Skeletal Atlas of Child Abuse
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We dedicate this atlas to our spouses, whose support enabled us to be successful in this endeavor.

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Child abuse investigation is one of the most contentious areas of forensic pathology. The forensic pathologist must determine, within a reasonable degree of medical certainty, the cause and manner of death. This opinion must be rendered with a comprehensive knowledge of the case – a knowledge that includes the medical history, scene findings, police investigation, history of terminal events, and a complete autopsy. The autopsy is one component of the overall investigation, and it is imperative that all injuries be thoroughly documented. Although other pathologists may offer alternative opinions regarding the mechanism of injury, or even the cause of death, the actual anatomic findings should never be in question. The collaborative approach taken at the Harris County Institute of Forensic Sciences (HCIFS) reflects this philosophy. The team of forensic anthropologists who work within the office and alongside the pathologists is immeasurably important in completing the requisite documentation. In our experience, anthropologists bring to the autopsy table another dimension of knowledge that is rarely tapped in the medical examiner setting. In this collaborative setting, suspected child abuse deaths are first examined by a forensic pathologist, with complete documentation of soft tissue injuries including visceral injuries, neuropathology, and examination of subcutaneous tissues. A forensic anthropologist is then brought in with the pathologist to take the examination another step, exposing all skeletal elements. The need for additional studies of the exposed elements (including histology and further anthropologic processing) is determined at that time. In this approach, the traditional role of the forensic anthropologist (examination of skeletal remains) is greatly expanded while keeping within the scope of his or her expertise.

The value of the collaborative approach between forensic pathologists and anthropologists is perhaps best illustrated by the numerous examples of classic metaphyseal lesions presented in this text. These lesions are difficult – and sometimes impossible – to identify radiographically. They must be sought at autopsy. Only with careful analysis of many cases using the approach described in this text (including abusive injuries, accidental injuries, and deaths from disease) will the true incidence, mechanism, and healing process of these injuries be appreciated.

Skeletal Atlas of Child Abuse is an illustrated guide to anthropologists’ role in investigating child abuse. The book offers a fairly comprehensive literature review of injuries within each body region or skeletal element and then illustrates injuries using real case examples. The literature review is presented in a nonbiased manner. The authors walk the reader through published data regarding the mechanics of injuries and the interpretation of injuries, including what the injuries may imply regarding mechanism. Also included is a review of literature that describes limitations of interpretation of a particular injury as being inflicted or accidental. The case examples are provided to demonstrate for the reader effective ways of viewing and then photographically documenting in situ examples of skeletal injuries as well as examples of processed skeletal elements. The abundance of in situ photographs sets this volume apart from the typical anthropology text, and this reflects the close collaboration between pathologists and anthropologists in the HCIFS. Information provided with each case example includes the mechanism of injury, if that can be determined, and describes limitations of interpretation. Information regarding the cause and manner of death is provided for many of the cases, but this determination is not the intent of the illustrations or the thrust of the book.

A chapter on natural disease conditions affecting the bones provides a good overview of several conditions that often are invoked as “mimics” of child abuse. This is a good starting point and will be of value to forensic pathologists and anthropologists, as these conditions frequently are suggested as explanations for unexplained injuries in abuse cases. Although isolated skeletal findings may well suggest one of these natural disease processes, differential diagnosis involves analysis of the entire case, including not only anatomic findings, but history – a history including the terminal events, the complete medical history, and birth history in some cases. The pathologist, working in tandem with the anthropologist and investigative personnel, must consider all facets of the case to arrive at a proper cause and manner of death determination and to opine regarding antemortem treatment of the child.

This book will serve as a valuable reference for practicing anthropologists, pathologists, and possibly radiologists in recognition of skeletal trauma. Although Skeletal Atlas of Child Abuse focuses on pediatric trauma, much of the information within the text and photographs of this volume is applicable to the analysis of skeletal trauma in general. Additionally, this photographic atlas of bone injury and pathology creates a comprehensive instructional resource for the forensic course classroom or for postgraduate training in the medical examiner office setting.
Perhaps the biggest contribution of this book is an implicit statement: forensic anthropology is a science that is most valuable when practiced as an integral component of the autopsy process, rather than a separate discipline unassociated with examination of the “soft tissue.” Examination of skeletal elements in situ as amply illustrated here, preferably in collaboration with the pathologist, allows both disciplines to arrive at a reasoned evaluation of each injury and, hence, of the total case. Forensic pathologists who have come to view anthropologists in their traditional role of analysis of skeletal remains will find this an eye-opening approach.

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Preface

The Centers for Disease Control and Prevention (CDC) defines child maltreatment as “any act or series of acts of commission or omission by a parent or other caregiver (e.g., clergy, coach, teacher) that results in harm, potential for, or threat of harm to a child.” The CDC further identifies four common types of abuse: physical, sexual, and emotional abuse, and neglect. The 2008 Child Maltreatment report by the US Department of Health and Human Services states that the fatality rate among abused and neglected children is 2.3 per 100,000 children. Eighty percent of the deaths occur in children younger than 3 years. Physical abuse accounts for 22.9% of all child maltreatment fatalities.

Despite the frequency of child fatalities attributed to physical abuse, the recognition of child abuse is challenging in the forensic setting. Signatures of nonaccidental injury may be subtle and differentiating accidental from nonaccidental injury difficult. Yet, successful adjudication of a child abuse case often depends on comprehensive documentation of injury, including recognition, documentation, and interpretation of skeletal injury.

The goal of this volume is not to showcase all skeletal injury observed in children, but to illustrate common skeletal findings in child abuse fatalities investigated by a medical examiner’s office serving a large urban area. The Harris County Institute of Forensic Sciences (HCIFS) serves a population of approximately 4 million. In 2009, HCIFS certified 4,153 deaths, 3,383 of which required an autopsy. Infants and toddlers (ages birth to 4 years) accounted for 204 of these deaths; 31 were classified as homicide, 59 as natural, 48 as accident, and 66 as undetermined.

In 2006, the HCIFS Forensic Anthropology Division was formed with the primary responsibility of providing expertise in skeletal analysis at the request of the medical examiner. The Texas Code of Criminal Procedures permits the involvement of a forensic anthropologist in the attempt to establish the cause and manner of death. Furthermore, under the Texas statute, the autopsy must include the retention of body fluid samples, tissues (including bone), and organs to ascertain the cause of death or whether a crime was committed.

During the past 4 years, the HCIFS anthropologists have consulted on 75 cases of suspected child abuse. Typically, a consultation involves a full skeletal examination and the retention of the skeletal element(s) of known or suspected trauma for a complete anthropologic analysis (see Chap. 1). Per protocol, all elements are photographed after processing to document the trauma and the condition of the bone. The photographs taken following standard operational procedure during daily casework are shown in this volume. Also included are photographs of skeletal elements in situ during the autopsy. The purpose of the autopsy photographs is to provide the readers with examples of the trauma when first recognized.

The skeletal injuries most commonly encountered from our examination of child abuse fatalities were the impetus for this volume. The text accompanying the images summarizes the current literature explaining the mechanism of each injury. Additionally, skeletal development, most notably of the skull, is included to assist new practitioners with the differentiation of normal developmental features from trauma.

The goal of Chap. 7 was not to generate a compendium of infant and childhood conditions that affect the quality of bone, but to familiarize the reader with a few disorders that have been described as possible mimics of child abuse in the literature. Most of the diseases described are rare, and we have no direct experience investigating child fatalities associated with them. However, we feel a basic understanding of these conditions is necessary to adequately analyze skeletal lesions in infants and children and to offer effective testimony in child abuse cases.

A forensic anthropology division is relatively rare among medical examiner offices in the United States. Even rarer is the involvement of forensic anthropologists in nondecomposed child abuse cases, such as is the practice of the HCIFS. The numerous child abuse cases investigated by the HCIFS and the strong collaboration between the medical examiners and the forensic anthropologists have led to a large collection of skeletal injury photographs. Given the unique opportunity to analyze a substantial amount of nonaccidental trauma, we feel we have amassed a valuable resource for the medicolegal field and have attempted to share this resource here.

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Sharon M. Derrick, PhD
Jason M. Wiersema, PhD
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1 Skeletal Examination Method

Critical to the appropriate interpretation of possible child abuse is consistent and complete documentation of skeletal injury by all practitioners, including radiologists, clinicians, pathologists, and anthropologists [1, 2]. Complete documentation of skeletal injury by anthropologists requires a standardized method for evaluating the entire skeleton in any case in which injury is suspected. The authors employ a method referred to as skeletal examination to evaluate infants and children with medical histories and/or soft tissue injuries suspicious for inflicted trauma. Skeletal examination involves first incising and reflecting the skeletal muscle and periosteum overlying all long bones, scapulae, and ribs to facilitate detailed inspection for traumatic injury in situ, then removing and chemically processing elements with suspected trauma to generate a comprehensive interpretation of the injury. This method is intended to identify occult fractures typically not recognized during standard radiograph surveys or autopsy by providing greater visibility of the appendicular and axial skeleton [3].

Radiographic Examination

The skeletal examination is preceded by the acquisition of a complete set of skeletal radiographs that clearly illustrate the shafts and articular ends of each of the long bones and ribs, as well as the skull. The radiograph views should include anterior-to-posterior and lateral views of the skull; anterior-to-posterior and oblique (from both sides) views of the chest and abdomen; overall anterior-to-posterior views of the extremities taken in anatomic position, thus avoiding the crossing of long bones and obscuring the underlying bone surfaces; and images of each joint taken with a collimated beam. The anthropologist reviews these radiographs as the initial avenue to identify possible fractures, but the radiographs also represent an additional image-based record of the bones and fractures in their anatomic locations (Figs. 1.1–1.3).
Figure 1-1. Negative anterior-to-posterior chest radiograph showing skeletal elements of the thorax and abdomen of a 5-month-old male. The cause of death was classified as drowning and the manner as homicide.

Figure 1-2. Close-up radiograph of the wrist and hand of a 5-month-old male. Typically, close-up joint radiographs are taken with a collimated beam to reduce X-ray scatter and to facilitate inspection of bones prior to skeletal examination.

Figure 1-3. Radiograph of the arm of a 5-month-old male, with improper orientation of the forearm. The proximal ends of the radius and ulna are superimposed, obstructing the metaphysis of the radius.
Skeletal Examination

Ribs

The ribs are evaluated with the child lying supine; the long bones are examined with the child lying prone. For this reason, the authors advise beginning the skeletal examination by evaluating the ribs. Examination of the ribs first requires removal of the serous pleural membrane adhering to the internal surface of the rib cage. Removal of the pleura involves peeling the membrane laterally and anteriorly from the rib heads to the sternal rib ends. Following removal of the pleural membrane, the internal intercostal muscles are reflected to provide greater visibility of the rib bodies. A horizontal cut is then made through the periosteum along the long axis of each rib from the rib heads to the sternal rib ends. A Cushing periosteal elevator is then used to reflect the periosteum to expose fractures and areas of subperiosteal new bone formation (SPNBF) along the rib bodies. Special attention should be paid to both the costochondral junctions and to the rib head–vertebral articulations for evidence of disruption of the joints. To remove the ribs with suspected trauma, a scalpel cut is made through the cartilaginous portion of the rib head at an approximate 45° angle toward the posterior apex of the transverse process of the vertebra to which the rib is attached. Caution should be exercised to prevent scalpel contact with the delicate cortical bone overlying the rib head (Figs. 1.4 and 1.5).

Figure 1-4. Autopsy photograph of ribs prior to skeletal examination. The image shows the left side of the rib cage of a 2-month-old male following removal of the serous pleural membrane but prior to incision of the periosteum. Note that the pleural surface of several ribs is obstructed by muscle.
Examination of the long bones begins with the decedent lying prone with the extremities in anatomic position to prevent crossing of the long bones of the forearms. Incisions made to examine the subcutaneous tissue of the back, buttocks, arms, and legs during the autopsy are extended through the periosteum to the surface of the long bone, and the overlying muscle is resected with attention given to minimizing the amount of resulting soft tissue destruction. Ligaments and tendons, for example, are cut to facilitate reflection of complete muscle bodies rather than focal destruction of areas of musculature, which results in unnecessary disfigurement. Reflection of the overlying muscles is followed by a longitudinal incision through the periosteum, along the shaft of each bone. Caution should be taken not to cut into the cartilaginous epiphyses. A Cushing periosteal elevator is then used to reflect the periosteum and expose the underlying bone surface and the metaphyses. Additional incisions through the periosteum often are necessary to fully visualize the epiphyses and the chondro-osseus junctions (COJs). Caution should be exercised during this process to recognize and avoid destruction of adherent SPNBF. Certain long bones require additional attention because of their specific morphology. For example, the elbow is flexed during evaluation to provide visibility of the olecranon process of the ulna and the olecranon fossa of the humerus. Elements with suspected trauma are removed for analysis, preferably in their entirety. To remove the element, the attaching ligaments are incised with care not to cut the cartilaginous epiphysis or the bone. Separating the bones by side during collection, processing, and storage helps avoid the confusion associated with siding subadult bones (Figs. 1.6 and 1.7).
**Figure 1-6.** Normal tibial and fibular COJs of a 2-month-old male. The interface between the bone and cartilage is readily visible after the periosteum is removed.

**Figure 1-7.** Close-up view of normal tibial and fibular COJs of a 2-month-old male. Note the regular and straight interface between the bone and cartilage.
**Clavicle**

The same procedure used to expose the surface of the long bones is applied to the clavicles. The authors recommend examining the clavicles after the ribs, before placing the decedent in the prone position.

**Scapula**

To evaluate the scapulae, the infraspinatus and supraspinatus muscles are cut along the medial margin of the scapula and along the scapular spine. The muscles are reflected laterally to expose the underlying periosteum. The periosteum is incised along the same axes, and the periosteal elevator is used to reflect the periosteum in the same fashion as the overlying muscle tissue.

**Documentation and Analysis**

Once the bones are exposed, overview photographs are taken to document the completeness of the skeletal examination; then, photographs are taken of each fracture in situ. As stated earlier, each skeletal element with suspected trauma is removed in its entirety if possible. Artifacts of the autopsy and/or skeletal examination are documented in the bench notes as the examination proceeds. Detailed bench notes record which bones were retained and the location of the suspected fracture(s).

Following removal, the suspect elements are processed to remove all adherent soft tissue and to provide maximum visibility of the bone surfaces. The recommended method for processing the elements is to first fully submerge them in a solution composed of one part of concentrated liquid detergent containing alkaline builders and two parts water, one part detergent. The container in which the specimens are processed should be closed with a lid or cling wrap to prevent evaporation of the processing solution. The temperature of the solution is elevated to a consistent temperature of 60°C for approximately 24 h. The cycle may be repeated if necessary. An incubator facilitates maintenance of the appropriate temperature and ensures minimal if any destruction of the bone during the process. Fragmented elements are reconstructed prior to analysis. The processed bone is then available for gross inspection. All processed elements are evaluated in their entirety both grossly and via a stereomicroscope. An overall photograph of the complete array of elements recovered is taken in approximate anatomic position, and subsequent individual photographs are taken of each element and each injury (Figs. 1.8 and 1.9).
Figure 1-8. Processed skeletal elements. Shown are the elements removed from a 1-month-old male for an anthropologic analysis. All elements were removed during the skeletal examination because of the presence of trauma or suspected trauma to the bone.
Figure 1-9. Postprocessing view of the right tibia of a 4-month-old male. A healing midshaft fracture of the tibia is observed following processing.

References


Head injury, common in children, is the most frequent cause of death among abused children under 2 years of age [1–5]. Skull fractures reportedly are present in between 2 and 20% of children who present to U.S. hospitals for outpatient evaluation of head injuries [6]. Although head injury and skull fracture statistics generally are not separated from each other in the clinical literature, the leading causes of head injury in children, in decreasing order of frequency, are falls, motor vehicle crashes, pedestrian and bicycle injuries, sports-related trauma, and child abuse [7, 8]. Skull fractures are described in the following bones in order of decreasing frequency: parietal, occipital, frontal, and temporal bones [9–12]. Linear fractures are most common in children, followed by depressed fractures and fractures of the basilar skull [9].

Distinguishing accidental from nonaccidental head trauma is an important component of the forensic autopsy, and the thorough detection and appropriate interpretation of skull fractures is critical to the distinction between nonaccidental and accidental skull fractures. The impression of accidental versus nonaccidental cranial trauma has changed considerably during the past few decades. Skull fractures related to inflicted trauma once were described as simple linear fractures most commonly located on the parieto-occipital region of the skull [11], and more complex depressed, comminuted, and/or diastatic fractures of the skull were simply considered uncommon [11]. The consensus in more recent literature is that simple fractures of the type formerly considered suspicious for inflicted trauma are more or less ambiguous in the absence of other injury, and that complex fractures, particularly of the cranial base, are more suspicious for inflicted trauma [13, 14]. Depressed and complex skull fractures remain highly suspicious for inflicted trauma, but abusive skull fractures are often linear and similar to their accidental counterparts in both location and extent [13, 15, 16]. Fractures are more likely to have resulted from inflicted trauma if any of the following characteristics are present: (1) a stellate configuration, (2) depressed margins, (3) a fracture that crosses a suture, (4) multiple intersecting fractures, or (5) involvement of the skull base [10, 17].

Often it is the constellation of skeletal injuries that provides clarity in distinguishing accidental from nonaccidental trauma, because as many as 50% of children who have inflicted rather than accidental head injury also have evidence of other and/or prior skeletal injury [3, 12, 18]. The American Academy of Pediatrics [19] recommends that all children under 2 years of age whose circumstances of injury are suspicious for nonaccidental injury undergo a full radiographic skeletal survey. The relationship between acute abusive head trauma and prior head injury also has been demonstrated by Ewing-Cobbs et al. [20]. These authors indicated that 40–45% of children diagnosed with acute abusive head trauma have CT or MRI evidence of prior head injury. For this reason, the ability to distinguish healing from acute cranial fractures is important.

Most published data regarding the frequency and characteristics of childhood head injury are from a clinical context and thus are based on radiographic interpretation. However, numerous authors have acknowledged the difficulties associated with recognizing and characterizing skull fractures based on radiographs [21, 22]. Normal anatomic features may superimpose fractures, obscuring them in conventional radiographs [21]. Linear fractures that fall in the plane of the CT scan, particularly those of the vertex and basilar skull, also may be overlooked [22]. False positive and/or negative findings may
result from incorrect interpretation of unusual suture and vascular marking locations and atypical morphologic features on radiographs [21], suggesting that a significant underrepresentation of childhood skull fractures in the clinical literature is likely.

This chapter describes the manifestation of skeletal injury to the subadult cranium as seen during the skeletal examination process. Compared with radiologic evaluation, this method facilitates a more detailed gross and microscopic evaluation of suspect cranial bones for the presence and types of fractures, as well as a more detailed interpretation of forces associated with the fracture and the duration of time since it occurred. The chapter first provides an overview of pediatric cranial fracture types and proceeds with a discussion of pediatric cranial anatomy as it pertains to the interpretation of skull fracture, a review of the literature regarding the manifestation of fractures to the various regions of the skull, and a discussion of the distinctions between accidental and nonaccidental fractures; it concludes with a series of recent anthropologic case examples from the Harris County Institute of Forensic Sciences (HCIFS).

**Skull Fracture Types**

Skull fractures are separated into four types: linear, depressed, diastatic, and basilar. Linear fractures are nondisplaced fractures that take a linear path across the skull and involve the entire cross section of bone. In infants and children, linear fractures most often involve the bones of the vault, including the parietal, occipital, and frontal bones. Linear skull fractures result from low-energy transfer associated with blunt trauma over a wide area of the skull [23]. Linear fractures in children may become what are referred to as growing skull fractures (GSFs) in cases in which progressive enlargement of a skull fracture occurs in association with an underlying dural tear or defect [24–26]. GSFs are most common in children under 3 years old and are most commonly located on the parietal bone [27, 28]. GSFs also have been described as rarely occurring in the posterior cranial fossa or the orbital roof in children [29]. GSFs sometimes are referred to as leptomeningeal cysts, traumatic meningocele, cerebrocranial erosion, cephalhydrocele, or meningocele because of the associated soft tissue response. These fractures present themselves in association with herniated cerebral tissue, so they are often easily recognizable.

Depressed skull fractures are associated with high-energy transfer to a relatively small area of the skull. These fractures range in severity from compound comminuted fractures with inwardly displaced fragments located at the point of maximum impact force to simple depressions in the skull without accompanying fracture. The parietal bones are the most common location of accidental depressed skull fractures in both children and adults [11, 30]. Depressed fractures of the cranial vault are particularly common among neonates. These fractures sometimes are referred to as ping-pong fractures because they resemble the indented surface of a plastic ping-pong ball [31, 32].

The original article describing the ping-pong fracture states a single case and notes that the depression was not associated with a fracture [32]. Ping-pong fractures are seen primarily during the first few months of life and are the cranial analog to the greenstick fractures that appear in subadult long bones. Ping-pong fractures are most commonly associated with short falls and rarely with birth trauma [31]. Skull fractures associated with birth trauma are more often depressed than linear and are most often located on the frontal and parietal bones [26, 33].

Diastatic skull fractures are those in which the sutures are widened. These fractures may be associated with a fracture that transverses the suture, or they may be found in the absence of associated fracture or trauma. They are most common in children, because the cranial sutures have not yet fused [34]. Suture diastasis also has been associated with idiopathic brain swelling and an array of congenital disorders, including osteogenesis imperfecta [35, 36].

Basilar skull fractures are linear fractures involving the base of the skull. Most basilar skull fractures involve the temporal bone [37] and may occur at either the squama or base of the temporal bone, or in the area of the occipital condyles.

Crushing head injuries also are relatively common among children [38]. These injuries generally involve accidental crushing forces applied to a stationary head and are characterized by multiple comminuted and intersecting fractures. The forces applied in these injuries generally are slow loading (<200 ms) [38]. Examples of crushing head injuries include those resulting from the wheel of a car passing over the head or from a television or other heavy object being pulled or falling onto the head. Plastic deformation often is associated with these injuries as a result of the slow-loading forces.

