CHAPTER 34

Bone and Joint Infections

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Definition

Osteomyelitis is an inflammation of bone, with the term generally used to indicate pyogenic infection. Osteomyelitis can refer to a process that is acute, subacute, or chronic in nature. *Septic arthritis* refers to bacterial inflammation of a joint. Osteomyelitis and septic arthritis usually are of hematogenous origin, but either condition can arise through direct inoculation.

Historical Aspects

Osteomyelitic lesions have been found in Egyptian mummies dating as far back as 4,000 years.[255] Before the advent of modern antibiotic therapy in the mid-twentieth century, the mortality from chronic osteomyelitis was as high as 50 percent.[330] After the introduction of penicillin in 1944, the mortality eventually decreased to less than 1 percent.[231] However, despite the availability of antibiotics and the resultant reduction in mortality, pyogenic infection of bones and joints continues to be one of the most frequent and potentially devastating problems that the pediatric orthopaedist will encounter.

Although bone and joint infections are quite common, the diagnosis of osteomyelitis and septic arthritis is not always straightforward. A high index of suspicion must be maintained in order to make an accurate and prompt diagnosis of bone or joint sepsis. Medical advances have changed the classic presenting symptoms of children with osteoarticular infections.[81,100] Fulminating, chronic osteomyelitis is observed much less frequently today than it was in the past. Children today are more likely to be seen in the early stages of disease, often with a clinical picture clouded by previous oral antibiotic therapy, or they may be seen with a subacute form of the disease.

Although osteomyelitis and septic arthritis are caused by bacteria, cultures will frequently be negative. Because cultures are often nondiagnostic, a number of different criteria for the diagnosis of osteomyelitis and septic arthritis have been proposed (Tables 34–1 and 34–2).[247,248,273] Ultimately, however, clinicians may have to rely on their clinical skills and expertise rather than on laboratory tests and imaging studies to make the correct diagnosis. If the diagnosis is accurate and appropriate therapy is initiated in a timely manner, pyogenic osteoarticular infections can be successfully treated with little long-term morbidity.[249]

Acute Hematogenous Osteomyelitis and Septic Arthritis

EPIDEMIOLOGY

Acute hematogenous osteomyelitis (AHO) and *septic arthritis* are most common in the first decade of life.[132,133,169,273] Although some studies show an equal male-female ratio,[100] most investigators have reported a predominance among males.[133,136,231,248] Some authors have attributed the male preponderance to the role of trauma in the etiology of osteomyelitis and septic arthritis.[100,250,371,379] A seasonal variation and an ethnic predisposition have been reported in some studies.[133,385]

ETIOLOGY AND PATHOPHYSIOLOGY

To effectively treat osteoarticular infections, it is important to understand what is currently known of their etiology and pathophysiology. Although both osteomyelitis and septic arthritis usually are hematogenous infections that can and often do occur concurrently, their pathophysiology is different.

PATHOPHYSIOLOGY OF ACUTE HEMATOGENOUS OSTEOMYELITIS

Several factors have been implicated in the pathogenesis of AHO. These factors include the vascular and cellular anatomy of the juxtaphyseal region, trauma, and cellular mediators of inflammation.

Vascular Anatomy. Hematogenous osteomyelitis most frequently originates in the metaphysis of long bones. Early animal studies of osteomyelitis demonstrated that when either India ink particles or bacteria were injected intravenously (IV), both would lodge in the vascular spaces of the metaphysis.[157,193] Hobo theorized that the blood supply to the metaphysis consisted of arterial loops that emptied into venous sinusoids. He believed that the resulting turbulence

TABLE 34–1 **Criteria for the Diagnosis of Osteomyelitis**

Morrey and Peterson's Criteria

Definite: The pathogen is isolated from the bone or adjacent soft tissue, or there is histologic evidence of osteomyelitis.

Probable: A blood culture is positive in the setting of clinical and radiographic features of osteomyelitis.

Likely: Typical clinical findings and definite radiographic evidence of osteomyelitis are present, and there is a response to antibiotic therapy.

Peltola and Vahvanen's Criteria

The diagnosis is established when two of the following four criteria are met:

- Pus is aspirated from bone.
- A bone or blood culture is positive.
- The classic symptoms of localized pain, swelling, warmth, and limited range of motion of the adjacent joint are present.
- Radiographic features characteristic of osteomyelitis are present.

and slow flow created an opportune environment for bacteria to lodge and proliferate.[157] Subsequent studies using electron microscopy have shown that these loops are actually terminal branches that originate from "sprouts." These sprouts possess gaps that allow blood cells and bacteria to pass out of the vascular system and into the extravascular space.[149,249,314] Once in the extravascular space, these pathogens may develop into a nidus of infection.

In addition to predisposing to the development of osteomyelitis, the vascular anatomy influences the potential severity and sequelae of infection of the proximal femur in the infant. In the neonate and young infant, before a secondary ossification center develops within the epiphysis, the metaphysis and epiphysis share a common blood supply. Thus, in the infant, osteomyelitis originating in the metaphysis can easily spread to the epiphysis, resulting in total destruction of both the physis and epiphysis (Fig. 34–1).[266,267,351,354] With growth, the physeal plate develops into a barrier between the metaphysis and epiphysis, with each region maintaining its own independent blood supply.

Cellular Anatomy. The unique cellular anatomy of both the metaphysis and the diaphysis also plays a role in the development of AHO. The cancellous bone of the metaphysis is relatively acellular compared to the diaphysis. Consequently, fewer cells of the reticuloendothelial system are present for phagocytosis.[249] In an animal model of AHO, Morrissy and Haynes were able to confirm Hobo's observations that, although the nidus of infection was in the metaphysis, the inflammatory response started in the medullary cavity of the diaphysis.[250] Immediately following a bacteremia, the pathogens were found primarily in the medullary cavity of the diaphysis; however, because of this area's rich reticuloendothelial system, the bacteria were removed during the first 24 hours. Morrissy and Haynes also noted little phagocytic activity in the area of the metaphysis immediately adjacent to the physis, and hypothesized that bacteria in this area were able to proliferate because of the relatively acellular environment. They also theorized that proliferation of bacteria away from the nidus of inflammation might help explain the predilection of AHO for rapidly growing bones. The primary spongiosa (and subsequently the metaphysis) is larger in more rapidly growing bones. Thus, inflammatory cells migrating to the proliferating bacteria have farther to travel, which presumably allows the bacteria more time to establish an active focus of infection (Fig. 34–2).

The cellular anatomy of the cortical bone of the metaphysis also affects the development of AHO. Unlike the dense cortical bone of the diaphysis, the relatively woven cortical bone of the metaphysis permits fairly easy communication with the subperiosteal space. As the inflammatory response progresses, purulent material may eventually pass out of the cortical bone of the metaphysis into the subperiosteal space. If the metaphysis is intra-articular (as it is in the proximal femur, proximal humerus, proximal radius, and distal tibia and fibula), concurrent septic arthritis may develop (Fig. 34–3). If untreated, the infection can also travel down the diaphysis through the haversian canals, eventually depriving the bone of its blood supply.[267] The resulting dead bone, or *sequestrum,* is surrounded by the hypertrophying periosteum, which develops into an *involucrum.* Eventually the involucrum is perforated by sinuses, and drainage occurs.[182] These changes mark the classic stages of chronic osteomyelitis (Fig. 34–4).

Trauma. In 1932, Burrows coined the term *locus minoris resistentiae* to describe the effect that trauma had in lowering resistance to infection.[60] Since then, numerous clinical studies have identified trauma as a factor predisposing to the development of AHO in children.[29,85,94,136,152,222] In addition to these clinical studies, several studies in animals have investigated the role of trauma in the development of osteomyelitis, with ambiguous results. In a series of experiments on rabbits, Whalen and associates successfully produced metaphyseal osteomyelitis by combining bacteremia and injury (trauma),[371] although neither insult alone produced osteomyelitis. In a later study by Morrissy and Haynes, occasional small, limited foci of osteomyelitis were identified in rabbits after the experimental induction of bacteremia alone, whereas significant osteomyelitis developed in almost all cases in which bacteremia was combined with injury.[250] Further investigation is needed to better define the role of trauma in the pathogenesis of AHO.

Cellular Mediators of Inflammation. A complete understanding of the etiology of AHO and the effects of AHO on bone metabolism requires knowledge of the events that occur at the cellular level during the disease process. Although this knowledge is still incomplete, investigators have discovered some of the mediators of this cellular response. The

TABLE 34–2 **Criteria for the Diagnosis of Septic Arthritis in Patients with Negative Cultures**

Morrey and Associates' Criteria

The diagnosis is established when five of the following six criteria are present:

- Temperature exceeds 38.3°C.
- Swelling of the suspected joint is present.
- Pain occurs in the suspected joint and is exacerbated with movement.
- Systemic symptoms are present.
- No other pathologic processes are present.
- There is a satisfactory response to antibiotic therapy.

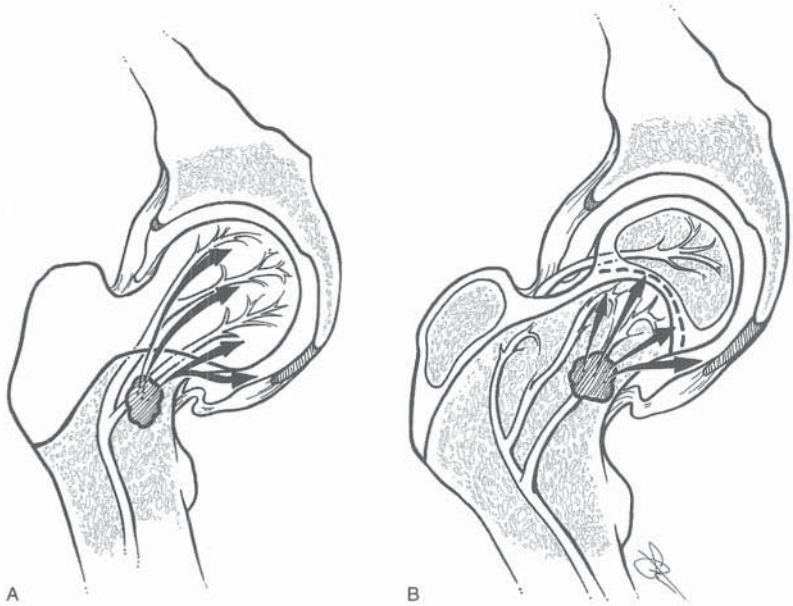


FIGURE 34–1 Vascular anatomy of the proximal femur. **A,** In the neonate the entire epiphysis shares a blood supply with the metaphysis. Thus, infection in the metaphysis can spread into the epiphysis, producing devastating osteonecrosis of the proximal femur. **B,** After development of the secondary ossification center, the epiphysis and metaphysis have separate blood supplies. Thus, in the older child the physis prevents the spread of infection into the epiphysis. However, the metaphysis remains intra-articular, and infection may decompress into the joint, producing septic arthritis.

initial inflammatory reaction is facilitated by the release of interleukin-1 (IL-1) from polymorphonuclear leukocytes (PMNs).[348] IL-1 in turn triggers a further inflammatory response as well as the release of prostaglandin E_2, which is a stimulator of bone resorption.[91,96] Leukotriene B_4 (LTB_4), tumor necrosis factor-alpha (TNF-α), IL-1β, IL-6, and IL-8 have all been shown to be elevated in acute osteomyelitis in both humans and rats.[192,211] The importance of these mediators is perhaps demonstrated by the fact that *Staphylococcus aureus,* the most common organism in AHO, also directly produces prostaglandins.[91]

PATHOPHYSIOLOGY OF SEPTIC ARTHRITIS

Bacteria may enter a joint in several different ways: hematogenously, through direct extension from adjacent osteomyelitis that has decompressed through an intra-articular metaphysis, or by direct inoculation. Once the bacteria are intra-articular, the relatively avascular joint space serves as an excellent culture medium. Although the mechanism of hematogenous seeding of joints is not well understood, the cellular response responsible for the destruction of the articular cartilage is being clarified.

It is believed that the normal bacteremia of childhood leads to intra-articular bacteremia,[164] presumably through the vascular synovium, which lacks a basement membrane.[99,281] The presence of bacteria within the joint causes an increase in the production of synovial fluid and an initial decrease in its viscosity. Within a few days, frank pus accumulates.

In 1924, Phemister hypothesized that chondrolysis secondary to infection was caused by enzymes released from PMNs within the synovium.[277] Curtiss and Klein showed that the destruction of cartilage was a two-stage process. The cartilaginous matrix (glycosaminoglycans) was lost first, followed by a subsequent decrease in collagen.[83,84] Smith and Schurman expanded on this work by using a rabbit model for both *E. coli* and *S. aureus* septic arthritis.[329] Forty-eight hours after induction of infection, the authors found that the glycosaminoglycan content was decreased by 20 percent

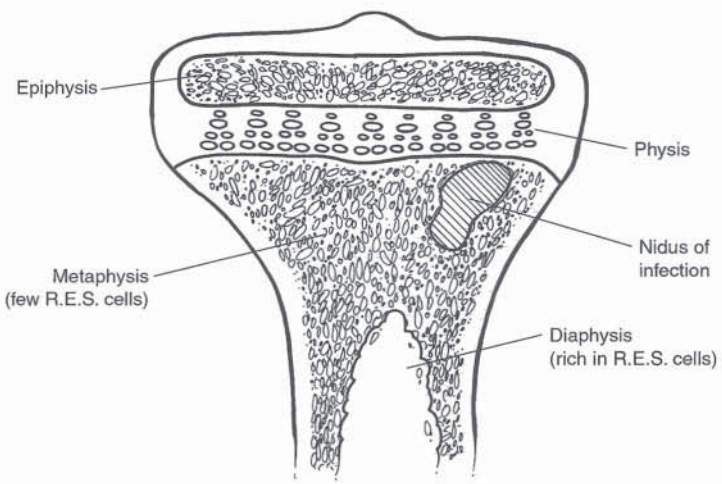


FIGURE 34–2 Acute hematogenous osteomyelitis (AHO) most frequently begins in the metaphysis, where there are fewer reticuloendothelial cells. The host response originates in the diaphysis, which is rich in cells from the reticuloendothelial system. These cells must travel a greater distance in long bones, which may explain the predilection of AHO for the metaphysis of long bones.

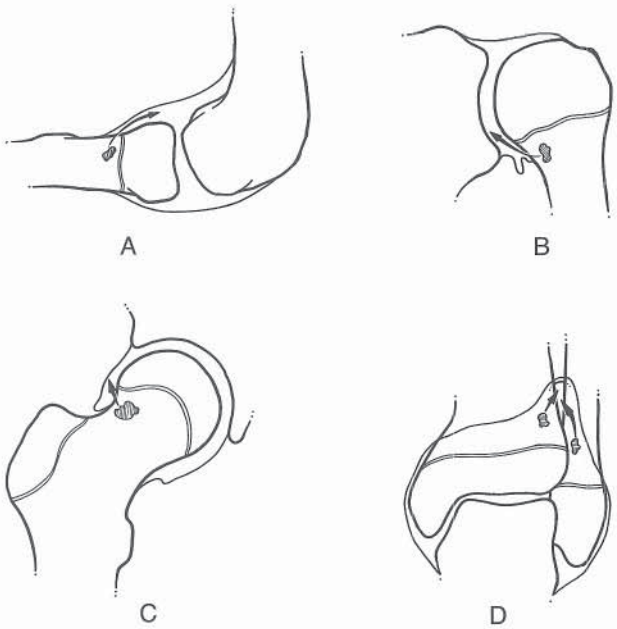


FIGURE 34–3 The metaphyses of the proximal radius (**A**), proximal humerus (**B**), proximal femur (**C**), and distal tibia/fibula (**D**) are intra-articular. Osteomyelitis in these locations may decompress into the joint, producing a concomitant septic arthritis.

in those rabbits with *E. coli* infection and by 42 percent in those with staphylococcal arthritis. Decreases in collagen were not significant in either group until the third week. There was no difference between the two groups in regard to the relative loss of chondroitin sulfate and keratan sulfate. Research by Riegels-Nielsen and associates suggested that this chondrolysis was due to the release of lysosomal enzymes from the synovial membrane.[293] More recent work has identified IL-1β, IL-6, TNF-α, and immunoglobulin G as specific mediators of pyogenic arthritis.[53,189,271]

Other studies have shown that some strains of *S. aureus* may possess unique properties that contribute to the development of septic arthritis. Patti and associates found that when mice were injected with a *Staphylococcus* strain possessing a collagen-binding adhesion (Cna) gene, more than 70 percent of the animals developed septic arthritis; when mice were injected with a *Staphylococcus* strain without the Cna gene, only 27 percent of the animals developed septic arthritis.[271] Other investigators have noted the predilection of *Staphylococcus* to bind to cartilage.[253,332] Williams and associates reported that a sterile staphylococcal medium increased the release of gelatinolytic, collagenolytic, and caseinolytic activity into the medium of isolated chondrocytes.[375,376]

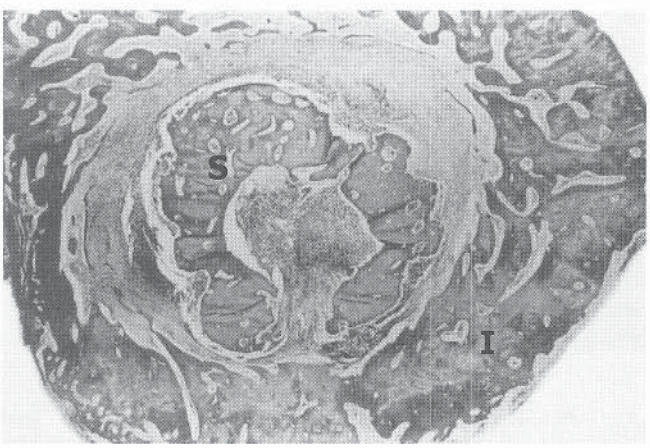


FIGURE 34–4 Cross section of a long bone showing involucrum (I) surrounding sequestrum (S) (necrotic cortex) (hematoxylin-eosin, ×2.6). (From Kahn DS, Pritzker KP: The pathophysiology of bone infection. Clin Orthop 1973;96:12.)

