CHAPTER 16

Orthopaedic Problems in the Pediatric Patient

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Nontraumatic Disorders

The nontraumatic disorders of adulthood are typically degenerative, whereas the nontraumatic disorders of childhood are typically developmental. Therapy for adult disorders usually focuses on restoration of function and retardation of degeneration, while therapy for childhood disorders usually focuses on correction of distorted anatomy and prevention of accelerated degeneration.

CONGENITAL MUSCULAR TORTICOLLIS

Congenital muscular torticollis is a contracture of one of the sternocleidomastoid muscles, more commonly seen on the right side. The cause of this deformity is unknown, although it has recently been studied with MRI and appears to be related to an intrauterine compartment syndrome. The deformity is characterized by a "cock robin" position of the head in which the head is tilted toward the side of the shortened muscle and the chin rotated to the contralateral side. On pathologic section of excised specimens, the muscle appears to be replaced by fibrous tissue.

Clinical Characteristics

At birth, the torticollis may or may not be evident but usually is identified in the first 2 months of life. In children who are normal at birth, the deformity and mass in the sternocleidomastoid usually become obvious at 2 to 3 weeks. As the child grows, the abnormal posture progresses, and secondary structural changes occur in the face and skull, resulting in asymmetric molding of these structures. Palpation of the sternocleidomastoid may reveal a firm, nontender, fusiform "olive" within the substance of the muscle or replacing the muscle. When present at birth, the mass commonly enlarges over the subsequent several weeks. Regression of the "tumor" usually occurs over a period of 2 to 6 months. Without treatment, facial asymmetry, including changes in the levels of the ears, eyes, and skull, may develop. The superficial and deep muscular tissues of the neck may become shortened.

Because of an increased incidence of clubfeet, congenital hip dysplasia, and other congenital abnormalities in patients with torticollis, these infants should be carefully examined. Patients with torticollis often have a significant history of difficult delivery, and infants with such a history should be carefully checked for this deformity. Infants with congenital torticollis do not have any neurologic abnormality. Although pain may occur with passive stretching, it does not occur at rest.

X-ray studies are usually unremarkable in the muscular type of torticollis. However, x-rays are necessary because there may be congenital bony deformities of the cervical spine (Klippel-Feil syndrome and primary cervical synostosis) that need to be excluded. The differential diagnosis includes traumatic conditions such as fracture or rotatory subluxation, inflammation of the cervical lymph nodes, tumors of the spinal cord or brain, and Sprengel's deformity (congenital elevation of the scapula).

Treatment

Early recognition and treatment are important. Stretching of the involved sternocleidomastoid should be performed several times a day. A good stretching program is usually successful in completely correcting the deformity but must be closely supervised. The physician or an experienced physical therapist should instruct the parents in the technique of stretching. One hand is used to stabilize the chest and shoulders, while the other hand tilts the head away from the contracted muscle and rotates the chin toward the contracted side. Each stretching maneuver is best held to a count of 10 with 15 repetitions. The initial tenderness of the muscle mass with stretching subsides gradually, and a great majority of infants respond to these conservative measures.

If the deformity does not respond to passive stretching in 1 to 2 months, orthopaedic referral is indicated. If a child is over 1 year of age at initial presentation, the contracture and progressive deformity are probably best treated by surgical release of the sternocleidomastoid muscle.

KLIPPEL-FEIL SYNDROME

Klippel-Feil syndrome is a congenital fusion of two or more cervical vertebrae. Clinically, the neck is short, stiff, and webbed. The hairline is often low and horizontal. This syndrome is often associated with torticollis. These patients should be referred for orthopaedic evaluation.

ROTATORY SUBLUXATION OF C-1 ON C-2

Rotatory subluxation of the atlantoaxial joint is a relatively common problem in children. It may be the result of an injury or may present without a history of significant trauma. In these circumstances, this disorder often occurs in association with pharyngitis (Grisel's syndrome). It is thought that inflammation, edema, and hyperemia lead to weakening of the supportive ligaments at C-1/C-2, allowing spontaneous subluxation of the facet joints. This disorder also may be a consequence of an otherwise minor injury.

Clinical Characteristics

The diagnosis is based on a fixed, painful torticollis with muscle spasm. Cervical spine x-rays, particularly the open-mouth odontoid view, reveal the pathognomonic asymmetry of the C-1/C-2 joint with the odontoid deviated to one side. In some children, painful torticollis may be present without radiographic evidence of C1/C2 subluxation. In equivocal situations, the diagnosis can be confirmed using dynamic CT scanning with the head maximally rotated to each side.

Treatment

Treatment consists of bedrest, analgesics, and constant head-halter traction until painful spasms subside and the subluxation reduces. Children who have torticollis without subluxation can be treated with a closely supervised stretching program with a physical therapist. After reduction, the child's neck should be protected in a soft cervical collar (Fig. 16.1) until the neck is completely comfortable and stable with a full, painless cervical range of motion. If the subluxation does not respond to traction within 5 to 7 days, an orthopaedic surgeon should be consulted.

BIRTH INJURIES OF THE BRACHIAL PLEXUS

The two common types of brachial plexus injury, Erb's palsy and Klumpke's palsy, are caused by strong lateral flexion of the infant's head and neck, producing traction on the brachial plexus. These injuries typically occur during a difficult delivery in which there is cephalopelvic disproportion when the infant must be extracted quickly to ensure viability.

Clinical Characteristics

Erb's palsy is the more common of the two conditions. The infant presents with mixed sensory and motor defects of the upper nerve roots (C-5/C-6). These roots control shoulder abduction and external rotation, and elbow flexion. At birth, the arm is typically motionless and lies adducted and extended in internal rotation at the infant's side. The hand

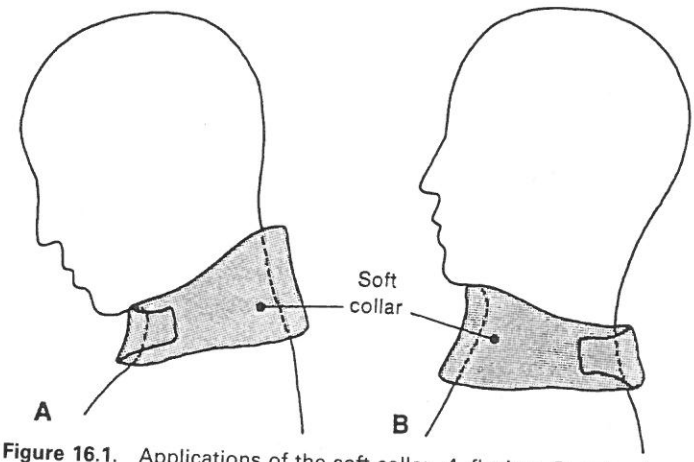

Figure 16.1. Applications of the soft collar. *A*, flexion. *B*, extension.

and wrist are usually functional. Although occasionally transient and reversible, the condition frequently results in residual paralysis and sensory deficits of the shoulder and upper arm. In contrast, "pseudoparalysis" may result from a perinatal fracture of the clavicle or humerus and can easily be confused with a brachial plexus injury. X-rays may be indicated to rule these out.

Klumpke's palsy is a rare condition. It involves the lower roots (C-8/T-1), resulting in a flail hand and wrist with atrophy of the forearm. The upper arm is functional. Spontaneous recovery is not likely.

Treatment

The initial treatment for both types of palsy is gentle passive range of motion exercises and splinting of the paralyzed muscle groups to prevent contractures. These exercises should be taught to the family by a skilled physical or occupational therapist so that the routine can be carried out several times each day. The prognosis depends on the severity of injury to the brachial plexus. If the root avulsion is adjacent to the spinal cord, the prognosis is poor, and nerve repair in the first year of life may be indicated. Return of biceps function by 3 to 6 months carries a good prognosis. Reconstructive orthopaedic surgery is sometimes useful to improve function of the impaired extremity. Orthopaedic consultation is necessary.

SCOLIOSIS

Scoliosis is an abnormal lateral curvature of the spine. When viewed from the front or back, the spine should be straight, centering the head over the sacrum. A number of conditions can produce an apparent scoliosis without a true, intrinsic spinal deformity. These conditions include a leg length discrepancy, muscular spasm, poor posture, or hysteria. We use the term "scoliosis" to describe a true, intrinsic spinal deformity.

Scoliosis may be secondary to congenital bone malformation, bony dysplasia, or metabolic or paralytic conditions. If no primary cause is obvious, it is designated as idiopathic scoliosis. Curvatures under 10°, as measured radiographically, that are not progressive do not require treatment or referral to an orthopaedist. Because scoliosis is often progressive, even the child with a mild degree of curvature greater than 10° should be referred to an orthopaedic surgeon for initial evaluation and possible treatment. Idiopathic scoliosis is the most common type of childhood scoliosis. This condition occurs in approximately 1% of the adolescent population and predominantly affects females.

With the advent of mandatory school screening programs, a large number of children with spinal asymmetries that do not represent true scoliosis are seen by the primary care physician. In this setting, the evaluation should define the exact nature of the condition. Abnormal posture, leg length discrepancy, or some painful condition producing an apparent spinal asymmetry must be ruled out. The presence of developmental, traumatic, or paralytic etiology must be evaluated. In the absence of these causes, the diagnosis of idiopathic scoliosis is confirmed. This is an inherited disorder characterized by a sex-linked or autosomal dominant genetic pattern with a variable expressivity and incomplete penetrance.

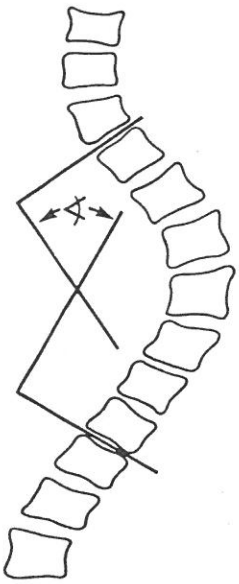

Figure 16.2. The Cobb method of measuring spinal curvature. (Copyright 1989. Novartis. Reprinted with permission from Clinical Symposia, Volume 41/4, Plate 12, illustrated by Frank H. Netter, MD. All rights reserved.)

Clinical Characteristics

Idiopathic scoliosis is generally a painless disorder in the adolescent, although the onset of a deformity may be noticed at an earlier age. A complaint of back pain warrants thorough investigation to rule out other etiologies and should not be attributed to the underlying scoliosis. On physical examination, the patient presents with asymmetry in the levels of the shoulders and in the waist crease, prominence of one iliac crest, and a posterior rib hump prominence most obvious on forward bending. Leg length measurements will not show significant discrepancy, and neurologic examination is usually normal. If there is evidence of neuromuscular disease or metabolic abnormality, or if x-rays reveal a congenital basis, the scoliosis is not classified as idiopathic.

Radiographs of the spine are essential for the diagnosis and treatment of scoliosis. To diminish the radiation exposure of the breasts in adolescent girls, the frontal radiograph should be performed in an anterior to posterior projection and breast shields should be used. The radiograph will establish the extent and the severity of the curvature.

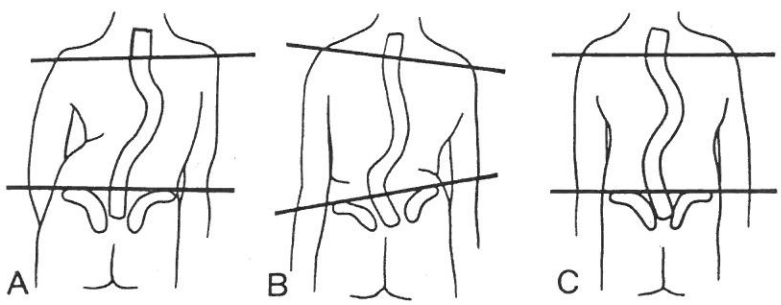

Figure 16.3. Examples of body contour with scoliosis. *A*, right thoracic curve. *B*, left lumbar curve. *C*, double major curve. (Copyright 1989. Novartis. Reprinted with permission from Clinical Symposia, Volume 41/4, Plate 3, illustrated by Frank H. Netter, MD. All rights reserved.)

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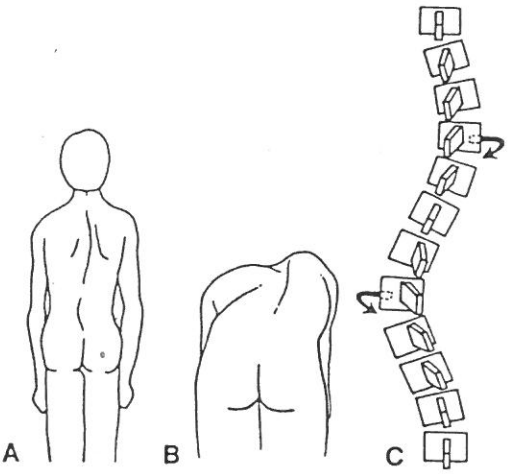

Figure 16.4. Right thoracic, left lumbar curve. *A*, patient standing upright. *B*, patient bending forward. *C*, the rotation accompanying lateral curvature. (Vertebral bodies rotate toward the convexity, hence clockwise rotation accompanies right thoracic curve, and counterclockwise rotation accompanies left lumbar curve, as viewed from overhead.) (Copyright 1989. Novartis. Reprinted with permission from Clinical Symposia, Volume 41/4, Plate 10, illustrated by Frank H. Netter, MD. All rights reserved.)

