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Invited Review

Evaluating long bone fractures in children: a biomechanical approach with illustrative cases☆

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Introduction

"... an understanding of the biomechanics of extremity injuries helps in determining the trauma that caused an injury"

"An understanding of how bones respond to loads that cause failure (fractures) can help in understanding the forces that cause the damage"(Levine, 2002, p. 491)

Knowledge of the basic biomechanics and anatomy of developing bone is fundamental to understanding long bone fractures in children. It is the knowledge of possible mechanisms for a given fracture type in combination with other information that increases the certainty with which the diagnosis of inflicted versus non-inflicted trauma may be made. By understanding *how* and *why* bones respond to different types of loading forces, and by understanding what types of fracture patterns different types of loads generate, the physician can better assess the validity of the stated injury mechanism in relation to the

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resultant fracture type (Daly & Calvert, 1991; Grant, Mata, & Tidwell, 2001; Hymel & Jenny, 1996; Joffe & Ludwig, 1988; John, Wherry, Swischuk, & Phillips, 1996; Kress et al., 1995; Pierce, Bertocci, Vogeley et al., 2001). The purpose of this article is to provide a *biomechanical basis for analyzing fracture patterns in children* in order to provide an objective method for evaluating consistency between injury and history.

Children age 4 years and younger are at the greatest risk of fatal or near fatal child abuse, and are the least able to defend themselves or explain what caused their injury. Children less than 1 year of age are especially at risk, with 40–80% of long bone fractures in this age group resulting from child abuse (Anderson, 1982; Beals & Tufts, 1983; Gross & Stranger, 1983; Leventhal, Thomas, Rosenfield, & Markowitz, 1993; McClelland & Heiple, 1982; Schwend, Werth, & Johnston, 2000; Thomas, Rosenfield, Leventhal, & Markowitz, 1991). In abuse cases, identification of the high-risk environment is *dependent on diagnosing the true mechanism of injury*. If abuse is not identified, there is a significant risk for recurrent and escalating inflicted injury (Jenny, Hymel, Ritzen, Reinert, & Hay, 1999; King, Diefendorf, Apthorp, Negrete, & Carlson, 1988; O'Neill, Meacham, Griffin, & Sawyers, 1973; Rivara, Kamitsuka, & Quan, 1988; Skellern, Wood, Murphy, & Crawford, 2000; Southall, Plunkett, Banks, Falkov, & Samuels, 1997). As a corollary, an abuse investigation of a long bone fracture that results from non-inflicted trauma consumes limited resources and exacts a considerable toll on falsely accused families (Scherl et al., 2000; Shaw, Murphy, Shaw, Oppenheim, & Myracle, 1997).

Identifying the *origin* of forces that has caused a long bone fracture in a young child can be extremely difficult. When a child sustains a fracture that has occurred in a public setting and is witnessed by a non-caretaker, the *nature* of the mechanism and origin of forces is known and the focus is the actual injury. When a young child or infant presents with a long bone fracture where the injury occurred in the privacy of the home, or with only caretaker witnesses, an additional focus must be on clarification of the mechanism. This additional focus helps to insure that the child is not being returned to a potentially life-threatening environment where the *origin* of forces is abuse. Judging *consistency* between fracture and history can be extremely difficult. When evaluating fractures in children, one must consider the stated injury history and resultant fracture type in terms of the potential magnitude and direction of forces and the expected structural bone responses. The type and location of a fracture is a direct reflection of the type, direction, and magnitude of forces required to cause that *specific fracture type* in that *specific bone and location* in that *age-specific child*. The fracture itself is not diagnostic (Carty, 1993; Rex & Kay, 2000; Shaw et al., 1997; Strait, Siegel, & Shapiro, 1995) of abusive or "accidental" energy, but rather diagnostic of the type and magnitude of loading forces that caused the resultant injury. Fractures resulting from a high energy event such as a car crash or a fall from a second story window, can be predictive of injury severity as well as co-existing injuries (Holmes, Sokolove, Brant, & Kuppermann, 2002; Taylor, Banerjee, & Alpar, 1994). In cases of child abuse, the injury mechanism is caretaker violence and the fracture is a marker of the severity of forces to which the child is being subjected.

A fracture is a direct reflection and result of the destructive energy generated by the injury event. The fracture morphology reflects: (1) the forces and resultant stress generated by the specific mechanism and (2) the ability of the bone (with its surrounding soft tissues) to resist these forces. This article will use a biomechanical approach to address: long bone fractures in infants and young children, how bone structure and material properties affect bone strength and likelihood of fracture, and how a specific fracture morphology reflects a specific load that has caused the failure or fracture pattern.

Biomechanical terms and concepts

The purpose of mechanics is to explain and predict physical phenomena associated with space, time, mass, and force (Beer & Johnston, 1977, p. 2). Nigg defines *biomechanics* as "the science that examines forces acting upon and within a biological structure and effects produced by such forces" (Nigg & Herzog, 1999, p. 2). Thus, biomechanics includes the study of forces and their effects within the human body and can be used to understand the injury mechanism associated with the application of force (Hall, 1999, p. 3).

Qualitative biomechanics emphasizes descriptive aspects of the injury event and bone morphology while quantitative biomechanics focuses on numeric analysis. Qualitative biomechanical reconstruction of an injury event requires extremely detailed and focused assessments. A qualitative biomechanical approach is practical for the physician evaluating the patient in the acute setting, whereas a quantitative biomechanical approach may not be practical or possible due to limited available data.

Force is an action that changes the state of motion of a body or changes the relative position of the molecules composing the body (Evans, 1974; Panjabi & White, 2001, p. 43) and can be thought of as a push or pull acting on a body. There are three pure forms of force: compression, tension, and shear. A *compressive force* is a pressing or squeezing force that is directed *axially* through the body or region (Hall, 1999, p. 73). *Tension* is a force that is also directed axially but results in stretching or pulling, rather than compression. *Shear* force tends to cause one part of the body to slide with respect to an adjacent part (Evans, 1974; Hall, 1999, p. 73). Each force is characterized by its magnitude, direction, and point of application relative to a given body (Panjabi & White, 2001, pp. 45, 63). A *moment* is generated by a force couple. When the force couple is acting on a structure, either twisting or bending moments result, depending on how the force couple acts on the structure. Torque, or twisting moment, is the rotary effect of an eccentric (non-axial) force (Hall, 1999, pp. 47, 72, 78). A bending moment is a non-axial, asymmetric force couple that causes the structure to bend (Hall, 1999, p. 75). A *load* is "a general term describing the application of force and/or moments to a structure" (Panjabi & White, 2001, p. 51). The overall loading is the sum of all the forces and moments acting on an object (Nigg & Herzog, 1999, p. 55). Loads create stress within the tissue, that is, *stress* is the "distribution of force within a body" (Hall, 1999, p. 74). Depending on the loads applied to the bone, tissue may undergo compressive, tensile, and shear stress. A *bending load* results when a non-axial, offset force is applied, producing tension on one side and compression on the other (Hall, 1999, p. 75). *Torsional loading*, when a structure is caused to twist about its longitudinal axis, produces pure shear in the transverse plane and a combination of tension compression at a 45° angle from the longitudinal axis. *Combined loading* refers to the presence of more than one form of loading and is the most common type of loading acting on the human body. "The amount of mechanical stress created by a force is inversely related to the size of the area over which the force is spread" (Nigg & Herzog, 1999, p. 75). Impact with the edge of a coffee table can result in a concentration of force over a small area, resulting in greater stress. The effects of force acting on a body include acceleration (or deceleration), and *deformation* (Hall, 1999, p. 77; Newman, 2002, p. 306). Factors that influence deformation include strength, *elasticity* and geometry *of the object*. If the *yield point or elastic limit* is exceeded and deformation is permanent, a bowing or plastic deformation of bone tissue results (Mabrey & Fitch, 1989). When deformation exceeds the *ultimate failure point*, mechanical failure of the structure occurs, which in the case of bone is manifested as a fracture.