PATHOGENS ASSOCIATED WITH AHO AND SEPTIC ARTHRITIS

An organism will not be identifiable in all cases of AHO or septic arthritis. However, if due diligence is taken to ensure that culture material is obtained from all possible sources before antibiotic therapy is initiated, an organism can be identified in approximately 70 to 80 percent of cases.[117,169,251,273,378] Most studies have shown a higher rate of positive cultures in AHO than in septic arthritis.[117,273] Placing a sample of synovial fluid in an "isolator" culture tube has been shown to increase the yield of culture results in septic arthritis.[386]

It is important to obtain blood for culture from all patients suspected of having osteoarticular infection, as well as to test with Gram stain all material cultured. Blood cultures have been reported to be positive in 40 percent of cases and the Gram stain in 30 percent. Often, these tests are the only laboratory studies that reveal the pathogen.[169,273,378] Mariani and associates recently described a processing protocol that uses polymerase chain reaction (PCR) technology to analyze synovial fluid for bacterial DNA.[224] This technique has had some early clinical success and may prove to be a valuable aid in identifying bacterial organisms in osteoarticular sepsis.[62,225] Because it specifically identifies bacterial DNA rather than an immune response to bacteria, PCR technology offers significant advantages over counterimmunoelectrophoresis in the diagnosis of osteoarticular infections.[93,353,362]

The pathogens involved in pediatric osteoarticular infections have traditionally been stratified by patient age (Table 34–3). *S. aureus* is by far the most common organism in all age groups.[94,117,169,231,273]

TABLE 34–3 **Organisms Associated with AHO and Septic Arthritis Based on Patient Age**

Age Group	Organism
Neonate	*Streptococcus*
	Gram-negative organisms
	Neisseria gonorrhoeae
Older infant and child	*Staphylococcus aureus*
	Hemophilus influenzae (decreasing or eliminated in areas with high vaccination rates)
	Kingella kingae
Children over 5 years	*Staphylococcus aureus*
	Salmonella (in patients with sickle cell anemia)
	Pseudomonas aeruginosa (secondary to puncture wounds)
Young adolescents	*Staphylococcus aureus*
	Neisseria gonorrhoeae (in those who are sexually active)

In the neonate, *Streptococcus* is the second most common pathogen. Neonates may also present with gram-negative or *Neisseria gonorrhoeae* infections. Polyostotic disease is seen in up to 50 percent of patients in this age group, and osteoarticular infections are often associated with such risk factors as indwelling umbilical catheters or the performance of invasive procedures.*

In the older infant and young child, *Hemophilus influenzae* has typically been a significant pathogen.[113,117,169,204,273] Awareness of this organism was once of particular importance because 20 percent of patients with *H. influenzae* osteoarticular infections were found to have meningitis.[204] With the availability of the conjugated *H. influenzae* vaccine, this bacterium has essentially disappeared as a pathogen in areas where vaccination rates are high.[2,50] Interestingly, as the incidence of *H. influenzae* osteoarticular infection has decreased, that of infection with *Kingella kingae* (another gram-negative rod) has been increasing.[197,216,385] *K. kingae,* a normal inhabitant of the nasopharynx, was first identified in 1976. Lacour and associates described findings in two cases and reviewed the literature on osteoarticular infections due to *K. kingae.*[197] They found a total of 51 cases of osteoarticular infections, of which 88 percent occurred in children less than 5 years old. All but four of the children had a normal immune system, and most of the cases followed a benign course. *K. kingae* has been reported as the etiologic pathogen in osteomyelitis, septic arthritis, concomitant osteomyelitis and septic arthritis, dactylitis, and diskitis.[197,385] The organism, which should be cultured in a CO_2-enriched medium,[72] is sensitive to all antibiotics, including penicillin and cephalosporins.[173]

In children older than 5 years, *S. aureus* remains the most common pathogen. However, there are a few unique circumstances in which other organisms are seen. Children with sickle cell anemia are predisposed to *Salmonella* infections,[137,341] although *Staphylococcus* is the most frequent infectious organism. *Pseudomonas aeruginosa* is commonly seen in puncture wounds to the foot, particularly when the puncture goes through a shoe.[107,113] Finally, septic arthritis may be caused by *N. gonorrhoeae* in young adolescents who are sexually active.[169]

CLINICAL PRESENTATION

History. The child who presents with an osteoarticular infection usually complains of bone pain and may be unable to move the affected extremity. If the infection involves a lower extremity, the child will often have a limp or will be unable to bear weight on the limb. Classically, the patient has a fever and general malaise, as well as a history of a recent upper respiratory infection or other infection that offers a clue as to the source of the bacteremia. However, a number of studies have shown that these classic constitutional signs of infection are often absent. In a study comprising 116 cases of AHO, Scott and associates found that only 30 percent of the patients had a history of recent bacteremia and only 36 percent had a temperature above 37.5°C.[317] Similarly, in a study comprising 44 cases of AHO and 25 cases of septic arthritis, Peltola and Vahvanen reported that only 24 percent of the patients had a temperature above 37.5°C.[273]

A history of antecedent trauma is quite common in children with bone or joint sepsis.[29,85,94,136,152,222] In fact, all too often the history of trauma results in a delay in the diagnosis and treatment of existing infection. Thus, it is extremely important to take a careful and meticulous history. Trauma rarely results in a delayed limp. The clinician should elicit specific details regarding any traumatic event. Was the injury witnessed? Did the child cry and limp or refuse to bear weight immediately after the trauma? If the answers to these questions are no and there was a period of time between the injury and the time symptoms appeared, the suspicion of infection should increase. It is easy to attribute musculoskeletal complaints to a child's most recent trivial fall event and overlook the possibility of infection.

Finally, assessment of the patient's general health is important in order to identify those children who may have a compromised immune system and therefore are at greater risk for infection, or children who may be at risk for musculoskeletal infection from specific pathogens (e.g., the child with sickle cell anemia).

Physical Examination. The physical examination of the child suspected of having an osteoarticular infection should start with a general assessment of the patient's overall health and then proceed to a general assessment of the musculoskeletal system. Only after these two steps have been completed should a more localized examination be conducted. Adherence to this approach will help the clinician avoid missing the forest for the trees, as it is not uncommon to overlook the presence of knee sepsis or a toddler's fracture in the "hunt" for hip sepsis. The general musculoskeletal assessment should include an observation of the patient's gait and a careful examination of the spine as the diagnosis of diskitis is classically delayed.[370] After these assessments, a careful examination of the suspected joint should be performed.

A thorough orthopaedic examination includes inspection and palpation of both hips, knees, tibiae, and feet, as well as the spine. Every effort should be made to keep the child quiet during the examination. Often this can be accomplished by examining the child with the child sitting on a parent's lap, and by starting the examination slowly and away from the area of greatest suspicion and apprehension (i.e., by examining the well leg first).

Palpating the bones and joints for tenderness and the presence of effusion helps to localize the area of inflammation. Range of motion of the joints also provides valuable information. If a joint is infected, the patient usually is unable to fully extend or flex the extremity. Pain may be present throughout the range of motion. In an examination of the spine or sacroiliac region, percussion and compression are more effective than palpation for evoking symptoms.

The classic posture of a septic hip is that of hip flexion, abduction, and external rotation. The child with a tense effusion of the hip joint holds the hip in this position of maximum capsular volume in an attempt to eliminate the pain associated with capsular distention. If the hip joint has a smaller, less painful effusion, the findings on examination are often much less obvious. In a less severe case, the careful examiner will usually find asymmetric abduction and internal rotation. Acute osteomyelitis of the proximal femur may

*See references 104, 117, 122, 123, 163, 167, 169, 273, 381.

cause a sympathetic effusion and manifest with findings similar to those of septic arthritis of the hip. The examination of specific joints for effusion or irritability is described in Chapter 3, The Orthopaedic Examination.

Neonatal and Infantile Osteoarticular Infection. Diagnosing neonatal or infantile osteoarticular infection is particularly difficult because there are usually few subjective or objective findings in the history or examination.[29,75,264] In general, AHO or septic arthritis develops in infants in two distinctly different groups.[249] The first is the sick, premature neonate who is hospitalized and may have obvious risk factors for infection, such as indwelling catheters.[167] The second is the neonate who is not systemically ill and has been discharged from the hospital, is developing and feeding normally, and may only exhibit irritability or fever. In both groups the examination is often unremarkable, although occasionally there will be soft tissue swelling or "pseudoparalysis" of an affected extremity. Some authors have reported multifocal involvement in more than half of their neonates.* In many cases these multiple sites of infection are not apparent at presentation. Thus, the clinician must maintain a high degree of suspicion for multifocal infection when evaluating and managing infants with osteoarticular sepsis. In fact, because the physical findings during the acute illness are so unremarkable and the potential complications of untreated or undertreated hip sepsis are so devastating, Morrissy recommends routine aspiration of both hips in all infants known to have osteomyelitis or septic arthritis.[249]

LABORATORY STUDIES

If osteoarticular infection appears likely, laboratory tests and plain radiography are usually the first diagnostic studies performed. Appropriate laboratory studies include a complete blood cell (CBC) count with differential, an erythrocyte sedimentation rate (ESR), and a C-reactive protein (CRP) concentration. Blood cultures should also be performed.

When musculoskeletal sepsis is present, the white blood cell (WBC) count often is elevated and the differential may show a predominance of polymorphonuclear cells (left shift). A "bandemia," or increase in immature cells, may also be present.[117,169,272,273] However, newborns and infants, as well as immunocompromised patients, may have only a slight elevation in WBC count. A number of reports indicate how frequently the WBC count is normal in patients with osteoarticular infection, with elevated counts found in only 21 percent to 43 percent.[317,356,378] The clinician should also review the hemoglobin and hematocrit values, platelet count, and peripheral smear to ensure that the patient has normal bone marrow function, since leukemia must be considered in the differential diagnosis of a limping child.[57,179,358]

Traditionally, the Erythrocyte Sedimentation Rate (ESR) has been used to monitor the clinical course of patients with osteoarticular sepsis.[67,184,195,272,317,356] The ESR is the rate at which erythrocytes sediment and therefore depends on several physical factors including the size and shape of red cells, plasma concentrations, hormone levels, and the nutritional status of the patient.[1a] More recently, the C-reactive protein (CRP) concentration has been shown to be more a more sensitive index of infection.[195,301,356,357] CRP is an acute phase protein that is hepatic in origin. The plasma concentration of CRP can increase several hundred–fold within 24 to 48 hours of the onset of infection or soft tissue injury. Because the CRP clearance rate is constant, the plasma CRP level is dependent solely on synthesis. Additionally, determination of CRP levels is a direct measurement of a specific molecular quantity and is subsequently independent of any physical circumstances.[1a] In a study of 44 children with AHO, Unkila-Kallio and associates reported an elevated ESR in 92 percent and an elevation in CRP in 98 percent. The ESR returned to normal in approximately 3 weeks (mean, 18 days), whereas CRP levels normalized within 1 week (mean, 6.9 days).[356]

Although laboratory studies play an important role in the diagnostic evaluation of suspected osteoarticular infection, laboratory values can be normal in patients with osteoarticular sepsis. Thus, normal values by themselves do not rule out the presence of AHO or septic arthritis.

IMAGING STUDIES

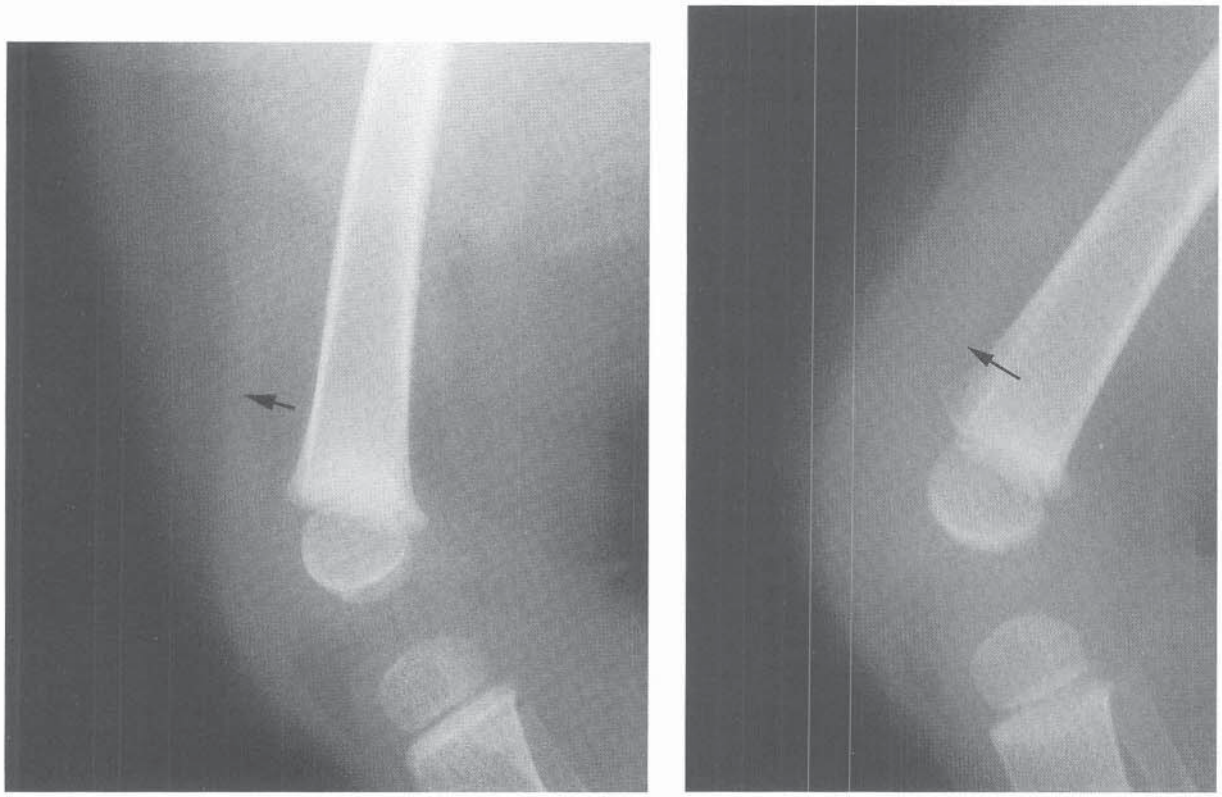
Plain Radiography. Plain radiographs are an essential part of the early diagnostic evaluation of the child believed to have an orthopaedic infection. The initial radiographs must be complete and of good quality. If the suspected area of anatomic focus is easily localized on clinical examination, the minimal study is two orthogonal views of the area. However, when it is difficult to localize the area of concern clinically, radiographs of the entire extremity may be necessary.

Diskitis, toddler's fractures, and subtle injuries to the feet, in addition to osteoarticular infection, must all be considered in the differential diagnosis of a limping child. Frequently these conditions can be diagnosed with plain radiographs without the need for more invasive and costly studies.* Radiographs of the knee must include a true lateral view, as the presence of an effusion may be difficult to ascertain on an oblique view (Fig. 34–5). The presence of an aggressive lytic lesion may suggest the possibility of neoplasm (Fig. 34–6). If so, further investigation with magnetic resonance imaging (MRI) may be indicated, and a biopsy may be necessary to establish the diagnosis.

Occasionally the findings on plain radiographs are dramatic and, in combination with clinical findings, may be diagnostic. If an infant or young child has an irritable hip and radiographs show a subluxed joint, the presence of infection must be assumed until proven otherwise (Fig. 34–7). In most cases, though, initial radiographs obtained in children with osteoarticular infections are normal or unremarkable because bony changes caused by osteomyelitis typically do not appear on plain radiographs for 7 to 14 days.[352] Most commonly, the initial radiographic findings associated with osteoarticular infection are soft tissue swelling or subtle lucencies within the metaphysis. If the infection is untreated, the complete radiographic appearance of chronic osteomyelitis will develop. These changes include subperiosteal new bone formation and dense sequestered dead bone (Fig. 34–8).

*See references 34, 104, 122, 167, 267, 275, 323.

*See references 20, 41, 45, 177, 269, 298, 322, 324, 333, 370.



A B

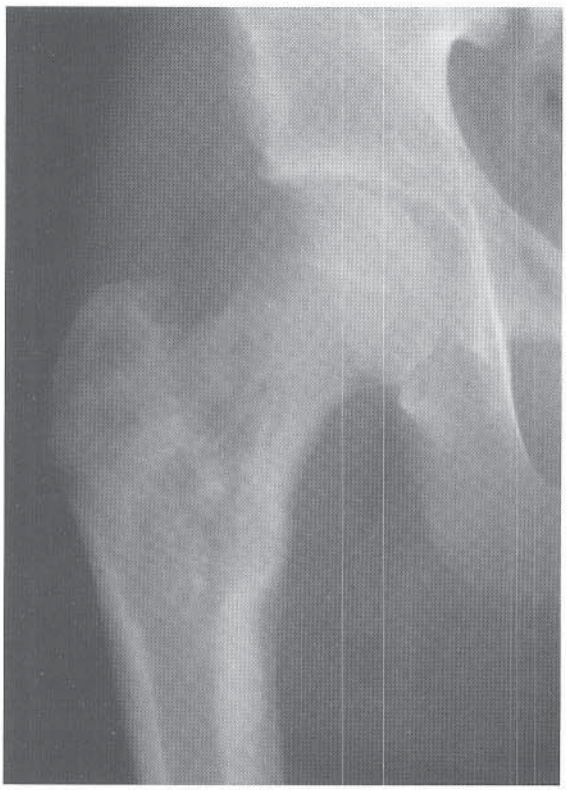
FIGURE 34–5 **A,** Lateral radiograph in normal 9-month-old child. Note the fat density along the anterior metaphysis of the distal femur. The soft tissue density between this fat pad and the subcutaneous fat represents the quadriceps tendon (*arrow*). **B,** Lateral radiograph in a 9-month-old child with a knee effusion. The increased soft tissue density between the suprapatellar fat pad and the subcutaneous fat represents an effusion (*arrow*).

Radionuclide Scintigraphy. When the clinical examination and laboratory findings support the diagnosis of bone or joint sepsis but the clinician is unable to localize the source of infection, radionuclide scintigraphy will often pinpoint the locus of infection.[20,49,229,344,352,355] The most commonly used radiopharmaceutical agent today is technetium 99m diphosphonate, although gallium 67 citrate and indium 111 are occasionally used.[201,208]

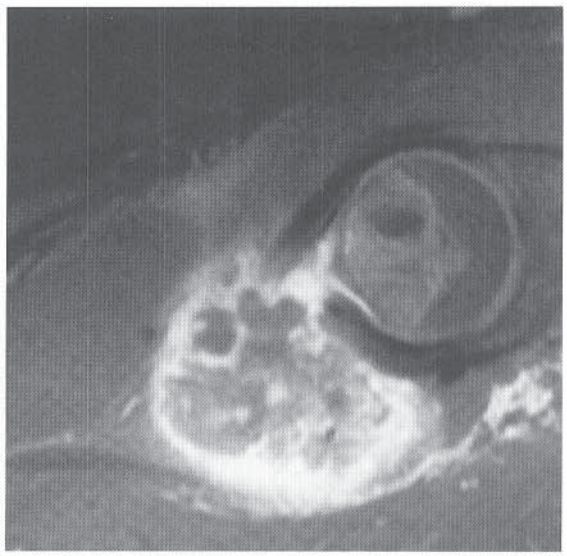
The value of bone scans has been demonstrated in a number of studies. Aronson and associates used scintigraphy to evaluate 50 consecutive children who presented with a limp whose exact cause could not be ascertained by an orthopaedist.[20] A bone scan was essential in localizing the lesion in 27 patients (54 percent). There were two false negative studies and one false positive, and no cases of infection were missed. In the presence of infection, a bone scan may show either increased uptake ("hot" scan) or decreased uptake ("cold" scan). Areas of decreased uptake may indicate an abscess. In a prospective study conducted in 86 children, Tuson and associates assessed the value and accuracy of bone scintigraphy in the diagnosis of suspected AHO and septic arthritis.[355] In AHO there was increased or decreased uptake of radionuclide extending beyond the confines of the joint capsule, whereas in septic arthritis, uptake was largely limited to and uniform within the joint capsule. The overall accuracy of bone scintigraphy was 81 percent. The positive predictive value was 100 percent for a cold scan and 82 percent for a hot scan. The negative predictive value was 63 percent. The findings from this study emphasize the fact that bone scans can be normal in patients with osteoarticular infection.

The efficacy of bone radionuclide scintigraphy in the diagnosis of infection in the neonate has been the topic of considerable debate. In 1980, Ash and Gilday reported that bone scans identified only 32 percent of infected loci in 10 neonates with multifocal osteomyelitis.[21] Subsequent studies, however, have reported sensitivities between 90 and 100 percent in the detection of focal skeletal involvement in neonates with osteomyelitis and septic arthritis.[6,54] These improved results may be attributable to technological improvements, such as obtaining "pinhole" images or areas of high suspicion. Despite these encouraging reports, it is important to remember that infants with osteoarticular sepsis are particularly likely to have false negative studies.