The amount of curvature is determined by the Cobb method (Fig. 16.2). Any number of combinations of curves may be encountered (Fig. 16.3). Lateral curvature is always accompanied by rotation of the vertebrae. The rotational component is an important determinant of clinical deformity and is most noticeable when the child bends forward and the examiner sites down the axis of the spine (Fig. 16.4).

Treatment

The treatment of idiopathic scoliosis is based on data obtained from long-term longitudinal studies of the natural history of scoliosis. A curvature of less than 30° at skeletal maturity appears to cause no prolonged disability as a person matures in later life. However, curves that have reached 50° at the time of skeletal maturity tend to progress later in life and may cause restrictive pulmonary disease, cor pulmonale, and severe back pain. Therefore, the treatment of idiopathic scoliosis in the adolescent attempts to prevent the curve from progressing to 50°. A curvature of greater than 10° in a skeletally immature patient needs to be followed by periodic clinical and radiographic examinations until skeletal maturity has been reached. An interval of 4 to 6 months between each visit is an acceptable follow-up program. After initial orthopaedic consultation, the primary care physician may monitor children in this early stage. However, while under observation if the curve progresses more than 5°, or on presentation if the curve is greater than 20°, referral to an orthopaedic surgeon for follow-up and treatment is recommended.

At this degree of deformity, brace therapy is initiated. The spinal orthosis, or brace, consists of a polypropylene shell that is contoured to provide a corrective force to the spine while the child grows. It is necessary that the child wear the brace for at least 16 hours a day until skeletal maturity is reached. The Charleston Bending brace is a new design that uses overcorrection of the spine to prevent further progression. This brace can be used in a nighttime only program. A bracing program usually involves treatment for approximately 3 to 4 years. This is difficult for the adolescent patient to accept, and the primary care physician is very helpful in providing guidance, direction, and emotional support to the patient and family. A scoliosis clinic where other similarly affected adolescents are treated can provide a supportive treatment environment.

If the curvature progresses to a point of 45 to 50°, the patient is at high risk for further progression in adulthood. Therefore, surgical stabilization of the spine with corrective instrumentation is performed to correct the curvature and prevent further progression.

ADOLESCENT KYPHOSIS (SCHEUERMANN'S DISEASE)

Adolescent kyphosis, also known as Scheuermann's disease, is thought to be secondary to repetitive trauma and stress fractures of the anterior aspect of the vertebral endplates in the growing adolescent. Untreated, the disease causes permanent kyphosis

of a variable degree, which will be established at the end of the adolescent growth period. Nonoperative treatment is effective only when applied before the cessation of skeletal growth.

Clinical Characteristics

The patient presents in early adolescence with thoracic or lumbar back pain or a kyphotic postural deformity. The child is often taller and heavier than other children of his or her age. Girls are affected as often as boys. When the disease process is in the thoracic area, there is an increased thoracic kyphosis; however, in a few children, the disease affects the lumbar region, causing that portion of the spine to look abnormally flat. The pain usually is aggravated by prolonged activity or standing for long periods of time and is relieved by rest. There may be local tenderness. The sensory and motor examinations are normal. The diagnosis is confirmed by a lateral radiograph of the spine that should be obtained while the patient is standing. The normal thoracic kyphosis measured from T4 to T12 by the Cobb method is between 20 and 40°. An increase in the amount of kyphosis of at least 5° in three or more consecutive vertebral bodies associated with anterior wedging of the vertebral body establishes the diagnosis. Concave osteolytic defects at the endplates, known as Schmorl's nodes, may be noted. These represent herniation of disc material into the weakly ossified vertebral endplate (Fig. 16.5).

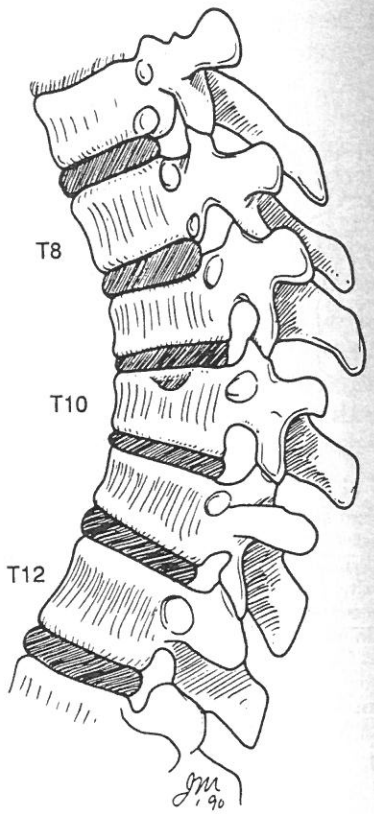

Figure 16.5. Juvenile kyphosis. Anterior vertebral body wedging on three or more consecutive vertebrae.

Treatment

The primary care physician should refer children to the orthopaedist if the radiographic findings of Scheuermann's disease are present. The treatment of Scheuermann's disease in a skeletally immature patient incorporates the use of a thoracolumbar brace and active physical therapy programs. Such treatment may decrease the amount of inevitable deformity. For severe deformity, however, surgical treatment may be necessary. Corrective spinal fusion with instrumentation can be performed in cases resistant to the aforementioned modalities. In the absence of radiographic findings of Scheuermann's disease, the "round back" deformity simply represents a postural habit. In these children, physical therapy is helpful in strengthening the thoracic extensor muscles and flattening the accentuated lumbar lordosis, which is commonly present. The therapy regimen should emphasize antilordotic lumbar exercises and thoracic extension exercises.

SPONDYLOLYSIS AND SPONDYLOLISTHESIS

The term spondylolysis refers to a bony defect in the pars interarticularis of the posterior elements of the vertebral body (Fig. 16.6B). Spondylolysis is thought to be the result of nonunion of a stress fracture in the

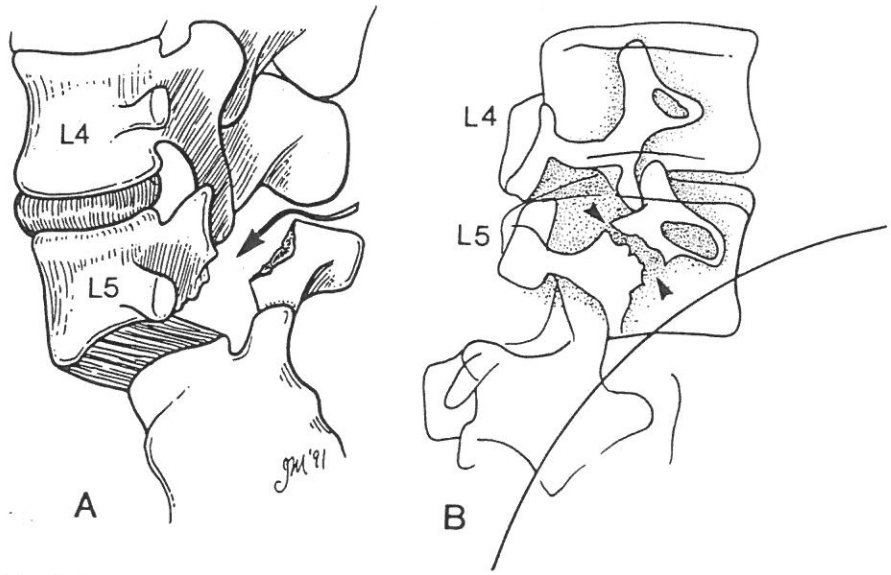

Figure 16.6. *A*, lateral view of spondylolytic spondylolisthesis. *B*, spondylolysis. A 45° oblique view of the lumbar space will show a defect in the pars interarticularis.

posterior elements. Defects of the pars interarticularis may be due to infection, trauma, tumor, congenital or developmental defects. The condition most commonly occurs at the L-5 lamina but has been seen throughout the spine. A high incidence of spondylolysis occurs in athletes who undergo constant hyperextension activity of the lumbar spine, such as gymnasts and football linemen. Spondylolisthesis refers to an anterior displacement of the vertebral column on a lower vertebra, most commonly an anterior slippage of L-5 on S-1 (Fig. 16.6A). This may be the result of spondylolysis, articular process malformation, or elongation of the pars interarticularis. Moderate or severe degrees of spondylolisthesis may cause nerve root impingement, and may consequently present with signs and symptoms of nerve root irritation.

Clinical Characteristics

Spondylolysis typically occurs in preadolescent and adolescent children. The initial symptom is an aching pain in the lumbar region, which is associated with activity. It may radiate into the buttock and thigh on the affected side. The pain typically is relieved by rest or limiting the aggravating activity. Spondylolysis is often associated with a feeling of stiffness. This is particularly true for gymnasts or ballerinas, who complain that they are unable to perform their usual routine. This may be difficult to detect on clinical examination because these children are so flexible that even with a decrease in their flexibility they still appear to be within normal standards. The physical examination reveals spasm in the paravertebral muscles, and a flattening of the normal lumbar lordosis may be present during the acute presentation with back pain. The child with long-standing and severe spondylolisthesis may have a kyphotic deformity at the lumbosacral junction with a compensatory hyperlordosis in the lumbar region. Hamstring tightness is common. This presents as diminished straight leg raising, increased popliteal angle, and diminished stride length on walking or running. The neurologic examination is usually normal except in those infrequent cases in which there is nerve root irritation. The diagnosis is confirmed by radiographs of the lumbar spine (Fig. 16.6B). A lumbosacral series should consist of standing lateral and anteroposterior (AP) views, as well

as 45° oblique views of the lumbar spine. Occasionally, back pain will be the presenting symptom in a child before the plain film radiographs show the bony defect. In these cases, a bone scan will be helpful.

Treatment

The goals of treatment in a child with spondylolisthesis or spondylolysis are (*a*) pain relief, (*b*) return to activity, and (*c*) prevention of deformity. It is necessary to monitor children with spondylolysis until they reach skeletal maturity. A child with spondylolysis may be treated by the primary care physician with a conservative program consisting of nonsteroidal anti-inflammatory medications, limiting activity below the pain threshold, and a physical therapy regimen. The physical therapy regimen emphasizes antilordotic exercises for the lumbar spine, such as pelvic tilts and modified situps, with stretching exercises for the hamstrings. Patients who do not respond to this therapy should be referred to an orthopaedic surgeon. Any patient who has pain associated with spondylolisthesis should be referred to an orthopaedic surgeon for evaluation. In the adolescent years, a spondylolysis may progress to spondylolisthesis, or a spondylolisthesis may increase in severity; however, this is unlikely to be true for the adult population. For children who do not respond to conservative treatment, a brace may be helpful to control their pain level and allow them to participate in activities. Surgical repair of the spondylolysis or posterolateral fusion of the involved vertebrae are indicated when the patient is unresponsive to conservative treatment, shows progression of a spondylolisthesis, or presents with a severe degree of spondylolisthesis. Surgical fusion is effective in controlling the pain and progression of the disease under these circumstances.

DEVELOPMENTAL DYSPLASIA OF THE HIP

Developmental dysplasia of the hip is the most common disorder of the hip presenting during the first 3 years of life. The term developmental dysplasia is commonly applied to the full spectrum of congenital and developmental hip diseases, which includes dysplasia without subluxation, the subluxatable hip, and the dislocated hip. The current terminology has gained acceptance as a result of the understanding that occasionally a hip that is clinically normal at birth will become dysplastic in the early months of life. The incidence of true congenital dislocation of the hip is approximately 1.2 in 1,000 live births. Developmental dysplasia of the hip is more commonly associated with intrauterine crowding, breech presentation, and ligamentous laxity. It is much more common in females, is most often unilateral with predilection for the left hip, and has a familial pattern. Other associated features include torticollis, hyperextension of the knee, and abnormalities of the feet. Because the development of the acetabulum and femoral head depend on normal physiologic stress, untreated dysplasia or dislocation may result in secondary developmental changes of both the acetabulum and femoral head, further compounding the initial deformity.

The pathology of developmental dysplasia of the hip is variable, depending on the severity of the condition. Infants may be born with minimal dysplasia in which some deformity of the acetabulum is evident, but the hip is not actually dislocated. In other infants, the hip may be dislocatable or dislocated; in the most severe form, there is obvious teratologic deformity of the joint and soft tissue with complete dislocation. Teratologic hip dislocation refers to a particular subset of hips associated with a syndrome in which the dislocation occurred earlier in intrauterine life. This type of dislocation is usually more difficult to treat.