Fractures represent incomplete or complete breaks in the continuity of bones, and are an indication that the stresses within the structure have exceeded the specific strength of the material in the region of

failure. Failure of bone may represent excessive stress and/or an abnormally weak bone or area of bone (Levine, 2002, p. 496; Pathria, 2002). Bone is *anisotropic* meaning that its biomechanical properties differ according to direction of force application. *Fracture morphology* is a detailed qualitative description of the failed structure, including location of bone failure (metaphyseal, diaphyseal, proximal 1/3, middle 1/3, or distal 1/3), line of fracture propagation (transverse, oblique, spiral, buckle/torus, and corner/bucket handle/classic metaphyseal lesion), and fracture segment relationships (angulation, displacement, fracture separation, and fragments or comminution). *The details of the failed structure give critical information as to the type and magnitude of the loading forces*.

The likelihood of fracture is dependent upon both extrinsic and intrinsic factors; both determine whether a fracture will occur and how the bone will respond to that stress.

Extrinsic factors

Extrinsic factors to the body include magnitude and direction of force, rate of loading, and area over which the force is distributed. Environmental factors such as surface type, height of fall, and initial velocity (standing, walking, running, propelled . . .) influence loading. Magnitude of load is also affected by the degree to which the impact surface can absorb and dissipate energy. Softer surfaces result in less energy available for injury while harder surfaces are more efficient in transmitting the energy to the body.

The rate of impact loading also influences the likelihood of fracture with bone responding differently to different loading rates. Bone may withstand a higher force (ultimate load) when the force is rapidly applied than it may sustain when a lower force is applied slowly (Hyde, 1992; Sammarco, Burstein, Davis, & Frankel, 1991). In other words, bone is stronger under dynamic loading than static loading (Mow & Hayes, 1991 p. 112). For example, applying a large load to the lower extremity over an extended period of time would produce a greater risk of fracture than if that same load was applied rapidly.

Intrinsic factors

Overview

The intrinsic biomechanical factors are the structural and material properties of the affected tissue (Gozna, 1982; Hall, 1999, pp. 73, 77). Because bone is a non-homogeneous, anisotropic material, its mechanical properties vary according to region of bone and direction of force application. Specifically, the biomechanical properties of bones depend on both tissue composition and distribution, and direction and rate of loading (Nigg & Herzog, 1999, p. 75). Both material and geometry of the structure determine how it will behave under any given loading condition (Hall, 1999, p. 91; Martin, 1991; Morild, Gjerdet, & Giertsen, 1993). The structural properties relate to the size and shape of a structure, both macro- and microscopically. Material properties are independent of structural size and shape and describe the actual properties of the local material or tissue. The local material properties of bone tissue depend on several intrinsic factors (apparent density, mineralization, microstructure, etc.), but they are independent of the overall size and shape of the entire bone. These tissue material properties may also vary depending upon the location of the bone within the body, the type of bone tissue (cortical vs. trabecular), developmental states, and disease (Gomez & Nahum, 2002, p. 210).

Bone material properties also vary depending upon the direction of loading, with bone tissue being able to resist failure in one direction (axis) better than another (Ford, Keaveny, & Hayes, 1996; Gomez & Nahum, 2002; Nigg & Herzog, 1999, pp. 72, 75; Turner & Burr, 1993). For example, adults' bone strength in compression is greater than in tension; but in children, bone is weaker in compression, leading to different fracture morphologies than in adults (Hall, 1999, p. 93; Ogden, 2000, p. 48). Bone matrix is a composite of organic (collagen and other proteins) and inorganic (mineral) materials. The collagen fibers are mineralized via inorganic, hydroxyapatite crystals containing calcium and phosphorus. The mineral content provides the hardness, or stiffness, and provides bone its compressive strength (the ability to resist failure under compression). The fibrous content provides elasticity, or flexibility and toughness, and provides bone with its tensile strength (ability to resist stretching forces).

Bone tissue

Long bones are made of two categories of bone: compact (cortical) bone and trabecular (spongy, cancellous) bone, with each having different biomechanical properties. The degree of porosity of bone marks the difference between the two types, with cortical bone being more dense, or less porous, allowing for greater mineralization and added stiffness, but less flexibility. Cortical, or compact bone has multiple haversian systems, made of concentric rings of lamellae of mineralized collagen fibers and lacunae, which help distribute nutrients through a cannilicular microchannel system. The number, size, and degree of mineralization of the osteons or haversian systems affect the way cortical bone responds to loading. Trabecular bone is found on the inside of the bone shaft and in each epiphysis and metaphysis. Trabecular bone is less compact, mineralized connective tissue with high porosity. This type of bony material consists of an open network of rods and bone plates called trabeculae. These plates are mineralized and their thickness and direction of alignment affects load-bearing capacity. The pores are fluid-filled with marrow and fat and provide energy absorption. The energy absorbing capacity of spongy bone is different from that of thick cortical bone due to these differences in porosity (Dormans & Flynn, 2001; Gomez & Nahum, 2002; Hall, 1999, pp. 91–93; Johnstone & Foster, 2001; Ogden, 2000, pp. 27, 48; Turner & Burr, 1993).

Bone is covered by the periosteal membrane that supplies nutrients to the underlying tissues. This membrane in children is especially vascular and loosely attached in the diaphyseal region. It is tightly adherent through fibrous and ligamentous insertion at the metaphyseal and epiphyseal region, adding to the biomechanical strength of that region. Muscles attach to the periosteum rather than directly to the bone to allow for coordination of growth (Johnstone & Foster, 2001). This unique interface between periosteum and bone provides some insight into how tensile and shear loading of long bone could lead to classic metaphyseal lesions, or corner fractures and subperiosteal new bone formation.

Bone biomechanics and strength

The biomechanical properties of the diaphysis and metaphysis are different due to their structural and material differences. The diaphysis is composed primarily of hard, less porous, compact bone, while the metaphysis has a thinner, fenestrated cortex surrounding trabecular bone. The thinner porous cortex of the metaphyseal region makes compression and resultant buckle fractures more likely (Figure 1). A biological edge effect, or internal "stress riser" exists where metaphyseal bone transitions to cortical bone, making this specific region more susceptible to failure in compression, resulting in a buckle fracture (Johnstone & Foster, 2001; Levine, 2002; Ogden, 2000, pp. 5–8).

