A wide variety of disorders of the musculoskeletal system are manifested clinically by the phenomenon of inflammation and are therefore best considered as a broad group in relation to this basic pathological process. For some of these clinical disorders, such as osteomyelitis and septic arthritis, a specific causative microorganism can be incriminated; however, for others, such as ankylosing spondylitis and rheumatoid arthritis, the exact cause remains an unsolved and challenging mystery.

Before learning about the various disorders as clinical entities, you will find it helpful to review some of the general features of the inflammatory process and the reactions of the musculoskeletal tissue to this process.

THE INFLAMMATORY PROCESS: GENERAL FEATURES

Inflammation, a process of biological events, is best defined as “the local reaction of living tissues to an irritant” (Boyd). In this reactive process, cells and exudates accumulate in the irritated tissue and usually (but not invariably) tend to protect them from further injury. Once considered a disease entity in itself, inflammation is now known to be a tissue response, or reaction, to any one of many types of irritants. The four clinical manifestations of inflammation originally described by Celsus are rubor, tumor, calor et dolor (redness, swelling, heat, and pain). To these Galen later added a fifth—funicio laesa (loss of function). These five clinical manifestations are readily explained by the nature of the inflammatory process.

The redness and the heat are caused by the vascular response, namely, a dilatation of local blood vessels combined with an increased rate of flow. The swelling represents the formation of an exudate that results from the combination of increased hydrostatic pressure within the capillaries and increased capillary permeability. Added to this inflammatory exudate is the emigration of various types of leukocytes from the capillaries. The pain, which is most severe in the acute type of inflammatory process, is related to the marked increase in local pressure within the tissues. When the inflammatory process develops in a closed space, such as a bone or synovial joint, it is easy to understand why the pain may be severe. The initial loss of function of the involved part results from pain and swelling; however, subsequent loss of function may result from a combination of actual destruction of tissue, such as articular cartilage, and dense scar formation in soft tissues.

In the central zone of the inflammatory process, local tissue necrosis and liquefaction are frequently seen. By contrast, the reaction in the peripheral zone is hyperplasia of connective tissue cells, a reaction that initially serves to localize the process and subsequently aids in the repair of the inflammatory lesion.

REACTIONS OF THE MUSCULOSKELETAL TISSUES TO INFLAMMATION

Each specialized type of tissue in the body reacts in a characteristic way to the general process of inflammation. Thus, a knowledge of the characteristic reactions of the various musculoskeletal tissues will enhance your understanding not only of the clinical, radiographic, and laboratory manifestations of inflammatory musculoskeletal disorders in your patients but also of the underlying reason for the principles and methods of their treatment. The characteristic reactions to infection and other types of inflammation in bone, epiphyseal plate, articular cartilage, synovial membrane, capsule, and ligaments are discussed and illustrated in Chapter 3. They are of sufficient importance that you may wish to review these reactions in Chapter 3 before proceed-
ing to a discussion of the various clinical disease entities that result from inflammation of musculoskeletal tissues.

**TYPES OF INFLAMMATORY DISORDERS OF BONES AND JOINTS**

The various musculoskeletal disorders discussed in this chapter have in common as their most prominent feature the phenomenon of inflammation. They are best considered in four broad groups.

First is the group of *specific infections* for which causative organisms can be detected. Of these, many are *pyogenic* (pus-producing) infections, such as osteomyelitis, septic arthritis, and tenosynovitis. Others are *granulomatous* (granuloma-producing) infections, such as tuberculous osteomyelitis and tuberculous arthritis.

A second broad group of inflammatory disorders includes the nonspecific and idiopathic *inflammatory types of rheumatic diseases*, which include entities such as rheumatic fever, transient synovitis, rheumatoid arthritis, and ankylosing spondylitis.

A third group includes inflammation of musculoskeletal tissue secondary to a chemical irritant, as seen in the form of *metabolic arthritis* known as gout.

