

## CHAPTER

# 42

# Alterations in the Skeletal System: Trauma, Infection, and Developmental Disorders

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The musculoskeletal system includes the bones, joints, and muscles of the body together with associated structures such as ligaments and tendons. This system, which constitutes more than 70% of the body, is subject to a large number of disorders. These disorders affect persons in all age groups and walks of life and cause pain, disability, and deformity. The discussion in this chapter focuses on alterations and the effects of trauma, infections, ischemia, and neoplasms on structures of the musculoskeletal system. Disorders of skeletal growth and development in children are discussed at the end of the chapter.

## INJURY AND TRAUMA OF MUSCULOSKELETAL STRUCTURES

A broad spectrum of musculoskeletal injuries results from numerous physical forces, including blunt tissue trauma, disruption of tendons and ligaments, and fractures of bony structures. Many of the forces that cause injury to the musculoskeletal

system are typical for a particular environmental setting, activity, or age group. Trauma resulting from high-speed motor accidents is ranked as the number one killer of adults younger than 45 years of age.<sup>1</sup> Motorcycle accidents are especially common in young men, with fractures of the distal tibia, midshaft femur, and radius occurring most often. Trauma in children is usually the result of an accident. The most common causes of childhood injuries are falls, bicycle-related injuries, and sports injuries. Falls are the most common cause of injury in people 65 years of age and older. Impaired vision and hearing, dizziness, and unsteadiness of gait contribute to falls in older persons. These falls are often compounded by osteoporosis, or bone atrophy, which makes fractures more likely. Fractures of the hip and proximal humerus are particularly common in this age group.

### Soft Tissue Injuries

Most skeletal injuries are accompanied by soft tissue injuries. These injuries include contusions, hematomas, and lacerations. They are discussed here because of their association with musculoskeletal injuries.

A *contusion* is an injury to soft tissue that results from direct trauma and is usually caused by striking a body part against a hard object. With a contusion, the skin overlying the injury remains intact. Initially, the area becomes ecchymotic (*i.e.*, black and blue) because of local hemorrhage; later, the discoloration gradually changes to brown and then to yellow as the blood is reabsorbed.

A large area of local hemorrhage is called a *hematoma*. Hematomas cause pain as blood accumulates and exerts pressure on nerve endings. The pain increases with movement or when pressure is applied to the area. The pain and swelling of a hematoma take longer to subside than that accompanying a contusion. A hematoma may become infected because of bacterial growth. Unlike a contusion, which does not drain, a hematoma may eventually split the skin because of increased pressures and produce drainage. The treatment for a contusion and a hematoma consists of elevating the affected part and applying cold for the first 24 hours to reduce the bleeding into the area. A hematoma may need to be aspirated.

A *laceration* is an injury in which the skin is torn or its continuity is disrupted. The seriousness of a laceration depends on the size and depth of the wound and on whether there is contamination from the object that caused the injury. Puncture wounds from nails or rusted material may result in the growth of toxic bacteria, leading to gas gangrene or tetanus. Lacerations are usually treated by wound closure, which is done after the area is sufficiently cleaned; the closed wound is covered with a sterile dressing. It is important to minimize contamination of the wound and to control bleeding. Contaminated wounds and open fractures are copiously irrigated and debrided, and the skin usually is left open to heal to prevent the development of an anaerobic infection or a sinus tract.

### Joint (Musculotendinous) Injuries

Joints, or articulations, are sites where two or more bones meet. Joints (*i.e.*, diarthrodial) are supported by tough bundles of collagenous fibers called *ligaments* that attach to the joint capsule and bind the articular ends of bones together, and by *ten-*

*ons* that join muscles to the periosteum of the articulating bones. Joint injuries involve mechanical overloading or forcible twisting or stretching.

### Strains and Sprains

A *strain* is a stretching injury to a muscle or a musculotendinous unit caused by mechanical overloading. This type of injury may result from an unusual muscle contraction or an excessive forcible stretch. Although there usually is no external evidence of a specific injury, pain, stiffness, and swelling exist. The most common sites for muscle strains are the lower back and the cervical region of the spine. The elbow and the shoulder are also supported by musculotendinous units that are subject to strains. Foot strain is associated with the weight-bearing stresses of the feet; it may be caused by inadequate muscular and ligamentous support, overweight, or excessive exercise such as standing, walking, or running.

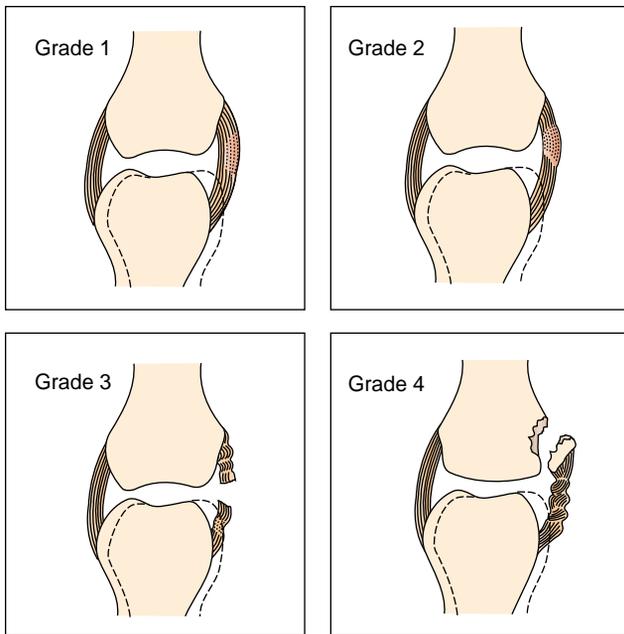
A *sprain*, which involves the ligamentous structures surrounding the joint, resembles a strain, but the pain and swelling subside more slowly. It usually is caused by abnormal or excessive movement of the joint. With a sprain, the ligaments may be incompletely torn or, as in a severe sprain, completely torn or ruptured (Fig. 42-1). The signs of sprain are pain, rapid swelling, heat, disability, discoloration, and limitation of function. Any joint may be sprained, but the ankle joint is most commonly involved, especially in higher-risk sports such as basketball. Most ankle sprains occur in the lateral ankle when the foot is turned inward under a person, forcing the ankle into inversion beyond the structural limits. Other common sites of sprain are the knee (the collateral ligament and anterior cruciate ligament) and elbow (the ulnar side). As with a strain, the soft tissue injury that occurs with a sprain is not evident on the radiograph. However, occasionally a chip of bone is evident when the entire ligament, including part of its bony attachment, has been ruptured or torn from the bone.

Healing of the dense connective tissues in tendons and ligaments is similar to that of other soft tissues. If properly treated,

#### KEY CONCEPTS

##### JOINT INJURIES

- Joints are the weakest part of the skeletal system and common sites for injury due to mechanical overloading or forcible twisting or stretching.
- Injury can include damage to the tendons, which connect muscle to bone; ligaments, which hold bones together; or the cartilage that covers the articular surface.
- Healing of the dense connective tissue involved in joint injuries requires time to restore the structures so that they are strong enough to withstand the forces imposed on the joint. Ligamentous injuries may require surgical intervention with approximation of many fibrous strands to facilitate healing.



■ **FIGURE 42-1** ■ Degrees of sprain on the medial side of the right knee: grade 1, mild sprain of the medial collateral ligament; grade 2, moderate sprain with hematoma formation; grade 3, severe sprain with total disruption of the ligament; and grade 4, severe sprain with avulsion of the medial femoral condyle at the insertion of the medial collateral ligament. (Adapted from Spickler L.L. [1983]. Knee injuries of the athlete. *Orthopedic Nursing* 2 [5], 12-13)

injuries usually heal with the restoration of the original tensile strength. Repair is accomplished by fibroblasts from the inner tendon sheath or the loose connective tissue that surrounds the tendon. Capillaries infiltrate the injured area during the initial healing process and supply the fibroblasts with the materials they need to produce large amounts of collagen. Formation of the new collagen fibrils begins within 4 to 5 days and although tensile strength increases steadily thereafter, it is not sufficient to permit strong tendon pulls for about 7 weeks.<sup>2</sup> During the first 3 weeks, there is a danger that muscle contraction will pull the injured ends apart, causing the tendon to heal in the lengthened position. There is also a danger that adhesions will develop in areas where tendons pass through fibrous channels, such as in the distal palm of the hands, rendering the tendon useless.

The treatment of muscle strains and ligamentous sprains is similar in several ways. For an injured extremity, such as the ankle, elevation of the part followed by local application of cold may be sufficient.<sup>3</sup> Compression, accomplished through the use of adhesive wraps or a removable splint, helps reduce swelling and provides support. A cast is applied for severe sprains, especially those severe enough to warrant surgical repair. Immobilization for a muscle strain is continued until the pain and swelling have subsided. In a sprain, the affected joint is immobilized for several weeks. Immobilization may be followed by graded active exercises. Early diagnosis, treatment, and rehabilitation are essential in preventing chronic ligamentous instability.

## Dislocations

Dislocation of a joint is the loss of articulation of the bone ends in the joint capsule caused by displacement or separation of the bone end from its position in the joint. It usually follows a severe trauma that disrupts the holding ligaments. Dislocations are seen most often in the shoulder and acromioclavicular joints. A *subluxation* is a partial dislocation in which the bone ends in the joint are still in partial contact with each other.

Dislocations can be congenital, traumatic, or pathologic. Congenital dislocations occur in the hip and knee. Traumatic dislocations occur after falls, blows, or rotational injuries. For example, car accidents often cause dislocations of the hip and accompanying acetabular fractures because of the direction of impact. In the shoulder and patella, dislocations may become recurrent, especially in athletes. They recur with the same motion but require less and less force each time. Less common sites of dislocation, seen mainly in young adults, are the wrist and midtarsal region. They usually are the result of direct force, such as a fall on an outstretched hand. Pathologic dislocation in the hip is a late complication of infection, rheumatoid arthritis, paralysis, and neuromuscular diseases.

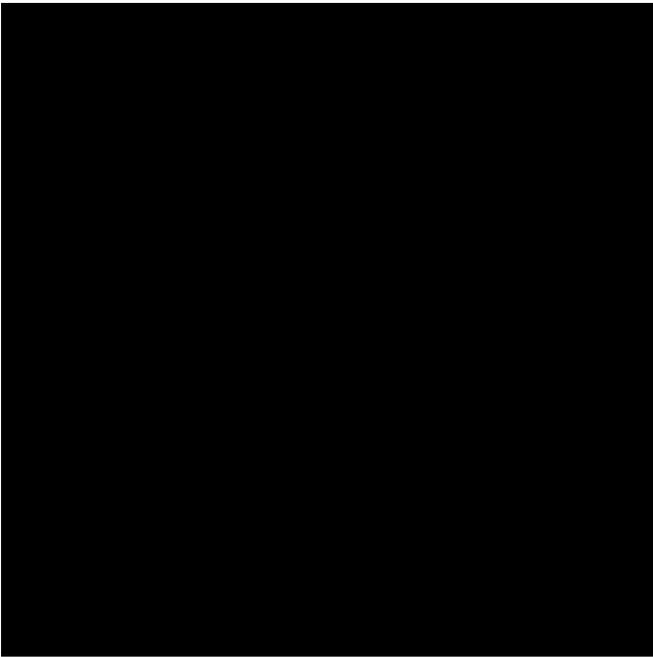
Diagnosis of a dislocation is based on history, physical examination, and radiologic findings. The symptoms are pain, deformity, and limited movement. The treatment depends on the site, mechanism of injury, and associated injuries such as fractures. Dislocations that do not reduce spontaneously usually require manipulation or surgical repair. Various surgical procedures also can be used to prevent redislocation of the patella, shoulder, or acromioclavicular joints. Immobilization is necessary for several weeks after reduction of a dislocation to allow healing of the joint structures. In dislocations affecting the knee, alternatives to surgery are isometric quadriceps-strengthening exercises and a temporary brace. Surgical procedures, such as joint replacement, may be necessary in certain pathologic dislocations.

## Loose Bodies

Loose bodies are small pieces of bone or cartilage within a joint space. These can result from trauma to the joint or may occur when cartilage has worn away from the articular surface, causing a necrotic piece of bone to separate and become free floating. The symptoms are painful catching and locking of the joint. Loose bodies are commonly seen in the knee, elbow, hip, and ankle. The loose body repeatedly gets caught in the crevice of a joint, pinching the underlying healthy cartilage; unless the loose body is removed, it may cause osteoarthritis and restricted movement. The treatment consists of removal using operative arthroscopy.

## Shoulder and Rotator Cuff Injuries

The glenohumeral (shoulder) joint is a ball-and-socket joint that permits a wide range of motion, a factor that makes the joint relatively unstable.<sup>4</sup> The support and movement of the shoulder joint relies heavily on the support of four relatively small muscle-tendon groups collectively known as the rotator cuff (Fig. 42-2). The space between the acromion of the shoulder blade and superior part of the humeral head is called the impingement interval. This space is normally narrow and is maximally narrow when the arm is abducted or moved above the horizontal.



Rotator cuff impingement, tendonitis, and tears are common among athletes. The rotator cuff is commonly injured during repetitive movements that carry the arm above the shoulder, such as those used by baseball pitchers, tennis and racquet ball players, swimmers, and weight lifters.<sup>4</sup> Among the relevant muscles, the supraspinous is the one most often affected. Rotator cuff tears may be partial or full thickness. Partial tears do not completely sever the tendon and respond well to nonsurgical treatment. Full thickness tears require surgery.

Recurrent inflammation or tendonitis of the rotator cuff inflammation, also known as *shoulder impingement syndrome*, is characterized by pain and swelling of the cuff tendons and the surrounding bursa. It occurs most often in people with loose joints, people with abnormal bony anatomy in the shoulder, and people who do repetitive lifting above the shoulder level. It is common in baseball pitchers. Lifting overhead or just moving the arm above the head may cause pinching or impingement of one of the tendons of the rotator cuff. Tendonitis may progress to a partial or complete rotator cuff tear caused by progressive weakening of the tendon fibers.

Calcific tendonitis is a self-limiting calcification of the rotator cuff.<sup>5</sup> It involves the deposition of calcium crystals in the tendon of the rotator cuff. It is most common in women, ages 30 to 60 years, and workers in sedentary jobs. The disorder involves four stages: (1) precalcific phase with asymptomatic fibrocartilaginous transformation within the cuff, (2) deposition of calcium crystals, (3) the resorptive phase, and (4) the postcalcific healing and repair phase. The third phase is usually the most incapacitating because the movement of the calcium crystals in the subacromial bursa during the resorptive process may cause constant and severe pain and restriction of motion that typically lasts for several weeks.

