk factors for upper-extremity injury in sports-related activities. Most injuries occurred while playing soccer and upper-extremity injuries were most common. Risk factors for injury were young age and playing individual sports, no-contact sports, or no-ball sports. Women were at risk in speed skating, in-line skating, and basketball, whereas men mostly were injured during skiing and snowboarding.[113]

In the United States, football- and basketball-related injuries are common complaints presenting to pediatric emergency departments, with fractures occurring more frequently in football.[22] In a 5-year survey of the NEISS National Electronic Injury Surveillance System

(NEISS)-All Injury Program, injury rates ranged from 6.1 to 11 per 1,000 participants/year as age increased, with fractures and dislocations accounting for nearly 30% of all injuries receiving emergency room evaluation.[64]

Recreational Activities and Devices

In addition to increasing participation in sports, new activities and devices[65] have emerged that expose children to increased fracture risk. Traditional activities such as skateboarding, roller skating, alpine sports, and bicycling have taken on a new look in the era of extreme sports where such activities now involve high speeds and stunts. Many of these activities have safety equipment available but that does not assure compliance. Organizations such as the American Academy of Pediatrics and the American Academy of Orthopaedic Surgeons (AAOS) have issued position statements regarding the proper use and supervision of such devices, but it remains within the duty of the physician to educate and reinforce to patients and families to promote safety around these activities.[54]

Playground Equipment

Play is an essential element of a child's life. It enhances physical development and fosters social interaction. Unfortunately, unsupervised or careless use of some play equipment can endanger life and limb.[44] When Mott et al.[66] studied the incidence and pattern of injuries to children using public playgrounds, they found that approximately 1% of children using playgrounds sustained injuries. Swings, climbers, and slides are the pieces of playground equipment associated with 88% of the playground injuries.[58]

In a study of injuries resulting from playground equipment, Waltzman et al.[110] found that most injuries occurred in boys (56%) with a peak incidence in the summer months. Fractures accounted for 61% of these injuries, 90% of which involved the upper extremity and were sustained in falls from playground equipment such as monkey bars and climbing frames. Younger children (1 to 4 years old) were more likely to sustain fractures than older children. Lillis and Jaffe[51] made similar observations in a study in which upper-extremity injuries, especially fractures, accounted for most of hospitalizations resulting from injuries on playground equipment. Older children sustained more injuries on climbing apparatus, whereas younger children sustained more injuries on slides.

Loder[53] used the NEISS dataset to explore the demographics of playground equipment injuries in children. Monkey bars were the most common cause of fractures. In another study looking specifically at injuries from monkey bars, the peak age group was the 5- to 12-year-old group, with supracondylar humeral fractures being the most common fracture sustained.[59]

The correlation of the hardness of the playground surface with the risk of injury has been confirmed in numerous studies.[43,53,67,69] Changing playground surfaces from concrete to more impact-absorbing surfaces such as bark reduced the incidence and severity of head injury but increased the tendency for long-bone fractures (40%), bruises, and sprains.

Public playgrounds appear to have a higher risk for injuries than private playgrounds because they usually have harder surfaces and higher pieces of equipment,[78] although playground injury was most likely to occur at school compared to home, public, and other locations.[79]

Bicycle Injuries

Bicycle injuries are a significant cause of mortality and morbidity for children.[82] Bicycle mishaps are the most common causes of serious head injury in children.[112] Boys in the 5- to 14-year age group are at greatest risk for bicycle injury (80%). Puranik et al.[82] studied the profile of pediatric bicycle injuries in a sample of 211 children who were treated for bicycle-related injury at their trauma center over a 4-year period. They found that bicycle injuries accounted for 18% of all pediatric trauma patients. Bicycle/motor vehicle collisions caused 86% of injuries. Sixty-seven percent had head injuries and 29% sustained fractures. More

than half of the incidents occurred on the weekend. Sixteen percent were injured by ejection from a bicycle after losing control, hitting a pothole, or colliding with a fixed object or another bicycle. Fractures mainly involved the lower extremity, upper extremity, skull, ribs, and pelvis in decreasing order of incidence. Over the last decade, youth participation in mountain biking has seen an increase and with that so has the number of injuries related to mountain biking increased with many caused by unpredictable terrain and falls as one rides downhill.[2,3] As public awareness of both the severity and preventability of bicycle-related injuries grows, the goal of safer bicycling practices and lower injury rates can be achieved.[82]

Skateboarding

Skateboarding and in-line skating have experienced a renewed surge in popularity over the past three decades. With the increasing number of participants, high-tech equipment development, and vigorous advertising, skateboard and skating injuries are expected to increase. Since the late 1990s, there has been an increase in the number of skateboard injuries.[41] Because the nature of skateboarding encompasses both high speed and extreme maneuvers, high-energy fractures and other injuries can occur, as highlighted by several published reports.[25,75,80] Studies have shown that skateboarding-related injuries are more severe and have more serious consequences than roller skating or in-line skating injuries.[75] In a study of skateboarding injuries, Fountain et al.[25] found that fractures of the upper or lower extremity accounted for 50% of all skateboarding injuries. Interestingly, more than one-third of those injured sustained injuries within the first week of skateboarding. Most injuries occurred in preadolescent boys (75%) from 10 to 16 years of age; 65% sustained injuries on public roads, footpaths, and parking lots. In a study over a 5-year period of time using data from the National Trauma Data Bank, skateboarding injuries were associated with a higher incidence of closed-head injuries and long-bone fractures with children under age 10 more likely to sustain a femur fracture.[55] Several authors[25] have recommended safety guidelines and precautions such as use of helmets, knee and elbow pads, and wrist guards, but such regulations seldom are enforced.

Trampolines

Trampolines enjoyed increasing popularity in the 1990s and are a significant cause of morbidity in children. Several studies have noted a dramatic increase in the number of pediatric trampoline injuries during the past 10 years, rightfully deeming it as a “national epidemic.”[26,100] Using the NEISS data, Smith[100] estimated that there are roughly 40,000 pediatric trampoline injuries per year. Younger children had a higher incidence of upper-extremity fractures and other injuries. Furnival et al.,[26] in a retrospective study over a 7-year period, found that the annual number of pediatric trampoline injuries tripled between 1990 and 1997. In contrast to other recreational activities in which boys constitute the population at risk, patients with pediatric trampoline injuries were predominantly girls, with a median age of 7 years. Nearly a third of the injuries resulted from falling off the trampoline. Fractures of the upper and lower extremities occurred in 45% and were more frequently associated with falls off the trampoline. In a later study, Sandler et al.[87] reported injuries requiring surgery in over 60% of patients with 20% requiring operative fixation for upper-extremity fractures. These researchers, along with others,[26]rightly concluded that use of warning labels, public education, and even direct adult supervision were inadequate in preventing these injuries and have called for a total ban on the recreational, school, and competitive use of trampolines by children.[20,26,87,100,101]

Motor Vehicle Accidents

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

The injury patterns of children involved in MVAs differ from those of adults. In all types of MVAs for all ages, children constitute a little over 10% of the total number of patients injured.[45,89] Of all the persons injured as motor vehicle occupants, only about 17% to 18% are children. Of the victims of vehicle-versus-pedestrian accidents, about 29% are children. Of the total number of children involved in MVAs, 56.4% were vehicle–pedestrian accidents, and 19.6% were vehicle–bicycle accidents.[23]

The fracture rate of children in MVAs is less than that of adults. Of the total number of vehicle–pedestrian accidents, about 22% of the children sustained fractures; 40% of the adults sustained fractures in the same type of accident. This has been attributed to the fact that children are more likely to "bounce" when hit.[23]

Children are twice as likely as adults to sustain a femoral fracture when struck by an automobile; in adults, tibial and knee injuries are more common in the same type of accident. This seems to be related to where the car's bumper strikes the victim.[7,8] MVAs also produce a high proportion of spinal and pelvic injuries.[7]

All-Terrain Vehicles

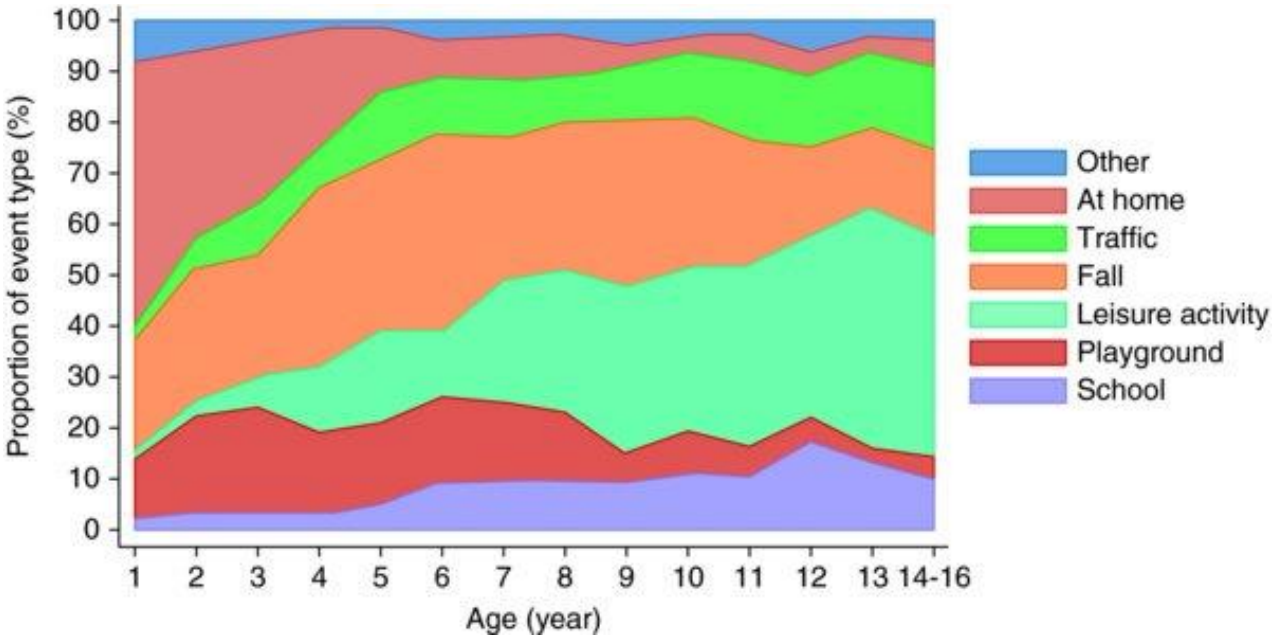
Recreational all-terrain vehicles (ATVs) have emerged as a new cause of serious pediatric injury. Despite product training and safety education campaigns, ATV accidents continue to cause significant morbidity and mortality in children and adolescents.[28,31,38,39,88,92] In contrast to other etiologies of injury, children who sustained ATV-related fractures had more severe injuries and a higher percentage of significant head trauma, with 1% of these injuries resulting in inhospital death. These statistics point to the failure of voluntary safety efforts to date and argue for much stronger regulatory control.

In a review of 96 children who sustained injuries in ATV-related accidents during a 30-month period, Kellum et al.[38] noted age-related patterns of injury. Younger children (≤12 years) were more likely to sustain an isolated fracture and were more likely to sustain a lower-extremity fracture, specifically a femoral fracture, than older children. Older children were more likely to sustain a pelvic fracture. In a recent review of the Kids' Inpatient Database, Sawyer et al.[88] found that despite the known risks associated with ATV use in children, their use and injury rate continue to increase. The injury rate for children from ATV accidents has increased 240% since 1997, whereas the spinal injury rate has increased 476% over the same time frame. The authors found that injuries to the spinal column occurred in 7.4% of patients with the most common level of fracture was thoracic (39%), followed by lumbar (29%) and cervical (16%). Pelvic fractures were the most common associated fractures, accounting for 44% of all musculoskeletal injuries, followed by forearm/wrist fractures (15%) and femoral fractures (9%). Despite educational and legislative efforts, children account for a disproportionate percentage of morbidity and mortality from ATV-related accidents. The sport of motocross has also been shown to have a high rate of musculoskeletal injuries requiring hospitalization in children.[47]

The etiologic aspects of children's fractures are summarized in Figure 1-5.

Figure 1-5 Accident types correlated to age.

