The Pathology of Acute Chondro-Osseous Injury in the Child

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Skeletal tissues from children sustaining acute skeletal trauma were analyzed with detailed radiologic and histologic techniques to assess the failure patterns of the developing skeleton. In the physis- and epiphysis-specific fracture propagation varied, usually going through the portion of the hypertrophic zone adjacent to the metaphysis. However, the physeal fracture in types 1 and 2 sometimes involved the germinal zone. There may also be microscopic propagation at oblique angles from the primary fracture plane, splitting cell columns apart longitudinally. The cartilage canals supplying the germinal zone appear to be "weak" areas into which the fracture may propagate, especially in infancy. Incomplete type 1 physeal fractures, which cannot be detected by routine radiography, may occur. Types 1, 2, and 4 physeal injuries may be comminuted. In type 3 injuries, discrete segments of physis that include the germinal zone may "adhere" to the metaphysis, separating the cells from their normal vascularity. In types 2 and 3, comminution may occur at the site of fracture redirection from the physis. Direct type 5 crushing of the physeal germinal zone does not occur, even in the presence of significant pressure-related changes within other areas of the epiphysis. Type 7 separation between cartilage and bone at any chondro-osseous epiphyseal interface may occur, but is similarly impossible to diagnose radiographically.

In the metaphysis torus, fractures result from plastic deformation of the cortex, coupled with a partial microfracturing that may be difficult to visualize with clinical radiography. Some of the energy absorption may also be transmitted to the physis, causing metaphyseal hemorrhage adjacent to the growth plate and variable microscopic damage within the physis.

In the diaphysis, the greenstick fracture is associated with longitudinal tensile failure through the developing osteons of the "intact" cortex. The inability of these failure patterns to "narrow" after the fracture force dissipates is the probable cause of retained bowing (plastic deformation). In both torus and greenstick fractures, the fractured bone ends show micro-splitting through the osteoid seams.

In the diaphysis, metaphysis, and epiphyseal ossification center there may be areas of focal hemorrhage and microfracture that correlate with the reported MRI phenomenon of "bone bruising." Again, such injury cannot be diagnosed during routine radiography.

INTRODUCTION

The literature concerning the actual histology of human chondro-osseous failure prior to skeletal maturity is virtually nonexistent. Smith, Geist, and Cooperman [1] described a type 1 growth mechanism fracture of the distal tibia in a single specimen from a 9-year-old boy who sustained a traumatic amputation. Johnson and Jones [2] assessed biopsy material obtained during open reduction of physeal fractures; this material, however, was never formally published after the oral presentation. Kleinman, Marks, and Blackbourne [3] described pathologic physeal findings in several infants with

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"corner" fractures following fatal child abuse. There are no other published reports of the detailed histopathology of actual human physeal fractures that occurred during spontaneous injury. Similarly, while bowing, torus, and greenstick fractures are extremely common failure mechanisms to the developing, maturing skeleton in the child, there are no published reports of the histopathology of these acute injury patterns in the human.

Aufdermaur [4] removed spines from children and adolescents fatally injured in automobile accidents. He was able to define the histologic patterns of physeal failure in the spine and demonstrated that occult (i.e., radiologically invisible) injuries were much more frequent than realized, and were probably the cause of spinal cord injury without radiographic abnormality (SCIWORA).

One of the senior authors (JAO) and others have assessed the histopathology of chronic trauma prior to skeletal maturity. In particular, we have analyzed histologic material from patients with bipartite patella, accessory navicular, os trigonum, bipartite sesamoid and accessory malleolar ossification to better understand the actual injury and to improve treatment [5–10]. We have also looked at some pathologic material often fortuitously obtained following injury [11–19] and after experimental injury [20–23].

In an effort to similarly understand the biologic responses of the different components of the developing human skeleton (diaphysis, metaphysis, physis, and epiphysis) to trauma, a more extensive collection of tissues from acutely injured children has been assimilated for study.