The initial suspicion of hip instability or dislocation is clinical, requiring careful examination of the newborn and infant at the "well baby visits." Any child with a significantly abnormal walking gait requires x-rays of the hips. This does not usually include the common problems such as isolated in-toeing or out-toeing, or the expected variations of a newly walking child.

Clinical Characteristics

The clinical presentation of developmental dysplasia varies according to the age of presentation and the pathologic state of the joint. In infants, the most common findings are a positive Barlow test and Ortolani test. With the Barlow test, the hip can be felt subluxing posteriorly with an adduction-axial loading maneuver. With the Ortolani test, the dislocated hip can be felt reducing with an abduction maneuver that is often accompanied by an audible or palpable "clunk" (Fig. 16.7). This should be distinguished from the distinctive "click" that often is seen in normal hips and is related to myofascial motion in the hip or knee. Asymmetry of the gluteal, inguinal, and thigh skin folds may exist. In most infants older than 3 to 4 months, abduction will be limited, and the Barlow and Ortolani tests may no longer be positive. The affected extremity may appear short in comparison with the normal side.

When an infant presents at age 3 to 6 months and the femoral head has been dislocated for several months, it may not be possible to relocate the hip during examination. The hip appears more adducted. This is secondary to contracture of the adductor musculature, which can be demonstrated by palpation of the adductor tendon at the pubic tubercle. Significant asymmetry of skin folds is more common at this stage. Slight proximal migration of the hip also may be observed. Palpation of the greater trochanter will indicate that the involved hip is higher than the opposite hip. This abnormality can be confirmed by leg length measurement.

When a child presents after walking has begun, the chief complaint may be a limp. This gait pattern is the result of ipsilateral hip abductor weakness.

When congenital dysplasia of the hip is suspected in the infant, ultrasonography of the hips should be obtained. This should be performed by an experienced radiologist because this is the most sensitive method of making the proper diagnosis. In the child older than 3 or 4 months, x-rays should be obtained (Fig. 16.8). When the hip is subluxed or dislocated, Shenton's line (the continuous curve of the inner margin of the femoral neck and the inner margin of the obturator foramen) is broken. Ossification of the capital femoral epiphysis often is delayed; therefore, the bony portion of the epiphysis may be absent or appear smaller in the dysplastic hip. When the femur is laterally subluxed, its malposition may be demonstrated relative to a vertical line (Perkins line) drawn from the lateral lip of the acetabulum perpendicular to a line drawn through the triradiate cartilage of the two hips (Hilgenreiner line). The beak of the medial metaphysis of the normally articulating femur lies well medially to this line, while the beak of the neck of the dislocated femur lies closer to or laterally to this line. When the acetabulum is dysplastic, the slope of its superior margin is increased.

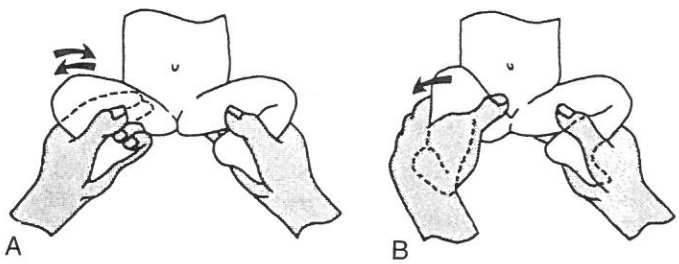

Figure 16.7. Infant hip examination for instability. *A*, Barlow sign. Adduction causes posterior subluxation or dislocation of the reduced hip. *B*, Ortolani sign. Abduction causes reduction of the subluxed or dislocated hip.

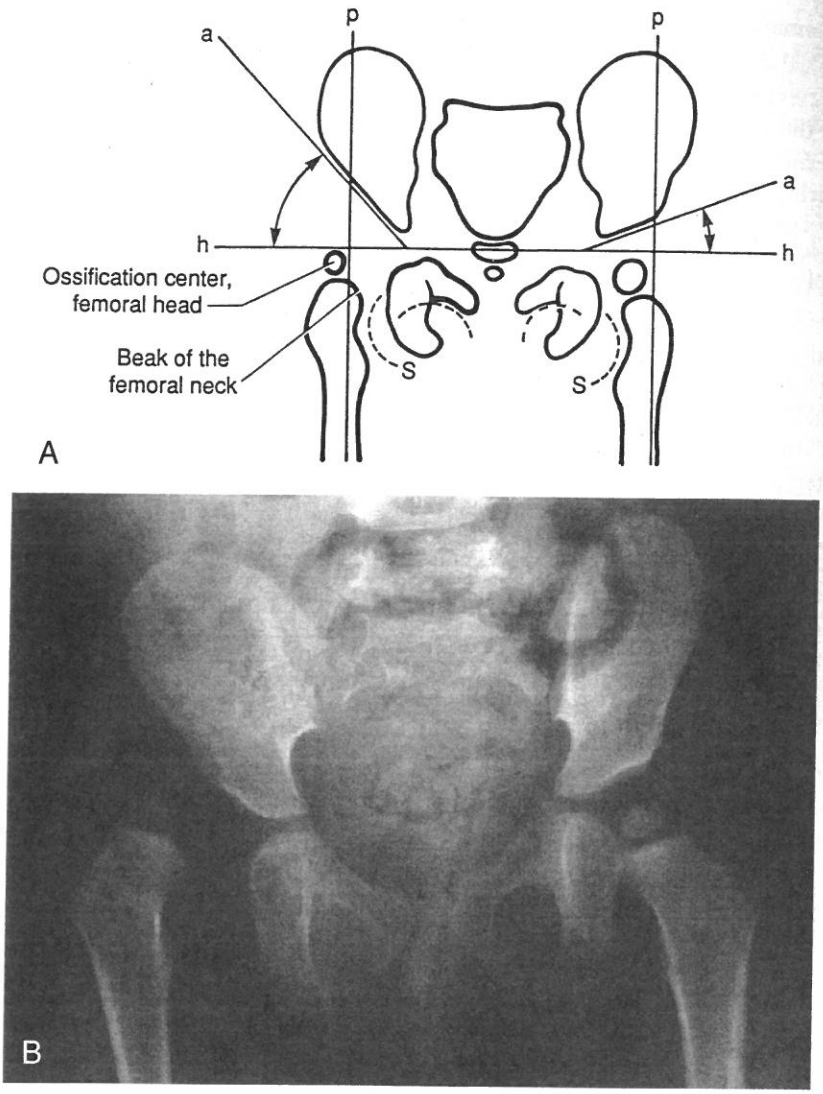

Figure 16.8. *A,* x-ray characteristics of congenital hip dislocation. 1. The arc of Shenton's line (*S*) is discontinuous on the dislocated side. 2. The beak of the femoral neck and the ossification center of the femoral head are displaced laterally and upward toward or beyond the Perkins line (*p*) and the Hilgenreiner line (*h*). 3. The angle between the acetabular line (*a*) and the Hilgenreiner line (*h*) is increased. *B,* plain x-ray of a 12-month-old female shows changes analogous to those in part *A*.

Treatment

Once the diagnosis of developmental dysplasia of the hip is made, referral to the orthopaedist is indicated. The principle in treatment of the dysplastic hip is to obtain or maintain reduction to allow for normal development. For patients who present from birth to 6 months, successful treatment can be achieved with a Pavlik harness. This is successful in up to 90% of cases. Failure is more commonly associated with bilaterality, "Ortolani negative" hips, and older infants. If treatment with the harness is unsuccessful, further measures are required including traction, closed reduction under anesthesia, and possible surgical intervention. In a child presenting

older than 6 months, treatment is more difficult. Success of the abduction device is less predictable, and the need for closed reduction under anesthesia or surgical treatment is more likely.

TRANSIENT OR TOXIC SYNOVITIS OF THE HIP

This is a self-limited synovitis of the hip joint most commonly seen in children between the ages of 3 and 5 years. The etiology is unclear, but unrecognized injuries and viral infections have been considered. This condition must be differentiated from pyogenic arthritis and from avascular necrosis of the hip (Legg-Calvé-Perthes disease). When suspicion of pyogenic arthritis exists, the joint must be aspirated. The distinction from aseptic necrosis is made by clinical follow-up and x-ray.

Clinical Characteristics

The child presents with complaints of pain in the groin, anteromedial thigh, or knee. Also the child has an associated limp. Duration of pain is from several days to 2 weeks. Low-grade fever may be present with slight leukocytosis and slight increase in the sedimentation rate. The affected hip is maintained in a flexed and externally rotated position. The child resists efforts to move the hip rapidly or to the extremes of motion. This contrasts with septic arthritis in which the pain is usually more severe and the fever, white blood count, and sedimentation rate are more elevated. X-rays are unremarkable in both conditions except for evidence of soft tissue swelling and an effusion, which may be subtle.

Treatment

Initial treatment should consist of bed rest. If septic arthritis cannot be ruled out on the basis of the physical examination and hematology, hospital admission for close observation should be considered. Crutches should be used with nonweightbearing gait. Acetaminophen or ibuprofen may be given on a symptomatic basis. If simple bed rest is not adequate, a few days of Buck's traction (Chapter 10) will almost always produce symptomatic relief. This can be done at home with orthopaedic consultation. Patients may begin full weightbearing when they have regained full, painless range of motion and can ambulate without pain and without a limp. They may then return to normal activity as tolerated. If pain is persistent or recurrent, or if pyogenic arthritis or aseptic necrosis is suspected, orthopaedic consultation should be obtained promptly. Even if the child becomes asymptomatic and returns to normal activity without restriction, a follow-up visit at 8 to 12 weeks is recommended. At that time, a repeat x-ray should be obtained to rule out aseptic necrosis.

AVASCULAR NECROSIS OF THE FEMORAL HEAD (LEGG-CALVÉ-PERTHES DISEASE)

This condition affects children between the ages of 2 and 11 years old. The cause of the interruption of the blood supply to the femoral head is not known. After infarction, reparative tissue grows into the necrosed head, and healing occurs by a process of creeping substitution with resorption of dead bone and deposition of new bone. The process of necrosis and reconstitution can take 2½ years. Deformity of the femoral head and acetabulum may occur if the disease is extensive and exceeds the remodeling capacity of the developing epiphysis. Younger children have a better prognosis as do those children with smaller areas of involvement of the femoral head.

Clinical Characteristics

Although the disease may occur in children between the ages of 2 and 11 years, the incidence is highest in children between 5 and 9 years of age. Pain is usually in the groin, anterior thigh, and sometimes the knee. Some children present without pain, but almost all limp. The onset is usually insidious, and the patient has frequently been symptomatic for several months before presentation. Limping increases with activity and may be intermittent. The condition frequently presents with synovitis, which is associated with muscle spasm and restricted hip abduction and

rotation. X-ray changes are variable depending on the stage of the disease. The first bony change noted may be a subtle subchondral crescent-shaped radiolucency of the femoral head. This is followed by more diffuse sclerosis and later, an irregular mottled appearance as the head undergoes fragmentation (Fig. 16.9). Patients with a poorer prognosis are older and have more extensive involvement of the femoral head. They are at risk of developing early osteoarthritis. The condition is bilateral in 15 to 20% of cases.