There is a predominance of fractures due to accidents at home throughout the first 4 years of life, whereas leisure activities become the leading cause during school-age and adolescence. (Redrawn from Joeris A, Lutz N, Wicki B, et al. An epidemiological evaluation of pediatric long bone fractures: a retrospective cohort study of 2716 patients from two Swiss tertiary pediatric hospitals. *BMC Pediatr*. 2014;14:314 . © Joeris et al.; licensee BioMed Central. 2014.)



Gunshot and Firearm Injuries

Gunshot or missile wounds arise from objects projected into space by an explosive device. Gunshot wounds have become increasingly common in children in the United States. In a reflection of the changing times and pervasive gun culture, firearms are determined to be second only to motor vehicles as the leading cause of death in youths. In considering the prevalence of firearms in the United States, it has been estimated that there are about 200 million privately owned guns in the United States and that approximately 40% of US households contain firearms of some type.[17]

Two recent epidemiologic reports from pediatric trauma centers, orthopedic injuries related to firearms, demonstrated varying rates of complications and need for operative intervention (35% and 54%) in gunshot wound–related injuries and complications.[72,76] Perkins et al.[76] found that in 46 patients with 50 injuries, gunshot-related fractures had an increased incidence of permanent neurologic deficits, infection (11%), and fracture non-union rates (9%).

The two most common complications associated with firearm-related injuries are growth arrest and infection. Other complications included delayed union and malunion. The treatment of fractures associated with gunshot wounds in children is never simple. Bone defects, associated peripheral nerve injuries, and involvement of the joint can negatively influence outcomes.[4]

Firearm-related injury and safety have received much attention nationally and internationally in the wake of the events over the last decade. Rather than modifying behavioral or environmental issues, which are more complex, strategies to reduce firearm-related injuries and deaths among the youth include reducing the number of guns in the environment through restrictive legislation, gun buy-back programs, gun taxes, physician counseling, and modifying the design of guns to make them more childproof and prevent unauthorized and unintended use.

Evolving Epidemiology of Fractures in Children

Preventive Programs

While studying the epidemiology of fractures, it is important to focus on the etiology of fractures and the settings in which they occur. Fractures do not occur in a vacuum, and well-researched studies that analyze the physical and social environment in which they occur are extremely valuable. Efforts can be made toward creating a safer environment for play and

recreation. It is hoped that by targeting these areas, programs can be designed to decrease the risk factors.

National Campaigns

Several national organizations have developed safety programs. The foremost is the American Academy of Pediatrics, which has committees on injury and poison prevention and sports medicine and fitness that has produced guidelines for athletics,[15] playgrounds, trampolines,[20] ATVs,[18] and skateboards.[19] The AAOS has also produced a program designed to decrease the incidence of playground injuries. These programs offer background data and guidelines for various activities, but their effectiveness has not been fully studied. In addition, the AAOS, the Orthopaedic Trauma Association (OTA), and Pediatric Orthopaedic Society of North America (POSNA) have issued updated position statements regarding the safe use of ATVs, trampolines, skateboards, and in-line skating.

Expanded Opportunities to Examine the Epidemiology of Pediatric Trauma

Several sources of administrative, national, and regional data have recently become available, providing significantly improved investigation into various areas within pediatric trauma. The Healthcare Cost and Utilization Project (HCUP) is a family of databases including the State Inpatient Databases (SID), the Nationwide Inpatient Sample (NIS), and the KID. Although administrative data may lack clinical detail for certain purposes, these datasets provide a comprehensive overview of health care utilization in the United States and are available without purchase (http://www.ahrq.gov/research/data/hcup/index.html).[104] The KID database has been increasingly used to examine the incidence of pediatric trauma as well as practice patterns in pediatric trauma. Data for KIDS are collected and published every 3 years. In 2011 study, using the 2006 HCUP KID dataset, Gao[27]reported on lower-extremity fractures requiring hospitalization and found there were about 11,500 admission records for children aged 0 to 20 with lower-extremity fractures. Urban hospitalizations accounted for 93% of cases and 66% of admissions were to teaching hospitals in Gao's study. There was an increased mortality risk among patients cared for in nonteaching hospitals and hospitals located in a rural region. An additional study using this dataset, Nakaniida et al.[70] found femur and humerus fractures as the most common injuries requiring hospitalization, with pelvic and vertebral fractures largely due to MVAs representing the most costly injuries. Trauma registries are another source for injury data that document clinical and demographic information regarding acute care delivered to hospitalized patients with injuries at trauma centers. These databases are designed to provide information that can be used to study the effectiveness and quality of trauma care, collect information on rare injuries,[81,109] and identify areas for quality improvement. Although the amount of information available through regional and national databases allowed is immense, the creation and maintenance of these registries require a significant amount of time and financial resources. Several limitations of these databases include the focus on adult over pediatric injuries and the data that do not always reflect population-based samples. Currently, the American College of Surgeons National Trauma Data Bank serves as the largest database, producing annual reports on pediatric injury from trauma centers from the United States and Canada (http://www.ntdb.org). In the future, databases such as these may provide the infrastructure needed to study pediatric musculoskeletal trauma care.

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Annotated References

Reference	*Annotation*
Clark EM, Ness AR, Bishop NJ, et al. Association between bone mass and fractures in children: a prospective cohort study. *J Bone Miner Res*. 2006;21:1489–1495.	The largest prospective study on bone mass in nearly 6,000 children demonstrated low bone density as a risk factor for fractures in children.
Garay M, Hess J, Armstrong D, et al. Pediatric ATV injuries in a statewide sample: 2004 to 2014. *Pediatrics*. 2017;140(2).	Data over an 11-year period from a statewide trauma database was reviewed to report the incidence of ATV-related injuries, mortality rates, as well as associated fracture patterns.
Hedstrom EM, Waernbaum I. Incidence of fractures among children and adolescents in rural and urban communities: analysis based on 9,965 fracture events. *Inj Epidemiol*. 2014;1:14.	In a study of nearly 10,000 fracture events in children, known risk factors of age and sex were predictive of fracture incidence. The authors also found a lower rate of fractures in rural and less densely populated municipalities.
Joeris A, Lutz N, Wicki B, et al. An epidemiological evaluation of pediatric long bone fractures: a retrospective cohort study of 2716 patients from two Swiss tertiary pediatric hospitals. *BMC Pediatr*. 2014;14:314.	Using the AO Pediatric Comprehensive Classification of Long Bone Fractures (PCCF) the morphologic patterns of over 2,700 upper- and lower-extremity fractures are presented along with age, sex, and injury data.
Lempesis V, Rosengren BE, Nilsson JA, et al. Time trends in pediatric fracture incidence in Sweden during the period 1950–2006. *Acta Orthop*. 2017;88:440–445.	Epidemiologic studies on pediatric fractures have been ongoing in the Swedish city of Malmö since 1950. This most recent report describes the fracture epidemiology including etiology comparing this to historical data and describes changes in age- and sex-adjusted pediatric fracture incidences to identify time trends independent of changing population demographics.
Monteilh C, Patel P, Gaffney J. Musculoskeletal injuries associated with hoverboard use in children. *Clin Pediatr (Phila)*. 2017;56:909–911.	Hoverboards, which are self-balancing, elevated motorized scooters, have recently been introduced as recreational devices to children and adolescents. This early report describes the increase in fractures especially of the upper extremity with falls associated with this device.
Naranje SM, Gilbert SR, Stewart MG, et al. Gunshot-associated fractures in children and adolescents treated at two level 1 pediatric trauma centers. *J Pediatr Orthop*. 2016;36:1–5.	A recent retrospective review from two trauma centers reports on 49 patients with 58 gunshot-associated fractures in the pediatric population. While nearly two-thirds of patient's fractures were successfully managed nonoperatively, more than one-third required surgery to manage their fracture or associated complications.
Wren TA, Shepherd JA, Kalkwarf HJ, et al. Racial disparity in fracture risk between white and nonwhite children in the United States. *J Pediatr*. 2012;161:1035–1040.	A longitudinal study of nearly 1,500 children with data collected on diet, activity, ethnicity, and body composition including DXA measures reported on risk factors for fracture. Skeletal age 10–14 and white race along with increased sports

Reference	*Annotation*
	participation were some predictors of fracture risk in this population.

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2 The Injured Immature Skeleton

Karl E. Rathjen, Harry K. W. Kim, Benjamin A. Alman

Introduction

Roughly 20% of children will sustain a fracture, and the incidence of pediatric fractures is increasing over time.[62,93,123] Thus, childhood fractures are not only important from an orthopedic standpoint, but are also an important societal health issue. A child's bones heal quicker, are surrounded by thicker periosteum, and have a substantially greater remodeling potential than adults. These differences change the way fractures are treated. In a growing child, injury can damage the growth plate, which can be the weakest region of bone.[123] Such injury can result in temporary or permanent growth arrest. The growth plate can also remodel less than perfect reductions and there can be tremendous remodeling potential. As an example, the distal radius can correct deformities at a rate of one degree a month in line with the flexion and extension arc of motion of the wrist.[80,97,131]

Children can have underlying disorders that weaken bone or slow healing, such as osteogenesis imperfecta,[82,182] neurofibromatosis,[73] or a bone tumor.[105] The first presentation of such conditions may be a referral to an orthopedist for a fracture. Understanding how the bone normally behaves to an injury can help in the identification of a pathologic bone injury, which may be caused by such an underlying condition. For instance, an avulsion fracture, such as in the ulna, can be the first presentation for a preteen with osteogenesis imperfecta.[149] Identification of unusual fracture patterns can also help in the identification of nonaccidental trauma, and an orthopedist can save a child's life by the early identification of nonaccidental trauma in childhood.[110] For these reasons it is critical to understand the normal response of a child's bone to trauma to know when there is something unusual in the presentation or course of healing, to make these diagnoses.

Anatomic Considerations of the Immature Skeleton

Five regions characterize long bones: The bulbous, articular cartilage-covered ends (epiphyses) tapering to the funnel-shaped metaphyses, with the central diaphysis interposed between the metaphyses (Fig. 2-1). During growth, the epiphyseal and metaphyseal regions are separated by the organized cartilaginous physis, which is the major contributor to longitudinal growth of the bone. The larger long bones (clavicle, humerus, radius, ulna, femur, tibia, and fibula) have physes at both ends, whereas the smaller tubular bones (metacarpals, metatarsals, and phalanges) usually have a physis at one end only. The relative contribution to longitudinal growth of the proximal and distal physes of the long bones is variable and listed for the upper and lower extremities in Figure 2-2.

Figure 2-1 A schematic diagram of femur illustrating the terminology used to describe various anatomical regions of a growing bone.