**MATERIALS AND METHODS**

Over the past 20 years, the authors have collected samples of skeletal tissue from acute injuries to the developing skeleton. The trauma material has included specimens from non-fatal accidents and has especially concentrated on specimens from traumatic avulsions at various limb levels. Additionally, small fragments were sometimes removed during open reduction of comminuted growth mechanism fractures, but only if the fragment was felt to be unnecessary for, or detrimental to satisfactory healing. Altogether, specimen material from 57 patients sustaining acute injury were available for study. The specimens ranged in size from small fragments to a complete limb due to acute scapulothoracic dissociation. The patients ranged in age from 7 months to 15 years.

Each specimen was photographed and radiographed. Whenever possible, CT scans, 3-D CT reconstruction, and MRI scans were obtained on significantly-sized traumatic amputation specimens to further delineate the various skeletal and soft tissue changes. After fixation specimens were sagittally or coronally sectioned in slabs approximately 0.5 to 1 cm thick, and again photographed and radiographed. Each slab specimen was then decalcified and embedded for selective sectioning and staining for histologic analysis.

Dr. Cooperman [1] kindly supplied an original tissue block, as well as additional unembedded material of the rest of the injured distal tibia, for further processing and comparison.

**RESULTS**

*Type 1*

The fracture line propagates principally but variably along and through the calcified hypertrophic zone or the primary spongiosa (Figure 1). In no instance was there smooth, invariable propagation completely within a single cellular zone. Instead, there was undulation, usually into the non-calcified portion of the physis or into the primary spongiosa. Fractures tended to follow physeal contours. While the main direction of fracture was transverse, there were longitudinal extensions that split the extracellular matrix between
Figure 1. A. A type 1 physeal separation is evident at the junction of the primary spongiosa and the hypertrophic cells (double ended arrows). The separation also propagates within the hypertrophic zone (open arrows). B. The primary fracture is directed transversely across the hypertrophic zone (solid arrows). However, there are longitudinal micro-separations between cell column clusters (small open arrows). Note the difference in the hypertrophic zone histology between 1A and 1B. Such differences among physes undoubtedly affect the pattern of fracture propagation.

cell columns. There also were smaller, parallel fractures that tended to separate the hypertrophic zone from the germinal zone. Microscopic damage was infrequently evident in the secondary ossification center adjacent to the physeal fracture. Small islands of physis (hypertrophic cells and primary spongiosa) were sometimes present as free fragments (Figure 2). The blood vessels supplying the germinal zone, at least in the infants, appeared to act as "stress risers", allowing longitudinal extension into the cartilage canal and potentially damaging the circulation to the physeal chondrons. In rare instances the line of separation extended through the germinal zone.

In older patients, a similar fracture pattern was evident. However, there was a natural loss of cell columns as the region was beginning spontaneous epiphysiodesis. The zone of separation was between the former hypertrophic zone and the transversely oriented, thickened subchondral plate of the primary spongiosa.

In 4 instances, unsuspected type 1 incomplete physeal fractures were found (Figure 3). These injuries principally propagated through the junction between columnar forma-
Ogden et al.: Growth mechanism of injuries

Figure 2. Separation through the "upper" hypertrophic zone, with small comminuted fragments (arrow) of the physis. The metaphyseal bone and "lower" hypertrophic zone are at the top, while the epiphysis is below.

tion and hypertrophy of the columnar cells (Figure 4). There were long streaks of separation as well as small foci, often only involving two or three cell columns. Two of these fractures propagated to the periphery (zone of Ranvier), but did not disrupt the periosteum (Figure 3). These fractures appeared to be intrinsically stable.

Type 2

The only acute type 2 injury available for study showed the main fracture propagation turning into the metaphysis. However, there was microscopic transverse extension beyond the "turning point", including some fragmentation into the germinal zone. If this transverse extension had continued, the metaphyseal fragment might have come completely separate.

Type 3

Type 3 injuries followed the pattern of transverse fracture propagation followed by extension into the epiphysis. The basic fracture line was similar to that described for the types 1 and 2 injuries, namely through the hypertrophic zone. However, segments of physis, especially the germinal zone, remained attached to the metaphysis (Figure 5). In a specimen of the distal humerus (lateral condylar fracture), this separated, devascularized segment was extensive.