**The Pediatric Cranium**

The pediatric skull differs significantly in its structure, mechanics, and anatomy from the adult skull. This distinction has a significant effect on the pattern, type, and frequency of traumatic injury that characterizes the pediatric skull. The following paragraphs summarize the anatomic and structural characteristics of the pediatric skull.
Pediatric Cranial Anatomy

The primary anatomic differences between the adult and pediatric skull are most evident in the perinatal and infant skull. These include several features that are not present in the adult skull, including remnant sutures and fontanelles. These features influence the morphology and propagation of fractures in the infant skull [39, 40]. The bones of the pediatric skull are also generally more elastic than those of the adult, a distinction that also is manifest in the form of variation between adult and pediatric skull fractures [23]. Following is a review of the anatomic characteristics of the pediatric skull that distinguish it from that of the adult.

The neonatal and infant skulls are marked by several developmental sutures and fontanelles that are not present in the adult skull. The anterior fontanelle is diamond shaped and located at the intersection of the frontal and the right and left parietals, at the landmark of bregma in the adult skull (Fig. 2.1). The posterior fontanelle is located at the intersection of the right and left parietals and occipital, the landmark of lambda. The sphenoid (anterolateral) fontanelle is located at the intersection of the temporal squama, sphenoid, parietal, and coronal, at the landmark of pterion. The mastoid (postero-lateral) fontanelle is located at the intersection of the parietal, occipital, and temporal bones, at the landmark of asterion. Additionally, several bones of the vault, including the frontal, occipital, and temporal bones, are composed of independent segments during much of the fetal period. The multiple component development of the bones results in fetal sutures (present in the neonatal period) that may cause confusion during trauma analysis, including the sutura mendosa, which is located on the squamous part of the occipital bone.

The perinatal frontal bone consists of two separate halves, each of which ossifies from a single center, divided vertically by the metopic suture. The opposing sides of the frontal bone are in contact but unfused in infancy. A widening of the metopic suture located near its midpoint is referred to as the metopic fontanelle. The suture mendosa (Fig. 2.2) is the remnant line of fusion between the fetal pars interparietalis and the pars supraoccipitalis. These extend superomedially from the lateral margins of the pars squama toward the center and may extend as much as half the distance between the lateral border and the center of the bone. In the articulated skull, the suture mendosa are continuous with the mastoid fontanelle, and are often referred to as the lateral fissures. There typically is a median fissure that extends some distance from the superior margin of the pars squama into the bone during the perinatal period. This fissure is continuous with the posterior fontanelle. The margins of the pars squama are serrated in infancy. Vascular and nervous grooves appear on the pars squama during the perinatal period. Fusion of the separate components of the occipital bone begins during infancy and continues until the fifth or sixth year. The sutura mendosa begin to close during the fourth month and generally are closed by the end of the first year, although some have suggested they may remain in some form into the third year of life [45]. The pars lateralis and pars squamos initialy are separated by the sutura intraoccipitalis posterior, which begins to fuse by the end of the first year and is completely fused during the third year of life. The pars lateralis fuses with the pars basilaris between the fifth and seventh years of life.

The perinatal temporal bone consists of two separate components: the combined squamotympanic and the petromastoid. These elements fuse during the first year of life, along with gradual expansion of the mastoid processes and the bony auditory meatus. The lateral growth of the petrous portion generally and the meatus specifically results in a dramatic change in the orientation of the tympanic membrane. At birth, the membrane is oriented horizontally and located more inferiorly on the skull. The membrane assumes its more vertical adult orientation by age 4 or 5. The mastoid process grows in length and width according to multiple growth periods that vary between males and females. Overall growth of the squamous proceeds rapidly until age 4, then continues at a slower pace until age 20 [43].

The bones of the face covary in their morphology and development in a very complex way [41]. At birth, the maxilla is very small and its shape is determined largely by the underlying developing deciduous tooth buds, whose crypts form noticeable bulges along the anterior surface. The tooth buds are very near the base of the eye orbit, and the maxillary sinuses are present but very small as a result. The alveolar bone is primarily cancellous and continuously remodels as the tooth buds grow. The overall growth of the maxilla is dictated by two major influences: growth in response to the need to accommodate more and larger teeth and growth in response to the development of the surrounding anatomy, including the eyes and the nose. The maxillary sinuses expand from the postnatal period into adulthood. Growth of the mandible keeps pace with growth of the maxilla, and also is dictated by the growth of the dentition. At birth, the mandible consists of two halves divided at a centrally located symphysis. The symphysis fuses during the first year. The pediatric mandible consists primarily of trabecular bone wrapped in thin, constantly changing cortical bone. The dimensions of the
mandible, including the depth of the chin, height of the ramus, angle of the mandible, and bigonial breadth, change significantly during childhood as a result of the growth of the dentition [45].

At birth, the zygomatic bones are triradiate in shape, with recognizable overall morphology [41]. They achieve adult proportions, but not size, by the second or third year in conjunction with the completion of the deciduous dentition. Growth of the zygomatics proceeds along with the development of the attached maxilla as it grows to accommodate rapid dental development. This process results in a shift in the location of the zygomaticomaxillary junction and the orientation of the zygomaticomaxillary suture.

Pediatric Cranial Mechanics

Several variables influence both the likelihood and the pattern of skull fracture, including differences in level of force, impact surface area and type, skull cross-sectional thickness, thickness of the scalp and hair, and impact location and direction [46–48]. Additionally, the structure/morphology of the subadult cranium is distinct from that of the adult in several ways that critically influence its response to traumatic insult [21, 49–52]. This distinction is the result of physiologic influences on cranial anatomy related to growth and development. The primary anatomic distinctions between the subadult and adult skull are the relative dimensions of face and vault, variation associated with the development of the dentition, the degree of pneumatization of the frontal and paranasal sinuses, and overall changes in the relative size of the various components of the skull [49]. The impact of each of these factors is addressed under "Skull Fracture Location."

Specific anatomic differences also exist between the structure of adult and pediatric cranial bone that can influence the manifestation of traumatic injury. For example, the neurocranium bones of infants and very young children are thinner and may lack the typical three-layered structure that characterizes mature cranial bones (Fig. 2.3). The diploe that separates the inner and outer tables of the bone of the cranial vault is generally thought to develop in early childhood, although the timing is debated [38, 53]. The development of the three-layered structure of cranial bone has implications beyond the mechanical response of the cranial bones to trauma because the meningeal artery grooves arise with the development of the diploe, which increases the susceptibility of the meningeal arteries to damage in adulthood [54]. Another structural distinction between adults and children is the relatively tighter adherence of the dura mater to the intracranial surface in children under 2 years of age relative to adults [55].

Decades of experimental and clinical research have attempted to elucidate the biomechanical response of the skull to impact loads in both adults [56–61] and children [62, 63]. Gurdjian [58, 59] first argued that skull fractures initiate at a site remote from the impact rather than at the site of impact. This often-cited opinion is supported by significant recent experimental literature [64, 65]. Researchers suggest that fractures initiate at sites of outbending surrounding the central area of
Skull Fractures

The specific location of fractures on the subadult skull reflects the type and circumstances of injury. The epidemiology of cranial fracture changes with age, and the resemblance between adult and subadult cranial bone fracture location frequencies increases with age. The adult skull is prone to fracture at anatomic sites that are thin, and fractures are more common in portions of the skull that do not have a three-layered structure (i.e., do not have diploe). These areas include the squamous temporal bones, the sphenoid ala, the floor of the middle cranial fossa, the cribriform plate, the roofs of the eye orbits, and the region of the posterior cranial fossa located between the mastoid and dural sinuses (sigmoid and transverse sulci) [41]. The following paragraphs describe the patterns of fracture common to specific regions of the skull and their general etiology. This volume divides the skull into three anatomic regions based on consideration of the fracture types and locations as they relate to trauma type: the vault, facial, and cranial base regions (Fig. 2.4). The vault region includes the frontal bone superior to the frontal sinus, the complete parietales, and the occipital bone from the posterior margin of the foramen magnum posteriorly. The facial region includes the inferior segment of the frontal bone including the frontal sinus; the upper margins of the orbits, maxilla, zygomatics, and mandible; and the floor of the anterior cranial fossa, including the orbital roofs. The cranial base includes the segment of the occipital bone anterior to the posterior margin of the foramen magnum (including the occipital condyles), the temporal bones in their entirety, and the sphenoid bone.

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 Vault Fractures

Skull fractures documented in the clinical literature as associated with nonaccidental childhood injury are most commonly located on the parietals and occipital [67]. Geddes et al. [68] noted that 18 of the 19 skull fractures identified in a sample of 53 children aged 1 month to 8 years who died as a result of inflicted head trauma were located on either the parietal or the occipital. Hobbs [10] listed the following fractures as being more common in nonaccidental than accidental injury cases: depressed skull fractures, diastatic fractures wider than 3 mm, nonparietal fractures, fractures having a complex configuration, multiple calvarial fractures, bilateral fractures, and fractures that cross sutures. He stated that the presence of one or more of these features is reason to suspect abuse, and that most of the cases in his sample had two or more of them; however, as Meservy et al. [11] pointed out, he provided no statistical support for his conclusion. Meservy et al. [11] used a sample of radiographs of 134 children under 2 years of age to investigate distinctions between accidental and nonaccidental skull fractures. The parietal bone was the most frequently fractured in both the abuse and accidental groups, occurring in 88% of the abused children and 91% of the accidental sample. The authors found that the presence of multiple fractures, bilaterally located fractures, and fractures that cross sutures were more common in the abuse sample than in the accidental one. Their conclusion is distinct from Hobbs’s [10] in that they found no significant difference between accidental and abusive fractures in terms of the specific morphology of depressed, diastatic, nonparietal, or complex fractures. In concurrence with Hobbs’s [10] conclusion, Meservy et al. [11] attached greater suspicion to the presence of multiple fractures, bilateral fractures, and fractures crossing sutures. Of note, the sample of Meservy et al. [11] comprised children who survived their injuries, and it is likely that the children in Hobbs’s [10] sample, some of whom were deceased, were injured more severely as a whole.

Oon and Yu [69] completed a survey of 50 children aged 3 months to 4 years who had either accidental or nonaccidental fractures on the posterior aspect of the parietal bone extending anteriorly from the lambdoidal suture, most frequently in the approximate superior–inferior midpoint of the suture. The authors noted a frequent (32 of 50) association between these fractures and Wormian bones, an inca bone, or accessory posterior parietal sutures, arguing for a causal relationship between the fractures and these anomalies. Figures 2.5–2.46 are case examples of pediatric cranial vault fractures.
Figure 2-7. Postprocessing view of the endocranial surface of the occipital bone of the individual in Figs. 2.5 and 2.6. The fracture intersects the intraoccipital suture (arrowhead) at the margin of the foramen magnum (paired arrowheads). Early fracture bridging is present along the fracture (arrow).

Figure 2-8. Endocranial surface of the occipital bone of the individual in Figs. 2.5–2.7. The fracture intersects the intraoccipital suture at the margin of the foramen magnum (arrowhead).

Figure 2-9. Posterior autopsy view of the skull of a 2-year-old female. A complex pattern of perimortem intersecting fractures is located on the occipital bone. Remote fractures are present on the appendicular and thoracic skeleton. The cause of death was classified as blunt trauma of the head with subdural hematoma and skull fracture, the manner as homicide.

Figure 2-10. Postprocessing view of the endocranial surface of the posterior skull of the individual in Fig. 2.9. A complex pattern of intersecting fractures that incorporates a portion of the lambdoidal suture is located on the occipital bone (arrowhead). A portion of the fracture is depressed. The fracture pattern is consistent with a posterior-to-anterior-directed impact with an object of relatively small surface area.
Figure 2-11. Postprocessing ectocranial view of a stellate fracture pattern on the superior portion of the occipital bone of the individual in Figs. 2.9 and 2.10. The arrowheads indicate the path of the fracture.

Figure 2-12. Posterior (A) and lateral (B) autopsy views of the skull of a 6-month-old female. A complex pattern of fractures is visible on the posterior right parietal (arrowheads). The cause of death was classified as blunt force head trauma, the manner as homicide.

Figure 2-13. Postprocessing view of the right and left parietals and a segment of the occipital bone from the individual in Fig. 2.12. A complex pattern of fracture is located on the right parietal (arrowhead). The fracture is three pronged with radiating fractures extending superior-anteriorly, anterior-inferiorly, and posteriorly from the point of intersection. The two anteriorly radiating fractures terminate within the right parietal bone, and the bone between them is slightly depressed.
Figure 2-14. Close-up view of the right parietal and occipital bones in Fig. 2.13. An additional linear fracture (arrowhead) extends inferiorly from the lambdoidal suture and is continuous with the aforementioned fracture via the diastatic fracture of the lambdoidal suture. The fracture pattern is consistent with a superior-to-inferior, posterior-to-anterior-directed impact. The fracture margins are sharp, and there is no gross evidence of healing at or near the fracture sites.

Figure 2-15. Autopsy view of the right side of the skull of a 9-month-old male. A stellate fracture pattern is located on the posterior right parietal (single arrowhead), and a widely separated radiating fracture extends anteriorly to terminate in the coronal suture (paired arrowheads). The cause of death was classified as blunt trauma of the head with skull fractures and subdural hemorrhage, the manner as homicide.

Figure 2-16. Endocranial view of the right parietal bone in the individual in Fig. 2.15. The fracture margins are sharp, with no indication of a healing response. The pattern of fracture is consistent with an impact in a right-to-left direction with an object of relatively small surface area.
Figure 2-17. Cranial bones. Shown are right lateral (A), oblique right frontal (B), posterior (C), and left lateral (D) autopsy views of the skull of a 1-month-old male with multiple visible fractures (arrowheads). The entire calotte was recovered for anthropologic analysis. The fractures were of both acute and remote origin. The cause of death was classified as blunt trauma of the head with skull fractures and brain contusions, and the manner as homicide.
Figure 2-18. Postprocessing view of the bones retained for analysis from the individual in Fig. 2.17. The autopsy cut transects the skull through the parietal bones in the coronal plane. A perimortem, tri-pronged stellate fracture is located near the right parietal eminence, with radiating fractures extending anteriorly, posteriorly, and medially from the impact site. The fracture pattern is consistent with a minimum of one direct blow to the right cranium in a right-to-left direction with a relatively broad object.

Figure 2-19. Postprocessing view of the left parietal bone from the individual in Figs. 2.17 and 2.18. The single arrowheads indicate a remote transparietal fracture across the body of the parietal bone. The fracture extends anteroposteriorly from the coronal suture to the lambdoid suture. Primary new bone formation is present along the fracture margins, and the fracture is partially stabilized. Note that the bone is transected by the autopsy cut (paired arrowheads). An acute fracture also is visible superior to the remote fracture (arrow).
Figure 2-20. Postprocessing views of the frontal bone pictured in Fig. 2.18. (A) Ectocranial view showing only a subtle indication of a fracture (arrowhead). (B) Close-up view of the ectocranial surface. A depressed fracture is observed near the coronal suture (arrowhead).

Figure 2-21. Posterior autopsy view of the skull of a 4-month-old male. A complex fracture pattern is present involving the left parietal and occipital bones near lambda. The fracture is curvilinear and courses through the parietal bone anteriorly and to the left from lambda, then extends posteriorly and inferiorly across the lambdoidal suture, terminating in the occipital squama just left of and superior to the external occipital protuberance (fracture path indicated by arrowheads). The fracture creates a displaced fragment of parietal bone. The fracture pattern is consistent with an impact to the posterosuperior skull immediately left of lambda with an object of relatively small surface area. Note the diastatic separation of the lambdoidal and sagittal sutures. The cause of death was classified as blunt trauma of the head and the manner as homicide.

Figure 2-22. Postprocessing view of the occipital skull fracture pictured in Fig. 2.21. The margins of the fractures are sharp, without evidence of a healing response. Evidence of previous skeletal injury was located on the ribs and long bones.
Figure 2-23. Close-up postprocessing view of the occipital fracture pictured in Fig. 2.22. The photograph illustrates the sharp edges of the fracture and the associated lack of a healing fracture response.

Figure 2-24. Autopsy view of the posterior right side of the skull of a 15-month-old female. A stellate fracture is observed on the occipital bone (arrowhead). The horizontal component of the fracture is located at the level of the sutura mendosa, a developmental suture within the forming occipital squama. The vertical component of the fracture extends inferiorly from the horizontal component and terminates in the sutura intraoccipitalis posterior. The cause of death was classified as complications of blunt trauma of the head with subdural hematoma and skull fracture, the manner as homicide.

Figure 2-25. Close-up view of the fracture pictured in Fig. 2.24 (arrowheads mark the fracture). A segment of the skull including a portion of the occipital and right parietal bones was retained for anthropologic analysis.

Figure 2-26. Endocranial view of the occipital skull fractures pictured in Figs. 2.24 and 2.25. The photograph depicts the path (as indicated by arrowheads) of the fracture on the endocranial surface of the occipital bone.
Figure 2-27. Postprocessing view of the cranial segment recovered from the individual in Figs. 2.24–2.26 for anthropologic analysis. Evidence of a healing response is located along the fracture margins.

Figure 2-28. Healing linear skull fracture. Shown are close-up postprocessing views of the fracture pictured in Figs. 2.24–2.27. The fracture is in the initial stage of healing, as shown on the endocranial (A) and the ectocranial (B) surfaces. It is open, with fibrous tissue bridging and minimal SPNBF. The fracture type is consistent with a direct impact to the posterior right skull in a posterior-to-anterior direction with an object of relatively small surface area.
Figure 2-29. Autopsy views of the left (A) and right (B) sides of the posterior skull of a 4-month-old male. Note the fractures inferior to the lambdoidal suture on both the left and right sides of the bone (arrowheads). A specimen including the occipital squama, a posterior segment of the left pars lateralis, posterior segments of the right and left parietal, and a segment of the squama of the left temporal was recovered for anthropologic analysis. The cause of death was classified as blunt trauma of the head and the manner as homicide.

Figure 2-30. Postprocessing views of the ectocranial (A) and endocranial (B) surfaces of the occipital bone from the individual in Fig. 2.29. Bilateral linear fractures (arrowheads) were identified extending from the right and left sides of the lambdoidal suture near asterion superiorly and medially and meeting inferior to lambda. Diastatic fractures were identified on the left lambdoidal and left posterior squamosal suture.
Figure 2-31. Close-up views of the left (A) and right (B) fractures of the occipital bone pictured in Figs. 2.29 and 2.30. There is partial union of both fractures with woven new bone formation, as well as fracture bridging (arrowheads). The fracture pattern is consistent with a minimum of two blows: one to the right posterior cranium and one to the left posterior cranium. The healing pattern is consistent with a single traumatic event, evident by the uniform new woven bone along the fractures.

Figure 2-32. Postprocessing view of the occipital bone of a 1-month-old female. A segment of the left parietal also was recovered for anthropologic analysis. Three perimortem fractures were noted on the bones of the skull: two on the occipital and one on the left parietal. The first occipital fracture is curvilinear and extends inferomedially from the midpoint of the left half of the lambdoidal suture (arrowhead). The second occipital fracture is linear and extends superomedially from the left sutura inoccipitalis posteriorly into the squama left of the external protuberance (paired arrowheads). The fracture margins are sharp, and there is no bony reaction at or near the fracture sites. The cause of death was classified as hypoxic ischemic encephalopathy due to multiple blunt force injuries, including skull fractures and subarachnoid hemorrhage, and the manner as homicide.
Figure 2-33. Left side of the occipital bone pictured in Fig. 2.32. (A) Note the fracture (arrowhead) and a second defect (arrow). The second defect is a remnant of the sutura mendosa. (B) Close-up view of the sutura mendosa (arrowhead).

Figure 2-34. Fracture located on the left parietal from the case shown in Figs. 2.32 and 2.33. The fracture is linear and extends inferiorly from the sagittal suture to an area immediately posterior to the parietal eminence.

Figure 2-35. Autopsy view of the superior surface of the skull of a 10-month-old male. The decedent underwent a craniotomy prior to death (note the burr hole indicated by the single arrowhead). The bones were evaluated in situ (the craniotomy defect is indicated by paired arrowheads). Both the cause and manner of death were classified as undetermined.
Figure 2-36. Left lateral autopsy view of the skull pictured in Fig. 2.35. The trauma in this case was limited to the anterior, posterior, and right sides of the skull (there were no acute or remote fractures on the left side).

Figure 2-37. Postero-inferior autopsy views of the skull pictured in Figs. 2.35 and 2.36. (A) Two acute linear fractures are observed on the occipit: one vertically oriented (arrowheads) and one horizontally oriented (arrow). The horizontal fracture extends medially from the sutura mendosa. The fracture pattern is consistent with a direct impact to the posterior cranium against an object of relatively broad surface area. (B) The first fracture is vertically oriented and extends from the right lambdoidal suture, continuing through the squama, and terminating immediately right of the posterior midline of the foramen magnum (arrowheads indicate the fracture path). (C) The fracture bifurcates between the inferior nuchal line and the foramen magnum, creating a triangular fragment.
Figure 2-38. Autopsy view of the occipital skull from the case shown in Figs. 3.35–3.37. The second linear fracture (arrowhead) is horizontally oriented and extends medially from left asterion. Laterally the fracture is diastatic, involving the sutura mendosa.

Figure 2-39. Autopsy view of the right side of the skull shown in Figs. 2.35–2.38. The right side of the skull was the site of the craniotomy (note the burr holes indicated by the arrowheads). The craniotomy defect noted in Fig. 2.35 is visible along the superior margin of the skull.

Figure 2-40. Autopsy view of the anterior surface of the skull shown in Figs. 3.35–3.39. The autopsy cut traverses the frontal bone and intersects the craniotomy cut.

Figure 2-41. Autopsy view of the right side of the skull of a 2-month-old male. Note the linear fracture located on the right parietal (arrowheads). No other trauma was noted during the skeletal examination. Segments of the right parietal and the occipital bones were retained for anthropologic analysis. Both the cause and manner of death were classified as undetermined.
Figure 2-42. Postprocessing view of the segment of the right parietal recovered from the individual shown in Fig. 2.41. A linear fracture extends from the approximate midpoint of the right squamosal suture posteriorly to the lambdoidal suture (arrowheads). The fracture margins are sharp, and there is no evidence of healing. The fracture location and type are consistent with an impact to the right side of the skull with an object of relatively broad surface area.