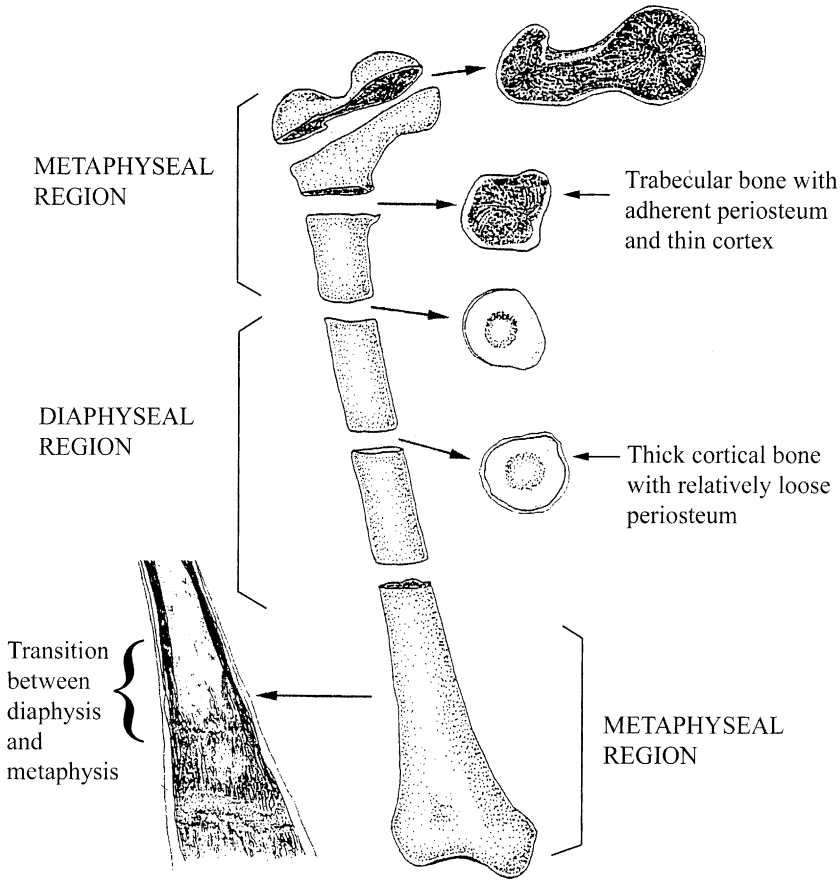

Figure 1. Illustration of bone architecture.

The strength of bone is related to bone mass and the apparent density of the bone, which is related to its mineral content (Gomez & Nahum, 2002; Rogers, 1992, p. 35). Weaker bone, or bone with lower bone strength and lower bone density, will potentially fracture at lower levels of force (Dabezies & Warren, 1997; Levine, 2002; Manzoni et al., 1996; Pathria, 2002). Bones of children have a lower mineral content than normal adult bone (Ogle et al., 1995), and are more elastic but less stiff. Children's bones can absorb relatively more energy before permanent deformation and fracture occurs (Currey & Butler, 1975; Hirsch & Evans, 1965; John et al., 1996). Research suggests that bone mineral density may be lower in some children who sustain fractures (Chan, Hess, Hollis, & Book, 1984; Davie & Haddaway, 1994; Henderson, Lin, & Greene, 1995; Landin & Nilsson, 1983; Mabrey & Fitch, 1989; Ogden, 2000, p. 23). This is an important consideration in children with potential bone density issues such as prematurity, severe failure to thrive or malnutrition, cystic fibrosis, spina bifida, or osteogenesis imperfecta (Dabezies & Warren, 1997; Davie & Haddaway, 1994; Henderson, Lin, & Greene, 1995).

Bone density has a direct relation to a bone's material properties and ultimate breaking strength (Foltz, 1977; Hui, Slemenda, & Johnston, 1988; Jurist & Foltz, 1977; Leichter et al., 1982; Mather, 1967).

Non-invasive techniques such as dual-energy X-ray absorptiometry or DXA, can be used to determine bone mineral density (BMD) and bone mineral content (BMC) (Braillon et al., 1992; Cameron, Mazess, & Sorenson, 1968; Ellis, Shypailo, Pratt, & Pond, 1994; Genant, Faulkner, & Glüer, 1991; Henderson, 1991; Mazess, 1996; Southard et al., 1991; Venkataraman & Ahluwalia, 1992). Research shows that these non-invasive measurements are predictive of bone strength in immature animal models (Crenshaw, 1986; Koo, Yang, Begeman, Hammami, & Koo, 2001; Pierce, Bertocci, Kambic, & Valdevit, 2001; Pierce, Valdevit, Anderson, Inoue, & Hauser, 2000), and in bone strength and fracture risk prediction models in adults (Beck, Ruff, Warden, Scott, & Rao, 1990; Beck et al., 1996; Gordon, Burns, & Keller, 1992; Martin, 1991; Mourtada, Beck, Hauser, Ruff, & Bao, 1996; Strømsøe, Hoiseth, Alho, & Kok, 1995). Clinical research to develop models for predicting the likelihood of fracture in a given child from a given mechanism is underway (Bertocci, Pierce, Deemer, Aguel, & Vogeley, 2001; Pierce, Bertocci et al., 2000; Pierce et al., 2003). Additional research is needed to develop models of fracture prediction in children.

Fracture morphology

"The biomechanical properties of bone dictate that a particular level of load and a particular mechanism of loading are necessary to cause a particular type of bone fracture" (Gomez & Nahum, 2002, p. 226). "In the clinical setting, the type, direction, and magnitude of a fracturing force can be inferred from the radiographic appearance of the resultant fracture" (Rogers, 1992, p. 22).

Bone may fail secondary to shear, tension, compression, bending, torsion, or combined loading. The fracture morphology reflects the failure mode (Pathria, 2002).

Spiral fractures

When a torque moment is applied to a bone such as a femur, the mechanical properties of bone in shear and tension are tested. That is to say, the ability of the bone to withstand failure from shear and tension stresses determines the likelihood of fracture. Torsion creates a state of pure shear between parallel transverse planes. In other planes (at other angles with respect to the longitudinal axis) tensile and compressive stresses are present, and they become maximum at a 45° angle to the longitudinal axis (Turner & Burr, 1993). Because failure generally occurs in tension, the resulting fracture pattern associated with torsion is spiral (Pathria, 2002). In experimental testing of human cadaver long bones, spiral fractures were only produced from torsional loading (Kress et al., 1995). Torsional loading occurs when a structure is caused to twist about its longitudinal axis. A small child running who gets his foot caught and trips can result in torsional forces being indirectly distributed to the femur. A spiral fracture may result (Figure 2). Spiral fractures were believed to be highly associated with abusive trauma due to the mechanism of twisting. Studies indicate that other fracture types such as transverse may be more common, and that a spiral fracture in itself is not diagnostic of abuse (Frasier, 2003; Leventhal et al., 1993; Scherl et al., 2000; Thomas et al., 1991). Spiral fractures can occur from seemingly innocuous trauma (Schwend et al., 2000) such as tripping while running. It is an uncommon fracture type to occur from falls higher than 10 feet (Bertocci, Pierce, Aguel et al., 2001; Pierce, Bertocci, Vogeley et al., 2001).

Figure 2. Spiral fracture.

Buckle or torus fractures

This fracture type is common in children and results typically from axial loading. The location of failure is usually at the proximal or distal 1/3 of the bone in the metaphyseal region. When the buckle fracture is located at the mid-diaphysis, a bending rather than axial load may have occurred, with immature bone failing under compression first. The loading forces may be insufficient to cause fracture completion, resulting in a unicortical buckle in this region. A torus fracture typically implies that circumferential cortical buckling has occurred. This distinction is sometimes difficult to identify on plain X-ray.