Many physical examination maneuvers are used to define shoulder pathology. The history and mechanism of injury are important. In addition to standard radiographs, an arthrogram,

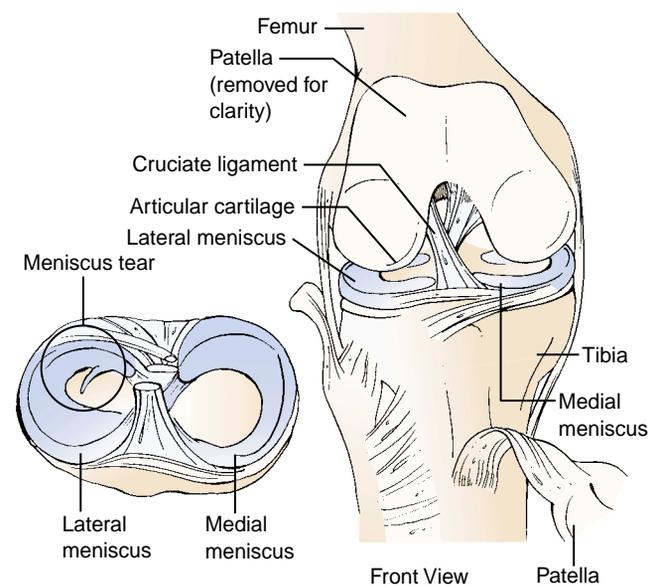
computed tomography (CT) scan, or magnetic resonance imaging (MRI) scan may be obtained. Arthroscopic examination under anesthesia is done for diagnostic purposes and operative arthroscopy to repair severe tears. Conservative treatment with anti-inflammatory agents, corticosteroid injections, and physical therapy often is done. A period of rest is followed by a customized exercise and rehabilitation program to improve strength, flexibility, and endurance.

### Knee Injuries

The knee is a common site of injury, particularly sport-related injuries in which the knee is subjected to abnormal twisting and compression forces. These forces can result in injury to the menisci, rupture of the anterior cruciate ligament, patellar subluxation and dislocation, and chondromalacia. Knee injuries in young adulthood and both knee and hip injuries in middle age substantially increase the risk of osteoarthritis in the same joint later in life.<sup>6</sup>

**Meniscus Injuries.** The menisci are C-shaped plates of fibrocartilage that are superimposed between the condyles of the femur and tibia. The menisci play a major role in load bearing and shock absorption; they help to stabilize the knee; they assist in joint lubrication; and they serve as a source of nutrition for articular cartilage in the knee. There are two menisci in each knee, a lateral and medial meniscus (Fig. 42-3). The menisci are thicker at their external margins and taper to thin, unattached edges at their interior margin. They are firmly attached at their ends to the intercondylar area of the tibia and are supported by the coronary and transverse ligaments of the knee.

Any action of the knee that causes injury to the knee ligaments can also cause a meniscal tear.<sup>7</sup> Meniscus injury commonly occurs as the result of a rotational injury from a sudden or sharp pivot or a direct blow to the knee, as in hockey, basketball, or football. The injured knee is edematous and painful,



■ **FIGURE 42-3** ■ The knee showing the lateral and medial meniscus (with the patella removed for clarity). Insert (**lower left**) shows meniscus tear.

especially with hyperflexion and hyperextension. A loose fragment may cause knee instability and locking.

Diagnosis is made by examination and confirmed by methods such as arthroscopy and radiologic, CT scans, MRI, and radionuclide imaging.<sup>8</sup> Initial treatment of meniscal injuries may be conservative. The knee may be placed in a removable knee immobilizer. Isometric quadriceps exercises may be prescribed. Activity usually is restricted until complete motion is recovered. Arthroscopic meniscectomy may be performed when there is recurrent or persistent locking, recurrent effusion, or disabling pain.

**Rupture of the Anterior Cruciate Ligament.** The cruciate ligaments (CL) secure the femur to the tibia in a crossed position (Fig. 42-3). The anterior CL (ACL) and posterior CL (PCL) control flexion and lateral rotation of the knee. The ACL is the weaker ligament and is often ruptured with a lateral blow to the knee. People with knees that are unstable as the result of ACL tears are at risk for early degenerative joint disease, as well as subsequent damage to other structures of the knee.<sup>9</sup>

About 85% of people who have an ACL tear are immediately disabled and not able to continue their activity. They often report the feeling of “giving way” and hearing or feeling a “pop” at the time of injury. Swelling is usually caused by hemarthrosis.

Diagnosis is based on the history, physical examination of the knee, and MRI. The selection of treatment methods, which include surgical repair, alterations in lifestyle, and bracing, depend on the person’s activity level and expectation. Surgical repair is usually the treatment of choice for athletes who want to continue activities requiring twisting or rapid changes in direction.

**Patellar Subluxation and Dislocations.** Recurrent subluxation and dislocation of the patella (*i.e.*, knee cap) are common injuries in young adults. They account for approximately 10% of all athletic injuries and are more common in women. Sports such as skiing or tennis may cause stress on the patella. These sports involve external rotation of the foot and lower leg with knee flexion, a position that exerts rotational stresses on the knee. Congenital knee variations are also a predisposing factor.

There is often a sensation of the patella “popping out” when the dislocation occurs. Other complaints include the knee giving out, swelling, crepitus, stiffness, and loss of range of motion.

Treatment can be difficult, but nonsurgical methods are used first. They include immobilization with the knee extended, bracing, administration of anti-inflammatory agents, and isometric quadriceps-strengthening exercises. Surgical intervention often is necessary.

**Chondromalacia.** Chondromalacia, or softening of the articular cartilage, is seen most commonly on the undersurface of the patella and occurs most frequently in young adults. It can be the result of recurrent subluxation of the patella or overuse in strenuous athletic activities. Persons with this disorder typically report pain, particularly when climbing stairs or sitting with the knees bent. Occasionally, the person experiences weakness of the knee.

The treatment consists of rest, isometric exercises, and application of ice after exercise. Part of the patella may be surgi-

cally removed in severe cases. In less severe cases, the soft portion is shaved using a saw inserted through an arthroscope.

## Fractures

A fracture, or discontinuity of the bone, is the most common type of bone lesion. Normal bone can withstand considerable compression and shearing forces and, to a lesser extent, tension forces. A fracture occurs when more stress is placed on the bone than it is able to absorb. Grouped according to cause, fractures can be divided into three major categories: (1) fractures caused by sudden injury, (2) fatigue or stress fractures, and (3) pathologic fractures. The most common fractures are those resulting from sudden injury. The force causing the fracture may be direct, such as a fall or blow, or indirect, such as a massive muscle contraction or trauma transmitted along the bone. For example, the head of the radius or clavicle can be fractured by the indirect forces that result from falling on an outstretched hand.

A *fatigue fracture* results from repeated wear on a bone. Pain associated with overuse injuries of the lower extremities, especially posterior medial tibial pain, is one of the most common symptoms that physically active persons, such as runners, experience. *Stress fractures* in the tibia may be confused with “shin splints,” a nonspecific term for pain in the lower leg from overuse in walking and running, because they frequently do not appear on x-ray films until 2 weeks after the onset of symptoms.

A *pathologic fracture* occurs in bones that already are weakened by disease or tumors. Fractures of this type may occur spontaneously with little or no stress. The underlying disease state can be local, as with infections, cysts, or tumors, or it can be generalized, as in osteoporosis, Paget’s disease, or disseminated tumors.

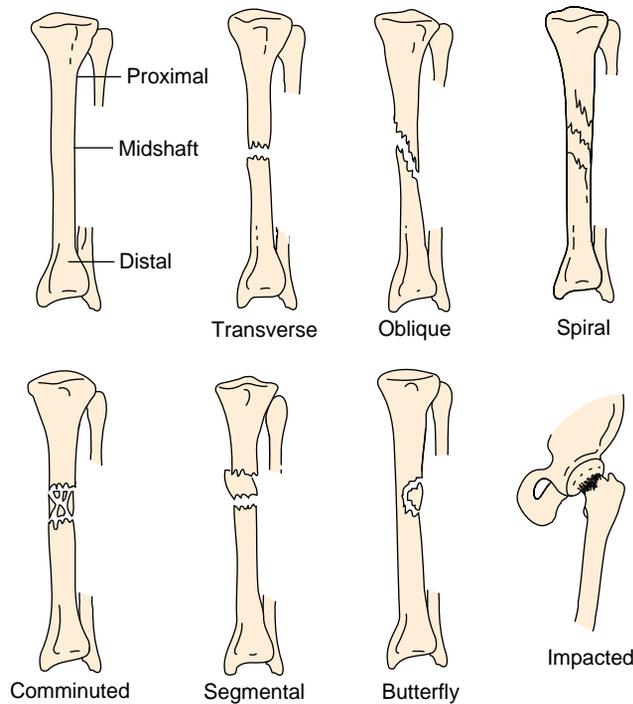
## Classification

Fractures usually are classified according to location, type, and direction or pattern of the fracture line (Fig. 42-4). A fracture of the long bone is described in relation to its position in the bone—proximal, midshaft, and distal. Other descriptions are used when the fracture affects the head or neck of a bone,

### KEY CONCEPTS

#### FRACTURE HEALING

- Fractures are caused by forces that disrupt the continuity of bone.
- Fracture healing depends on the extent of the injury, the ability to align the bone fragments, and immobilizing the fracture site so that healing can take place.
- Bone healing occurs by replacement of injured bone cells. It involves formation of a hematoma that provides the foundation for blood vessel and fibroblast infiltration, proliferation of bone repair cells (osteoblasts), callus formation, ossification of the callus, and remodeling of the fracture site.



■ **FIGURE 42-4** ■ Classification of fractures. Fractures are classified according to location (proximal, midshaft, or distal), the direction of fracture line (transverse, oblique, or spiral), and type (comminuted, segmental, butterfly, or impacted).

involves a joint, or is near a prominence such as a condyle or malleolus.

The type of fracture is determined by its communication with the external environment, the degree of break in continuity of the bone, and the character of the fracture pieces.<sup>10</sup> A fracture can be classified as open or closed. When the bone fragments have broken through the skin, the fracture is called an *open* or *compound fracture*. In a closed fracture, there is no communication with the outside skin.

The degree of a fracture is described in terms of a partial or complete break in the continuity of bone. A *greenstick fracture*, which is seen in children, is an example of a partial break in bone continuity and resembles that seen when a young sapling is broken. This kind of break occurs because children's bones, especially until approximately 10 years of age, are more resilient than the bones of adults.

The character of the fracture pieces may also be used to describe a fracture. A *comminuted fracture* has more than two pieces. A *compression fracture*, as occurs in the vertebral body, involves two bones that are crushed or squeezed together. A fracture is called *impacted* when the fracture fragments are wedged together. This type usually occurs in the humerus, often is less serious, and usually is treated without surgery.

The direction of the trauma or mechanism of injury produces a certain configuration or pattern of fracture. *Reduction* is the restoration of a fractured bone to its normal anatomic position. The pattern of a fracture indicates the nature of the trauma and provides information about the easiest method for reduction. *Transverse fractures* are caused by simple angular forces. A *spiral fracture* results from a twisting motion, or torque. A transverse fracture is not likely to become displaced

or lose its position after it is reduced. On the other hand, spiral, oblique, and comminuted fractures often are unstable and may change position after reduction.

### Manifestations

The signs and symptoms of a fracture include pain, tenderness at the site of bone disruption, swelling, loss of function, deformity of the affected part, and abnormal mobility. The deformity varies according to the type of force applied, the area of the bone involved, the type of fracture produced, and the strength and balance of the surrounding muscles.

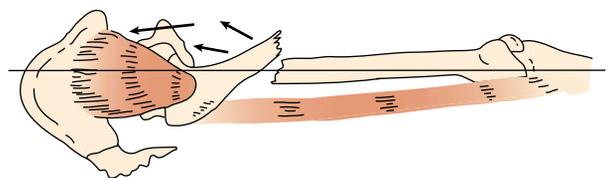
In long bones, three types of deformities—angulation, shortening, and rotation—are seen. Severely angulated fracture fragments may be felt at the fracture site and often push up against the soft tissue to cause a tenting effect on the skin. Bending forces and unequal muscle pulls cause angulation. Shortening of the extremity occurs as the bone fragments slide and override each other because of the pull of the muscles on the long axis of the extremity (Fig. 42-5). Rotational deformity occurs when the fracture fragments rotate out of their normal longitudinal axis; this can result from rotational strain produced by the fracture or unequal pull by the muscles that are attached to the fracture fragments. A crepitus or grating sound may be heard as the bone fragments rub against each other. In the case of an open fracture, there is bleeding from the wound where the bone protrudes. Blood loss from a pelvic fracture or multiple long bone fractures can cause hypovolemic shock in a trauma victim.

Shortly after the fracture has occurred, nerve function at the fracture site may be temporarily lost. The area may become numb, and the surrounding muscles may become flaccid. This condition has been called *local shock*. During this period, which may last for a few minutes to half an hour, fractured bones may be reduced with little or no pain. After this brief period, the pain sensation returns and, with it, muscle spasms and contractions of the surrounding muscles.

### Diagnosis and Treatment

Diagnosis is the first step in the care of fractures and is based on history and physical manifestations. X-ray examination is used to confirm the diagnosis and direct the treatment. The ease of diagnosis varies with the location and severity of the fracture. In the trauma patient, the presence of other, more serious injuries may make diagnosis more difficult.

Treatment depends on the general condition of the person, the presence of associated injuries, the location of the fracture and its displacement, and whether the fracture is open or closed. A *splint* is a device for immobilizing the movable fragments of a fracture. When a fracture is suspected, the injured part always should be splinted before it is moved. This is essential for preventing further injury.



■ **FIGURE 42-5** ■ Displacement and overriding of fracture fragments of a long bone (femur) caused by severe muscle spasm.