Figure 2-43. Close-up view of the fracture shown in Figs. 2.41 and 2.42. The quality of the bone in this decedent is abnormally thin and porous. The compromised quality of the bone likely decreased its ability to withstand impact, possibly leading to fracture from forces associated with normal activity. The arrowhead indicates a postmortem fracture (autopsy artifact).

Figure 2-44. Postprocessed occipital bone segment recovered from the individual shown in Figs. 3.41–3.43. Panel (B) is a close-up of panel (A). Note the areas of abnormally thin translucent bone indicated by the arrowheads (B). A remnant sutura mendosa (A) is present on the bone and is not of forensic concern (paired arrowheads).
Figure 2-45. Crushing head injury. (A) Superior view of the calotte. (B) Endocranial surface of the skull base. The 4-year-old female was thrown from the back of a horse, and the horse subsequently stepped onto the side of her head. The cause of death was classified as blunt head trauma, the manner as accident.
Figure 2.46. Calotte of the individual pictured in Fig. 2.45. Shown are the superior (A), endocranial (B), right lateral (C), and left lateral (D) surfaces. The calotte is marked with two complex patterns of radiating and concentric fractures. One of the fracture patterns is large, extends anteriorly and medi- ally from the posterior left quadrant of the calotte, and involves the left and right parietal, occipital, and frontal bones. The other fracture pattern is small, located on the right anterior region of the calotte, and concentrated primarily on the frontal bone. The calotte also is marked with significant cranial deformation, resulting in artificial plagiocephaly.

The fracture patterns and deformation are consistent with two opposing forces applied to the skull simultaneously. The larger fracture pattern of the left posterior calotte is consistent with a slow-loading impact with an object of broad surface area, and the small fracture pattern of the right anterior calotte is consistent with an impact with an object of a relatively small surface area. All fracture margins are sharp, with no evidence of healing. No additional antemortem or perimortem trauma is observed on the specimen. The quality and development of the bone is as expected for the individual’s reported age of 4 years.
Maxillofacial injuries/fractures are rare, and less frequent in children than in adults [50–52, 70–72]. Pediatric facial fractures account for approximately 5% of all facial fractures [71]. The frequency of subadult facial fractures is lowest in infancy and increases with age [72]. The low occurrence of pediatric facial fracture is attributed to the higher cranial vault-to-facial skeleton ratio in children, the elastic composition of pediatric facial bones, the presence of protective thick soft tissues, and the lack of pneumatization of the paranasal sinuses [73]. Some authors suggest that facial fractures in children may also be underreported because of difficulties associated with their recognition on radiographs, particularly as a result of the presence of unerupted tooth buds, and the minimal pneumatization of the paranasal sinuses [49, 70]. The pattern of facial bone fracture also differs between children and adults [49, 70, 72]. This variation is attributed to differences in the developmental status of the bones of the face and dentition and to the degree of pneumatization of the involved sinuses. Skeletal injuries of the subadult face vary in severity from minor fractures to complex fractures involving some combination of orbital zygomatic and maxillary complexes.

There is a considerable lack of consensus in the literature as to the primary causes of pediatric facial fracture, primarily because of the variation in ages of the children surveyed. However, most pediatric facial fractures are the result of motor vehicle accidents, followed by sports-related injury, falls, and nonaccidental trauma. The number attributed to nonaccidental injury varies widely (3.7–61%), with most authors reporting it in the 10% range [52, 62, 72, 73]. Most fractures involving the subadult face are associated with very high-energy forces and are regularly associated with other neurocranial injuries [63].

### Inferior Frontal Bone Fractures

In young children, particularly those younger than 7 years, the large cranial vault is more exposed than the relatively smaller face to trauma and frontal bone fractures are common [50, 70]. The increased proportion of the cranial vault relative to the face in children has been associated with a higher proportion of upper facial fractures in children, as opposed to the greater proportion of lower facial fractures in adults [74]. In the absence of the frontal sinus, the forces associated with impacts sustained directly to the frontal eminence transfer either superiorly across the body of the frontal bone or posteriorly across the orbital roof, resulting in fractures at these locations [62]. Fractures of the orbital roof are relatively common and often radiographically occult in young children [75]. Greenwald et al. [75] associated isolated orbital roof fractures with younger children (mean age, 2.8 years). Most of these fractures are linear and often associated with relatively minor trauma. The authors used a fall from a height of less than 10 feet as an example of minor trauma. Messinger et al. [76] found that maxillofacial fractures were associated with orbital roof fractures in 30% of a sample of children with an average age of 3.3 years. They concluded that most children with orbital roof fractures do not have associated facial fractures and that each patient in the fracture sample lacked frontal sinus pneumatization. Orbital roof fractures often are characterized as nondisplaced, blow-out, or blow-in [77]. Figures 2.47 and 2.48 are photographs of an acute orbital roof fracture in a 9-month-old female.
Midface, and Orbital Rim Fractures

The reduced proportional emphasis on the orbits relative to the adult, in combination with the lack of frontal sinus pneumatization and the associated tendency toward fracture of the frontal bone and orbital roofs, limits the number of orbital rim fractures in children [63]. Midface fractures including the floors of the orbits and the maxilla are rare in children, particularly those under 2 years of age. The frequency of these fractures increases with age-related structural changes of the face, particularly sinus pneumatization. Midface fractures in children generally are associated with high-energy impacts such as motor vehicle collisions [70]. Fractures of the midface become increasingly common as development of the paranasal sinuses progresses [51, 70, 78, 79]. Fractures to the medial and lateral rims of the orbits increase in frequency after age 7, and there is a concomitant decrease in orbital roof fractures [51, 70]. Blow-out or trapdoor fractures of the orbital floor generally are associated with direct impacts to the eye in children [79]. These fractures are circular defects in the orbital floor in which one of the fracture margins is displaced inferiorly.

Zygomatic fractures are more common than other midfacial fractures in children [51, 70]. In children, these fractures often manifest as greenstick fractures of the zygomatic bone and/or the lateral eye orbit. Zygomatic fractures are categorized as low energy with minimal or no displacement, medium energy with minimal displacement and mild comminution, and high energy with severe displacement and comminution of the zygomatic processes and arch [80].

The reduced anterior protrusion of the nose relative to the vault is associated with a lower incidence of nasal bone fractures in children than in adults [81, 82]. Nasal fractures, however, are the most common facial fractures among children [81]. Nasal fractures in children often are limited to the septum rather than the nasal bones. The septum may be either fractured longitudinally or dislocated entirely. Asymmetric nose tip deformity, characterized by laterally displaced nasal bones and septum, is sometimes present in newborns and has been attributed to both birth trauma and an abnormal intrauterine position. With the exception of cases in which the septum is completely dislocated from the septum, the nose tip deformity pattern generally is limited to newborns. There is considerable debate regarding the appropriate means to correct these deformities [82–84].

Complex fractures such as Le Fort fractures represent a very small number of facial fractures in children [51] and are exceedingly rare in children less than 2 years old [70]. Most of these fractures are present in children over 10 years old [79]. The Le Fort classification system is used to classify complex facial fractures in the same way for adults and children.
Mandible Fractures

Mandible fractures are second to nasal fractures in their frequency among facial fractures in children [62, 70]. Keniry [85] suggests that between 8 and 15% of mandible fractures occur in children younger than 15, but MacLennan [86] found that only 1% of mandible fractures occurred in children under 6 years old. The ratio of bone to tooth tissue is high in children, and the thin cross section of the mandibular cortical bone predisposes the pediatric mandible to greenstick fracture [87, 88]. The condyles are the site most commonly associated with fractures in children, followed by the symphyseal region. Condyle fractures were present in 72% of children with mandible fractures in a 1992 study of 220 children under the age of 15 [89]. Fractures of the angle and body regions are relatively less common but increase in frequency with age. The common pediatric mandible fracture pattern is attributed to the thin cortical bone overlying the heavily vascularized medullary bone in the mandibular condyles in children [70]. An additional difference between the adult and pediatric mandible is that a single impact to the mandible in children tends to cause a single fracture, whereas in adults it tends to cause multiple fractures. The distinction between the adult and subadult mandible fracture pattern ceases after about age 9 [88]. Figures 2.49–2.51 are photographs of an acute mandible fracture in a 9-month-old female.

**Figure 2.49.** Autopsy view of the maxilla and mandible. An incomplete vertical fracture is located near the symphysis (arrowheads) of a 9-month-old female. No fractures were located on or near the condyles. The cause of death was classified as blunt force head trauma with skull fractures and brain injury, the manner as homicide.

**Figure 2.50.** Mandibular fracture. Shown is an inferior view of the mandible of the individual pictured in Fig. 2.49. Note the incomplete mandibular fracture (arrowhead). The location and characteristics of the fracture are consistent with an anterior-to-posterior-directed impact with an object with a medium to wide surface area.
Cranial Base Fractures

In children, cranial base or basilar skull fractures are most commonly located on the temporal bone and the anterior occipital bone in the area of the occipital condyles. There are three common varieties of temporal basilar skull fractures: longitudinal, transverse, and mixed [36]. Longitudinal fractures are the most common and typically involve the squamous portion of the temporal bone. Transverse fractures are less common and travel from the foramen magnum through the inner ear into the middle cranial fossa. The remainder of cranial base fractures share components of both longitudinal and transverse fractures, thus are referred to as mixed. Occipital condylar fractures (OCFs) are also subdivided into three varieties: type I, type II, and type III [90]. A type I fracture is associated with axial compression resulting in a comminuted fracture of the occipital condyle. A type II fracture is associated with a direct blow and often manifests as extensive fractures of the basioccipital region. A type III fracture is an avulsion injury associated with hyperrotation and lateral bending. In general, basilar skull fractures are associated with high-energy transfer from blunt trauma, axial compression, lateral bending, or rotational injury.

In a sample of 62 patients under 18 years old with basilar skull fractures, Liu-Shindo and Hawkins [91] attributed the injury most commonly to pedestrians struck by motor vehicles (42%), followed by falls (27%), motor vehicle collisions (23%), and being hit by an object (i.e., baseball bat) (8%). OCFs are rare in children and most often result from motor vehicle collisions and sporting accidents with other associated head injury [92]. Cranial base fractures are difficult to recognize on both CT and conventional radiography [54]. Figure 2.52 is a case illustration of an acute pediatric cranial base fracture.
Figure 2-52. Autopsy views of the endocranial surface of a 9-month-old female. Note the linear fracture of the squamous temporal bone indicated by arrowheads (A) and in close-up (B). The fractures are devoid of any indication of a healing response. The cause of death was classified as blunt force head trauma with skull fractures and brain injury, the manner as homicide.

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Rib fractures are recognized as strong indicators of nonaccidental injury in infants and children and are more prevalent than any other type of skeletal injury in cases classified as child abuse [1–13]. They remain strong indicators of nonaccidental injury in older children (>18 months) when other types of fractures become more prevalent with accidental injury [1]. Several researchers have found that the average number of rib fractures is significantly higher in cases of nonaccidental injury than in those of accidental injury and that rib fractures often are the only skeletal injury observed in cases classified as child abuse [1, 2].

The consensus among researchers is that regardless of the type of injury, rib fractures are associated with a major traumatic event (i.e., motor vehicle crash), and without an adequate history, nonaccidental injury must be considered a cause [5, 14, 15]. Exceptions include pathologic fractures (see Chap. 7) and rib fractures secondary to organ recovery. The standard method for recovery of the heart and lungs involves a median sternotomy followed by spreading of the ribs. Kleinman and Schlesinger [8] produced rib fractures in the posteromedial region by opening a median sternotomy with a sternal retractor. A median sternotomy with forcible lateral spread of the anterior ribs places excessive force on the posteromedial region of the ribs, increasing the potential to cause posterior rib fractures (see section “Posterior Rib Fractures”).

**Rib Fracture Location**

The location of a rib fracture is considered a valuable indicator of the mechanism of trauma [2, 3, 7, 8, 16, 17]. However, a literature review of infant rib fracture studies shows there is no standard definition for identifying the regions of a rib. Most simply, the rib is divided into four regions: posterior, posterolateral, anterolateral, and anterior (Fig. 3.1). The posterior region is the area from the medial tip of the rib head to the lateral margin of the rib tubercle. The posterolateral region is the area from the lateral margin of the rib tubercle to the most lateral point of the rib. The anterior and anterolateral regions of the rib span the most lateral point of the rib to the sternal end. In the upper and midrange ribs, the demarcation between the anterior and anterolateral regions is the midpoint that approximately aligns with the middle of the clavicle bone (midclavicular). In the lower-range ribs, the anterolateral and anterior regions of the rib become increasingly smaller. In the 11th and 12th ribs, there is no anterior or anterolateral portion of the rib; the complete rib lies in the posterior region of the thorax.
Of the four regions of the rib, fractures located in the posterior region are regarded as the strongest indicator of nonaccidental injury [2, 3, 7, 8, 15, 16, 18]. Posterior rib fractures are often multiple, arranged in a serial pattern, and bilateral [16]. Kleinman et al. [7] found that of 84 rib fractures identified in 31 infant victims of nonaccidental injury, 53 fractures (63%) were located in the posterior region of the rib. Bulloch et al. [2] conducted a retrospective study of infants treated at two children’s hospitals over a 3-year period and identified 39 cases with rib fractures; 32 (82%) of the cases were classified as child abuse. The authors found posterior rib fractures were more common than anterior ones but found no significant difference in location of the fracture between nonaccidental and accidental injury.

Mechanisms postulated to cause posterior rib fractures include avulsive forces applied to the rib head and posterior levering of the rib over the transverse process of the thoracic vertebra [2, 8, 15, 19]. The developing rib head is primarily hyaline cartilage and is analogous to the metaphysis of a long bone at the chondro-osseous interface. The structure is held in place with strong ligamentous attachments. When excessive force is applied to the costovertebral articulation, an osseous fragment is avulsed from the rib head (Figs. 3.2–3.9) [8].
Rib Fractures

Figure 3-2. Rib head fractures in situ. Rib head fractures (arrows) are shown in situ in a 2-month-old male. Note the absence of hemorrhage at the fracture sites. The cause of death was classified as blunt trauma of the head with skull fractures and subdural hemorrhage, the manner as homicide.

Figure 3-3. Avulsion fracture of the rib head. An avulsion fracture of a rib head is shown after processing; the view is of the articulating surface (same individual as shown in Fig. 3.2). Note the exposed trabecular bone of the articulating surface of the rib head. The fracture is similar in structure to a partial classic metaphyseal lesion (see Chap. 5).

Figure 3-4. Avulsion fracture of the rib head. An avulsion fracture of a rib head is shown after processing (same individual as shown in Fig. 3.2). The cortical bone of the articulating surface of the head has been fractured, exposing the trabecular bone.

Figure 3-5. Avulsion fracture of the rib head in situ. An avulsion fracture of a rib head (arrow) is shown in situ in a 2-month-old female. The cause of death was classified as methicillin-resistant Staphylococcus aureus (MRSA) chest wall abscess with disseminated infection and a contributing factor of chronic stress associated with multiple blunt trauma injuries, the manner as homicide.
Figure 3-6. Partial avulsion rib head fracture. This partial avulsion fracture of the rib head was observed in a 2-month-old male; the view is of the pleural surface. The cause and manner of death were classified as undetermined.

Figure 3-7. Healing rib head fractures in a 2-month-old male. Note the thickened cortical bone (arrows) and the lipping of the bone callus over the articulation surface of the rib head. The cause and manner of death were classified as undetermined.

Figure 3-8. Healing rib head fracture. This healing rib head fracture (arrow) was observed in an 11-week-old female with minimal fracture callus formation. A healing fracture of the rib neck (arrowhead) also is present. The cause of death was classified as blunt force head trauma, the manner as homicide.

Figure 3-9. Rib head fractures in a 4-month-old male who underwent a median sternotomy during organ recovery. Several healing rib fractures and an acute cranial fracture were noted in the decedent. The rib head fractures were the only acute rib fractures observed. Because of the surgical history, organ recovery could not be excluded as a possible cause of the fractures. The cause of death was classified as blunt force trauma to the head, the manner as homicide.
Kleinman et al. [7, 8, 16] found that when the chest is forced posteriorly, a classic level 1 lever is formed at or near the costovertebral articulation point (the rib tubercle and vertebral transverse process). The costovertebral ligaments are stronger than the bone, and excessive forces result in the failure of the rib in the region of the rib neck and tubercle (Figs. 3.10–3.15). Based on animal experimentation and assailant testimony, Kleinman [16] suggested that posterior rib fractures result when an infant is held by the chest while the thorax is squeezed in the anteroposterior plane. The infant is held facing the adult with the adult’s palms on the infant’s sides, fingers on the back, and thumbs near the midline of the chest. Kleinman [16] also postulated that similar forces occur when the chest is forced onto a broad surface while the back is unsupported. Kleinman and Schlesinger [8] reported that posterior rib fractures do not occur when the anterior chest is compressed and the back is supported on a broad surface (such as with the administration of cardiopulmonary resuscitation [CPR]).

**Figure 3-10.** Posterior rib fractures. These acute posterior rib fractures (arrows) were found in a 2-month-old female. The cause of death was classified as acute bacterial bronchopneumonia associated with bronchopulmonary dysplasia due to prematurity, the manner as natural. Decreased bone quality has been documented as associated with prematurity in the neonatal period (see Chap. 7). The decedent was born at 27 weeks’ gestation.

**Figure 3-11.** Posterior fracture of the rib head (same individual as shown in Fig. 3.10). Note the porotic cortical bone of the rib head (arrow). The hyperporosity reduced the bone’s ability to withstand low-level forces.

**Figure 3-12.** Acute incomplete fracture of the rib neck in a 21-month-old female. The fracture was interpreted as a failure in tension due to posterior levering of the rib over the transverse process of the vertebra. The cause of death was classified as blunt trauma of the torso with rib fractures, liver laceration, and traumatic transection of the pancreas, the manner as homicide.

**Figure 3-13.** Healing fracture of the rib neck in a 2-month-old male. Note the beginning formation of a fracture callus. The healing precludes interpreting the direction of forces associated with the fracture, but the location is consistent with posterior levering of the rib over the transverse process of the vertebra. The cause and manner of death were classified as undetermined.


**Posterolateral Rib Fractures**

Posterolateral rib fractures are less common than posterior rib fractures (Figs. 3.16–3.26) [16]. Posterolateral fractures are typically observed adjacent to ribs with neck fractures [16]. Kleinman et al. [7] studied 31 infants who died with nonaccidental skeletal injuries. Of the 84 total rib fractures observed, eight (5%) were identified as posterolateral.

Fractures located at the most lateral point of the rib body (the posterolateral and anterolateral interface) are postulated to be the result of anteroposterior compression that causes the bone to fail in tension [17]. Additionally, the body of the rib (the region that includes the posterolateral, anterolateral, and anterior regions) is vulnerable to direct impacts [2, 19].
Figure 3-18. Healing fractures of left ribs 1–12 from the individual pictured in Fig. 3.17. The rectangle marks the posterolateral/anterolateral rib fractures marked by arrows in Fig. 3.17. Numerous posterior and anterior healing rib fractures are present as well.

Figure 3-17. Posterolateral/anterolateral rib fractures. Multiple healing posterolateral/anterolateral rib fractures (arrows) are shown in situ in an 8-week-old male; also note the anterior rib fractures (arrowheads). The cause of death was classified as blunt trauma of the head with skull fractures and brain contusions, the manner as homicide.

Figure 3-16. Posterior rib fractures in a 21-month-old female. (A–C) Note the splaying apart of the bone, especially in panel (B). The fracture types are consistent with a posterior-to-anterior-directed impact. The cause of death was classified as blunt trauma of the head and torso with bilateral rib and scapular fractures, visceral lacerations, and multiple contusions, the manner as homicide.
Figure 3-19. Healing posterolateral incomplete fracture of the right ninth rib of a 3-year-old female. The fracture was interpreted to be the result of an anterior-to-posterior compression of the chest with posterior levering. The cause of death was classified as multiple blunt trauma of the head, torso, and extremities, with fractures; lacerations of the heart, liver, and mesentery; and subdural hemorrhage. The manner was classified as homicide.

Figure 3-20. Close-up of the fracture pictured in Fig. 3.19. Note the rounded margins and the subperiosteal new bone formation (SPNBF) around the fracture along the pleural surface of the rib (see Chap. 6 for a description of bone healing).

Figure 3-21. Rib fractures in situ. A healing posterolateral rib fracture (arrow) is shown in situ in an 11-month-old male. The arrowheads indicate additional posterolateral-to-anterolateral fractures. The cause and manner of death were classified as undetermined.

Figure 3-22. Healing complete posterolateral fracture of right rib 6 after processing. The fracture is shown in situ in Fig. 3.21 (arrow). Note the formation of the bone callus (see Chap. 6 for a description of bone healing).
Figure 3-23. Healing posterolateral rib fracture in left rib 9, an additional rib from the individual pictured in Fig. 3.21. Note that the fracture callus has fully stabilized the fracture (see Chap. 6 for a description of bone healing).

Figure 3-24. Healing incomplete transverse fracture of the right 12th rib of a 5-year-old female. Note that the rib bows inward at the fracture site (arrow). Although the fracture is located in the anterior region of the rib body, it is classified as a posterior rib fracture because of its position within the torso. The fracture was the only skeletal injury observed in this case and was interpreted as a result of a direct blow to the right back in a posterior-to-anterior direction. The cause of death was classified as blunt force abdominal trauma with jejunal perforation and peritonitis, the manner as homicide.

Figure 3-25. Posterolateral rib fractures observed in situ after the pleura was stripped in a 2-year-old male. The fractures are marked by the orange clips. The cause and manner of death were classified as undetermined.

