Disturbance of skeletal growth occurs often in children and results in considerable lifelong disability. When acquired, it is most frequently post-traumatic but may also be caused by other insults, such as infection, ischemia, tumoral lesions, and radiation. The complications of growth disturbance (leg length discrepancy, angular deformity, and altered joint mechanics) cause significant morbidity. MR imaging, with its ability to depict the cartilaginous structures of developing bones, has become the modality of choice for evaluating children with growth disorders and directing surgical management. An understanding of normal enchondral ossification and the anatomy of the ends of growing bones is essential for appreciation of the MR imaging findings in such patients.

ENCHONDRAL OSSIFICATION

The developing ends of long bones can be divided into segments based on their tissue composition and contribution to growth: cartilaginous epiphyseal unit, osseous metaphysis, and fibrous perichondrium.

The epiphyseal unit includes the epiphysis and the physis (or growth plate) and contributes most to the longitudinal growth of the bone. The physis is a specialized part of the epiphysis divided into zones based on histology and function (Fig. 1). The region of the physis adjacent to the epiphysis is the germinatal, or reserve, zone, which contains abundant matrix and poorly organized chondrocytes functioning as the stem cells of the physis. The proliferative zone is comprised of columns of flattened chondrocytes that are rapidly dividing; this cellular proliferation results in increased length of the bone. In the hypertrophic zone, the chondrocytes enlarge and vacuolate. The zone of provisional calcification, the juxtametaphyseal portion of the hypertrophic zone, is the transition between bone and cartilage. It is here that the matrix is invaded by the metaphyseal vessels and becomes mineralized. Overall physeal thickness remains constant throughout growth until maturity because of the exquisite balance between the rates of cartilage production in the proliferative zone and chondrocyte removal by mineralization in the hypertrophic zone.

Prenatally, the epiphysis is entirely cartilaginous. With time, the hyaline cartilage is converted to bone beginning centrally at the secondary center of ossification (SOC). The SOC is surrounded by a spherical growth plate that undergoes enchondral ossification analogous to that of the primary physis. The SOC is initially spherical, but with growth it conforms to the contours of the epiphysis and becomes more hemispherical and eventually abuts the physis.

The metaphysis is primarily osseous, and...
it contributes to the regulation of enchondral ossification. Another important role of the metaphysis is structured removal of chondrocytes and osteocytes at the periphery. This metaphyseal remodeling leads to the normal funnel shape of the ends of bones.

The perichondrial structures are fibrous and located at the periphery of the physis. The perichondrial groove of Ranvier is a wedge-shaped band of osteoblasts, chondrocytes, and fibrous cells at the periphery of the germinal zone, which is responsible for latitudinal growth of the physis (Fig. 1). The perichondrial ring of LaCroix is a fibrous band that surrounds the groove of Ranvier and is continuous with the periosteum of the metaphysis. The ring provides mechanical support to the otherwise weak chondro-osseous junction.

VASCULAR ANATOMY

The epiphyseal artery supplies the epiphysis and the SOC. Its branches course through small canals to the germinal and upper proliferative zones of the physis. Early in life, some of these canals extend from the epiphysis into the metaphysis allowing direct extension of metaphyseal pathology across the physis (Fig. 2). As the SOC enlarges, however, the vascular canals converge and the transphyseal vessels atrophy by the first year of life.

The metaphysis has a rich blood supply from the nutrient artery (80%) and the metaphyseal artery (20%). The nutrient artery nurtures the central metaphysis, whereas the metaphyseal vessels supply the periphery. The nutrient and metaphyseal arteries loop before reaching the zone of provisional calcification.
fication, leaving the hypertrophic zone of the physis avascular. The physis has a dual vascularity, with each component contributing uniquely to enchondral ossification. Whereas the epiphyseal vessels nurture the chondrocytes of the physis, the metaphyseal vessels regulate enchondral ossification by invading the physis and triggering cell death and bone synthesis.