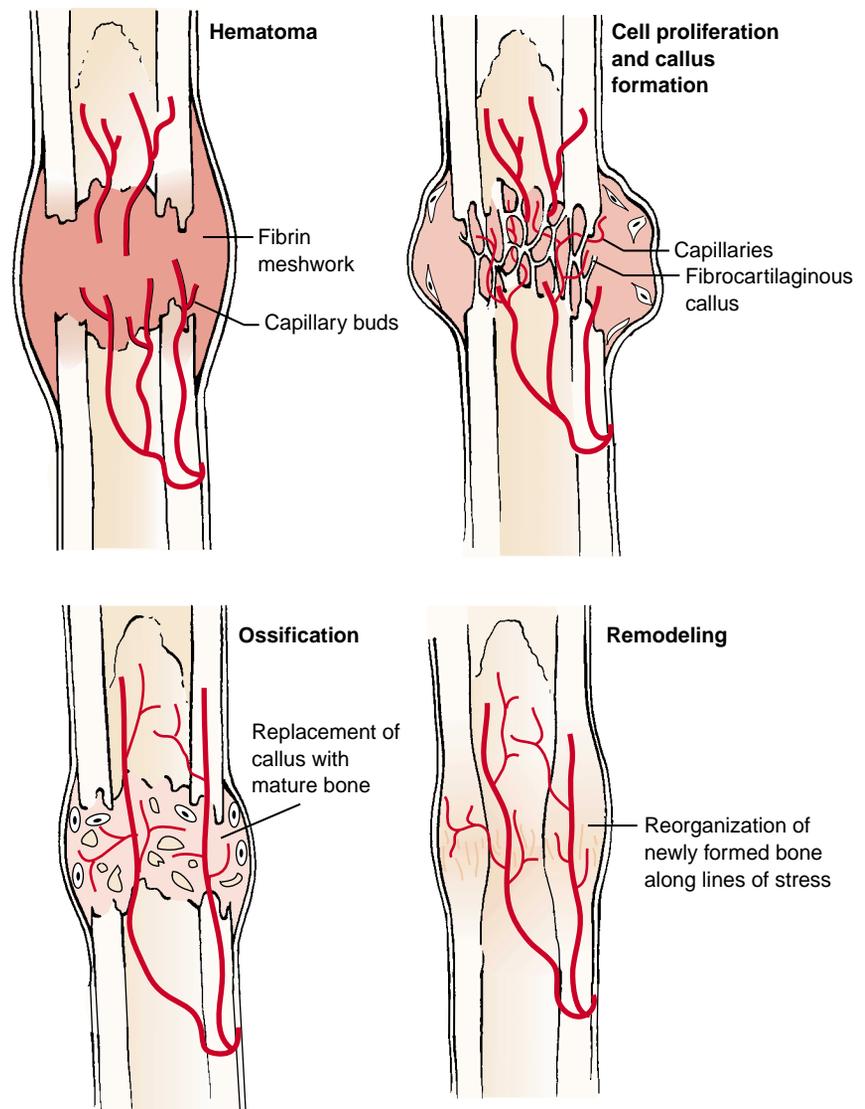
There are three objectives for treatment of fractures: (1) reduction of the fracture, (2) immobilization, and (3) preservation and restoration of the function of the injured part. Reduction of a fracture is directed toward replacing the bone fragments to as near normal anatomic position as possible. This can be accomplished by closed manipulation or surgical (open) reduction. Closed manipulation uses methods such as manual pressure and traction. Fractures are held in reduction by external or internal fixation devices. Surgical reduction involves the use of various types of hardware to accomplish internal fixation of the fracture fragments (Fig. 42-6). Immobilization prevents movement of the injured parts and is the single most important element in obtaining union of the fracture fragments. Immobilization can be accomplished through the use of external devices, such as splints, casts, external fixation devices, or traction, or by means of internal fixation devices inserted during surgical reduction of the fracture. Preservation and restoration of the function of muscles and joints are an ongoing process in the unaffected and affected extremities during the period of immobilization required for fracture healing. Exercises designed to preserve function, main-

tain muscle strength, and reduce joint stiffness should be started early.

### Bone Healing

Bone healing occurs in a manner similar to soft tissue healing. However, it is a more complex process and takes longer. There are five stages involved in bone healing: (1) hematoma formation, (2) cellular proliferation, (3) callus formation, (4) ossification, and (5) remodeling (Fig. 42-6). The degree of response during each of these stages is in direct proportion to the extent of trauma.

**Hematoma Formation.** Hematoma formation occurs during the first 48 to 72 hours after fracture. It develops as blood from torn vessels in the bone fragments and surrounding soft tissue leaks between and around the fragments of the fractured bone. The hematoma is thought to be necessary for the initiation of the cellular events essential to fracture healing.<sup>11-13</sup> As the result of hematoma formation, clotting factors remain in the injured area to initiate the formation of a fibrin meshwork, which serves as a framework for the ingrowth of fibroblasts and new



■ **FIGURE 42-6** ■ The stages of bone healing. The hematoma stage provides the fibrin meshwork and capillary buds needed for subsequent cellular invasion. Cellular proliferation and callus formation represent the stages during which osteoblasts enter the area and form the fibrocartilaginous callus that joins the bone fragments. The ossification stage involves the mineralization of the fibrocartilaginous callus; and the remodeling stage, the reorganization of mineralized bone along the lines of mechanical stress.

capillary buds. Granulation tissue, the result of fibroblast and new capillary growth, gradually invades and replaces the clot. When a large hematoma develops, healing is delayed because macrophages, platelets, oxygen, and nutrients for callus formation are prevented from entering the area.

**Cellular Proliferation.** Three layers of bone structure are involved in the cellular proliferation that occurs during bone healing: the periosteum, or outer covering of the bone; the endosteum, or inner covering; and the medullary canal, which contains the bone marrow. During this process, the osteoblasts, or bone-forming cells, multiply and differentiate into a fibrocartilaginous callus. The fibrocartilaginous callus is softer and more flexible than callus. Cellular proliferation begins distal to the fracture site, where there is a greater supply of blood. After a few days, a fibrocartilage “collar” becomes evident around the fracture site. The collar edges on either side of the fracture eventually unite to form a bridge, which connects the bone fragments.

**Callus Formation.** During the early stage of callus formation, the fracture site becomes “sticky” as osteoblasts continue to move in and through the fibrin bridge to help keep it firm. Cartilage forms at the level of the fracture, where there is less circulation. In areas of the bone with muscle insertion, periosteal circulation is better, bringing in the nutrients necessary to bridge the callus. The bone calcifies as mineral salts are deposited. This stage usually occurs during the third to fourth week of fracture healing.

**Ossification.** Ossification involves the final laying down of bone. This is the stage at which the fracture has been bridged and the fracture fragments are firmly united. Mature bone replaces the callus, and the excess callus is gradually resorbed by the osteoclasts. The fracture site feels firm and immovable and appears united on the radiograph. At this point, it is safe to remove the cast.

**Remodeling.** Remodeling involves resorption of the excess bony callus that develops in the marrow space and encircles the external aspect of the fracture site. The remodeling process is directed by mechanical stress and direction of weight bearing.

**Healing Time.** Union of a fracture has occurred when the fracture is solid enough to withstand normal stresses and it is clinically and radiologically safe to remove the external fixation. Healing time depends on the site of the fracture, the condition of the fracture fragments, hematoma formation, and other local and host factors. In children, fractures usually heal within 4 to 6 weeks; in adolescents, they heal within 6 to 8 weeks; and in adults, they heal within 10 to 18 weeks. The increased rate of healing among children compared with adults may be related to the increased cellularity and vascularity of the child’s periosteum.<sup>13</sup> In general, fractures of long bones, displaced fractures, and fractures with less surface area heal more slowly. Function usually returns within 6 months after union is complete. However, return to complete function may take longer.

**Impaired Bone Healing.** Factors that influence bone healing are specific to the person, the type of injury sustained, and local factors that disrupt healing (Chart 42-1). Individual factors that

#### CHART 42-1 Factors Affecting Fracture Healing

- Nature of the injury or the severity of the trauma, including fracture displacement, edema, and arterial occlusion with crushing injuries
- Degree of bridge formation that develops during bone healing
- Amount of bone loss (*e.g.*, it may be too great for the healing to bridge the gap)
- Type of bone that is injured (*e.g.*, cancellous bone heals faster than cortical bone)
- Degree of immobilization that is achieved (*e.g.*, movement disrupts the fibrin bridge and cartilage forms instead of bone)
- Local infection, which retards or prevents healing
- Local malignancy, which must be treated before healing can proceed
- Bone necrosis, which prevents blood flow into the fracture site

may delay bone healing are the patient’s age; current medications; debilitating diseases, such as diabetes and rheumatoid arthritis; local stress around the fracture site; circulatory problems and coagulation disorders; and poor nutrition.

**Malunion** is healing with deformity, angulation, or rotation that is visible on x-ray films.<sup>13</sup> Early, aggressive treatment, especially of the hand, can prevent malunion and result in earlier alignment and return of function. Malunion is caused by inadequate reduction or alignment of the fracture.

**Delayed union** is the failure of a fracture to unite within the normal period (*e.g.*, 20 weeks for a fracture of the tibia or femur in an adult). Intra-articular fractures (those through a joint) may heal more slowly and may eventually produce arthritis. **Nonunion** is failure to produce union and cessation of the processes of bone repair. It is seen most often in the tibia, especially with open fractures or crushing injuries. It is characterized by mobility of the fracture site and pain on weight bearing. Muscle atrophy and loss of range of motion may occur. Nonunion usually is established 6 to 12 months after the time of the fracture.<sup>13</sup> The complications of fracture healing are summarized in Table 42-1.

Treatment methods for impaired bone healing encompass surgical interventions, including bone grafts, bracing, external fixation, or electrical stimulation of the bone ends. Electrical stimulation is thought to stimulate the osteoblasts to lay down a network of bone. Three types of commercial bone growth stimulators are available: a noninvasive model, which is placed outside the cast; a seminoninvasive model, in which pins are inserted around the fracture site; and a totally implantable type, in which a cathode coil is wound around the bone at the fracture site and operated by a battery pack implanted under the skin.

#### Complications of Fractures and Other Musculoskeletal Injuries

The complications of fractures and other orthopedic injuries are associated with loss of skeletal continuity, injury from bone fragments, pressure from swelling and hemorrhage (*e.g.*, frac-

**TABLE 42-1** Complications of Fracture Healing

Complication	Manifestations	Contributing Factors
Delayed union	Failure of fracture to heal within predicted time as determined by x-ray	Large displaced fracture Inadequate immobilization Large hematoma Infection at fracture site Excessive loss of bone Inadequate circulation
Malunion	Deformity at fracture site Deformity or angulation on x-ray	Inadequate reduction Malalignment of fracture at time of immobilization
Nonunion	Failure of bone to heal before the process of bone repair stops Evidence on x-ray Motion at fracture site Pain on weight bearing	Inadequate reduction Mobility at fracture site Severe trauma Bone fragment separation Soft tissue between bone fragments Infection Extensive loss of bone Inadequate circulation Malignancy Bone necrosis Noncompliance with restrictions

ture blisters, compartment syndrome), involvement of nerve fibers (*e.g.*, reflex sympathetic dystrophy and causalgia), or development of fat emboli.

**Fracture Blisters.** Fracture blisters are skin bullae and blisters representing areas of epidermal necrosis with separation of epidermis from the underlying dermis by edema fluid. They are seen with more severe, twisting types of injuries (*e.g.*, motor vehicle accidents and falls from heights) but can also occur after excessive joint manipulation, dependent positioning, and heat application, or from peripheral vascular disease. They can be solitary, multiple, or massive, depending on the extent of injury. Most fracture blisters occur in the ankle, elbow, foot, knee, or areas where there is little soft tissue between the bone and the skin. The development of fracture blisters reportedly is reduced by early surgical intervention in persons requiring operative repair.<sup>14</sup> This probably reflects the early operative release of the fracture hematoma, reapproximation of the disrupted soft tissues, ligation of bleeding vessels, and fixation of bleeding fracture surfaces. Prevention of fracture blisters is important because they pose an additional risk of infection.

**Compartment Syndrome.** Compartment syndrome is the result of increased pressure in a limited anatomic space that compromises circulation and threatens the viability and function of the nerves and muscles (see Chapter 15). It can be acute or chronic. Acute compartment syndrome can occur after a fracture or crushing injury, when excessive swelling around the site of injury results in increased pressure in a closed compartment. This increase in pressure occurs because fascia, which covers and separates muscles, is inelastic and unable to stretch and compensate for the extreme swelling. The most common sites are the four compartments of the lower leg (*i.e.*, deep posterior, superficial posterior, lateral, and anterior compartments) and the dorsal and volar compartments of the forearm.

The condition is characterized by pain that is out of proportion to the original injury or physical findings.<sup>14,15</sup> Nerve compression may cause changes in sensation (*e.g.*, paresthesias such as burning or tingling or loss of sensation), diminished reflexes, and eventually the loss of motor function. Symptoms usually begin within a few hours but can be delayed as long as 64 hours.<sup>14</sup> Compression of blood vessels may cause muscle ischemia and loss of function. In contrast to the diminished or absent pulses that occur when ischemia is caused by a tight bandage or cast, the arterial pulses often are normal in compartment syndrome. Pallor and loss of the pulse, when they occur, are late findings.

Treatment of compartment syndrome is directed at reducing the compression of blood vessels and nerves. Constrictive dressings and casts are loosened. Intracompartmental pressure can be measured by means of a catheter or needle inserted into the compartment. A fasciotomy, or surgical transection of the fascia that is restricting the muscle compartment, may be required. Delay in diagnosis and treatment of compartment syndrome can lead to irreversible nerve and muscle damage.

**Reflex Sympathetic Dystrophy and Causalgia.** Reflex sympathetic dystrophy, also known as the complex regional pain syndrome, is a complication of orthopedic injuries that causes pain out of proportion to the injury and autonomic nervous system dysfunction manifested by hyperhidrosis (increased sweating) and vasomotor instability (either flushed and warm or cold and pale).<sup>14,16</sup> The disorder often produces long-term disability and chronic pain syndromes (see Chapter 39).

Pain, which is the prominent symptom of the disorder, is described as severe, aching, or burning. It usually increases in intensity with movement and with noxious and non-noxious stimuli. The cause of the pain is unclear but is thought to have a sympathetic nervous system component. Muscle wasting, thin and shiny skin, and abnormalities of the nails and bone

can occur. Decreased muscle strength and disuse can lead to contractures and osteoporosis.

Treatment focuses on pain management and prevention of disability. Physical therapy interventions such as hot/cold baths and elevation of the limb are used to maximize range of motion and minimize pain. Medications include anti-inflammatory agents, vasodilators, and antidepressant medications. Sympathetic nerve blocks may be used.