Figure 3-26. Postprocessing view of posterior fractures in the ninth rib from the child pictured in Fig. 3.25. The arrows are pointing to two posterolateral fractures. Note the SPNBF at both fracture sites. The more anterior fracture is subtle and observed primarily as a concentrated thickening of the SPNBF. The more posterior fracture was interpreted as a result of posterior levering of the rib over the transverse process of the vertebra. The subtlety and healing of the more anterior fracture preclude interpretation of the associated force.
Anterolateral Rib Fractures

Anterolateral and anterior rib fractures are considered the least indicative of nonaccidental trauma, primarily because these fractures have been associated with CPR [6, 15, 18] (see section "Cardiopulmonary Resuscitation Fractures"). The morphologic features of an acute fracture in this region are the best indicator of the mechanism of force – that is, anteroposterior compression or a direct blow (Figs. 3.27–3.33) [16]. However, healing may obscure the features of compression and tension failure, precluding determining the mechanism of force.

**Figure 3-27.** Posterolateral/anterolateral rib fracture. Shown is an incomplete fracture at the posterolateral/anterolateral interface (arrow) observed in a 13-month-old female. The fracture involved only the external surface of the rib and was interpreted as a failure in tension due to anteroposterior compression of the chest. The cause of death was classified as blunt force injuries of the head, the manner as homicide.

**Figure 3-28.** Close-up of the fracture pictured in Fig. 3.27. Note that the fracture is coated with SPNBF, indicating a relatively short interval between injury and death (see Chap. 6 for a description of bone healing and fracture healing rates).

**Figure 3-29.** Incomplete fracture of the anterolateral region of the left seventh rib of the individual pictured in Fig. 3.27. Note the slight deformation of the bone at the fracture site. The fracture type is consistent with a failure in tension along the pleural surface of the rib.

**Figure 3-30.** Close-up of the fracture pictured in Fig. 3.29. The fracture is a failure in tension along the pleural surface of the rib and is interpreted as a result of a direct force applied to the bone at the fracture site.
Anterior Rib Fractures

The anterior region of the rib is vulnerable to direct blows as well as anteroposterior compression (Figs. 3.34–3.44). When one considers the anterior region, the fractures of the sternal face are more informative as to the mechanism of trauma. Kleinman et al. [7] found that fractures of the costochondral junction (CCJ) are analogous to Salter–Harris type II physeal injury (Figs. 3.38–3.44). The fractures involve the chondro-osseous junction, with the greatest disruption typically occurring along the internal margin of the face (pleural surface). These fractures may occur without significant involvement of the periosteum, thus resulting in minimal hemorrhage. Furthermore, during the healing process, the fractured sternal face reforms in the absence of a callus formation.

Weber et al. [15] reviewed 546 autopsies of sudden unexpected death in infancy and found rib fractures in 24 cases. Four CCJ fractures were found; all were associated with healing rib fractures, and none was associated with CPR. Smeets et al. [10] reported a single case of a 9-month-old female who presented to the first aid department of a hospital with a history of a fall 2 days prior. During a radiologic examination, posterior rib fractures, cranial fractures, and classic metaphyseal lesions were identified. A sonogram of the chest and abdomen revealed costochondral distraction of the lower left ribs in association with subcutaneous hema- toma. Based on these findings, a diagnosis of battered child syndrome was made. Ng and Hall [20] presented three case studies (7-, 18-, and 36-month-old children) with a total of ten CCJ fractures. The fractures involved ribs 6–9, were bilateral in two cases, and were not associated with CPR. Conversely, Kleinman [16] postulated that CCJ fractures are a result of depression of the sternum and/or cartilages during CPR.
Figure 3-34. Healing anterior rib fractures observed in a 2-month-old female. The healing precludes interpretation of the direction of force. The cause of death was classified as blunt force head trauma, the manner as homicide.

Figure 3-35. Anterior rib fractures. Shown are acute bilateral incomplete fractures (arrows) of the anterior region of the right and left first ribs (view of the inferior surface) observed in the case described in Fig. 3.33.

Figure 3-36. Close-up of the fractures pictured in Fig. 3.35. Note that the bone is minimally traumatized (arrows). The injury was not recognized until the periosteum was stripped from the internal surface of the ribs (see Chap. 1 for a description of the skeletal examination).

Figure 3-37. Sternal surface of the ribs pictured in Fig. 3.35. Note that the fracture of the left rib extends onto the sternal surface. The sternal surface of the right rib is normal.
Figure 3-38. Normal sternal surface of a rib of a 2-month-old male. Note the even texture of the bone and the well-defined rim. Lipping of the rim is normal in the midrange ribs of infants and should not be mistaken as a rachitic rosary. The cause and manner of death in this case were classified as undetermined.

Figure 3-39. Acute partial fracture of the sternal surface of the left fourth rib of a 3-year-old female. The bone is oriented so that the pleural surface is down. Note that the trabecular bone is exposed. The cause of death was classified as multiple blunt trauma of the head, torso, and extremities, with fractures; lacerations of the heart, liver, and mesentery; and subdural hemorrhage. The manner was classified as homicide.

Figure 3-40. Acute partial fracture of the sternal surface of the right sixth rib of a 2-month-old female. The pleural surface is toward the top of the photograph. The lipped rim of the non-fractured area is normal. The cause of death was MRSA chest wall abscess with disseminated infection with the contributory factor of chronic stress associated with multiple blunt trauma injuries. The manner was classified as homicide.

Figure 3-41. Pleural surface of the rib pictured in Fig. 3.40. The fracture involves only the rim along the pleural surface. The anterior (external) region of the rib is atraumatic.
Cardiopulmonary Resuscitation Fractures

As stated previously, CPR fractures are rare in children. When they do occur, they typically are located in the midclavicular (anterolateral) region of the rib, are multiple and bilateral, are present on the midrange ribs (third through sixth), and have no to very little hemorrhage (Figs. 3.45–3.50) [15, 18]. Additionally, CPR fractures are not associated with posterior rib fractures. The direct anterior-to-posterior force in the absence of posterior levering, due to the supported back, protects the posterior and posterolateral regions of the rib.

In 2005, the American Heart Association released new guidelines for CPR and emergency cardiovascular care of pediatric and neonatal patients. The new guidelines require the administrator of CPR to encircle the infant’s chest with both hands and compress it in an anteroposterior direction. The thumbs are placed together on the lower half of the sternum, and the fingers are wrapped around the thorax. The two-thumb technique is recommended for infants (birth to 12 months) and is to be administered by trained medical personnel [21]. Reportedly, the two-thumb method generates increased pressure compared with the one-hand anterior compression method [21]. The two-thumb method is very similar to the position Kleinman [16] documented as described by assailants in child abuse cases. To date, no report of rib fractures associated with the two-thumb method has been published.
Rib Fractures

Figure 3-45. CPR fractures in a 2-month-old female. Shown in situ are CPR fractures (arrows) of left ribs 2–4. Note the lack of hemorrhage. The cause of death was classified as acute bacterial bronchopneumonia associated with bronchopulmonary dysplasia due to prematurity, the manner as natural.

Figure 3-46. Postprocessing view of the CPR fractures shown in Fig. 3.45. The incomplete buckle fractures (arrows) are located in the anterolateral-to-anterior region of the rib and are interpreted as resulting from an anterior-to-posterior compression of the mid-chest. Note the hyperporosity of the anterior region of the ribs. The decrease in density of the cortical bone is consistent with the decedent’s history of prematurity (born at 27 weeks’ gestation).

Figure 3-47. CPR fractures in a 5-month-old male. Shown in situ are CPR fractures (arrows) of right ribs 3–6 after the pleura was stripped. Note the absence of hemorrhage in the soft tissue surrounding the fractures. The cause and manner of death were classified as undetermined.

Figure 3-48. CPR fracture of the left fifth rib after processing (pictured in situ in Fig. 3.47). The buckle fracture is located on the anterior region of the rib and is interpreted as a result of anterior-to-posterior compression. A well-healed posterolateral fracture also was observed in the decedent. The decedent had an extensive medical history including extreme prematurity (delivered at 23 weeks’ gestation).
First Rib Fractures

Fractures of the first rib have received special attention in the literature (Fig. 3.51) [16, 19, 22, 23]. The first rib is uniquely located within the chest. It is positioned under the shoulder girdle and therefore protected from direct blows. The rib is directly united to the sternum by a synchondrosis and has reduced movement at the costo-vertebral articulation in comparison with the other ribs. Strouse and Owings [22] stated that the first rib typically fractures through the subclavian groove, a weak point of the rib. They and others postulated that the bone fails because of muscle contractions associated with violent shaking [19, 22, 23]. Vikramaditya [23] reported two cases of isolated first rib fractures identified in adolescents after vigorous activity. The author interpreted the fractures as a result of stress generated from opposing forces applied by the scalene and serratus muscles.
Rib fractures are considered highly suspicious for nonaccidental injury. The location of a rib fracture is informative as to the mechanism of trauma. The rib is conventionally divided into four regions: posterior, posterolateral, anterolateral, and anterior. Rib fractures in the posterior region are postulated to result from anterior-to-posterior constriction with posterior levering of the chest. Posterolateral, anterolateral, and anterior regions of the rib may fracture as a result of anterior-to-posterior constriction of the chest but also are vulnerable to direct impact. Fractures of the rib CCJ also have been reported in cases of nonaccidental injury. Typically, CCJ fractures involve the internal margin of the sternal end plate. CPR-related rib fractures normally are incomplete fractures of the pleural surface of the rib at the interface of the anterior and anterolateral regions. Fractures of the first rib tend to be located within the subclavian groove and to be the result of opposing muscle constriction rather than direct impact.

Summary

Rib fractures are considered highly suspicious for nonaccidental injury. The location of a rib fracture is informative as to the mechanism of trauma. The rib is conventionally divided into four regions: posterior, posterolateral, anterolateral, and anterior. Rib fractures in the posterior region are postulated to result from anterior-to-posterior constriction with posterior levering of the chest. Posterolateral, anterolateral, and anterior regions of the rib may fracture as a result of anterior-to-posterior constriction of the chest but also are vulnerable to direct impact. Fractures of the rib CCJ also have been reported in cases of nonaccidental injury. Typically, CCJ fractures involve the internal margin of the sternal end plate. CPR-related rib fractures normally are incomplete fractures of the pleural surface of the rib at the interface of the anterior and anterolateral regions. Fractures of the first rib tend to be located within the subclavian groove and to be the result of opposing muscle constriction rather than direct impact.

References


Vertebral, sternal, and scapular fractures are rare in children and are described in the literature as highly specific for child abuse injury [1–7]. However, current research suggests that sternal fracture may actually occur more often as a result of low-force accidental impact [3, 5]. Clavicular fractures are a very common accidental injury in children but may become more suspicious for nonaccidental injury when they occur on the lateral or medial thirds of the shaft or when there are concurrent fractures of the scapula or vertebrae [8–10]. This chapter takes a holistic approach to the torso by describing fractures of the neck, the thoracic skeleton, the pectoral girdles, and the thoracolumbar and sacral regions of the spine. Although the ribs are components of the thoracic skeleton, the common and complex nature of pediatric rib fractures requires a full chapter of detailed description; therefore, these fractures are not discussed here (see Chap. 3).

The human vertebral column is a segmented bony structure typically comprising 33 vertebrae cushioned by intervertebral fibrocartilaginous disks and held in place by strong elastic ligaments. The vertebral column extends from the base of the skull to the inferior pelvic aperture, enclosing and protecting the spinal cord, distributing body weight from the head to the lower limbs, and providing stability and flexibility of motion to the torso. The vertebrae are classified by specialized function into five sections: the cervical column (C1–C7), thoracic column (T1–T12), lumbar column (L1–L5), sacrum (S1–S5, usually fused), and coccyx (4 vestigial, usually fused).

The cervical column protects the most delicate and vital portion of the spinal cord, provides a bony fulcrum for head movement, and is the attachment site for the neck muscles. The thoracic column is a stabilizing and flexible component of the thoracic skeleton. The robust lumbar column functions as a strong base for transfer of weight to the pelvic girdle and lower limbs and encloses the termination of the spinal cord. At birth the spinal cord terminates at the second or third lumbar vertebra, but by adulthood the termination lies within the vertebral foramen of either the first or second lumbar vertebra. The sacrum articulates superiorly with the fifth lumbar vertebra and laterally with the right and left innominates. The sacrum functions in the transfer of weight from the lumbar region to the lower limbs and encloses the sacral and coccygeal nerve roots. The coccyx is vestigial and small and is variable in presence and number of fused vertebrae. Fractures of the coccyx are not discussed in this chapter.

The thoracic skeleton consists of the thoracic vertebral column, the ribs, the sternum, and the costal cartilage that articulates the sternum to the ribs (Figs. 4.1–4.3). The adult sternum is composed of three segments: the manubrium, the corpus sterni, and the xiphoid process. The manubrium is located at the level of the third and fourth thoracic vertebrae and the corpus sterni at the fifth through ninth thoracic vertebrae. The xiphoid process articulates with the inferior corpus sterni and varies in length and ossification. (The characteristics of the immature sternum are somewhat different and are discussed later in reference to pediatric sternal fractures.) The thoracic skeleton encloses and protects the heart and lungs and serves as a large framework for muscle attachments that enable respiration. The flexibility of the bone and cartilage construction of this region allows for expansion and contraction of the thoracic cage during respiratory movement.

The right and left pectoral girdles, each comprising one clavicle and scapula, articulate with the manubrium of the sternum through the medial clavicle. The clavicle stabilizes the...
The arm to the thoracic skeleton and holds the glenohumeral joint in place through ligamentous attachment of the lateral clavicle to the acromion and coracoid process of the scapula. The scapula, which articulates with the humeral head, slides over the posterior surface of the thorax protected by the supraspinatus, infraspinatus, and subscapularis muscles. The anatomy of the pectoral girdle provides the mobility required for directional movement of the arm.

**Figure 4-1.** Autopsy view of the articulated clavicles, sternum, and ribs of a 3-year-old female. To provide access to the internal organs, the costal cartilages will be cut to remove a roughly triangular chest plate containing the sternum.

**Figure 4-2.** Autopsy view of the articulated thoracolumbar region of a 2-year-old male. The chest plate and internal organs have been removed as part of the examination procedure.

**Figure 4-3.** Autopsy view of the articulated right scapula of a 3-year-old male. The supraspinatus and infraspinatus muscles that form a protective cushion for the relatively delicate scapula (arrow) are not reflected from the bone.
Vertebral Fractures

Spinal fractures account for only a small percentage of nonaccidental pediatric fractures (estimated at 1–3%), possibly as a result of the segmented and ligamentous construction of the vertebral column, which is particularly flexible in the young child [1, 7, 11–14]. The mechanisms associated with spinal fractures are distraction, hyperflexion, and/or hyperextension of the vertebral column with associated axial loading and rotation [1, 15].

The vertebral body is the most common site for vertebral fractures [14]. When a portion of the posterior column, such as the posterior spinous process, is fractured, it typically is accompanied by a vertebral body fracture. Vertebral body fractures have been categorized into three patterns: (1) mild compression deformity with intact end plates; (2) fracture of the anterosuperior end plate with no loss of vertebral body height; and (3) a combination of compression deformity with anterosuperior end plate fracture [1, 16]. The fracture of the anterosuperior end plate usually results in a fragment of bone [1, 16].

Kemp et al. [7] conducted an in-depth literature review of published studies and case reports of spinal trauma identified in child abuse victims less than 18 years old. They found reports of 25 child abuse victims with nonaccidental spinal injuries: 12 individuals had cervical injuries (median age, 5 months), 12 individuals had thoracolumbar injuries (median age, 13.5 months), and one child had cervical, thoracolumbar, and sacrum injuries (no age given). The type of musculoskeletal injuries observed in their review included C2–C3 fractures, anterior vertebral dislocation, compression fracture of the vertebral body, and bilateral pedicle fractures. The mechanism of injury was confirmed in eight cases. Four children were shaken, two children were thrown, one child was forcefully spanked, and one child with a sacral injury may have been forcibly seated [7].

Neck Fractures

Infants are particularly vulnerable to cervical spine injuries because of the size and weight of the head relative to the body (Figs. 4.4–4.6). At this developmental stage, the infant neck flexes at the C2–C3 segment rather than at the C5–C6 segment, as is the case in older children and adults; the vertebral facet joints are underdeveloped and horizontally oriented; and the neck musculature is immature in tone [7]. When suddenly subjected to forces of deceleration, the neck may fail in support of the heavy head, resulting in hyperflexion or hyperextension [7]. Bilateral avulsion of the pedicles or their synchondroses from the C2 vertebral body (spondylolysis) and spondylolysis with spondylolisthesis (anterior dislocation of a vertebra) are recognized as an injury secondary to hyperextension. This type of neck fracture in infants and children most frequently is the result of motor vehicle crash or fall injuries. However, several case reports describing nonaccidental injury have documented bilateral spondylolysis of the C2 vertebral body with associated spondylolisthesis [1, 15, 17, 18].

Figure 4-4. Superior view of a fractured C1 vertebra. A nondisplaced transverse fracture of the posterior synchondrosis is present in the C1 vertebra of a 3-year-old female. The synchondrosis was partially fused at injury. The anterior synchondrosis is open and atraumatic. The cause of death was classified as blunt trauma of the head, neck, torso, and extremities with cervical vertebral fractures and cervicomedullary spinal cord injury, the manner as homicide.

Figure 4-5. Magnified view of a C1 fracture. A magnified view of the C1 fracture pictured in Fig. 4.4 shows details of the fracture margins where the posterior synchondrosis was in partial fusion at injury.
Fractures of the Thoracolumbar and Sacral Regions

Nonaccidental fractures of the thoracolumbar region are typically compression fractures of the vertebral bodies resulting from relatively low-energy forces (Fig. 4.7) [14]. However, the more severe thoracolumbar fracture with listhesis (dislocation) has been described in several reports of child abuse injuries [12, 14, 19]. Levin et al. [19] reviewed plain films, CT scans, and MRI scans of seven thoracolumbar fractures with listhesis in child abuse victims aged 6 months to 7 years. The authors noted that the most common location of the fracture was at the L1–L2 segment. Bode and Newton [12] and Sieradzki and Sarwark [14] each reported a single case of child abuse with thoracolumbar fracture and listhesis. The level of the fracture was the T12/L1 segment in both cases. Eleven of the 12 thoracolumbar injuries reviewed by Kemp et al. [7] were located at the T11–T12 segment, and included compression of the vertebral body with associated listhesis.

Sacral fractures are rarely diagnosed in children, comprising an estimated 0.16% of pediatric trauma cases [20, 21]. However, fractures of the sacrum may be underreported because most sacral fractures are not associated with neurologic symptoms, may not be acutely painful, and may be difficult to assess on radiography if no CT is performed [11, 20, 22, 23]. Hart et al. [20] conducted a retrospective review of 4,876 cases of pediatric injury presenting at a children’s hospital over a 7-year period. Only eight children had documented sacral fractures, and each patient’s injuries were reported as sustained in motor vehicle crashes or falls from height. In a review of 89 case files of patients aged 3–16 years with documented thoracolumbar and/or sacral injuries sustained in motor vehicle crashes (66%), sports activities (21%), and falls (13%), Dogan et al. [21] found that thoracolumbar spine injuries were more frequent in children over 9 years of age. The L2–L5 region was the most common injury site (29.8% of cases), and the sacrum was the least common injury site (5% of cases). In this study, all the sacral injuries were fractures of the ala extending from the lateral margin to the lateral border of the neural foramen (zone 1 fracture). Zone 1 sacral fractures are typically the result of lateral compression of the pelvis. All the patients with sacral fractures were intact neurologically [21].
Fractures of the Vertebral Column, Sternum, Scapulae, and Clavicles

**Fractures of the Sternum**

The sternum is composed of at least four centers of ossification (sternebrae) at birth (Fig. 4.8). The manubrium cannot be identified in isolation until the child is approximately 1 year of age [24]. All the sternebrae are usually formed by 6 years of age and are fused into a single bone, the corpus sterni, by late adolescence [24]. The flexibility associated with the late development of the sternum has been cited as one of the reasons for the rarity of documented pediatric sternal fractures and the likelihood that sternal fractures are associated with significant blunt force injury to the thorax [1, 25]. Kleinman [1] stated that in the absence of a significant traumatic event, sternal fractures should be considered suspicious for inflicted injury. However, several researchers have found sternal fractures resulting from relatively minor trauma.

Hechter et al. [3] reported that pediatric sternal fractures may be underdiagnosed, and therefore underreported, because of difficulty in obtaining a clear image of the immature sternum in standard radiographic view. They stated that each sternebra must receive a dedicated radiographic view to eliminate or diagnose fractures of the sternebrae. The authors conducted a retrospective review of radiographic and clinical charts of all documented sternal fractures observed at a large pediatric hospital over an 11-year period. They found 12 documented fractures: four in children under 3 years and 8 in children over 3 years of age. Ten of the 12 fractures were the result of accidental injury. The other two fractures, one in a 1-year-old and one in a 2-year-old, were suspicious for nonaccidental injury, but the circumstances of injury were not determined. The most common location of sternal fractures in the 12 cases was the proximal region of the corpus sterni, followed by the manubrium and the lower region of the corpus sterni. The most common mechanism of injury was a direct blow to the sternum. In nine cases, there were no associated fractures of the thorax or other regions of the body. The authors concluded that although fractures of the sternum are uncommon in children, they are not highly specific for nonaccidental injury.

Ferguson et al. [5] conducted a retrospective study of sternal fractures treated at a children’s hospital over a 40-month period. They reviewed the hospital records of children who received a plain radiograph or CT scan of the thorax after trauma. Twelve cases with sternal fractures were identified: the age range was 5 years to 12 years. None of the sternal fractures was sustained in a motor vehicle crash, and all were isolated injuries without associated fractures. Eleven of the 12 fractures were nondisplaced fractures of the anterior cortical bone of the first and second sternebrae. Seven fractures resulted from direct blows to the sternum, and five resulted from hyperflexion of the torso. One direct blow resulted in a fracture at the manubriosternal joint and a posterior displacement of the corpus sterni. All 12 injuries were accidental and occurred during common activity, such as a fall from a trampoline or bouncy structure (four cases), a fall from a bicycle (four cases), a fall from a 5- to 6-ft height (two cases), and a fall from a standing position (two cases).