This fracture type is unique to developing bone and is due to both increased elasticity and decreased stiffness. When a parent is carrying a child and then falls or drops the child, the child's knee could potentially experience a compressive load from upward forces of the impact surface and the downward forces of her own weight, resulting in a buckle or torus fracture (Figure 3). This type of axial loading

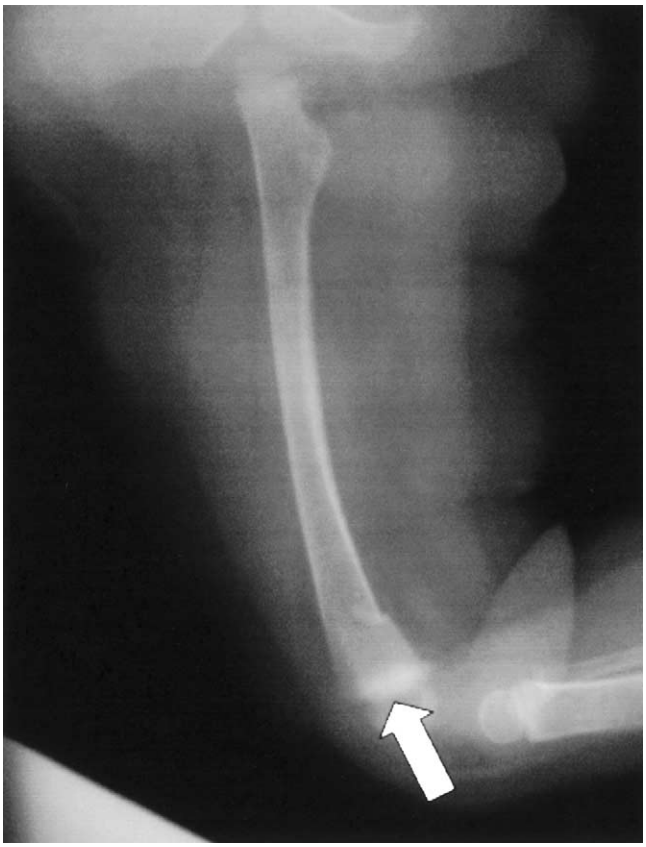

Figure 3. Buckle fracture.

results in compressive forces to the knee. Because immature bone fails in compression first (Pathria, 2002), the fracture line occurs at the weakest point, resulting in a buckle fracture of the distal femur. Compression failure results as hard cortical bone is compressed into the softer, trabecular bone of the metaphysis.

Buckle fractures in any child under 9 months of age are of concern due to a lack of mobility and underdeveloped protective reflexes that might lead to an “accidental” mechanism such as falling on an outstretched arm. Buckle fractures from abusive causes may result when a child or infant is thrown or slammed down onto a hard surface. This type of axial loading can cause a buckle type failure of bone. Cases also exist where a caretaker will intentionally bend a child’s extremity backward to cause pain. This bending mechanism can result in a buckling of the side of the bone that is being compressed as it is bent backwards.

Transverse fractures

A transverse fracture is characterized by a fracture line that is perpendicular to the long axis of the bone. Transverse fractures can occur from failure under tensile loading and from bending loads. During

bending, one portion of the bone will experience compressive stresses while the opposite region will experience tensile stresses. The bone fails and the fracture pattern is a combination of both tensile and compressive failure modes (Levine, 2002, p. 505; Turner & Burr, 1993). The forces may be delivered directly to the bone, as when the leg or arm is directly struck with an object. Forces may also be delivered indirectly to the diaphyseal region, as might occur when a child falls from a significant height and lands on her knee. This type of fall could generate a combined bending and axial compressive loading that causes shear stresses proximal to the point of impact. Direct trauma to the bone often results in transverse fractures that become increasingly more comminuted with progressively greater force (Pathria, 2002). A transverse fracture could result from the type of loading generated from a parent falling onto steps while carrying a child. If the child's leg impacted between the caretaker and the edge of the step, this could potentially result in a bending load with a resultant transverse fracture (Figure 4). Tension loading can also result in transverse fractures of the distal fibula, for example, with inversion injuries of the ankle.

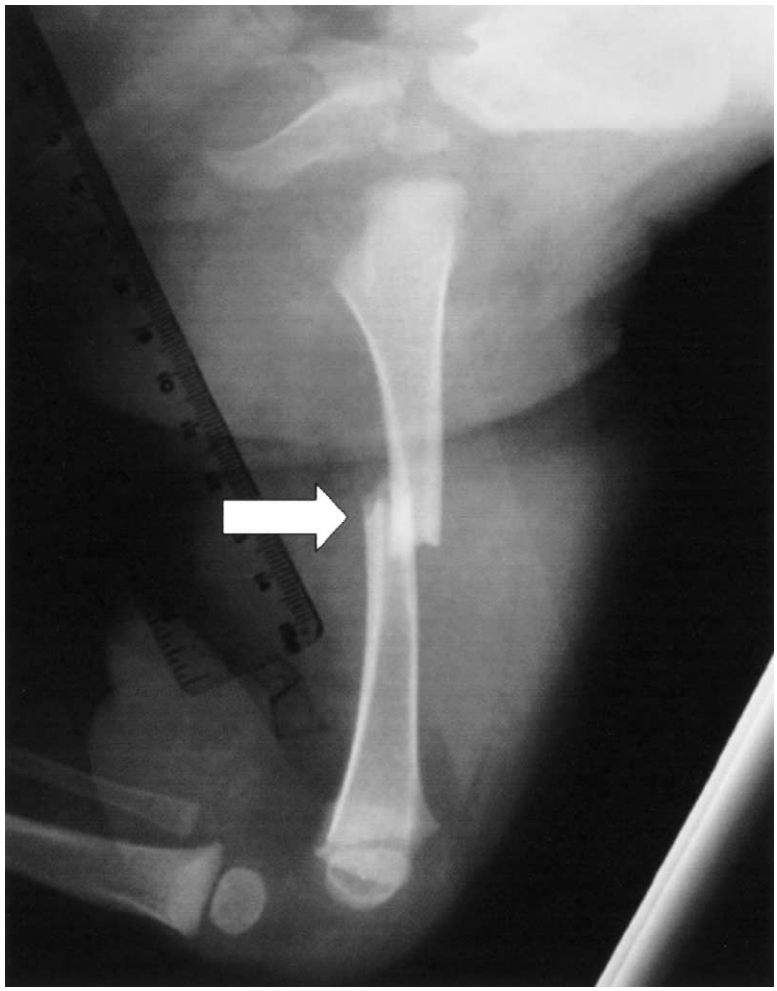

Figure 4. Transverse fracture.

Completed (non-greenstick) and displaced transverse fractures often result from mechanisms of high energy such as injuries involving encounters with cars or falls from significant heights (Bertocci, Pierce, Aguel et al., 2001; Pierce, Bertocci, Vogeley et al., 2001).

Studies suggest that transverse fractures may be a more common type of fracture to occur in abusive trauma (King et al., 1988; Leventhal et al., 1993; Scherl et al., 2000). When a child is diagnosed with a transverse fracture, the mechanism should reflect the specific type and magnitude of forces required to cause this specific fracture morphology.