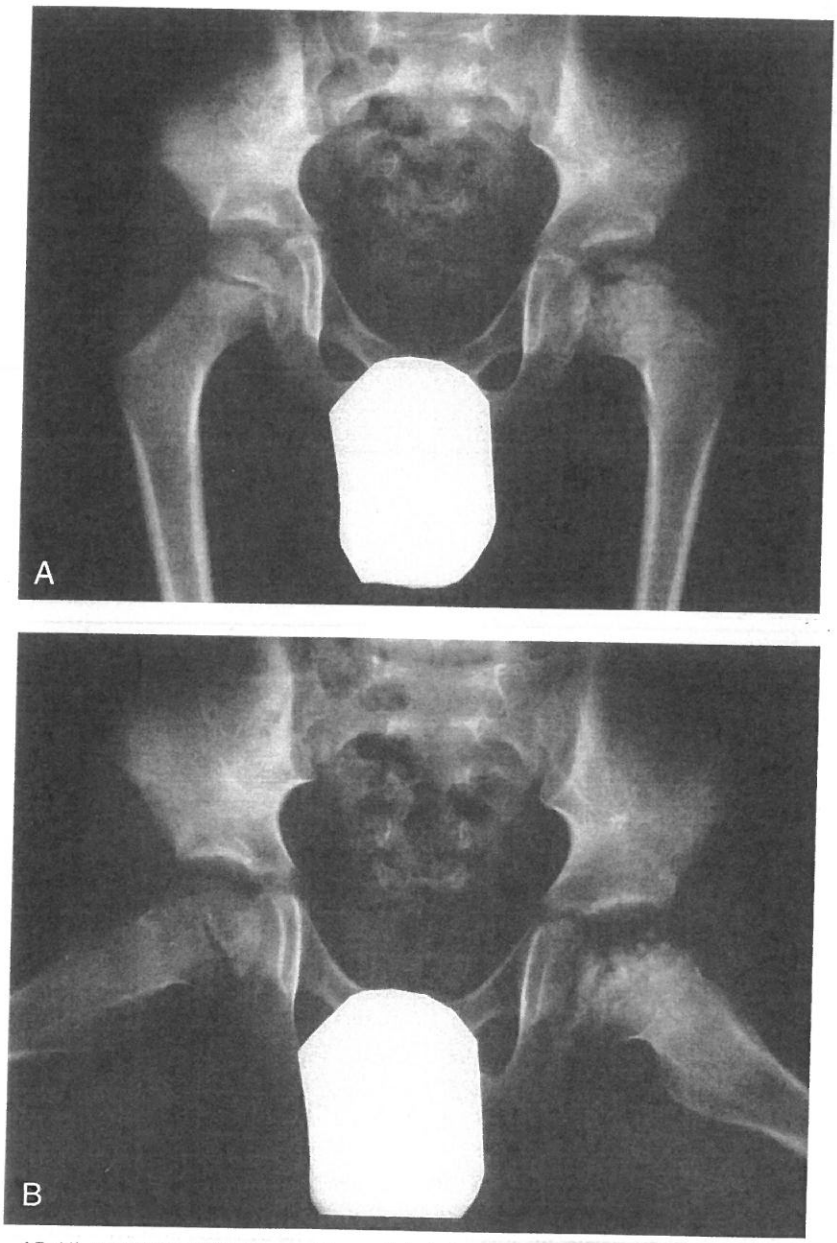

Figure 16.9. AP (*A*) and lateral (*B*) views of Legg-Calvé-Perthes of the left hip. X-rays demonstrate partial collapse, lateral subluxation, and irregular, mottled appearance as a result of advanced fragmentation and early repair.

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Treatment

Orthopaedic referral should be made. Treatment is based on the principle that hip mobility and containment of the femoral head within the acetabulum will lead to the best healing and remodeling of the infarcted femoral head. Patients who are young (4 to 6 years old) and who have involvement of less than 50% of the head simply may be observed without active treatment. For patients with a less optimistic prognosis, containment of the femoral head within the acetabulum can be achieved with physical therapy, home traction, and sometimes an external brace or cast. For some patients, osteotomy of the femur or pelvis is necessary.

One of the problems for the primary practitioner is distinguishing the child with avascular necrosis from the child with toxic synovitis of the hip. X-rays are most important in making this distinction. Orthopaedic consultation is indicated if avascular necrosis is suspected. It also is important to re-examine patients who present with symptoms of "nonspecific synovitis" to make certain that the diagnosis is correct.

SLIPPED CAPITAL FEMORAL EPIPHYSIS

During the period of rapid growth during adolescence, the capital femoral epiphyseal plate is relatively weak. The exact cause of this condition remains unknown. Various etiologies have been suggested including hormonal dysfunction. Whatever the cause, it appears that the stresses of normal activity exceed the strength of the growth plate through the zone of cartilage hypertrophy. This results in progressive slippage of the capital femoral epiphysis in a posterior and medial direction. In most patients, the condition has an insidious onset; however, in some there is an acute presentation associated with injury. Many patients report some preinjury pain and have an acute slip superimposed on a chronic condition.

Clinical Characteristics

A child between the ages of 10 and 16 years presents with a history of insidious hip, thigh, or knee pain associated with a limp. The patient may also present with acute pain after an injury, superimposed on a history of intermittent pain. Males are more commonly affected, and the patient is often obese with somewhat delayed development of secondary sexual characteristics. The condition may be bilateral and the opposite hip must be examined. A "silent slip" on the contralateral side should always be sought when the diagnosis is made on one side. In the acute phase, there is significant muscle spasm and synovitis with restricted range of motion; internal rotation usually is significantly limited. Because of the limitation in internal rotation, the extremity tends to externally rotate when the hip is flexed. X-rays demonstrate posterior and medial displacement of the epiphysis (Fig. 16.10). The change may be subtle and often apparent only on the lateral view of the hip.

Treatment

Once the diagnosis is made, orthopaedic referral and treatment should be obtained. The treatment is surgical (Fig. 16.11), unless the diagnosis is late and the growth plate is already closed. With small or moderate degrees of slip, fixation of the epiphysis may be performed in situ with a single screw. This results in fusion of the epiphysis and long-term stability. Shortening of the extremity usually is not a problem. In slips of a greater degree, more extensive surgical reconstruction may be necessary.

ANTERIOR KNEE PAIN

Knee pain is a common complaint in childhood, especially among rapidly growing adolescents. The differential diagnosis includes a variety of disorders that often can be identified by careful physical examination. Fortunately, the most worrisome diagnoses are seen infrequently and include juvenile rheumatoid arthritis, osteochondritis dissecans of the lateral femoral condyle, infection, and tumor. Conversely, the most common diagnoses are usually self-limiting and may resolve with analgesics and physical therapy. These include

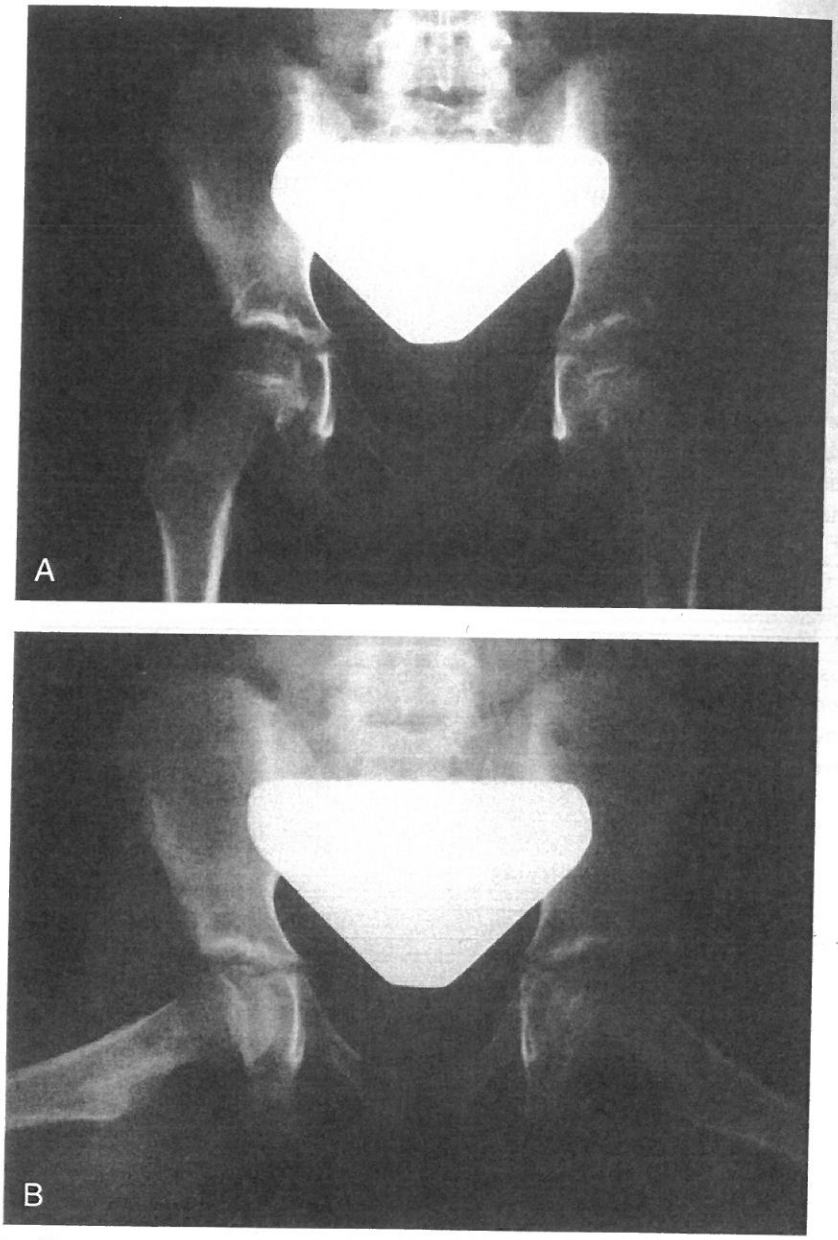
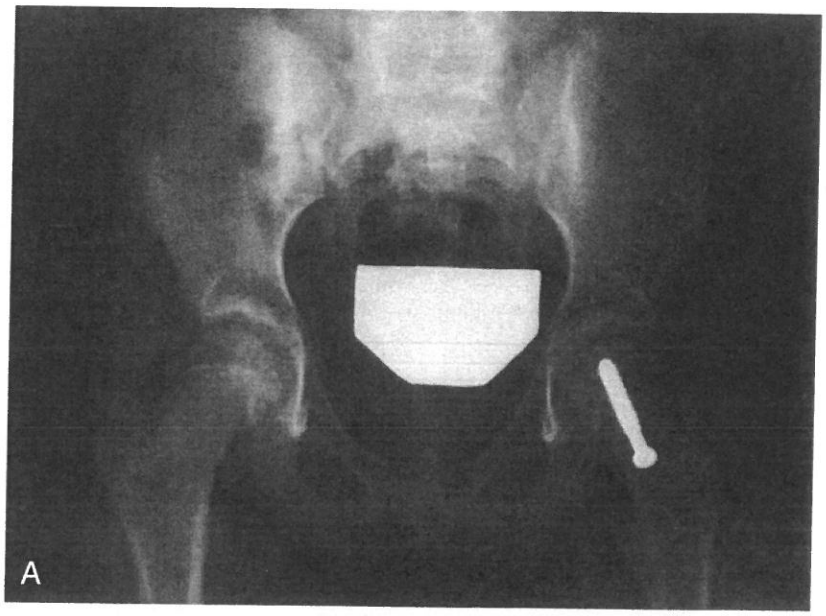


Figure 16.10. AP and lateral views of a low-grade slipped capital femoral epiphysis. *A*, normal appearing AP view of both hips. *B*, note the subtle loss of the contour of the femoral neck and head on the lateral view.

Osgood-Schlatter disease (tibial tubercle apophysitis), Sinding-Larsen-Johansson disease (apophysitis of the distal patella), abnormalities of patellar tracking, bursitis, and tendinitis.

Chondromalacia patella is a pathological term that has been grossly overused in the diagnosis of anterior knee pain in adolescents. It describes gross histologic abnormalities of the patellar articular cartilage, which is infrequently present. The preferred terminology is patellofemoral disorder if symptoms arise from this articulation, or simply adolescent



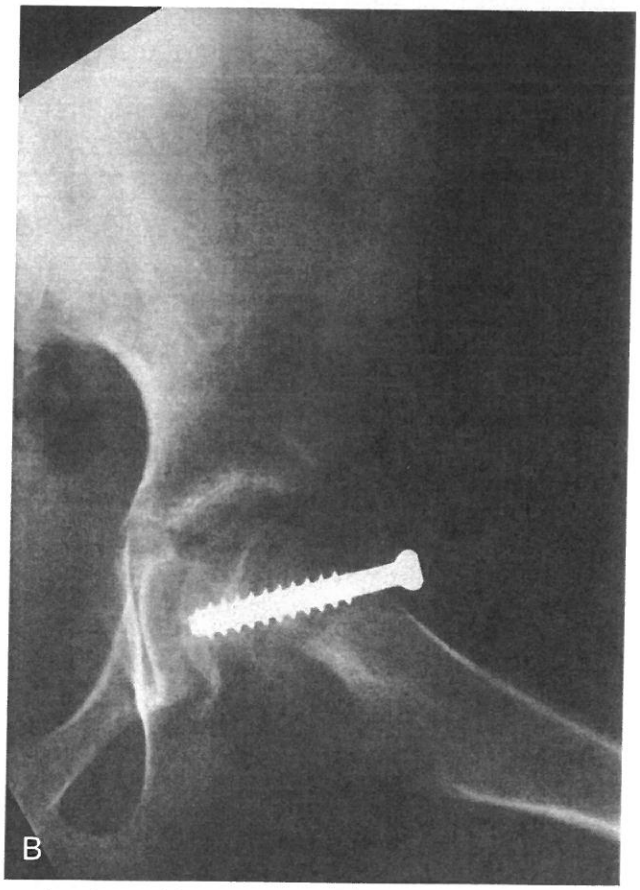


Figure 16.11. Postoperative views of the same patient seen in Figure 16.10. The close-up of the lateral x-ray shows the true magnitude of the slip.

anterior knee pain if a more precise diagnosis cannot be made. (For a more detailed discussion of several of these topics, see Chapter 7.)