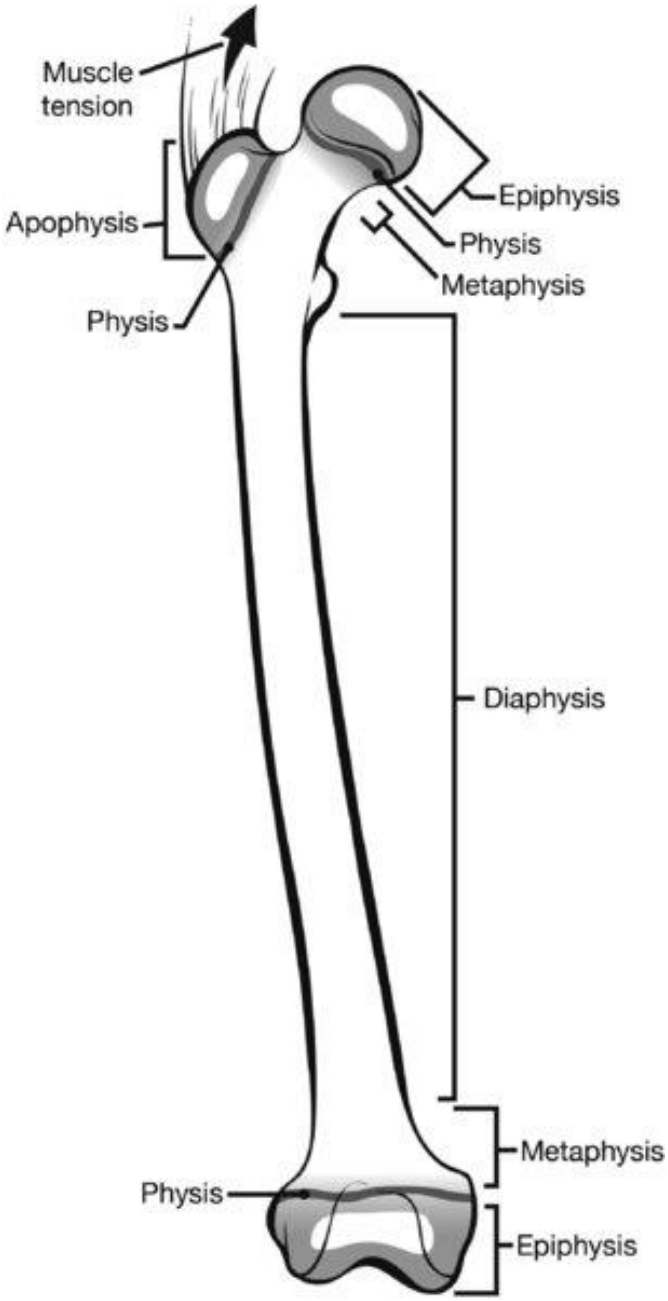
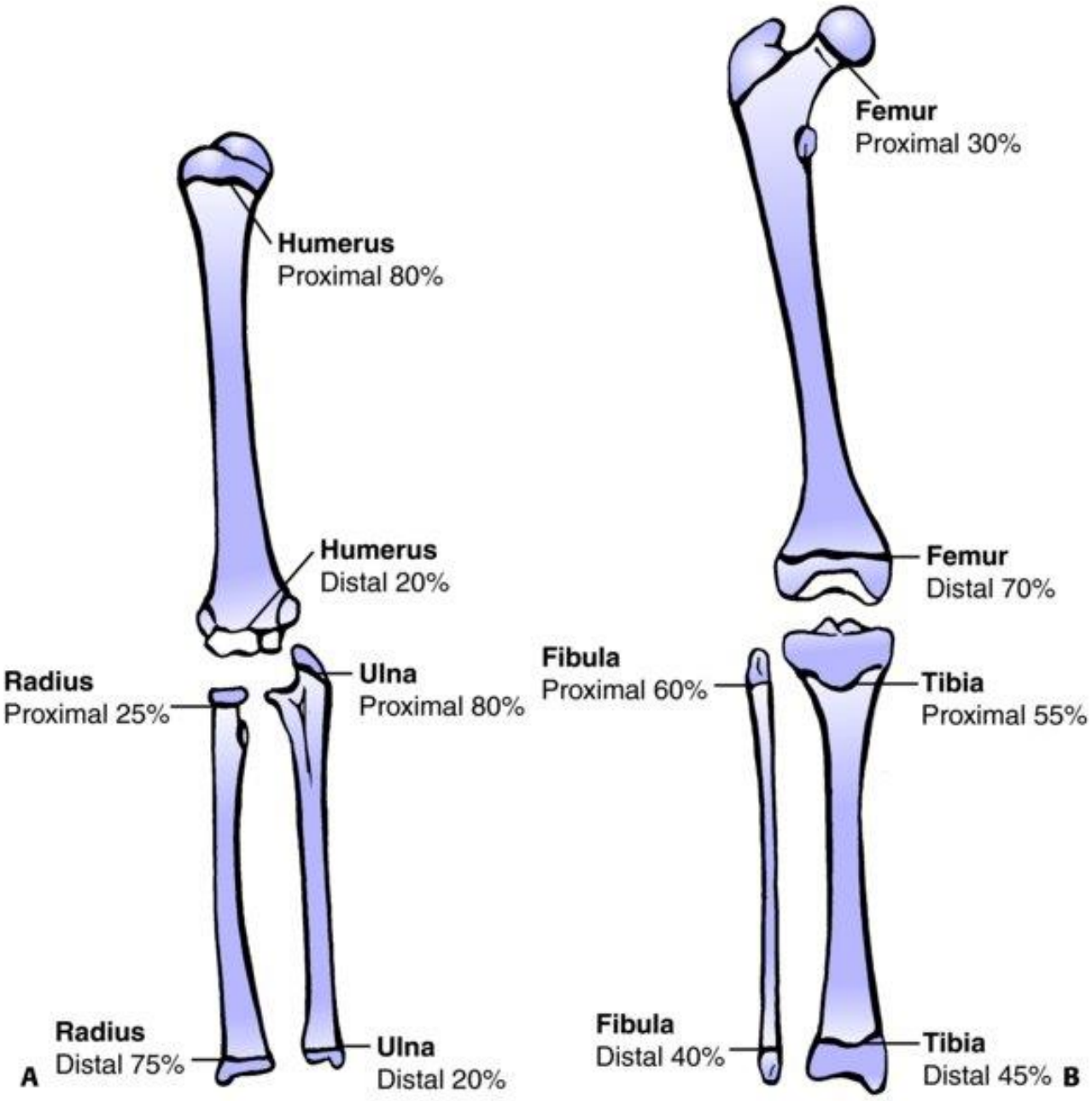


Figure 2-2 Approximate percentage of longitudinal growth provided by the proximal and distal physes for each long bone in the upper (A) and lower (B) extremities.



Epiphysis

The epiphysis is the region of a long bone between the end of the bone and the growth plate (or physis). At birth, the end of the bones is completely cartilaginous (except for the distal femur and occasionally the proximal tibia), and termed as chondroepiphysis. A secondary center of ossification forms at a specific time for each chondroepiphysis, which gradually enlarges until the cartilaginous area has been almost completely replaced by bone at skeletal maturity. The appearance of the ossification centers differs between different bones (Figs. 2-3 and 2-4), and this needs to be taken into account when diagnosing fractures of these regions.[22,136] As the ossification center matures, there is increased rigidity at the end of the bone, and this increase in rigidity is responsible for changes in the fracture pattern with age. Indeed, injuries that might not result in a fracture in this region in the very young may do so as the skeleton becomes more rigid.

Figure 2-3 Typical age (and range) of development of the secondary ossification centers of the epiphyses in the (A) upper extremity and (B) lower extremity.

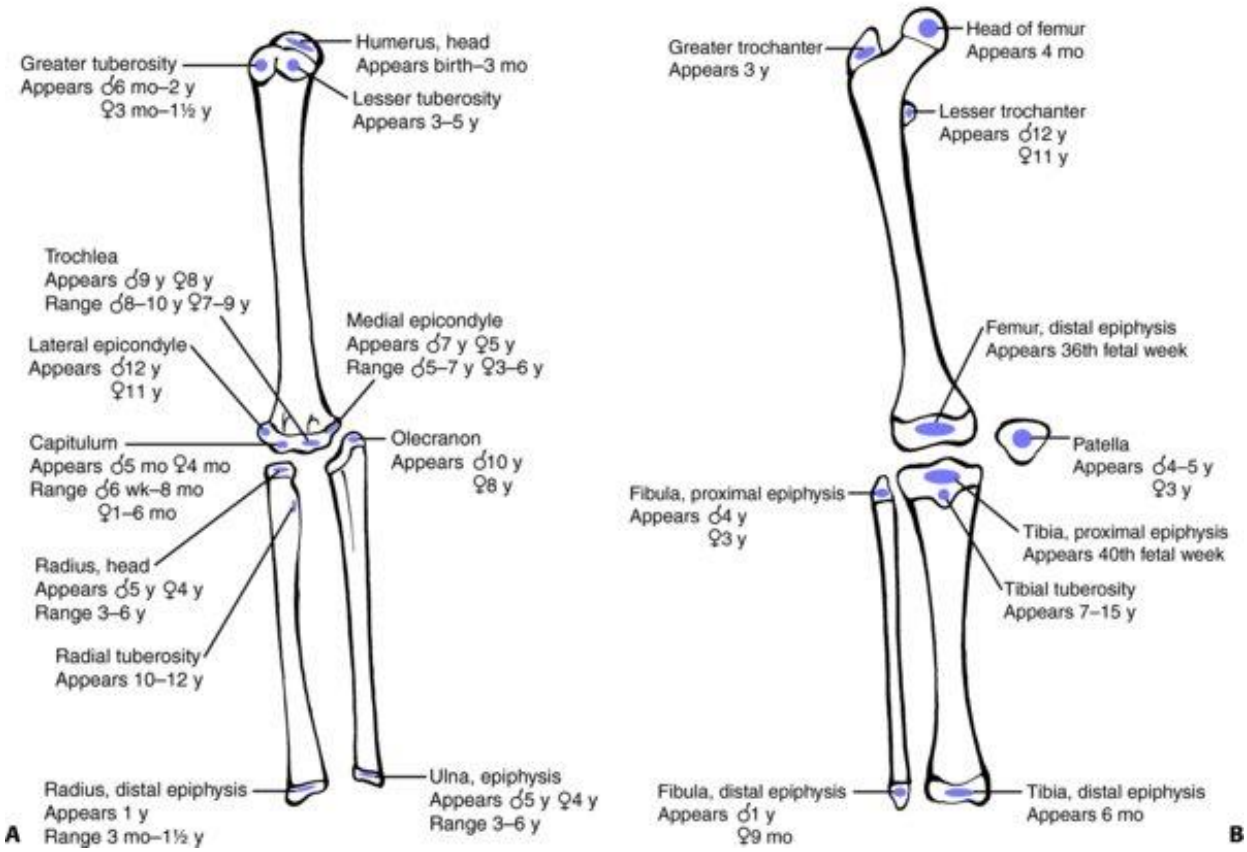
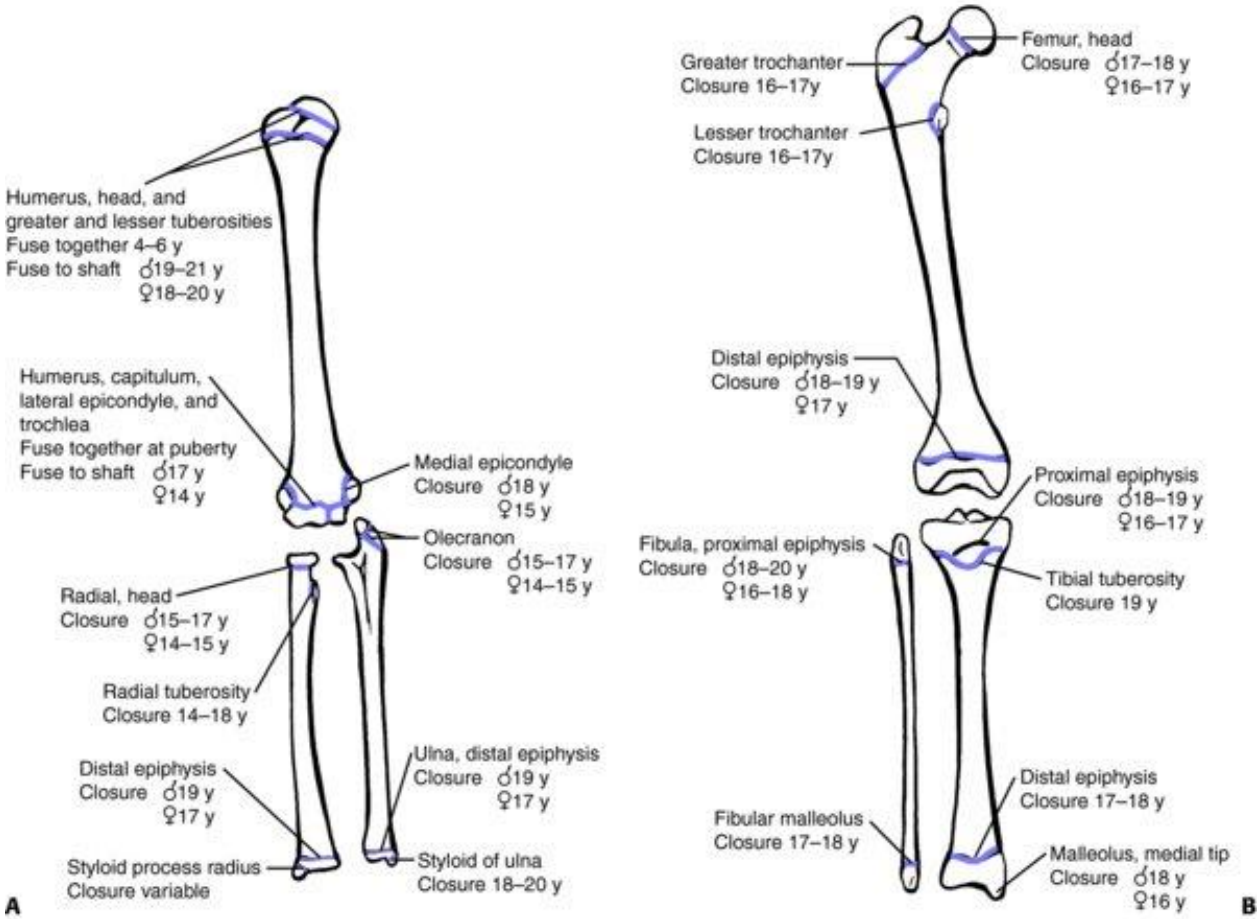