A fourth group is characterized by chronic inflammation caused by *repeated physical injury*—now known as *chronic repetitive strain injury*—usually minor injury (microtrauma), or mechanical irritation. Bursitis and tenosynovitis stenosans, which are examples of this type of inflammation secondary to chronic repetitive strain injury, are described in Chapter 11.

**Pyogenic Bacterial Infections**

Pyogenic bacterial infections in bones and joints continue to represent a serious threat to both life and limb. Although chemotherapeutic and antibiotic drugs have dramatically reduced the mortality of the various pyogenic infections involving the musculoskeletal system, the incidence of these infections and their morbidity have been less dramatically reduced. Indeed, drug therapy may mask the clinical manifestations of infection without completely controlling the local lesion, thereby creating an altered clinical picture.

**Principles of Antibacterial Therapy**

Acute pyogenic infection is an exceedingly rapid process measured in hours and days. Thus, even a short delay in treatment may lead to serious consequences for the patient. Antibiotics such as tetracycline, chloramphenicol, and erythromycin exert their effect on the metabolism of bacteria and thereby greatly decrease their rate of multiplication; their action, therefore, is bacteriostatic. Other antibacterial drugs, such as the penicillins and cephalosporins, actually kill bacteria and hence are *bactericidal*.

To control an infection, the concentration of the appropriate antibiotic in the blood and at the site of infection must exceed the level necessary to kill the infecting organism. The ideal antibiotic is *bactericidal* (as opposed to bacteriostatic), should be known to be effective against the most likely infecting bacteria, must reach the infected tissues in high concentrations (which can be difficult in bone), should be nontoxic, and should have little effect on the normal flora. The parenteral (intravenous or intramuscular) route of administration is more effective than the oral route in achieving adequate serum and tissue levels of the antibiotic and is therefore preferable in the initial treatment, especially if the patient is unable to take medications by mouth.

Since patients vary in their response to antibiotics and since the infecting organisms vary in their resistance, both clinical and laboratory monitoring of the patient are essential. An effective laboratory method of such monitoring is the weekly determination of the serum bacterial titer.

Antibacterial therapy must be continued for a longer period to control infection in bone than in soft tissues in order to achieve a permanent cure and thereby prevent either chronic or recurrent infection. Empirically, this period is from 3 to 4 weeks.

The relatively slow diffusion of antibacterial agents into the area of bacterial inflammation is dependent on an intact local blood supply. When the local pressure within the inflamed
Acute Hematogenous Osteomyelitis

One of the most serious inflammatory disorders of the musculoskeletal system is acute hematogenous osteomyelitis, a rapidly developing blood-borne bacterial infection of bone and its marrow in children.

Incidence

At the beginning of the era of specific antibacterial drug therapy, there was a sharp fall in the incidence of acute hematogenous osteomyelitis; indeed, some clinicians optimistically predicted the eradication of this disease. Subsequently, however, the incidence returned almost to its former level. This phenomenon—which has been paralleled by bacterial infections involving other tissues—is explained by the combination of the emergence of resistant strains of bacteria (especially staphylococci) and the failure of too many clinicians to understand and apply the principles of antibacterial and surgical therapy in relation to bone and joint infections.

Hematogenous osteomyelitis is primarily a disease of growing bones and, therefore, of children; boys are afflicted three times as often as girls. The long bones most frequently involved (in order of decreasing frequency) are the femur, tibia, humerus, radius, ulna, and fibula, and the characteristic site in any given bone is the metaphyseal region—possibly because of the unique blood vessels and low-flow state to this part of the bone during childhood.