TIBIAL APOPHYSITIS (OSGOOD-SCHLATTER DISEASE)

Apophysitis of the tibial tubercle occurs at the time the apophysis undergoes the transition from cartilage to bone. This disease commonly occurs between 12 and 14 years of age. Traction of the patellar ligament on the apophysis may result in microfractures.

Clinical Characteristics

Pain in the region of the tibial tubercle is related to activity, and it may be relieved by rest. Swelling and tenderness are present over the tibial tubercle and along the patellar ligament. A lateral x-ray may or may not show irregular ossification or fragmentation of the tibial tubercle. Although this is not evident early in the disease, irregular ossification or fragmentation may persist long after the disease has become asymptomatic.

Treatment

Instruct the child to avoid all activity that requires resisted knee extension, such as climbing, running, or kicking, until pain and tenderness have fully remitted. This restriction usually applies for 6 to 8 weeks. In addition, a program of gentle quadriceps and hamstring stretching may be combined with a mild analgesic and application of ice when symptoms are acute. This treatment applies to many of the common knee pain syndromes and is likely to be more effective than strengthening programs. Prolonged restriction of activity is not appropriate. Although activity may exacerbate symptoms, it does not cause long-term morbidity.

When pain is severe and recurrent, casting in a walking cylinder for 2 to 3 weeks lessens symptoms. When pain persists, the usual cause is the presence of ossicles within the tuberosity or tendon, which can be seen on x-ray. Surgical excision of the loose ossicles, although rarely necessary, will relieve symptoms. This procedure may be indicated either before or after skeletal maturity.

NECROSIS WITHIN THE POLES OF THE PATELLA (SINDING-LARSEN-JOHANSSON DISEASE)

This condition occurs in children 8 to 13 years of age. Its presentation is similar to Osgood-Schlatter disease except that maximal tenderness is localized to the distal pole of the patella. The initiating event is unknown. Clinical characteristics include pain that occurs during resisted extension of the knee and during kneeling. Onset is insidious and activity related. Swelling and tenderness also is present over the inferior pole of the patella. X-rays may or may not show fragmentation of bone near the affected pole. Treatment and prognosis are the same as those presented for Osgood-Schlatter disease.

NECROSIS WITHIN THE CONDYLAR EPIPHYSES OF THE FEMUR (OSTEOCHONDRITIS DISSECANS)

Osteochondritis dissecans (OCD) is the result of avascular necrosis in the area of subchondral bone. The lateral aspect of the medial femoral condyle is involved in 75% of cases, but the lateral condyle or patella may be affected (Fig. 16.12). The result is a small area of bone, usually around 1 cm, which is biologically separated from the remaining bone by an area of necrosis. These lesions may heal, particularly if the growth plates are open. The etiology is probably traumatic from impingement of the tibial spine on the femoral condyle.

Clinical Characteristics

OCD is characterized by aching pain in the knee at rest that worsens with weightbearing, causing the patient to limp. The onset is insidious. Physical examination may demonstrate a restricted range of motion, but it is usually normal. Rarely, an effusion will be evident. If the fragment detaches, it may produce locking or symptoms of a loose body. AP, lateral, and

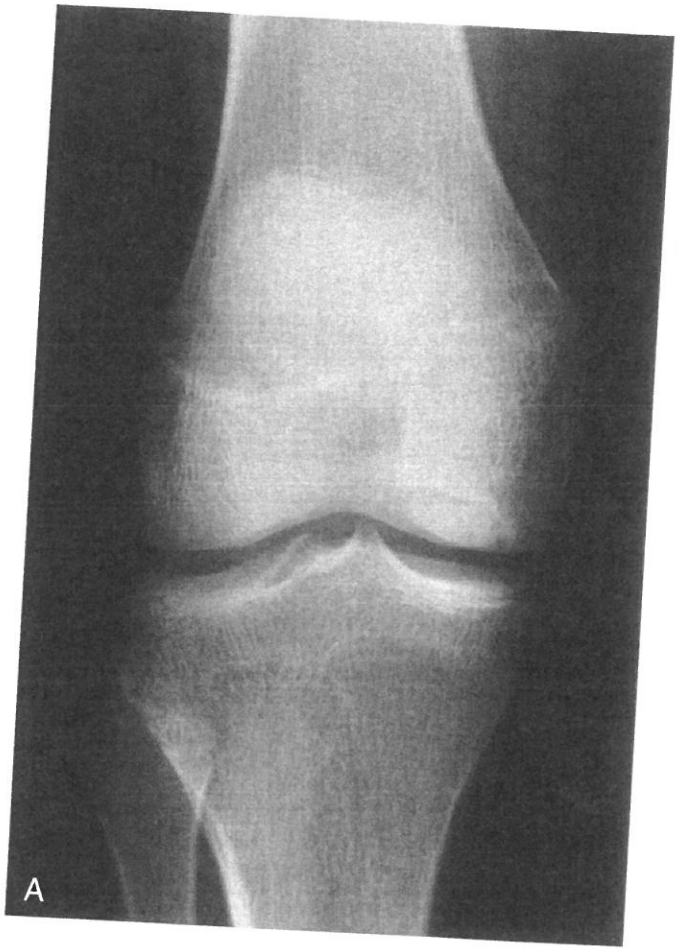


Figure 16.12. AP *(A)* and lateral *(B)* views of the knee of a 14-year-old female with anterior knee pain show the typical location of osteochondritis dissecans in the lateral aspect of the medial femoral condyle.

tunnel x-rays of the knee will usually demonstrate a characteristic lesion that appears as a half-moon defect or irregularity of the subchondral bone. The physician should always obtain bilateral x-rays, because physiologic irregular ossification of the femoral condyles can easily be confused with OCD, although the former is almost always bilateral and the latter, less commonly.

Treatment

Because the prognosis is unpredictable, at the outset the patient should be referred to an orthopaedist. A decrease or elimination of weightbearing with immobilization may be prescribed until the lesion heals. This treatment may be prolonged, and its efficacy has not been confirmed. If the fragment separates completely, it can become a loose body that may have to be removed. The remaining defect can be drilled to promote revascularization and healing, or if the fragment is large, it can be fixed by screws. Most recent technology allows cartilage grafting for large defects in the weightbearing area, although this is experimental.

ABNORMALITIES OF PATELLAR TRACKING

Anterior knee pain may be a manifestation of abnormal patellar tracking. This is more common in females, especially near the adolescent growth spurt. The spectrum of abnormality ranges from subtle deviation of the patella from the center of the femoral condyles to frank dislocation. Predisposing factors

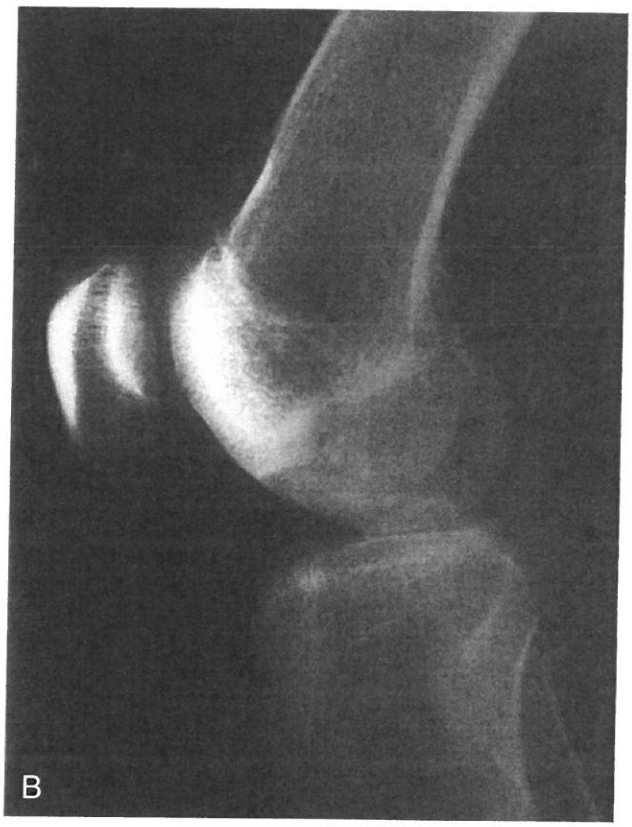


Figure 16.12. *(continued)*

include relative weakness of the vastus medialis muscle, ligamentous laxity, and valgus alignment of the tibio-femoral "Q-angle." Observing the position of the patella as the straight knee is flexed will demonstrate lateral deviation in a "J" pattern. In addition, it may be possible to markedly translate the patella laterally, and the patient may experience pain or apprehension of the patella dislocating if the examiner attempts to translate it too far. For maltracking without dislocation, strengthening the quadriceps muscle, especially the vastus medialis, is indicated. Acute treatment of patellar dislocation should include a 4- to 6-week period of immobilization in a knee immobilizer, or a cylinder cast. This should be followed by a period of vigorous quadriceps strengthening. Recurrent dislocation may require surgical realignment of the extensor mechanism.

ALIGNMENT PROBLEMS

Angular and rotational problems are among the most common reasons for referrals to a pediatric orthopaedic surgeon. Among these, bowlegs and in-toeing are by far the most frequently seen. The bones of the lower extremities remodel rapidly until 6 to 8 years of age, and most physiologic deformities will completely resolve without treatment by this time. The challenge is to identify the small subset of children who are at risk of progressive deformity.

Angular Deformities

Angular deformities may occur in the coronal plane or in the sagittal plane. They are named according to the apex of the angle. In the coronal plane, these include bowlegs (genu vara) and knock-knees (genu valga). Either of

these may be physiologic or pathologic. In the sagittal plane, the deformities are usually posteromedial or anterolateral—these are always pathologic.

Physiologic tibia vara usually is noted from infancy to approximately 24 months of age. This is replaced by a variable amount of valgus from 24 months through the age of 5 to 6 years. By then, the knee will usually have assumed the normal adult value of 5 to 8° of knee valgus. In contrast, pathologic tibia vara does not follow the aforementioned pattern of growth and becomes progressively worse. Initially, it may be impossible to distinguish between physiologic and pathologic bowlegs by clinical evaluation alone, and x-rays of the entire limb from hips to ankles are often indicated. These x-rays may show that the deformity represents Blount's Disease (Fig. 16.13) in which an abnormality of the medial tibial growth plate is present or a metabolic bone disease is present in which the epiphysis, physis, or metaphysis is affected. If it is impossible to determine with certainty whether bowing is physiologic or pathologic, a child with a significant deformity should be referred to an orthopaedic surgeon. Surgery is limited to severe or progressive deformities.

Valgus deformity of the lower extremities is less common than varus deformity. Like bowlegs, this valgus deformity is usually a physiologic alignment that will remodel. Valgus deformity can be associated with certain pathologic states such as juvenile rheumatoid arthritis or hypoplasia of the lateral femoral condyle. Bracing is not usually effective or indicated. As in bowlegs, surgical treatment is only indicated in severe cases. Early arthritis is not usually a problem.

Angular deformities in the sagittal plane are much less common and include anterior and posterior angulation. These are usually noted at birth and invariably require specialized treatment by an orthopaedic surgeon.

Posteromedial Angulation

In posteromedial bowing, the angle of the bow is almost always directed posteriorly and medially, but rarely may be purely posterior. The etiology is unknown but it may be caused by intrauterine positioning of the infant, resulting in tight anterior compartment musculature and limited plantar flexion of the ankle.

Treatment

Treatment is initially nonoperative. Passive stretching of the tight musculature is required and occasionally a total contact orthosis is used. The angular deformity usually corrects by about 4 years of age, but a residual limb length discrepancy usually remains and may require a limb length equalization procedure at the appropriate time. Osteotomies of the tibia and fibula are not indicated in this deformity.

Anterolateral Angulation

This direction of angulation is more serious, and the deformity may be caused by congenital deficiency of the fibula, congenital pseudarthrosis of the tibia, or fibrous dysplasia.

In fibular hemimelia, the fibula may be short or completely absent. It is usually associated with abnormalities of the foot and often the femur as well. Treatment may range from limb length equalization procedures to an amputation of the foot. Congenital pseudarthrosis of the tibia is a condition in which a portion of the diaphysis of the tibia fails to develop normally and is partially or completely replaced by fibrous tissue. This condition is associated with neurofibromatosis over 50% of the time and is infrequently seen with polyostotic fibrous dysplasia. In either case, management is extremely complex and involves making the correct diagnosis, correcting the angular deformity, and achieving bony union and maintaining it through adulthood.

Rotational Problems

In-Toeing

In-toeing is a common pediatric lower extremity disorder. Most cases can be evaluated and definitively treated by the primary care physician.