The perichondrial artery supplies the peripheral perichondrial groove of Ranvier and ring of LaCroix. Small branches also provide vascularity to the periphery of the growth plate. The tenuous vascular supply to the central portion of the growth plate makes it particularly vulnerable. This explains the cupping deformity seen following severe ischemic states, such as meningococcemia (Fig. 3).4

**MECHANISMS OF GROWTH DISTURBANCE**

The most widely described type of growth disturbance is physeal arrest with bone bridge formation across the physis. Additionally, physeal dysfunction without bridge formation occurs with resultant physeal irregularity or wid-

![Figure 3. Bilateral distal tibial growth arrest following meningococcemia. A, Anteroposterior (AP) radiograph in an 8-year-old boy who had meningococcemia as an infant. There is central growth arrest and metaphyseal cupping caused by a bony bridge. B, Coronal gradient-recalled echo imaging of both ankles shows that the high signal intensity physes of both distal tibias are interrupted by central bony bridges (arrows).](image-url)
en. Epiphyseal growth disruption generally leads to a small SOC with abnormal contour or fragmentation. Disturbance of metaphyseal growth disrupts remodeling with loss of the normal funnel shape. Membranous growth impairment resulting from injury to the perichondrial groove limits latitudinal growth.

Direct Physeal Injury

The mechanisms of growth disturbance include direct physeal injury, epiphyseal injury, and metaphyseal injury (Fig. 4). Direct physeal insult is usually traumatic, but may also occur with metaphyseal infections and tumoral lesions, such as bone cysts and enchondromas, which breach the physis. Fracture across the physis, especially if vertical as in Salter-Harris type 4 injuries, permits transphyseal vascular communication between the epiphysis and metaphysis. Osteoprogenitor cells accompany the vessels and deposit bone leading to a bridge across the physis. Physeal fractures are discussed in detail later in this article.

Indirect Physeal Injury: Epiphyseal

Epiphyseal injury leads to two types of growth disturbance. Compromise of the epiphyseal vascularity causes focal ischemia of the physeal germinal and proliferative zones. This chondrocyte death may result in bony bridge formation. Alternatively, the growth may cease in the ischemic portion of the physis but continue in the unaffected areas. The growth plate loses its discoid shape and the area of slowed growth curves toward the metaphysis with resultant angular deformity. Additionally, epiphyseal injury and ischemia lead to decreased epiphyseal size and diminished signal intensity of the fatty marrow of the SOC on T1-weighted MR images. Growth disturbance caused by epiphyseal ischemia may be post-traumatic, but it is more common in patients with developmental dysplasia of the hip (DDH) following abduction therapy, Legg-Calvé-Perthes disease, and ischemia caused by infection and radiation (Fig. 5).

Indirect Physeal Injury: Metaphyseal

The third mechanism of growth disturbance is metaphyseal injury, which unlike

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**Figure 4.** Mechanisms of injury leading to growth disturbance. Epiphyseal injury (A) leads to physeal ischemia and physeal disorganization or bridge formation. Acute physeal injury (B) results in transphyseal vascular communication and subsequent bridging. Repetitive physeal injury (C) can cause bridging or physeal widening. Metaphyseal injury (D) perturbs endochondral ossification, resulting in diffuse or focal cartilage remnants in the metaphysis.
Figure 5. Proximal femoral epiphyseal ischemia leading to growth arrest. A, Axial fat-suppressed fast spin echo T2-weighted images of an 11-day-old boy obtained with the hips in flexion shows that there is effusion and posterior subluxation of the right hip. B, Coronal gadolinium-enhanced fat-suppressed T1-weighted image demonstrates decreased enhancement of the right femoral head (arrow), which contrasts with the normal enhancement of the epiphyseal vascular canals on the left. C, AP radiograph of the hip 10 months later shows that the right physis is obliterated indicating early growth arrest.