**Fat Embolism Syndrome.** The fat embolism syndrome (FES) refers to a constellation of clinical manifestations resulting from the presence of fat droplets in the small blood vessels of the lung or other organs after a long-bone fracture or other major trauma.<sup>14</sup> The fat emboli are thought to be released from the bone marrow or adipose tissue at the fracture site into the venous system through torn veins.

Clinically, the incidence of fat embolization is related to fractures of bones containing the most marrow (*i.e.*, long bones and the bones of the pelvis). An increase in intramedullary pressure in the femur is the most important pathogenic factor in the development of emboli. Although fat embolization occurs with fractures or operative fixation of fractures, FES occurs in only a small percentage of cases, supporting the hypothesis that factors other than mechanical forces may be necessary in the development of FES.

The main clinical features of FES are respiratory failure, cerebral dysfunction, and skin petechiae.<sup>14,17</sup> Initial symptoms begin within a few hours to 3 to 4 days after injury and do not appear beyond 1 week after the injury. The first symptoms include a subtle change in behavior and signs of disorientation resulting from emboli in the cerebral circulation combined with respiratory depression. There may be complaints of substernal chest pain and dyspnea accompanied by tachycardia and a low-grade fever. Diaphoresis, pallor, and cyanosis become evident as respiratory function deteriorates. A petechial rash that does not blanch with pressure often occurs 2 to 3 days after the injury. This rash usually is found on the anterior chest, axillae, neck, and shoulders. It also may appear on the soft palate and conjunctiva. The rash is thought to be related to embolization of the skin capillaries or thrombocytopenia.

Three degrees of severity are seen: subclinical, overt clinical, and fulminating. Although the subclinical and overt clinical forms of FES respond well to treatment, the fulminating form often is fatal. An important part of the treatment of fat emboli is early diagnosis. Arterial blood gases should be assayed immediately after recognition of clinical manifestations. Treatment is directed toward correcting hypoxemia and maintaining adequate fluid balance. Mechanical ventilation may be required. Corticosteroid drugs are administered to decrease the inflammatory response of lung tissues, decrease the edema, stabilize the lipid membranes to reduce lipolysis, and combat the bronchospasm. Corticosteroids are also given prophylactically to high-risk persons. The only preventive approach to FES is early stabilization of the fracture.

## Osteonecrosis

Osteonecrosis, or death of a segment of bone, is a condition caused by the interruption of blood supply to the marrow, medullary bone, or cortex. It is a relatively common disorder and can occur in the medullary cavity of the metaphysis and

the subchondral region of the epiphysis, especially in the proximal femur, distal femur, and proximal humerus. It is a common complicating disorder of Legg-Calvé-Perthes disease, sickle cell disease, steroid therapy, and hip surgery.<sup>18,19</sup> The rates of osteonecrosis among persons treated with corticosteroids range from 5% to 25%. More than 10% of 500,000 joint replacements performed annually in the United States are for treatment of osteonecrosis.<sup>18</sup>

Although bone necrosis results from ischemia, the mechanisms producing the ischemia are varied and include mechanical vascular interruption such as occurs with a fracture; thrombosis and embolism (*e.g.*, sickle cell disease, nitrogen bubbles caused by inadequate decompression during deep sea diving); vessel injury (*e.g.*, vasculitis, radiation therapy); and increased intraosseous pressure with vascular compression (*e.g.*, steroid-induced osteonecrosis). In many cases, the cause of the necrosis is uncertain. Other than fracture, the most common causes of bone necrosis are idiopathic (*i.e.*, those of unknown cause) and prior steroid therapy. Chart 42-2 lists disorders associated with osteonecrosis.

Bone has a rich blood supply that varies from site to site. The flow in the medullary portion of bone originates in nutrient vessels from an interconnecting plexus that supplies the marrow, trabecular bone, and endosteal half of the cortex. The outer cortex receives its blood supply from periosteal, muscular, metaphyseal, and epiphyseal vessels that surround the bone. Some bony sites, such as the head of the femur, have only limited collateral circulation so that interruption of the flow, such as with a hip fracture, can cause necrosis and irreversible damage to a substantial portion of medullary and cortical bone.

The pathologic features of bone necrosis are the same, regardless of cause. The site of the lesion is related to the vessels involved. There is necrosis of cancellous bone and marrow. The cortex usually is not involved because of collateral blood flow. In subchondral infarcts (*i.e.*, ischemia below the cartilage), a triangular or wedge-shaped segment of tissue that has the subchondral bone plate as its base and the center of the epiphysis as its apex undergo necrosis. When medullary infarcts occur in

**CHART 42-2 Causes of Osteonecrosis**

Mechanical disruption of blood vessels
Fractures
Legg-Calvé-Perthes disease
Blount's disease
Thrombosis and embolism
Sickle cell disease
Nitrogen bubbles in decompression sickness
Vessel injury
Vasculitis
Connective tissue disease
Systemic lupus erythematosus
Rheumatoid arthritis
Radiation therapy
Gaucher's disease
Increased intraosseous pressure
Steroid-induced osteonecrosis

fatty marrow, death of bone results in calcium release and necrosis of fat cells with the formation of free fatty acids. Released calcium forms an insoluble “soap” with free fatty acids. Because bone lacks mechanisms for resolving the infarct, the lesions remain for life.

One of the most common causes of osteonecrosis is that associated with administration of corticosteroids.<sup>18,19</sup> Despite numerous studies, the mechanism of steroid-induced osteonecrosis remains unclear. The condition may develop after the administration of very high, short-term doses, during long-term treatment, or even from intra-articular injection. Although the risk increases with the dose and duration of treatment, it is difficult to predict who will be affected. The interval between corticosteroid administration and onset of symptoms rarely is less than 6 months and may be more than 3 years. There is no satisfactory method for preventing progression of the disease.

The symptoms associated with osteonecrosis are varied and depend on the extent of infarction. Typically, subchondral infarcts cause chronic pain that is initially associated with activity but that gradually becomes more progressive until it is experienced at rest. Subchondral infarcts often collapse and predispose the patient to severe secondary osteoarthritis.

Diagnosis of osteonecrosis is based on history, physical findings, radiographic findings, and the results of special imaging studies, including CT scans and technetium-99m bone scans. MRI is particularly effective in the diagnosis of osteonecrosis. Plain radiographs are used to define and classify the course of the disease, particularly of the hip.

Treatment of osteonecrosis depends on the underlying pathology. In some cases, only short-term immobilization, non-steroidal anti-inflammatory drugs, exercises, and limitation in weight bearing are used. Osteonecrosis of the hip is particularly difficult to treat. In persons with early disease, limitation of weight bearing through the use of crutches may allow the condition to stabilize. Although several surgical approaches have been used, the most definitive treatment of advanced osteonecrosis of the knee or hip is total joint replacement.

**In summary,** many external physical agents can cause trauma to the musculoskeletal system. Particular factors, such as environments, activity, or age, can place a person at greater risk for injury. Some soft tissue injuries such as contusions, hematomas, and lacerations are relatively minor and easily treated. Muscle strains and ligamentous sprains are caused by mechanical overload on the connective tissue. They heal more slowly than the minor soft tissue injuries and require some degree of immobilization. Healing of soft tissue begins within 4 to 5 days of the injury and is primarily the function of fibroblasts, which produce collagen. Joint dislocation is caused by trauma to the supporting structures. Repeated trauma to the joint can cause articular softening (*i.e.*, chondromalacia) or the separation of small pieces of bone or cartilage, called *loose bodies*, in the joint. The knee and shoulder are common sites for injuries in athletes. The rotator cuff is a common site for shoulder injuries. Knee injuries include injury to the menisci, anterior cruciate ligament tears, patellar subluxation and dislocation, and chondromalacia.

Fractures occur when more stress is placed on a bone than the bone can absorb. The nature of the stress determines the type of fracture and the character of the resulting bone frag-

ments. Healing of fractures is a complex process that takes place in five stages: hematoma formation, cellular proliferation, callus formation, ossification, and remodeling. For satisfactory healing to take place, the affected bone has to be reduced and immobilized. This is accomplished with external fixation devices (*e.g.*, splints, casts, or traction) or surgically implanted internal fixation devices. The complications of fractures are associated with loss of skeletal continuity (malunion or nonunion), pressure from swelling and hemorrhage (fracture blisters and compartment syndrome), involvement of nerve fibers (reflex sympathetic dystrophy and causalgia), or development of fat emboli.

Osteonecrosis, or death of a segment of bone, is a condition caused by the interruption of blood supply to the marrow, medullary bone, or cortex. Sites with poor collateral circulation, such as the femoral head, are most seriously affected. It is a common complicating disorder of Legg-Calvé-Perthes disease, sickle cell disease, steroid therapy, and hip surgery. Symptoms include pain that varies in severity, depending on the extent of infarction. Total joint replacement is the most frequently used treatment for advanced osteonecrosis.

## BONE INFECTIONS

Bone infections are difficult to treat and eradicate. Their effects can be devastating; they can cause pain, disability, and deformity. Chronic bone infections may drain for years because of a sinus tract. This occurs when a passageway develops from an abscess or cavity in the bone to an opening through the skin.

### Osteomyelitis

Osteomyelitis represents an acute or chronic pyogenic infection of the bone. The term *osteo* refers to bone, and *myelo* refers to the marrow cavity, both of which are involved in this disease. Osteomyelitis can be caused by direct extension or contamination of an open fracture or wound (contiguous invasion); seeding through the bloodstream (hematogenous

## KEY CONCEPTS

### BONE INFECTIONS

- Bone infections may be caused by a wide variety of microorganisms introduced during injury, during operative procedures, or from the bloodstream.
- Once localized in bone, the microorganisms proliferate, produce cell death, and spread within the bone shaft, inciting a chronic inflammatory response with further destruction of bone.
- Bone infections are difficult to treat and eradicate. Measures to prevent infection include careful cleaning and debridement of skeletal injuries and strict operating room protocols.

spread); or from skin infections in persons with vascular insufficiency. In most cases, *Staphylococcus aureus* is the infecting organism.<sup>20,21</sup> *S. aureus* has two characteristics that favor its ability to produce osteomyelitis: (1) it is able to produce a collagen-binding adhesion molecule that allows it to adhere to the connective tissue elements of bone; and (2) it has the ability to be internalized and survive in osteoblasts, making the microorganism more resistant to antibiotic therapy.<sup>20</sup> The term *acute osteomyelitis* is used to describe a newly recognized bone infection. *Chronic osteomyelitis* refers to recurrence of a previously treated or untreated infection.

The pathogenesis of osteomyelitis includes the presence of the infecting agent, inflammation and the protective efforts of inflammatory cells, and bone destruction. The infective microorganisms present in osteomyelitis incite an inflammatory process with recruitment of phagocytic cells. In an attempt to contain the invading microorganisms, the phagocytes generate toxic oxygen radicals and release proteolytic enzymes that destroy surrounding tissues.<sup>20</sup> The purulent drainage that ensues spreads into blood vessels in the bone, increasing the intraosseous pressure and impairing blood flow. The loss of blood flow leads to ischemic necrosis of bone. The blood supply to the bone may become obstructed by septic thrombi, in which case the ischemic bone becomes necrotic. As the process continues, the necrotic bone separates from the viable surrounding bone to form devascularized fragments, called *sequestra* (Fig. 42-7). One of the characteristics of chronic osteo-

myelitis is the presence of necrotic bone and the absence of living osteocytes.

### Direct Contamination

The most common cause of osteomyelitis is the direct contamination of bone from an open wound. It may be the result of an open fracture, a gunshot wound, or a puncture wound. Inadequate irrigation or debridement, introduction of foreign material into the wound, and extensive tissue injury increase the bone's susceptibility to infection. If the infection is not sufficiently treated, the acute infection may become chronic. Osteomyelitis may also occur as a complication of surgery, such as in the sternum after open heart surgery or in extremities after bone allograft or total joint replacement.

Osteomyelitis after trauma or bone surgery usually is associated with persistent or recurrent fevers, increased pain at the operative or trauma site, and poor incisional healing, which often is accompanied by continued wound drainage and wound separation. Prosthetic joint infections present with joint pain, fever, and cutaneous drainage.

Diagnosis requires both confirming the infection and identifying the offending microorganism. The diagnosis of skeletal infection entails use of various imaging strategies, including conventional radiology, nuclear imaging studies, CT scans, and MRI. Bone biopsy may be used to identify the causative microorganisms. Treatment includes the use of antibiotics and selective use of surgical interventions.



■ **FIGURE 42-7** ■ Hematogenous osteomyelitis of the fibula of 3 months' duration. The entire shaft has been deprived of its blood supply and has become a sequestrum (S) surrounded by new immature bone, involucrum (lv). Pathologic fractures are present in the lower tibia and fibula. (Wilson F.C. [1980]. *The musculoskeletal system*. [2nd ed., p. 150]. Philadelphia: J.B. Lippincott)

### Hematogenous Osteomyelitis

Hematogenous osteomyelitis occurs as the result of localization of a blood-borne infection in the bone. It is seen most commonly in children younger than 10 years of age<sup>21</sup> but is seen occasionally in the elderly.<sup>20</sup>

In children, it commonly begins in the metaphyseal region of long bones and usually is preceded by staphylococcal or streptococcal infections of the skin, sinuses, teeth, or middle ear. Thrombosis occurring as the result of local trauma may predispose to localization of the infection consequent to bacteremia.<sup>21</sup>

In the adult, hematogenous osteomyelitis usually affects the axial skeleton and the irregular bones in the wrist and ankle. It is most common in debilitated patients and in those with a history of chronic skin infections, chronic urinary tract infections, intravenous drug use, and in those who are immunologically suppressed. Intravenous drug users are at risk for infections with *Streptococcus* and *Pseudomonas*.

The signs and symptoms of acute hematogenous osteomyelitis are those of bacteremia accompanied by symptoms referable to the site of the bone lesion. Bacteremia is characterized by chills, fever, and malaise. There often is pain on movement of the affected extremity, loss of movement, and local tenderness followed by redness and swelling. X-ray studies may appear normal initially, but they show evidence of periosteal elevation and increased osteoclastic activity after an abscess has formed.

The treatment of hematogenous osteomyelitis begins with identification of the causative organism through blood cultures, aspiration cultures, and Gram's stain.<sup>22</sup> Antibiotics are given first intravenously and then orally. Debridement and surgical drainage also may be necessary.