Although in-toeing frequently needs no

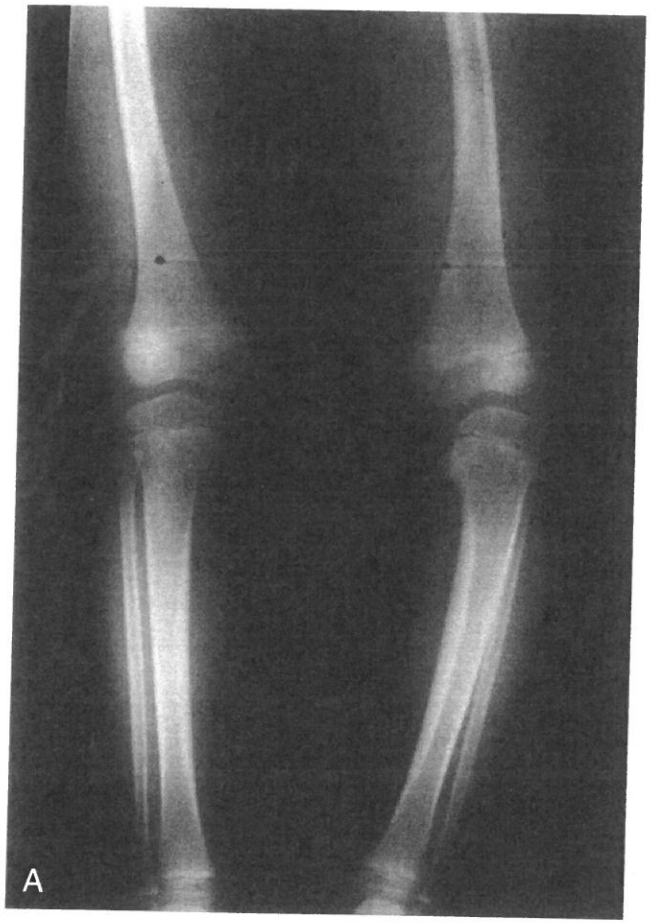


Figure 16.13. *A*, a 3½-year-old female with a 1-year history of progressive bowing of the left knee. X-rays show genu varum with sloping of the medial proximal tibial epiphysis and metaphysis consistent with Blount's disease. The tibia is subluxed on the femoral condyles. Surgery is indicated in this child with advanced disease. *B*, close-up of the left knee.

specific treatment, the physician must understand the causes and explain the natural history of the deformity to the patient's parents. The foot progression angle is used to document the presence and magnitude of in-toeing (Fig. 16.14). The foot progression angle has a wide range of normal, from 5° of turning in to 20° of turning out. Although the patient will present with a chief complaint of turning in of the feet, it is important to remember that the etiology may be abnormal rotation of the femur, the tibia, or the foot. In-toeing may be caused by one or more of the following: (*a*) increased femoral anteversion (Fig. 16.15), (*b*) increased internal tibial torsion (Fig. 16.16), or (*c*) metatarsus adductus. Each of these entities has specific physical findings, natural history, and treatment options, and each will be discussed separately. It is important to be aware that the natural history of these deformities is spontaneous resolution in over 90% of cases. Those individuals with residual anatomical deformities can almost always overcome their problem by consciously altering the gait, leaving few children who actually require active treatment.

Femoral Anteversion

Femoral anteversion is the angle of the femoral neck relative to the femoral condyles in the sagittal plane (Fig. 16.15). In the average adult standing with the femoral condyles directed straight ahead, the femoral necks will angle forward about 15° to enter the acetabula. Normal infants may have up to 45° of anteversion, which gradually decreases to an average

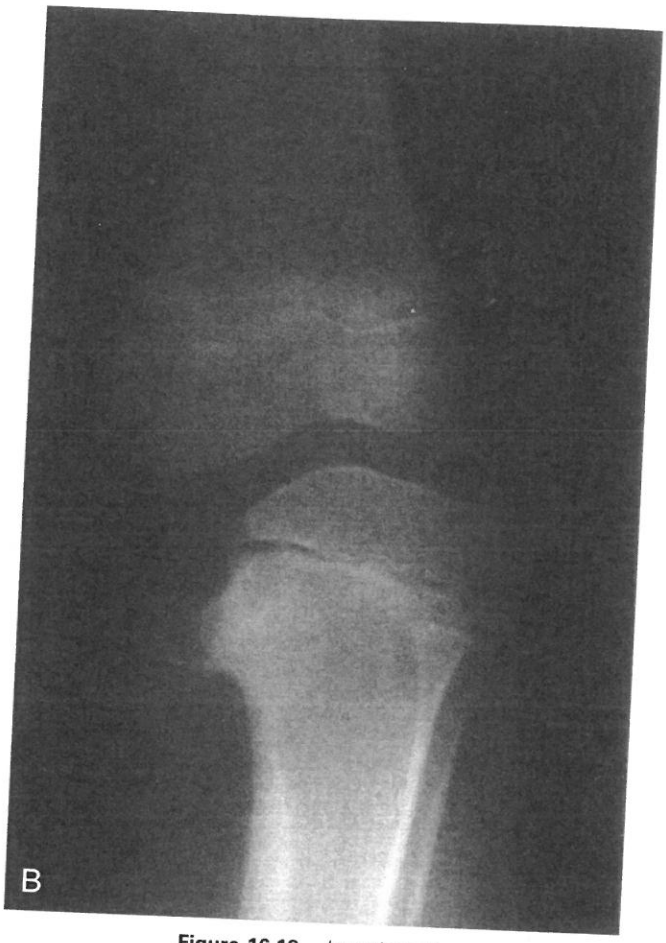


Figure 16.13. (*continued*)

of 15° during adolescence. If the amount of anteversion is increased, the hip will have more internal rotation and less external rotation. This will allow increased internal rotation of the entire leg during gait, thus causing in-toeing. Because of soft tissue contracture, normal infants may have much more external rotation of the hips than internal rotation. Normally, an older child will have slightly more external rotation than internal rotation of the hips, measured with the child in the prone position and the knees flexed (Fig. 16.17). If a patient is found to have more than 70° of internal rotation of the hips and less than 30° of external rotation of the hips, then femoral anteversion is causing or contributing to the in-toeing.

Treatment Various devices such as twister cables, external rotation (Denis Browne) splints, and shoe wedges have been used in the past to treat femoral anteversion. However, these are either ineffective or may cause secondary deformities such as excessive external tibial torsion or foot deformities and are rarely indicated. The parents can be reassured that most femoral anteversion resolves by adolescence and that the use of braces has not been shown to be any more effective than no treatment at all. The physician should explain to the parents that femoral anteversion has not been shown to cause back pain, hip pain or hip arthritis, flatfeet, or any problems with sports participation. Femoral anteversion plus abnormal external tibial torsion, however,

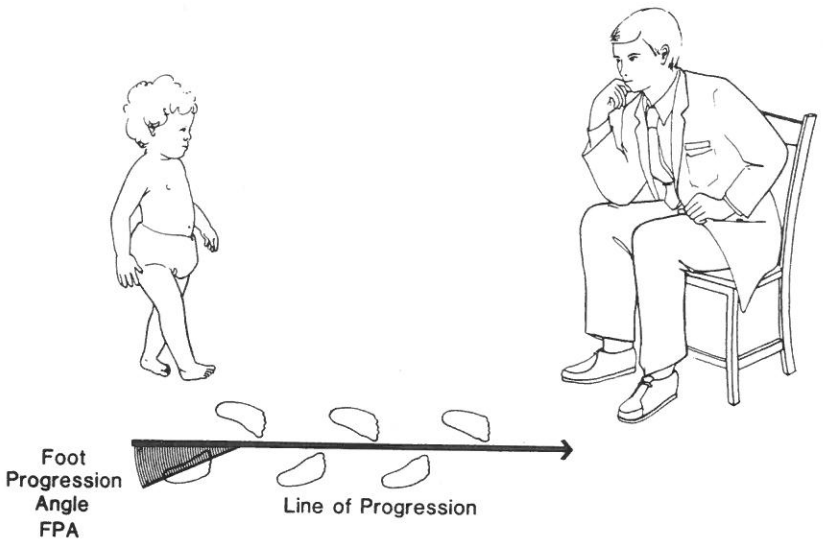


Figure 16.14. Foot progression angle. The angle formed by the direction of the foot relative to the line of progression of gait.

may cause patellar malalignment and subsequent knee symptoms. In the rare case of severe femoral anteversion persisting in late childhood, only derotational osteotomies of the femurs will change the anteversion and correct the in-toeing, and orthopaedic referral will be necessary.

Internal Tibial Torsion

Tibial torsion is the angular relationship of the medial and lateral malleoli to the coronal plane of the knee. This is best measured as the thigh-foot angle with the child prone (Figs. 16.16 C and D). If the transmalleolar axis and, therefore, the foot are internally rotated compared with the thigh, then internal tibial torsion is present. If the foot and ankle are externally rotated to the thigh, then external tibial torsion is present. Tibial torsion may be associated with physiologic genu vara, and the combination of the two deformities can be striking. The range of normal tibial torsion is variable, and up to 20° of internal tibial torsion is normal in infants. The transmalleolar axis normally becomes more externally rotated during childhood, resulting in 15 to 20° of external tibial torsion by adolescence. If internal tibial torsion persists, the ankle joint will be internally rotated compared with the knee, and in-toeing will result. During gait, the child with in-toeing secondary to femoral anteversion will be noted to have both patellae turned inward toward each other (because the inward rotation occurs at the level of the hips), whereas in a child with internal tibial torsion, the feet will turn in, but the patellae will not (because the inward rotation occurs below the knees).

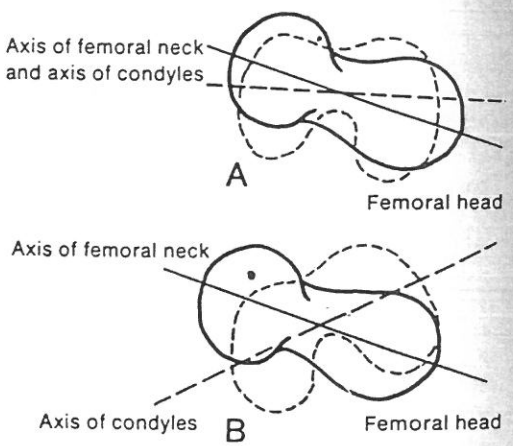


Figure 16.15. Anteversion of the femur.
A, normal axis. *B*, the axial twist of anteversion.

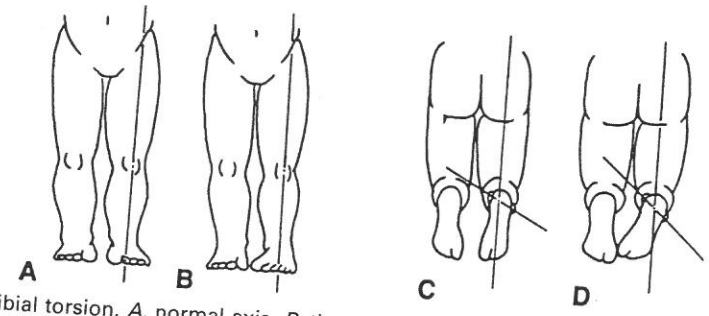


Figure 16.16. Tibial torsion. *A*, normal axis. *B*, the axial twist of internal tibial torsion. *C*, normal thigh-foot angle. *D*, thigh-foot angle in internal tibial torsion.

Treatment Internal tibial torsion, like femoral anteversion, improves spontaneously with growth, and usually only reassurance is needed rather than active treatment. Special shoes, shoe wedges, and casts are ineffective in treating internal tibial torsion, but in severe cases, an external rotation splint may be recommended. The indications for this device and its efficacy are questionable, and many clinicians will prefer to simply observe the child. If severe internal tibial torsion persists, orthopaedic referral is indicated for possible tibial osteotomies near skeletal maturity.

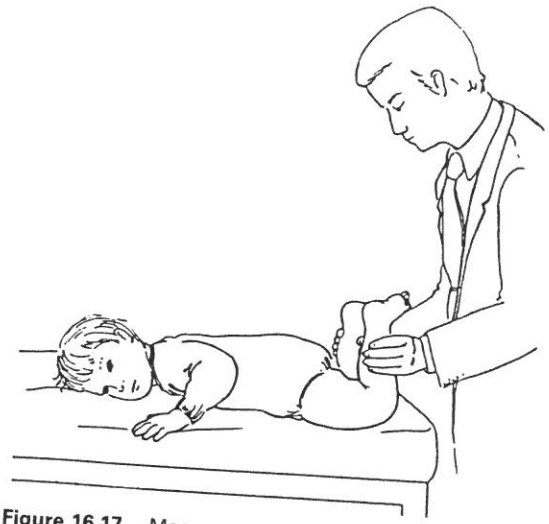


Figure 16.17. Measurement of hip rotation in prone position.