Type 4

The typical longitudinal separation across the physis, ossification center, and articular cartilage was evident (Figure 6). However, these both showed transverse comminution and fracture propagation where the injury traversed the physis. In one case, the transverse propagation was on the epiphyseal side of the physis, effectively devascularizing this segment of the physis.

Type 5

A below knee amputation specimen from a boy with congenital sensory neuropathy was specifically examined for evidence of type 5 injury. The surgery was undertaken because of progressive destruction of the hind foot. The distal tibia had an irregular, dam-
Figure 3A. Undisplaced "occult" type 1 fracture of the proximal tibial physis (straight arrows). The periosteal-perichondrial tissue gradient was intact grossly and histologically. Note the chondro-osseous fracture of the tibial spine (curved arrow). There are also focal areas of bone bruising in the tibial metaphysis and fibular epiphyseal ossification center. B. Histologic appearance of "occult" type 1 physeal fracture (open arrows) with extension up to the zone of Ranvier (solid arrow). However, the periosteum is intact, still attached to the perichondrium and adherent to the recently formed cortical bone.

Aged articular surface. Microscopically, the cartilage had formed uncharacteristic spherical clones in the region between ossification center and articular surface (this is a pressure response; see DISCUSSION). In contrast, with the exception of one small focal area, the distal tibial growth plate showed virtually no evidence of crushing (compression) damage. None of the metatarsals or phalanges from this specimen had growth plate damage.

Type 7

The developing ossification center may be fragmented either acutely or chronically as a type 7 injury. Figure 6 shows occult chondro-osseous separation of the distal tibia and fibula following traumatic injury. A significant fibular injury, not evident radiographically accompanied the Type 4 distal tibial fracture. This fibular injury was a combination of avulsion of the distal tip at the chondro-osseous interface, further propagation between the bone and cartilage along the inner side of the distal fibular ossification center, and
Figure 4. Histologic appearance of an "occult", undisplaced type 1 physeal fracture of the distal radius. This is a transverse fracture (arrows) propagating in the central physis, with an area of comminution and fracture extension into the primary spongiosa.

Figure 5. Incomplete type 3 fracture (open arrows) of the lateral condyle. Note the separation of areas of the physis from the epiphysis (solid arrows). This included all zones of the physis.

final propagation across the epiphyseal cartilage toward the lateral margin of the physis.

Occult distant injury

In eight instances, mid-diaphyseal fractures were associated with hemorrhage and trabecular metaphyseal or physeal injury several cm away from the grossly evident injury. In none of these cases, even in a retrospective review of clinical and specimen radiographs, could the distant extent of injury be fully appreciated. For example, Figure 7 shows the distal tibia of a 9-year-old girl injured by a car. She had a mid-diaphyseal fracture of the tibia and popliteal artery and femoral artery lesions. She was initially placed in an external fixator but subsequently underwent a BK amputation. There was trabecular hemorrhage bone bruising in the metaphysis. More importantly, the mid-portion of the physis was obviously abnormal compared to the peripheral regions.

Greenstick ulnar and radial torus fractures were also associated with distant, occult, incomplete 1 growth mechanism fractures of the physes (see Figure 4).
Type 4 Injury extending through the distal tibial physis (open arrows). Also evident is a type 7 injury (solid arrows) of the distal fibular epiphysis (see text for details).

Figure 6. Type 4 Injury extending through the distal tibial physis (open arrows). Also evident is a type 7 injury (solid arrows) of the distal fibular epiphysis (see text for details).

Figure 7. Distal tibia showing focal hemorrhage in the central metaphysis (white arrows), with a loss of the normal white (opaque) appearance of the entire central physis (black arrows) as compared to the more lateral and medial regions.

Bone bruises

Figure 8 shows areas of bone bruising, comprised of microtrabecular damage and hemorrhage involving the distal tibia and fibula of a patient who sustained a traumatic amputation and a diaphyseal fracture several inches proximal to the "bruising." These focal areas of hemorrhage would probably be evident by MRI.