Etiology

Staphylococcus aureus is by far the most common causative organism, being responsible for at least 90% of acute hematogenous osteomyelitis cases. The portal of entry is usually through the skin secondary to infected scratches, abrasions, pimples, or boils; sometimes it is through the mucous membranes of the upper respiratory tract as a complication of a nose or throat infection. Even vigorous brushing of the teeth in the presence of inflamed gums can result in transient bacteremia. In the presence of bacteremia, local trauma seems to play a significant role in determining the particular bone in which osteomyelitis develops (perhaps because of local thrombosis and hence decreased resistance to infection); this may account, in part, for the higher incidence in boys and also in the lower extremities. Streptococcus or Pneumococcus may on occasion be the offending bacteria, particularly in infants. Hemophilus influenzae has almost been eliminated as a cause of osteomyelitis by the development of an effective vaccine.

Pathogenesis and Pathology

The early and rapid development of untreated hematogenous osteomyelitis is characterized by an initially small focus of bacterial inflammation with early hyperemia and edema in the cancellous bone and marrow of the metaphyseal region of a long bone (Fig. 10.1). Unlike soft tissues, which are capable of expanding
to accommodate swelling, the bone represents a rigid closed space; therefore, the early edema of the inflammatory process produces a sharp rise in the intraosseous pressure, which explains the symptom of severe and constant local pain. Pus forms, thereby increasing the local pressure even further with resultant compromise of the local circulation which, in turn, leads to vascular thrombosis and consequent necrosis of bone.

The untreated infection rapidly spreads by several routes, destroying bone in its path by osteolysis (Fig. 10.2). Through damaged vessels in the local lesion, large numbers of bacteria re-invade the bloodstream; the clinically undetectable bacteremia becomes a septicemia, which is manifest by the onset of malaise, anorexia, and fever. Local spread of the infection by direct extension, aided by increased local pressure, penetrates the relatively thin cortex of the metaphyseal region and involves the highly sensitive periosteum, which accounts for the exquisite local tenderness. The periosteum, being loosely attached to bone during childhood, is readily separated and elevated from the bone. The result is a subperiosteal abscess that may either remain localized or spread along and around the entire shaft of the bone; such elevation of the periosteum disrupts the blood supply to the underlying cortex, thereby increasing the extent of bone necrosis.

After the first few days, the infection penetrates the periosteum to produce a cellulitis and eventually a soft tissue abscess. In sites where the metaphyseal region is within the synovial joint, as in the upper end of the femur and the upper end of the radius, penetration of the periosteum carries the infection directly into the joint, with resultant septic arthritis (Fig. 10.3). In other sites where the metaphyseal region is outside but close to the joint, a sterile synovial effusion frequently develops.
Meanwhile, local spread of the infection within the medullary cavity further compromises the internal circulation. The resultant area of bone necrosis, which may vary in extent from a small spicule to the entire shaft, eventually becomes separated, or sequestrated, from the living bone, thereby forming a separated fragment of infected dead bone, a *sequestrum*. Extensive new bone formation from the deep layer of the elevated periosteum produces an enveloping bony tube, or *involution*, which maintains the continuity of the involved bone, even when large segments of the shaft have died and sequestrated (Fig. 10.2). The epiphyseal plate usually acts as a barrier to direct spread of infection, but if it is damaged in the process, a serious growth disturbance will become apparent at a later date.

If uncontrolled, the septicemia may produce metastatic foci of infection in other bones at any time; more important; it may produce these foci in other organs, particularly the lungs and the brain. Indeed, in the days before antibacterial drugs, 25% of all children with acute hematogenous osteomyelitis died of the associated septicemia. If the child survives the septicemia, the local bone lesion—unless adequately treated—gradually passes into a chronic state. Chronic osteomyelitis, which is perpetuated by the presence of infected dead bone, is discussed in a subsequent section of this chapter.

**Clinical Features and Diagnosis**

The clinical features of acute hematogenous osteomyelitis are readily correlated with the foregoing description of its pathogenesis. The onset is acute and the infection progresses with remarkable rapidity. There is a history of recent local injury in 50% of the children; frequently you will find evidence of a pre-existing bacterial infection either in the skin or in the upper respiratory tract.