Oblique fractures

With respect to the longitudinal axis, failure of bone at a 30–45° angle produces an oblique fracture line. This fracture type usually results from combination loading, and the fracture morphology reflects the predominate loading type. A longitudinal compressive force with rotation is an example of combined loading as might occur if a child falls off the top bunk bed and twists as the knee impacts the floor (Figure 5). A long oblique is common when torsion is the predominant force. Of note, a long oblique and a spiral fracture often appear very similar on X-ray, and may be indistinguishable in some cases. A shorter line of fracture propagation commonly occurs when the predominant force is bending or compression that results in a more transverse orientation and a “shorter” oblique fracture (Levine, 2002, p. 505; Rogers, 1992, p. 23). If a compressive load is applied during bending, a butterfly fragment may result, or in children with greater elasticity, an oblique fracture can occur. Alternatively, transverse loading of the shafts of long bones can result in an oblique fracture pattern as well as fragmentations. This pattern of bone failure is most likely due to the bone’s intrinsic response to tensile stresses (Kress et al., 1995).

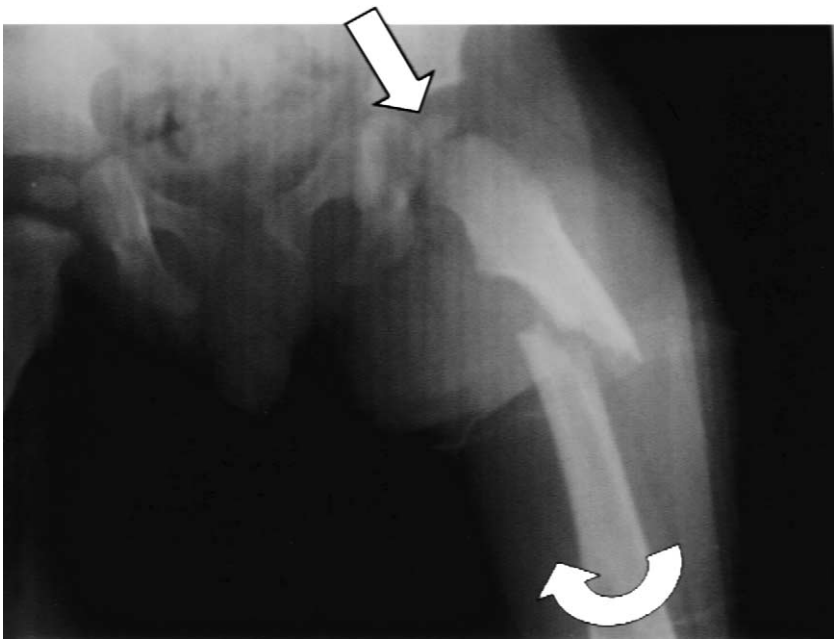

Figure 5. Oblique fracture.

Figure 6. Classic metaphyseal lesion.

Classic metaphyseal lesions (CML, corner/bucket handle fractures)

The CML is a subepiphyseal planer fracture through immature metaphyseal bone (Figure 6). The appearance of a corner, avulsion or bucket handle depends on the extent of bone failure and radiographic projection (Kleinman, Marks, & Blackbourne, 1986). Bone can fail in this pattern from both shearing and tensile stresses. The weakest area of bone for a given loading condition determines the injury threshold for that structure. Histological studies have shown that the immature metaphyseal bone, or

the primary spongiosa fails and a planer fracture through this region results (Cooperman & Merten, 2001; Kleinman et al., 1986; Osier, Marks, & Kleinman, 1993). We know of no published experimental model that investigates the biomechanics of CML bone failure. Because trabecular bone does not, in general, contain haversian systems in its architecture, a trabecular bone specimen would separate more easily under tension or shear (Gomez & Nahum, 2002, p. 221). This may explain biomechanically how or why CMLs occur when a child is pulled or forcefully yanked up by an extremity and/or shaken violently. As indirect tensile and shearing forces are applied axially and non-axially to an extremity, a planar fracture may result (Kleinman, 1998, p. 22). CMLs have been diagnosed in cases where the inflictor confesses to "yanking" the child up by the extremity, slamming the child onto a surface by using an extremity as the "lever" and, with violent shaking of the child. Both violent shaking and forceful pulling by an extremity could potentially produce both shear and tensile loading to the long bone. This specific fracture morphology is reflective of an unusual failure mode and is diagnostic of a unique type of mechanism required to cause this fracture type. This pattern of bone failure is highly associated with abusive trauma (Kleinman et al., 1986; Kleinman, Marks, Richmon, & Blackbourne, 1995; Kleinman et al., 1996) but has occurred in non-abusive cases from the type of loading forces generated during surgery for repair of clubfoot (Grayev, Boal, Wallach, & Segal, 2001).

Subperiosteal new bone formation (SPNBF)

Shear and torsional loading applied along the outer bone surface parallel to the long bone axis can result in periosteal separation and hemorrhage, leading to sub-periosteal new born formation. SPNBF can be identified radiographically as a thin or hazy layer of cortical bone separated from the original by a thin dark line, representing hemorrhage (Kleinman, 1998, p. 11) (Figure 7). SPNBF is not a discrete fracture per say, but its presence may indicate healing of an underlying bone injury. Both direct and indirect forces can cause subperiosteal hemorrhage. There are several causes of SPNBF, but their presence may be an indication that the child was subjected to shearing, tensile or torsional forces that are known to cause this morphologic and radiographic finding. Its presence is important when trying to identity the types of forces a child may have been subjected to. The caretakers of infants with SPNBF often confess to shaking the child; others describe grabbing and yanking the involved extremity and/or slamming the baby down onto a surface. SPNBF is a non-specific finding that reflects subperiosteal hemorrhage from any cause (Kleinman, 1998, pp. 10–12).

Magnitude of force

Certain fracture morphologies and locations (such as displaced, transverse, comminuted and femoral neck fractures) may be associated with larger magnitudes of force. A high-energy direct blow to an adult bone will cause a markedly comminuted fracture (Levine, 2002, p. 504). "Highly comminuted fractures typically are associated with extensive soft tissue injury and indicate a large amount of energy dissipation in conjunction with a rapid loading rate" (Pathria, 2002). In children, high-energy direct blow fractures are more likely to be displaced transverse fractures without comminution due to their elasticity, although exceptions exist. The presence of a comminuted fracture in a healthy child is most often indicative of a direct, high-magnitude impact to the leg (Figure 8) and is highly unlikely to be associated with a simple

Figure 7. Subperiosteal new bone formation.

fall or low energy event. Other fractures, such as a small buckle fracture or a spiral or oblique hairline fracture without displacement, are associated with lower energy mechanisms of injury (Levine, 2002, pp. 497, 514). Subtle buckle and hairline fractures are often associated with subtle and delayed clinical findings (Daly & Calvert, 1991; Pierce et al., 2003).

Figure 8. Comminuted fracture.

Limitations

Concepts and the current level of understanding of fractures in children of the magnitude and direction of loading required to produce specific fracture types in developing bone is limited. This limitation of knowledge is due, in part, to the lack of experimental research that has been done in human infant specimens, and to a lack of an ideal model for investigation of bone strength and likelihood of fracture in immature bone. Much research is still needed to help address remaining questions of how immature bone responds to different types of loading forces at different stages of development, and how different disease processes affect the likelihood of fracture.