FOOT DEFORMITIES

Metatarsus Adductus

The final cause of in-toeing is metatarsus adductus, a deformity of the foot itself. In patients with metatarsus adductus, the alignment of the legs will be normal from the hips down to and including the hindfoot. However, the forefoot will be adducted or turned inward relative to the hindfoot (Fig. 16.18E). This is best seen when the foot is examined from the bottom. Normally, the lateral border of the foot will form a straight line from the heel to the fifth toe. In metatarsus adductus, there will be a break or curve at the midfoot such that, even with the entire leg and hindfoot directed straight ahead, the forefoot will turn in toward the midline of the body.

Treatment

Metatarsus adductus is a common positional deformity of the newborn and, unless associated with a deformity of the hindfoot (such as a true clubfoot), it usually corrects with growth and stretching. Parents should be instructed to perform passive stretching exercises with each diaper change. If the metatarsus adductus is rigid at birth, serial casting may be instituted to assist in rapidly correcting the deformity. When the infant is 1 to 3 months old, the metatarsus adductus is evaluated again to determine severity and also flexibility, which is measured by how easily the foot can be passively overcorrected by the examiner. If significant metatarsus adductus persists to the age of 4 to 6 months, it can usually be well-corrected by manipulation and serial

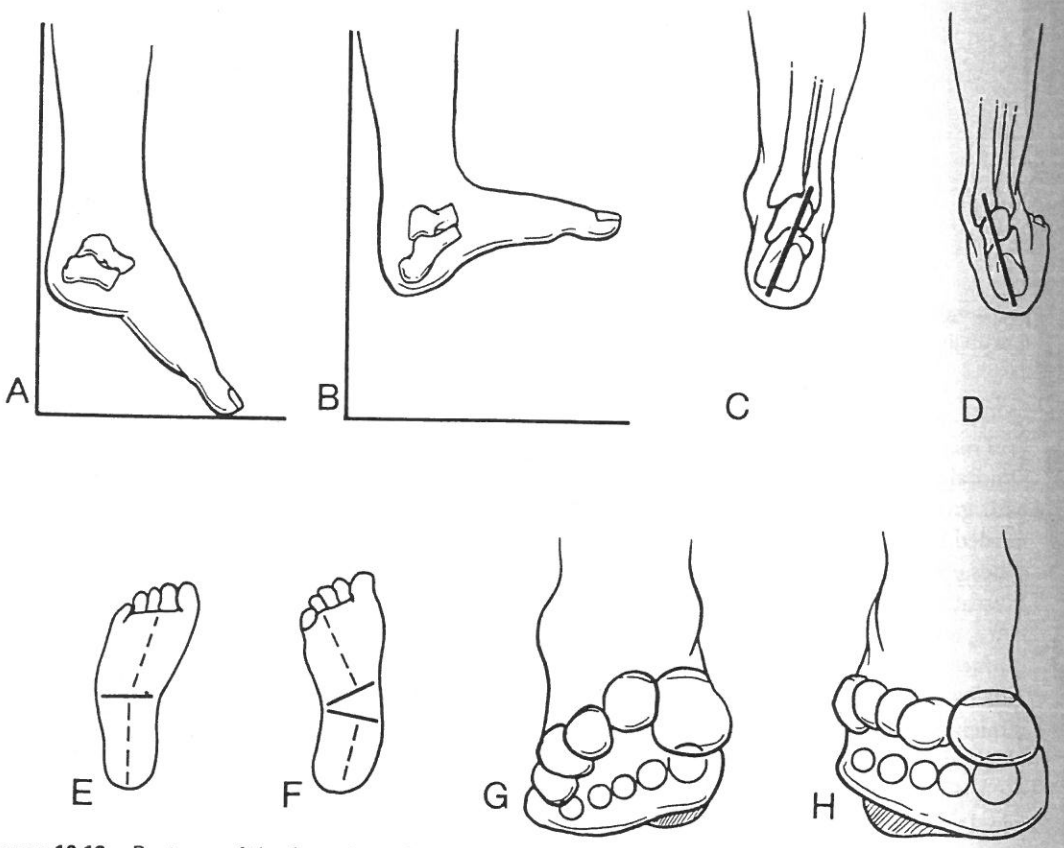


Figure 16.18. Postures of the foot. *A*, equinus. *B*, calcaneus. *C*, hindfoot varus (viewed from behind). *D*, hindfoot valgus (viewed from behind). *E*, metatarsus adductus (viewed from sole of foot). *F*, metatarsus abductus (viewed from sole of foot). *G*, forefoot supination (varus). *H*, forefoot pronation.

corrective casts. The proper molding of these corrective casts is difficult to perform, and because of potential skin problems, the authors recommend that these children be referred to the orthopaedic surgeon for cast treatment of persistent metatarsus adductus. A variety of shoes with modifications are available to assist in obtaining or maintaining the correction.

Flatfeet

Flatfeet, or pes planus, is divided into the common and rarely symptomatic flexible flatfoot and the uncommon but symptomatic rigid flatfoot. The differentiation between a flexible and rigid flatfoot is made by physical examination. In both types of pes planus, there is loss of the normal plantar arch when the child stands. However, in a flexible flatfoot, there will be a normal arch when the patient is sitting with the feet hanging over the side of the examination table as well as when the patient stands on tiptoes. Also, a flexible flatfoot will have normal motion of the subtalar joint. The patient with flexible flatfeet often will have other findings of increased ligamentous laxity, e.g., hyperextension of the knee or elbow, or a history of being "double-jointed." In children up to the age of 2, the fat pad on the medial side of the foot may give a false appearance of a flatfoot when the toddler is standing. Flexible pes planus is usually bilateral, often hereditary, and as mentioned previously, usually asymptomatic.

Longitudinal studies of large groups of children have shown that some flexible flatfeet

will gradually develop a plantar arch without treatment. No study has shown any significant difference in arch development in children with flexible flatfeet treated with orthoses versus simple observation. Therefore, orthotic treatment of asymptomatic flexible flatfeet is not usually recommended. However, orthotics may decrease symptoms in an older child with flexible flatfeet who has discomfort in the legs or feet.

In contrast to flexible pes planus, the rigid flatfoot is usually symptomatic. The rigid flatfoot will be flat with loss of the arch even when the child is nonweightbearing or standing on tiptoes. The rigid flatfoot has limited or absent motion of the subtalar joint. Peroneal muscle spasm also is frequently noted on physical examination, and hence the term "peroneal spastic flatfoot" is sometimes used interchangeably with rigid flatfoot. The most common cause of a rigid flatfoot is a tarsal coalition or fusion between the calcaneus and either the talus or navicular. The radiographic diagnosis of tarsal coalition is sometimes difficult and may require conventional tomography, CT scanning, or MRI evaluation, in addition to routine x-rays. However, any pathology involving the subtalar joint, such as arthritis, infection, or trauma, may result in a rigid flatfoot and must be considered in the differential diagnosis.

Treatment

If symptoms of a tarsal coalition persist after conservative treatment with immobilization, orthoses, and anti-inflammatory medication, surgery is often indicated. Therefore, patients with rigid flatfeet should be referred to the orthopaedist for evaluation.

NEWBORN FOOT DEFORMITIES

Significant foot deformities in the newborn usually will be referred to the orthopaedic surgeon, but these congenital deformities must first be recognized by the primary care physician. These include talipes equinovarus (clubfoot), calcaneovalgus foot deformity, and congenital vertical talus.

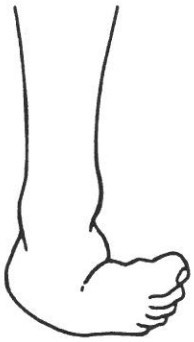


Figure 16.19. Talipes equinovarus (clubfoot).

Talipes Equinovarus (Clubfoot)

Talipes equinovarus is a congenital foot deformity in which the heel is in equinus (plantar flexed) and varus, and the forefoot is adducted (Fig. 16.19). The initial treatment is stretching combined with corrective casting. This manipulative treatment of clubfoot is much more effective when started immediately. Therefore, the orthopaedic surgeon should be consulted promptly so that treatment can begin within the first days of life. The foot is then treated by serial manipulation and casting, often for several months. If manipulation and casting are not successful in completely correcting the foot, surgery may be necessary. Surgery may be performed as early as 3 or 4 months of age, but in some cases can be delayed until the child is 1 to 2 years old.

Calcaneovalgus

The calcaneovalgus foot presents with dorsiflexion of the ankle and eversion and abduction of the forefoot (Figs. 16.18 B and F). In severe cases, the dorsal surface of the foot will be resting on the anterolateral aspect of the lower leg. Although this deformity may initially look as severe as a clubfoot, its prognosis is much better. A calcaneovalgus foot usually will respond within 1 to 3 months with simple passive stretching exercises. The parents are taught to stretch the foot down out of the dorsiflexed position and inward toward the midline of the body with each diaper change. In a severe case that does not correct well by the age of 2 to 3 months with passive stretch-

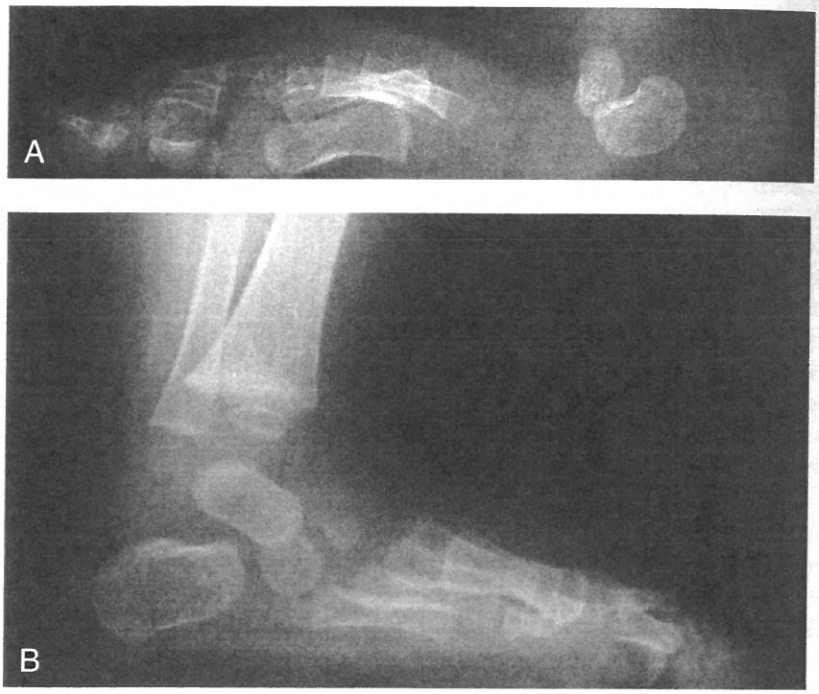


Figure 16.20. *A,* right foot of a 2-month-old child with congenital vertical talus. Note that the talus is nearly parallel to the axis of the tibia and is perpendicular to the axis of the calcaneus. *B,* comparison to a normal foot shows the talus at almost 90° to the tibia and the calcaneus at approximately 45° to the talus.

ing exercises, manipulation by the orthopaedic surgeon and a corrective cast may be necessary. Surgery is almost never required for a calcaneovalgus deformity of the foot. Because calcaneovalgus foot is the result of an intrauterine "packaging problem," there is a strong association with hip dysplasia, thus the hips should be carefully and repeatedly examined to rule this out.

Vertical Talus

A rare but severe congenital foot deformity is a congenital vertical talus (Fig. 16.20). In this deformity, the navicular is dislocated onto the dorsal surface of the talus, forcing the talar head down into a plantar-flexed position. The Achilles tendon is tight, pulling the calcaneus into a plantar-flexed position also. This results in a rigid flatfoot with a rounded "rocker bottom" appearance on the sole of the foot caused by the displaced head of the talus. This uncommon deformity should be referred to the orthopaedic surgeon immediately. This deformity is usually a manifestation of an underlying neurologic problem that should be carefully investigated. Manipulation may partially correct the deformity, but surgery usually is necessary to gain a satisfactory correction of this foot deformity.