The first and most significant symptom the afflicted child experiences is severe and constant pain near the end of the involved long bone—this is accompanied by exquisite local tenderness and the child's unwillingness to use the limb (Fig. 10.4). Within 24 hours, the associated septicemia is evidenced by malaise, anorexia, and fever; the child appears acutely ill. Increasing pain and local tenderness near the end of a long bone, combined with systemic manifestations of infection, in a child
always justify the *clinical* diagnosis of acute hematogenous osteomyelitis—at least until there is definite evidence to the contrary. Soft tissue swelling is a relatively late sign appearing only after a few days and indicating that the infection has already spread beyond the confines of the bone (Fig. 10.5).

It is extremely important for you to appreciate that the early diagnosis of acute hematogenous osteomyelitis must be made on *clinical* grounds alone. During at least the first week of illness, there is absolutely no concrete radiographic evidence of bone infection, despite severe local involvement of bone. There may be radiographic evidence of soft tissue swelling after the first few days (Fig. 10.6). Such swelling can also be detected by ultrasonography. However, only after the first week does the radiograph reveal the first evidence of destruction of bone in the metaphysis and the first signs of reactive new bone from the periosteum (Fig. 10.7). During this first week before radiographic changes become apparent, scintigraphy—that is, a bone scan—may be of value in establishing the diagnosis. With magnetic resonance imaging (MRI), the combination of a dark focus on T1-weighted im-

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**Figure 10.4.** This boy has early acute hematogenous osteomyelitis of the upper end of the left tibia; he is unable to bear weight on his left foot and is unwilling to move his knee. He is able to localize the point of pain and tenderness very accurately.

**Figure 10.5.** Soft tissue swelling secondary to osteomyelitis of the right tibia in a child. This child had severe pain in the right leg for 7 days prior to this photograph. The infection has already spread from the bone into the soft tissues to produce an extensive cellulitis.

**Figure 10.6.** Radiographic evidence of soft tissue swelling secondary to acute hematogenous osteomyelitis. A. Normal lower end of the femur of a child. B. Soft tissue swelling posterior to the lower end of the opposite femur in the same child with acute hematogenous osteomyelitis 3 days after the onset of symptoms. At this stage there is no evidence of bone destruction.

**Figure 10.7.** Radiographic evidence of bone destruction in the metaphyseal region of the lower end of the femur in a child with acute hematogenous osteomyelitis of 10 days' duration. Note also the evidence of subperiosteal new bone formation along the shaft of the femur in the lateral projection.
ages and a bright signal on T2-weighted images is consistent with osteomyelitis.

In infants, the systemic manifestations of infection are often less apparent than they are in children. Furthermore, the localization of the osteomyelitis is obviously more difficult because of the lack of communication and requires careful examination of all the major long bones and joints.

The white blood cell count and the sedimentation rate are usually elevated, but despite the underlying bacteremia, and the later septicemia, a single blood culture gives positive results in only about half of the patients.

The clinical manifestations of acute hematogenous osteomyelitis—particularly the systemic manifestations—may be masked during the first few days of the illness by the casual and speculative use of inadequate antibacterial therapy for what is loosely considered “a little infection.” This deplorable type of management obscures the true diagnosis until irreparable changes in the bone have developed and the local infection has progressed relentlessly to chronic osteomyelitis (Fig. 10.8).

In its early stages, acute hematogenous osteomyelitis must be differentiated from rheumatic fever, cellulitis of soft tissues, and local trauma to soft tissues or bone. After the first week or more, particularly if the systemic manifestations have been masked by antibacterial drugs, the radiographic changes of irregular