Computed Tomography. Although computed tomography (CT) is excellent for defining bony pathology, CT has limited application in the diagnosis or management of osteoarticular infections because the bony changes associated with AHO occur late in the course of the disease and usually are easily seen on plain films. However, CT may be helpful in demonstrating soft tissue abscess of the spine or pelvis.[143] In addition, CT-guided percutaneous biopsy often is the preferred method of obtaining tissue from axial locations for evaluation (Fig. 34–9).[3,145,158,230]



A



B

FIGURE 34–6 **A,** AP radiograph showing a lytic lesion in the proximal femur. The patient had a 2-month history of pain and an elevated ESR. The permeative nature of the lesion suggested a neoplastic rather than an infectious etiology. **B,** Axial MR image of the proximal femur demonstrating a large soft tissue mass posteriorly. Biopsy confirmed the diagnosis of Ewing's sarcoma.

Magnetic Resonance Imaging. Advancements in MRI technology have greatly improved the ability to delineate soft tissue pathology. This is often extremely beneficial when managing patients with osteoarticular infections, particularly patients who have received partial treatment, those whose lesions involve the axial skeleton, and those in whom plain radiographs suggest the possibility of neoplasm (see Fig. 34–6).[35,110,146,327]

Because normal fatty tissue is replaced by inflammation and edema in cases of acute osteomyelitis, there will be a decrease in signal intensity on T1-weighted images and an increased signal intensity on T2-weighted images.[35,327] MRI can also demonstrate fluid collections associated with abscesses (Fig. 34–10). A number of authors have used the fact that the MRI findings of diskitis are the same as those of osteomyelitis to support the argument that diskitis is a bacterial infection (Fig. 34–11).[237,298,327]

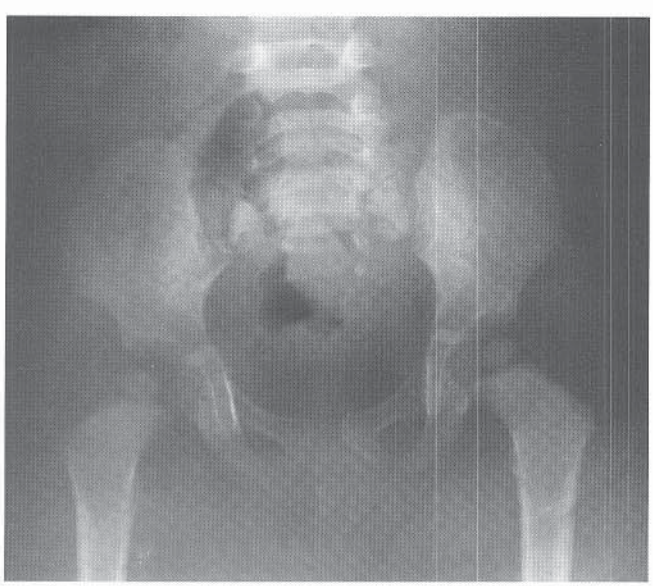


FIGURE 34–7 AP radiograph demonstrating subluxation of the right hip secondary to septic arthritis.

Ultrasonography. Ultrasonography is another noninvasive imaging modality that has been used to evaluate children suspected of having osteoarticular infections.* Ultrasound is most useful in demonstrating a fluid collection, whether the effusion is in the hip joint or in a subperiosteal location (Fig. 34–12). Although ultrasound does not show the cause of an effusion, it can serve as an aid when performing aspiration.† Attempts have been made to correlate sonographic features with the likelihood of infection. Shiv and associates reported both hypoechoic and hyperechoic synovial fluid in septic arthritis, with hyperechoicity and thickening of the articular capsule the most characteristic findings.[321] Dorr and associates noted that synovial hypertrophy and a thickened capsule distinguished septic arthritis from transient synovitis.[101] Normal or unremarkable ultrasound findings do not rule out infection, as AHO often exists without subperiosteal fluid collection.

*See references 10, 101, 105, 164, 183, 220, 254, 321, 389.
†See references 10, 101, 183, 220, 254, 321, 389.

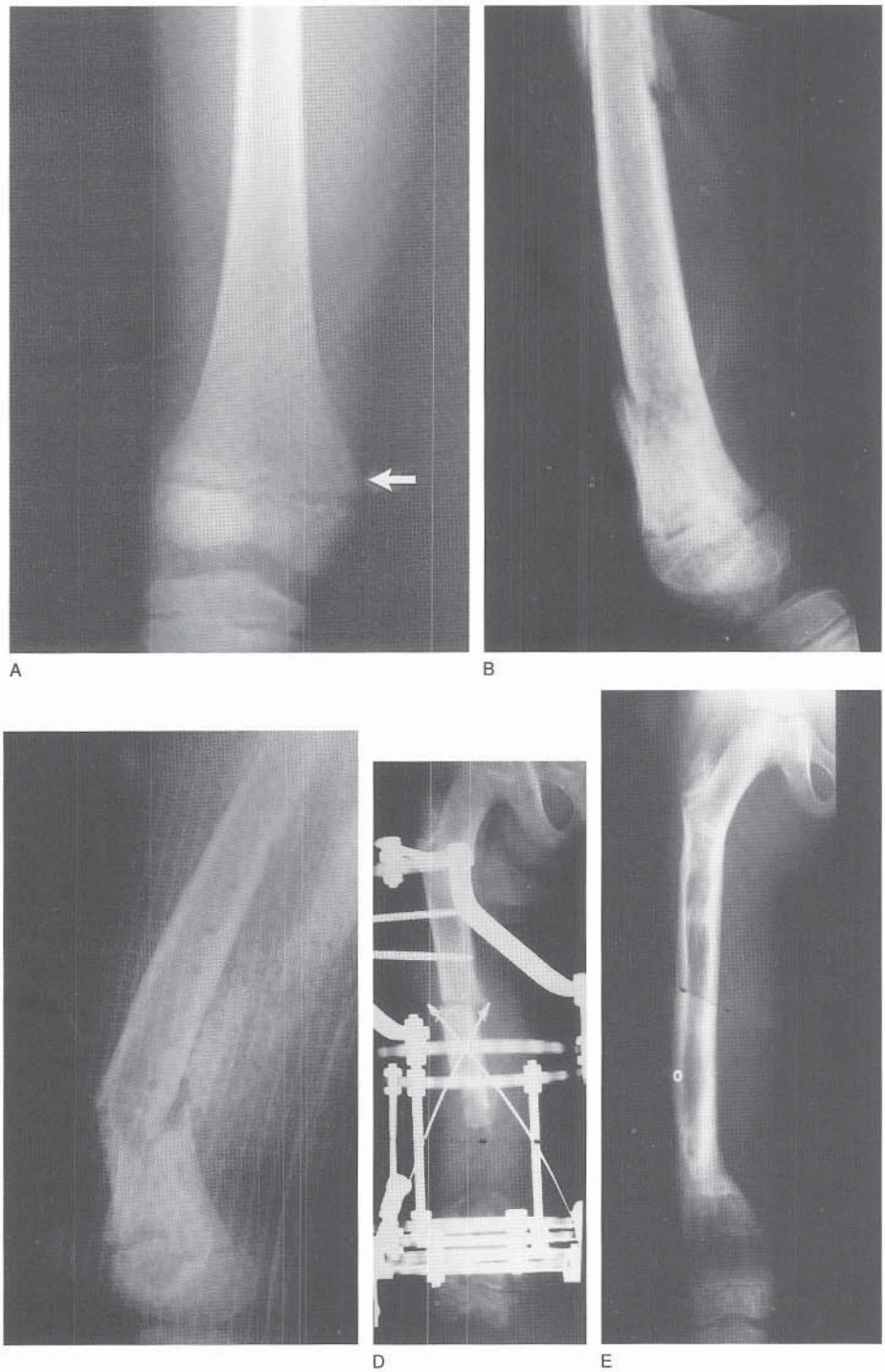
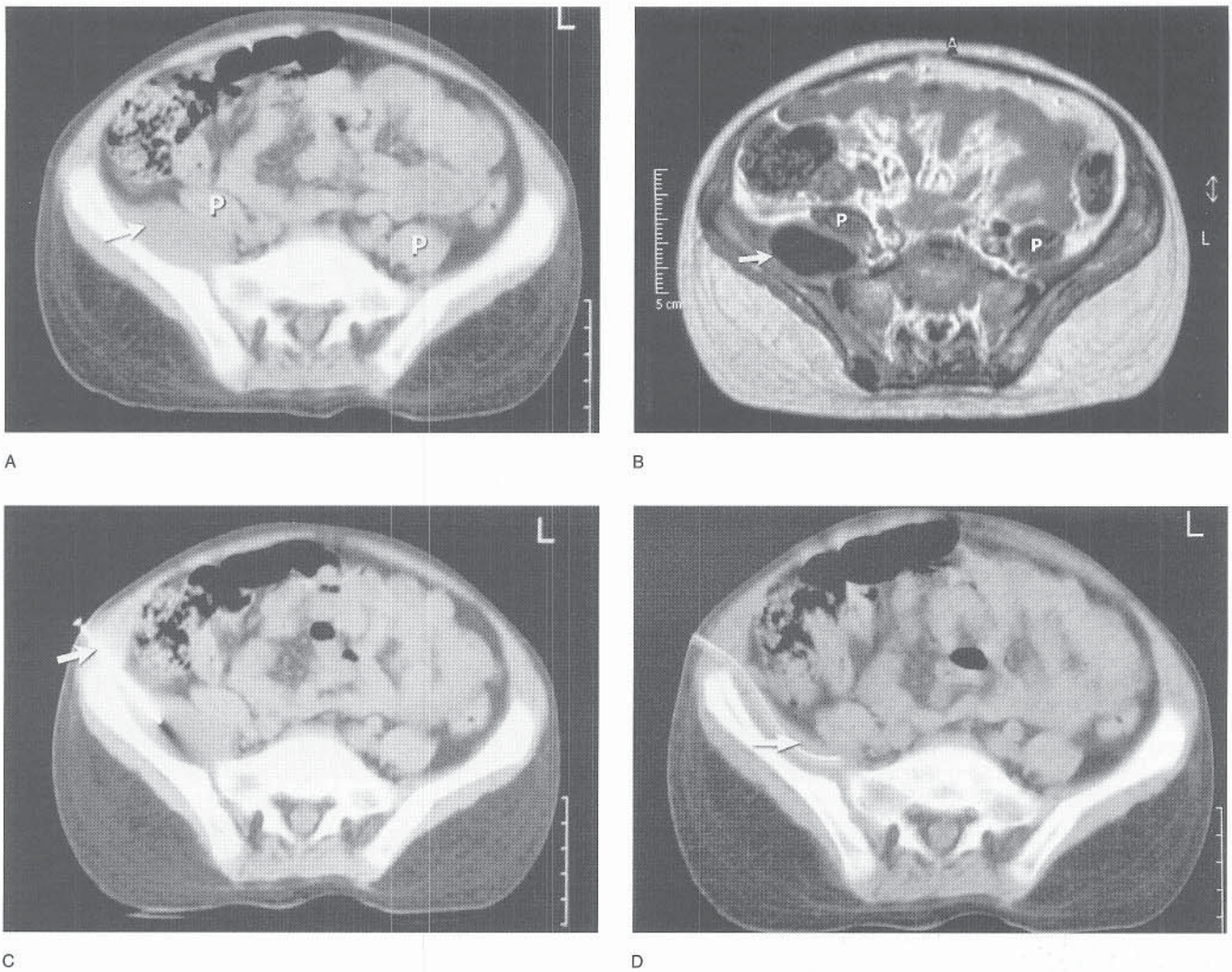


FIGURE 34–8 Progression of acute to chronic osteomyelitis. **A,** AP radiograph of the femur shows subtle lucency in the medial distal femoral metaphysis (*arrow*). **B,** Lateral radiograph of the same patient, untreated, 6 weeks later. Note the periosteal reaction as well as the progression of the radiolucency in the metaphysis. **C,** AP and lateral radiographs obtained after an attempt at surgical decompression without debridement. Note the pathologic fracture at the metaphyseal-diaphyseal junction. The drill holes visible in the lateral view are residua of an attempt to decompress the infection. Adequate debridement of necrotic tissue was not achieved. **D,** AP radiograph obtained after complete debridement and application of an Ilizarov frame for bone transport. **E,** AP radiograph obtained after a successful bone transport.



A B C D

FIGURE 34–9 CT scan (**A**) and MR image (**B**) demonstrating a psoas abscess (*arrow*) displacing the right psoas muscle (P). CT-guided percutaneous drainage (**C**) with placement of a drainage tube (**D**).

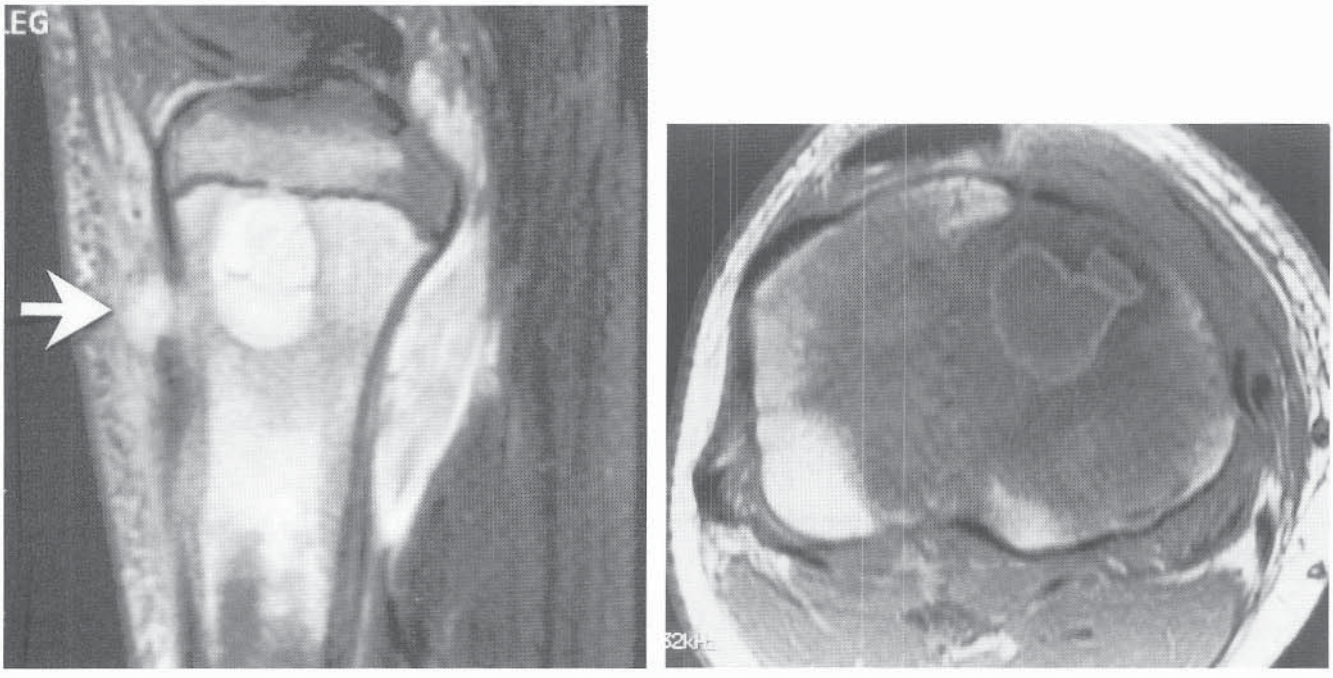
DIFFERENTIAL DIAGNOSIS

The differential diagnosis of AHO or septic arthritis is diverse and includes all of the entities discussed in Chapter 6, The Limping Child. Most often the focus is on differentiating osteomyelitis from septic arthritis and toxic or transient synovitis. Other important causes of a limp are diskitis, toddler's fractures, and subtle foot pathology.* Although leukemia[118,151,179,358] and inflammatory arthritis[38,115,128,243,244,315] are not as common, it is important to include both conditions in the differential diagnosis of a limp.

Distinguishing osteomyelitis from septic arthritis can be quite straightforward in some patients and nearly impossible in other cases. It is not uncommon for the two conditions to occur concomitantly. A bone scan is often helpful in making the distinction. Cultures of aspirated material may also help determine the particular pathology. However, in some cases the clinician will find it impossible to localize the infection specifically to bone or joint. Fortunately, it is possible to successfully treat patients with osteomyelitis or septic arthritis without knowing the exact location of the infection.

Making a clinical distinction between toxic synovitis, septic arthritis of the hip, and osteomyelitis of the proximal femur is also frequently quite difficult. In each entity the clinical signs may range from a mild limp with an irritable hip to refusal to bear weight or walk. Classically, children with toxic synovitis have milder signs and symptoms that often improve rapidly with bedrest. Patients with true sepsis more often present with the hip in a flexed, abducted position. Laboratory results can be unremarkable to alarmingly abnormal in all three conditions. Findings on imaging studies are often equally frustrating when evaluating an irritable hip. Bone scintigraphy and ultrasound are quite helpful in establishing the presence of an effusion, but both studies are of questionable value in identifying the cause of the effusion.[10,49,101,321] Making the diagnosis often requires aspiration of synovial fluid for appropriate culture and analysis. The decision to proceed with aspiration of the hip must be made based on the pertinent clinical signs and symptoms and on the expertise of the treating physician. A number of authors have stressed the relative benefit of "overtreating"

*See references 20, 41, 45, 177, 269, 298, 322, 324, 333, 370.



A B

FIGURE 34–10 MR image demonstrating intraosseous abscess. **A,** Sagittal image of a metaphyseal lesion abutting the physis. There is also extrusion into the anterior pretibial soft tissues (*arrow*). **B,** Axial view demonstrating gadolinium enhancement surrounding the rim of the lesion. The abscess is nonenhancing.

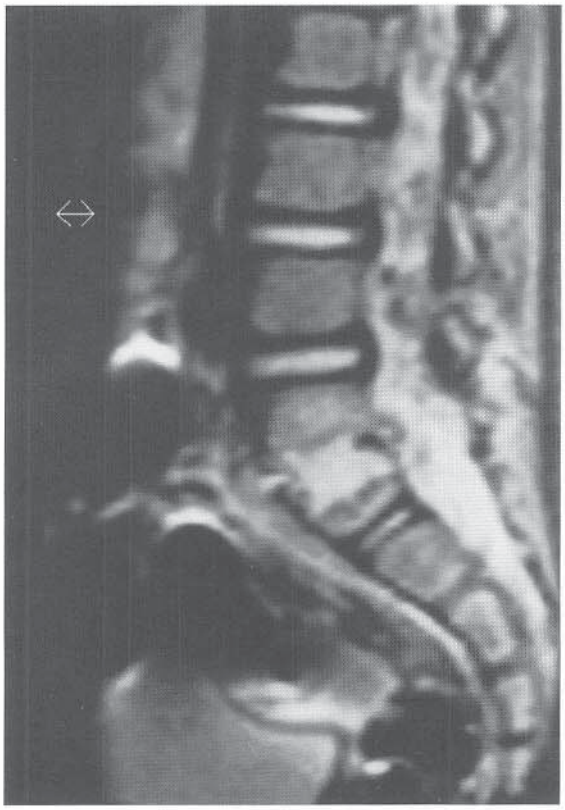


FIGURE 34–11 MRI of L5–S1 diskitis. Note the increased signal intensity of the entire disk space as well as both end-plates.

transient synovitis in order to avoid a missed or delay in treatment of septic arthritis of the hip.[259,270,369]

Diskitis, toddler's fractures, and subtle foot pathology can all cause limping in a child.* When these conditions are included in the differential diagnosis, they usually can be accurately established or ruled out without delay. However, if they are not part of the initial differential diagnosis, an appropriate examination will not be performed and necessary radiographs will not be obtained, resulting in possible delay in making the correct diagnosis and an increase in the number of unnecessary tests performed for other conditions.