Summary

The following should be considered when evaluating a child with a long bone fracture:

1. What are the biodynamics of the injury event, the energies generated by the event, and how could certain factors of the injury environment contribute to the likelihood of injury?
2. What injuries are expected, and what is the likelihood that the event generated the specific load required to cause each and all of the injuries?
3. Did the energy of the event exceed the injury threshold, or was there a biological abnormality such as decreased bone density that resulted in a lowering of the actual threshold for injury? Is there evidence of bone weakness or disease?
4. Is the fracture morphology consistent with the direction, magnitude, and rate of loading of the described mechanism?
5. Is the fracture pattern unusual, and one that requires an extremely unusual loading condition, as is the case with a CML?
6. What is the child's developmental capabilities and could the child have generated the necessary energy, independent of "outside" forces, to cause the observed injury?
7. Does the fracture reflect a high-energy fracture? Did the event generate enough energy to cause a high-energy fracture? Or is the fracture a small cortical defect, or hairline crack, reflecting a smaller amount of energy required for propagation of the fracture type?
8. What regions of the bone have been injured and what are the structural components that affect the ultimate pattern of fracture that is being observed? Were there structural factors that contributed to the likelihood of fracture?

Mechanisms of injury generate specific forces called loads that have the potential to cause structural damage. If the mechanism results in forces that exceed the injury threshold of a long bone, then a fracture is generated. The fracture morphology is a direct reflection of the degree and direction of the forces and the ability of the tissue to resist those forces. Mechanisms that generate greater amounts of energy with greater magnitudes of force may result in a completed fracture with the fracture type depending on the resultant combination of forces and moments. The fracture may be angulated or displaced, again reflecting a greater magnitude of force that has propagated the fracture. Fracture patterns such as classic metaphyseal lesions reflect unusual types of loading forces. When fracture morphology seems inconsistent with the history, or reflects a high-energy fracture pattern, or an unusual type of loading, it is paramount to determine

how and if the explained mechanism generated the necessary forces to create the observed injury. If an inconsistency exists, further investigation to evaluate for abusive trauma is warranted.

When evaluating fractures in children, it is critical that the clinician determine if the injury and stated mechanism are consistent. Observations of the resultant damage (fracture, and other injuries if present) and historical details of the scenario provide necessary input for reconstruction from a biomechanical, physics-based consideration of the injury, and evaluation of injury plausibility. Approaching childhood injuries from a biomechanical point of view allows for a better understanding of the type and magnitude of forces to which a child is being subjected. This in turn helps to better define the level of risk involved and the means necessary to ensure the future safety of the child. The response and intervention must equal the injury risk that biomechanics helps clarify scientifically. If the trauma is minimized, underestimated, or misinterpreted, the intervention needed to protect the child may fall short.

References

Anderson, W. A. (1982). The significance of femoral fractures in children. *Annals of Emergency Medicine*, *11*, 174–177.

Beals, R. K., & Tufts, E. (1983). Fractured femur in infancy: The role of child abuse. *Journal of Pediatric Orthopedics*, *3*, 583–586.

Beck, T. J., Ruff, C. B., Mourtada, F. A., Shaffer, R. A., Maxwell-Williams, K., Kao, G. L., Sartoris, D. J., & Brodine, S. (1996). Dual-energy X-ray absorptiometry derived structural geometry for stress fracture prediction in male US Marine Corps recruits. *Journal of Bone and Mineral Research*, *11*, 645–653.

Beck, T. J., Ruff, C. B., Warden, K. E., Scott, W. W., Jr., & Rao, G. U. (1990). Predicting femoral neck strength from bone mineral data. A structural approach. *Investigative Radiology*, *25*, 6–18.

Beer, F., & Johnston, R. (1977). *Vector mechanics for engineers*. New York: McGraw Hill.

Bertocci, G., Pierce, M. C., Aguel, F., Deemer, E., Vogeley, E., & Szczepanski, J. (2001). Energy associated with pediatric accidents resulting in femur fractures: A means to differentiate between accidents and abuse [abstract]. *Pediatric Research*, *49*, 92A.

Bertocci, G., Pierce, M. C., Deemer, E., Aguel, F., & Vogeley, E. (2001). Femur fractures and child abuse: Using engineering principles to develop a radiographic model for evaluating bone strength and likelihood of fracture [abstract]. *Pediatric Research*, *49*, 92A.

Braillon, P. M., Salle, B. L., Brunet, J., Glorieux, F. H., Delmas, P. D., & Meunier, P. J. (1992). Dual energy X-ray absorptiometry measurement of bone mineral content in newborns: Validation of the technique. *Pediatric Research*, *32*, 77–80.

Cameron, J. R., Mazess, R. B., & Sorsenson, J. A. (1968). Precision and accuracy of bone mineral determination by direct photon absorptiometry. *Investigative Radiology*, *3*, 141–150.

Carty, H. M. L. (1993). Fractures caused by child abuse. *The Journal of Bone and Joint Surgery*, *75B*, 849–857.

Chan, G. M., Hess, M., Hollis, J., & Book, L. S. (1984). Bone mineral status in childhood accidental fractures. *American Journal of Disease of the Child*, *138*, 569–570.

Cooperman, D. R., & Merten, D. F. (2001). Skeletal manifestations of child abuse. In R. M. Reece & S. Ludwig (Eds.), *Child abuse: Medical diagnosis and management* (pp. 123–156). Philadelphia: Lippincott Williams & Wilkins.

Crenshaw, T. D. (1986). Reliability of dietary Ca and P levels and bone mineral content as predictors of bone mechanical properties at various time periods in growing swine. *Journal of Nutrition*, *116*, 2155–2170.

Currey, J. D., & Butler, G. (1975). The mechanical properties of bone tissue in children. *The Journal of Bone and Joint Surgery*, *57-A*, 810–814.

Dabezies, E. J., & Warren, P. D. (1997). Fractures in very low birth weight infants with rickets. *Clinical Orthopaedics and Related Research*, *335*, 233–239.

Daly, K. E., & Calvert, P. T. (1991). Accidental femoral fracture in infants. *Injury: The British Journal of Accident Surgery*, *22*, 337–338.

Davie, M. W. J., & Haddaway, M. J. (1994). Bone mineral content and density in healthy subjects and in osteogenesis imperfecta. *Archives of Disease in Childhood*, *70*, 331–334.

Dormans, J. P., & Flynn, J. M. (2001). Pathologic fractures associated with tumors and unique conditions of the musculoskeletal system. In J. H. Beaty & J. R. Kasser (Eds.), *Rockwood and Wilkins' Fractures in children* (5th ed., pp. 139–240). Philadelphia: Lippincott Williams & Wilkins.

Ellis, K. J., Shypailo, R. J., Pratt, J., & Pond, W. G. (1994). Accuracy of dual-energy X-ray absorptiometry for body-composition measurements in children. *American Journal of Clinical Nutrition*, *60*, 660–665.

Evans, F. G. (1974). The mechanical properties of human cortical bone. *Accademia Nazionale dei Lincei*, *208*, 3–6.

Foltz, A. S. (1977). Human ulnar bending stiffness, mineral content, geometry and strength. *Journal of Biomechanics*, *10*, 455–459.