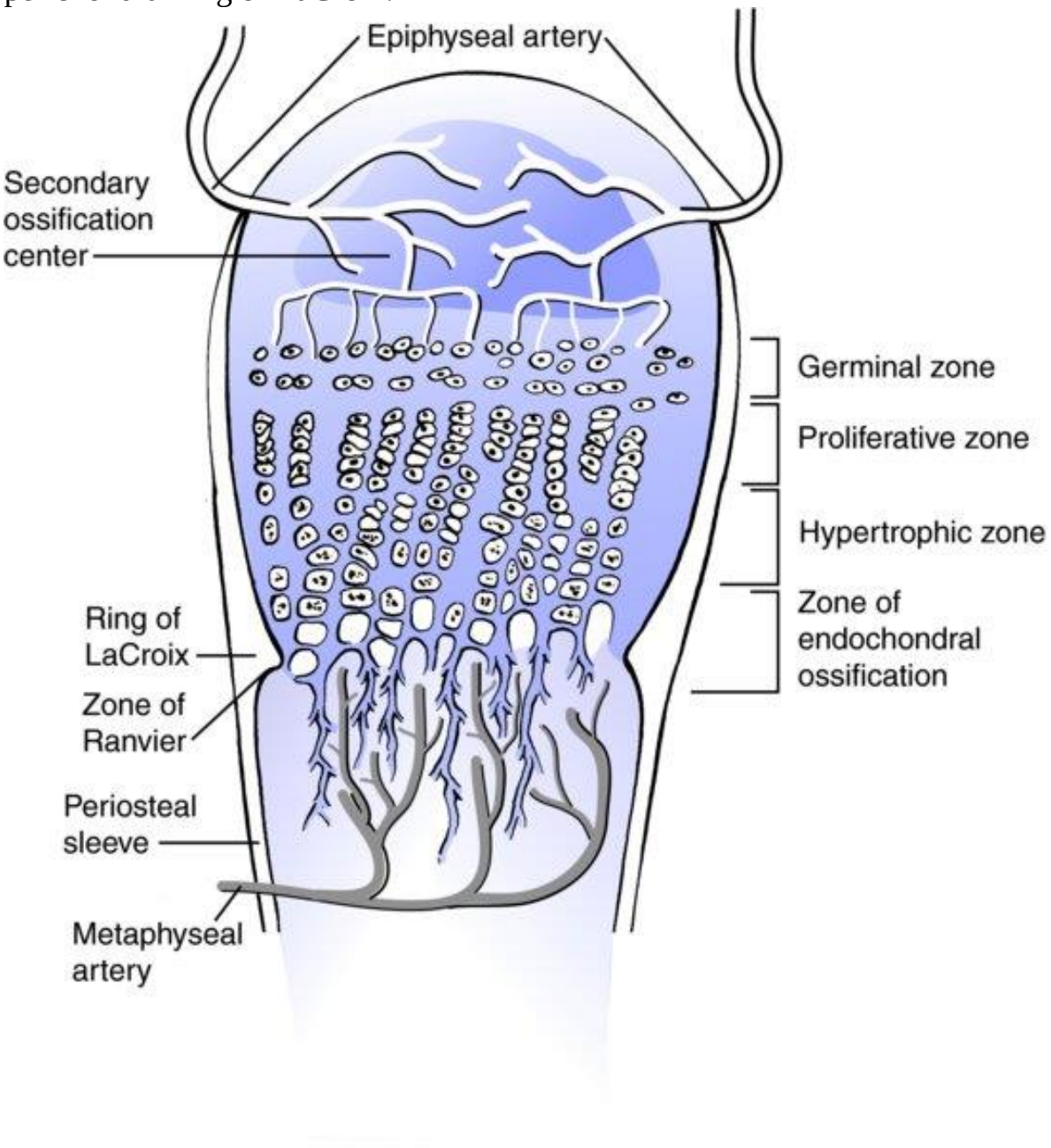
Figure 2-4 Typical age (and range) of closure of physes in the (A) upper extremity and (B) lower extremity.



The external surface of the epiphysis is composed of articular cartilage or perichondrium. Muscle fibers, tendons, and ligaments attach to the perichondrium, which also contributes to the centrifugal enlargement of the epiphysis. The perichondrium blends into the periosteum. This perichondrial/periosteal tissue continuity contributes to the biomechanical strength of the epiphyseal/metaphyseal junction at a region that is called the zone of Ranvier (Fig. 2-

5).[42,190] Hyaline cartilage below the articular cartilage contributes to the growth of the epiphysis. As skeletal maturity is reached, a tidemark develops at the demarcation between the articular and calcified epiphyseal hyaline cartilage.

Figure 2-5 Schematic diagram of the organization of the physis.
Four zones are illustrated: The germinal, proliferative, hypertrophic, and provisional calcification (or enchondral ossification) layers. Note also the groove of Ranvier and the perichondral ring of LaCroix.



Physis

Physis is a highly organized, yet dynamic structure that consists of chondrocytes undergoing proliferation, differentiation, and formation of complex extracellular matrix. The extracellular matrix is composed of type II collagen fiber network, aggrecans, and noncollagenous proteins, such as cartilage oligomeric protein and matrilin-3. Type IX and XI collagens are minor collagens found in the physis. Type X collagen is also found in the physis; however, its synthesis is limited to the hypertrophic zone and is a distinguishing feature of hypertrophic chondrocyte.

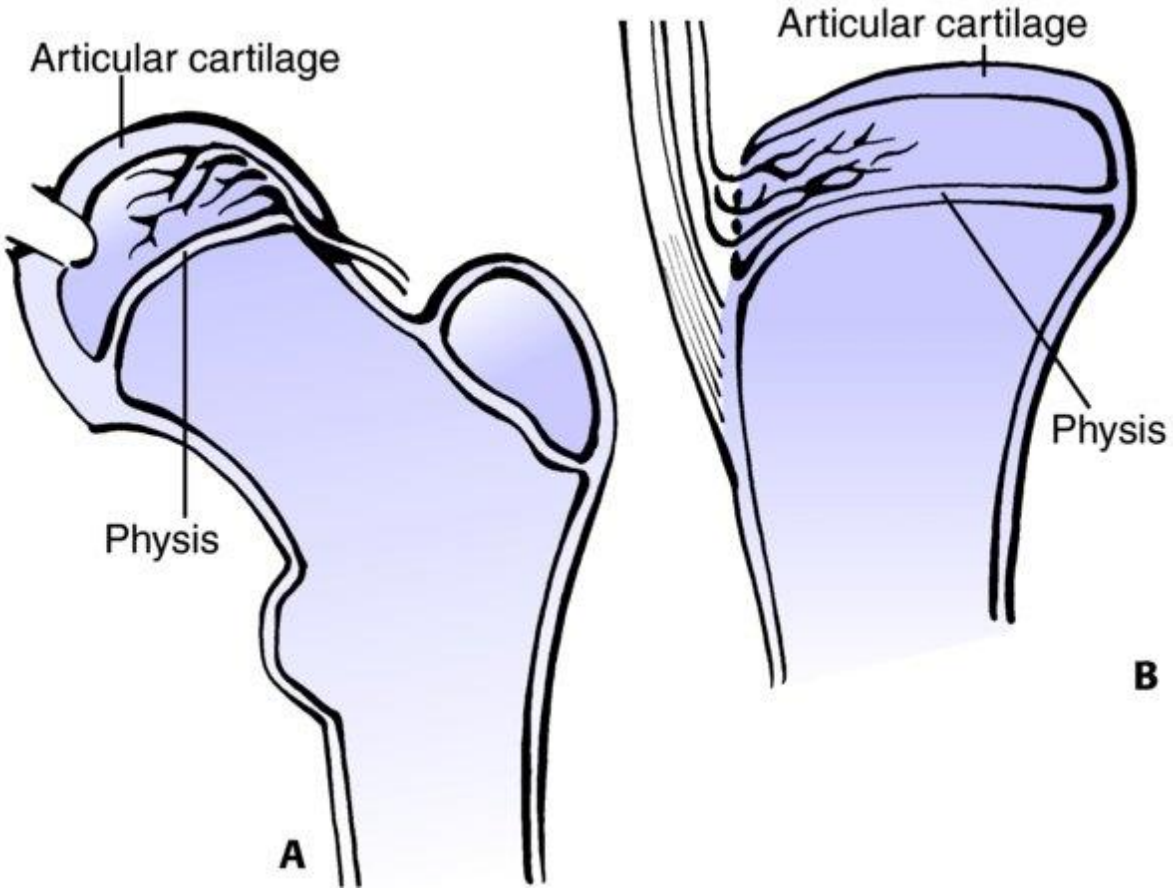
Understanding of physeal injuries requires knowledge of normal physeal morphology.[179] Histologically, the physis is divided into four zones oriented from the epiphysis to the metaphysis: Germinal (reserve), proliferative, hypertrophic, and provisional

calcification (see Fig. 2-5). The proliferative zone is the location of cellular proliferation, whereas the hypertrophic and provisional calcification zones are characterized by extracellular matrix production, cellular hypertrophy, apoptosis, extracellular matrix calcification, and vascular invasion of the lacunae of the terminal hypertrophic chondrocytes. Collagen fiber orientation is horizontal in the germinal zone whereas it is vertical in the proliferative and hypertrophic zones, in line with growth and columnar arrangement of cells.[11] Collagen content is lower in the proliferative and hypertrophic zones compared with the germinal zone. The differences in the collagen content and fiber orientation of different physeal zones have important implications in the mechanical behavior of each zone to mechanical loading.[12] For instance, greater strains are observed in the proliferative and hypertrophic zones compared with the germinal zone following compression loading.

The peripheral margin of the physis comprises two specialized areas important to the mechanical integrity and peripheral growth of the physis (see Fig. 2-5). The zone (or groove) of Ranvier is a triangular microscopic structure at the periphery of the physis, containing fibroblasts, chondroblasts, and osteoblasts. It is responsible for peripheral growth of the physis. The perichondral ring of LaCroix is a fibrous structure overlying the zone of Ranvier, connecting the metaphyseal periosteum and cartilaginous epiphysis, and has the important mechanical function of stabilizing the epiphysis to the metaphysis.

The epiphysis and secondary ossific nucleus must receive blood supply for viability.[204] Dale and Harris[63] identified two types of blood supply to the epiphysis (Fig. 2-6). Type A epiphyses (such as the proximal humeral and proximal femoral epiphyses) are nearly completely covered with articular cartilage; therefore, most of the blood supply to the epiphysis must enter from the perichondrium in a distal to proximal direction. The blood supply to these epiphyses may be easily compromised by epiphyseal separation. A complete disruption of the epiphyseal vasculature, however, may not produce an extensive ischemic damage to the physis if the metaphyseal vasculature is intact.[117] The studies using multiphoton microscopy also suggest that growth plate nutrition is not unidirectional from the epiphysis to the metaphysis as traditionally believed but is contributed by the epiphyseal, metaphyseal, and circumferential perichondrial vasculature.[72,209] Type B epiphyses (such as the proximal and distal tibia and the distal radius) have only a portion of their surface covered with articular cartilage and are theoretically less susceptible to devascularization from epiphyseal separation.