### Chronic Osteomyelitis

Chronic osteomyelitis has long been recognized as a disease. However, the incidence has decreased in the past century because of improvements in surgical techniques and antibiotic therapy. Chronic osteomyelitis includes all inflammatory processes of bone, excluding those in rheumatic diseases, that are caused by microorganisms. It may be the result of delayed or inadequate treatment of acute hematogenous osteomyelitis or osteomyelitis caused by direct contamination of bone. Acute osteomyelitis is considered to have become chronic when the infection persists beyond 6 to 8 weeks or when the acute process has been adequately treated and is expected to resolve but does not. Chronic osteomyelitis can persist for years; it may appear spontaneously, after a minor trauma, or when resistance is lowered.

The hallmark feature of chronic osteomyelitis is the presence of infected dead bone, a sequestrum, that has separated from the living bone. A sheath of new bone, called the involucrum, forms around the dead bone. Radiologic techniques such as x-ray films, bone scans, and sinograms are used to identify the infected site. Chronic osteomyelitis or infection around a total joint prosthesis can be difficult to diagnose because the classic signs of infection are not apparent and the blood leukocyte count may not be elevated. A subclinical infection may exist for years. Bone scans are used in conjunction with bone biopsy for a definitive diagnosis.<sup>23</sup>

Treatment includes the use of antibiotics. Wound cultures are used to identify the microorganism and its sensitivity to

antibiotic therapy. Initial antibiotic therapy is followed by surgery to remove foreign bodies (e.g., metal plates, screws) or sequestra and by long-term antibiotic therapy. Immobilization of the affected part usually is necessary, with restriction of weight bearing on a lower extremity.

### Osteomyelitis With Vascular Insufficiency

In persons with vascular insufficiency, osteomyelitis may develop from a skin lesion. It is seen most commonly associated with chronic or ischemic foot ulcers in persons with long-standing diabetes or other chronic vascular disorders. It is characterized by local cellulitis with inflammation and necrosis. Treatment depends on the oxygen tension of the involved tissues. Debridement and antibiotic therapy may benefit persons who have good oxygen tension in the infected site. Amputation is indicated when oxygen tension is inadequate.

### Tuberculosis of the Bone or Joint

Tuberculosis can spread from one part of the body, such as the lungs or the lymph nodes, to the bones and joints. When this happens, it is called *extrapulmonary* or *miliary tuberculosis*. It is caused by *Mycobacterium tuberculosis*. The disease is localized and progressively destructive but not as contagious as primary pulmonary tuberculosis. In approximately 50% of cases, it affects the vertebrae, but it also frequently is seen in the hip and knee.<sup>24</sup> Tuberculosis also can affect the joints and soft tissues. The disease is characterized by bone destruction and abscess formation. Local symptoms include pain, immobility, and muscle atrophy; joint swelling, mild fever, and leukocytosis also may occur. Diagnosis is confirmed by a positive culture. The most important part of the treatment is antituberculosis drug therapy. Conservative treatment is usually as effective as surgery, especially for earlier and milder cases.

Because of improved methods to prevent and treat tuberculosis, its incidence had diminished in recent decades. However, tuberculosis has re-emerged as a health problem, affecting one third of the global population and 10 million people in the United States.<sup>25</sup> Tuberculosis has increased because of the spread of disease in communal settings (e.g., jails, shelters, nursing homes), the human immunodeficiency virus epidemic, and the influx of immigrants who have come to the United States from countries where the disease is endemic.<sup>26</sup> Unfortunately, the diagnosis of tuberculosis in the bones and joints still may be missed, especially when the musculoskeletal infection is the sole presenting sign. CT scans and MRI can be used as aids for early diagnosis.

**In summary,** bone infections occur because of the direct or indirect invasion of the skeletal circulation by microorganisms, most commonly *S. aureus*. Osteomyelitis, or infection of the bone and marrow, can be an acute or chronic disease. Acute osteomyelitis is seen most often as a result of the direct contamination of bone by a foreign object. Chronic osteomyelitis is a long-term process that can recur spontaneously at any time throughout a person's life. Tuberculosis of the bone, which is characterized by bone destruction and abscess formation, is caused by spread of the infection from the lungs or lymph nodes.

## NEOPLASMS

Neoplasms in the skeletal system usually are referred to as *bone tumors*. Like other types of neoplasms, bone tumors may be benign or malignant (see Table 42-2). Primary bone tumors may arise from any of the skeletal components, including osseous bone tissue, cartilage, and bone marrow. Primary malignant tumors of the bone are uncommon, constituting approximately 1% of all adult cancers and 15% of pediatric malignancies.<sup>27</sup> However, metastatic disease of the bone is relatively common.

There are three major symptoms of bone tumors: pain, presence of a mass, and impairment of function (Chart 42-3). Pain is a feature common to almost all malignant tumors but may or may not occur with benign tumors. For example, a benign bone cyst usually is asymptomatic until a fracture occurs. Pain that persists at night and is not relieved by rest suggests malignancy. A mass or hard lump may be the first sign of a bone tumor. A malignant tumor is suspected when a painful mass exists that is enlarging or eroding the cortex of the bone. The ease of discovery of a mass depends on the location of the tumor; a small lump arising on the surface of the tibia is easy to detect, whereas a tumor that is deep in the medial portion of the thigh may grow to a considerable size before it is noticed. Benign and malignant tumors may cause the bone to erode to the point where it cannot withstand the strain of ordinary use. In such cases, even a small amount of bone stress or trauma precipitates a pathologic fracture. A tumor may produce pressure on a peripheral nerve, causing decreased sensation, numbness, a limp, or limitation of movement.

### Benign Neoplasms

Benign bone tumors usually are limited to the confines of the bone, have well-demarcated edges, and are surrounded by a thin rim of sclerotic bone. The four most common types of be-

### CHART 42-3 Symptoms of Bone Cancer

- Bone pain in an adult or child that comes on slowly but lasts for as long as a week, is constant or intermittent, and may be worse at night.
- Unexplained swelling or lump on the bones of the arms, legs, thighs, or other parts of the body that is firm and slightly tender and may be felt through the skin. It may interfere with normal movement and can cause the bone to break.

These symptoms are not sure signs of cancer. They also may be caused by other, less serious problems. Only a physician can tell for sure.

(Adapted from U.S. Department of Health and Human Services [1993]. *What you need to know about cancers of the bone*. NIH publication no. 93-1517. Bethesda, MD: U.S. Government Printing Office)

nign bone tumors are osteoma, chondroma, osteochondroma, and giant cell tumor.

An *osteoma* is a small bony tumor found on the surface of a long bone, flat bone, or the skull. It usually is composed of hard, compact (ivory osteoma), or spongy (cancellous) bone. It may be excised or left alone.

A *chondroma* is a tumor composed of hyaline cartilage.<sup>10</sup> It may arise on the surface of the bone (*i.e.*, *ecchondroma*) or within the medullary cavity (*i.e.*, *enchondroma*). These tumors may become large and are especially common in the hands and feet. A chondroma may persist for many years and then take on the attributes of a malignant chondrosarcoma. A chondroma usually is not treated unless it becomes unsightly or uncomfortable.

An *osteochondroma* is the most common form of benign tumor in the skeletal system, representing 50% of all benign bone tumors and approximately 15% of all primary skeletal lesions.<sup>27</sup> It grows only during periods of skeletal growth, originating in the epiphyseal cartilage plate and growing out of the bone like a mushroom. An osteochondroma is composed of cartilage and bone and usually occurs singly but may affect several bones in a condition called *multiple exostoses*. Malignant changes are rare, and excision of the tumor is done only when necessary.

A *giant cell tumor*, or *osteoclastoma*, is an aggressive tumor of multinucleated cells that often behaves like a malignant tumor, metastasizing through the bloodstream and recurring locally after excision. It occurs most often in young adults, predominantly females, and is found most commonly in the knee, wrist, or shoulder. The tumor begins in the metaphyseal region, grows into the epiphysis, and may extend into the joint surface. Pathologic fractures are common because the tumor destroys the bone substance. Clinically, pain may occur at the tumor site, with gradually increasing swelling. X-ray films show destruction of the bone with expansion of the cortex.

The treatment of giant cell tumors depends on their location. If the affected bone can be eliminated without loss of function, such as the clavicle or fibula, the entire bone or part of it may be removed. When the tumor is near a major joint, such as the knee or shoulder, a local excision is done. Irradiation may be used to prevent recurrence of the tumor.

**TABLE 42-2** Classification of Primary Bone Neoplasms

Tissue Type	Benign Neoplasm	Malignant Neoplasm
Bone	Osteoid osteoma Benign osteoblastoma	Osteosarcoma Parosteal osteogenic sarcoma
Cartilage	Osteochondroma Chondroma Chondroblastoma Chondromyxoid fibroma	Chondrosarcoma
Lipid	Lipoma	Liposarcoma
Fibrous and fibroosseous tissue	Fibrous dysplasia	Fibrosarcoma Malignant fibrous histiocytoma
Miscellaneous	Giant cell tumor	Malignant giant cell
Bone marrow		Ewing's sarcoma Multiple myeloma Reticulum cell sarcoma

## Malignant Bone Tumors

In contrast to benign tumors, malignant tumors tend to be ill defined, lack sharp borders, and extend beyond the confines of the bone, showing that it has destroyed the cortex. Specific types of bone tumors affect different age groups. They are virtually unknown in infancy, rare in children younger than 10 years of age, and peak during the teenage years. Adolescents have the highest incidence, with a rate of 3 cases per 100,000.<sup>27</sup> The two major forms of bone cancer in children and young adults are osteosarcoma and Ewing's sarcoma. It is unusual for either condition to be seen after 20 years of age.<sup>12</sup> Chondrosarcoma is most common in those 40 years of age and older.<sup>10</sup>

The methods used in the diagnosis of malignant bone tumors include radiographic studies, CT scans, MRI, and bone biopsy. Radiographs give the most general diagnostic information, such as malignant versus benign and primary versus metastatic status. The radiograph demonstrates the region of bone involvement, extent of destruction, and amount of reactive bone formed. Radioisotope scans are used to estimate the local intramedullary extent of the tumor and screen for other skeletal areas of involvement. CT scans further aid diagnosis and anatomic localization and can identify small pulmonary metastases not seen by conventional radiographs. MRI is the most accurate method of evaluating the intramedullary extent of bone tumor and can demarcate the soft structures in relation to neurovascular structures without the use of contrast media. It is best used in conjunction with a CT scan.<sup>27</sup> A bone biopsy is used to determine the type of tumor that is present.

### Osteosarcoma

Osteosarcoma represents 60% of all bone tumors occurring in children and adolescents. Osteosarcoma has a bimodal distribution, with 75% occurring in persons younger than 20 years of age. A second peak occurs in the elderly with predisposing factors such as Paget's disease, bone infarcts, or prior irradiation.<sup>10</sup> The male-to-female ratio increases to approximately 1.6 to 1 during late adolescence and adulthood. It is seen most commonly during periods of maximal growth. In younger persons, the primary tumor most often is located at the anatomic sites associated with maximum growth velocity—the distal femur, proximal tibia, and proximal humerus. Persons affected with osteosarcoma usually are tall and are found to have a high plasma level of somatomedin. Bone tumors in the elderly are more common in the humerus, pelvis, and proximal femur.

Osteosarcoma is a malignant tumor of mesenchymal cells, characterized by the direct formation of osteoid or immature bone by malignant osteoblasts. These cells synthesize thin, wispy, and purposeless fragments of bone. Osteogenic sarcomas are aggressive tumors that grow rapidly; they often are eccentrically placed in the bone and move from the metaphysis of the bone out to the periosteum, with subsequent spread to adjacent soft tissues.

The causes of osteosarcoma are unknown. The correlation of age and location of most of the tumors with the period of maximum growth suggests some relation to increased osteoblastic activity. Paget's disease, which is linked to osteosarcoma in adults, also is associated with increased osteoblastic activity. Irradiation from an internal source, such as the radio-

active pharmaceutical technetium used in bone scans, or an external source, such as x-ray films, also has been associated with osteosarcoma.

The primary clinical feature of osteosarcoma is localized pain and swelling in the affected bone, usually of sudden onset. Patients and their families often associate the symptoms with recent trauma.<sup>28</sup> The skin overlying the tumor may be warm, shiny, and stretched, with prominent superficial veins. The range of motion of the adjacent joint may be restricted. Osteosarcoma usually begins as a firm white or reddish mass and later becomes softer with a viscous interior (Fig. 42-8). The tumor infrequently metastasizes to the lymph nodes because the cells are unable to grow in the node. Nodal metastases usually occur only in the late course of disseminated disease. Most often, the tumor cells exit the primary tumor through the venous end of the capillary, and early metastasis to the lung is common. Lung metastases, even if massive, usually are relatively asymptomatic. The prognosis for a patient with osteosarcoma depends on the aggressiveness of the disease, radiologic features, presence or absence of pathologic fracture, size of the tumor, rapidity of tumor growth, and gender of the person.

The treatment for sarcomas is surgery in combination with multiagent chemotherapy used both before and after surgery.<sup>28,29</sup> Osteosarcomas are relatively resistant to radiation therapy. In the past, treatment usually entailed amputation above the level of the tumor. Limb salvage surgical procedures, using a metal prosthesis or cadaver allografts, are becoming a standard alternative. Studies have shown that limb salvage surgery has no adverse effects on the long-term survival



■ **FIGURE 42-8** ■ Juxtacortical osteosarcoma. The lower femur contains a malignant tumor arising from the periosteal surface of the bone and sparing the medullary cavity. (Rubin E., Farber J.L. [1999]. *Pathology* [3rd ed., p. 1386]. Philadelphia: Lippincott Williams & Wilkins)

of persons with osteosarcoma. Chemotherapy using various drug combinations is the most effective treatment for metastatic osteosarcoma.

### Ewing's Sarcoma

Ewing's sarcoma is the third most common type of primary bone tumor, and it is highly malignant. It commonly occurs in males younger than 25 years of age, with the incidence highest among teenagers.<sup>28</sup> Ewing's tumor arises from immature bone marrow cells and causes bone destruction from within. It usually occurs in the shaft of long bones or any portion of the pelvis and often metastasizes to bone marrow.

Manifestations of Ewing's tumor include pain, limitation of movement, and tenderness over the involved bone or soft tissue. It often is accompanied by systemic manifestations such as fever or weight loss, which may serve to confuse the diagnosis. There also may be a delay in diagnosis when the pain and swelling associated with the tumor are attributed to a sports injury. Pathologic fractures are common because of bone destruction.