Torus fracture

Torus injuries were present in four specimens (Figure 9). Morphologically, the buckling of the cortex extended incompletely around the cortical periphery. The periosteum was intact, although subperiosteal hemorrhage and variable elevation were evident. In all instances, the fracture was grossly stable to manipulation. Sectioning revealed that the cortex was damaged in the region of transition from fenestrated metaphyseal cortex to
Figure 8. Bone bruising or areas of focal hemorrhage and microtrauma. Multiple foci of hemorrhage (dark areas in the trabecular bone) are evident along the metaphysis, as well as in the medial malleolar and most lateral portions of the epiphyseal ossification center (arrows).

Figure 9. Torus fracture of the distal radius. Note how curvilinear bowing of the cortex (straight arrows) ends in cortical microdisruption (curved arrow).

osteon bone. The disruption did not extend all the way around the periphery. As described in the previous section, there was evidence of concomitant occult injury in the metaphyseal region adjacent to the physis.

The cortical fracture appeared to propagate irregularly through vascular foramina, as well as splitting longitudinally along osteoid seams. All cases demonstrated variable degrees of actual curvature of the cortex, more evident on the compression side than the tension side. Closer examination revealed that the compression buckling really became
possible because of microscopic tensile failure of the outer portion of the cortex and the splitting of osteoid seams, as well as distortion of the seams. On the opposite cortex there was microscopic failure evident as hemorrhage and splitting of developing osteons.

**Periosteal injury**

The torus fractures particularly allowed demonstration of the highly variable stripping of the periosteum. In most instances the periosteum stripped, as a full thickness, away from the underlying bone. Beyond the macroseparation, however, there were areas of further incomplete microseparation. In one instance the periosteum was separated within its layers, leaving the osteogenic inner layer (cambium) still attached to the metaphyseal cortex.

**Greenstick fracture (plastic deformation)**

There were two examples of retained bowing in the diaphyseal cortex. In the first example the ulna had an oblique complete fracture. However, on the "compression" side, the longer segment of cortex retained an accentuated permanent deformation. The bone probably failed initially as a greenstick injury and then subsequently failed completely, splitting from the inner to outer cortex. This is compatible with the splitting evident longitudinally. Another specimen had a greenstick injury of the ulna associated with a dislocation of the radial head (Monteggia injury). Stress radiography accentuated the injury, showing that there is continued, longitudinally directed tensile failure on the endosteal portion of the "compression" side. The splitting apart and subsequent reversal of deformity may entrap soft tissue. Microscopically there is a combination of splitting and widening of the developing Haversian systems, as well as compressive distortion along osteoid seams (micro-plate tectonics).

**DISCUSSION**

Based on experimental studies in small mammals (rats, rabbits) Salter and Harris [24], among others, postulated that all physeal fractures consistently occurred through the third layer (i.e., the hypertrophic cells) at approximately the junction of the calcified and uncalcified matrices, and that the "most striking microscopic feature of experimental separation is the constancy of the plane of cleavage." While this may hold for small physis and epiphyses in animals that attain skeletal maturity within few months to a year following birth, the observations may or may not hold true for failure patterns in the human (or any animal, especially primates) with a more extended period of skeletal maturation, or when there is significant undulation of the physis in any skeletally immature individual. Studies have shown that even in the experimental animal the extent of skeletal maturation and the direction and rate of application of deforming forces may affect physeal fracture patterns [23–25]. In particular, the proliferative layer is most susceptible to tensile forces, whereas the zone of calcification and the metaphyseal bone appear to be more vulnerable to compressive forces. When the shearing force is dominant, the tendency is to type 1 physeal injury. An increase in the compression component increases the likelihood of a type 2 fracture with the typical metaphyseal fragment [20, 23, 24].