Figure 2-6 Classification of epiphyseal blood supply according to Dale and Harris.
A: Type A epiphyses are nearly completely covered by articular cartilage. Blood supply must enter via the perichondrium. This blood supply is susceptible to disruption by epiphyseal separation. The proximal femur and proximal humerus are examples of type A epiphyses. B:Type B epiphyses are only partially covered by articular cartilage. Such epiphyses are more resistant to blood supply impairment by epiphyseal separation. The distal femur, proximal and distal tibia, and distal radius are clinical examples of type B epiphyses.



In the past decade there has been a substantial increase in research into the control of growth plate chondrocyte function. Such work is the first step in developing approaches to modulate growth plate function, such as using a drug, a cell, or a biologic approach. Such treatments could someday be used to treat a partial growth arrest following an injury. Chondrocytes develop from an undifferentiated mesenchymal precursor cell, sometimes called MSCs, which differentiate into a common osteochondroprogenitor cell. The Wnt/β-catenin signaling pathway plays a key role in determining if these cells become osteoblasts or chondrocytes, as in the absence of β-catenin they develop into chondrocytes. Several drugs are available that modulate β-catenin, and these could someday be used to modulate growth plate activity.[96,100,120] Once cells become committed to be growth plate chondrocytes, they undergo a coordinated process of differentiation, with expression of various genes (*SOX9, IHH, PTHrP, RUNX2*, and then *type X collagen*)[7,101,215] marking the states of differentiation. Resting cells proliferate, then hypertrophy, before undergoing terminal differentiation, where they express *type X collagen* and become replaced by bone. The resting cells maintain the growth plate cells, and as such these cells located nearest to the epiphysis are critical to normal bone growth, and damage to these cells will permanently disrupt growth.

The hedgehog (Hh) signaling pathway is crucial in the regulation of chondrocyte fate in the growth plate. Prehypertrophic chondrocytes in the growth plate express the Hh ligand Indian Hh (IHH).[28,135,161] IHH serves as a key regulator of endochondral ossification, acting in a negative feedback loop with parathyroid hormone-like hormone (PTHrP), also called parathyroid hormone-related protein (PTHrP). IHH regulates chondrocyte differentiation and induces ossification of the perichondrium in a PTHrP-independent manner.[121] The regulation of PTHrP by IHH involves mediators such as BMPs which also play a role initiating the chondrocyte differentiation cascade.[146] In this way, IHH and PTHrP act in a feedback loop controlling the pace of growth plate chondrocyte differentiation.

Metaphysis

The metaphysis is the flared portion of the bone at each end of the diaphysis. It has a decreased cortical thickness and increased volume of trabecular bone in the secondary

spongiosa. During growth, endochondral modeling centrally and peripherally initially forms the primary spongiosa, which then is remodeled into the more mature secondary spongiosa by osteoclasts and osteoblasts. For this reason, there is considerable bone turnover in the metaphysis compared to other regions of the bone. The metaphyseal cortex is thinner and is more porous than the diaphysis, and there are cortical fenestrations, which contain fibrovascular soft tissue elements that connect the metaphyseal marrow spaces with the subperiosteal region. The metaphyseal region does not develop extensive secondary and tertiary haversian systems until the late stages of skeletal maturation. These microscopic and anatomic changes correlate with changing fracture patterns, and the ability of bone to deform without breaking in this region is why buckle (or torus) fractures are more likely to occur than complete metaphyseal or epiphyseal/physeal fractures.[115,163,205]

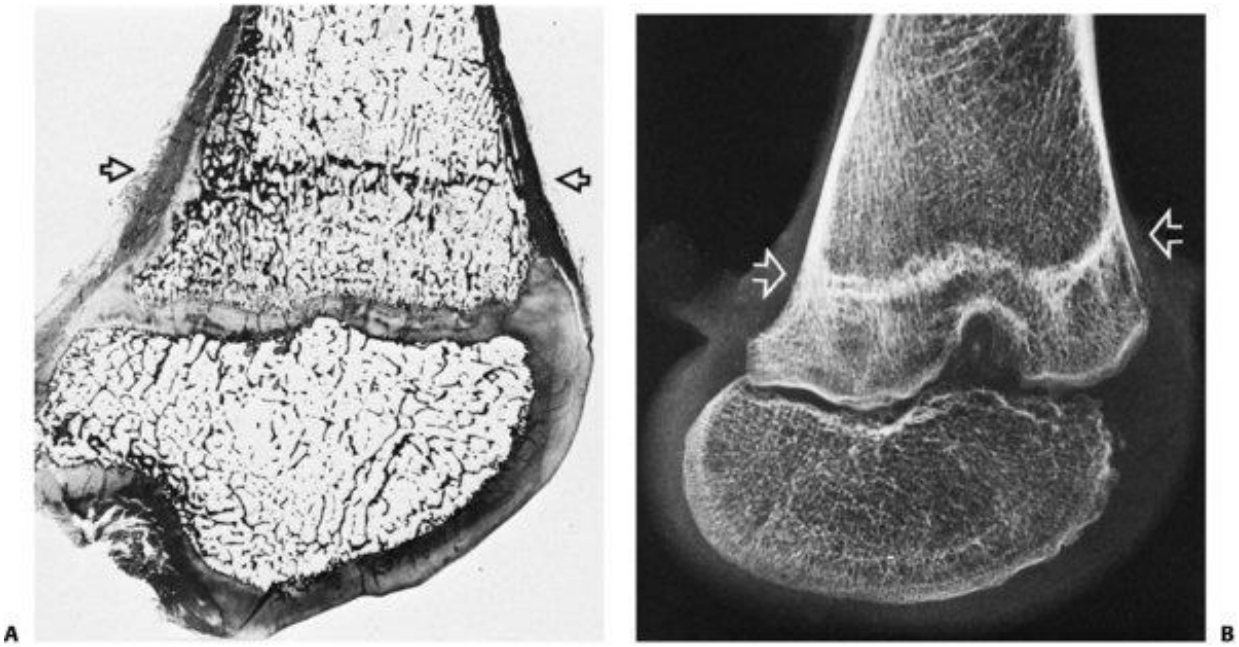
Although the periosteum is attached relatively loosely to the diaphysis, it is firmly fixed to the metaphysis because of the increasingly complex continuity of fibrous tissue through the metaphyseal fenestrations. The periosteum subsequently attaches densely into the peripheral physis, blending into the zone of Ranvier as well as the epiphyseal perichondrium. The zone of Ranvier is a specialized region between bone and cartilage formation, and cells in this zone contribute to growth plate remodeling over time.[42,190]The fenestrated metaphyseal cortex extends to the physis as the thin osseous ring of LaCroix. There are no significant direct muscle attachments to the metaphyseal bone; instead, muscle fibers primarily blend into the periosteum. The medial distal femoral attachment of the adductor muscles is a significant exception.

Growth Lines of Park and Harris

Many bones exhibit transversely oriented, dense trabecular linear bone patterns within the metaphysis. These lines duplicate the contiguous physeal contour, and appear after processes which transiently slow growth or increase mineralization. As such, they are seen after generalized illnesses, treatment with bisphosphonate drugs (which inhibit osteoclasts, and therefore decrease bone resorption and remodeling of the primary spongiosa), or after localized processes within the bone, such as infection or growth plate trauma. The lines are called Harris or Park growth slowdown or arrest lines. Once the normal longitudinal growth rate resumes, longitudinal trabecular[8] orientation is restored. The thickened, transversely oriented osseous plate is left behind, will be gradually remodeled, and with time will disappear (Fig. 2-7).[193]

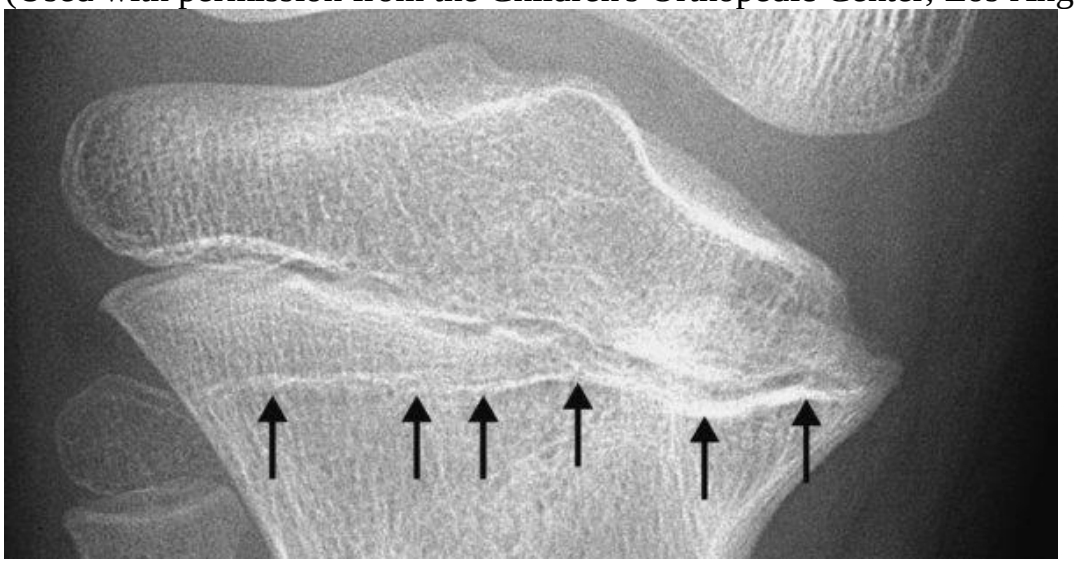
Figure 2-7 Growth lines.

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



In a systemic problem slowing bone growth, the lines are distributed relatively symmetrically throughout the skeleton, and are thickest in metaphyses from bones that grow most rapidly. The lines are important in analyzing the effects of a fracture on growth. They can be measured and the sides compared to corroborate femoral overgrowth after diaphyseal fracture and eccentric overgrowth medially after proximal tibial metaphyseal fracture. A line that converges toward a physis suggests localized growth damage that may result in an osseous bridge and the risk of angular deformity (Fig. 2-8).

Figure 2-8 Example of a growth arrest line converging toward the area of physeal arrest at the medial proximal tibial physis.
(Used with permission from the Children's Orthopedic Center, Los Angeles.)



Diaphysis

The diaphysis constitutes the major portion of each long bone, and is formed from bone remodeled from the metaphysis. Mature, lamellar bone is the dominant feature of the diaphyseal bone, and the developing diaphyseal bone is extremely vascular. When analyzed in cross section, the center is much less dense than the maturing bone of older children, adolescents, and adults. Subsequent growth leads to increased complexity of the haversian (osteonal) systems and the formation of increasing amounts of extracellular matrix, causing a relative decrease in cross-sectional porosity and an increase in hardness. Some bones, such as the tibia, exhibit a decrease in vascularity as the bone matures; this factor affects the rate of healing and risk of nonunion.[40,162] The vascularity of bone is important not only because it

brings nutrients to the bone, but also because pericyte cells surrounding blood vessels contribute to new osteoblasts.[19]

Periosteum

A child's periosteum is thicker and more readily elevated from the bone than in adults. It also has a much greater osteogenic potential than that of an adult. Indeed, in young children, one can remove the entire diaphysis of a bone, but leave the periosteum, and the bone will regrow. The thicker and stronger periosteum affects fracture displacement, ease of closed reduction, and the capacity to form new bone. The periosteum usually remains intact on the concave (compression) side of an injury. This intact periosteal hinge or sleeve may lessen the extent of displacement of the fracture fragments, and it also can be used to assist in the reduction, because the intact portion contributes to the intrinsic stability. Thus, accentuating the deformity, unlocks the periosteum, helping with the reduction. Because the periosteum allows tissue continuity across the fracture, the subperiosteal new bone that forms quickly bridges the fracture gap and leads to more rapid long-term stability.[10,20]

The periosteum comprises two tissue layers. An outer fibroblast layer provides fibrous attachment to subcutaneous connective tissue, muscles, tendons, and ligaments, whereas the inner cambium layer contains a pool of cells that support bone formation and repair. The periosteum, rather than the bone itself, serves as the origin for muscle fibers along the metaphysis and diaphysis. This mechanism allows coordinated growth of bone and muscle units; something that would be impossible if all the muscle tissues attached directly to the developing bone or cartilage. Exceptions include the attachment of muscle fibers near the linea aspera and into the medial distal femoral metaphysis. The latter pattern of direct metaphyseal osseous attachment may be associated with significant irregularity of cortical and trabecular bones. Radiographs of this area often are misinterpreted as showing a neoplastic, osteomyelitic, or traumatic response, even though this is actually a variation of skeletal development. The periosteum in the growing child also plays a critical role in remodeling, as the tissues in tension over the concave side of a deformity will produce new bone. The bone on the tension, or convex, side of a deformity will be resorbed over time, ultimately resulting in a straight diaphysis.[10,20,106]