Epiphyseal injury does not usually lead to a transphyseal bridge because the metaphysis does not contribute to the physisal blood supply. Injury to the metaphyseal vascularity has no effect on chondrogenesis or chondrocyte maturation, but rather blocks enchondral ossification, the final conversion of cartilage to bone. Disruption of the balance between chondrocyte growth and removal by mineralization results in physisal thickening in the region of metaphyseal injury. As growth continues, the physis migrates from the site of injury and cartilage remains in the metaphysis. This persistent metaphyseal cartilage may take the form of a tongue perpendicular to the physis or a broad band extending from the physis (Fig. 6). With reconstitution of metaphyseal blood supply, the cartilage remnants are gradually ossified. The normal funnel-shape may be lost because of altered remodeling in the area of injury (Fig. 7). Experimental injury to the periphery of the metaphysis has lead to osteochondroma formation, especially when the periosteum is involved. Metaphyseal ischemia is likely responsible for the persistent metaphyseal cartilage in patients with meningococcemia, leukemia, disseminated intravascular coagulation, and prior radiation therapy. Repetitive metaphyseal injury also explains the physisal widening seen in some athletes, especially wrists of gymnasts, and patients with myelodysplasia.

PHYSEAL FRACTURES AND GROWTH ARREST

Approximately 15% of all pediatric fractures involve the physis and 15% of those lead to growth arrest requiring surgical management. The phalanges and the distal radius are the most common sites of physisal fracture but infrequently result in growth arrest. Conversely, the distal femur and proximal tibia are infrequent sites of physisal fracture (1.4% and 0.8%, respectively) with disproportionately high incidences of post-traumatic bridge formation (35% and 16%, respectively). The most frequent site of post-traumatic bridge formation is the distal tibia. Post-traumatic physisal bridge formation is much more common at the distal ends of long bones compared with the proximal ends, and in the lower extremities compared with the upper extremities.
Figure 6. Metaphyseal injury leading to persistence of physeal cartilage in the metaphysis. A, Sagittal fat-suppressed fast spin echo proton density image of the distal femur in a 12-year-old boy with severe genu valgus shows a tongue of high signal intensity cartilage in the metaphysis (arrow). B, Sagittal fat-suppressed spoiled gradient-recalled echo image of the proximal tibia in a 14-old-boy with Blount disease shows multiple areas of physeal widening.

Figure 7. Altered modeling caused by metaphyseal injury. AP radiograph of the left hip in an 11-year-old with a remote history of metaphyseal osteomyelitis shows widening of the femoral neck and protuberance of the medial proximal femoral metaphysis.
The risk of growth disturbance following physeal fracture is dependent in descending order of importance on the severity of the injury; the patient’s growth potential (age and skeletal maturity); anatomic site involved; and fracture type. Fractures with significant displacement, comminution, or those with loss of a segment of the germinal or proliferative physeal layers are at greatest risk. Younger patients have a poorer prognosis because there is more time for deformity to develop. The importance of the anatomic site relates to the size and contour of the physis and its rate of growth. Undulating, multiplanar physes like those of the proximal tibia and distal femur are particularly prone to growth arrest, and because these sites contribute 60% to 70% of the growth of their respective bones, leg length discrepancy and angular deformity frequently result.

Post-traumatic bone bridge formation is also dependent on the fracture type. This has less to do with Salter-Harris classification than with the amount of physis involved and the intracartilaginous path of the fracture (Fig. 8). Longitudinal fractures extend from the epiphysis to the metaphysis perpendicular to the plane of the physis. This allows development of transphyseal vascularity leading to bridge formation in 75% of cases. Transverse fractures parallel to the physis, on the other hand, develop bone bridges in only 25% of cases, presumably because they are juxtametaphyseal extending through the avascular chondro-osseous junction. In a recent study of transverse physeal fractures in rabbits, however, 60% of the distal femoral fractures involved the germinal and proliferative zones both at MR imaging and histologically. This juxtaepiphyseal extension occurred most frequently in the central physeal undulation, which is the site of earliest normal physiologic epiphysiodesis. This may explain the high incidence of post-traumatic physeal bridging in the distal femur.