Leukemia, the most common malignancy of childhood, often manifests with bone pain as the initial symptom.[118,151,179,358] In a review of 296 patients with acute lymphoblastic leukemia (ALL), Jonsson and associates reported that 22 percent had some bone pain and 18 percent presented with bone pain as the predominant symptom.[179] Patients with prominent bone pain were more likely to have normal or near normal hematologic values, a feature the authors believed may have contributed to a delay in the diagnosis of ALL. These findings emphasize the importance of a careful and complete evaluation of every patient who presents with musculoskeletal pain. Every CBC count should include a differential; however, because the differential may appear normal early in the course of the disease, the entire CBC count needs to be carefully assessed. Patients with early leukemia may show subtle signs of myelosuppression or dysfunction (e.g., anemia, thrombocytopenia) before blast cells are seen in the peripheral smear. Today, many differentials are analyzed with automated equipment, which is

*See references 20, 41, 45, 177, 269, 298, 322, 324, 333, 370.

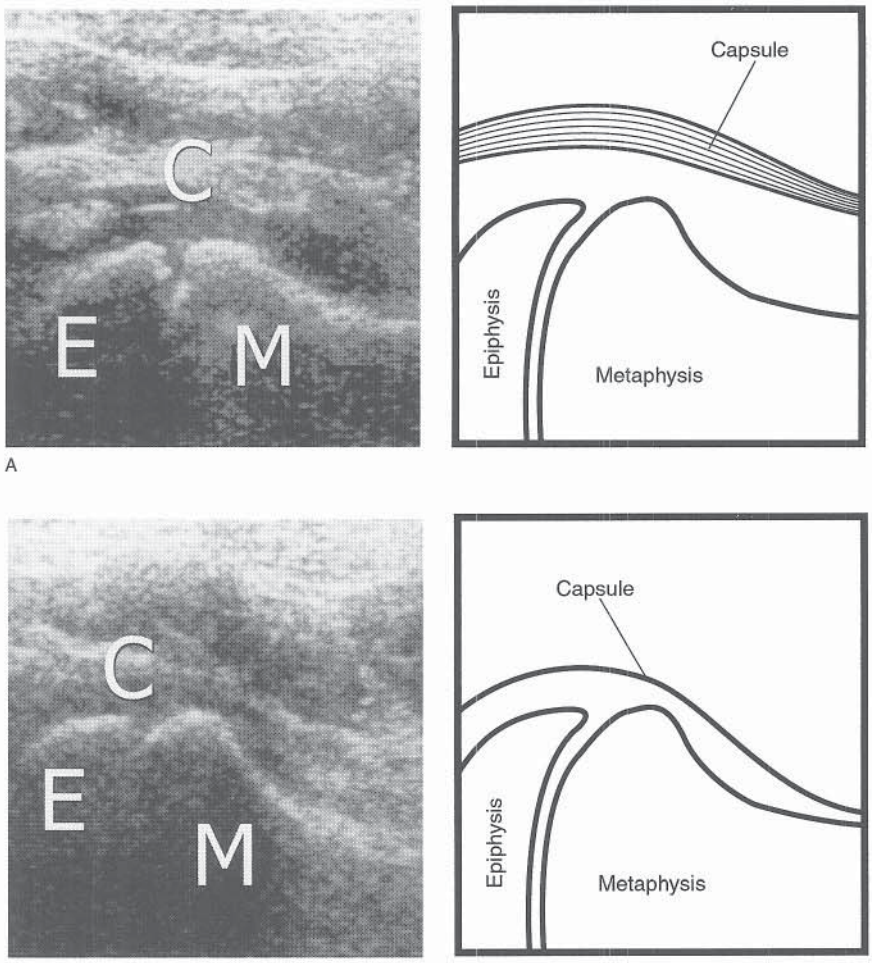


FIGURE 34–12 Sonograms with line drawing of a patient with septic arthritis of the hip (*E,* epiphysis; *M,* metaphysis; *C,* capsule). **A,** Intra-articular fluid displaces the capsule into a convex position. The capsule is also thickened. **B,** The capsule on the normal side is concave (following the contour of the metaphysis) and not thickened.

more likely to miss early or subtle signs of marrow dysfunction than is a manual review of a peripheral smear.[25,343] Thus, if there is a high degree of suspicion of leukemia, the orthopaedic surgeon should request a manual review of a peripheral smear or consult a pediatric hematologist-oncologist.

It may be difficult to distinguish inflammatory arthritis from AHO or septic arthritis. In fact, AHO may produce a reactive or "sympathetic" effusion in an adjacent joint. Postinfective arthritis is another reactive inflammatory arthritis that may be seen after systemic bacterial or viral infection.[38,128,243,244,315] Inflammatory arthritis also exists as a primary disease process, best known as juvenile arthritis.[115,116] Classically, juvenile arthritis manifests with a painless limp. Symptoms usually are worse early in the day and improve as the day progresses. Distal joints, such as the hands and feet, are commonly involved, whereas "central" joints, such as the hip, are rarely symptomatic on initial presentation. If the patient has an obvious joint effusion and the history and laboratory tests suggest an infection, the joint should be aspirated and the synovial fluid cultured and analyzed to make a definitive diagnosis. The etiology, diagnosis, and prognosis of the various inflammatory arthritides are discussed in greater detail in Chapter 33, Arthritis.

TREATMENT

Aspiration. The first goal in treating AHO or septic arthritis is to confirm the diagnosis and, if possible, identify the specific pathogen involved. Needle aspiration of all possible loci is the most expeditious method of establishing the diagnosis. Any fluid obtained by aspiration should be sent to the laboratory for Gram stain and appropriate cultures. Placing a small amount of synovial fluid in an "isolator" culture tube may increase the chances of identifying the organism.[386] Synovial fluid should also be sent to the laboratory for a count of nucleated cells.

When aspirating bones or joints, it is important to use careful sterile technique. The procedure can often be performed in an emergency room setting using local anesthetic or by sedating the patient. Aspirating a metaphysis that may be involved in AHO is a two-part process. First, the needle is advanced into the subperiosteal space and aspiration is performed. If no pus is encountered, the needle is then advanced through the soft metaphyseal cortex and the metaphyseal bone is aspirated. If no pus is withdrawn, a few milliliters of blood can be aspirated and sent for culture.

The timing and even the necessity of aspirating bone or

joints have received considerable attention. In the past there was some concern that needle aspiration of bones or joints might cause false positive results on ensuing bone scans. However, a number of clinical and animal studies have demonstrated that aspiration (even with arthrography with contrast material) does not adversely affect the outcome of subsequent bone scans.[7,61,229,350] With the increased availability of highly sophisticated imaging studies such as bone scintigraphy and MRI, some authors have questioned the need for needle aspiration in every case of AHO. However, because blood cultures are positive in only 40 percent of cases,[169,262,273] it is much less likely that a specific organism will be identified without aspiration. Although *S. aureus* remains the most common pathogen in AHO and septic arthritis, many cases are caused by other organisms. In addition, a number of recent reports have noted an increase in the frequency of community-acquired methicillin-resistant *S. aureus* (MRSA).[203,245,338] Failure to correctly identify these pathogens with cultures can make treatment more difficult and can adversely affect the outcome.[347] Thus, we continue to advocate the expeditious aspiration of any suspected foci of osteoarticular infection.

Interpreting the synovial fluid nucleated cell count is often difficult. There is no absolute value that is pathognomonic for septic arthritis. The cell count and, if possible, its differential must be interpreted in light of clinical findings. Traditionally, nucleated cell counts in excess of 50,000/mm^3 have been considered representative of infection.[24] In a review of 129 synovial fluid analyses, Kunnamo and Pelkonen found that a nucleated cell count greater than 40,000/mm^3 was greater than 90 percent sensitive and specific in differentiating septic arthritis from other causes of inflammatory arthritis.[196] They also noted that patients with juvenile arthritis were more likely to have mononuclear cells in the synovial fluid. A subsequent study by the same authors demonstrated the importance of the entire clinical picture. A review of 278 children with arthritis showed that the presence of either an elevated CRP level or a temperature above 38.5°C had a sensitivity of 100 percent and a specificity of 87 percent for septic arthritis.[195] Not all patients with synovial fluid nucleated cell counts greater than 50,000/mm^3 will have septic arthritis. Baldassare and associates reported findings in six patients with juvenile arthritis who had synovial fluid nucleated cell counts greater than 88,000/mm^3, three of whom had counts greater than 100,000/mm^3.[24] These studies highlight the difficulties that may be encountered in diagnosing septic arthritis, particularly in light of the fact that cultures may be negative in 30 to 60 percent of cases.[247,273]

Histologic evaluation of aspirated material may help in the diagnosis. In 30 patients in whom osteomyelitis was suspected, Howard and associates reported that fine-needle bone biopsy had a sensitivity of 87 percent and a specificity of 93 percent for osteomyelitis.[161] In a study of 25 patients, White and associates reported that culture of aspirated material alone had only a 42 percent sensitivity for osteomyelitis, whereas the combination of culture and histologic findings had a sensitivity of 84 percent.[372] Obtaining a histologic evaluation of any tissue thought to harbor infection is a prudent step that can expedite making an accurate diagnosis, particularly when there is suspicion of unusual pathogens or neoplasm.[170]

Surgery—Septic Arthritis. The role of surgery in the treatment of septic arthritis is poorly defined and somewhat controversial. While few reports mention successful medical management of septic arthritis of the hip,[67,112,247,378] *prompt surgical drainage of hip sepsis* remains one of the absolutes in the management of children with osteoarticular infections.* Good results have been reported with aspiration, irrigation, and IV antibiotic therapy, without surgical decompression or synovectomy as well as with arthroscopic drainage of septic arthritis of the hip.† However, we still prefer to perform a formal open capsulotomy to allow continued drainage of septic material.

The surgeon can approach the hip anteriorly, posteriorly, or medially. We prefer an anterior approach because it can be performed through a cosmetic incision, results in minimal blood loss, and most importantly does not jeopardize the circumflex femoral vessels, which provide the blood supply to the proximal femoral physis. In addition, the anterior approach allows the surgeon to perform a capsulorrhaphy and a psoas and adductor release to treat a subluxated or dislocated femoral head.[369] Fortunately, it is rare today for hip sepsis to progress to the point of subluxation or dislocation. In a retrospective review of 94 septic hips, Rosenblatt and Bennett reported that patients younger than 3 years had a statistically significant lower rate of complications when treated through an anterior approach.[304] In 22 of 33 hips drained through a posterior approach, complications included joint narrowing, coxa magna, acetabular dysplasia, and osteonecrosis, whereas no complications occurred in 16 hips drained through an anterior approach. The anterior approach for drainage of the hip is illustrated in Plate 34–1.

The indications for surgical drainage of septic joints other than the hip are less well defined. Many authors agree that an initial trial of aspiration, irrigation, and IV antibiotic therapy is reasonable for patients with "early" septic arthritis.‡ In these series, "early" is generally defined as cases in which symptoms have been present for less than 4 days. Surgery is reserved for patients who have had symptoms for more than 4 days at presentation and for patients who fail to improve with aspiration, irrigation, and IV antibiotics. Although all of these studies have documented the effectiveness of this treatment method, other authors still advocate immediate surgical drainage of all septic joints.[270,363,364] This opinion is supported by animal studies in which rabbits treated with synovectomy 3 days after infection was established had less cartilaginous destruction than rabbits treated by antibiotics alone.[294,295] However, when synovectomy was not performed until 7 days after infection had been induced, the macroscopic and microscopic characteristics of the cartilage were similar to those of the cartilage in animals treated with antibiotics alone.

In addition to controversy over surgical indications, there also is some debate regarding the technique of surgical drainage. Recent reports have discussed arthroscopic drainage of knees, shoulders, and hips in patients with septic arthritis.[40,48,70,175,325,346] The reported advantages of arthroscopic treatment include complete joint visualization and less soft tissue dissection, which may result in decreased postopera-

Text continued on page 1858

*See references 31, 36, 117, 156, 162, 165, 242, 270, 276, 363, 364, 369.
†See references 40, 48, 70, 117, 156, 164, 247, 248, 272, 276, 317, 342, 356.
‡See references 85, 117, 156, 169, 213, 247, 251, 257, 262, 273, 342, 378.

Surgical Technique for Anterior Approach to the Hip for Drainage of Septic Arthritis

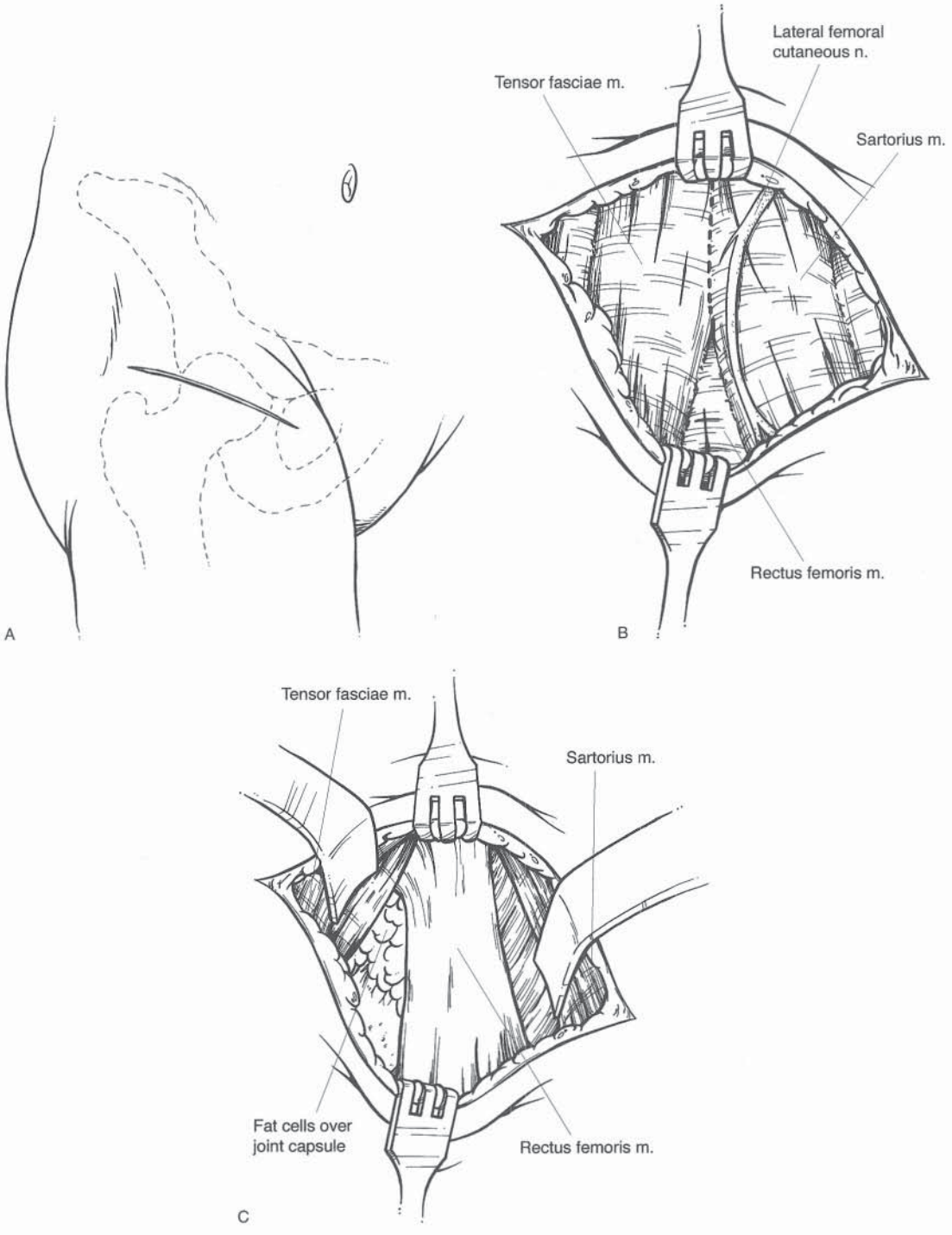
OPERATIVE TECHNIQUE

A, The patient is placed supine with a roll under the back. The entire extremity is prepared and draped. A 4- to 6-cm skin incision is made within Langer's lines, centered on and 2 cm distal to the anterior superior iliac crest. The incision is carried down to the fascia. The subcutaneous tissues can be mobilized to "functionally lengthen" the incision.

B, The interval between the tensor fascia lata and sartorius is identified and opened. The lateral femoral cutaneous nerve can be identified.

C, Blunt retractors are used to develop the plane between the tensor and sartorius. On the floor of this space will be the rectus tendon. Blunt retraction is used to mobilize the rectus either medially or laterally, exposing the hip joint capsule.

PLATE 34–1. Surgical Technique for Anterior Approach to the Hip for Drainage of Septic Arthritis

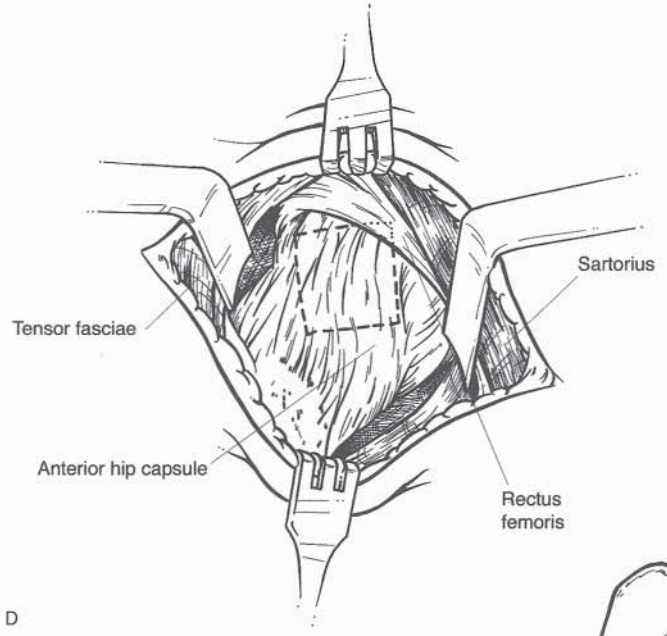


Surgical Technique for Anterior Approach to the Hip for Drainage of Septic Arthritis *Continued*

D, A 1- to 2-cm square capsulectomy is then performed. The cuts are made through the full thickness of the capsule. This can be difficult, as the underlying synovium is hypertrophied.

E, Once the capsulectomy has been performed, the articular surface of the femoral head should be inspected. The joint is copiously irrigated with sterile saline. Suction drainage is then placed within the capsule inferior to the neck and brought out through the skin. The subcutaneous tissue and skin are closed completely with absorbable sutures. Spica cast immobilization should be considered in the infant if the hip is unstable.