Because of the de-emphasis on physeal long-term damage and the difficulty in classifying other patterns of injury affecting the various growth mechanisms of bone, one of the authors (JAO) has developed a modified classification system of growth mechanism injuries [25–27]. This system, in part, is based upon the various specimens and histologic descriptions included in this study. The necessity for trying to create anatomic bases for fracture concepts was emphasized by Kleinman et al. [3] in that they felt the removal of epiphyseal/metaphyseal segments at autopsy was justified in suspected cases of child
abuse to look for occult injuries that would support the diagnosis of multiply inflicted injury.

Gomes et al. [28] duplicated types 1 through four fractures in rats. They found that fracture propagation in type 1 and 2 injuries could involve the germinal zones. In our studies, the human type 1 injury fracture propagation is similar to that described in these small experimental animals. However, there is considerable variation that seems to relate to the particular physis being examined and the degree of maturation of this physis. The height of the cell columns, both the zone of column formation (uncalcified matrix) and the zone of hypertrophy (calcified matrix) varies, and reflects the degree of physiologic activity. The principle zone of fracture was through the junction of column formation and hypertrophy, with propagation into the primary spongiosa. Undulation did not affect the level of propagation, since the fracture usually turned to follow major contour changes.

In older patients, especially those approaching maturity, the subchondral bone of the metaphysis thickens and cellular (cartilage) hypertrophy decreases. The zone of separation is distinctly between cartilage and bone.

A significant finding was that smaller fractures, no matter which physal fracture pattern, often extended from the main fracture plane at oblique angles, propagating between cell columns to split them longitudinally. Further, such extensions were associated with smaller transverse fractures extending across the less mature levels of the hypertrophic zone, separating it from the germinal zone. In specimens from infants and toddlers the longitudinal fracture extension went toward and into epiphysial vessels within the germinal zone. In two instances, the fracture propagated into the germinal zone, separating it from the undifferentiated portions of the physis. The observations of germinal zone involvement and fracture extension into the E-vessel cartilage canals (which appeared to act as microscopic stress risers) might elucidate why there is an infrequent association of growth slowdown and arrest with type 1 physal fractures.

Another extremely important finding of this study was the presence of "occult" type 1 physal fractures. Each of these microinjuries was associated with a much more obvious injury of the bone. The line of separation was between the columnar and hypertrophic zones, with the separation being only a few cells wide. The transverse propagation varied, from only a few cell columns to larger expanses. The propagation did not always expand to the periphery, and when it did, the peristomeum was always intact. Thus, these fractures would have an inherent stability. Further, finding such comitant obvious metaphysial and diaphysial fractures and occult physal fractures may explain the cases reported in the literature in which there is "unexplained" growth arrest following shaft fractures [25].

These types of "occult" microfractures through the physis were described in experimental animals by Bright and co-workers [29]. They felt they represented the earliest stages of physal failure, preceding periosteal failure and displacement. Accordingly, it would appear that physal separation takes place within the substance of the physis and then extends toward the periphery. If the periosteum subsequently fails, the physis may then be grossly separated. This is in accord with the observation that an intact periosteum significantly strengthened the physis and provided a resistance to complete failure [25, 30]. The fact that these occult physal fractures may occur reinforces the need to make a clinical judgement of physal injury even if the radiograph does not show an obvious injury [31–33]. These occult injuries also explain the unexpected examples of premature cessation of growth in a bone or extremity following a seemingly innocuous shaft fracture [25].

Experimental fractures of the proximal femur in stillborns showed unexpected longitudinal splitting of the physis, with these side-directed fractures extending into the epiphysis, even to the point of creating small type 3 or 4 physal fractures [21]. This type of
peripheral disruption may explain the long-term growth impairment (humerus varus, genu valgum, etc.) often seen after physeal fracture at birth or in the first few months of life [25].

In the type 2 physeal fracture the transverse propagation is redirected into the metaphysis to create a variable sized fragment. While only one acute specimen was available for study, there was further microscopic fracture extension, including germinal zone involvement. This area of redirection of the main fracture line may thus be associated with microcomminution, which may be a factor in those cases that develop a growth arrest following a type 2 injury.