**IMAGING OF GROWTH ARREST**

The goal of imaging patients with growth disturbance is accurate demonstration of the physeal cartilaginous pathology and depiction of the size and location of bone bridges relative to the remainder of the physis to guide surgical management. Bridge size determines the surgical procedure. Bridges comprising less than 50% of the physis may be resected with good prognosis. Larger bridges may require ipsilateral epiphysiodesis to prevent angular deformity, contralateral epiphysiodesis to avoid leg length discrepancy, corrective osteotomy, or a combination of procedures. Bridge location determines the surgical approach with the aim of sparing as much normal physis as possible.

Radiographs usually provide the initial clues to growth disorder. Physeal widening or narrowing are the early findings. Approximately 3 months following injury, radiographs may show bone bridge development. MR images can reveal transphyseal abnormalities earlier, but some of these heal spontaneously. Centrally located bridges lead to decreased growth and leg length discrepancy. Peripheral bridges result in focally arrested growth and angular deformity. Although CT and scintigraphy have been used for further evaluation of these patients, MR imaging has proved to be the modality of choice with its unique ability to visualize cartilage.

The authors typically image patients with physeal pathology using coronal T1-weighted images, sagittal fat-suppressed fast spin echo proton density and T2-weighted images, and a three-dimensional fat-suppressed spoiled gradient echo (SPGR) sequence. The latter is usually acquired in the coronal plane prescribed off an axial localizer, with slice thickness less than 1 mm. The advantages of the three-dimensional SPGR sequence are
multiplanar reformations and the ability to generate an axial maximum intensity projection map of the physis (Fig. 9). On a workstation, the physis and bridge can be traced, areas determined, and a bridge-to-physis ratio calculated in less than 15 minutes. These maps have become critical to surgical planning.

On fat-suppressed three-dimensional SPGR images, all cartilage has bright signal intensity. A bone bridge appears as a low signal intensity interruption in the normally high signal intensity physis (see Fig. 9A). Physeal dysfunction without bridge appears as high signal intensity physeal thickening or irregularity. The T1 signal intensity of physeal bridges varies with size: small bridges are of low signal intensity whereas larger bridges are

Figure 9. Three-dimensional spoiled gradient-recalled echo (3D SPGR) MR imaging of a bony bridge producing distal tibial growth arrest. A, Coronal fat-suppressed 3D SPGR image in a 14-year-old boy who had suffered a fracture 6 months earlier. There is a low signal intensity bone bridge interrupting the high signal intensity physis (arrow). The bridge is at Kump's bump, which is the site of physiologic physeal closure. B, Isolation of the juxtaphyseal area for creating a maximum intensity projection (MIP) axial map of the physis. C, Axial MIP reveals the low signal intensity bony bridge (arrows) anteromedially within the heterogeneous high signal intensity physis. Ridges within the normal physis correspond to the mammillary processes. The traced physeal area measures 1404 mm$^2$ and the bridge measures 411.8 mm$^2$. D, Coronal T1-weighted image obtained at a more anterior level shows that the large bridge is isointense with fatty marrow (arrow).
IMAGING OF GROWTH DISTURBANCE IN CHILDREN

GROWTH RECOVERY LINE

Figure 10. Growth recovery line following an anterior growth arrest of the distal tibia. Sagittal T1-weighted image in an 11-year-old girl shows a high signal intensity anterior bridge tethering growth. The physis abuts the growth recovery line (arrows) anteriorly at the bridge but has migrated distally posteriorly.

isointense to fatty marrow (Fig. 9C). T2-weighted images are helpful in young patients for differentiating physeal cartilage, which is bright, from epiphyseal cartilage, which is dark. Proton density and T2-weighted images are also helpful for identifying associated marrow and soft tissue including edema, persistent metaphyseal cartilage, areas of avascular necrosis, and ligamentous or meniscal injury.