PLATE 34–1. Surgical Technique for Anterior Approach to the Hip for Drainage of Septic Arthritis



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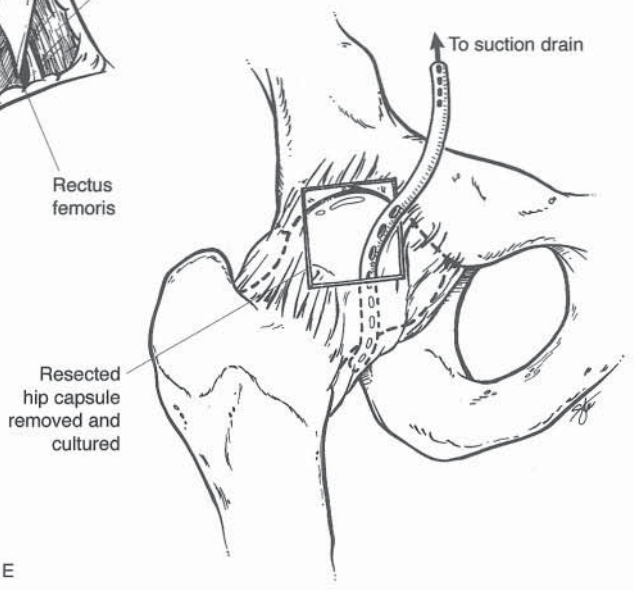


E

tive pain and a quicker return of range of motion. The method and even the necessity of postoperative drainage after arthrotomy or arthroscopy have also been debated. Both proponents of no drainage[253,270] and those who favor the use of a closed irrigation and drainage system[78,142,325] have reported good results. However, in a critical review, Letts and Wong reported a 60 percent complication rate and a 30 percent superinfection rate (most commonly from *Pseudomonas*) with an irrigation-drainage system.[206] In addition, patients treated with irrigation and drainage systems averaged 30 days in the hospital, compared to 16 days for patients treated by simple incision and drainage alone.

For joints other than the hip, we believe that aspiration, irrigation, and IV antibiotic therapy is a reasonable first line of treatment. If the patient fails to respond to these treatments within 48 to 72 hours, we believe the joint should be surgically drained. We also recommend surgical drainage for patients who have had prolonged symptoms, patients with radiographic evidence of osteomyelitis in the metaphysis adjacent to the affected joint, and patients from whom thick, purulent material is obtained on initial aspiration of the joint. Generally we prefer a formal open arthrotomy rather than arthroscopic drainage because the arthrotomy is quicker to perform and places fewer demands on operating room personnel; however, we realize that in some settings, there may be advantages to an arthroscopic approach. We prefer routine skin closure with either no drain or a simple suction drain rather than using an irrigation-drainage system. We strongly agree with those authors who contend that there is less harm in the occasional "unnecessary" arthrotomy than in the undertreatment of septic arthritis.[251,270,369]

Surgery—Acute Hematogenous Osteomyelitis. As in septic arthritis, the role of surgery in AHO is poorly defined. A number of authors have suggested that surgery is not required in all cases of AHO.* In a review of 77 patients, Cole and associates defined three groups of patients with AHO.[75] The first group consisted of patients older than 1 year who presented within 48 hours of the onset of illness. These patients did well with antibiotic therapy alone. The second group comprised patients more than 1 year old who presented 5 or more days after the onset of illness with severe osteomyelitis and an abscess. These patients generally underwent multiple operations and prolonged antibiotic therapy. In the final group of patients were infants less than 1 year old in whom the exact diagnosis was difficult to make, and who generally required a single operation and antibiotic therapy.

The classification by Cole and associates serves as an excellent general guideline for our preferred treatment approach to patients with AHO. We believe that in every patient suspected of having AHO, all suspected loci should be aspirated and blood cultures should be done before antibiotics are administered. Patients who have no radiographic evidence of bone destruction and no pus on aspiration can be initially managed with IV antibiotics. Patients who have substantial radiographic bony destruction or in whom pus is aspirated require surgical debridement followed by IV antibiotics. Regardless of the initial treatment, any patient who fails to improve within 48 to 72 hours should undergo surgical (or repeated surgical) debridement.

For children less than 1 year old, we have a lower threshold for performing surgical decompression, particularly when the infection is in the proximal femur, where damage to the common blood supply of the metaphysis and epiphysis can have devastating consequences (see Fig. 34–1). In addition, because of the predilection for multifocal disease in neonates and infants and the difficulty of examining these patients clinically, we often obtain a bone scan early in the course of treatment to help ensure an accurate and complete diagnosis.

The management of patients who have early radiographic findings is controversial. Although the conventional tenet is that the area of radiographic abnormality may contain pus that requires surgical decompression, there are numerous reports of good results when these patients were treated nonsurgically.[75,136,265,272,317] We believe that each case must be managed on an individual basis and that the only absolute maxim when treating patients with AHO is that they must be followed vigilantly to ensure an adequate and complete response to therapy, whether that therapy is medical or surgical.

Antibiotic Therapy. After appropriate cultures have been obtained, IV administration of antibiotics should be started. Empirical antibiotic coverage usually is best begun with a first-generation cephalosporin; however, the choice of antibiotics should provide coverage for any specific pathogens that the patient's history suggests may be present.

The route (IV versus oral) and duration of antibiotic therapy for AHO and septic arthritis have received considerable attention in the literature.* Historically, patients were managed with prolonged parental antibiotic therapy. When oral antibiotic therapy was first introduced, it was in an inpatient setting with close monitoring of serum bactericidal levels.[258,261,345] Subsequently oral antibiotics have been administered on an outpatient basis and efforts have been made to shorten the duration of treatment.[260,272,342]

Recently, Peltola and associates reported results in treating 50 consecutive patients with a short course of IV antibiotics (average of 4 days), followed by oral antibiotics, for a total of 3 to 4 weeks of antimicrobial therapy.[272] Antibiotic serum concentrations and bactericidal activity were not monitored, but the patients' clinical course, CRP, and ESR were closely followed. The average hospital stay was 11 days and the total duration of antibiotic therapy averaged 23 days. No treatment failures or long-term complications were noted. Despite these encouraging results, Nelson has warned against a " 'cookbook' approach of standardized management" for a "disease with varied manifestations and variable course."[259]

We believe that the only absolute dictum in the management of patients with osteoarticular infections is that they require close clinical follow-up. A short course of antibiotic therapy (2 to 3 weeks) is often adequate for patients who present in the first 48 hours of illness and who have a rapid clinical response to drug treatment, whereas patients who

*See references 75, 100, 117, 134, 136, 169, 231, 248, 252, 256, 265, 272, 273, 276, 317, 342, 379.

*See references 75, 85, 94, 117, 134, 136, 156, 213, 231, 247, 248, 252, 256, 257, 259, 262, 272, 273, 276, 292, 307, 317, 342, 345.

present early but fail to demonstrate an adequate clinical response to antibiotics may require surgical debridement and prolonged antibiotic therapy. All patients should be followed on a long-term basis to ensure that there are no late sequelae, as these may not appear until well after the acute stage of the disease.[162,200]

Complications

Because of an increased awareness of osteoarticular infection and improved diagnostic techniques, complications from AHO and septic arthritis are becoming less common. Potential complications include systemic sepsis, growth deformity, pathologic fracture, osteonecrosis, and chronic infection. The propensity of osteomyelitis and septic arthritis to cause systemic illness is demonstrated by the 50 percent mortality in the preantibiotic era.[231,330] Today, systemic illness due to AHO is rarely seen, although there have been reports of cardiopulmonary complications secondary to osteomyelitis (e.g., bronchopneumonia, cardiac failure from septic pericarditis, septic pneumonias).[168,283,340]

Fortunately, orthopaedic complications such as physeal arrest (resulting in shortening or angular deformity, or both), physeal stimulation (resulting in overgrowth), pathologic fracture, osteonecrosis, and chronic osteomyelitis are also increasingly uncommon.[162,247,256,273,377] The treatment of physeal arrest is discussed in detail in Chapter 39, General Principles of Managing Orthopaedic Injuries.

OSTEONECROSIS OF THE PROXIMAL FEMUR

Osteonecrosis following musculoskeletal sepsis is most common in the proximal femur of infants. The predilection of this potentially devastating complication to infants is a result of the unique vascular anatomy of the infantile proximal femur. Prior to the development of a secondary ossification center within the proximal femur, the metaphysis and epiphysis share a common blood supply (see Fig. 34–1). Thus, if osteomyelitis of the proximal femur in an infant is inadequately treated, the infection can spread through the common blood supply to the proximal femoral epiphysis. This can lead to osteonecrosis of the entire proximal femoral epiphysis and, in severe cases, to destruction of the physis of the proximal femur. The sequelae of such an insult range from mild coxa magna to complete physeal arrest with dislocation of the hip. This devastating complication has been reported in up to 50 percent of cases of infantile hip sepsis, particularly if the diagnosis was delayed.[148,264]

In an effort to determine the best treatment for these hips, Hunka and associates classified the types of growth arrest following inadequately treated hip sepsis.[165] Their classification is based on the presence or absence of a capital femoral epiphysis and the stability of the hip (Fig. 34–13). In a review of ten hips treated with a wide range of surgical procedures, the authors found eight satisfactory outcomes and two unsatisfactory results. In both of the unsatisfactory cases there was pseudarthrosis of the femoral neck (type III hip).

A number of different surgical procedures have been described to treat the sequelae of proximal femoral osteonecrosis. Hunka was one of the first to modify Albee's greater trochanteric osteotomy[8] and use it to treat proximal femoral growth arrest secondary to sepsis. Lloyd-Roberts later added a varus osteotomy.[214] Others, including Colonna, have reported results in small series of modifications of this procedure.[22,77,154,205,207,339,368] More recently, Freeland and associates reported an 11-year follow-up in 17 patients treated with a greater trochanter hip arthroplasty.[124] The results were unpredictable but, in general, were better when the procedure was combined with a proximal femoral varus osteotomy. The benefit of varus and acetabular arthroplasty has been noted by others.[69,148,339,368] All of these authors stress the unpredictability of greater trochanteric arthroplasty and the need for close follow-up to address limb length inequality with a well-timed epiphysiodesis.

Cheng and associates used a vascularized iliac crest graft to replace the damaged femoral head and neck as an alternative to greater trochanteric arthroplasty.[68] Unfortunately, their results were equally unpredictable, with excellent graft incorporation in only three of seven hips. Choi and associates reviewed the late sequelae of infantile hip sepsis in 34 hips, which represents one of the largest series in the literature.[69] The authors modified the classification of Hunka and associates and included hips that had less severe deformity. Not surprisingly, they found improved function in those patients with milder deformity. Eleven of 13 hips with total destruction of the femoral head and neck were treated by reconstructive surgery, but only four of these 11 hips had a satisfactory outcome.

In an effort to determine whether attempts at reconstruction are warranted, Wopperer and associates reviewed nine hips at an average of 31.5 years after an episode of infantile sepsis.[382] None of the hips had been treated by reconstructive surgery. The Harris hip score was excellent in six hips, good in one, and poor in two. Based on these findings, the authors recommended a more conservative approach, avoiding attempts at reconstruction and performing only contralateral epiphysiodesis.

When confronted with an infant with untreated or undertreated hip sepsis, we believe that initial efforts to reduce the hip joint are warranted. For patients with complete proximal femoral growth arrest, the decision to perform salvage procedures must be made on an individual basis. The unpredictable results of these procedures and the relatively good long-term results of conservative treatment favor a more conservative approach. Unquestionably, the best solution is continued improvement in the acute management of infantile hip sepsis.

CHRONIC OSTEOMYELITIS

With the advent of antibiotics, greater awareness of osteoarticular infection, and improved diagnostic tests, chronic osteomyelitis has also become an uncommon problem.

The hallmark of chronic osteomyelitis is the presence of dead bone and, often, gross purulence. As such, the condition clearly requires surgical management. The principles of treatment in the child are similar to those in the adult (see Fig. 34–8). First the infection must be eradicated. This requires removal of all purulent and necrotic tissue. During debridement, it is important to remember that children

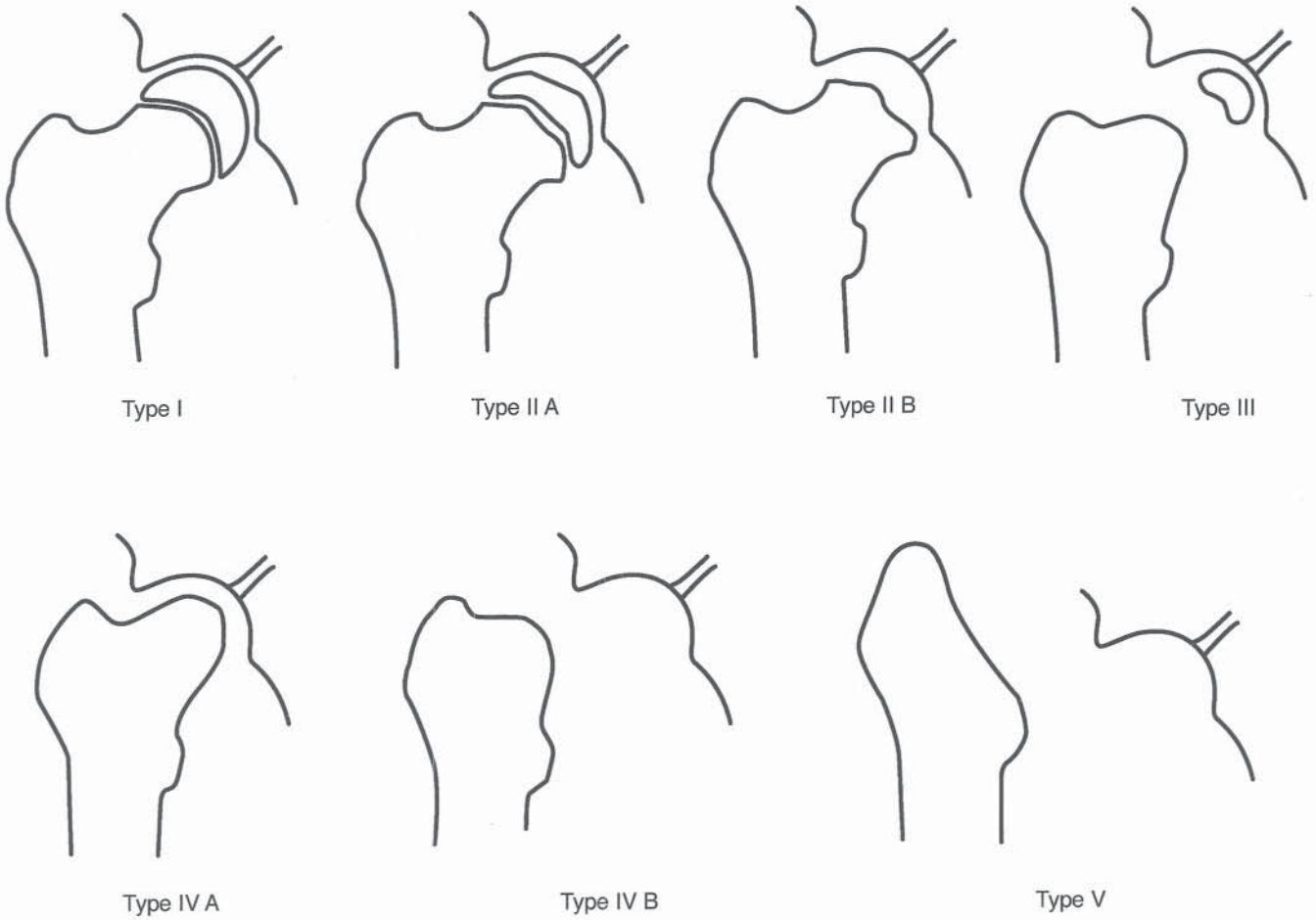


FIGURE 34–13 Hunka's classification of late sequelae of septic arthritis of the hip.
Type I—Minimal femoral head changes.
Type IIA—Femoral head deformity with a normal growth plate.
Type IIB—Femoral head deformity with growth arrest.
Type III—Pseudarthrosis of the femoral neck.
Type IVA—Complete destruction of the proximal femoral epiphysis, with a stable neck segment.
Type IVB—Complete destruction of the proximal femoral epiphysis, with a small unstable neck segment.
Type V—Complete destruction of the head and neck to the intertrochanteric line, with dislocation of the hip.

often have an involucrum present. If so, care should be taken to remove only the sequestered bone and, if possible, to leave behind the hypertrophying periosteum. Once the infection has been controlled with adequate debridement and appropriate antibiotics, reconstruction of any residual bony or soft tissue defects can be undertaken.

Cierny has stressed the importance of the condition of the host in the treatment of adult osteomyelitis.[71] These systemic and nutritional factors are also important in the treatment of children with chronic osteomyelitis, as they frequently have coexisting diseases or nutritional deficiencies. A serum albumin level greater than 3.5 g/dL and a total lymphocyte count greater than 1,500/mm^3 have both proved to be good predictors of wound healing in orthopaedic surgery.[95]

Recent reports on the management of chronic osteomyelitis have highlighted technological advances such as free tissue transfer, hyperbaric oxygen therapy, and Ilizarov techniques that may assist reconstructive procedures.[28,46,71,388] However, the unique healing properties of children may allow for equally good results with less complex procedures, such as simple Papineau bone grafting.[26,111,120,121,306]

Special Considerations

SUBACUTE OSTEOMYELITIS

The primary characteristic of subacute osteomyelitis is the insidious onset of extremity pain without signs of systemic illness. At the time of presentation, the pain usually has been present for at least 2 weeks, findings from laboratory studies are often unremarkable, and the radiographic appearance may mimic that of a number of benign and malignant neoplasms.[138,150,190,210,300] The first description of subacute osteomyelitis is credited to Sir Benjamin Brodie, in 1836, when he described a localized abscess of the tibia in a patient who had no history of previous acute illness.[56] Subsequently other authors also reported the presence of this entity,[155,278] and in 1973 Gledhill proposed a classification of the condi-