In type 3 injuries, variable sized foci of physis, including the germinal zone, may remain attached to the metaphysis. This effectively removes the cells from the E-vessel supply necessary for continued growth within the physis. Even with accurate anatomic restoration, this type of physeal fracture would lead to chronic alteration or disruption of growth. This observation would also explain the increased risk of injury as a vascular related function.

The unique opportunity to observe the potential effects of excessive pressure on the physis confirmed the concept of Peterson and Burkhart that direct crushing of the germinal zone, as originally proposed by Salter and Harris, probably does not occur.[24, 34]. Previous experiments in the Yale Growth and Development and Orthopaedic laboratories also failed to cause germinal zone crushing [20]. The fact that this region was subjected to unusual chronic compression may be seen in the unique clone formation throughout the cartilage between the articular surface and secondary ossification center. This type of clone formation in cartilage in response to chronically altered compression has been described experimentally [35]. The only small focus of physeal abnormality resembled the focal changes that lead to striations within the physis and metaphysis. This appearance is certainly compatible with epiphyseal ischemia, as described by Trueta [36].

The type 7 injury is confined to the epiphyseal ossification center and epiphysis, and does not routinely involve the physis. It may occur acutely, as an avulsion of a tibial spine, or chronically, as an Osgood-Schlatter lesion. In the latter, chronic tension is applied to the anterior portion of the developing tuberosity center, gradually pulling a segment away. The gap may fill in with fibrovascular inflammatory tissue, or develop a pseudarthrosis. Treatment of such a lesion as a fracture (acute or chronic) will usually result in asymptomatic relief. The Osgood-Schlatter lesion may also predispose to avulsion of the entire tuberosity, which usually occurs in adolescence [13, 25].

Ischemic necrosis is probably one of the major underlying factors of any growth abnormality or physeal bridging, especially in types 1, 2, and 5 injuries, and it probably interacts with direct bone apposition (metaphysis and ossification center) in types 3 and 4, 37, 38. Salter and Harris [24] felt that the "crux of the problem" was not mechanical damage to the physis, but whether the separation interferes with blood supply. Gomes et al. [28] felt vascular anastomosis across the physis preceded the formation of an osseous bridge. The vascular damage is to variable sized epiphyseal vessel regions, and may lead to either bridge formation or to slowdown of growth and endochondral ossification without discrete bridge formation. The ischemia causes the involved segment to cease growing, causing it to be "left behind" by the still functional growth plate.

The damaged vessel may or may not revascularize. Trabecular bone forms along the vessel and, over time, bridges between ossification center and metaphysis. Large gaps may be associated with considerable fibrovascular bridging which will eventually ossify. The damaged area cannot form a discrete bridge until the secondary ossification center extends to the damaged area. This would explain the delayed appearance of osseous bridges.
The transphyseal linear longitudinal striation, often described in the distal radius and ulna in the radiologic literature, may be a localized focus of ischemic damage. Trabecular bridging surrounds a core of abnormal physeal tissue, and often extends along this tissue into and across the physis to an epiphyseal vessel that exhibits loss of normal vascular morphology [19]. The trabecula do not, however, extend from the epiphyseal vessel to the secondary ossification center. Interestingly, if there are microscopic areas of damage in a region frequently injured in the child, are they a possible anatomic cause of ulnar plus and ulnar minus variants of the distal radius and ulna?

It is well recognized that generalized slowdown of longitudinal growth leads to increased transverse bone formation patterning in the juxaphyseal metaphysis. With the resumption of normal rates of longitudinal growth, the primary spongiosa again becomes oriented in line with the cell columns, leaving a transversely oriented "Harris growth slowdown line" [17, 25]. In contrast, these linear striations in the metaphysis and physis appear to be caused by focal areas of permanent physeal damage, with remnants of physeal cartilage "left behind" as the rest of the physis grows. Unlike the contained remnants of hypertrophic cartilage matrix in the primary spongiosa, these physeal remnants do not undergo remodeling and replacement. This may relate to a lack of the necessary antecedent calcification.