Growth recovery lines (GRL) seen on radiographs and MR images provide important clues to physeal health. These lines, also called Parks or Harris lines, represent disks of transversely oriented, as opposed to the normal longitudinally oriented, bony trabecula. They form at the physis during slowed growth because of injury, immobilization, or illness. As growth resumes, the physis migrates away from the line, which remains in the metaphysis. If the line parallels the physeal contour, growth is normal. A GRL that is angled or obliquely oriented relative to the physis indicates growth tethered by a bone bridge (Fig. 10). A GRL in the fibula or ulna without a corresponding line in the tibia or radius suggests growth arrest of the entire physis. GRL are best seen on T1-weighted images as low signal intensity bands but can also be seen on proton density and gradient recalled echo (GRE) images.

GROWTH ARREST BY ANATOMIC LOCATION

Proximal Femur

Growth disturbance of the proximal femur is most commonly ischemic secondary to hyperabduction therapy for DDH, Legg-Calvé-Perthes disease, and rapidly developing effusions caused by hip infection. The proximal femoral physis is particularly vulnerable because the epiphyseal artery is intra-articular. Attenuation of the artery by abduction or increased intra-articular pressure leads to ischemia of the epiphysis and germinal and proliferative zones of the physis. The proximal femur has a unique "bifid" growth plate that results in typical patterns of growth arrest. The lateral portion is slow growing and forms in relation to the greater trochanteric apophysis. The medial physis grows twice as rapidly as the lateral portion and forms the femoral neck. The junction between the medial and lateral portions, the vertex, is the most active area of growth and is particularly prone to ischemia.

Growth recovery lines often appear following reduction for DDH. If growth is normal, the distance between the GRL and the medial physis should be twice what it is laterally (Fig. 11). Deviations from this pattern imply physeal dysfunction. Medial physeal impairment leads to a short, wide femoral neck and

Figure 11. Normal growth recovery line in a 7-month-old girl following reduction of developmental dysplasia of the hip. The distance between the physis and the growth recovery line medially (arrows) is twice that seen laterally (arrowheads).
coxa vara deformity (Fig. 12), whereas vertex arrest causes coxa valga (Fig. 13). True lateral growth arrest is infrequent.

Knee: Distal Femur and Proximal Tibia

The undulating, multiplanar course of the physis of the distal femur and proximal tibia predisposes to juxtaepiphyseal fracture paths and bony bridge formation. Physeal fractures of the distal femur have the highest incidence of post-traumatic arrest with nearly 40% developing a bony bridge. Most occur at the central undulation of the distal femoral physis, which is also the initial site of normal physiologic physeal closure (Fig. 14). These large central bridges often cause sufficient limb shortening to warrant contralateral epiphysiodesis.

Proximal tibial growth arrest is often post-traumatic but also occurs in patients with Blount disease. Unlike post-traumatic bridges, which are most often central in the proximal tibia, growth arrest in Blount disease specifically involves the medial physis (Fig. 15). The etiology of bridge formation in Blount disease is uncertain but may be related to chronic stress from altered weight bearing. T2-weighted MR images in these patients often reveal metaphyseal cartilage rests (see Fig. 6B) and medial meniscal and epiphyseal cartilage abnormalities. Proximal tibial bone bridges that involve the anterior tibial tubercle usually occur after fractures and cause genu recurvatum deformity (Fig. 16).

Distal Tibia

Greater than 10% of physeal fractures involve the distal tibia and about one third develop growth arrest. Normal physiologic closure of this physis begins at an anteromedial undulation called Kump’s bump, which can be seen radiographically and with MR imaging (Fig. 17). This is also the most common location for post-traumatic premature physeal closure of the distal tibia (Fig. 18). These eccentric, peripheral physeal bridges lead to varus deformity. The differential positions of the distal tibial and fibular physes should not be misinterpreted as Text continued on page 837