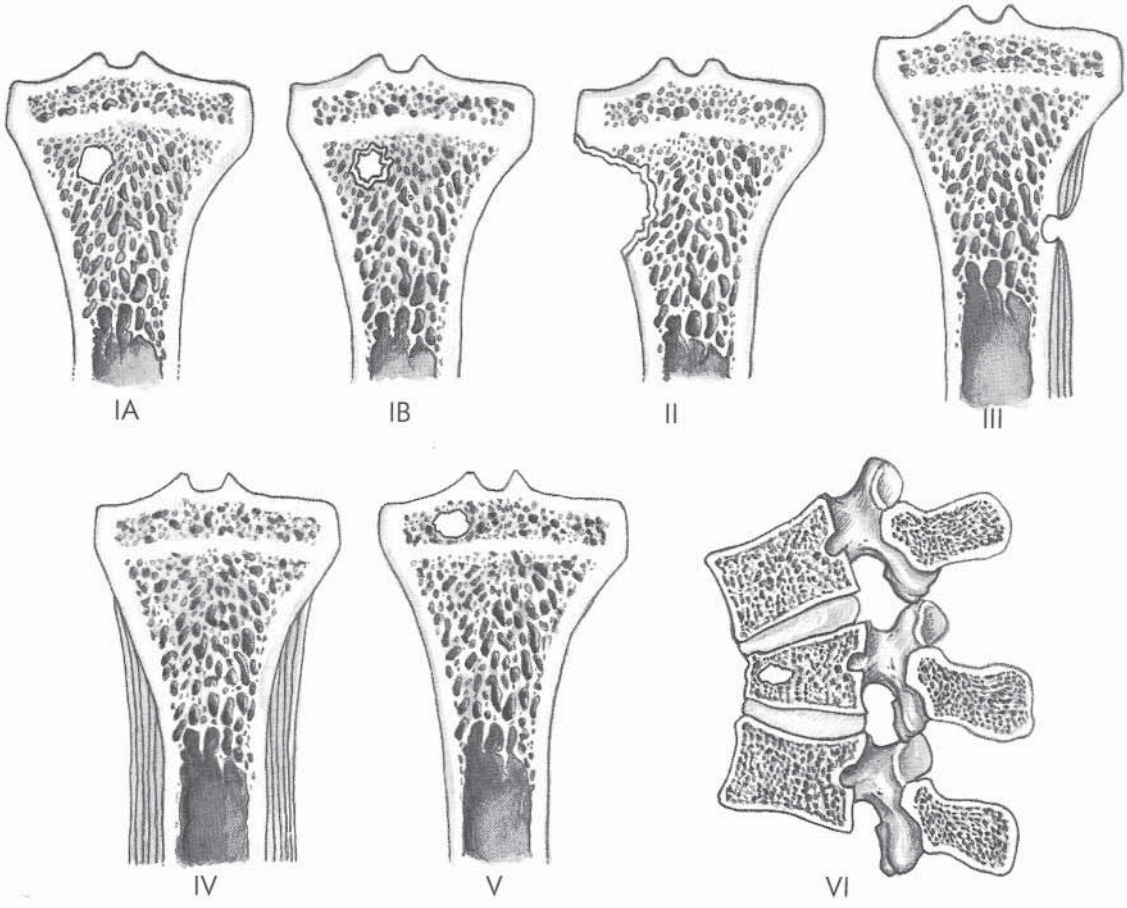


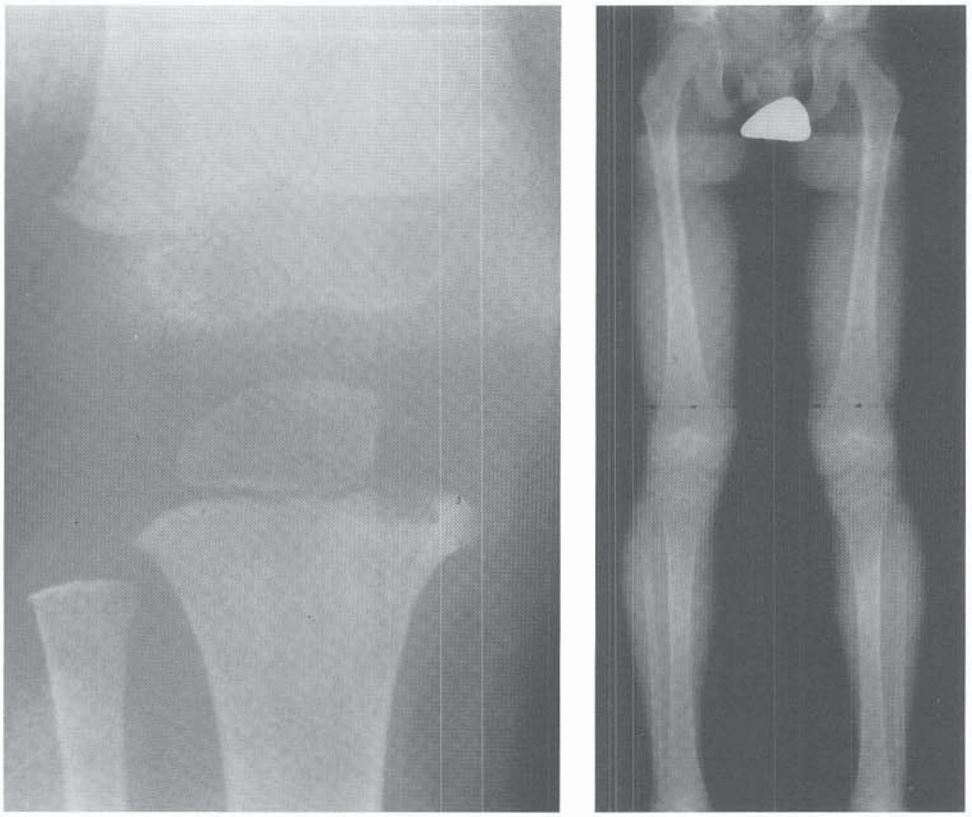
FIGURE 34–14 Radiographic classification of subacute osteomyelitis according to Roberts and associates. This classification system is based on the anatomic location, the response of the surrounding tissue to the infection, and the similarity to benign or malignant tumors:
Type IA—Metaphyseal area of radiolucency without marginal sclerosis.
Type IB—Metaphyseal area of radiolucency with surrounding reactive bone, the classic Brodie's abscess.
Type II—Metaphyseal area of radiolucency with cortical erosion (may be misinterpreted as an osteosarcoma).
Type III—Localized diaphyseal lesion with periosteal reaction (may be misinterpreted as an osteoid osteoma).
Type IV—Diaphyseal lesion with subperiosteal new bone formation (may be misinterpreted as Ewing's sarcoma).
Type V—Epiphyseal lesion.
Type VI—Vertebral lesion. This entity is also known as diskitis.
(Redrawn after Roberts JM, Drummond DS, Breed AL, et al: Subacute hematogenous osteomyelitis in children: a retrospective study. J Pediatr Orthop 1982;2:249.

tion.[138] Subacute osteomyelitis is believed to be the result of an altered host-pathogen relationship, either from an increase in host resistance, or from a decrease in bacterial virulence, or from partial treatment of AHO with oral antibiotics. The incidence of subacute osteomyelitis is reportedly increasing, but whether this represents a true increase in the occurrence of the primary disease or an increase in the number of cases of AHO attenuated by partial treatment with antibiotics is unknown.[81,138,379]

There are a number of unique characteristics of subacute osteomyelitis that are best discussed in the context of Roberts and associates' modification of Gledhill's initial classification (Fig. 34–14).[138,300] The lesions are most often metaphyseal, but they may be found in the diaphysis and in the epiphysis. The lesions usually are "obviously" benign, but they may have an aggressive radiographic appearance. Occasionally the lesions cross the physis. Somewhat surprisingly, lesions involving the physis or epiphysis seldom cause subsequent growth disturbance. Finally, the lesions may appear in the spine—a condition synonymous with diskitis.

The course of subacute osteomyelitis has been noted to be uniformly mild, with most cases responding to surgical or medical management (or both).[135,138,150,190,305] Because results have been so consistently good, Hamdy and associates have questioned whether surgery is routinely indicated.[150] In their review of 44 patients, 24 of the patients were successfully managed without surgical biopsy. They believed that benign radiographic features provided an accurate diagnosis, without the need for biopsy, in more than 90 percent of cases. Benign radiographic features included (1) lesions surrounded by sclerosis, (2) lesions that crossed the physis, (3) lesions with a "serpentine" shape or multiple cavities, (4) epiphyseal lesions, and (5) lesions without surrounding destruction.

Our approach to subacute osteomyelitis follows the rec-



A B

FIGURE 34–15 Epiphyseal subacute osteomyelitis. **A,** AP radiograph of the knee shows lytic lesion involving the proximal tibial metaphysis and epiphysis. **B,** Standing AP radiograph of the lower extremities obtained 5 years after treatment shows normal growth of the proximal right tibial physis.

ommendations of Hamdy.[50] We favor an initial trial of antibiotics for "radiographically obvious lesions." We reserve surgical treatment for lesions that fail to respond to medical treatment or that have an aggressive radiographic appearance. Somewhat surprisingly, lesions involving the physis or epiphysis seldom cause subsequent growth disturbance (Fig. 34–15).

DISKITIS

Diskitis—an inflammatory lesion of the disk space—has long been recognized as a clinical entity that may manifest in children with back or abdominal pain or refusal to bear weight.* Most studies have described the relatively benign course of the disease process, the frequent delay in diagnosis, and the predilection of the inflammatory lesions to localize in the lumbar and thoracolumbar spine. Most early authors recommended treatment with immobilization, with or without antibiotic therapy.[236,326]

Both a bacterial and a viral/"inflammatory" etiology have been proposed as the cause of diskitis.[299,370] A recent study by Ring and associates shed considerable light on the etiology of diskitis.[298] In a review of 47 patients, the authors reported a prolonged course of disease or recurrent symptoms in 67 percent of patients who did not receive antibiotic therapy, compared with 50 percent of patients who were treated with oral antibiotics and 18 percent of patients who received IV antibiotics. They also noted that most studies show a 30 to 50 percent positive culture rate, which is not significantly different from the positive culture rate found in patients with septic arthritis.* In addition, both plain radiography and MRI have shown similarities between diskitis and adult pyogenic spondylitis (see Fig. 34–11).[237,298]

We agree with Ring and associates[298,299] that diskitis is a bacterial disease. Presumably the infection begins in the endplate and then communicates via vascular channels to the avascular disk space.[374] Initially, we treat all cases of diskitis with IV antibiotics until the clinical findings and infectious indices (WBC count with differential, ESR, CRP) improve, at which time we change to oral antibiotic therapy. We perform blood cultures on all patients before beginning antibiotic therapy. Biopsy is indicated only when patients do not respond to antibiotic therapy, and in these cases, the procedure can often be performed percutaneously under CT guidance.[3,143,145,158,161,170]

Our practice is to treat infection of the disk space with 4 weeks of antibiotic therapy. If necessary, we have the patient use a removable orthosis to help alleviate symptoms during the early phase of treatment. There is no long-term need or biomechanical requirement for bracing in these patients. It has been reported that adolescents may have a subacute clinical course.[370] We have found that adolescents often present initially with less remarkable findings on examination and less elevation in infectious indices; however, they frequently have a more protracted clinical course,

*See references 9, 12, 43, 47, 102, 103, 227, 236, 239, 285, 298, 308, 326, 370.

*See references 94, 117, 169, 257, 262, 272, 273, 342.

often with mild back pain persisting over a period of several months.

INFECTION SECONDARY TO PUNCTURE WOUNDS

Puncture wounds of the foot are a common problem in children. Most puncture wounds do not result in serious sequelae.[291] However, a small number will progress and develop either superficial (cellulitis)[119] or deep infection (osteomyelitis or septic arthritis).[202] Because of these infrequent but potentially serious complications, the acute management of puncture wounds is controversial. Most reports on the treatment of puncture wounds are either retrospective analyses or anecdotal reports, with few prospective studies available to elucidate the best treatment approach.[359] Recommendations for the acute care of puncture wounds range from simple wound care and tetanus prophylaxis to empirical "prophylactic" antibiotic therapy to routine surgical debridement for all cases.[180,199,274,359]

Patients in whom either superficial or deep infections develop after punctures typically complain of pain and swelling at the area of the wound 2 to 5 days after the injury, a time when symptoms have usually resolved in noncomplicated cases. Initially it may be difficult to distinguish among cellulitis, osteomyelitis, and septic arthritis. Patients with cellulitis usually respond to elevation of the foot and oral antibiotics active against *Staphylococcus.* If the symptoms continue with the patient on this treatment regimen, deep infection should be suspected. Although cellulitis is most often due to *Staphylococcus, Pseudomonas aeruginosa* is the primary pathogen in osteomyelitis complicating puncture wounds of the foot.[176] If a deep infection does develop, surgical intervention is usually required, although there are reports of successful medical management.[171,199,238]

Because puncture wounds usually do not result in infection, most wounds should be carefully cleaned, the child should be given tetanus prophylaxis, and the affected foot should be rested and elevated. The parents should be informed of the potential for infection developing and the child should be closely followed on an outpatient basis until the wound has healed and the risk of deep infection has passed.

Initially, we treat patients with established infections with antibiotics (either oral or IV) unless there is clinical or radiographic evidence of deep infection. Patients with obvious deep infection and those who fail to improve with antibiotic therapy are treated with surgical debridement. In general, we favor a plantar approach, as it allows easier debridement of the puncture tract. After surgical debridement, most cases can be managed successfully with 7 to 10 days of IV antibiotics.[172]

The use of oral fluoroquinolone antibiotics (e.g., ciprofloxicin) as antipseudomonal agents in children is controversial. For many years, quinolone-induced cartilage toxicity, observed in experiments with some skeletally immature animals, was considered a contraindication to the use of these agents in children. However, accruing data indicate the safety and effectiveness of ciprofloxicin in children.[141,289,311,312] Thus, we occasionally use oral ciprofloxicin in pediatric patients. However, we do not advocate its use (or the use of any other antibiotic) for routine prophylaxis following puncture wounds.

SEPTIC ARTHRITIS OF THE SACROILIAC JOINT

Pyogenic infection of the sacroiliac joint is more common in late childhood than in early childhood, and the disease often has an insidious onset. Patients may complain of pain in the back, the hip, or the leg. Findings on physical examination may include a positive Patrick or FABER (flexion, abduction, external rotation) test. (In a positive FABER test, any of the listed movements of the hip elicits pain.) The diagnosis of sacroilitis is often delayed because of the frequent subacute onset, the nonspecific symptoms, and the generalized findings on physical examination. However, nuclear imaging has dramatically reduced the delay in diagnosis.[42,87,313] MRI may also be helpful in achieving an accurate and timely diagnosis.[146]

Staphylococcus is the most common organism responsible for septic arthritis of the sacroiliac joint, but *M. tuberculosis* and *Brucella* have also been commonly reported.[18,240,268] Most authors report successful results with a 3- to 6-week regimen of antibiotics alone. Surgical decompression is reserved for cases in which an abscess or sequestrum is present.[42,87,268,313] Long-term sequelae are rare, even though most patients will have residual radiographic "sclerosis" of the sacroiliac joint.[313]

OSTEOMYELITIS IN PATIENTS WITH SICKLE CELL DISEASE

Bone pain in children with sickle cell disease is a common occurrence. Although it is most often the result of sickle cell crisis, the orthopaedist frequently becomes involved because of the possibility of osteoarticular infection coexisting with crisis/infarction. A number of studies have attempted to determine the incidence of osteoarticular infection in children with sickle cell disease who were admitted for musculoskeletal pain. Results ranged from 0.2 to 5 percent.[27,86,109,185,280,341]

Despite this rather dramatically low incidence, diagnosing osteoarticular infection in patients with sickle cell disease remains a frequent and difficult problem. In many studies, the clinical, laboratory, and radiographic findings in patients in sickle cell crisis are identical to those in patients with osteoarticular infection.*

Patients in acute vaso-occlusive sickle cell crisis may present with point tenderness, swelling, and warmth. Erythema and effusion, although less common in sickle cell crisis, may also be present.[86,108,185,280,331] Fever may be present with either crisis or infection, and the temperature is not predictably higher with infection.[109,185] Laboratory studies, including WBC count, differential, and ESR, have not been shown to be reliable in differentiating crisis from infection.[86,221,341] Unfortunately, plain radiographs are equally ambiguous. Periosteal new bone formation is the earliest radiographic feature of both osteomyelitis and bone infarction.[32,108,109]

The effectiveness of bone scans in distinguishing infection from infarction is a matter of controversy.[185,187,287,296] Initially,

*See references 86, 185, 187, 221, 287, 296, 341.

infarction causes a decrease in vascularity, which results in a decrease in uptake in that area. Approximately 3 to 7 days after the infarction, an inflammatory reaction is established. The increase in vascularity causes an increase in radionuclide uptake. At this point the bone scan will appear similar for both infection and infarction. Thus, to be of benefit, a bone scan should be performed within 72 hours of the onset of symptoms. However, both Keeley and Buchanan[185] and Dalton and associates[86] failed to find radionuclide scans universally diagnostic in differentiating between infarction and infection. Bone marrow scans with gallium 67 citrate[14] and ultrasound performed in conjunction with bone scans[296] have helped to identify patients with infection, even though the accuracy of these imaging studies is less than 100 percent.

Because sickle cell patients with bone pain are much more likely to be in vaso-occlusive crisis than to have osteoarticular infection, and because there are few objective clinical findings or tests to distinguish infection from infarction or crisis, we initially treat these patients with appropriate medical treatment (i.e., analgesics, hydration, and oxygen) for crisis. We do not routinely treat these patients with antibiotics. If patients fail to improve in 2 to 4 days, we obtain blood cultures and aspirate all suspected foci prior to initiating antibiotic therapy.

Although the predisposition of patients with sickle cell disease to develop *Salmonella* osteoarticular infection is well recognized, there is some controversy as to whether *S. aureus* or *Salmonella* is the most common pathogen overall. A recent review of the literature by Burnett and associates found the incidence of *Salmonella* infection to be almost twice that of *S. aureus.*[59] This predilection is significantly greater than was reported in studies published before 1981. These results are also supported by the 1993 report by Piehl and associates.[280] Epps and associates, however, found a slight predominance of *S. aureus* (8 of 15 patients were infected with *S. aureus,* compared to 6 of 15 patients infected with *Salmonella*).[109] Because both organisms are so common, initial antibiotic therapy should cover *S. aureus* and *Salmonella* until cultures confirm which pathogen is present.[384]

The recommendations for surgical debridement vary among authors, with some reporting that surgery provides the best outcome[309] and others believing that nonsurgical management yields good results.[4,15,221,341] Although specific indications for surgery are not clear, early diagnosis of osteomyelitis and prompt surgical drainage of an abscess will reduce complications. It is important to remember that these patients have an altered immune status and an inadequate oxygen delivery system. As a result, they are prone to complications. Thus, an inadequate or delayed response to medical treatment should lower the threshold for aggressive surgical debridement.[109] The use of a tourniquet in patients with the sickle cell gene has been considered contraindicated, as the ischemia may lead to thrombosis and crises. Several studies, however, have described the safe use of tourniquets during surgery on sickle cell patients.[5,130,131,337]

Sickle cell dactylitis (hand-foot syndrome) has been reported in approximately 10 to 20 percent of children with sickle cell disease.[365,383] This condition usually is seen in infants and children less than 4 years old. The condition is characterized by the acute onset of symmetric or asymmetric painful swelling of the hands and feet. In most cases the condition is benign[383]; however, there have been reports of *Salmonella* osteomyelitis presenting as hand-foot syndrome.[198,263,365,367] Distinguishing the two conditions can be difficult. Laboratory tests and radiographic studies have not been found helpful in the differential diagnosis.

CHRONIC RECURRENT MULTIFOCAL OSTEOMYELITIS

Chronic recurrent multifocal osteomyelitis (CRMO) is an unusual inflammatory bone disease that mimics osteomyelitis and most often occurs in children and adolescents. The clinical picture is one of gradual onset of malaise and pain at multiple sites. The disease is characterized by subsequent resolution with relapse occurring months later, with the findings and symptoms similar each time. This cycle of onset and relapse can occur over a period of 2 to 5 years.

Radiographic findings are similar to those of osteomyelitis. Clinically silent lesions can often be detected on bone scans. Histologic findings from biopsy are consistent with osteomyelitis. Repeated cultures of bone and blood are negative. Lesions are most often seen in the clavicle, the spine, and the long bones of the lower extremities, but they may occur anywhere.[64,126,387] Associated palmoplantar pustulosis has been reported.[33,39,181]

Antibiotics and anti-inflammatory agents have been ineffective in treating CRMO.[126,191,223,316] There are a few case reports of successful treatment of painful symptoms with interferon or other chemotherapeutic agents.[16,125] However, because the disease is generally benign and self-limiting, patient management chiefly entails making the correct diagnosis and limiting unnecessary biopsies and antibiotic treatment.

GONOCOCCAL SEPTIC ARTHRITIS

Gonococcal septic arthritis occurs in up to 5 percent of patients with gonorrhea and may manifest anywhere from 5 days to months after the initial infection. It is frequently seen as part of a spectrum of the syndrome of disseminated gonococcal infection (DGI), although DGI can occur without gonococcal arthritis (GCA) and GCA can occur without DGI. The syndrome of DGI includes arthritis, tenosynovitis (usually affecting the dorsum of the upper extremity), meningitis, myopericarditis, and dermatitis (the classic papular, petechial skin lesions on the distal extremities).[63,140] Disseminated infection has been reported to be more common in females and associated with periods of low progesterone activity, such as menstruation and pregnancy.[159]

There are two primary situations in which gonococcal arthritis is seen by the pediatric orthopaedist: in the neonate, who contracts the disease from the birth canal of the mother, and in the sexually active adolescent. If gonococcal arthritis is seen in patients after the neonatal period but before puberty, or in sexually inactive adolescents, the orthopaedist needs to be particularly alert to the possibility of sexual abuse.[297,373]

Up to 60 percent of patients with gonococcal disease will present with polyarthritis.[140,246] Any joint may be affected, although more distal joints (wrist, ankle, and knee) tend to be involved more frequently.[63,140,380] Because of its fragility, *N. gonorrhoeae* is difficult to culture in the laboratory. Thus, it is important that specimens for culture be obtained from

all possible sources, including blood, synovial fluid, and all mucous membranes (e.g., pharynx, vagina, rectum). Material from normally sterile sites (blood, synovial fluid) should be isolated on chocolate agar, a heated blood medium. Thayer-Martin, a medium that contains antibiotics, should be used to isolate material from the pharynx and anogenital region. Classically, cultures from blood and synovial fluid are mutually exclusive, that is, patients with positive synovial fluid cultures and gonococcal arthritis will not have positive blood cultures, as is seen in patients with disseminated *N. gonorrhoeae* infection.[52,63,140] Gram stains of all culture material should be evaluated for the characteristic gram-negative intracellular diplococci.