In a published case study, DeFriend and Franklin [26] described two cases in which children sustained similar fractures of the proximal region of the corpus sterni during a fall from a swing. One child, an 8-year-old, fell directly onto her chest. The other child, a 7-year-old, fell onto her back, and her legs went up over her shoulders, causing hyperflexion of her torso.

There appears to be consensus in the current literature that sternal fractures are rare in children but are not specific for child abuse in the absence of other suspicious injuries. Pediatric sternal fractures occur most frequently in the proximal region of the corpus sterni, are rarely displaced, and are often the result of accidental injury at relatively low levels of force.
Fractures of the Scapula

Scapular fractures in adults are relatively uncommon and are typically caused by a direct impact of significant force [27]. Several mechanisms may result in fracture of the adult scapula: (1) a direct downward impact to the shoulder, fracturing the acromion; (2) a direct impact to a flexed elbow, transmitting force to and fracturing the glenoid rim; (3) a direct impact to the lateral shoulder, causing a stellate glenoid fracture; (4) a direct blow to the superior aspect of the shoulder, fracturing the coracoid; and (5) forceful muscular contractions, resulting in avulsion fracture of the acromion or the coracoid [27]. Pediatric scapular fractures also are uncommon and are recognized as highly specific for nonaccidental injury. However, it has been postulated that scapular fractures in infants and young children are more likely the result of indirect forces than direct impact [1, 4, 25, 28].

Scapular fractures are considered highly specific for child abuse and are documented most often in children with typical child abuse fracture patterns involving the posterior region of the upper ribs and the pectoral girdle [1, 4, 25, 28]. In an early paper on child abuse fractures, Kogutt et al. [25] reviewed the case histories and radiographs of 100 infants and children diagnosed with “battered child syndrome” at a teaching hospital from 1967 to 1971. The age of the patients ranged from 6 weeks to 8 years. Skeletal fractures were visible on radiograph in 52 (55%) of the cases, with multiple fractures occurring in 22 (23%) of the cases. In the three scapular fractures documented, the authors observed two types of fracture: transverse blade fracture (Figs. 4.9–4.13) and acromial process fragmentation. Kogutt et al. [25] suggested that transverse blade fracture results from direct impact and acromion fragmentation is caused by severe twisting of the arm or shaking, similar to the mechanism associated with classic metaphyseal lesions of long bones.

The most common location of nonaccidental scapular fractures is the middle third of the acromion, although fractures of the coracoid and inferior glenoid margin also have been documented [1]. In concordance with the conclusions of Kogutt et al. [25], Kleinman [1] theorized that nonaccidental scapular fractures are likely a result of indirect forces generated by violent traction and torsion of the humerus. His theory is based on the anatomic structure of the shoulder and the concomitant pattern of fractures observed in abused children. The acromioclavicular and coracoclavicular ligament attachments to the clavicle create a stable unit with the predilection for the bone to fail along the two scapular processes.

Several researchers have reported avulsive fractures of the acromion resulting from muscle spasms in pediatric cases similar to those documented in adults. Kalideen and Satyapal [29] prospectively studied 171 neonates with tetanus over a 5-year period and found ten patients with bilateral symmetric avulsive fracture of the right and left acromions. Tetanus results in muscle hypertonia and spontaneous contraction of agonist/antagonist muscles. In the shoulder, the deltoid muscle abducts against the concomitant adduction forces, resulting in the avulsion of the acromion. Coote et al. [28] reported a case of bilateral acromial fractures in a patient with malignant infantile osteopetrosis. The fractures were noted on chest films taken a day after the patient had two witnessed seizures. The authors stated that the acute deltoid muscle contraction against the weak bone resulted in the avulsive acromial fractures at the deltoid muscle attachment sites. These findings support the theory that acromial fractures in children may occur through indirect avulsive forces.
Fractures of the Vertebral Column, Sternum, Scapulae, and Clavicles

Figure 4-9. Scapular blade fractures. Two healing incomplete transverse fractures, one on the medial border and one on the lateral border (arrows), are present in the left scapula of a 3-year-old female. The cause of death was classified as multiple blunt trauma of the head, torso, and extremities with fractures; lacerations of the heart, liver, and mesentery; and subdural hemorrhage. The manner was classified as homicide.

Figure 4-10. Magnified view of a lateral border scapular fracture. This view of the left scapula pictured in Fig. 4.9 shows callus formation at the margins of the lateral border transverse fracture. The direction of force cannot be determined because of healing, but fractures at this location typically occur as the result of a direct impact to the scapular blade.

Figure 4-11. Magnified view of a medial border scapular fracture. This view of the left scapula pictured in Figs. 4.9 and 4.10 shows rounding of the margins and subperiosteal new bone formation (SPNBF) around the transverse fracture at the medial border (arrow). The direction of force cannot be determined because of healing, but fractures at this location typically occur as the result of a direct impact to the scapular blade.

Figure 4-12. Posteromedial fractures of the right and left scapulae of a 21-month-old female. The infraspinous processes are buckled and the medial border of the right scapula is splayed apart. The fractures (arrows) are consistent with a posterior-to-anterior impact to the back. The cause of death was classified as blunt trauma of the torso and head with bilateral rib and scapular fractures, visceral lacerations, and multiple contusions; the manner was classified as homicide.
Fractures of the Clavicle

Although the clavicle is classified as a long bone, it does not contain a medullary cavity. The medial articular end is approximately triangular in shape, and the lateral articular end is relatively flat. The shaft and articular ends form a double curvature (S shape). The clavicle is located just under the skin at the root of the neck, leaving it relatively unprotected from impact. Because it is connected to the acromial and coracoid processes of the scapula by ligamentous attachment, the clavicle also may be injured by an impact to the glenohumeral joint. Falls, sports injuries, and motor vehicle crashes often result in clavicle fractures, which are reported as the most common pediatric fracture [9, 30].

The S shape of the clavicle is divided into thirds for mechanical classification of fractures by location [9, 31, 32]. Group I fractures are those of the middle one third, group II fractures are of the lateral third (Figs. 4.13–4.18), and group III fractures are of the medial third. The mid-clavicular shaft contains a weak spot, which is a product of the double-curvature shape, and this is the most frequent site of accidental clavicular fractures (group I fractures). Group I fractures typically result from direct impact to the lateral shoulder, as may occur in a fall.

Group II fractures and group III fractures are those of the lateral third and medial third, respectively. Neer [32] further categorized group II fractures into five types based on the relationship of the fracture to the coracoclavicular ligaments. Group II fractures in an infant or young child with no history of major accidental trauma are more suspicious for nonaccidental injury, because fracture of the lateral third of the clavicle typically is caused by a direct blow to the top of the shoulder [9, 10]. Impact to the top of the shoulder is unlikely to occur in a fall. Group III fractures are categorized into five types based on the severity of the fracture (e.g., degree of displacement, involvement of the articular surface, epiphyseal separation, and comminution). Group III fractures of the medial third are the least common clavicular injury [33]. Medial clavicular fractures are caused by direct impact to the anterior chest, which may occur through several accidental mechanisms [9, 30]. In the absence of an accidental cause, a medial clavicle fracture in an infant or young child, as is the case in sternal fracture, may increase suspicion of non-accidental injury.

Clavicle fractures also may result from birth trauma; typically these injuries are group I fractures of the mid-shaft [34]. Current research shows that fractures during birth may not be clearly associated with a traumatic birth event or be the result of congenital skeletal pathology. Beall and Ross [35] conducted a retrospective study of all deliveries at a large urban teaching hospital from January 1996 to March 1999 to observe the incidence of birth-related clavicle fracture and to quantify the association of clavicle fracture with shoulder dystocia. Shoulder dystocia is the inability of the infant to pass through the birth canal in a timely manner because the shoulders are wedged within the pelvic girdle. Shoulder dystocia traditionally has been associated with clavicle fractures [35]. The researchers found 26 clavicle fracture cases among the 4,297 births (5%). In these cases, clavicle fracture was significantly associated with increased maternal age and birthweight greater than 4,000 g, but not with documented shoulder dystocia events.

As further supporting evidence, Groenendaal and Hukkelhoven [36] concluded from an extensive retrospective study of neonate fractures in the Netherlands that clavicle fractures may be sustained during birth without a documented birth trauma event. The authors researched the incidence of unexplained clavicle fractures in full-term neonate admissions during the perinatal period to ten neonatal intensive care units and 60% of the level 2 hospitals in the Netherlands from 1997 to 2004. The study data were accessed from The Netherlands Perinatal Registry. Documented birth trauma and/or congenital bone disease cases were excluded. Of the 158,035 full-term births recorded, 1,174 had fractures and 227 of the fractures were unexplained by birth trauma. Clavicle fractures were noted in 212 of the 227 unexplained fracture cases; the additional 15 fractures were humeral (12) and femoral (3).
Fractures of the Vertebral Column, Sternum, Scapulae, and Clavicles

Figure 4-14. Osteopenic left clavicle of a 1-month-old female with a complete oblique fracture of the lateral third of the shaft. SPNBF is present on the acromial end and on the shaft adjacent to the fracture. The fracture likely is a result of hyper-rotation of the clavicle during birth. The cause of death was classified as sudden infant death syndrome, the manner as natural.

Figure 4-15. Magnified view of a clavicular fracture. This view of the oblique fracture described in Fig. 4.14 shows the fracture margins and SPNBF on the acromial end and shaft. Note the normal absence of a medullary cavity in the clavicular shaft.

Figure 4-16. Stereomicroscopic view of the inferior surface of the clavicular fracture described in Figs. 4.14 and 4.15. Note the rounded fracture margins and early soft callus formation (arrow).

Figure 4-17. Remodeled clavicular fracture. A remodeled fracture callus is present on the lateral third of the right clavicular shaft of a 12-month-old female (arrow). The cause of death was classified as multiple blunt force injuries and malnutrition, the manner as homicide.

Figure 4-18. Clavicle injury associated with humeral fracture. SPNBF is present on the lateral third of the left clavicle of a 3-year-old male (arrow). The injury is associated with a complete fracture of the left proximal humerus. The cause of death was classified as blunt trauma of the head, the manner as homicide.
Summary

Fractures of the neck, sternum, scapula, lateral or medial clavicle, and the thoracolumbar and sacral regions of the spine are uncommon in infants and young children. Without an explanatory history of accidental trauma, these fractures may raise suspicions of child abuse injury. Infants are at higher risk for C1–C2 fractures because of their immature neck structures, but the most common type and site of pediatric vertebral fracture are vertebral body compression fractures in the thoracolumbar region. Fractures of the immature sternum are rare but may not be as specific to child abuse injury, as previously thought. Scapular fracture is highly specific for nonaccidental injury and may be the result of abnormal forces generated during shaking or abnormal rotation or traction of the arm. Although fracture at the clavicle midshaft is a very common pediatric injury, fractures of the lateral or medial third of the clavicle are more suspicious for nonaccidental injury. Birth trauma is a possible mechanism for clavicle injury and is a consideration when healing clavicle fractures are observed during the infant postmortem skeletal examination.

References


Fractures occurring in the humerus, ulna, radius, femur, tibia, and fibula are collectively categorized as long bone fractures. Long bone fractures are further classified by type and location. Interpretation of the forces associated with the fracture depends on the location, type, and extent of the fracture. Common long bone fracture types include spiral, oblique, transverse, torus (or buckle), greenstick, and classic metaphyseal lesion (CML) [1]. A spiral fracture circles the shaft and typically results from a rotational force. An oblique fracture crosses the bone diagonal to the long axis, and a transverse fracture crosses the bone at a right angle to the long axis. A torus (or buckle) fracture, in which the cortical bone balloons out, is typically the result of a compression force loading through the long axis of the bone. A greenstick fracture is an incomplete fracture, most often an incomplete transverse fracture, and is commonly found in children as a result of the increased elasticity of the developing bone. CML is a fracture of the physeal plate of a long bone.

In terms of location, a developing long bone is conventionally divided into four regions: the shaft (or diaphysis), metaphysis, physis, and epiphysis. The shaft, the tubular midsection of the bone, is subdivided into three regions: the mid, distal, and proximal one third. The metaphyses are the flared regions of the proximal and distal ends of the long bone. At the transition between the shaft and the metaphysis, the cortical bone thins and the density of the trabecular bone increases. The physis is the proximal or distal surface (end plate) of the long bone at the chondro-osseous interface. The epiphysis is the cartilaginous end of the bone containing the secondary ossification site.

**Classic Metaphyseal Lesions**

The CML is considered the long bone fracture with the greatest specificity for nonaccidental injury (Figs. 5.1 and 5.2) [2, 3]. A CML is a transmetaphyseal disruption of trabeculae of the primary spongiosa (the mineralized cartilage in the developing metaphysis) (Fig. 5.3). It occurs when indirect torsional or tractional forces are applied to an extremity during pulling and/or twisting of an infant’s arm or leg, or flailing of unsupported limbs during shaking. The delicate nature of the immature trabeculae, the loose attachment of the periosteum to the diaphysis, and the firm attachment of the perichondrium to the epiphysis all contribute to the bone failing at the specific location [2, 4–7]. The loose periosteum transmits most of the dynamic force associated with the trauma to the metaphysis, where the trabeculae of the primary spongiosa are weaker than the cartilage of the epiphysis [2, 4]. CMLs are most common during the first and second years of life, when the trabeculae of the primary spongiosa are elongating rapidly, making them vulnerable to torsional and tractional forces (Figs. 5.4 and 5.5) [2].

Historically, CMLs have been called bucket handle or corner fractures. The terminology is based on the radiologic appearance of the fractures. The series of microfractures through the primary spongiosa cause the physeal surface of the bone to separate from the metaphysis, creating a fragment of bone that remains adhered to the epiphysis.
Typically the fragment is thicker along the periphery and thinner in the middle (Figs. 5.6 and 5.7). Radiologically, the fracture appears as a line or a translucency through the metaphysis that dips deeper into the metaphysis at the edges of the bone, creating the bucket handle or corner fracture appearance [8–10].

A CML is considered highly suspicious for nonaccidental injury, primarily because the forces associated with the fracture are not generated by accidental falls [2, 4, 6]. Kleinman and Marks [9] performed a histologic and radiologic examination of 31 deceased victims of child abuse and found 64 CMLs. The authors reported the most common sites for a CML were the proximal and distal tibia and the distal femur. Worlock et al. [5] retrospectively compared the medical records of 35 children with nonaccidental skeletal injury with those of 826 children with accidental skeletal injury. The records documented 17 CMLs; all were observed in cases of nonaccidental injury in which the child was under the age of 18 months. The authors concluded that CMLs are uncommon, making up only 17% of the fractures recorded in the nonaccidental injury cases, but that they are specific for nonaccidental injury. Conversely, Caffey [11] noted that birth trauma, especially breech delivery, may result in a CML.

The reported infrequency of CMLs may reflect the difficulties associated with recognizing the fracture rather than their true occurrence. A CML is radiologically subtle; the fracture is rarely displaced, there is minimal hemorrhage associated with it, and little to no fracture callus forms during the healing process [2]. In fact, Lonergan et al. [12] reported that CMLs often are radiologically occult. CMLs also are difficult to recognize during a standard autopsy procedure. Figures 5.8 and 5.9 show a normal chondro-osseous interface and an acute CML after the periosteum was removed. Prior to the skeletal examination, the fracture shown in Fig. 5.9 was not recognized, radiologically or grossly (see Chap. 1). Kleinman et al. [8], recognizing the difficulty associated with identifying a CML radiologically, have advocated the removal of seemingly normal high-risk metaphyses for histopathologic examination in decedents with even low-level suspicion of nonaccidental injury.

As the CML heals, the exposed trabeculae become rounded and thickened. Eventually, the physeal surface reforms. (The healing process of CMLs is discussed in detail in Chap. 6.) Figure 5.10 shows a radiograph of a healing CML. Figure 5.11 shows the same fracture after processing. Figures 5.12–5.17 show CMLs at various stages of healing.

**Figure 5-1.** Normal physeal surface of a 21-month-old female, after processing. Notice the smooth, regular quality of the bone. The cause of death was classified as blunt trauma of the torso and head with bilateral rib and scapular fractures, visceral lacerations, and multiple contusions, the manner as homicide.

**Figure 5-2.** Complete CML of the proximal tibia of a 1-month-old male. Note the exposed trabeculae. The cause of death was classified as blunt trauma of the head with skull fractures and brain contusions, the manner as homicide.
**Figure 5-3.** Partial CML of the proximal tibia of a 1-month-old male. (A, B) Note the partial separation of the physis from the metaphysis through the primary spongiosa. (C) The physeal surface with the trauma along the posterior margin (the arrow is pointing to the tibial tuberosity of the anterior surface). The cause of death was classified as blunt trauma of the head with skull fractures and brain contusions, the manner as homicide.

**Figure 5-4.** Complete CMLs of the right and left distal radii of a 2-month-old male. The cause of death was classified as blunt trauma of the head with skull fractures and subdural hemorrhage, the manner as homicide.

**Figure 5-5.** Partial CML of the left distal tibia of a 2-month-old female. Note the fractures through the remaining physeal surface. The cause of death was classified as methicillin-resistant *Staphylococcus aureus* chest wall abscess with disseminated infection and a contributing factor of chronic stress associated with multiple blunt trauma injuries; the manner was classified as homicide.
**Figure 5-6.** Classic metaphyseal lesion. Notice that a small disk of bone remains attached to the epiphysis. The bone fragment is thicker at the periphery of the bone.

**Figure 5-7.** Complete CML. Shown is a partially processed proximal tibia of a 2-month-old female (same individual as pictured in Fig. 5.5). Note the thin disk of bone adhered to the epiphysis that becomes thicker at the periphery (arrow).

**Figure 5-8.** Normal metaphyses in situ. (A) The chondro-osseous junction (COJ; arrow) after the muscle has been reflected. (B) The COJ after the periosteum has been removed (arrow). Note the COJ is uniform and straight.
Figure 5-9. Complete CML. A CML of the right proximal fibula of a 1-month-old male (A) and a close-up (B) are shown in situ. Note the absence of hemorrhage in the soft tissue surrounding the injury. The cause and manner of death were classified as undetermined.

Figure 5-10. Radiograph showing healing CMLs of the distal ulna and radius of a 3-month-old female. Note the frayed appearance of the distal end of both bones (arrows). The cause and manner of death were classified as undetermined.
Figure 5-11. Processed ulnar CML shown in Fig. 5.10. Note the thickened trabeculae of the reforming physeal surface (A) and the bony spiculae extending from the bone collar (B).

Figure 5-12. Healing CML of the right proximal fibula of a 2-year-old male. The decedent survived in the hospital for 3 weeks after the date of the injury. Note the thickened trabeculae (arrow). The cause of death was classified as blunt force head trauma, the manner as homicide.

Figure 5-13. Healing CML of the proximal humerus of a 3-year-old female. The trabeculae are thickened and rounded. The cause of death was classified as multiple blunt trauma of the head, torso, and extremities with fractures; lacerations of the heart, liver, and mesentery; and subdural hemorrhage. The manner was classified as homicide.
Figure 5-14. Healing CML of the distal fibula of a 2-month-old male. The cause of death was classified as blunt trauma of the head with skull fractures and subdural hemorrhage, the manner as homicide.

Figure 5-15. Healing partial CML of the distal fibula of a 21-month-old female. The healing pattern suggests the fracture was incomplete. The cause of death was classified as blunt trauma of the torso and head with bilateral rib and scapular fractures, visceral lacerations, and multiple contusions; the manner was classified as homicide.

Figure 5-16. Healing CML with involvement of the metaphysis and epiphysis in a 12-month-old female. (A) Note that the fracture callus extends above the physeal surface. (B) Exposed trabecular bone of the fractured metaphysis. (C) Radiograph of the injury [arrow]. The cause of death was classified as multiple blunt force injuries and malnutrition, the manner as homicide.
Shaft, Metaphyseal, and Epiphyseal Fractures

Reconstructing the mechanism of force from the location and type of fracture has been a focus of much research. Researchers have described relationships between fractures of the shaft, metaphysis, and epiphysis and the type of forces applied to the bone [6, 7, 13]. Johnson [7] classified four types of long bone fractures and described the force associated with each. He stated that spiral fractures are caused by a twisting or torsional force (Figs. 5.18 and 5.19), oblique fractures by a levering or bending force (Fig. 5.20), transverse fractures by a direct impact (Figs. 5.21 and 5.22), and buckle or torus fractures by compression (Figs. 5.23–5.25). Pierce et al. [6] added that buckle or torus fractures are most common at the metaphysis–diaphysis interface. They stated that the predilection results from the structural transition from the dense cortical bone of the diaphysis to the dense trabecular bone of the metaphysis, creating an area more susceptible to failure in compression.

Epiphyseal separations, in contrast to CMLs, are primarily cartilaginous injuries, although they may extend into the bone (Figs. 5.26 and 5.27). Epiphyseal separation occurs more often with accidental injury, especially in older children. Salter and Harris [13] studied these fractures experimentally induced in rabbits and identified five types (Fig. 5.28). Three of the fracture types involve only the cartilage, one of which parallels the physeal surface (type I). Fracture types II and IV dip into the metaphysis in a manner similar to that of the CML. Kleinman [14] theorized that given the shared features between CMLs and epiphyseal separations, the forces associated with each are similar and the type of fracture depends on the magnitude of the force. However, the authors of this text hypothesize that the robustness of the trabeculae of the primary spongiosa is a contributing factor. CMLs are more common in infants and very young children, in whom the trabeculae of the rapidly elongating long bones are more vulnerable to traumatic forces [5]. In contrast, Salter–Harris fractures are more common in older children with robust trabeculae resulting from the slower growth rate. However, Jones et al. [15] presented two cases of nonaccidental physeal injury of the proximal femora in infants (9 and 3 months old). The authors described the mechanism of injury as a combination of traction and torque of the femur resulting from simultaneously pulling and externally rotating the extremity. They inferred that physeal separation occurs because the ligamentum teres is more resistant to stress than is the fibrous anchoring ring of the physis.
Figure 5-18. Spiral fracture of the right humerus of a 1-month-old male. The cause of death was classified as blunt trauma of the head with skull fractures and brain contusions, the manner as homicide.