Ford, C. M., Keaveny, T. M., & Hayes, W. C. (1996). The effect of impact direction on the structural capacity of the proximal femur during falls. *Journal of Bone and Mineral Research*, *11*, 377–383.

Frasier, L. D. (2003). Child abuse-or mimic? *Consultant for Pediatricians*, *2*, 212–215.

Genant, H. K., Faulkner, K. G., & Glüer, C.-C. (1991). Measurement of bone mineral density: Current status. *The American Journal of Medicine*, *91*, 49S–53S.

Gomez, M. A., & Nahum, A. M. (2002). Biomechanics of bone. In A. M. Nahum & J. W. Melvin (Eds.), *Accidental injury* (pp. 206–227). New York: Springer-Verlag.

Gordon, K. R., Burns, P., & Keller, G. (1992). Experimental changes in mineral content of juvenile mouse femora. *Calcified Tissue International*, *51*, 229–232.

Gozna, E. R. (1982). *Biomechanics of musculoskeletal injury*. Baltimore, MD: Williams & Wilkins.

Grant, P., Mata, M. B., & Tidwell, M. (2001). Experience and reason. Femur fracture in infants: A possible accidental etiology. *Pediatrics*, *108*, 1009–1011.

Grayev, A. M., Boal, D. K., Wallach, D. M., & Segal, L. S. (2001). Metaphyseal fractures mimicking abuse during treatment for clubfoot. *Pediatric Radiology*, *31*, 559–563.

Gross, R. H., & Stranger, M. (1983). Causative factors responsible for femoral fractures in infants and young children. *Journal of Pediatric Orthopedics*, *3*, 341–343.

Hall, S. J. (1999). *Basic biomechanics* (3rd ed.). Boston: WCB McGraw-Hill.

Henderson, H. C. (1991). Assessment of bone mineral content in children. *Journal of Pediatric Orthopaedics*, *11*, 314–317.

Henderson, R. C., Lin, P. P., & Greene, W. B. (1995). Bone-mineral density in children and adolescents who have spastic cerebral palsy. *The Journal of Bone and Joint Surgery*, *77-A*, 1671–1679.

Hirsch, C., & Evans, F. G. (1965). Studies on some physical properties of infant compact bone. *Acta Orthopedica Scandinavia*, *XXXV*, 300–313.

Holmes, J. F., Sokolove, P. E., Brant, W. E., & Kuppermann, N. (2002). A clinical decision rule for identifying children with thoracic injuries after blunt torso trauma. *Annals of Emergency Medicine*, *39*, 492–499.

Hui, S. L., Slemenda, C. W., & Johnston, C. C., Jr. (1988). Age and bone mass as predictors of fracture in a prospective study. *Journal for Clinical Investigation*, *81*, 1804–1809.

Hyde, A. (1992). *Crash injuries: How and why they happen*. Biscayne, FL: HAI Publishers.

Hymel, K. P., & Jenny, C. (1996). Abusive spiral fractures of the humerus: A videotaped exception. *Archives Pediatric Adolescent Medicine*, *150*, 226–227.

Jenny, C., Hymel, K. P., Ritzen, A., Reinert, S. E., & Hay, T. C. (1999). Analysis of missed cases of abusive head trauma. *Journal of the American Medical Association*, *281*, 621–626.

Joffe, M., & Ludwig, S. (1988). Stairway injuries in children. *Pediatrics*, *82*, 457–461.

John, S. D., Wherry, K., Swischuk, L. E., & Phillips, W. A. (1996). Improving detection of pediatric elbow fractures by understanding their mechanics. *RadioGraphics*, *16*, 1443–1460.

Johnstone, E. W., & Foster, B. K. (2001). The biological aspects of children's fractures. In J. H. Beaty & J. R. Kasser (Eds.), *Rockwood and Wilkins' fractures in children* (5th ed., pp. 21–47). Philadelphia: Lippincott Williams & Wilkins.

Jurist, J. M., & Foltz, A. S. (2001). Human ulnar bending stiffness, mineral content, geometry and strength. *Journal of Biomechanics*, *10*, 455–459.

King, J., Diefendorf, D., Apthorp, J., Negrete, V. F., & Carlson, M. (1988). Analysis of 429 fractures in 189 battered children. *Journal of Pediatric Orthopaedics*, *8*, 585–589.

Kleinman, P. K. (1998). *Diagnostic imaging of child abuse*. St. Louis: Mosby.

Kleinman, P. K., Marks, S. C., & Blackbourne, B. (1986). The metaphyseal lesion in abused infants: A radiologic-histopathologic study. *American Journal of Roentgenology*, *146*, 895–905.

Kleinman, P. K., Marks, S. C., Jr., Richmond, J. M., & Blackbourne, B. D. (1995). Inflicted skeletal injury: A postmortem radiologic-histopathologic study in 31 infants. *American Journal of Roentgenology*, *165*, 647–650.

Kleinman, P. K., Nimkin, K., Spevak, M. R., Rayder, S. M., Madansky, D. L., Shelton, Y. A., & Patterson, M. M. (1996). Follow-up skeletal surveys in suspected child abuse. *American Journal of Roentgenology*, *167*, 893–896.

Koo, M. W. M., Yang, K. H., Begeman, P., Hammami, M., & Koo, W. W. K. (2001). Prediction of bone strength in growing animals using noninvasive bone mass measurements. *Calcified Tissue International*, *68*, 230–234.

Kress, T. A., Porta, D. J., Snider, J. N., Fuller, P. M., Paihogios, J. P., Heck, W. L., Frick, S. J., & Wasserman, J. F. (1995). Fracture patterns of human cadaver long bones. *International Research Counsel on the Biomechanics of Impact* (pp. 155–169).

Landin, L., & Nilsson, B. E. (1983). Bone mineral content in children with fractures. *Clinical Orthopaedics and Related Research*, *178*, 292–296.

Leichter, I., Margulies, J. Y., Weinreb, A., Mizrahl, J., Robin, G. C., Conforty, B., Makin, M., & Bloch, B. (1982). The relationship between bone density, mineral content, and mechanical strength in the femoral neck. *Clinical Orthopaedics and Related Research*, *163*, 272–281.

Leventhal, J. M., Thomas, S. A., Rosenfield, N. S., & Markowitz, R. I. (1993). Fractures in young children. Distinguishing child abuse from unintentional injuries. *American Journal of Diseases of Children*, *147*, 87–92.

Levine, R. S. (2002). Injury to the extremities. In A. M. Nahum & J. W. Melvin (Eds.), *Accidental injury* (pp. 491–522). New York: Springer-Verlag.

Mabrey, J. D., & Fitch, R. D. (1989). Plastic deformation in pediatric fractures: Mechanism and treatment. *Journal of Pediatric Orthopaedics*, *9*, 310–314.

Manzoni, P., Brambilla, P., Pietrobelli, A., Beccaria, L., Bianchessi, A., Mora, S., & Chiumello, G. (1996). Influence of body composition on bone mineral content in children and adolescents. *American Journal of Clinical Nutrition*, *64*, 603–607.

Martin, R. B. (1991). Determinants of the mechanical properties of bones. *Journal of Biomechanics*, *24*, 79–88.

Mather, B. S. (1967). Correlations between strength and other properties of long bones. *The Journal of Trauma*, *7*, 633–638.

Mazess, R. B. (1996). Bone densitometry using dual-energy X-ray absorptiometry. *Current Opinion in Orthopedics*, *7*, 5–11.