Figure 12. Medial proximal femoral growth arrest following avascular necrosis complicating abduction therapy for developmental dysplasia of the hip resulting in coxa vara. A, AP radiograph of the proximal left femur of a 3-year-old girl shows abnormal growth. The distance between the physis and the growth recovery line is less medially (arrow) than it is laterally. B, Coronal gradient-recalled echo image also shows the growth recovery line (arrows), the varus deformity, and the unossified high-riding greater trochanter. Capital femoral ossification center is not in the plane of this section.
Figure 13. Vertex proximal femoral growth arrest following avascular necrosis complicating abduction therapy for developmental dysplasia of the hip resulting in coxa valga. A, AP radiograph of the left hip in a 16-month-old girl shows severe acetabular dysplasia and dislocation of the femoral head. B, At 3 years of age there is coxa valga deformity and avascular necrosis of the femoral head. C, At 7 years of age, the radiograph shows coxa valga and a bone bridge at the vertex (arrow). D, Coronal 3D fat-suppressed spoiled gradient-recalled echo image of the hip obtained at the same age shows a small bony bridge at the vertex interrupting the physis (arrow). E, AP radiograph at 7 years of age following medial epiphysiodesis to prevent further angular deformity.
Figure 14. Post-traumatic bone bridge formation in the distal femur. A, Coronal fat-suppressed fast spin echo T2-weighted image of the distal femur in a 12-year-old boy obtained at the time of injury shows a metaphyseal fracture. There is associated metaphyseal marrow edema medially and widening of the physis laterally (solid arrow). At the distal femoral central undulation, the fracture path approaches the epiphysis and there is associated epiphyseal marrow edema (open arrow). B, Coronal 3D fat-suppressed spoiled gradient-recalled echo image of the knee obtained 6 months later reveals a central bone bridge across the distal femoral physis (arrow). C, The corresponding MIP axial map of the physis confirms that the bone bridge involves less than 50% of the physis. The patient underwent bridge excision.
IMAGING OF GROWTH DISTURBANCE IN CHILDREN

Figure 15. Medial proximal tibial growth arrest in Blount disease. Coronal gradient-recalled echo image of the right knee in a 7-year-old girl with Blount disease demonstrates downward sloping of the medial aspect of the proximal tibial metaphysis and a small peripheral physeal bridge (arrow). There is increased unossified cartilage in the medial tibial epiphysis.

Figure 16. Genu recurvatum deformity because of anterior tibial tubercle bone bridge. Sagittal (A) and coronal (B) 3D fat-suppressed spoiled gradient-recalled echo images of the knee in an 11-year-old girl with spondyloepiphyseal dysplasia reveal premature physeal closure of the anterior tibial tubercle (arrow) resulting in genu recurvatum. Abnormal foci of epiphyseal and metaphyseal cartilage typical of this dysplasia are also demonstrated.

Figure 17. Kump's bump. Coronal 3D fat-suppressed spoiled gradient-recalled echo image of the distal tibia in a 10-year-old boy shows the upward undulation in the anteromedial physis, Kump's bump (arrow), which is the site of initial physiologic physeal closure and the most common location of post-traumatic premature physeal bridging.
Figure 18. Anteromedial distal tibial growth arrest. A, Coronal T1-weighted spin echo image of the ankle in a 14-year-old boy obtained 1 year following a Salter-Harris 2 fracture shows a low signal intensity growth recovery line (arrow), which converges with the physis medially. Sagittal 3D fat-suppressed spoiled gradient-recalled echo image (B) and the corresponding axial MIP physeal map (C) demonstrate the low signal intensity anteromedial bridge that obliterates 20% of the physeal area.
IMAGING OF GROWTH DISTURBANCE IN CHILDREN

Figure 19. Physeal arrest without bony bridging caused by repetitive physeal injury. Coronal gradient-recalled echo images of the wrist in a 15-year-old female gymnast show areas of high signal intensity physeal widening (white arrows) related to chronic stress. A, Demonstration of the positive ulnar variance and the distal deviation of the triangular fibrocartilage (black arrow) that is intact. B, Obtained at a more volar level, this image shows the physeal irregularity to better advantage.

evidence for growth arrest. The fibular physis is just distal to the tibial physis in early life; by age 3 years, the fibular physis migrates to the level of the tibial articular surface.2,17

Distal Radius

Despite the high frequency of distal radial physeal fractures, bony bridging rarely develops in this smooth, uniplanar growth plate. Physeal abnormalities, however, are frequent in the distal radius of gymnasts because of repetitive trauma.22 MR imaging in these patients shows diffuse physeal widening and increased T2 signal intensity (Fig. 19). In patients with Madelung’s deformity chronic stress and proximal curvature of the physis predispose to medial physeal dysfunction and even bony bridging (Fig. 20). MR imaging often reveals relative ulnar lengthening and triangular fibrocartilage complex abnormalities in the wrists of gymnasts and patients with Madelung’s deformity.