Figure 5-19. Spiral fracture of tibial shaft observed in a 4-month-old male. Note the early formation of a fracture callus. The cause of death was classified as blunt force head trauma, the manner as homicide.

Figure 5-20. Vertical ulnar fracture. Shown is an incomplete vertical fracture of the ulnar midshaft observed in a 14-month-old male (A) and a close-up (B). The cause of death was classified as blunt force head trauma, the manner as homicide.
Figure 5-21. Transverse humeral fracture. (A–C) Transverse fracture with anterior/posterior compression located on the left proximal humeral shaft of a 3-year-old male. According to the child’s family, the decedent’s left arm was shut in a car door. Note the early formation of a fracture callus. The cause of death was classified as blunt trauma of the head, the manner as homicide.

Figure 5-22. Transverse ulnar fracture. Shown is an incomplete transverse fracture of the ulnar shaft of a 3-year-old female (A) and a close-up of the fracture (B; same individual as shown in Fig. 5.13). Note the early signs of healing, rounded fracture margins, and minimal subperiosteal new bone formation (SPNBF).

Figure 5-23. Torus ulnar fracture. Shown is a torus fracture of the proximal ulna (arrow) of a 14-month-old male. The cause of death was classified as blunt force head trauma, the manner as homicide.
Figure 5-24. Torus ulnar fracture. Shown are a torus fracture (arrow) of the distal ulnar shaft of a 16-month-old female (A) and a close-up of the fracture (B). Panel (B) shows the posterior surface of the bone. The cause of death was classified as blunt force head trauma, the manner as homicide.

Figure 5-25. Healing fracture of the left distal radial shaft of a 2-year-old female. The cause of death was classified as blunt trauma of the head with subdural hematoma and skull fracture, the manner as homicide.

Figure 5-26. Epiphyseal fracture. Shown is an epiphyseal fracture of a 12-month-old female, before processing (A) and after processing (B). The extension of the fracture into the metaphysis and the fracture callus (arrows) were not visible until the bone was processed. The cause of death was classified as multiple blunt force injuries and malnutrition, the manner as homicide.
Figure 5-27. Humeral condylar fracture. (A, B) Fracture of the lateral condyle of the right humerus of a 12-month-old female. Panel (B) shows the distal physeal surface. Note the rounded fracture margins and the thickened trabecular bone, indicating healing in the absence of a fracture callus and SPNBF. The fracture of the epiphysis was not noted prior to processing. The cause and manner of death were classified as undetermined.

Figure 5-28. Salter–Harris classification system. The five fracture types of the Salter–Harris classification system are shown. Type I is a cartilaginous fracture that parallels but typically does not involve the metaphysis. Type II involves a metaphyseal fragment. Type III involves only the cartilaginous epiphysis. Type IV involves both the epiphysis and metaphysis. Type V is a crushing injury involving the interface of the cartilage and bone.
Accidental Versus Nonaccidental Injury

The distinction between accidental and nonaccidental injury cannot be made on fracture type alone, because no long bone fracture type is pathognomonic for nonaccidental injury [16–19]. Differentiating accidental from nonaccidental long bone fractures depends on the age and developmental stage of the child and the history of the traumatic event as well as the type and location of the fracture [6, 12, 18–20]. Several researchers have emphasized the importance of considering the age of the patient when diagnosing nonaccidental injury [5–17, 21–26]. As mentioned previously, Worlock et al. [5] retrospectively compared nonaccidental to accidental long bone fractures and found that one in eight children under the age of 18 months who sustain a fracture is a victim of child abuse. Pandya et al. [24] compared skeletal injury patterns of 500 child abuse victims with 985 accidental injury cases (aged birth to 48 months) and found that the odds of a tibial/fibular fracture were 12.8 times, humeral fracture 2.3 times, and femoral fracture 1.8 times more likely the result of child abuse if the child was under 18 months of age. Conversely, in children 18 months of age and older, the occurrence of humeral and femoral fractures was 3.4 and 3.3 times more likely the result of an accidental injury. In a literature review, Pierce et al. [6] found that among infants, 40–80% of long bone fractures are from abuse. Coffey et al. [27] conducted a retrospective study of patients treated at a regional pediatric trauma center and found that 67% of lower-extremity fractures in patients less than 18 months of age were linked to child abuse whereas only 1% of lower-extremity fractures in patients 18 months of age and older were linked to abuse.

Researchers have studied the occurrence of nonaccidental fractures by long bone in an attempt to identify trends; however, there is no consensus. Kemp et al. [19] conducted a thorough literature review with the goal of distinguishing fractures resulting from accidental and nonaccidental injuries. The authors included studies that reported skeletal trauma in children less than 18 years of age and found that the probability of abuse given a humeral fracture was between 0.48 and 0.54 and that of a femoral fracture was between 0.28 and 0.43. Loder and Feinberg [25] conducted a retrospective review to examine the demographic and injury characteristics of children hospitalized with nonaccidental injury. Using the 2000 Healthcare Cost and Utilization Project Kids’ Inpatient Database of child abuse cases, the authors identified 1,794 cases with musculoskeletal trauma and a diagnostic coding of abuse. These cases were broken down into four groups based on age: infants (birth to 1 year), toddlers (1–2 years), children (3–12 years), and adolescents (13–20 years). Forty-nine percent of all the fractures occurred in the infant group. The most common location of a fracture was the skull, followed by the humerus and the femur. King et al. [28] reviewed the charts and radiographs of 189 battered children aged 1 month to 13 years and identified 429 fractures. The most common bone fractured was the humerus, followed by the femur and the tibia. Loder and Bookout [21] reviewed 75 cases of battered children with fractures (average age of the study sample was 16 months). The most common fracture occurred in the skull, followed by the tibia.

Additionally, researchers have searched for trends in fracture types among nonaccidental injuries. Worlock et al. [5] performed a retrospective comparison of fracture patterns observed in accidental and nonaccidental trauma in children under the age of 5 years and found spiral humeral fractures were significantly more common in the group with nonaccidental trauma (P<0.001). Beals and Tufts [29] conducted a retrospective review of medical charts and radiographs of children under the age of 4 years with femoral fractures treated at the University of Oregon Health Sciences Center over a 22-year period. The authors found that fractures of the subtrochanteric level and CMLs of the distal physis were more common in victims of nonaccidental injury. Additionally, Arkader et al. [30] examined the occurrence of complete metaphyseal fractures of the distal femur recorded by two level 1 trauma centers over a 10-year period. They found 29 fractures, 20 of which occurred in infants. Fifteen of the 20 fractures were confirmed to be the result of child abuse or highly suspicious for abuse. The five fractures resulting from accidental injury were the result of birth trauma, a fall from a swing, being dropped by a sibling, a fall from a bed, and a motor vehicle accident (Fig. 5.29). As mentioned earlier, Loder and Bookout [21] reviewed 75 cases of battered children with a total of 154 fractures and found the most common long bone fracture was transverse shaft fractures, followed by CMLs. Rewers et al. [31] conducted a review of the cases with documented femoral fractures entered into the Colorado Trauma Register over a 4-year period. They found that among children younger than 3 years, falls were the most common cause of femoral fractures, followed by nonaccidental injury. The nonaccidental femoral fractures tended to be more distal and to have a combined shaft–distal metaphysis pattern.

As noted earlier, humeral and tibial fractures are common in nonaccidental injury; however, these fractures are common in accidental injury as well. Of humeral fractures, shaft and supracondylar (Fig. 5.30) fractures are reported frequently in cases of accidental injury in infants and children [19]. Humeral shaft fractures have been documented as birth trauma, most often associated with macrosomic and breech presentation [32]. Hymel and Jenny [33] reported two cases of accidental spiral humeral shaft fractures occurring in infants when they were rolled onto their outstretched arm by a caregiver. Furthermore, Shaw et al. [34] conducted a retrospective study during which four physicians independently reviewed the medical charts of patients younger than 3 years who were treated...
for humeral shaft fractures (N=34). Based on the charts, the physicians ranked each case as probable not abuse, probable abuse, or indeterminate. Of the 34 cases, 62% were unanimously ranked or ranked by three of the four physicians as probable not abuse, and 20% were ranked as probable abuse by one or more of the physicians. The remainder of the cases (18%) were ranked as indeterminate by two or more of the physicians. Kemp et al. [19] reviewed 32 published studies of child abuse victims younger than 18 years and found that supracondylar fractures were reported to be more likely associated with accidental injury, specifically falls. Worlock et al. [5] found that in the 35 infants and toddlers with nonaccidental injuries (150 total fractures), only one fracture was identified as supracondylar. Of the 116 infants and toddlers with accidental injuries (135 fractures), 13 fractures were identified as supracondylar. Despite reporting a lower occurrence of supracondylar fractures in nonaccidental cases than accidental cases (3:70), Strait et al. [22] cautioned against dismissing supracondylar fractures as accidental based on fracture type alone.

Accidental tibial fractures require special attention. Childhood accidental spiral tibial (CAST) fractures typically are isolated spiral fractures located on the mid and distal regions of the tibial shaft that occur with relatively low levels of force. Often the spiral fracture is oriented superolaterally to inferomedially. CAST fractures are most common in children aged 2–6 years. The mechanism is a torque or rotational force applied to the lower extremity associated with a fall or an immobilized foot [35]. CAST fractures are easily recognized on radiographs. Toddler fractures, which are considered a subset of CAST fractures, are nondisplaced oblique or spiral fractures of the distal tibia that are difficult to recognize on radiographs [8]. Mellick and Ressor [35] conducted a 14-month prospective study during which ten CAST fractures were recognized, as well as a retrospective study of 5 years of social work service records of child abuse cases. Of the ten fractures examined during the prospective study, none were a result of accidental injury and were found in children ranging in age from 21 to 44 months. Only one fracture resulted from nonaccidental injury; the infant was 9 months old (nonambulatory). During the retrospective study of the child abuse cases, 33 cases were found with skeletal injury, only three of which were CAST fractures. Of the three CAST fractures, one was in a 2-month-old who on subsequent hospital visits had rib fractures. The other two cases involved a 19-month-old and a 17-month-old, and the suspicion of child abuse and neglect was based on an inability to exclude child abuse and a previous allegation of leaving the child unattended. Of interest, Schwend et al. [26] found that spiral femoral shaft fractures were common in ambulatory children and described them as analogous to toddler fractures. They stated that femoral shaft fractures may occur at relatively low-energy levels, including low-level simple falls, and are nonspecific for nonaccidental injury.

![](figure5-29.png)

**Figure 5-29.** Healing torus fracture. Shown is a healing torus fracture (arrow) of the distal femoral metaphysis observed in a 1-month-old female. Note the frayed appearance of the periphery of the physeal surface; the physeal surface is intact. The cause of death was classified as hypoxic–ischemic encephalopathy due to multiple blunt force injuries, including skull fractures and subarachnoid hemorrhage; the manner was classified as homicide.

![](figure5-30.png)

**Figure 5-30.** Supracondylar humeral fracture observed in a 3-year-old female. Note the SPNBF along the shaft proximal to the fracture. The cause of death was multiple blunt trauma of the head, torso, and extremities with fractures; lacerations of the heart, liver, and mesentery; and subdural hemorrhage. The manner was classified as homicide.
Summary

Long bone fractures are highly variable in type and location. The mechanism of the injury often can be reconstructed from the features of the fracture. The consensus among researchers is that no long bone fracture is pathognomonic for nonaccidental injury. The age of the child, inconsistencies between history and clinical presentation, delay in treatment, and injuries of various ages often are more telling than the fracture itself.

References

Healing and Interpretation

The ultimate goal of skeletal injury analysis in cases of suspected child abuse is to reconstruct the child’s life history with regard to the number, extent, and timing of the traumatic episodes. Recognizing and interpreting fracture distribution patterns throughout the body enable the anthropologist to move from the myopic assessment of the isolated fracture to the comprehensive evaluation of the inflicted injury that may have multiregional involvement. In turn, assessment of the stage(s) of healing observed throughout the skeleton often leads to an estimate of the minimum number of traumatic episodes and the age(s) of the injury. However, variation in healing rates associated with the type of bone injured and the extent of the initial injury must be recognized.

Healing

Conventionally, bone healing is broken into three phases: inflammation, reparation, and remodeling. The inflammation phase begins immediately following the trauma as a result of bleeding from the damaged periosteum, bone, and surrounding soft tissues. The hemorrhage leads to formation of a hematoma, which may be intramedullary, subperiosteal, or extraperiosteal, depending on the extent of the injury. The disruption of the vessels of the Haversian and Volkmann systems interrupts the blood supply to the osteocytes adjacent to the fracture site, resulting in a focal avascular trabecular and cortical bone. Necrosis of the bone, cartilage, and soft tissue occurs at the avascularized areas. With time, the hematoma is vascularized and inflammatory cells migrate into the injury site. The initial cellular repair processes are organization of the fracture hematoma and resorption of the necrotic tissues. Fibrovascular tissue replaces the clot. The collagen fibers eventually are mineralized to form the woven bone of the primary callus [1–5].

During the reparation phase, new bone is formed through two processes, endochondral and membranous; meanwhile, the necrotic bone of the fracture ends continues to be resorbed. Endochondral bone formation occurs within the hematoma, and membranous bone formation occurs under the periosteum. The organizing hematoma serves primarily as a fibrin scaffold that supports the repair cells. The repair cells are of mesenchymal origin and are pluripotent, forming collagen, cartilage, and bone. Initially, cartilage forms within the organizing hematoma and is referred to as the fracture callus. With time, the cartilage is resorbed and replaced by bone. The newly formed bone is highly disorganized woven bone. Concomitantly, the cells of the cambium layer of the periosteum and endosteum proliferate and begin to deposit bone matrix. The bone matrix envelops the forming callus. As the cartilage and bone form, the fracture becomes increasingly more stable; however, it remains vulnerable to deformation during this phase [3, 4].

McKibbin [5] and Chapman [4] described the formation of a medullary callus as part of the healing process. They stated that formation of the medullary callus occurs later in the healing process, forms in the absence of a cartilage model, and appears to be unaffected or possibly stimulated by motion at the fracture site. The primary function of the medullary fracture callus is to replace the fracture gap.
The final phase of healing is the remodeling phase, during which the disorganized woven bone is replaced with organized mature bone. Osteoclasts remove the superfluous and poorly organized woven bone, whereas osteoblasts form mature bone that is organized in accordance to biomechanical forces. Most likely, bone formation and resorption are controlled by piezoelectricity [3]. When the bone is subjected to stress – compressive, tensile, or shearing – electropositivity occurs along the more convex surface and electronegativity occurs along the more concave surface. Electropositivity appears to favor bone formation, and electronegativity favors bone resorption. Ultimately, the bone will return to its original contour or an altered form that enables the bone to sustain normal biomechanical forces [3, 4].

Subperiosteal new bone formation (SPNBF) is the bone formed by the cambium layer of the periosteum, as mentioned earlier. It requires special attention in infants and children because occasionally it is the only evidence of injury observed [2]. At very low levels of force, the periosteum may be disturbed in the absence of a fracture, causing SPNBF to occur (Fig. 6.1). When a fracture does occur, the weakly attached periosteum enables the hematoma to spread along the diaphysis and metaphysis, leading to diffuse new bone formation [3]. SPNBF also is laid down in response to frostbite, burns, and infection and as a function of normal growth and development [2, 6, 7]. Using high-detailed postmortem skeletal radiologic surveys, Kwon et al. [7] studied 101 neonates and infants who had died of sudden infant death syndrome. The authors found SPNBF in 35% of the sample. SPNBF peaked at ages 2–3 months, was uncommon in infants younger than 1 month and those older than 4 months, and did not exceed 2 mm in thickness. SPNBF was found more commonly on the bones of the lower extremities and bilaterally. Johnson [6] stated that SPNBF due to normal growth and development occurs most often in infants less than 4 months old. He also described pathologic SPNBF as typically layered, greater than 2 mm in thickness, having a convex border, and extending into the distal metaphyseal and physeal regions. Kleinman [2] found that the quantity, thickness, density, and longitudinal extension of SPNBF correlate to the severity of the injury and increase with movement at the fracture site.

Healing of physeal injuries follows a reparative course different from that of shaft and metaphyseal fractures. To understand the repair mechanisms, an understanding of normal growth and development is necessary. Growth at the epiphysis occurs through endochondral ossification. The epiphysis consists of hyaline cartilage, and at the furthest point from the physeal plate the cells are in a resting state. As the cells progress from the resting state to ossification, they transition through several phases. Histologically, the transition is broken down into five zones: (1) resting, (2) proliferation, (3) hypertrophic cartilage, (4) calcified cartilage, and (5) ossification (Fig. 6.2). In the resting zone, the chondrocytes are disorganized. In the proliferative zone, the chondrocytes divide rapidly and organize to form columns of stacked cells. The columns are oriented parallel to the long axis of the bone. In the hypertrophic cartilage zone, the chondrocytes become enlarged through the accumulation of glycogen, and the lacunar wall is resorbed, resulting in a thin septum between the cells. Some of the lacunae become confluent. In the calcified cartilage zone, the chondrocytes die and the thin septum of the chondrocytes becomes calcified by deposits of hydroxyapatite. In the ossification zone, blood capillaries and osteoprogenitor cells originating from the periosteum invade the cavities left by the chondrocytes. The osteoprogenitor cells give rise to osteoblasts, which deposit bone matrix over the calcified cartilage [2, 8, 9].

When the physeal plate is injured, the blood supply to the ossification zone is disrupted, interrupting the ossification of the matrix. In the absence of ossification, the hypertrophic and calcified cartilage zones thicken. Kleinman et al. [10] compared the thickness of the hypertrophic zones in traumatized distal tibiae to atraumatic distal tibiae and found it to be significantly greater at the site of injury. The authors found the thickening may be focal or diffuse, and they theorized the involvement is a reflection of the extent of the initial injury. When the thickening is focal, normal bone formation adjacent to the injury site creates the appearance of cartilaginous extensions into the physeal plate. Finally, the authors warned that diffuse hypertrophic thickening is observed in several pathologic conditions, such as rickets and mucolipidosis II.

![Figure 6-1](image_url). Subperiosteal new bone formation. Shown is the inferior surface of a rib angle of a 2-month-old male. Note the SPNBF in the absence of a fracture. The cause and manner of death were classified as undetermined.
Stages and Rates of Healing

In the dry bone under gross examination, the three phases of healing often are broken into four stages: induction, soft callus formation, hard callus formation, and remodeling. The induction stage spans from the moment of injury until SPNBF is observed at the fracture site (Figs. 6.3 and 6.4). Rounding of the fracture margins as a result of osteoclastic resorption occurs during this stage and is typically observed at 4–7 days post injury [2]. However, in very young infants, SPNBF may be observed prior to rounding of fracture margins (Fig 6.5).

The soft callus formation stage spans from the gross recognition of SPNBF to initial bridging of the fracture line. The fracture line is first bridged with fibrous matrix and then with primary woven bone (Figs. 6.6–6.8). The fibrous bridge typically disintegrates during the maceration process, but the SPNBF remains. During this stage, the callus is disorganized. Radiologically, SPNBF typically is observed 10–14 days post injury in children and 7–10 days in infants. SPNBF has been documented 4 days after injury in neonates with birth trauma [2]. The peak period for bridging of the fracture line is 14–21 days in children [2].

During the hard callus stage, the fracture callus fully bridges the fracture, the fracture line is in the process of obliteration, and the woven bone is replaced by lamellar bone, commonly termed consolidation (Figs. 6.9 and 6.10). During this stage, the osteoclasts are still active but most of the hematoma and inflammatory tissue is resorbed [2]. Saltter [11] gave a rough rule of thumb for aging consolidating femoral fractures: 3 weeks at birth, 8 weeks at 8 years, 12 weeks at 12 years, and 20 weeks at ≥20 years of age. Kleinman [2] stated that the hard callus stage may range from 14 to 90 days in children, but the peak period is 21–42 days.

The remodeling stage spans from the late conversion of the woven bone to lamellar bone to the reforming of the normal contour of the bone and reconstitution of the medullary cavity (Figs. 6.11 and 6.12). The fracture line may be visible during the early remodeling phase. According to Chapman [4], this phase may last throughout the growth and development of the child. Kleinman [2] found that the stage typically peaks around 1 year.

Islam et al. [12] examined 707 radiographs of forearm fracture in 141 patients, who ranged in age from 1 to 17 years, with a mean age of 8 years. The results of the study are listed in Fig. 6.13. This study presented a picture of the healing rate in subadults; however, the sensitivity of feature detection was affected by the variable interval between follow-up radiographs and the effects of the external stabilizing cast on the clarity of the radiographs.

The healing rate of physeal injuries is much less defined. There is a substantial paucity of research on physeal healing rates, most likely because of the difficulties associated with recognizing healing classic metaphyseal lesions (CMLs; see Chap. 5). SPNBF and callus formation typically do not occur with the healing of physeal injuries, and extensions of cartilage into the primary spongiosa are subtle [2, 10]. Kleinman [2] stated that a CML becomes inconspicuous by 4–8 weeks after the date of injury. Grossly, the fractured physeal surface transitions from sharp trabeculae to rounded thickened trabeculae to reformation of the physeal surface (Figs. 6.14–6.18). Thus far, these gross changes have not been correlated to the age of injury. However, the rate of healing most likely depends on the growth rate at the specific site as well as the extent of the injury, the age and health status of the individual, and whether the injury was repetitive [2, 10].
Figure 6-3. Early induction stage. Shown are acute posterior rib fractures of a 2-month-old female. Note there is an absence of healing, the fracture margins are sharp, and there is no SPNBF. The cause of death was classified as acute bacterial bronchopneumonia associated with bronchopulmonary dysplasia due to prematurity, the manner as natural.

Figure 6-4. Late induction stage. Shown is a healing fracture of the right ulnar shaft of a 3-year-old female. Note the rounding of the fracture margins with no definitive SPNBF. The cause of death was classified as multiple blunt trauma of the head, torso, and extremities with fractures; laceration of the heart, liver, and mesentery; and subdural hemorrhage. The manner was classified as homicide.
**Figure 6-5.** Healing radial fracture of a 9-month-old female. Note the sharp edges of the fracture surrounded by SPNBF. The cause of death was classified as blunt force head trauma with skull fractures and brain injury, the manner as homicide.