Once the diagnosis of gonococcal arthritis has been made, most patients can be managed successfully with local aspiration and irrigation of the joint and IV antibiotic therapy, without the need for a formal arthrotomy. Despite reports of increased penicillin sensitivity in cases of GCA,[92,140] the increasing prevalence of penicillin-resistant strains has led to recommendations for a third-generation cephalosporin as initial therapy.[380] Although 7 days of antibiotic therapy is generally sufficient for most cases of GCA, it is important to remember that gonococcal osteomyelitis has been reported as a complication of septic arthritis.[17,349] Finally, there may be a lower threshold for recommending arthrotomy when the hip joint is involved.

OSTEOARTICULAR INFECTIONS SECONDARY TO RARE PATHOGENS

Tuberculosis. Since antituberculous drug therapy became available, the incidence of tuberculosis bone or joint infection has generally declined.[144] However, recent reports indicate an increase in the number of cases of tuberculous bone and joint infection, possibly due to resistance to the pathogen *Mycobacterium tuberculosis*.[234,235]

Extrapulmonary tuberculosis is more common in children, especially those less than 5 years old. Thus, the condition needs to be included in the differential diagnosis of bone and joint infections in this age group. The lungs are the most common site of initial infection in children. If the lung infection is not treated, the tubercle bacilli will spread to bone or joints during hematogenous and lymphatic dissemination of the infection in 5 to 10 percent of children.[328]

CLINICAL MANIFESTATIONS. Osteoarticular tubercular infection can manifest as osteomyelitis (most commonly in the spine), septic arthritis, dactylitis, or multifocal cystic disease. The timing of the development of extrapulmonary infection is quite variable. It may take 1 to 3 years for long bones to be affected, whereas dactylitis may develop in a few months.

Tuberculous osteomyelitis most often involves the spine, with the lower thoracic and upper lumbar areas most commonly affected. The first description of tuberculosis of the spine is credited to Percivall Pott,[284] and the condition has often been referred to as Pott's disease. In tuberculous spondylitis, the child usually presents with back pain and stiffness. The affected child will walk with a protective gait, taking light steps and keeping the back hyperextended. The infection usually starts in the anterior third of the vertebral body near the end-plate, where blood perfusion and oxygen concentrations are higher. The formation of an abscess is the classic hallmark of spinal tuberculosis. Purulent material decompresses out the anterior cortex and spreads under the anterior or posterior longitudinal ligaments (Fig. 34–16). Often two contiguous vertebral bodies and the intervening disk become involved, although the infection can also skip levels. A kyphosis will develop if bone destruction continues and the vertebral bodies collapse. Untreated disease may progress to spinal cord compression with paralysis, which has been termed Pott's paraplegia. Caseous material collects in the front of the spine and may track inferiorly, producing a psoas abscess. Abscesses expand along fascial planes and can "point" in the groin or anterior chest wall. Cervical disease and abscess formation can lead to hoarseness (from recurrent laryngeal nerve paralysis), dysphasia, and stridor (Milar's asthma). Epidural abscess is more frequently associated with paralysis, which has been reported in up to 25 percent of cases. However, with improved diagnosis and medical treatment, this complication has diminished.

RADIOGRAPHIC FEATURES. Radiographic studies of spinal tuberculosis are highly suggestive of the diagnosis and often pathognomonic. The plain radiographs initially show radiolucency of the vertebral body. As the disease advances, the vertebral body expands and the borders become indistinct. With progressive destruction of bone, the vertebral body collapses. At first the intervertebral disk space narrows; later it is obliterated. Paraspinal abscesses appear as fusiform shadows along the vertebral column on the AP radiograph and as an anterior soft tissue mass on the lateral radiograph. Late involvement is characterized by a sharply angled kyphosis or gibbus at the level of vertebral destruction (Fig. 34–17).

MRI is useful in delineating the extent of the disease and differentiating it from other types of vertebral osteomyelitis. Vertebral and disk destruction and the location of paravertebral, abcesses are graphically demonstrated on MRI (see Fig. 34–16B).[188,212] Gadolinium studies show rim enhancement of lesions, an appearance more typical of tuberculosis than of septic conditions.[19,188] With tuberculosis of the spine more vertebrae are involved and paravertebral abscesses are larger than in pyogenic vertebral osteomyelitis.[19] Brucellar spondylitis may be differentiated from tubercular in that *Brucella* more commonly involves lumbar vertebrae while tuberculosis more often affects thoracic levels.[79]

Tubercular osteomyelitis in other locations most commonly involves the epiphysis or metaphysis. Unlike other bone infections, the physeal plate does little to stop the spread of the infection. As the infection progresses, the area of bony destruction expands centrifugally, typically appearing on radiographs as a round cystic lesion with obscure margins. Because the disease process is almost entirely lytic, there is little periosteal reaction and often no sclerotic margin. There may be widening and accelerated growth of the epiphysis when the condition is chronic and hyperemic.

Tubercular septic arthritis most commonly involves the hip and knee. As in pyogenic septic arthritis, it may result either from hematogenous spread or from direct extension from an adjacent area of tubercular osteomyelitis. Initially the patient will have joint swelling, and there will be proliferation and thickening of the synovium. Early in the disease process, the radiographic appearance is indistinguishable from that of other causes of chronic joint inflammation. As with tubercular osteomyelitis, hyperemia results in extensive

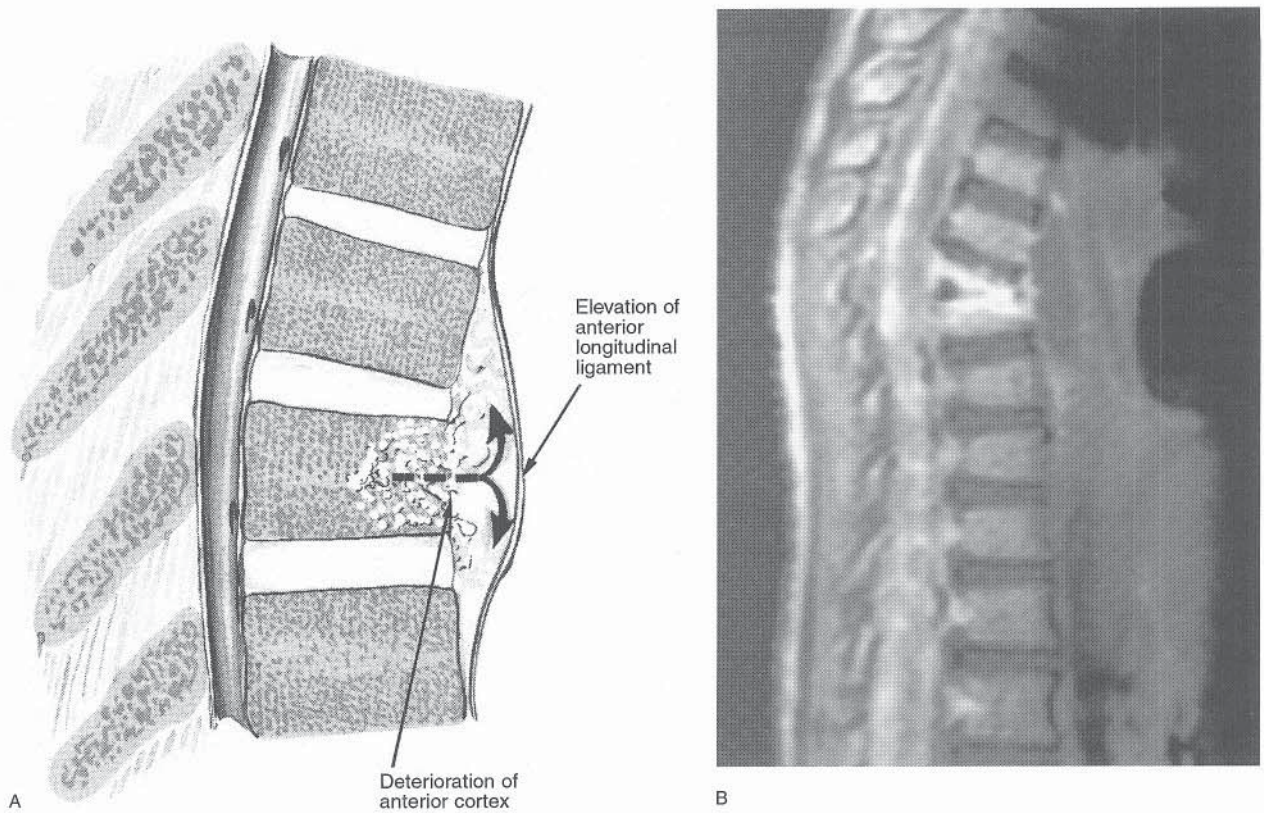


FIGURE 34–16 Pathogenesis of Pott's disease (tuberculosis of the spine). **A,** Infection egressing from bone elevates the anterior longitudinal ligament. **B,** MRI shows collapse of the anterior vertebral bodies and the intervening disk space as well as abscess formation anteriorly.

osteopenia and possible epiphyseal overgrowth. Pannus formation over the articular cartilage and subchondral bone erosion (starting at the synovial margins) cause joint space narrowing and subchondral cystic erosion.[277,279]

In tubercular dactylitis, children experience progressive, painless swelling of the phalanges, metacarpals, and metatarsals. The hand is affected more often than the foot. The disease may involve several digits, with shortening and contractures of the affected digits. Disability is minimal, however. Radiographs reveal a cystlike enlargement of the infected short tubular bones, with thinning of the cortex and some subperiosteal new bone formation (Fig. 34–18).[153] Patients may also present with multifocal cystic lesions of the skeletal system.[288] In these cases the lesions may occur simultaneously in flat bones and in the shaft of the long bones, with a tendency for symmetric development.[318]

Osteoarticular tubercular infection must be considered in the differential diagnosis of any chronic lytic bone or joint lesion. The bone and joint infections in tuberculosis typically have an insidious onset and there are few or no signs of inflammation. Often patients present with general malaise, easy fatigability, and a history of weight loss. The differential diagnosis includes bacterial spondylitis, leukemia, Hodgkin's disease, eosinophilic granuloma, aneurysmal bone cyst, and Ewing's sarcoma. All of these conditions can produce destruction and collapse of the vertebral bodies, narrowing and obliteration of disk spaces, and paraspinal soft tissue swelling.

LABORATORY EVALUATION. Patients with skeletal tuberculosis usually have a normal WBC count and an elevated ESR and CRP.[23,217] In most cases the purified protein derivative skin test will be positive.

TREATMENT. The diagnosis of tubercular bone or joint infection can be made by identifying acid-fast bacilli on Ziehl-Neelson stain (Fig. 34–19), by growing *M. tuberculum* in culture, or by identifying the characteristic caseating granulomas on histologic examination (Fig. 34–20).[58] In the past, patients with tuberculosis were treated with long periods of rest, fresh air, and joint immobilization. Current management is based on early diagnosis and the use of antimicrobial drugs to which the organism is sensitive.

Although surgical biopsy is usually necessary to establish the diagnosis of osteoarticular tuberculosis,[288,361] the first line of treatment is generally medical. The American Academy of Pediatrics' recommended chemotherapy regimen for active tuberculosis as of 1997 consists of isoniazid, rifampin, and pyrazinamide for 2 months, followed by isoniazid and rifampin for the remaining 4 months.[13] This 6-month treatment protocol has been reported to be effective against drug-susceptible *M. tuberculosis.* For tubercular bone and joint infections, the Academy recommends a 12-month regimen consisting of isoniazid, rifampin, pyrazinamide, and streptomycin for the first 2 months, followed by isoniazid and rifampin for the remaining 10 months of therapy.[13,288] Patients with active AIDS as well as those with visceral disease need more intensive nutritional and medical management.

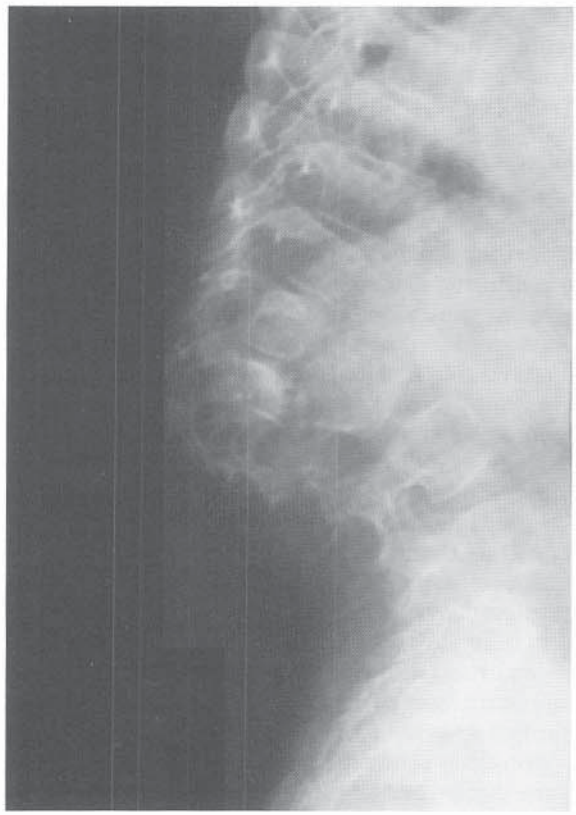


FIGURE 34–17 Kyphotic deformity of the lumbar spine secondary to tuberculosis.

The increasing incidence of drug-resistant tuberculosis complicates both control of the disease and the treatment of individual patients. In these cases an initial four-drug therapeutic regimen is recommended, with treatment including at least two bactericidal drugs, such as isoniazid, rifampin, streptomycin, other aminoglycosides, pyrazinamide, and high-dose ethambutol.[13] In vitro drug susceptibility testing and initial directly observed therapy are also recommended. The selection of drugs and the duration of treatment depend on the resistance pattern, the toxicities of the drugs, and the patient's response to therapy. Thus, deciding on an appropriate therapeutic regimen for drug-resistant tuberculosis is best done in consultation with a specialist in the field of infectious diseases who is familiar with the regional characteristics of the tuberculosis epidemic.[51,174]

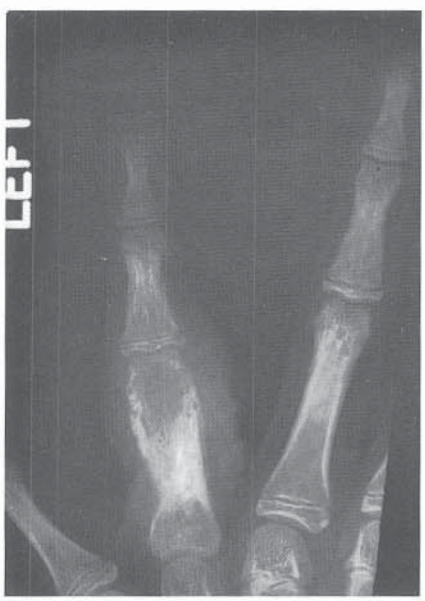


FIGURE 34–18 Radiographic changes of tuberculous dactylitis. Note the cystic changes in the proximal phalanx of the ring finger and the subperiosteal new bone.

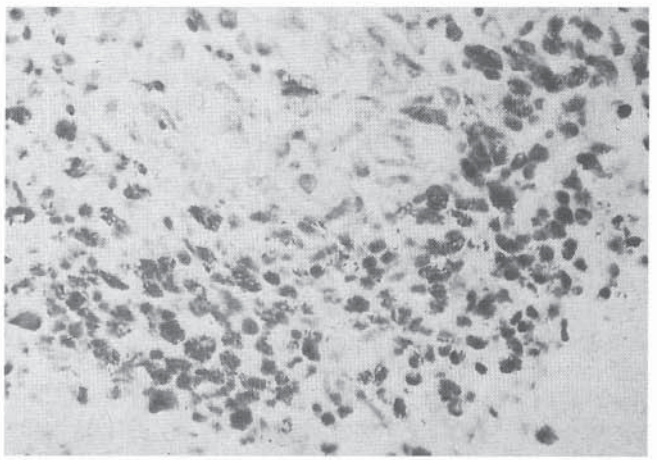


FIGURE 34–19 Ziehl-Neelson stain of a biopsy specimen from a patient with atypical mycobacterial arthritis. Multiple acid-fast bacilli are present, most within histiocytes. (From Bullough PG, Vigorita VJ: Atlas of Orthopaedic Pathology with Clinical and Radiologic Correlations. New York, Gower Medical Publishing, 1984.) (See color version.)