Apophysis

An apophysis is composed of fibrocartilage instead of columnar cartilage and grows primarily in response to tensile forces. They are generally attached to muscular structures. With growth secondary ossification centers can form in the apophysis. Because of the differing histologic composition of these structures, they fail differently than other parts of the bone, and excessive tensile stress may avulse the apophysis, especially during the late stages of closure. Such injuries can generate large amounts of new bone, and may be mistaken for tumors, especially around the pelvis. Healing of a displaced fragment to the underlying undisplaced secondary center creates the symptomatic reactive overgrowth, and in the tibial tuberosity apophysis, this is known as an Osgood–Schlatter lesion.[64,140,181]

Mechanisms of Fracture Healing

Fracture healing is a complex regenerative process initiated in response to injury, in which bone can heal by primary or secondary mechanisms. In primary healing, new bone is laid down without any intermediate. This type of healing is rare in a complete bone fracture, except when the fracture is rigidly fixed through certain types of surgery. In the more common secondary healing, immature and disorganized bone forms between the fragments, which is termed the callus.[45,70,142,164] During the fracture repair process, cells progress through stages of differentiation reminiscent of those that cells progress through during normal fetal bone development. In normal development of long bone, undifferentiated

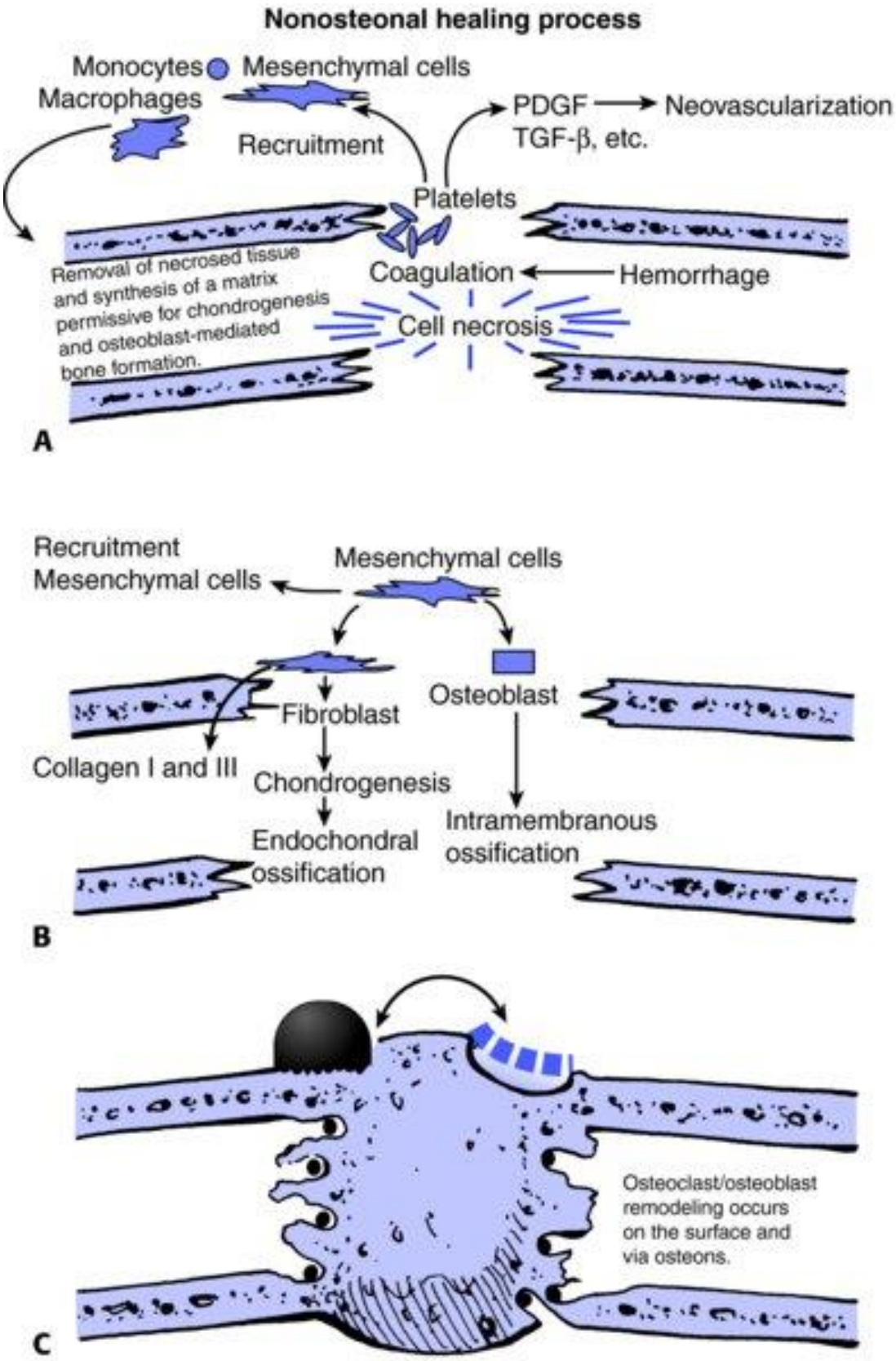
mesenchymal cells initially form a template of the bone, which differentiate to chondrocytes. This cartilaginous template is termed the bone's anlage. Following this phase, blood vessels enter the cartilaginous template, and osteoblasts, which differentiate from perivascular and other cells surrounding the bone, form bone.

There are, however, several important differences between bone repair and development. One is that repair does not need to progress through a cartilaginous template. Another is that the liberation of growth factors in the extracellular environment and inflammatory mediators initiates fracture repair, and the activation of these factors does not occur during development. Indeed, this inflammatory initiation of repair processes may be the fundamental difference between development and regeneration. This is one reason that agents that modulate inflammation can affect bone formation. Although some inflammatory pathways can have both positive and negative effects on bone repair, an inhibition of prostaglandin activity inhibits bone formation, and indeed this has been used clinically to prevent bone formation.[29,151,177]

Osseous repair progresses through closely integrated phases. In the *initial* phase of fracture repair, bleeding from the damaged tissues causes a hematoma at the fracture site, stopping blood loss and liberating growth factors and cytokines. Endothelial cells respond by increasing their vascular permeability, allowing leukocytes, monocytes, macrophages, and multipotential mesenchymal cells to reach the fracture site.[159] The blood supply is temporarily disrupted for a few millimeters on either side of the fracture site, producing local necrosis and hypoxia. In the *proliferative* phase, undifferentiated mesenchymal cells aggregate at the site of injury, proliferate, and differentiate presumably in response to growth factors produced by the injured tissues.[45] This process involves both intramembranous and endochondral ossification. Intramembranous ossification involves the formation of bone directly from committed osteoprogenitor cells and undifferentiated mesenchymal cells that reside in the periosteum, resulting in hard callus formation.[68] During endochondral ossification, mesenchymal cells differentiate into chondrocytes, producing cartilaginous matrix, which then undergoes calcification and eventually is replaced by bone. The formation of primary bone is followed by extensive *remodeling* until the damaged skeletal element regains its original shape and size (Fig. 2-9).[68,70,142]

Figure 2-9 Phases of fracture repair.

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



The strength of bone is a function of the intrinsic mechanical properties of the ossified tissue as well as the way the tissue is organized. When a child's fracture heals, the weaker callous has a larger diameter than the intact bone, but because the weaker material is farther from the center, the moment of inertia is increased, and as a whole unit the bone can be just as strong as if it had not fractured. Children form a larger diameter callous than adults, in part because the stronger periosteum comes off the bone easier and forms a wider barrier to the callous. In addition, during the proliferative phase of fracture repair, children form new bone faster than adults. These factors combine to make children's bone regain its strength much quicker following a fracture than an adult. Furthermore, these factors are responsible for the observation that the younger the child the quicker a fractured bone will regain its strength.

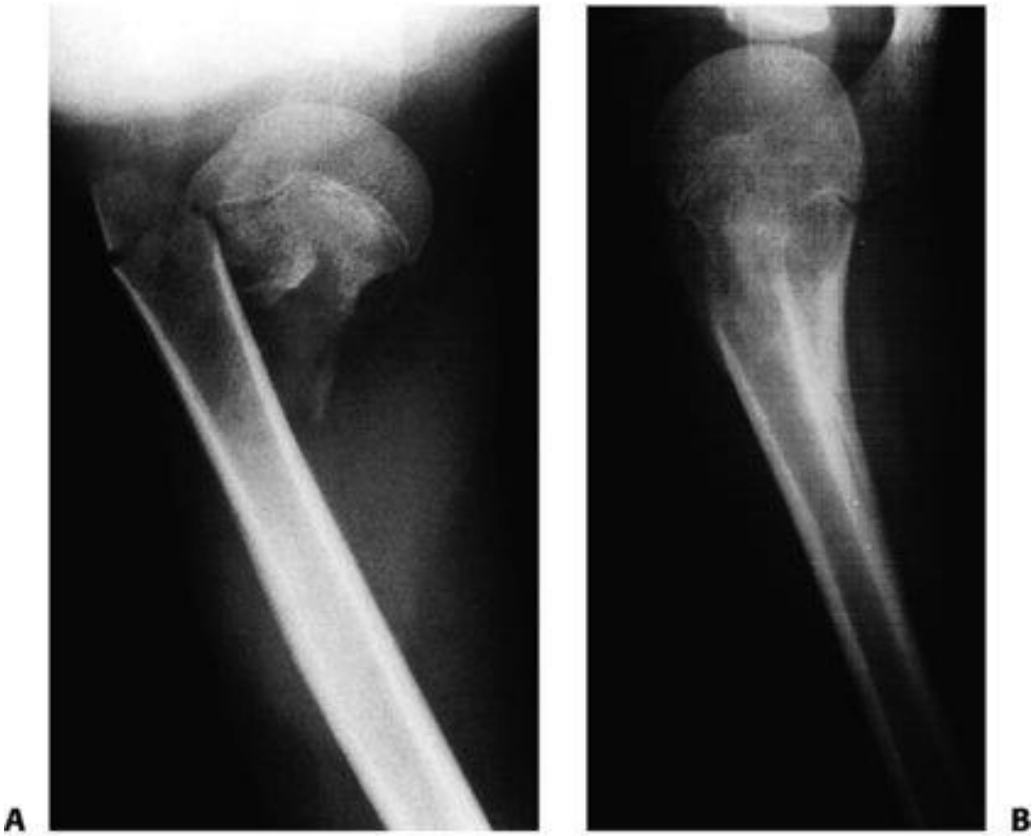
Several cell signaling pathways are normally activated during fracture repair, and many of these are the same ones that are activated during bone development. Certain BMPs are liberated early in the repair process, and they stimulate undifferentiated mesenchymal cells to achieve an osteoblastic phenotype. Tibial fractures are a high-risk injury for developing a nonunion and clinical studies show that treatment with select BMPs will improve the rate of healing in this situation.[85] Another pathway that plays an important role in bone repair is β-catenin. There is upregulation of β-catenin during the healing process,[21,55,56] and healing is repressed in mice lacking β-catenin. However, β-catenin functions differently at different stages of fracture repair. Because drugs that modulate β-catenin are in development, this is an area in which novel therapies could be used to improve delayed repair.[55,58,198]
The various signaling pathways which play a role in bone repair also interact with each other during the repair process. For instance, the inflammatory process activates prostaglandin synthesis, which regulates BMP expression in mesenchymal progenitors.[18] In a similar manner, prostaglandin activity also regulates β-catenin activity.[118]Furthermore, BMP stimulation requires β-catenin to produce bone.[56] Thus, the various signaling pathways involved in bone repair and regeneration do not act alone but in a coordinated manner to allow for bone regeneration.