**Figure 6-6.** Early soft callus formation stage. Shown is a healing lateral rib fracture of a 13-month-old female. Note the thin layer of SPNBF forming along the pleural surface of the rib (arrowheads). The cause of death was classified as blunt injuries of the head, the manner as homicide.

**Figure 6-7.** Soft callus formation stage. Shown are healing lateral rib fractures of a 2-month-old male. Note that the SPNBF is thicker and nearly completely surrounds the shaft. The cause of death was classified as blunt trauma of the head with skull fractures and brain contusions, the manner as homicide.
**Figure 6-9.** Early hard callus formation. Shown is a healing posterior rib fracture of a 2-year-old female. Note that the fracture callus is still disorganized and the fracture line is still visible, but there is complete union of the fracture. The cause of death was classified as multiple blunt force injuries of the torso, the manner as homicide.

**Figure 6-8.** Late soft callus formation stage. Shown are healing lateral rib fractures of the same individual pictured in Fig. 6.7. Note that the fracture calluses are large and consist primarily of woven bone. In situ, the calluses bridged the fractures, but after processing, the fractures remain open.

**Figure 6-10.** Late hard callus formation stage. Shown is a healing posterolateral rib fracture of the same individual pictured in Fig. 6.9. Note that the fracture line is still somewhat visible as the woven bone is being consolidated into lamellar bone.
Figure 6-11. Early remodeling stage. Shown are healing posterior rib fractures of the same individual pictured in Fig. 6.7. Note the loss of the fracture line and the consolidation of the disorganized callus.

Figure 6-12. Remodeling stage. Shown are healing posterior rib fractures of the same individual pictured in Fig. 6.7. Note the organization of the bone callus into lamellar bone and the reduction of the callus as the normal contour of the bone is reestablished.
<table>
<thead>
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<th>Feature</th>
<th>Time since Injury, wk</th>
<th>Status</th>
<th>Patients, % (n/n)</th>
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<td>Present</td>
<td>60 (42/70)</td>
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<tr>
<td>SPNBF</td>
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<td>100 (0/22)</td>
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<td>SPNBF incorporation in cortex</td>
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<td>Present</td>
<td>100 (33/33)</td>
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<td></td>
<td>3</td>
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<tr>
<td></td>
<td>10</td>
<td>Present</td>
<td>58 (31/53)</td>
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<tr>
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<td>Occurred</td>
<td>9 (11/177)</td>
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<td></td>
<td>4–6</td>
<td>Occurred</td>
<td>56 (84/150)</td>
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<td></td>
<td>7</td>
<td>Occurred</td>
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<td>Decrease in fracture gap</td>
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<tr>
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<td></td>
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<tr>
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<td>90 (26/29)</td>
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<tr>
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<td>Partial</td>
<td>50 (sample size not given)</td>
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<tr>
<td></td>
<td>10</td>
<td>Complete</td>
<td>40 (12/30)</td>
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<tr>
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<td>1 patient</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>Present</td>
<td>95 (91/96)</td>
</tr>
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Figure 6-13. Radiologic changes observed in healing forearm long bone fractures (data from Islam et al. [12]).

Figure 6-14. Acute CML of the right proximal fibula of a 1-month-old male. Note the sharply defined trabeculae. The cause of death was classified as blunt trauma of the head with skull fractures and brain contusions, the manner as homicide.

Figure 6-15. Early healing CML on the right proximal fibula of a 2-year-old male. Note the area of thickened trabeculae (arrow). The cause of death was classified as blunt force head trauma, the manner as homicide.
Figure 6-17. Healing CML of the left distal tibia of a 2-month-old male. Note the reforming of the physeal plate. The cause of death was classified as blunt trauma of the head with skull fractures and subdural hemorrhage, the manner as homicide.

Figure 6-18. Healing CML of the left distal fibula observed in a 21-month-old female. The original fracture most likely was incomplete and involved the lateral region only. Note the porotic but reforming physeal plate. The cause of death was classified as blunt trauma of the torso and head with bilateral rib and scapular fractures, visceral lacerations, and multiple contusions; the manner was classified as homicide.
Healing of the Skull

Gross examination of processed bone reveals that woven bone bridges fractures in the skull in a manner similar to that of other parts of the skeleton, but large fracture calluses typically do not form. The woven bone deposited does not exceed the thickness of the bone (Figs. 6.19–6.21). Concurrently, a very thin layer of SPNBF blankets the bone affected by the hematoma (Fig. 6.22).

Research has shown that the dura mater is active as a regulator of calvarial ossification in infants and children less than 2 years of age [13, 14]. The fibrous membrane allows for more effective reossification of defects in the calvaria of children than in that of adults. Greenwald et al. [14] suggested that it is the differential expression of growth factors and extracellular matrix molecules that distinguishes the osteogenic propensity of adult and subadult dura mater. The authors were able to demonstrate a relatively greater rate of proliferation of dural cells as well as significantly higher rates of production of proliferating cell nuclear antigen, transforming growth factor β1, basic fibroblast growth factor 2, alkaline phosphatase, osteocalcin, and collagen I gene expression in young children. Calcified bone nodules arose from the immature dural cultures but did not grow from the adult dural cell cultures.

The increased healing rate of cranial defects in infants and children is well described in the surgical literature [15–17], and surgeons have taken advantage of this characteristic of the infant skull for many years. Hassler and Zentner [16] studied infants who underwent radical osteoclastic craniectomy for sagittal synostosis. Using radiographs, they found that reossification did not occur at the craniectomy edges but rather as small bone islands that developed between the separated margins of the surgical cuts. These islands of bone expanded to join the fragment margins. The authors found initial reossification started at 2 weeks postoperation in all patients and was completed by 6 months in infants 6 months old and younger at the time of surgery, 12–18 months in infants 7–12 months old, and 2 years in young children (>12 months). In young children, the authors found a tendency for the formation of persistent pseudo-sutures at the surgical sites. Of note, during the procedure the surgeons obtained hemostasis of the bone fragments using bone wax. One of the most common complications associated with bone wax is inhibition of osteogenesis [18]. Therefore, the healing rates reported by Hassler and Zentner [16] may be slightly slower than expected in the absence of bone wax. Paige et al. [17], studying healing of frontal orbital advancement procedures performed in children with sagittal synostosis, also found a direct correlation between the age of the child and the completeness of skull regeneration. In a retrospective study, the authors reviewed the medical charts of 81 patients who underwent the procedure. The median age of the group was 12 months. The authors found closure of the surgical sites occurred within the first postoperative year, but there was an increased prevalence in the formation of bone closure defects in patients 9 months and older at the time of the surgery.

Figure 6-19. Healing linear transparietal skull fracture of a 2-month-old male (same individual as pictured in Fig. 6.7). Note that the fracture is bridged with woven bone in the absence of a callus swell.
Figure 6-20. Healing occipital skull fractures of a 4-month-old male. The decedent survived in the hospital for 37 days after the date of injury. (A) Ectocranial surface of the occiput. (B) Endocranial surface. (C) Close-up of the right fracture (ectocranial surface). Note the bridging of the fracture in the absence of a callus swell. The cause of death was classified as blunt trauma of the head, the manner as homicide.

Figure 6-21. Healing lambdoidal diastatic fracture of the individual pictured in Fig. 6.20. Note the lacy bone layered on the suture digitations (arrows).

Figure 6-22. Healing linear parietal fracture (endocranial surface) of a 9-month-old female. Note the roughened raised SPNBF (arrowheads). The SPNBF is encroaching on the fracture from the outer limits of the hematoma. The cause of death was classified as blunt force head trauma with skull fractures and brain injury, the manner as homicide.
Healing Rate

As stated previously, the rate of healing depends on the age of the individual [19]. Fractures in infants and children heal much faster than those in adults for the following reasons: Immature bones are more woven than mature ones and have a propensity to incompletely fail in compression rather than tension. Incomplete compression fractures initially are more stable and require less callus formation during the healing process [1]. Immature bones are more vascular and capable of a greater hyperemic response than mature bones, allowing the inflammatory response to occur more rapidly. The osteogenic environment in infants and children enables the periosteum to contribute immediately and immensely to the formation of new bone [1, 3]. In children, there also is a higher concentration of trabecular bone adjacent to thick cortical bone. Ogden [3] theorized that this medullary bone may respond faster than lamellar bone to injury, improving the fracture healing rate in children. Furthermore, he stated that the periosteum is stronger in children than adults and is more likely to remain intact and coordinate the osteogenic response. Figure 6.23 demonstrates how quickly an infant heals, even with compromised health. The infant from whom these ribs were removed was 8 weeks old at the time of death. He was born at 38 weeks’ gestation and spent 9 days in the neonatal intensive care unit for supplemental oxygen and a small patent foramen ovale. He was discharged in good condition. During the autopsy, the infant was noted to be malnourished with features consistent with dehydration and severe stress reaction. Despite the poor health status of the infant, several of the ribs progressed to the remodeling stage within 8 weeks.

When repetitive injury occurs, it can slow the healing rate and cause an exuberant callus to form (Figs. 6.24–6.26) [2, 5]. Kleinman [2] stated that the term repetitive injury should be reserved for additional injury to a single fracture site. Repetitive injury should not be used to describe the presence of fractures of various ages seen throughout the body. He also stated that any fracture that is not stabilized either externally or internally is at significant risk for repetitive injury, even in the absence of an assault. When repetitive injury occurs after an initial period of healing, the newly formed blood vessels of the hematoma are torn, reinitiating the inflammatory response, leading to additional callus formation.

Figure 6.23. Multiple rib fractures in various stages of healing observed in an 8-week-old infant. A total of 45 healing rib fractures were observed and represented a minimum of four traumatic episodes. The healing stages are described as open fractures with initial primary bone formation, open fractures with large calluses of primary bone, stabilized fractures with organizing bone calluses, and reducing calluses. The cause of death was classified as blunt trauma of the head with skull fractures and brain contusions, the manner as homicide.
Figure 6-24. Exuberant fracture callus observed on an anterior rib fracture in a 6-year-old male. The decedent had a history of repeated blunt force trauma to the chest over a 2-week period. The cause of death was classified as multiple blunt force trauma, the manner as homicide.

Figure 6-25. Healing humeral fracture. (A, B) Healing humeral fracture with exuberant callus formation of a 2-month-old female. Panel (B) shows the posterior surface of the distal portion of the callus. Note the large cloaca. The cause of death was classified as methicillin-resistant *Staphylococcus aureus* chest wall abscess with disseminated infection and a contributing factor of chronic stress associated with multiple blunt trauma injuries; the manner was classified as homicide.

Figure 6-26. Posterior rib fracture of a 14-month-old male. Note what appears to be a secondary deposit of bone callus, suggesting a possible repeat injury (arrowhead). The cause of death was classified as blunt head trauma, the manner as homicide.
Interpretation

Forces associated with nonaccidental injury often affect multiple bones in separate regions of the body. A good example is the violent shaking of an infant, which generates compression and posterior levering of the chest as well as flailing of the limbs. Figure 6.27 shows the distribution of healing fractures observed in a 3-month-old female. A total of 25 rib fractures and 15 long bone fractures were found on the decedent. All fractures were healing. Based on the healing stages observed in the ribs, a minimum of two traumatic episodes were identified (Fig. 6.28). The type and distribution of the rib fractures—posterior, posterolateral, and at the chondro-osseous junction—and CMLs of nearly every long bone are consistent with constriction and posterior levering of the chest and uniform flailing of the limbs. The fracture distribution suggests violent shaking.

Figures 6.29–6.32 show rib and scapular fractures observed on a 21-month-old female. A total of 51 fractures were observed on the decedent. The more severe rib fractures were located in the posterior and posterolateral regions. At the fracture sites, the ribs were buckled or splayed apart. The fractures in the anterior and anterolateral regions of the ribs were simple, consisting primarily of incomplete failure in compression. The right and left scapulae were fractured along the medial border at the level of the scapular spine. The infraspinous processes along the medial margin of the right and left scapulae were buckled, and the internal and external surfaces of the medial border of the right scapula were splayed apart. The fractures observed in the decedent were in two stages of healing: soft callus formation with minimal SPNBF and acute with sharp margins and no bony reaction. The types and distribution pattern of the fractures were consistent with a posterior-to-anterior-directed force applied while the anterior chest was supported by a broad surface. The uniformity of the fracture types and the healing stages suggest a similar force was applied on two separate occasions within a relatively short period.

**Figure 6-27.** Distribution pattern of fractures of a 3-month-old female. The blue dots mark the location of the fractures. The cause and manner of death were classified as undetermined (skeletal diagram adapted from Buikstra and Ubelaker [20]).

**Figure 6-28.** Posterior rib fractures demonstrating two stages of healing. The fractures were observed in the same individual represented in Fig. 6.25. (A) Fracture in the soft callus formation stage. Note that the fracture is open, with minimal SPNBF. (B) Fracture in the remodeling stage.
Figure 6-29. Posterior and posterolateral rib fractures. (A–C)
Posterior and posterolateral rib fractures of a 21-month-old female. Note the splaying apart of the bone, especially in panel (B), and the buckling, most notable in panel (C). The fracture types were observed bilaterally in all ribs except right and left rib 12. The fracture types and distribution are consistent with a posterior-to-anterior impact. The cause of death was classified as blunt trauma of the torso and head with bilateral rib and scapular fractures, visceral lacerations, and multiple contusions; the manner was classified as homicide.
Figure 6-30. Lateral rib fracture of the individual pictured in Fig. 6.29. Note the buckling of the cortical bone, resulting from a force directed parallel to the anterior/posterior plane of the bone.

Figure 6-31. Incomplete compression fractures observed in the anterior region of the rib of the individual pictured in Fig. 6.27. Note the subtle fracture with SPNBF (right arrow) in comparison with the absence of a healing response at the fracture site, indicated by the left arrow.

Figure 6-32. Scapular fractures observed in the individual pictured in Fig. 6.29. (A) Note the bilateral uniformity (arrows), suggesting that a single impact caused both fractures. (B) Also note the splaying apart of the bone, most notable at the arrow.
Summary

Recognition of various stages of healing and fracture distribution patterns is critical to the successful reconstruction of the traumatic event(s). Providing an estimation of the minimum number of traumatic episodes and a timeline based on the stage(s) of healing can contribute significantly to the investigation of the case. Identifying the trauma vector of each fracture and then, when appropriate, associating the individual forces with a single action enable a comprehensive reconstruction of the inflicted injury.

References

The skeletal system, one of the ten major organ systems of the human body, is integral to routine body functions. The bones, tendons, and ligaments provide support, protection of visceral organs, and leverage for movement. The bones of the skeleton also store and release necessary minerals, such as calcium, magnesium, and phosphorus, into the bloodstream. Because of the skeleton’s participation in these vital functions, natural disease conditions that significantly affect the health, growth, and development of the soft tissues frequently also produce a pathologic skeletal response. Bone reactions to the stresses of premature birth, various disease syndromes, and genetic and congenital disorders can be seen grossly on the postmortem pediatric skeletal examination. Bone involvement in natural disease frequently is observed as osteopenia, osteoporosis, or osteomalacia, although certain syndromes produce a hyperostotic (proliferative) cortical bone response.

Weakened bone, whether it is delicately thin (osteopenia) or delicately soft (osteomalacia), is predisposed to pathologic fracture. Although less prevalent, hyperostotic bone response to disease may resemble a healed fracture or a healing fracture callus. These findings on skeletal examination may raise the suspicion for child abuse injuries.

Osteopenia and osteomalacia are the most common bone reactions to prematurity and natural disease in the developing infant skeleton. Rauch and Schoenau [1] described osteopenia and osteomalacia as the result of two types of deficiencies: (1) an apposition/resorption deficiency in which either an insufficient amount of osteoid is synthesized and deposited by the osteoblasts or there is pathologically increased resorption of the osteoid by the osteoclasts and (2) an insufficient amount of mineral incorporated into the osteoid. The first type of deficiency leads to osteopenia and may progress to osteoporosis. A reduction in the formation of osteoid and/or increased resorption of the osteoid results in fewer trabeculae and a thinner cortex. Cartilage typically is not affected and continues to develop normally. The authors maintain that because pediatric osteoporosis cannot be accurately measured by bone density radiography, osteoporosis should be diagnosed only in the presence of pathologic fractures, which indicate that bone mass is inadequately low and cannot withstand normal mechanical requirements. The second type of deficiency leads to osteomalacia. Incorporation of mineral into the osteoid is disturbed, causing a reduction in the average mineral content of the developing bone. The bone becomes relatively soft, and the trabeculae have a “washed out” appearance on radiograph. Cartilage is usually also affected by this deficiency, and the metaphyses of the affected elements have characteristic widened and irregular borders in radiographic view.

Many natural conditions that produce an abnormal bone response, especially those with a genetic or congenital etiology, are so multifocal and life-threatening that they are diagnosed quickly during pregnancy or shortly after birth (the perinatal period). The symptoms of others, particularly certain metabolic diseases, usually prompt the pediatrician to order a battery of diagnostic tests that result in a timely diagnosis and treatment. However, when a condition that results in pathologic bone is not severe or even apparent in the perinatal period and/or a clinical diagnosis is not obtained prior to the death of an infant or young child, child abuse may be suspected. Although not meant as a comprehensive review of all such disorders, this chapter presents 11 examples of natural conditions commonly described in the literature as possible mimics of bone response to inflicted injury [2–10]. The published incidence statistics for these conditions vary by resource because they are not public health-related reportable diseases. Concomitantly, the
Sequelae of Prematurity

The World Health Organization defines premature birth as childbirth prior to 37 weeks’ gestation [11]. Prematurity carries a substantial risk for infant mortality. Globally, an estimated 28% of all infant deaths within the first week of life are the result of premature birth. In the United States and other developed nations, the prognosis for survival of premature infants, even those as young as 22–25 weeks’ gestation, has improved greatly because of access to advances in perinatal medicine. Currently, infants born at 32 weeks’ gestation in developed nations have a survival rate comparable with that of infants born at term [11]. Despite the improved survivability, however, premature infants remain at risk for long-term comorbidities resulting from their original premature condition (sequelae of prematurity). Bone fragility and fracturing are possible sequelae of prematurity that may be mistaken for inflicted injury (Figs. 7.1–7.3).

Poor prenatal nutritional status, early separation from maternal hormones, substrate deficiency related to inadequate postpartum nutritional supplementation, treatment drug side effects (e.g., from corticosteroids), lack of mechanical stimulation of the skeleton, and severe systemic disease all are recognized as contributing factors in the development of bone fragility of prematurity [1, 12]. However, substrate deficiency is a fundamental and common factor in premature infants that is known to be a significant detriment to bone health. Supplementation of the premature infant diet usually is required to prevent substrate deficiency because breast milk contains low levels of calcium and phosphate [13]. When premature infants are fed unsupplemented breast milk, they experience low plasma concentrations of phosphate and raised calcium excretion levels in the urine. Their bodies do not have enough phosphate to support metabolism and overall growth, so phosphate is pulled from the skeleton. The calcium in the skeleton then lacks sufficient phosphorus to use as a building block for bone growth and is excreted as waste. Bone apposition and mineralization slow, resulting in fragility.

Several published studies from the 1980s compared radiologic bone density, bone mineral content as assessed through photon absorptiometry (PA), and serum calcium and phosphorus levels of premature and term infants [12–17]. Brooke and Lucas [13] reported that although radiologic evidence of osteopenia may be hard to assess, the condition may occur in up to 57% of infants with a birthweight of less than 1,000 g. The authors also noted that poor mineralization is very common in infants born weighing less than 1,500 g. James et al. [12] used PA to measure the bone mineral content of the forearm in 17 infants at 40 weeks postconception. They also took a blood sample from each infant and assayed it for alkaline phosphatase activity. The researchers found that bone mineral content was significantly correlated with infant size. The premature infants were shorter, weighed less, and had a lower bone mineral content than their term counterparts. Because premature infants typically are smaller at 40 weeks than a term infant, size was removed from the statistical analysis. Even when the calculations were adjusted for size, the premature infants had a significantly lower bone mineral content than the term infants in the study. However, Horsman et al. [17] found in a study of 58 infants measured at later dates of postconception (65–100 weeks) that there is a “catch-up” period of mineralization between 40 and 60 weeks that results in normalization of the bone density.

Rauch and Schoenau [1] stated that the physical density of the long bone diaphysis of all infants, whether premature or term births, decreases by about 30% during the first 6 months postpartum. This decrease is a result of the expansion of the marrow cavity, which occurs at a faster rate than the increase in the cross-sectional area of the cortical bone. This process resolves later in the premature infant than it does in the term infant. Therefore, the authors contended that comparison of measured differences of physical density between the premature and term infant at the same postconception age may not actually be evidence of long-term bone disease in the healthy premature infant.

Improvements in maternal health and prenatal follow-up, nutritional supplementation, and adjusted protocols that reduce the amount of time spent immobilized on the ventilator likely have reduced the incidence of severe bone responses in the typical premature infant born in the hospital since the 1980s [1]. However, because our forensic casework reflects only the premature infants with poor outcomes, bone fragility due to sequelae of prematurity, usually exacerbated by immobility during a long hospitalization, is a very common observation.
Natural Disease May Mimic Child Abuse

Figure 7-1. Osteoporotic rib from a 2-month-old individual born prematurely. Note the porosity on the internal surface of the rib body at the sternal end. The cause of death was classified as acute bacterial bronchopneumonia associated with bronchopulmonary dysplasia due to prematurity, the manner as natural.

Figure 7-2. Osteoporosis in a premature male. The osteoporotic sternal end of the right seventh rib is pictured. The 3-month-old male was born at 33 weeks’ gestation, remained in the hospital postnatally for 2 months, and then was released home. After 1 week at home, the individual returned to the hospital and died during a subsequent 28-day hospital stay. The cause of death was classified as blunt force head trauma with skull fractures, the manner as undetermined.