Elbow

The distal humerus accounts for only 20% of the growth of the bone and growth disturbance at this location rarely causes clinically significant limb shortening. Most distal humeral fractures in children are supracondylar without physeal involvement. Although cubitus varus deformity is a common sequela of these injuries, this is a complication of the reduction rather than a progressive arrest of growth. Internal fixation is frequently used to reduce the risk of cubitus varus deformity. The risk of subsequent growth arrest caused by pins traversing the physis is being evaluated (Fig. 21).

Lateral condylar fractures account for 16% of elbow fractures in children and may lead to lateral physeal growth arrest and cubitus valgus deformity (Fig. 22). The more common result, however, is premature fusion of all of the distal humeral ossification centers with

Figure 20. Growth disturbance in Madelung deformity. Coronal gradient-recalled echo images of the wrist in a 12-year-old girl demonstrates downward sloping of the medial distal radial physis consistent with Madelung deformity. The medial epiphysis is deficient, and there is a small bone bridge across the medial physis (arrow).
Figure 21. Trochlear growth arrest following transphyseal pinning for supracondylar fracture. A, AP radiograph of the elbow in a 9-year-old boy obtained 1 year after internal fixation for a supracondylar fracture shows cubitus varus deformity and fragmentation of the trochlear ossification center. B, Sagittal 3D fat-suppressed spoiled gradient-recalled echo image of the elbow shows complete bony bridging between the small trochlear ossification center (arrow) and the distal humeral metaphysis. Closure of the trochlear portion of the distal humeral physis normally occurs at approximately 16 years of age.

mild shortening. This is of little consequence in the upper extremity.

Proximal Humerus

Physeal fractures of the shoulder are rare in children. This is a common site for benign tumoral lesions, however, such as bone cysts, osteochondromas, and enchondromas. If these lesions cross into the epiphysis allowing transphyseal vascular communication, bony bridging can result leading to shortening or angular deformity (Fig. 23).

SUMMARY

Growth disturbance of the long bones in children is frequently post-traumatic but also occurs because of physeal, epiphyseal, or metaphyseal ischemia. The imaging features of growth arrest depend more on the anatomic site involved than on the cause. The physes of the distal tibia and femur and proximal tibia are disproportionately at risk because of their complex geometry. The central undulation in the distal femur and the bump in the anteromedial physis (Kump's bump) in the distal tibia are the sites of initial physiologic closure and the most frequent areas of premature fusion.

The MR imaging features of growth disturbance are characteristic. T1-weighted images show low signal intensity GRL and variable signal intensity bony bridges. On GRE sequences, a bridge appears as low signal intensity interruption in the otherwise high signal intensity physeal cartilage. Physeal widening on GRÉ and T2-weighted images implies physeal dysfunction without bridge formation. Proton density and T2-weighted images best reveal associated metaphyseal and soft tissue changes.

Regardless of the cause, MR imaging exquisitely depicts cartilaginous pathology at the
Figure 22. Growth disturbance following lateral condylar fracture. A, AP radiograph of the elbow in an 11-year-old girl obtained several months after a Salter-Harris 4 lateral condylar fracture of the distal humerus suggests premature closure of the capitellar physis. Normal closure of the capitellar physis occurs between 13 and 16 years of age. B, Coronal gradient-recalled echo image confirms complete premature bridging of the capitellar portion of the distal humeral physis (arrow). Other imaging sequences (not shown) demonstrate avascular necrosis of the capitellum.
physis. MR evaluation should be considered in patients at high risk for growth disturbance including young children with extensive residual growth potential; those with involvement of particularly vulnerable growth plates; and those with severe, complex fractures.

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