Treatment methods incorporate a combination of multi-agent chemotherapy, surgery, and radiation therapy. There is controversy about the potential superiority of surgical resection compared with radiation therapy.<sup>28</sup> Radiation therapy is associated with risk of radiation-induced second malignancies, especially osteosarcoma.

### Chondrosarcoma

Chondrosarcoma, a malignant tumor of cartilage that can develop in the medullary cavity or peripherally, is the second most common form of malignant bone tumor. It occurs primarily in middle or later life and slightly more often in males. The tumor arises from points of muscle attachment to bone, particularly the knee, shoulder, hip, and pelvis. Chondrosarcomas can arise from underlying benign lesions such as osteochondroma, chondroblastoma, or fibrous dysplasia.<sup>10</sup>

Chondrosarcomas are slow growing, metastasize late, and often are painless. They can remain hidden in an area such as the pelvis for a long time. This type of tumor, like many primary malignancies, tends to destroy bone and extend into the soft tissues beyond the confines of the bone of origin. Chondrosarcomas mainly affect the bones of the trunk, pelvis, or proximal femur and rarely develop in the distal portion of a bone. Irregular flecks and ringlets of calcification often are prominent radiographic findings. Early diagnosis is important because chondrosarcoma responds well to early radical surgical excision. It usually is resistant to radiation therapy and available chemotherapeutic agents. Not infrequently, these tumors transform into a highly malignant tumor, mesenchymal chondrosarcoma, which requires a more aggressive treatment, including combination chemotherapy.

### Metastatic Bone Disease

Skeletal metastases are the most common malignancy of osseous tissue. Approximately half of all people with cancer have bone metastasis at some point in their disease.<sup>30</sup> Metastatic lesions are seen most often in the spine, femur, pelvis, ribs, sternum, proximal humerus, and skull, and are less common in anatomic sites that are further removed from the trunk of the body. Tumors that frequently spread to the skeletal sys-

tem are those of the breast, lung, prostate, kidney, and thyroid, although any cancer can ultimately involve the skeleton. More than 85% of bone metastases result from primary lesions in the breast, lung, or prostate.<sup>31</sup> The incidence of metastatic bone disease is highest in persons older than 40 years of age.

The major symptom of bone metastasis is pain with evidence of an impending pathologic fracture. It usually develops gradually, over weeks, and is more severe at night. Pain is caused by stretching of the periosteum of the involved bone or by nerve entrapment, as in the nerve roots of the spinal cord by the vertebral body. Pathologic fractures occur in approximately 10% to 15% of persons with metastatic bone disease. The affected bone appears to be eaten away on x-ray images and, in severe cases, crumbles on impact, much like dried toast. Many pathologic fractures occur in the femur, humerus, and vertebrae. In the femur, fractures occur because the proximal aspect of the bone is under great mechanical stress.<sup>31</sup>

X-ray examinations are used along with CT or bone scans to detect, diagnose, and localize metastatic bone lesions. Approximately one third of persons with skeletal metastases have positive bone scans without radiologic findings. This is because 50% of the trabecular bone must be destroyed before a lesion is visible on plain radiographs.<sup>32</sup> Arteriography using radiopaque contrast media may be helpful in outlining the tumor margins. A bone biopsy usually is done when there is a question regarding the diagnosis or treatment. A closed-needle biopsy with CT localization is particularly useful with spine lesions. Serum levels of alkaline phosphatase and calcium often are elevated in persons with metastatic bone disease.

The primary goals in treatment of metastatic bone disease are to prevent pathologic fractures and promote survival with maximum functioning, allowing the person to maintain as much mobility and pain control as possible. Treatment methods include chemotherapy, irradiation, and surgical stabilization. Radiation therapy is primarily used as a palliative treatment to alleviate pain and prevent pathologic fractures. After a pathologic fracture has occurred, bracing, intramedullary nailing of the femur, and spine stabilization may be done. Because adequate fixation often is difficult in diseased bone, cement (*i.e.*, methylmethacrylate) often is used with internal fixation devices to stabilize the bone.

**In summary,** bone tumors, like any other type of neoplasm, may be benign or malignant. Benign bone tumors grow slowly and usually do not destroy the surrounding tissues. Malignant tumors can be primary or metastatic. Primary bone tumors are rare, grow rapidly, metastasize to the lungs and other parts of the body through the bloodstream, and are associated with a high mortality rate. Metastatic bone tumors usually are multiple, originating primarily from cancers of the breast, lung, and prostate. The incidence of metastatic bone disease is increasing probably because improved treatment methods enable persons with cancer to live longer. Advances in chemotherapy, radiation therapy, and surgical procedures have substantially increased the survival and cure rates for many types of bone cancers. A primary goal in metastatic bone disease is the prevention of pathologic fractures.



## DISORDERS OF SKELETAL GROWTH AND DEVELOPMENT IN CHILDREN

During childhood, skeletal structures grow in length and diameter and sustain a large increase in bone mass. Alterations in musculoskeletal structure and function may develop as a result of normal growth and developmental processes or as a result of impairment of skeletal development caused by hereditary or congenital influences.

### Alterations During Normal Growth Periods

Infants and children undergo changes in muscle tone and joint motion during growth and development. Toeing-in, toeing-out, bowlegs, and knock-knees occur frequently in infancy and childhood.<sup>33</sup> These changes usually cause few problems and are corrected during normal growth processes. The normal folded position of the fetus in utero causes physiologic flexion contractures of the hips and a froglike appearance of the lower extremities (Fig. 42-9). The hips are externally rotated, and the patellae point outward, whereas the feet appear to point forward because of the internal pulling force of the tibiae. During the first year of life, the lower extremities begin to straighten out in preparation for walking. Internal and external rotation become equal, and the hips extend. Flexion contractures of the shoulders, elbows, and knees also are commonly seen in newborns, but they should disappear by 4 to 6 months of age.<sup>34</sup>

### Torsional Deformities

All infants and toddlers have lax ligaments that become tighter with age and assumption of the weight-bearing posture. The hypermobility that accompanies joint laxity coupled with the torsional, or twisting, forces exerted on the limbs during

## KEY CONCEPTS

### DEVELOPMENTAL SKELETAL DISORDERS

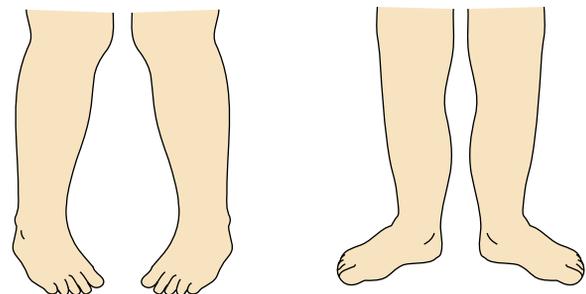
- Many disorders of early infancy are caused by intrauterine positions and resolve as the child grows.
- Infants and toddlers have lax ligaments that predispose to skeletal disorders caused by twisting or torsional forces.
- Bone growth in infants and children occurs at the epiphysis. Separation of the epiphyseal growth plate ruptures the blood vessels that nourish the epiphysis, causing cessation of growth and shortened extremity length.

growth are responsible for a number of variants seen in young children. Torsional forces caused by intrauterine positions or sleeping and sitting patterns twist the growing bones and can produce the deformities as a child grows and develops.

In infants, the femur normally is rotated to an anteverted position with the femoral head and neck rotated anteriorly with respect to the femoral condyles. Femoral anteversion (*i.e.*, medial rotation) decreases from an average 40 degrees at birth to approximately 15 degrees at maturity. The normal tibia is externally rotated approximately 5 degrees at birth and 15 degrees at maturity. Torsional abnormalities frequently demonstrate a familial tendency.

**Toeing-in and Toeing-out.** The foot progression angle describes the angle between the axis of the foot and the line of progression. It is determined by watching the child walking and running. Figure 42-10 illustrates the position of the foot in toeing-in and toeing-out.

Toeing-in (*i.e.*, metatarsus adductus) is the most common congenital foot deformity, affecting boys and girls equally. It can be caused by torsion in the foot, lower leg, or entire leg. Toeing-in caused by adduction of the forefoot (*i.e.*, congenital metatarsus adductus) usually is the result of the fetal position maintained in utero. It may occur in one foot or both feet. A supple deformity can be passively manipulated into a straight position and requires no treatment. Treatment consisting of



Toeing-in

Toeing-out

■ **FIGURE 42-10** ■ Position of feet in toeing-in and toeing-out.

serial long leg casting or a brace that pushes the metatarsals (not the hindfoot) into abduction usually is required in a fixed deformity (*i.e.*, one in which the forefoot cannot be passively manipulated into a straight position).

Toeing-out is a common problem in children and is caused by external femoral torsion. This occurs when the femur can be externally rotated to approximately 90 degrees but internally rotated only to a neutral position or slightly beyond. Because the femoral torsion persists when a child habitually sleeps in the prone position, an external tibial torsion also may develop. If external tibial torsion is present, the feet point lateral to the midline of the medial plane. External tibial torsion rarely causes toeing-out; it only intensifies the condition. Toeing-out usually corrects itself as the child becomes proficient in walking. Occasionally, a night splint is used. Toeing-in and toeing-out are less noticeable when the child is running or barefoot. Overcorrection of a supple foot deformity can cause flatfoot deformity, but a rigid deformity that is untreated can cause pain and improper fitting of footwear.

**Tibial Torsion.** Tibial torsion is determined by measuring the thigh-foot angle, which is done with the ankle and knee positioned at 90 degrees (Fig. 42-11A). In this position, the foot

normally rotates outward. *Internal tibial torsion* (*i.e.*, bowing of the tibia) is a rotation of the tibia that makes the feet appear to turn inward. It is the most common cause of toeing-in in children younger than 2 years of age. It is present at birth and may fail to correct itself if children sleep on their knees with the feet turned in or sit on in-turned feet. It is thought to be caused by genetic factors and intrauterine compression, such as an unstretched uterus during a first pregnancy or intrauterine crowding with twins or multiple fetuses. Tibial torsion improves naturally with growth, but this may take years.<sup>35</sup> The Denis Browne splint, a bar to which shoes are attached, may be used to put the feet into mild external rotation while the child is sleeping.

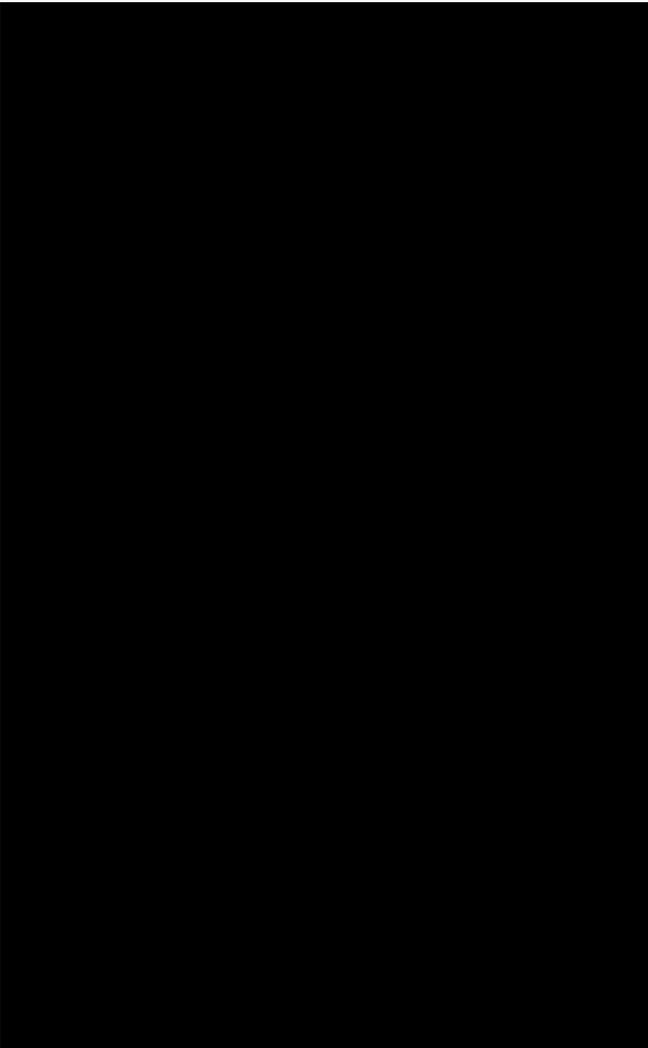
*External tibial torsion*, a much less common disorder, is associated with calcaneovalgus foot and is caused by a normal variation of intrauterine positioning or a neuromuscular disorder. It is characterized by an abnormally positive thigh-foot angle of 30 to 50 degrees. The condition corrects itself naturally, and treatment is observational. Significant improvement begins during the first year with the onset of ambulation and usually is complete by 2 to 3 years of age.<sup>34</sup> The normal adult exhibits 20 degrees of tibial torsion.

**Femoral Torsion.** *Femoral torsion* refers to abnormal variations in hip rotation. Hip rotation is measured at the pelvic level with the child in the prone position and the knees flexed at a 90-degree angle. In this position, the hip is in a neutral position. Rotating the lower leg outward produces internal or medial femoral rotation; rotating it inward produces external or lateral rotation (see Fig. 42-11B). During measurement of hip rotation, the legs are allowed to fall to full internal rotation by gravity alone; lateral rotation is measured by allowing the legs to fall inward and cross. Hip rotation in flexion and extension also can be measured with computed tomography (CT). By 1 year of age, there is normally approximately 45 degrees of internal and 45 degrees of external rotation.<sup>34</sup>

*Internal femoral torsion*, also called *femoral anteversion*, is a normal variant commonly seen during the first 6 years of life, especially in 3- and 4-year-old girls.<sup>33</sup> Characteristically, there is 80 to 90 degrees of internal rotation of the hip in the prone position.<sup>34</sup> The condition is thought to be related to increased laxity of the anterior capsule of the hip such that it does not provide the stable pressure needed to correct the anteversion that is present at birth. Children are most comfortable sitting in the "W" position with their hips between their knees (Fig. 42-12). It is believed that this position allows the lower leg to act as a lever, producing torsional changes in the femur. When the child stands, the knees turn in and the feet appear to point straight ahead; when the child walks, knees and toes point in. Children with this problem are encouraged to sit cross-legged or in the so-called *tailor position*. If left untreated, the tibiae compensate by becoming externally rotated so that by 8 to 12 years of age the knees may turn in but the feet no longer do. This can result in patellofemoral malalignment with patellar subluxation or dislocation and pain. A derotational osteotomy may be done in severe cases or if there is functional disability.