Figure 7-3. Osteopenic rib of a premature male with an extensive medical history. The sternal end of the left fourth rib from a 5-month-old male with an extensive medical history following premature birth shows marked fragility of the cortex with exposure of the trabeculae. The cause of death was classified as complications of extreme prematurity, the manner as natural.
**Osteogenesis Imperfecta**

Osteogenesis imperfecta (OI) is a broad term that refers to the skeletal system’s response to a disorder of the connective tissue in which type 1 collagen is of abnormal quality or is produced in reduced quantity. Type 1 collagen is a structural protein essential to the development and maintenance of the bones, dentin, organ capsules, fascia, corneas, sclera, tendons, meninges, and skin [18, 19]. OI is caused by missense mutations (point mutations that result in a codon that codes for an amino acid different from the one required) in the code for type 1 procollagen (COL1A1 and COL1A2). These mutations may be inherited as an autosomal dominant allele or an autosomal recessive allele (rare), or may occur as spontaneous mutations in the infant’s genome. OI is classified into types I–IV, based on the mode of inheritance and the severity of the phenotypic expression (Fig. 7.4). The most severe cases typically are lethal in the perinatal period [18]. A suite of major clinical features may be observed in an infant or child with OI: osteoporosis, cranial suture ossicles (Wormian bones), blue sclerae, hearing impairment, triangular positioning of facial features, crumbling teeth, and joint laxity. The limbs may be bowed, and multiple fractures and fenestrations related to bone fragility may occur throughout the skeleton (see Figs. 7.5 and 7.6). In some cases, the clinical signs of OI are more subtly expressed, and blue sclerae also may be observed in children who have thin sclerae but do not have OI [7]. Differentiation between OI and inflicted injury in the clinical setting may be complicated in mild cases, particularly when the collagen or DNA sequencing test results are inconclusive [7]. However, classic metaphyseal lesions (CML), which are highly suspicious for inflicted injury, are not typically reported in clinically diagnosed OI without severe loss of distal long bone mass [7, 18]. Plotkin [19] estimated the prevalence of OI at 1 in 20,000 live births.

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**Figure 7-4.** Classification of OI (adapted from Ablin et al. [18] and Plotkin [19]).

**Syndromes Resembling Osteogenesis Imperfecta**

Disorders that resemble OI in phenotype but originate through different genetic mutations are described in the literature and are sometimes listed as OI types V–VII, brittle bone diseases, or syndromes resembling OI (SROI). SROI may be caused by posttranslational modifications (as in posttranslational loss of cartilage-associated protein [CRTAP]) or by other mutations that affect the production of structural proteins. The mutations causing SROI may be inherited or of congenital origin and usually occur in recessive autosomal alleles [19]. As in OI, the morbidity and mortality of SROI depend on the severity of the phenotypic expression because the effects of these disorders on the skeleton do not typically resolve with time and growth and development.
Infantile Cortical Hyperostosis (Caffey Disease)

Infantile cortical hyperostosis, commonly referred to as Caffey disease because it was defined by Caffey and Silverman [20] in 1945, is a disease of bone proliferation that presents in tetuses and young infants. The fetal form, possibly inherited as an autosomal recessive trait, is severe and frequently fatal. The infantile forms, usually benign and self-limiting, do not typically result in mortality or permanent health deficits. The infantile forms are categorized into two types: heritable and sporadic. Both types initiate osteogenic changes that may be confused clinically and radiographically with bone response to trauma [21–23]. In the event an infant with undiagnosed Caffey disease receives a postmortem skeletal examination, cortical hyperostosis must be differentiated from healing trauma.

The etiology of sporadic infantile Caffey disease is not well defined. This form of the disease typically presents at 9–11 weeks of age. Multiple explanations for the development of the disease include an inflammatory response trigger, infection by a latent virus, or an allergic reaction. Cortical hyperostosis also has been observed in infants receiving therapeutic doses of prostaglandin E in preparation for surgery [21, 24].

The heritable infantile form typically presents at approximately 6–8 weeks of age and has been reported as following an autosomal dominant pattern of inheritance within families [21, 23, 25, 26]. These studies show that Caffey disease may result from a heterozygous missense mutation in COL1A1 with incomplete penetrance and variable expressivity, placing it in the same family of collagen disorders as OI.

The clinical presentation of Caffey disease is characterized by initial fever and soft tissue inflammation and swelling. Three pathologic phases have been described – early, subacute, and late [21] – that roughly correspond to the three stages of fracture healing – inflammation, repair, and remodeling – particularly as observed in cases of repetitive injury. In the early phase of Caffey disease, there is an acute intraperiosteal inflammatory reaction with swelling and thickening of the periosteum. During the subacute phase, the periosteum continues to thicken and undergoes increased subperiosteal new bone formation (SPNBF). As the phase progresses, osteoblastic activity produces exuberant and hyperplastic lamellar bone layers that thicken the bone cortex. Fibrosis of the bone marrow may develop. Disorganized bone also may be deposited in the peripheral soft tissues and form bony bridges between long bones. In the late phase, the underlying original cortex is removed by osteoclastic activity, resulting in a complete turnover of the cortical bone and reshaping of the element. Over time, active bone proliferation slows, peripheral bone is removed from the soft tissue, and as the disease self-resolves, additional cortical remodeling occurs. On postmortem skeletal examination, the bone response may resemble the exuberant bony callus of a healing fracture responding to repetitive injury. Caffey disease typically resolves before 2 years of age [21].
Caffey disease predilects the diaphysis of the long bones (including the clavicles), mandible, and ribs, but also may affect the scapulae asymmetrically [21, 26]. Other elements, such as the vertebrae and bones of the wrists and hands, rarely are involved. The incidence of Caffey disease is unclear, as it appears to be relatively rare but cases may be difficult to diagnose. Diagnosed cases also may be underreported.

Hypophosphatasia (Rathbun Syndrome)

Hypophosphatasia, also known as Rathbun syndrome [27], is an inherited or congenital metabolic disorder (inborn error of metabolism) that results in defective bone mineralization. This disorder is believed to be caused by a missense mutation in the autosomal recessive gene ALPL, which codes for tissue-nonspecific alkaline phosphatase (TNSALP) [28, 29]. The mutation results in a serum deficiency of the enzyme. Hypophosphatasia is a relatively rare disorder; an estimated incidence of 1 per 100,000 births was published in 1957 [30].

Hypophosphatasia has been classified into six forms based on the age when skeletal involvement occurs. Two of the forms – the infantile and childhood forms – may mimic child abuse. Infantile hypophosphatasia typically manifests with symptoms prior to 6 months of age. The infant may present with weight loss, vomiting, and hypotonia (reduced muscle tone). Blue sclera of the eyes may be observed, similar to the documented characteristic of OI. Laboratory studies typically reveal calcium in the urine and abnormally high levels of calcium in the blood. Osteopenia with pathologic fracture, premature craniosynostosis, irregularity of the long bone metaphyses, and “beading” of the ribs at the costochondral junction (similar to the rachitic ribs observed in rickets) often are observed on radiographic examination (Figs. 7.7–7.10). Mortality is high; approximately 50% of patients diagnosed with infantile hypophosphatasia do not survive [30].

The childhood form is somewhat less severe, although debilitating, and may be the result of compound heterozygosity in which there are two heterogeneous recessive alleles present at a particular locus [28]. Childhood hypophosphatasia may present with bone pain and/or pathologic fracture in a child who has experienced a delay in walking. The child may be short in stature and ambulate with a shuffling gait. Bone inflammation and osteomyelitis may develop without documented traumatic injury. Often there is premature loss of the deciduous teeth, particularly the incisors. Rachitic ribs and long bone metaphyses with radiolucent projections that extend from the epiphyseal plate into the metaphysis may be observed on radiographic examination. The projections at the long bone metaphyses are distinct radiographically from the poorly ossified epiphyses and flared metaphyses commonly observed in rickets but may resemble the focal thickening of healing metaphyseal injury [28, 31, 32]. The overall presentation of the child may be suspicious for chronic inflicted injury if further testing is not completed and a diagnosis rendered prior to death.
Figure 7-8. Transverse view of an osteoporotic left femur. This transverse view of the midshaft of the left femur pictured in Fig. 7.7 shows the sparse trabeculae and delicate cortical bone indicative of reduced bone quality.

Figure 7-9. Transverse view of an osteoporotic femur from a 5-year-old female. The trabeculae are sparse, and the cortical bone is relatively poor in quality. The individual spent 4 years as a quadriplegic following a motor vehicle crash. The cause of death was classified as asphyxia due to the detachment of a ventilator from the tracheostomy used to treat quadriplegia following blunt impact trauma with cervical spine injury; the manner was classified as an accident.

Figure 7-10. Healed fracture of the osteoporotic right femur pictured in Fig. 7.9. The shaft retains a slight anteroposterior bend from the fracture. An involucrum with large cloacae is present along the posterior surface, consistent with osteomyelitis (arrow).
Menkes Disease

The molecular basis of Menkes disease is an X chromosome-linked recessive trait that causes abnormal copper metabolism, impaired absorption in the intestines, and concomitant copper deficiency. Because of the impaired metabolism, copper accumulates in the duodenum, kidney, spleen, pancreas, and skeletal muscle. Copper deficiency during growth and development results in nerve sheath demyelination and interferes with neurodevelopment. The estimated international incidence of Menkes disease ranges from 1 in 50,000 to 1 in 250,000 births [33].

Menkes disease presents almost exclusively in males, because female carriers typically are asymptomatic. Infants with Menkes disease appear generally healthy from birth until approximately 8 weeks of age, when clinical manifestations begin to arise, including loss of developmental milestones, hypotonia, seizures, inguinal hernia, failure to thrive, abnormally kinky hair, and loose skin. Skeletal involvement in Menkes disease includes abnormal development of the sternum and sternal ribs that results in a “sunken” chest, osteoporosis with multiple fractures (see Figs. 7.7–7.10), diaphyseal periosteal reaction, metaphyseal spurring and widening, sutureal ossicles, and scalloping of the posterior portion of the vertebral bodies [33]. Arita et al. [34] and Bacopoulou et al. [35] reported separate cases in which male infants had radiologic results that increased the emergency room clinicians’ suspicions for inflicted injury. In both cases, the infants subsequently were diagnosed with Menkes disease. In the first case, the infant was hospitalized at 3 months with pneumonia, upper gastrointestinal bleeding, and seizure. At 9 months, he again was hospitalized with seizures. A cerebral CT scan suggested subdural hematoma. A radiographic skeletal survey showed metaphyseal spurs at the distal long bones. A preliminary diagnosis of “shaken baby” syndrome was investigated, but eventually the correct diagnosis of Menkes disease was obtained. In the second case, a 3-month-old infant presented to the hospital with seizures. MRI of the head showed bilateral subdural hematomas. A radiographic skeletal survey showed anterior flaring of the ribs and metaphyseal spurs of the distal femur, ulna, and radius. Biochemical and genetic studies confirmed Menkes disease in this case.

Very early recognition of Menkes disease in the perinatal period, followed by subcutaneous copper injections, has produced some success in achieving normal neurodevelopment. However, although there are milder variants, classic Menkes disease usually results in the child’s death by about 3 years of age [36]. Although Menkes disease is relatively rare, in the absence of a clinical diagnosis, familiarity with the skeletal manifestations of Menkes disease may inform the postmortem skeletal examination.

Mucolipidosis Type II

Mucolipidosis is a metabolic disorder of lysosomal storage inherited as an autosomal recessive allele. In this disorder, there is a functional deficiency of lysosomal enzymes that results in abnormal vacuolization and inclusions in the cytoplasm of mesenchymal cells, particularly the fibroblasts. In addition to producing valvular heart disease, mucolipidosis type II severely affects the trabecular and cartilage structures of the skeletal system [37, 38]. The disorder is slowly progressive from birth and typically fatal prior to 10 years of age. The most common mechanism of death is respiratory insufficiency due to mucosal thickening, congestive heart failure, and gradual stiffening of the rib cage. Reported global incidence estimates range from 1 in 123,500 to 1 in 625,500 live births [37].

Clinical symptoms of mucolipidosis type II include developmental delay and failure to thrive, noisy breathing, generalized hypotonia, frequent otitis media, thick skin, epicanthal folds, prominent abdomen with umbilical hernia, a flat midface with depressed nasal bridge and small orbits, widening of the rib bodies, kyphosis, concavities of the anterior vertebral bodies, pelvic dysplasia, joint limitation, diaphyseal expansion of the long bones with periosteal cloaking, short hands and fingers, and clubbing of the feet. Although some symptoms may be present at birth or shortly thereafter, the full clinical picture that includes many of the skeletal anomalies is not completely apparent until the infant is close to 12 months of age [37]. Therefore, despite the severe morbidity associated with this disorder, it is possible that a clear diagnosis of mucolipidosis type II may not be made prior to death and a subsequent postmortem examination.
Sickle Cell Disease

Sickle cell disease (SCD) is a heritable disorder that may result in sudden pediatric death from pulmonary distress. The hemoglobin-β gene (HBB) codes for the β-peptide chain of adult hemoglobin, β-globin. A mutation in HBB that produces the hemoglobin variant Hb S is the most common cause of SCD. In this variant, the amino acid valine is substituted for glutamic acid. This substitution causes aggregation of the hemoglobin molecules when they release oxygen. The red blood cells become rigidly sickle-shaped and clump together, forming blockages in the blood vessels. Additionally, anemia develops because the abnormal red blood cells are destroyed at a higher rate than is normal in humans. SCD is an autosomal recessive disorder. Individuals who have one copy of the normal HBB gene and one copy of the mutated gene have sickle cell trait and are less likely to experience symptoms of the disorder, although it does occur [39, 40].

SCD also may produce skeletal lesions as a result of sickle cell crises [41]. Acute and chronic bone infarcts (death of cellular elements of the bone marrow), medullary sclerosis, bone necrosis, periostitis, and sterile osteomyelitis with development of cloacae (see Figs. 7.7–7.10) are all discussed in the literature [42, 43]. Kepron et al. [42] reported an autopsy case of a 5-year-old boy who experienced “multifocal epiphyseal separations due to microinfarction and hemorrhage of the underlying bone.” The separations were accompanied by marrow fibrosis, subperiosteal hemorrhage, and SPNBF. The authors assert that on radiograph and during the skeletal examination, the epiphyseal separations resembled CML.

Hypovitaminosis D and Rickets

Vitamin D enhances absorption of calcium and phosphorus from the small intestine (see section “Short Bowel Syndrome”) and has a role in the maturation of osteoclasts that resorb calcium from the bones. Several disorders that cause malabsorption of nutrients, as well as poor dietary intake and even certain demographic characteristics of the individual, may result in insufficient vitamin D levels (hypovitaminosis D).

Vitamin D status is measured most accurately through analysis of serum 25-hydroxyvitamin D concentration. A concentration of less than 11 ng/mL indicates hypovitaminosis D. However, Heaney et al. [44] demonstrated that maximum calcium absorption occurs at levels greater than 32 ng/mL. Vitamin D is available from exogenous sources (absorption through diet and supplementation) but also may be synthesized endogenously. The skin acts as the major source of endogenous vitamin D production. A proper diet that includes vitamin D-supplemented milk and minimal exposure to sunlight typically provide sufficient access to vitamin D in healthy individuals. However, dark skin pigmentation, residence above the 35º latitude in winter, breastfeeding without vitamin D supplementation, and copious use of high-SPF sunscreen may decrease the amount of serum 25-hydroxyvitamin D concentration and are risk factors for hypovitaminosis D [45]. Hypovitaminosis D is associated with osteomalacia caused by lack of sufficient calcium and phosphorus uptake for bone health and with elevated parathyroid hormone levels, which may initiate increased movement of calcium from bone, resulting in further risk for demineralization and fractures [46]. The term rickets refers to the clinically and radiographically recognized metabolic bone disease that may ensue when hypovitaminosis D is not treated promptly.

Infants and children with congenital or untreated hypovitaminosis D are particularly susceptible to rickets because of the nutritional needs of active skeletal growth and development. The findings of rickets include diffuse bone rarefaction and osteomalacia, cranioptases, bowing of the weight-bearing long bones after the child begins to walk, softening of the ribs with flaring of the sternal ends, fraying and cupping of the metaphyses of the distal long bones on radiograph, and pathologic fractures with delayed healing (Figs. 7.11–7.14) [9, 45]. Metaphyseal changes in the long bones are usually bilateral and appear radiographically as a separation between the epiphysis and metaphysis. This finding is caused by demineralization in the calcified cartilage zone. The lack of mineralization at this location results in an area of diminished radiolucency that imitates a wide separation between the epiphysis and metaphysis [47]. Also as a consequence of the demineralization, the cartilage at the metaphysis loses normal columnar organization, resulting in the characteristic frayed and cupped appearance. Keller and Barnes [9] reported that although the affected metaphyses may become increasingly sclerotic and malformed, they are usually asymptomatic and resolve quickly with treatment of the hypovitaminosis D.
**Figure 7-11.** Comparison of typical and atypical development of the physeal surface. (A) Normal development of the physeal surface is shown in the left ulna, fibula, and radius from a 2-month-old individual. (B, C) In comparison, note the flared distal metaphyses in the left and right distal ulnae from a 3-month-old male with atypical bone development and quality. The physeal surfaces are deep, cup-shaped, and macroporotic. The cortical bone is slightly brittle, flaky, and easily chipped. On radiograph, the metaphyses appear frayed. The atypical quality and morphology of the bone in the 3-month-old individual is similar to that produced by a metabolic disease such as hypovitaminosis D. The cause of death was classified as undetermined, the manner as undetermined (co-sleeping).

**Figure 7-12.** Comparison of typical and atypical development of the distal left radius. The normal physeal surface of the distal left radius from a 5-month-old individual (A) and the atypical surface from the 3-month-old male (B) described in Fig. 7.11 are compared. Note the smooth, slightly convex surface with intact rim in panel (A) and the porous, rugged, slightly concave surface with chipped rim (arrow) in panel (B).
Metabolic Bone Disease in Short Bowel Syndrome and Biliary Atresia

Bone response to malabsorption of nutrients follows a pattern similar to that of rickets in both short bowel syndrome and biliary atresia, disorders that may affect the patient concurrently in pediatric cases. The etiology and health ramifications of these two disorders are discussed in this section.

Short Bowel Syndrome

Short bowel syndrome occurs when there is functional or anatomic loss of small intestine surface area to the extent that the capacity of the bowel to absorb nutrients is compromised. This disorder is characterized by diarrhea, steatorrhea (abnormal amount of fat in the feces), malabsorption, electrolyte imbalances, and malnutrition [48]. In children, short bowel syndrome usually is the result of necrotizing enterocolitis, in which the intestinal tissue dies; intestinal atresia (blockage or closure of the intestine); intestinal volvulus (abruption of the intestine by twisting); congenital short small bowel; congenital abdominal wall defects; or meconium peritonitis [49].

Skeletal growth, development, and maintenance depend on the skeletal system’s access to appropriate amounts of calcium, magnesium, and phosphorus. Approximately 30–35% of total dietary calcium and magnesium are absorbed in the small intestine. Although the duodenum is the site of active calcium transport, passive calcium transport occurs throughout the small intestine. Because of the longer period during which the contents are in the jejunum and ileum, these regions absorb a larger proportion of the total calcium. Magnesium also is absorbed throughout the small intestine, but the maximum absorption takes place in the proximal section of the small intestine. Approximately 65% of dietary
phosphorus is absorbed by the small intestine through a passive diffusional process stimulated when the concentration of phosphorus in the lumen exceeds 47 mg/L [50]. In short bowel syndrome patients, the absorptive surface area of the small intestine is so reduced that the essential minerals of calcium, magnesium, and phosphorus, as well as other important nutrients and fluids, are not made available in sufficient quantity for use by the body. Even with total or supplemented parenteral nutrition, inadequate absorption may occur. Growth and development of the skeleton slows, and overall bone density decreases. Radiographic findings include osteopenia, osteoporosis, and irregularities of the distal metaphyses of the long bones (Fig. 7.15).

**Figure 7-15.** Short bowel syndrome. The distal right and left ulnae of a 6-month-old infant with friable ossified metaphyseal rings, poor bone quality, and marked porosity are pictured. The cause of death was classified as short bowel syndrome with cholestasis and hepatic fibrosis due to partial small bowel resection and administration of parenteral nutrition for treatment of necrotizing enterocolitis associated with prematurity; the manner was classified as natural.

**Biliary Atresia**

The pathogenesis of biliary atresia is poorly understood. Various etiologies have been proposed that describe possible infectious origins and genetic anomalies [51]. Infants with biliary atresia may present with jaundice that persists longer than 2 weeks postbirth, dark urine, light stools, firm hepatomegaly, and splenomegaly. Biliary atresia has been classified into three types (I–III). Up to 90% of pediatric cases fall into the type III category, involving closure or destruction of the right and left hepatic ducts to the level of the hepatic port. In these cases, the ducts are typically open the first few weeks after birth but become inflamed and are progressively destroyed, resulting in cholestasis (reduced bile formation and flow). Surgical treatment prior to 2 months of age is essential to prevent death. Estimates of incidence range from 1 per 10,000 to 1 per 15,000 live births in the United States [52].

Skeletal findings of biliary atresia patients on radiographic examination include osteopenia, metaphyseal flaring, rachitic ribs, and pathologic fractures. DeRusso et al. [53] describe three cases of infants with previously diagnosed biliary atresia in which the skeletal findings in the emergency department raised suspicion of child abuse and resulted in investigation by child protective services. Katayama et al. [54] reported 13 biliary atresia cases in which radiologic manifestations of bone involvement were found, including fractures, rachitic ribs, and irregularities of the distal metaphyses.

**Summary**

Bone response to prematurity and/or natural disease may result in osteopenia, osteoporosis, osteomalacia, or proliferative bone formation in infants and young children. In the absence of a clear clinical diagnosis prior to the postmortem examination, fractures of delicate bone, frayed metaphyseal surfaces, or callus-mimicking bone formation are likely to raise suspicions of inflicted injury. It is important to include natural disease as a differential in the postmortem diagnostic process in pediatric cases.


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