In multiple instances there were distant injuries separated from the radiographically obvious fractures within either the metaphysis or diaphysis. These distant injuries included metaphyseal hemorrhage and microscopic disruption of the primary spongiosa, as well as incomplete type 1 fractures of the physis separating the hypertrophic zone from the columnar zone.

Two important clinical correlations stem from these observations. First, the developing skeleton, because of its intrinsic elasticity and decreased brittleness compared to an adult's skeleton, may absorb fracture energy in multiple portions of the bone, not just the clinically (i.e., radiographically) evident fracture. Such distant energy absorption and microdisruption may explain the variable long-term consequences of shaft fractures, such as overgrowth or undergrowth.

Second, the occult type 1 physeal fractures were incomplete. This suggests that growth variations may also be related to concomitant undiagnosed physeal injury. Further, the occurrence of these incomplete fractures of the physis suggests occult physeal injury may occur in any patient. The clinical import should be obvious. If a child has an injury mechanism associated with pain and swelling around a physeal region, then occult physeal injury must be suspected if the radiograph is negative. The child should be treated with appropriate immobilization to prevent the stable, incomplete fracture from progressing to a complete, unstable injury. Perhaps selective quantitative bone scans in such cases will allow a more definitive diagnosis.

Bone bruising has recently been described in the diagnostic imaging literature as a phenomenon evident during magnetic resonance imaging of injured patients.[39, 40]. However, there has been absolutely no anatomic correlation of what constitutes such "bruising." The microscopic trabecular damage and hemorrhage seen in several specimens in this study certainly supports the concept that such bruising indeed has an anatomic injurious basis.

Our specimens have also allowed elucidation of the torus fracture mechanism. First, a segment of the cortex is completely disrupted on the compression side. Second, at the site of cortical bowing there is microscopic tensile failure of osteons close to the periosteal surface and compression distortion through osteoid seams at the endosteal surface. There are multiple microscopic disruptions throughout the focus of bowing. The opposite cortex shows tensile failure through osteons. The failure was through the trans-
tion region wherein the fenestrated metaphyseal cortex was merging with modeling from primary osteons. This physiologically active area undoubtedly represents a stress riser because of the differing biomechanical responses of these two zones [16].

The torus fracture is usually considered a stable fracture. This is corroborated by these specimens. However, they also show that there is incomplete cortical disruption that could propagate if the child is not treated. The examples of associated occult type 1 physeal injury also help explain the reported instances in the literature of growth impairment after seemingly innocuous metaphyseal fractures.

As is evident clinically, the periosteum strips away from the diaphysis and much of the metaphysis relatively easily. The separation may also occur between the various layers of the peristomeum, leaving segments of the highly osteogenic layer intact and still attached to the contiguous cortical bone. Obviously the full thickness separation explains the subperiosteal new bone formation. However, the observation that the osteogenic layer may not always strip away would explain the lack of subperiosteal new bone formation in many cases.

Greenstick fractures represent a transition between retained plastic deformation (bowing) and complete cortical disruption. Figure 9 shows that even when there is completion of a fracture, some of the cortex may still retain the plastic deformation. This potential for retained deformity may be a factor in certain angular changes that occur during fracture treatment. It also should be remembered when attempting open reduction of both bone forearm fractures in children.

In the greenstick fracture the significant observation was widening of the osteons to "increase" the relative cortical width, the microscopic splitting (tensile failure) of osteons close to the endosteal surface, macroscopic longitudinal splitting on the "intact" side, and microscopic compression disruption of osteons and osteoid seams near the periosteal surface. The pattern is one of progressive tensile splitting. Accordingly, should one complete these fractures by continuing or reversing the direction of deformity?

Mabrey and Fitch [41] reviewed concepts of plastic deformation, but without any supporting histologic data. They stated there is a "difference of opinion among some authors as to its exact mechanism." Osteons surrounded by cement lines are potential sites of weakness [38, 42, 43]. This certainly was the failure pattern in what is observed in this study. Lamellae within each osteon comprise the second composition levels [44, 45]. These also may separate. These observations thus support mechanical concepts of failure that have been hypothesized by others [46].

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