Cavus Foot

The foot with a high arch and a plantar-flexed forefoot is called a cavus foot (Fig. 16.21). This foot deformity is frequently associated with hammertoes or claw toes and may or may not be symptomatic. A cavus foot should alert the physician to the possibility of an underlying neurologic disorder. Neurologic causes of a cavus foot include Charcot-Marie-Tooth disease, diastematomyelia, myelomeningocele, and polio. Therefore, examination of the spine, a careful neurologic examination, as well as a family history are necessary in the evaluation of a patient with

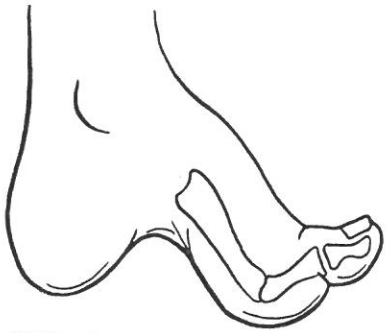


Figure 16.21. Cavus (high arch) foot. This deformity may be caused by calcaneus position of hindfoot (Fig. 16.18B) and/or equinus position of forefoot.

a cavus foot. Because of the plantar-flexed forefoot and the claw toes, there is frequently pain in the plantar aspect of the metatarsal heads. This pain may be relieved with orthoses and metatarsal pads or shoe modifications. Children with cavus feet should be referred to the orthopaedist for global evaluation, which may include consideration of surgical correction of the foot deformity.

TRAUMATIC DISORDERS

Developing bones and joints respond to injuries differently in several respects from fully developed and deteriorating bones and joints. The greater remodeling potential of growing bones and its effect on treatment plans were summarized under "General Principles of Treatment of Forearm Fractures" in Chapter 3. This ability of growing bones to remodel allows acceptance of positions that would not be acceptable in adult injuries. Growing joint capsules, ligaments, and muscles are more tolerant to prolonged immobilization than mature joint capsules, ligaments, and muscles. Thus, the need for prolonged immobilization does not commonly contraindicate closed methods of treatment in children as it does in adults. This is often important in the treatment of fractures of the radius, ulna, femur, and tibia in a child. Unfortunately, application of these principles is difficult to teach in a text. Consequently, until experience is gained, it is recommended that orthopaedic consultation be obtained when questions of this sort are problematic in the care of a specific injury.

Fracture patterns that involve the articular surface of a joint are much less common in children than they are in adults. Fractures in children that do involve the articular surface share the same risk involved in articular fractures in adults. Any fracture involving the articular surface in either a child or an adult should be referred to an orthopaedist.

PHYSEAL FRACTURES

Physeal fractures of the long bones usually do not interfere with the growth of the injured bone. These fractures occur through the zone of provisional calcification in the physis. The growth potential of the physis usually is not disturbed. However, if the fracture extends through the zone of proliferation into the epiphysis, there is a significant risk of interfering with the growth of the injured bone (Fig. 16.22). Fractures involving the growth plate have been classified into five groups, each of which presents different diagnostic and prognostic characteristics.

Salter I fractures are transverse fractures of the growth plate without injury to the bony metaphysis or epiphysis. When these fractures are undisplaced, they are not radiologically

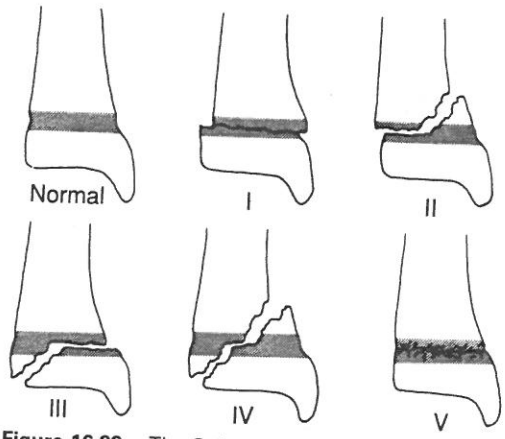


Figure 16.22. The Salter classifications of epiphyseal fractures.

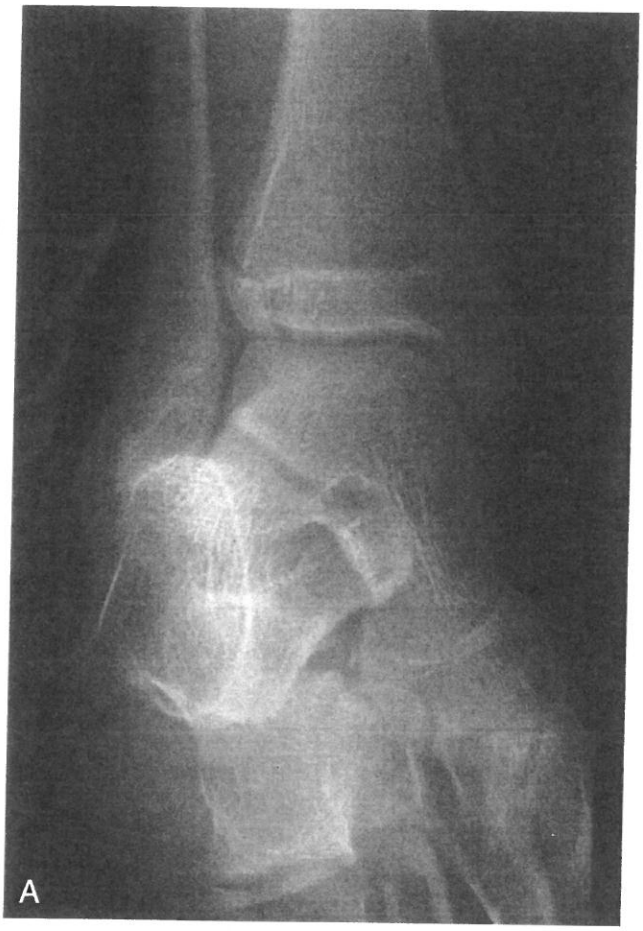


Figure 16.23. *A,* plain x-ray shows minimal displacement of the anterolateral corner of the distal tibial physis. *B,* CT scan more accurately demonstrates the degree of displacement. *C,* postoperative plain x-ray after percutaneous screw fixation.

evident at the time of injury. Tenderness over the level of the growth plate of a long bone and a normal x-ray imply a Salter I fracture until developments prove otherwise. Stress views may be diagnostic. Repeat x-rays after 2 weeks usually will show bone response characteristic of fracture healing if the Salter I fracture has actually occurred. Salter I fractures must be reduced and immobilized until clinical union is evident. In the upper extremity, union usually requires 3 to 4 weeks of immobilization, whereas in the lower extremity, clinical union is usually present by 6 weeks after the injury. In most cases, nondisplaced Salter I fractures do not interfere with growth (refer to Salter V fractures).

Salter II fractures are transverse fractures of the growth plate that extend obliquely into the bony metaphysis. This fracture and the Salter I fracture are by far the most common physeal fractures. The fracture separation of the distal radial epiphysis discussed in Chapter 3 in "Fractures of Both Bones of the Forearm" is an example. Since the fracture into the metaphysis is radiologically evident, a Salter II fracture is rarely missed at the time of injury. Treatment and prognosis are identical to those of the Salter I fracture. Special attention must be paid to injuries in the distal femoral physis. Growth inhibition after Salter I fractures has been reported. Salter II fractures of the distal physis are more likely to result in a complete or partial growth arrest than similar injuries at other growth plates. Therefore, children who have an injury to the distal femoral physis should be followed by clinical and

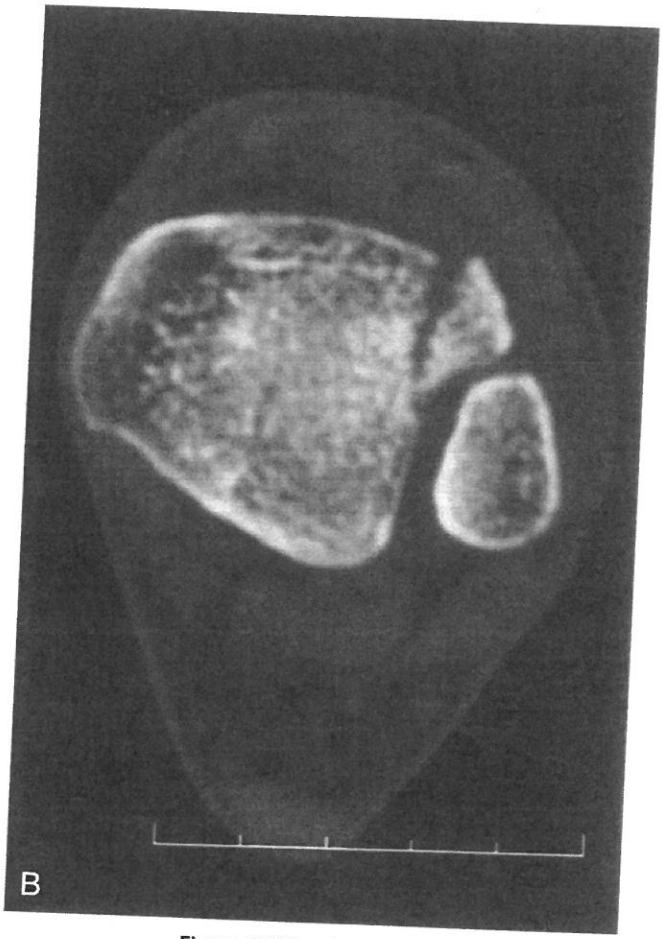


Figure 16.23. (*continued*)

radiographic examination for at least 2 years after their injury.

Salter III fractures are transverse fractures of the growth plate that have involved the bony epiphysis. The Salter III fracture occurs in adolescence and is commonly seen in the distal tibia (Fig. 16.23). When the physis is in the process of closing, the fracture pattern extends through the remaining open part of the physis, through the epiphysis, and into the nearby joint. Because this is an intra-articular fracture, reduction must be anatomic. This injury should be referred to an orthopaedist.

Salter IV fractures extend axially into the bony metaphysis and into the bony epiphysis. The fracture may appear to extend axially from the bony epiphysis to the bony metaphysis directly through the growth plate, or it may appear to extend axially through the bony epiphysis, transversely across part of the growth plate, then axially into the bony metaphysis. The danger of growth arrest is greatest in these fractures. Reduction must be anatomic, and close follow-up of leg length discrepancies and angular deformities is required. The injury must be referred to an orthopaedist.

Salter V fractures are compression injuries of the growth plate. The initial clinical characteristics are usually indistinguishable from those of the undisplaced Salter I fracture: no radiologic visibility, tenderness at the region of the growth plate, and eventual appearance of radiologic signs of bone healing (usually

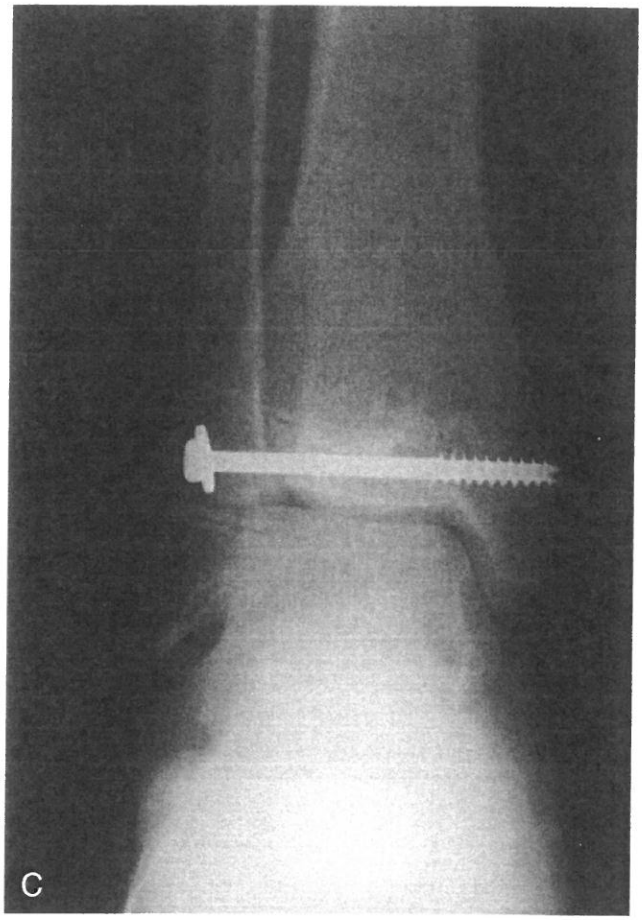


Figure 16.23. (*continued*)

within 2 to 3 weeks). The distinction occurs much later when it becomes evident that the injured physis is either no longer growing or is growing in an asymmetric fashion. Asymmetric closure of a physis can be recognized on a roentgenogram by a bridge of sclerotic bone spanning the normal lucency of the physis. The growth arrest line may be asymmetric as well, which would be indicative of a Salter injury. Even if the injury had been recognized on the first day, nothing could have been done to prevent the outcome. When the undisplaced Salter I/Salter V clinical presentation appears, parents must be warned of the poor prognosis of the Salter V fracture while the injury is treated as a Salter I fracture.

The prognosis of epiphyseal fractures differs not only among the types but among the locations as well. Epiphyseal fractures in the upper extremity are rarely followed by growth failure except after Salter V injuries. In the lower extremity, any epiphyseal fracture can be followed by growth failure, and the Salter III, IV, and V fractures carry a particularly guarded prognosis.

SUGGESTED READINGS

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