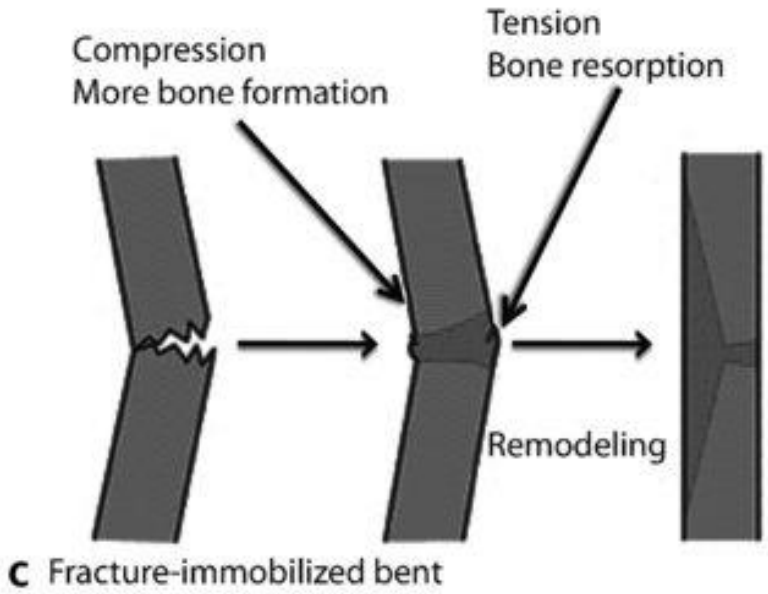
Unique Aspects of Injury in the Immature Skeleton

Remodeling of Bones After a Fracture in Children

In a growing child, the normal process of bone growth and remodeling may realign initially malunited fragments, making anatomic reduction less important than in an adult. Bone and cartilage generally remodel in response to normal stresses of body weight, muscle action, and joint reaction forces, as well as intrinsic control mechanisms such as the periosteum. The potential for spontaneous, complete correction is greater if the child is younger, the fracture site is closer to the physis, and there is relative alignment of the angulation in the normal plane of motion of the joint (Fig. 2-10).[201] This is particularly evident in fractures involving hinge joints such as the knee, ankle, elbow, or wrist, in which corrections are relatively rapid if the angulation is in the normal plane of motion. As an example, the distal radius can correct deformities at a rate of one degree a month.[80,97,131] However, spontaneous correction of angular deformities is unlikely in other directions, such as a cubitus varus deformity following a supracondylar fracture of the humerus. Similarly, rotational deformities usually do not correct spontaneously.[163,184]

Figure 2-10 Remodeling of a fracture.
A and ***B*** show the initial and follow-up radiographs of a proximal humeral fracture, illustrating an impressive degree of remodeling. ***C*** shows how bone deposition and resorption result in straightening of the deformity over time. Efficient remodeling requires an open growth plate, and as such is a unique feature of childhood injury.

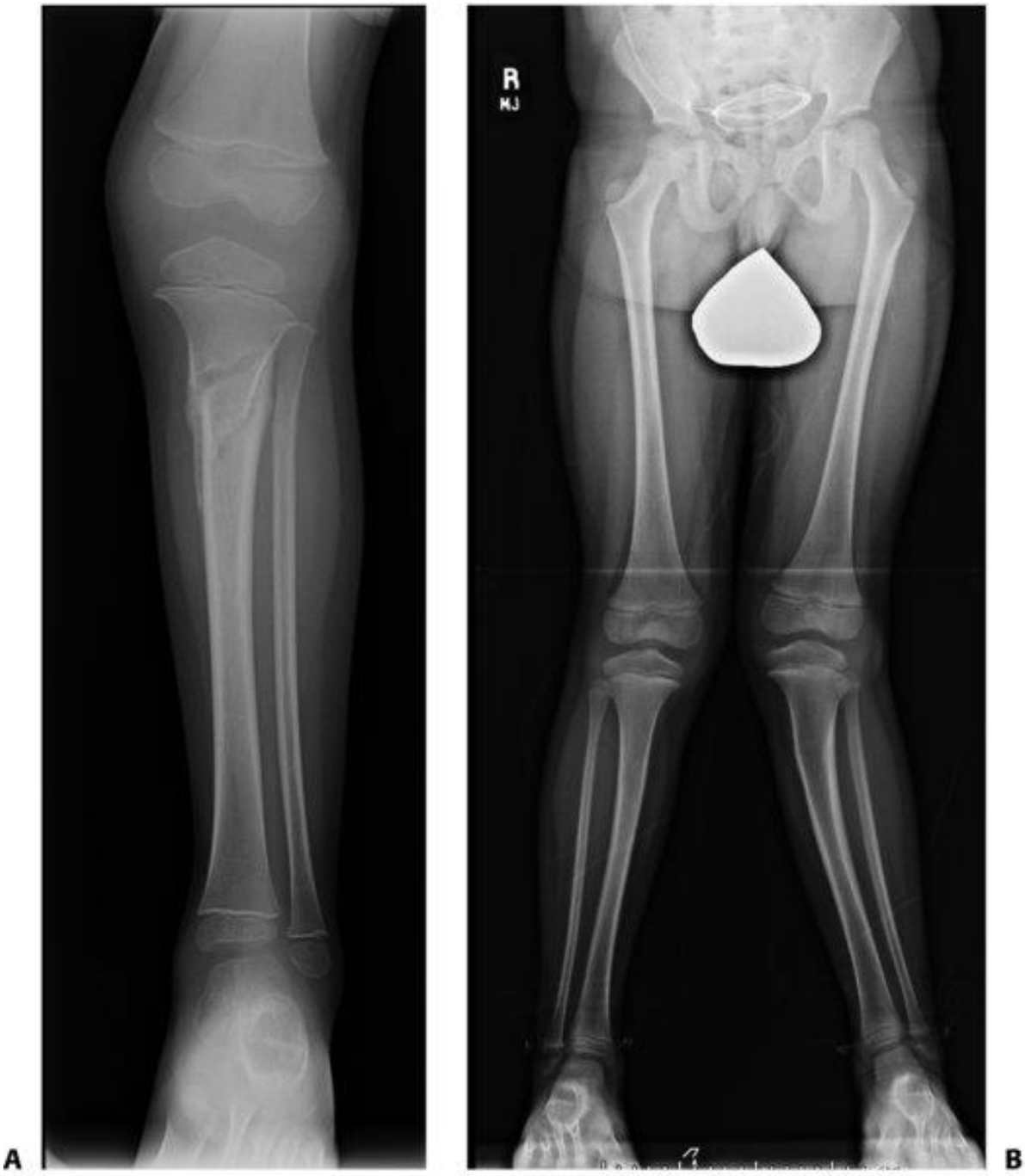




Growth Stimulation

Fractures may stimulate longitudinal growth by increasing the blood supply to the metaphysis, physis, and epiphysis, and at least on an experimental basis, by disrupting the periosteum and its physiologic restraint on the rates of longitudinal growth of the physes. Such increased growth may make the bone longer than it would have been without an injury. Eccentric overgrowth may also occur; this is particularly evident in tibia valgum following an incomplete fracture of the proximal tibial metaphysis (Fig. 2-11).[201]

Figure 2-11 A: AP radiograph of the tibia of a 4 year old shows a proximal metaphyseal fracture with minimal displacement in the fibula intact. B: Two years later note the significant valgus deformity.



Physeal Injuries

Etiology of Physeal Injuries

Physes can be injured in many ways, both obvious and subtle. Obviously, the most frequent mechanism of injury is fracture. Most commonly, physeal injury is direct, with a fracture involving the physis itself. Occasionally, physeal injury from trauma is associated with a fracture elsewhere in the limb segment, either as a result of ischemia[167] or perhaps compression[1,13,33,103,143,150,206] (see discussion of Salter–Harris type V physeal fractures below). Other mechanisms of injuries to the physes include infection[26,31,126,165]; disruption by tumor, cysts,[199] and tumor-like disorders; vascular insult[167]; repetitive stress[9,34,49,50,134,213]; irradiation[43,180]; and other rare etiologies.[25,39,48,189,213]

Repetitive Stress Physeal Injuries

Repetitious physical activities in skeletally immature individuals can result in physeal stress–fracture equivalents.[9,49,50] The most common location for such injuries is in the distal radius or ulna, as seen in competitive gymnasts (Fig. 2-12); the proximal tibia, as in running and kicking sports such as soccer (Fig. 2-13); and the proximal humerus, as in baseball pitchers.[49] These injuries should be managed by rest, judicious resumption of activities, and longitudinal observation to monitor for potential physeal growth disturbance.

Figure 2-12 Stress injury of the distal radius and ulna in both wrists of a competitive gymnast.
There was no history of specific injury. The wrists were tender to touch. Note distal radial and ulnar physeal widening and irregularity.

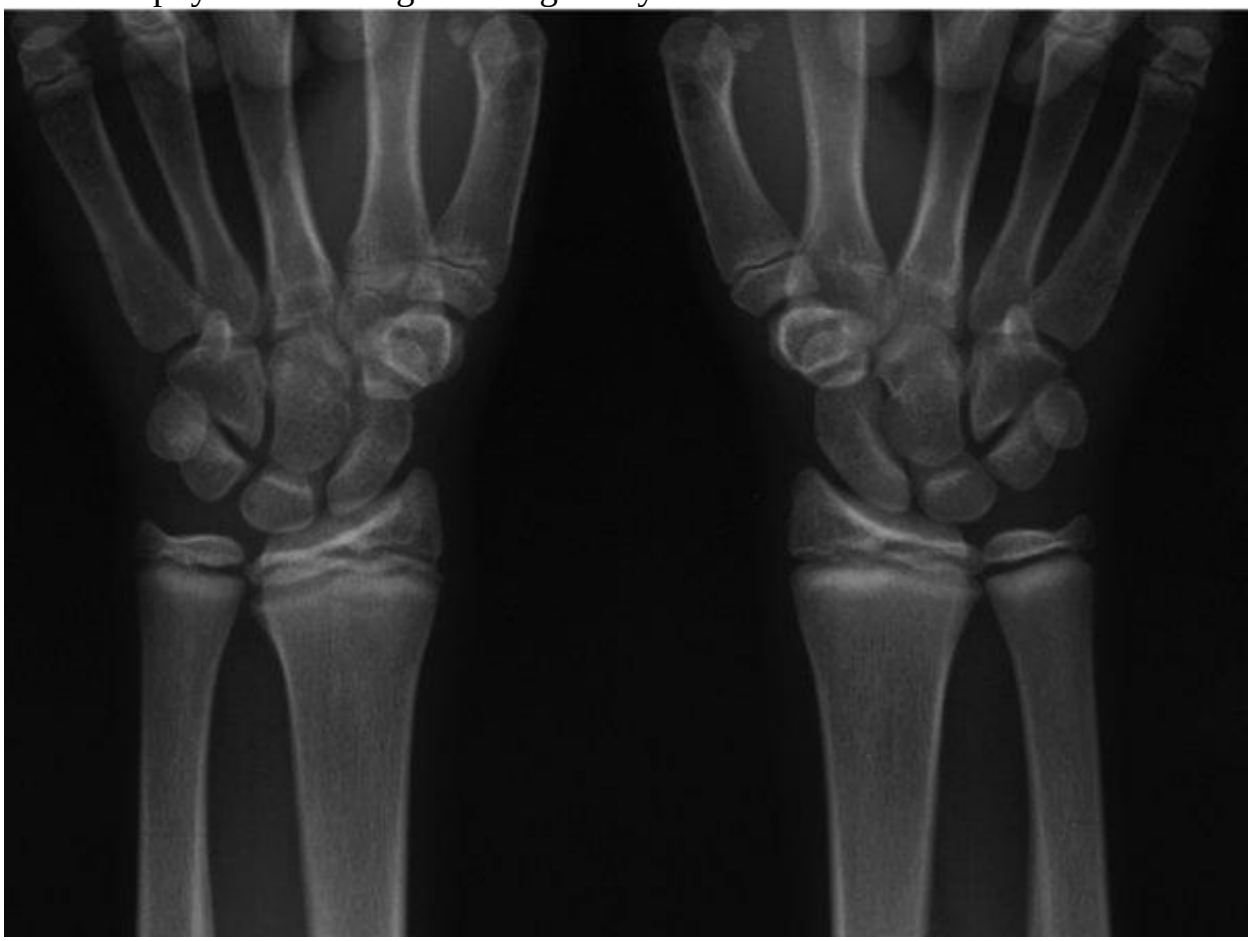
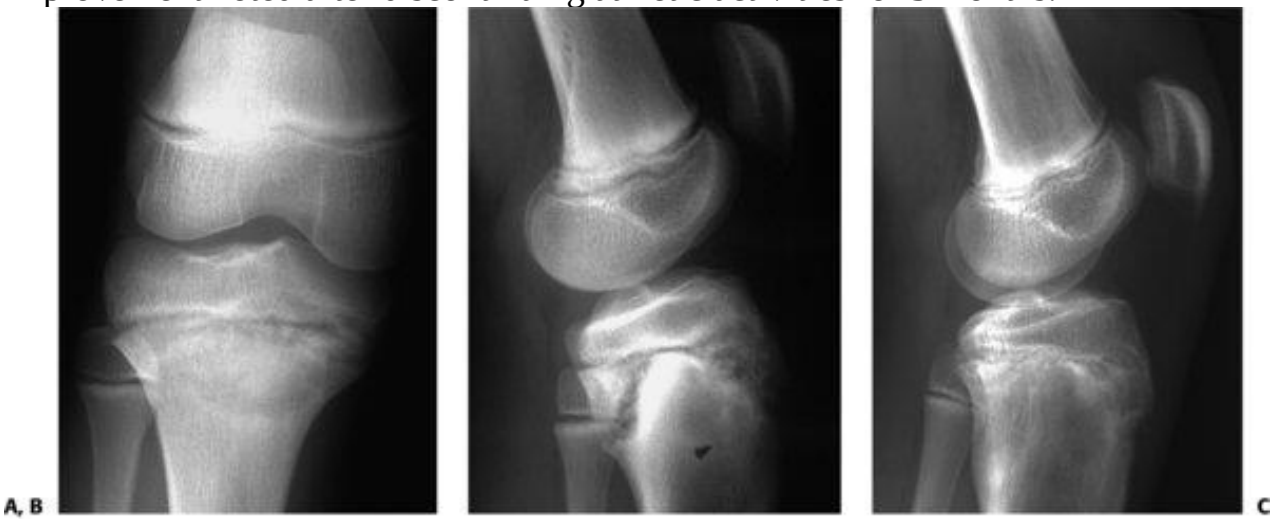