### Genu Varum and Genu Valgum

*Genu varum* or *bowlegs* is an outward bowing of the knees greater than 1 inch when the medial malleoli of the ankles are touching (Fig. 42-13). Most infants and toddlers have some





bowing of their legs up to age 2 years. If there is a large separation between the knees (>15 degrees) after 2 years of age, the child may require bracing. The child also should be evaluated for diseases such as rickets or tibia vara (*i.e.*, Blount's disease to be discussed).



■ **FIGURE 42-13** ■ Normal genu varum (bowlegs) in a toddler (*left*) and genu valgum (knock-knees) in a toddler (*right*), which is often seen in children between 2 and 6 years of age. (From Weinstein S.L., Buckwalter J.A. [1994]. *Turek's orthopaedics* [5th ed.]. Philadelphia: J.B. Lippincott)

*Genu valgum* or *knock-knees* is a deformity in which there is decreased space between the knees (see Fig. 42-13). The medial malleoli in the ankles cannot be brought in contact with each other when the knees are touching. It is seen most frequently in children between the ages of 3 and 5 years and should resolve by 5 to 8 years of age.<sup>34</sup> The condition usually is the result of lax medial collateral ligaments of the knee and may be exacerbated by sitting in the "M" position. Genu valgum can be ignored to age 7 years, unless it is more than 15 degrees, unilateral, or associated with short stature. It usually resolves spontaneously and rarely requires treatment. If genu varum or genu valgum persists and is uncorrected, osteoarthritis may develop in adulthood as a result of abnormal intra-articular stress. Genu varum can cause gait awkwardness and increased risk of sprains and fractures. Uncorrected genu valgum may cause subluxation and recurrent dislocation of the patella, with a predisposition to chondromalacia and joint pain and fatigue.

### Hereditary and Congenital Deformities

Congenital deformities are abnormalities that are present at birth. They range in severity from mild limb deformities, which are relatively common, to major limb malformations, which are relatively rare. There may be a simple webbing of the fingers or toes (syndactyly), the presence of an extra digit (*i.e.*, polydactyly), or the absence of a bone such as the phalanx, rib, or clavicle. Joint contractures and dislocations produce more severe deformity, as does the absence of entire bones, joints, or limbs. An epidemic of limb deformities occurred from 1957 to 1962 as a result of maternal ingestion of thalidomide. This drug was withdrawn from the market in 1961.

Congenital deformities are caused by many factors, some unknown. These factors include genetic influences, external agents that injure the fetus (*e.g.*, radiation, alcohol, drugs, viruses), and intrauterine environmental factors. Many of the organic bone matrix components have been identified only recently, and their interactions found to be more complex than originally thought. Diseases associated with abnormalities in bone matrix include those with deficient collagen synthesis and decreased bone mass.

### Osteogenesis Imperfecta

Osteogenesis imperfecta is a hereditary disease characterized by defective synthesis of type I collagen.<sup>36</sup> It is one of the most common hereditary bone diseases, with an occurrence rate of approximately 1 case in 10,000 births.<sup>12</sup> Although it usually is transmitted as an autosomal dominant trait, a distinct form of the disorder with multiple lethal defects is thought to be inherited as an autosomal recessive trait.<sup>10</sup> In some cases the defect is caused by a spontaneous mutation.

The clinical manifestations of osteogenesis imperfecta include a spectrum of disorders marked by extreme skeletal fragility. Four major subtypes have been identified.<sup>10</sup> Type II is at one end of the spectrum and is uniformly fatal in utero or during early perinatal life. Less severe disease occurs when the disorder is inherited as a dominant trait. The skeletal system is not so weakened, and fractures often do not appear until the child becomes active and starts to walk, or even later in childhood. These fractures heal rapidly, although with a poor-quality callus. Other problems associated with defective connective tissue synthesis include short stature, thin skin, blue or gray

sclera, abnormal tooth development, hypotonic muscles, loose-jointedness, scoliosis, and a tendency toward hernia formation. Hearing loss caused by otosclerosis of the middle and inner ear is common in affected adults.

There is no known medical treatment for correction of the defective collagen synthesis that is characteristic of osteogenesis imperfecta. Instead, treatment modalities focus on preventing and treating fractures.

### Congenital Clubfoot

Clubfoot, or talipes, is a congenital deformity of the foot that can affect one or both feet. Like congenital dislocation of the hip, its occurrence follows a multifactorial inheritance pattern. The condition has an incidence of 1 case per 1000 live births and occurs twice as often in males as in females.<sup>37</sup> Clubfoot is associated with chromosomal abnormalities and may be associated with other congenital syndromes that are transmitted by mendelian inheritance patterns (see Chapter 4). However, it is most commonly idiopathic and found in healthy infants in whom no genetic or chromosomal abnormality or other extrinsic cause can be found.

Although the exact cause of clubfoot is unknown, three theories are generally accepted: an anomalous development occurs during the first trimester of pregnancy, the leg fails to rotate inward and move from the equinovarus position at approximately the third month, or the soft tissues in the foot do not mature and lengthen. Maternal smoking is associated with occurrence of clubfoot, and the risk increases enormously when combined with a family history.<sup>38</sup>

In forefoot adduction, which accounts for approximately 95% of idiopathic cases, the foot is plantar flexed and inverted. This is the so-called *equinovarus type* in which the foot resembles a horse's hoof (Fig. 42-14). The other 5% of cases are of the calcaneovalgus type, or reverse clubfoot, in which the foot is dorsiflexed and everted. The reverse clubfoot can occur as an isolated condition or in association with multiple congenital

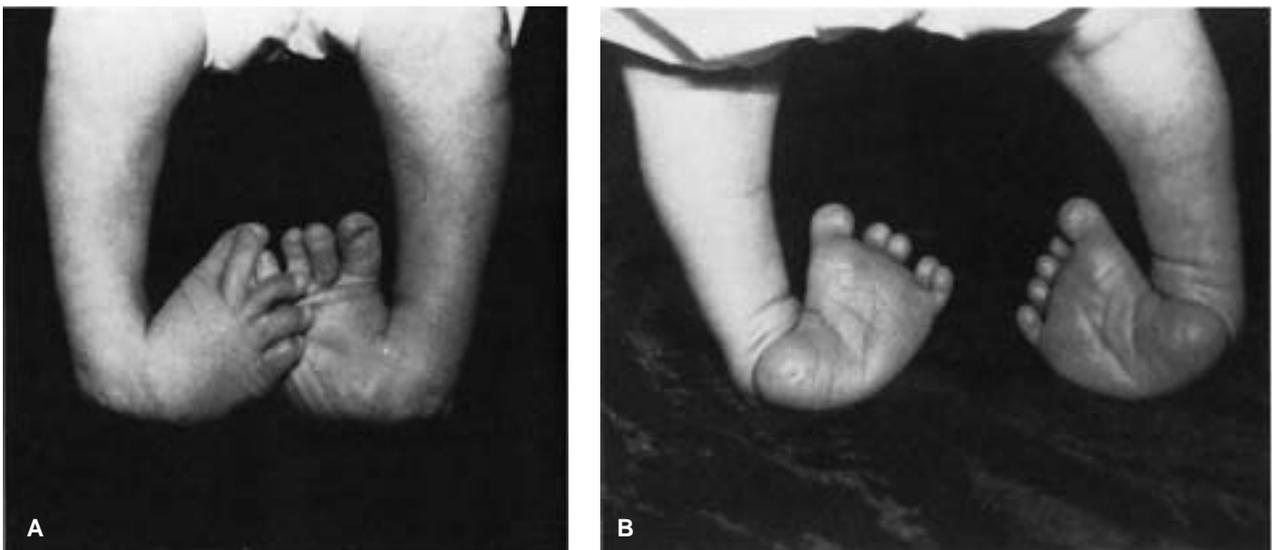
defects. At birth, the feet of many infants assume one of these two positions, but they can be passively overcorrected or brought back into the opposite position. If the foot cannot be overcorrected, some type of correction may be necessary.

Clubfoot varies in severity from a mild deformity to one in which the foot is completely inverted. Treatment is begun as soon as the diagnosis is made and usually is effective within a short period. Serial manipulations and casting are used to gently correct each component in the forefoot varus, the hindfoot varus, and the equinus. The treatment is continued until the foot is in a normal position with full correction evident clinically and on radiographic studies. Surgery may be required for severe deformities or when nonoperative treatment methods are unsuccessful.

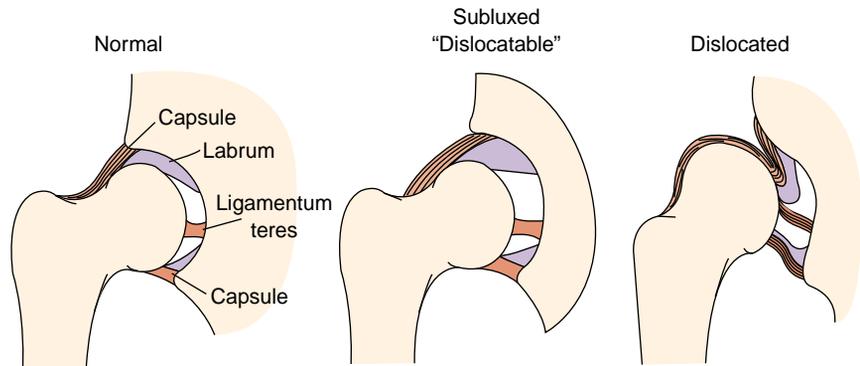
### Developmental Dysplasia of the Hip

Developmental dysplasia of the hip, formerly known as *congenital dislocation of the hip*, is an abnormality in hip development that leads to a wide spectrum of hip problems in infants and children, including hips that are unstable, malformed, subluxated, or dislocated.<sup>10-12</sup> In less severe cases, the hip joint may be unstable, with excessive laxity of the joint capsule, or subluxated, so that the joint surfaces are separated and there is a partial dislocation (Fig. 42-15). With dislocated hips, the head of the femur is located outside of the acetabulum.

The results of newborn screening programs have shown that 1 of 100 infants have some evidence of hip instability; however, dislocation of the hip is seen in 1.5 of every 1000 live births.<sup>12</sup> The left hip is involved three times more frequently than the right hip because of the left occipital intrauterine positioning of most infants.<sup>39</sup> In white infants, developmental dysplasia of the hips occurs most frequently in first-born children and is six times more common in female than in male infants.<sup>40</sup> The cause of developmental dysplasia of the hip is multifactorial, with physiologic, mechanical, and postural factors playing a role. A positive family history and generalized



■ **FIGURE 42-14** ■ Severe clubfoot deformity. (A) The heel is in severe varus, and the forefoot is adducted and inverted. (B) The cavus deformity results from the slightly pronated position of the forefoot in relation to the hindfoot. (From Weinstein S.L., Buckwalter J.A. [1994]. *Turek's orthopaedics* [5th ed.]. Philadelphia: J.B. Lippincott)



■ **FIGURE 42-15** ■ Normal and abnormal relationships of hip joint structure. (Adapted from Dunn P.M. [1969]. Congenital dislocation of the hip. *Proceedings of the Royal Society of Medicine* 62, 1035–1037)

laxity of the ligaments are related. The increased frequency in girls is thought to result from their susceptibility to maternal estrogens and other hormones associated with pelvic relaxation. Dislocation also may result from environmental factors such as fetal position, a tight uterus that prevents fetal movement, and breech delivery.

Early diagnosis of a developmental dysplasia of the hip is important because treatment is easiest and most effective if begun during the first 6 months of life. Repeated dislocation causes damage to the femoral head and the acetabulum. Clinical examinations to detect dislocation of the hip should be done at birth and every several months during the first year of life. In infants, signs of dislocation include asymmetry of the hip or gluteal folds, shortening of the thigh so that one knee (on the affected side) is higher than the other, and limited abduction of the affected hip (Fig. 42-16). The asymmetry of gluteal folds is not definitive but indicates the need for further evaluation. In the older child, instability of the hip may produce a delay in walking or eventually cause a characteristic waddling gait. Diagnosis is confirmed by radiography. Ultrasound is used to diagnose the conditions in newborns and infants from birth to 4 months of age.<sup>40</sup>

The treatment of a developmental dysplasia should be individualized and depends on whether the hip is subluxated or dislocated. Mild instability often resolves without treatment. The best results are obtained if the treatment is begun before

changes in the hip structure (e.g., 2 to 3 months) prevent it from being reduced by gentle manipulation or abduction devices. Infants with dislocated hips caused by anatomic changes and toddlers who may lack development of the acetabular socket require more aggressive treatment, such as open reduction and joint reconstruction. Treatment at any age includes reduction of the dislocation and immobilization of the legs in an abducted position. With children younger than 3 years, skin traction is used when reduction cannot be easily obtained. This treatment is followed by several months of immobilization in a hip spica cast, plaster splints, or an abduction splint. Older children or adults with an unreduced dislocatable hip may require hip surgery because of damage to the articulating surface of the joint.

## Disorders of Later Development

Developmental disorders of bones and joints are those that are not present at birth but make their appearance during later periods of development. Some may have a genetic component, some present during periods of rapid growth, and others may develop as the result of trauma and stress imposed by activities associated with various periods of development.

### Developmental Disorders of the Hip

**Legg-Calvé-Perthes Disease.** Legg-Calvé-Perthes disease, or coxa plana, is an osteonecrotic disease of the proximal femoral (capital) epiphysis, which is the growth center for the head of the femur. It occurs in 1 of 1200 children, affecting primarily those between ages 2 and 13 years, with a peak incidence between 4 and 9 years.<sup>41</sup> It occurs primarily in boys and is much more common in whites than African Americans. Although no definite genetic pattern has been established, it occasionally affects more than one family member.

The cause of Legg-Calvé-Perthes disease is unknown. The disorder usually is insidious in onset and occurs in otherwise healthy children. However, it may be associated with acute trauma. Affected children usually have a shorter stature. Undernutrition has been suggested as a causative factor. When girls are affected, they usually have a poorer prognosis than boys because they are skeletally more mature and have a shorter period for growth and remodeling than do boys of the same age. Although both legs can be affected, in 85% of cases, only one leg is involved.<sup>35</sup>

The primary pathologic feature of Legg-Calvé-Perthes disease is an avascular necrosis of the bone and marrow involving



■ **FIGURE 42-16** ■ An 18-month-old girl has congenital dysplasia of the left hip. (From Weinstein S.L., Buckwalter J.A. [1994]. *Turek's orthopaedics* [5th ed.]. Philadelphia: J.B. Lippincott)

the epiphyseal growth center in the femoral head. The disorder may be confined to part of the epiphysis, or it may involve the entire epiphysis. In severe cases, there is a disturbance in the growth pattern that leads to a broad, short femoral neck. The necrosis is followed by slow absorption of the dead bone during a period of 2 to 3 years. Although the necrotic trabeculae eventually are replaced by healthy new bone, the epiphysis rarely regains its normal shape.