McClelland, C. Q., & Heiple, K. G. (1982). Fractures in the first year of life. A diagnostic dilemma? *American Journal of Diseases of the Child*, *136*, 26–29.

Morild, I., Gjerdet, N. R., & Giertsen, J. C. (1993). Bone strength in infants. *Forensic Science International*, *60*, 111–119.

Mourtada, F. A., Beck, T. J., Hauser, D. L., Ruff, C. B., & Bao, G. (1996). Curved beam model of the proximal femur for estimating stress using dual-energy X-ray absorptiometry derived structural geometry. *Journal of Orthopaedic Research*, *14*, 483–492.

Mow, V. C., & Hayes, W. C. (1991). *Basic orthopaedic biomechanics* (p. 112). New York: Raven Press.

Newman, J. A. (2002). Biomechanics of head trauma: Head protection. In A. M. Nahum & J. W. Melvin (Eds.), *Accidental injury* (pp. 303–323). New York: Springer-Verlag.

Nigg, B. M., & Herzog, W. (Eds.). (1999). *Biomechanics of the musculo-skeletal system* (2nd ed.). Chichester: John Wiley & Sons, Ltd.

Ogden, J. A. (2000). *Skeletal injury in the child* (3rd ed.). New York: Springer-Verlag.

Ogle, G. D., Allen, J. R., Humphries, I. R. J., Lu, P. W., Briody, J. N., Morley, K., Howman-Giles, R., & Cowell, C. T. (1995). Body-composition assessment by dual-energy X-ray absorptiometry in subjects aged 4–26 y. *American Journal of Clinical Nutrition*, *61*, 746–753.

O'Neill, J. A., Jr., Meacham, W. F., Griffin, P. P., & Sawyers, J. L. (1973). Patterns of injury in the battered child syndrome. *The Journal of Trauma*, *13*, 332–339.

Osier, L. K., Marks, S. C., Jr., & Kleinman, P. K. (1993). Metaphyseal extensions of hypertrophied chondrocytes in abused infants indicate healing fractures. *Journal of Pediatric Orthopaedics*, *13*, 249–254.

Panjabi, M. M., & White, III., A. A. (2001). *Biomechanics in the musculoskeletal system*. Philadelphia: Churchill Livingstone.

Pathria, M. N. (2002). Radiologic analysis of trauma. In A. M. Nahum & J. W. Melvin (Eds.), *Accidental injury* (pp. 103–120). New York: Springer-Verlag.

Pierce, M. C., Bertocci, G., Aguel, F., Deemer, E., Janosky, J., Boal, D., Moreland, M., Herr, S., Zuckerbraun, N., & Garcia, S. (2003). Femur fractures resulting from stair falls: An injury plausibility model [abstract]. *Pediatric Research*, *53*, 1948A.

Pierce, M. C., Bertocci, G. E., Janosky, J., Hani, J., Deemer, E., Aguel, F., & Moreland, M. (2000). Biomechanical and biological characterization of accidental femoral fractures in young children. Model development to aid in differentiating accidental and non-accidental trauma [abstract]. *Pediatric Research*, *47*, 115A.

Pierce, M. C., Bertocci, G., Kambic, H., & Valdevit, A. (2001). *Prediction of pediatric bone strength based on radiographic geometry and DEXA: An in vitro porcine model for infant femur fracture* [abstract]. Rhodes, Greece: Orthopaedic Research Societies of the U.S.A., Canada, Europe, and Japan.

Pierce, M. C., Bertocci, G., Vogeley, E., Deemer, E., Aguel, F., & Szczepanski, J. (2001). Resultant fracture types from known injury mechanisms: Development of a database for identification of potential child abuse fractures [abstract]. *Pediatric Research*, *49*, 92A.

Pierce, M. C., Valdevit, A., Anderson, L., Inoue, N., & Hauser, D. L. (2000). Biomechanical evaluation of dual-energy X-ray absorptiometry for predicting fracture loads of the infant femur for injury investigation: An in vitro porcine model. *Journal of Orthopaedic Trauma*, *14*, 571–576.

Rex, C., & Kay, P. R. (2000). Features of femoral fractures in nonaccidental injury. *Journal of Pediatric Orthopaedics*, *20*, 411–413.

Rivara, F. P., Kamitsuka, M. D., & Quan, L. (1988). Injuries to children younger than 1 year of age. *Pediatrics*, *81*, 93–97.

Rogers L. F. (1992). *Radiology of skeletal trauma* (Vol. 1, 2nd ed.). New York: Churchill Livingstone Inc.

Sammarco, J., Burstein, A., Davis, W., & Frankel, V. (1991). The biomechanics of torsional fractures: The effect of loading on ultimate properties. *Journal of Biomechanics*, *4*, 113.

Scherl, S. A., Miller, L., Lively, N., Russinoff, S., Sullivan, C. M., & Tornetta, P., III. (2000). Accidental and nonaccidental femur fractures in children. *Clinical Orthopaedics and Related Research*, *376*, 96–105.

Schwend, R. M., Werth, C., & Johnston, A. (2000). Femur shaft fractures in toddlers and young children: Rarely from child abuse. *Journal of Pediatric Orthopaedics*, *20*, 475–481.

Shaw, B. A., Murphy, K. M., Shaw, A., Oppenheim, W. L., & Myracle, M. R. (1997). Humerus shaft fractures in young children accident or abuse? *Journal of Pediatric Orthopaedics*, *17*, 293–297.

Skellern, C., Wood, D. O., Murphy, A., & Crawford, M. (2000). Non-accidental fractures in infants: Risk of further abuse. *Journal Paediatric Child Health*, *36*, 590–592.

Southall, D. P., Plunkett, M. C. B., Banks, M. W., Falkov, A. F., & Samuels, M. P. (1997). Covert video recordings of life-threatening child abuse: Lessons for child protection. *Pediatrics*, *100*, 735–760.

Southard, R. N., Morris, J. D., Mahan, J. D., Hayes, J. R., Torch, M. A., Sommer, A., & Zipf, W. B. (1991). Bone mass in healthy children: Measurement with quantitative DXA. *Radiology*, *179*, 735–738.

Strait, R. T., Siegel, R. M., & Shapiro, R. A. (1995). Humeral fractures without obvious etiologies in children less than 3 years of age: When is it abuse? *Pediatrics*, *96*, 667–671.

Strømsøe, K., Hoiseth, A., Alho, A., & Kok, W. L. (1995). Bending strength of the femur in relation to non-invasive bone mineral assessment. *Journal of Biomechanics*, *28*, 857–861.

Taylor, M. T., Banerjee, B., & Alpar, E. K. (1994). Injuries associated with a fractured shaft of the femur. *Injury*, *25*, 185–187.

Thomas, S. A., Rosenfield, N. S., Leventhal, J. M., & Markowitz, R. I. (1991). Long-bone fractures in young children: Distinguishing accidental injuries from child abuse. *Pediatrics*, *88*, 471–476.

Turner, C. H., & Burr, D. B. (1993). Basic biomechanical measurements of bone: A tutorial. *Bone*, *14*, 595–608.

Venkataraman, P. S., & Ahluwalia, B. W. (1992). Total bone mineral content and body composition by X-ray densitometry in newborns. *Pediatrics*, *90*, 767–769.