Figure 2-13 Stress injury of the proximal tibia in an elite soccer player.
A: Anteroposterior radiograph film demonstrates subtle proximal tibial physeal widening. B:Lateral radiograph shows widening, a metaphyseal Thurston Holland fragment, and some posterior displacement of the proximal epiphysis. C: Significant radiograph improvement noted after discontinuing athletic activities for 3 months.



Classification of Physeal Fractures

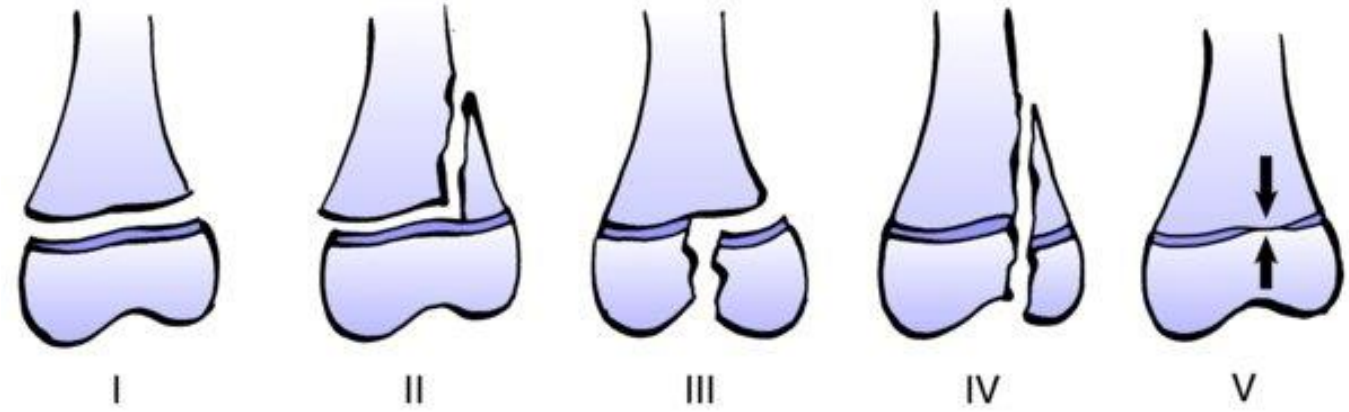
Physeal fractures have been recognized as unique since ancient times. Hippocrates is credited with the first written account of this injury. Poland reviewed accounts of physeal injuries in his 1898 book, *Traumatic Separation of the Epiphysis*,[174] and is credited with the first

classification of the patterns of physeal fracture. Modifications to Poland's original scheme have been proposed by a number of authors,[2345,63,66,137,153,155,168,169,172,185] including Aitken,[4] Salter and Harris,[185] Ogden et al.,[143,155] and Peterson.[168,169] Classifications of physeal fractures are important because they alert the practitioner to potentially subtle radiographic fracture patterns, can be of prognostic significance with respect to growth disturbance potential, and guide general treatment principles based on that risk and associated joint disruption. Currently, the Salter–Harris classification, first published in 1963,[185] is firmly entrenched in the literature and most orthopedists' minds. The reader also should be aware of some deficiencies in that classification, as pointed out by Peterson and discussed below.[168169170]

Salter–Harris Classification of Physeal Fractures

Salter–Harris Classification consists of five types of physeal injuries.[185] The first four types were adopted from Poland (types I, II, and III) and Aitken (Aitken type III became Salter–Harris type IV) (Fig. 2-14). Salter and Harris added a fifth type, which they postulated was an unrecognized compression injury characterized by normal radiographs and late physeal closure.[1,13,24,33,99,103,116,170,206]

Figure 2-14 Salter–Harris classification of physeal fractures.
In Salter–Harris type I fractures, the fracture line is entirely within the physis, referred to by Poland as type I. In Salter–Harris type II fractures, the fracture line extends from the physis into the metaphysis; described by Poland as type II and Aitken as type I. In Salter–Harris type III fractures, the fracture enters the epiphysis from the physis and almost always exits the articular surface. Poland described this injury as type III and Aitken as type II. In Salter–Harris type IV, the fracture extends across the physis from the articular surface and epiphysis, to exit in the margin of the metaphysis. Aitken described this as a type III injury in his classification. Salter–Harris type V fractures were described by Salter and Harris as a crush injury to the physis with initially normal radiographs with late identification of premature physeal closure.

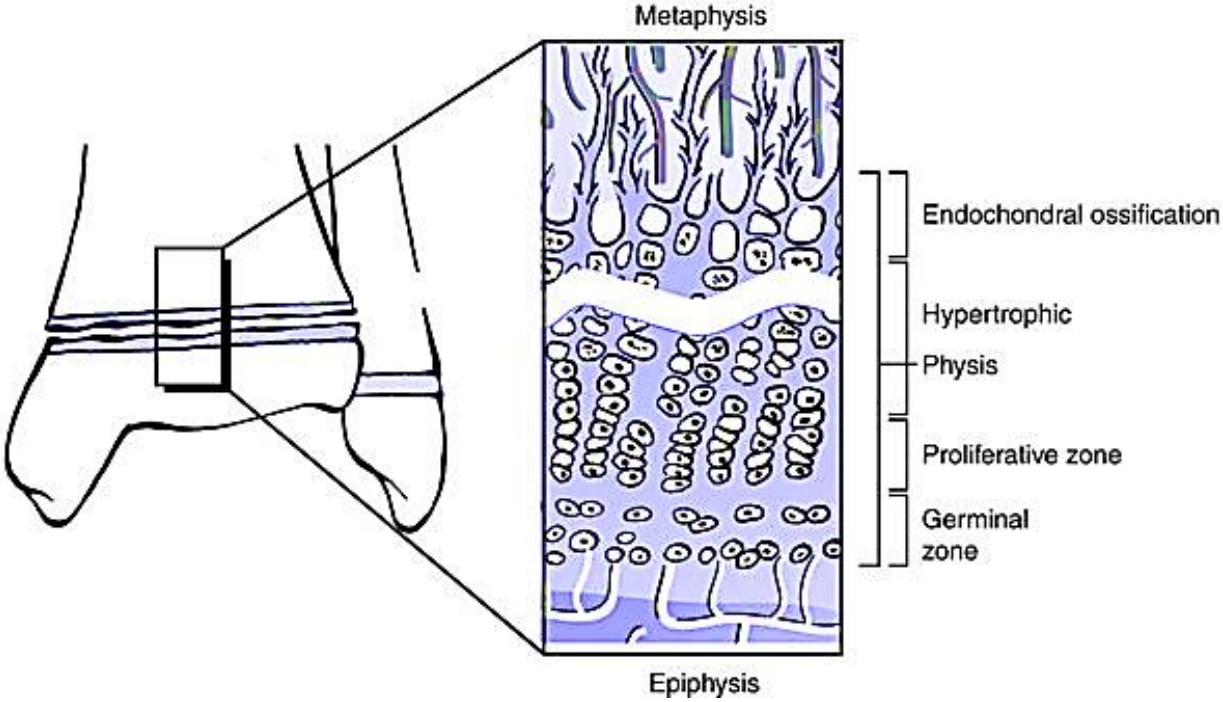


Type I

Salter–Harris type I injuries are characterized by a transphyseal plane of injury, with no bony fracture line through either the metaphysis or the epiphysis. Radiographs of undisplaced type I physeal fractures, therefore, are normal except for associated soft tissue swelling, making careful patient examination particularly important in this injury. In the Olmstead County Survey of physeal fractures,[171] type I fractures occurred most frequently in the phalanges, metacarpals, distal tibia, and distal ulna. Epiphyseal separations in infants occur most commonly in the proximal humerus, distal humerus, and proximal femur. If there is an urgency to make the diagnosis on patients suspected of having a type I injury, further imaging by ultrasound, magnetic resonance imaging (MRI),[46,60,108,172,196] or intraoperative arthrography may be helpful.[6,89,139] Ultrasound is particularly helpful for assessing epiphyseal

separations in infants (especially in the proximal femur and elbow regions) without the need for sedation, anesthetic, or invasive procedure.[37,66,67,98,188]
The fracture line of type I injuries is usually in the zone of hypertrophy of the physis, as the path of least resistance during the propagation of the injury (Fig. 2-15). As a consequence, in theory, the essential resting and proliferative zones are relatively spared, and, assuming that there is no vascular insult to these zones as a consequence of the injury, subsequent growth disturbance is relatively uncommon. As discussed above, however, studies have shown this to be a simplistic view of the fracture line through a physis, and that, because of uneven loading and macroscopic undulations in the physis, any zone of the physis can be affected by the fracture line.[35,109,148,183,189,197]

Figure 2-15 Scheme of theoretic fracture plane of Salter–Harris type I fractures.
Because the hypertrophic zone is the weakest zone structurally, separation should occur at this level. Experimental and clinical studies have confirmed that the fracture plane is more complex than this concept and frequently involves other physeal zones as well.



Because the articular surface and, at least in theory, the germinal and proliferative layers of the physis are not displaced, the general principles of fracture management are to secure a gentle and adequate reduction of the epiphysis on the metaphysis and stabilize the fragments as needed.

Type II

Type II injuries have physeal and metaphyseal components; the fracture line extends from the physeal margin peripherally across a variable portion of the physis and exits into the metaphysis at the opposite end of the fracture (Fig. 2-16). The epiphyseal fragment thus comprises all of the epiphysis and some portion of the peripheral metaphysis (the Thurston Holland fragment or sign). The physeal portion of this fracture has microscopic characteristics similar to those of type I injuries, but the fracture line exits the physis to enter the metaphysis (i.e., away from the germinal and proliferative layers) at one margin. Similar to type I injuries, these fractures should have a limited propensity to subsequent growth disturbance as a consequence of direct physeal injury. However, the metaphyseal “spike” of the diaphyseal/metaphyseal fragment may be driven into the physis of the epiphyseal fragment, which can damage the physis (Fig. 2-17). Similar to type I injuries, the articular surface is not affected and the general principles of fracture management are effectively the same.