Legg-Calvé-Perthes disease has an insidious onset with a prolonged course. The main symptoms are pain in the groin, thigh, or knee and difficulty in walking. The child may have a painless limp with limited abduction and internal rotation and a flexion contracture of the affected hip. The age of onset is important because young children have a greater capability for remodeling of the femoral head and acetabulum, so less flattening of the femoral head occurs. Early diagnosis is important and is based on correlating physical symptoms with radiographic findings that are related to the stage of the disease.

The goal of treatment is to reduce deformity and preserve the integrity of the femoral head. Conservative and surgical interventions are used in the treatment of Legg-Calvé-Perthes disease. Treatment involves abduction casts or braces to keep the legs separated in abduction with mild internal rotation. Surgery may be done to contain the femoral head in the acetabulum. The best surgical results are obtained when surgery is done early, before the epiphysis becomes necrotic.

**Slipped Capital Femoral Epiphysis.** Normally, the proximal femoral epiphysis unites with the neck of the femur between 16 and 19 years of age. Before this time (10 to 14 years of age in girls and 10 to 16 years in boys), the femoral head may slip from its normal position directly at the head of the femur and become displaced medially and posteriorly.<sup>41</sup> The head is held in the acetabulum by the ligamentum teres, and the neck of the femur is pulled upward and outward. This produces an anterolateral and superior adduction with extension deformity. The condition occurs with an estimated frequency of between 1 in 100,000 to 1 in 800,000.<sup>35</sup> It is the most common disorder of the hip in adolescents.

The cause of slipped capital femoral epiphysis is obscure, but it may be related to the child's susceptibility to stress on the femoral neck as a result of genetics or abnormal structure. Boys are affected twice as often as girls, and in approximately one half of cases, the condition is bilateral. Affected children often are overweight with poorly developed secondary sex characteristics or, in some instances, are extremely tall and thin. In many cases, there is a history of rapid skeletal growth preceding displacement of the epiphysis. The condition also may be affected by nutritional deficiencies or endocrine disorders such as hypothyroidism, hypopituitarism, and hypogonadism. Rapid growth after administration of growth hormone has been associated with displacement of the epiphysis.

Children with the condition often report referred knee pain accompanied by difficulty in walking, fatigue, and stiffness. The diagnosis is confirmed by radiographic studies in which the degree of slipping is determined and graded according to severity. Early treatment is imperative to prevent lifelong crippling. Avoidance of weight bearing on the femur and bed rest are essential parts of the treatment. Traction or gentle manipulation under anesthesia is used to reduce the slip. Surgical insertion of pins to keep the femoral neck and head of the femur aligned is a common method of treatment for children with moderate or

severe slips. Crutches are used for several months after surgical correction to prevent full weight bearing until the growth plate is sealed by the bony union.

Children with the disorder must be followed up closely until the epiphyseal plate closes. Long-term prognosis depends on the amount of displacement that occurs. Complications include avascular necrosis, leg shortening, malunion, and problems with the internal fixation. Degenerative arthritis may develop, requiring joint replacement later in life.

### Disorders of the Knee

**Blount's Disease.** *Idiopathic tibia vara*, or *Blount's disease*, is a developmental deformity of the medial half of proximal tibial epiphysis that results in a progressive varus angulation below the knee (Fig. 42-17). It is the most common cause of pathologic genu varum and seen most often in black children, females, obese children, and early walkers.<sup>42</sup> Onset can occur early in infancy or later, during adolescence. Adolescent Blount's disease occurs in the second decade of life, is seen in persons who are above the 95th percentile in height and weight, and is usually unilateral.<sup>42</sup>

Long leg braces are used for treatment in early-onset disease. If progression occurs, or onset is late, surgery is done to correct the angulation and prevent further progression.

**Osgood-Schlatter Disease.** Osgood-Schlatter disease involves microfractures in the area where the patellar tendon inserts into the tibial tubercle, which is an extension of the proximal tibial epiphysis.<sup>34</sup> This area is particularly vulnerable to injury caused by sudden or continued strain from the patellar tendon during periods of growth, particularly in athletic individuals. It occurs most frequently in boys between the ages of 11 and 15 years and in girls between 8 and 13 years.

The disorder is characterized by pain in the front of the knee that is associated with inflammation and thickening of the patellar tendon. The pain usually is associated with specific activities, such as kneeling, running, bicycle riding, or stair climbing. There is swelling, tenderness, and increased prominence of the tibial tubercle. The symptoms usually are self-limiting.



■ **FIGURE 42-17** ■ Rotational deformity of the proximal tibia, especially when unilateral, suggests tibia vara (Blount's disease). (From Weinstein S.L., Buckwalter J.A. [1994]. *Turek's orthopaedics* [5th ed.]. Philadelphia: J.B. Lippincott)

They may recur during growth periods but usually resolve after closure of the tibial growth plate. In some cases, limitations on activity, tibial bands or braces to immobilize the knee, anti-inflammatory agents, and application of cold are necessary to relieve the pain. The objective of treatment is to release tension on the quadriceps to permit revascularization and reossification of the tibial tubercle. Complete resolution of symptoms through healing (physical close) of the tibia tubercle usually requires 12 to 24 months.<sup>34</sup> Occasionally, minor symptoms or an increased prominence of the tibial tubercle may continue into adulthood. In some cases, a high-riding patella can cause dislocation with chondromalacia of the patella and result in degenerative arthritis.

## Scoliosis

Scoliosis is a lateral deviation of the spinal column that may or may not include rotation or deformity of the vertebrae. It has been estimated that more than 500,000 adults in the United States have scoliosis.<sup>43</sup> It is most commonly seen during adolescence and is eight times more common among girls than boys. Scoliosis can develop as the result of another disease condition, or it can occur without known cause. Idiopathic scoliosis accounts for 75% to 80% of cases of the disorder. The other 20% to 25% of cases result from more than 50 different causes, including poliomyelitis, congenital hemivertebrae, neurofibromatosis, and cerebral palsy. Although minor curves are relatively common (affecting approximately 2% of the population), it has been estimated that less than 0.1% of U.S. schoolchildren have severe idiopathic scoliosis.

### Types of Scoliosis

Scoliosis is classified as postural or structural. With postural scoliosis, there is a small curve that corrects with bending. It can be corrected with passive and active exercises. Structural scoliosis does not correct with bending. It is a fixed deformity classified according to the cause: congenital, neuromuscular, and idiopathic.

**Congenital Scoliosis.** Congenital scoliosis is caused by disturbances in vertebral development during the sixth to eighth week of embryologic development. There are structural anomalies in the vertebrae that can cause a severe curvature. The child may have other anomalies and neurologic complications if the spine is involved. Early diagnosis and treatment of progressive curves are essential for children with congenital scoliosis.

**Neuromuscular Scoliosis.** Neuromuscular scoliosis develops from neuropathic or myopathic diseases. Neuropathic scoliosis is seen with cerebral palsy, myelodysplasia, and poliomyelitis. There is often a long, "C"-shaped curve from the cervical to the sacral region. In children with cerebral palsy, severe deformity may make treatment difficult. Myopathic neuromuscular scoliosis develops with Duchenne's muscular dystrophy and usually is not severe.

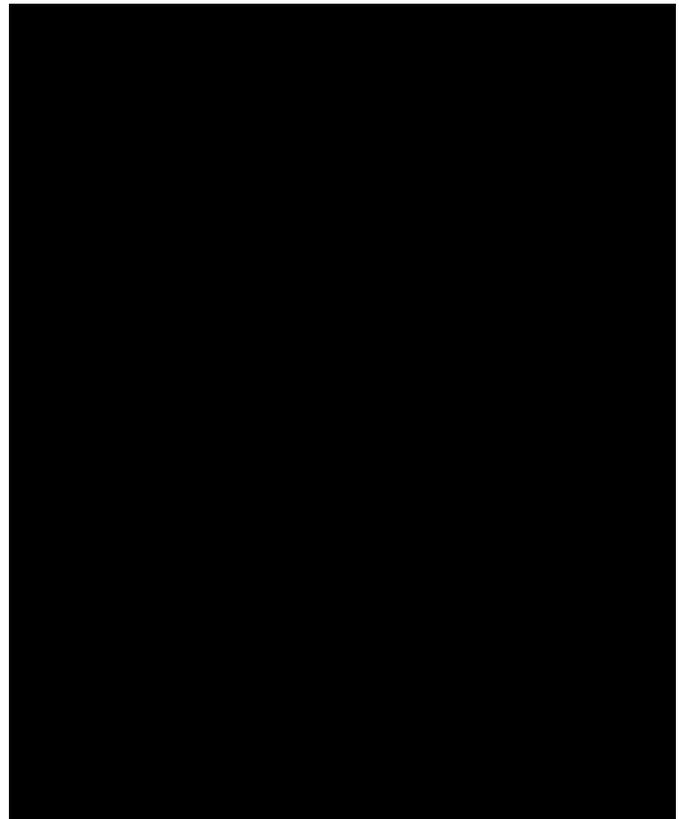
**Idiopathic Scoliosis.** Idiopathic scoliosis is a structural spinal curvature for which no cause has been established. It seems likely that genetics is involved, and mother-daughter pairings are common. Growth and mechanical factors also seem to play a role.

Idiopathic scoliosis can be divided into three groups on the basis of age at onset: infantile (birth to 3 years), juvenile (4 to

10 years), and adolescent (11 years and older).<sup>44</sup> Adolescent scoliosis is the most common type of idiopathic scoliosis. It accounts for approximately 80% of cases, and is seen most commonly in girls. An increase in joint laxity, which causes excessive joint motion and is found commonly in girls, has been associated with development of idiopathic scoliosis. Delayed puberty and menarche are other risk factors for the development of scoliosis.<sup>45</sup> Although the curve may be present in any area of the spine, the most common curve is a right thoracic curve, which produces a rib prominence on the convex side and hypokyphosis from rotation of the vertebral column around its long axis as the spine begins to curve.

Scoliosis usually is first noticed because of the deformity it causes. A high shoulder, prominent hip, or projecting scapula may be noticed by a parent or in a school screening program. Idiopathic scoliosis usually is a painless process, although pain may be present in severe cases, usually in the lumbar region. The pain may be caused by pressure on the ribs or on the crest of the ilium. There may be shortness of breath as a result of diminished chest expansion and gastrointestinal disturbances from crowding of the abdominal organs. Adults with less severe deformity may experience mild backache. If scoliosis is left untreated, the curve may progress to an extent that compromises cardiopulmonary function and creates a risk for neurologic complications.

Early diagnosis of scoliosis can be important in the prevention of severe spinal deformity. The cardinal signs of scoliosis are uneven shoulders or iliac crest, prominent scapula on the convex side of the curve, malalignment of spinous processes, asymmetry of the flanks, asymmetry of the thoracic cage, and rib hump or paraspinal muscle prominence when bending forward (Fig. 42-18). A complete physical examination is necessary for



children with scoliosis because the defect may be indicative of other, underlying pathology. Diagnosis of scoliosis is confirmed by radiographs, CT scans, MRI, or myelography.

The treatment of scoliosis depends on the severity of the deformity and the likelihood of progression. A brace may be used to control the progression of the curvature during growth and can provide some correction. Surgical intervention with instrumentation and spinal fusion is done in severe cases. Unlike bracing, which is intended to halt progression of the curvature, surgical intervention is used to decrease the curve.

**In summary,** skeletal disorders can result from congenital or hereditary influences or from factors that occur during normal periods of skeletal growth and development. Newborn infants undergo normal changes in muscle tone and joint motion, causing torsional conditions of the femur or tibia. Many of these conditions are corrected as skeletal growth and development take place.

Osteogenesis imperfecta is a rare autosomal hereditary disorder characterized by defective synthesis of connective tissue, including bone matrix. It results in poorly developed bones that fracture easily. Developmental dysplasia of the hip includes a range of structural abnormalities. Dislocated hips are always treated to prevent changes in the anatomic structure. Other childhood skeletal disorders, such as the osteochondroses, slipped capital femoral epiphysis, and scoliosis, are not corrected by the growth process. These disorders are progressive, can cause permanent disability, and require treatment. Disorders such as congenital dislocation of the hip and congenital clubfoot are present at birth. Both of these disorders are best treated during infancy.

Scoliosis represents a lateral deviation of the spinal column that may or may not include rotation or deformity of the vertebrae. It can occur as the result of congenital deformities of the vertebrae, neuromuscular diseases that produce weakness of the muscles that support the spinal column, or curvature for which no cause has been established (idiopathic scoliosis). Idiopathic scoliosis, which is the most common form, occurs more frequently in girls than boys. Treatment depends on the severity of the deformity and the likelihood of progression. If left untreated, large curves may progress to the extent that they compromise cardiopulmonary function and create a risk of neurologic complications.

## REVIEW QUESTIONS

- Compare muscle strains and ligamentous sprains.
- Describe the healing process of soft tissue injuries.
- Differentiate open fractures from closed ones.
- Describe the fracture healing process and relate individual and local factors to the healing process in bone.
- Explain the importance of immobilization for fracture healing.
- Describe the pathogenesis of osteomyelitis and differentiate among osteomyelitis caused by spread from a contaminated wound, hematogenous osteomyelitis, and osteomyelitis caused

by vascular insufficiency in terms of etiologies, manifestations, and treatment.

- Define *osteonecrosis* and cite major causes of osteonecrosis.
- Differentiate between the properties of benign and malignant bone tumors.
- Describe common torsional deformities that occur in infants and small children, proposed mechanisms of development, diagnostic methods, and treatment.
- Define *genu varum* and *genu valgum*.
- List the problems that occur because of defective tissue synthesis in *osteogenesis imperfecta*.
- Characterize the abnormalities associated with developmental dysplasia of the hip and methods of diagnosis.
- Define the term *osteochondroses* and describe the pathology and symptomatology of Legg-Calvé-Perthes disease and Osgood-Schlatter disease.
- Describe the pathology associated with a slipped capital femoral epiphysis and explain why early treatment is important.
- Discuss the diagnosis and treatment of idiopathic scoliosis.



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