ORTHOPAEDIC TREATMENT. Surgical indications for osteoarticular tuberculosis infections are poorly defined. The majority

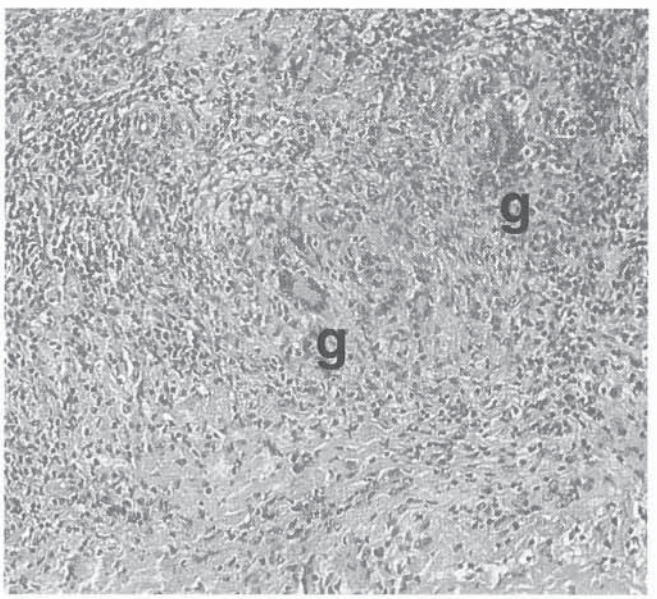


FIGURE 34–20 Micrograph of granulomatous tissue from a patient with tuberculous septic arthritis. Note the chronic inflammation with intermittent well-organized granulomas (*g*). The granuloma is circumscribed by plump, round epithelioid cells that have intermittent Langerhans'-type giant cells, as well as a caseating, necrotic central core. (From Bullough PG, Vigorita VJ: Atlas of Orthopaedic Pathology with Clinical and Radiologic Correlations. New York, Gower Medical Publishing, 1984.) (See color version.)

of studies on surgical management of skeletal tuberculosis have concentrated on spinal disease. Most investigators agree on surgical decompression for new or progressive neurologic deficits, marked spinal instability, and failure of medical treatment.[209,233,310] The Medical Research Council Working Party on Tuberculosis of the Spine reported early bony fusion and less kyphosis when radical debridement and bone grafting were performed.[232] However, the MRC working party also acknowledged that ambulatory chemotherapy yielded "very good" results and recommended this course of treatment if adequate facilities were lacking.[232,233] Lifeso and associates reserved surgical treatment for those patients with a neurologic deficit, spinal instability, or failure of medical management.[209] The authors noted more rapid and frequent neurologic recovery in those patients who were treated surgically, but progression of kyphosis did not occur in either the medically or the surgically treated group. Rajasekaran and Soundarapandian reported their results using anterior decompression and strut grafting.[286] They noted more complications in patients with large postdebridement deficits, marked preoperative kyphosis, and thoracic lesions. Grafts were more likely to fail if they spanned more than two disk spaces. The authors recommended either prolonged nonweightbearing or brace wear or a posterior arthrodesis if the graft exceeded two disk spaces. Surgical drainage of nonspinal osteoarticular tubercular abscesses has been reported to help ameliorate constitutional symptoms,[226,318,360] and curettage of bone lesions has been reported to promote earlier healing.[361]

Because of the reported good results obtained with chemotherapy alone, our initial treatment approach to the patient with osteoarticular tuberculosis is conservative. When an abscess is present, percutaneous CT-guided drainage may supply tissue for diagnostic purposes and provide therapeutic relief.[97,160,282] However, it is often necessary to remove diseased synovium by synovectomy and to debride bony lesions. Postoperative immobilization is not necessary, and early motion or continuous passive motion may help restore range of motion. Most patients are not debilitated and may continue reasonable activities while being treated medically. We also favor surgery for patients with progressive neurologic deficits or marked spinal instability, and for patients in whom medical treatment has failed. We perform operations under the "umbrella" of optimum chemotherapy. We prefer an anterior approach for decompression of spinal lesions, with adequate autogenous grafting. If the infection does not extend to the posterior spinal elements, the addition of posterior spinal fusion with instrumentation can be helpful in treating extensive spinal lesions and lesions associated with spinal instability. Laminectomy should be avoided, particularly in the cervical spine, because the procedure is associated with increasing kyphotic deformity and progressive neurologic deterioration.[209]

Lyme Disease. Lyme disease is a multisystem infection caused by the spirochete *Borrelia burgdorferi.* It is more common in children than in adults and is the most common vector-borne disease in the United States.[65,114,319] It was first recognized in 1975, when a cluster of children living near Lyme, Connecticut, were diagnosed with juvenile rheumatoid arthritis.[335] The most common vector is *Ixodes scapularis,* the black-legged or deer tick. Other *Ixodes* species are responsible for transmission in the western United States, Canada, and temperate areas of Eurasia.[65] It is most common in the northeast, mid-Atlantic, and north-central United States. In 1996, eight states (Connecticut, Delaware, Rhode Island, New York, New Jersey, Pennsylvania, Maryland, and Wisconsin) accounted for 91 percent of cases reported.[65]

Diagnostic criteria established by the CDC include the presence of the characteristic erythema migrans rash at least 5 cm in diameter or laboratory confirmation of infection and at least one musculoskeletal, neurologic, or cardiovascular manifestation of disease.[65] It is important to realize that the only reliable manifestation within the musculoskeletal system is the presence of an acute joint effusion. Joint pain, "fibromyalgia," and fatigue do not constitute orthopaedic manifestations.[139,319] Neurologic and cardiac manifestations include aseptic meningitis (confirmed by CSF testing), cranial neuropathy, radicular or peripheral neuropathy with CSF or EMG changes consistent with axonal disease, MRI changes consistent with leukoencephalopathy, and cardiac conduction defects. The CDC recommends that before the serologic diagnosis is established, all specimens testing positive or equivocal with a sensitive immunoassay or an immunofluorescent assay be confirmed with a Western blot. (During the first 4 weeks of infection, both IgM and IgG Western blot procedures should be used.[66])

Lyme disease begins with the often unnoticed bite of a deer tick. The characteristic erythema migrans rash develops 2 to 30 days later. Its presence is highly variable, having been reported in as low as 2 percent to as high as 90 percent of children with Lyme disease.[82,139,166,319] It is described as single or multiple expanding erythematous macules or papules with central clearing that reach a minimum diameter of 5 cm within hours or days.[303] *B. burgdorferi* IgM enzyme-linked immunosorbent assay (ELISA) serology usually does not become positive for 3 to 6 weeks following the development of the rash. The IgG ELISA response follows the IGM response.[114] Weeks, months, or years later, an oligoarthritis or neurologic or cardiac manifestations may develop. The knee is the most commonly involved joint. Because the ESR is usually elevated, septic arthritis is frequently prominent in the differential diagnosis.[82,89] Synovial fluid analysis has revealed nucleated cell counts ranging from 180 to 97,000/mm^3.[106] Fortunately, patients with acute arthritis (as well as neuropathies or meningitis) secondary to Lyme disease usually have positive serology.[334]

Most cases of Lyme disease respond to oral antibiotic therapy with amoxicillin, doxycycline, or ceftriaxone for 10 to 30 days.[82,89,302,334] Rarely, recurrent episodes of arthritis develop following oral antibiotic therapy. Such cases may respond to parental antibiotics.[82] Serious CNS or cardiac symptoms may be treated initially with IV ceftriaxone or penicillin for 14 to 28 days.[114,334] After treatment, *B. burgdorferi* antibodies may persist, and antibody titers should not be used to guide therapy.[114] Antibiotics are not recommended for prophylaxis after tick bites because the risk of developing Lyme disease after a documented tick bite is only 1.2 percent.[320] A vaccination for Lyme disease was recently developed. Its efficacy after three injections over 12 months was reported to be 76 percent. Thus, it is currently recom-

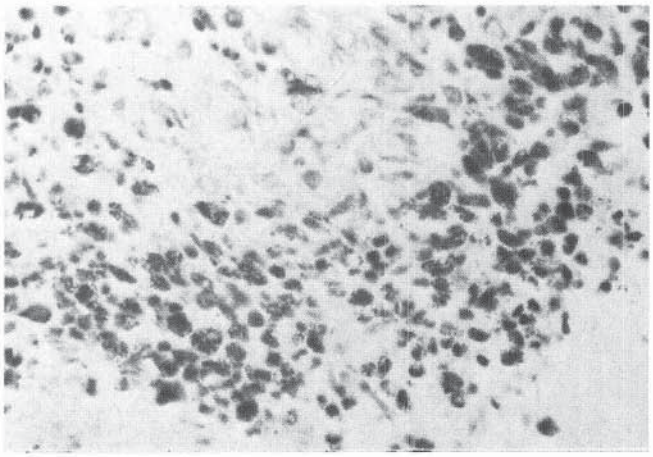


FIGURE 34–19 Ziehl-Neelson stain of a biopsy specimen from a patient with atypical mycobacterial arthritis. Multiple acid-fast bacilli are present, most within histiocytes. (From Bullough PG, Vigorita VJ: Atlas of Orthopaedic Pathology with Clinical and Radiologic Correlations. New York, Gower Medical Publishing, 1984.)

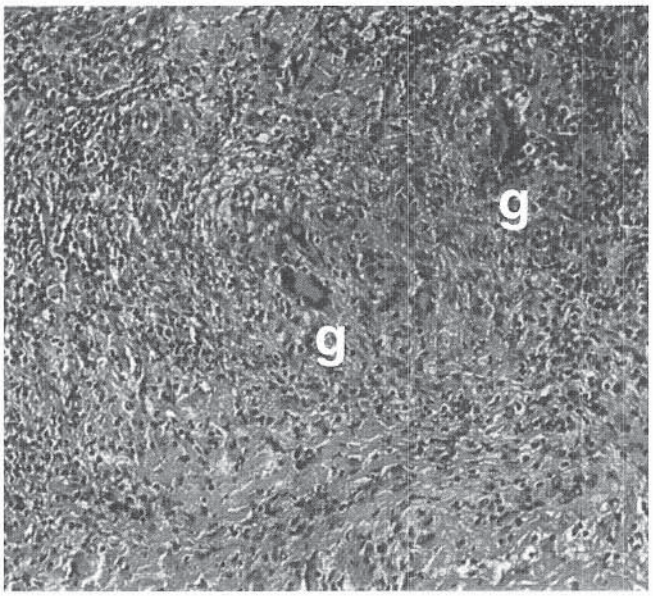


FIGURE 34–20 Micrograph of granulomatous tissue from a patient with tuberculous septic arthritis. Note the chronic inflammation with intermittent well-organized granulomas (*g*). The granuloma is circumscribed by plump, round epithelioid cells that have intermittent Langerhans'-type giant cells, as well as a caseating, necrotic central core. (From Bullough PG, Vigorita VJ: Atlas of Orthopaedic Pathology with Clinical and Radiologic Correlations. New York, Gower Medical Publishing, 1984.)

mended only for individuals at high exposure risk in endemic areas.[336]

Perhaps because of inordinate media attention, Lyme disease appears to be frequently overdiagnosed and mistreated. Ziska and associates documented marked differences between physicians' responses on a questionnaire regarding their practices and published recommendations for the diagnosis and treatment of Lyme disease.[390] Feder and Hunt reviewed the cases of 146 patients referred to a pediatric Lyme disease clinic and found that 38 percent of patients were overdiagnosed and 8 percent were underdiagnosed. Nineteen of the 75 who were correctly diagnosed received inappropriate treatment.[114]

Post-Lyme disease syndrome is characterized by recurrent arthralgias, myalgias, neck pain, headaches, fatigue, and irritability. Its pathogenesis is unknown. It usually resolves spontaneously within 6 months. Treatment with parental antibiotics is not recommended, and oral antibiotic therapy is controversial.[114,334]

Syphilis. With the advent of penicillin, the incidence of infection from *Treponema pallidum* has decreased markedly; however, it remains common in developing countries. Syphilis of bone, which may be either a congenital or an acquired infection, has been reported in up to 65 percent of cases of congenital syphilis.[129]

The organism reaches bone via hematogenous dissemination, and can be found in the bone marrow as early as 36 hours following infection. The pathogens tend to localize in the metaphysis and diaphysis and do not spread to the joints. The most common sites of involvement are the tibia, femur, humerus, and cranial bones; however, any bone may be affected.

Syphilitic metaphysitis is the usual finding in early infancy (Fig. 34–21). Symmetric involvement of multiple bones is characteristic. The physis becomes widened, irregular, and ill-defined. The epiphyses usually are not involved. Pathologic fractures may occur through the weakened metaphyseal area. Necrosis may develop, and frank pus can form if the disease process is not stopped.

In later childhood or adolescence, syphilitic osteoperiostitis produces a dense, circumscribed swelling over the convex side of the bone. In the tibia, the subperiosteal apposition of bone on the anterior cortical surface produces the "saber shin" of congenital syphilis (Fig. 34–22).

The treatment for syphilitic infection of bone is appropriate antibiotic therapy.

Brucellar Osteomyelitis. Brucellar osteomyelitis is caused by several of the *Brucella* species, including *B. abortus* (most commonly seen in North America and Europe), *B. melitensis,* and *B. suis.*[44,80,178,186] Brucellar osteomyelitis is most often seen in farmers, meat packers, and persons who drink unpasteurized milk. Patients may present with pain and tenderness at the site of infection as well as with symptoms of systemic brucellosis such as fever, malaise, weight loss, and leukocytosis. The diagnosis usually is made by identifying a high titer of antibodies in the serum. Patients are treated with a combination of tetracycline and streptomycin.[219] The recurrence rate is high, and chronic brucellar osteomyelitis may require wide surgical excision.[186]

Fungal Osteomyelitis. *Mycotic osteomyelitis* is a general term referring to several rare diseases caused by fungal infection within bone. The most common are coccidioidomycosis, blastomycosis, actinomycosis, and sporotrichosis, which tend to be endemic to specific areas (Fig. 34–23). Other, less common fungal infections that may involve bone are histoplasmosis, maduromycosis, and cryptococcosis. A rare case of primary vertebral osteomyelitis due to *Pseudalles-*

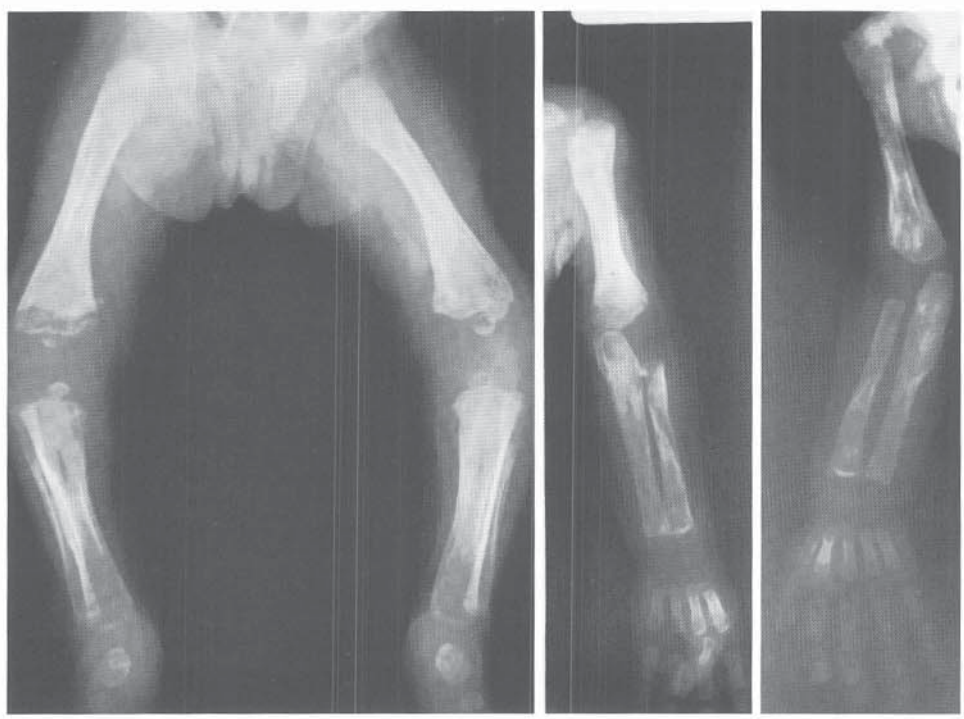


FIGURE 34–21 Congenital syphilis in a 3-month-old girl. Note the characteristic bilateral and symmetric metaphyseal erosions, which have progressed to diffuse osteochondritis with periosteal new bone formation.

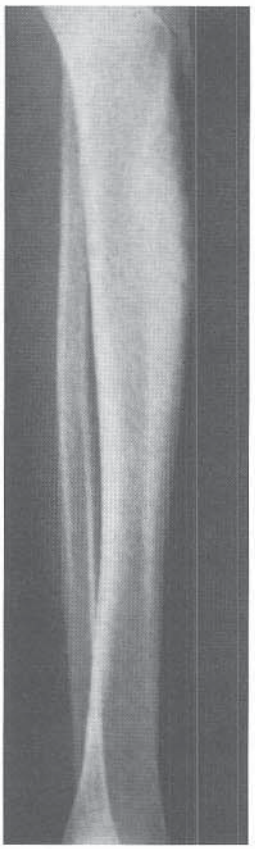


FIGURE 34–22 Classic "saber shin" of congenital lues in an adolescent with untreated congenital syphilis.

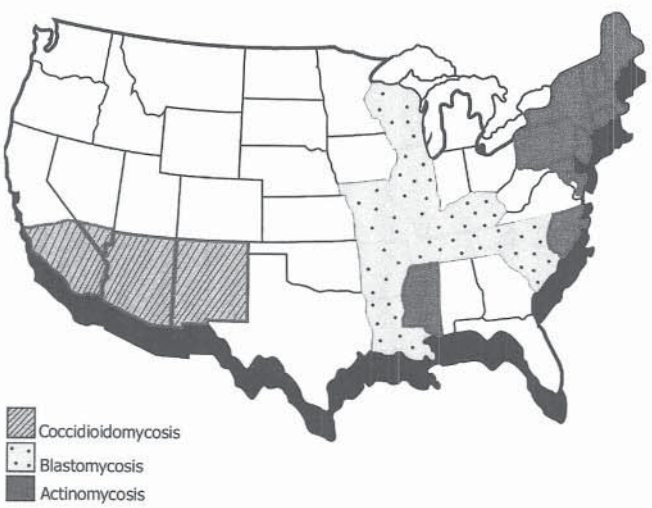


FIGURE 34–23 Endemic areas of fungal infection in the United States.

Coccidioidomycosis: Southwestern United States, particularly the San Joaquin Valley of California.
Blastomycosis: In areas extending from Wisconsin to Louisiana and from the Carolinas to Kentucky.
Actinomycosis: Mississippi, North Carolina, and the northeastern United States.

cheria boydii, an opportunistic fungus, has been reported.[127] Usually the primary infection originates in the soft tissues and later extends directly to bone. Fungal osteomyelitis is often seen in immunocompromised hosts.[98]

The radiographic features of fungal infections of bone are similar to those seen in tuberculosis and pyogenic osteomyelitis.[290] Although myotic osteomyelitis is rare, the various pathogens are more prevalent in some areas of the world and in specific regions of the United States. Thus, fungal infection of the bone should always be included in the differential diagnosis of chronic osteomyelitis.

Mycotic osteomyelitis and septic arthritis are often difficult to treat. Appropriate therapy includes both surgical debridement and systemic antifungal therapy.[37,98,241] Amphotericin B continues to be the preferred treatment for fungal infections.[98] The availability of the triazoles fluconazole and itraconazole has increased the therapeutic options for some mycoses.[234,235]

COCCIDIOIDOMYCOSIS. Coccidioidomycosis, a fungal infection caused by *Coccidioides immitis,* primarily affects the lungs. Dissemination is rare, but the infection does produce bone lesions, some of which are multiple. The diagnosis is made by identifying *C. immitis* spores under the microscope. Serologic and skin tests are not sufficiently specific for diagnosis. Coccidioidomycosis is endemic in the southwestern United States, particularly the San Joaquin Valley of California. Treatment consists of systemic amphotericin B and surgical excision of skeletal lesions when feasible.[34,241]

BLASTOMYCOSIS. Blastomycosis is a rare disease that primarily affects the skin and lungs;[11,30,147] however, bones and joints may be involved, either from direct extension of an adjacent soft tissue infection or by hematogenous spread.[218] The diagnosis is made by biopsy, direct smear, culture, skin testing, and serology. In the United States, blastomycosis is endemic in an area that extends from Wisconsin to Louisiana and from the Carolinas to Kentucky. Treatment is with antifungal agents such as amphotericin B.

ACTINOMYCOSIS. Actinomycosis is a chronic infection caused by the organism *Actinomycosis israeli* that usually involves the soft tissues of the head and neck, and may spread to the lungs and large intestine.[55,90,215,228] Bone becomes involved by direct extension. In North America, actinomycosis is endemic in Mississippi, North Carolina, and the northeastern United States. Patients are treated with long-term administration of penicillin.

SPOROTRICHOSIS. Sporotrichosis is a chronic granulomatous infection caused by the organism *Sporotrichum schenckii* that primarily affects the skin and subcutaneous tissues. Bone involvement may be by direct extension from a skin lesion or through hematogenous spread. Sporotrichosis may be associated with sarcoidosis or tuberculosis, and is treated with antifungal chemotherapy.

Viral. Variola (smallpox) was previously the most common cause of viral osteomyelitis. Detailed reviews of osteomyelitis variolosa have been published by Cockshott and MacGregor[74] and Davidson and Palmer.[88] However, in 1980 the World Health Organization officially documented the global eradication of smallpox.[194,366] Thus, viral osteomyelitis has become extremely rare. Bone lesions have been reported

following vaccination against smallpox[73] and as a complication of cat-scratch fever.[1,76]

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