**Figure 10.8.** Relentless progression from acute hematogenous osteomyelitis to chronic osteomyelitis. This series of radiographs of a girl’s forearm extends over a period of 10 years and demonstrates many of the radiographic changes of acute and chronic osteomyelitis of the ulna. A. One week after the onset of symptoms. There is soft tissue swelling, a small area of destruction in the metaphyseal region of the distal end of the ulna, and beginning new bone formation along the shaft of the ulna. This child had been thought to have a “a little infection” and had received a small amount of antibacterial therapy for a few days. At the end of 1 week her arm was significantly swollen and tender. The infection had already spread throughout the length of the ulna and at this stage even extensive therapy could not eradicate all the infection. B. Ten days later there is evidence of further destruction in the ulna and more subperiosteal new bone formation. C. One month later, there is involucrum formation and sequestration of the distal third of the ulna. At this stage, the child was still ill and in pain. Consequently, the large sequestrum was removed. D. Eight months later, there is still chronic osteomyelitis; there is a pathological fracture in that portion of the lower end of the ulna that has reformed from the deep surface of periosteaum. E. Three years later, there is evidence of premature cessation of growth at the distal ulnar epiphyseal plate secondary to the infection. There is still marked thickening of the proximal two thirds of the ulna because of residual chronic osteomyelitis. F. Ten years after the onset of the osteomyelitis, there is a small abscess in the upper end of the ulna and additional evidence of chronic osteomyelitis in the entire upper third of the ulna. This relentless progression from acute hematogenous osteomyelitis to chronic osteomyelitis could have been prevented by early adequate treatment.
metaphyseal rarefaction and subperiosteal new bone formation can mimic bone lesions such as Langerhans cell histiocytosis (eosinophilic granuloma), Ewing’s sarcoma, and osteosarcoma.

Treatment
Acute hematogenous osteomyelitis represents an extremely serious infection that demands urgent and vigorous treatment. As soon as the clinical diagnosis is strongly suspected on the basis of the previously mentioned symptoms and signs, the child should be admitted to hospital for intensive treatment. As soon as one blood sample has been taken for culture to seek the causative bacteria as well as its sensitivity to the various antibacterial drugs, antibacterial therapy is instituted. Since the incidence of bacterial resistance to antibiotics continues to increase and because the bacterial environment varies not only from one locality to another but also from year to year, the choice of the specific drug to be used initially will depend on existing conditions in your locale at the time. Nevertheless, general guidelines can be stated.

Currently, penicillin is still the safest antibiotic drug, but in many communities more than 70% of the staphylococci are penicillin-resistant. Therefore, at least initially, one of the newer antibiotics such as cloxacillin should be given for older children or, alternatively, one of the cephalosporins such as cefotaxime for neonates and cefuroxime for young children (all of which are effective in the presence of penicillinase). As soon as the culture and sensitivity results are known, antibiotic therapy can be modified appropriately if necessary. A consultant in the rapidly changing field of infectious diseases can be of much help in advising about the antibacterial therapy for these patients.

The following general plan of treatment has been found to be most effective:

1. Provide bed rest and analgesics for the child.
2. Supportive measures are given, including, when necessary, intravenous fluids.
3. Local rest for the involved extremity is provided by either a removable splint or traction to reduce pain, retard the spread of infection, and prevent soft tissue contractures.
4. For a child too sick to take drugs by mouth, immediate parenteral administration of appropriate antibacterial therapy (as soon as a blood sample has been taken for culture) is necessary, not only to control the bacteremia and sepsis but also to reach the area of osteomyelitis before it has become ischemic and therefore inaccessible to the circulating drug. For a child who is able to take drugs by mouth, oral administration of the antibiotic is an acceptable alternative from the beginning. After the first 2 weeks (provided that there has been a good clinical response), the antibiotic may be given orally (which has been proved effective and is certainly more comfortable for the child).
5. If local and systemic manifestations have not improved dramatically after 24 hours of intensive treatment, surgical decompression of the involved area of bone (evacuation of subperiosteal pus, drilling of bone) is performed to reduce the intraosseous pressure and to obtain pus for culture. Postoperatively, continuous local infusion of saline with an appropriate antibiotic, combined with drainage, may be required for severe infections for at least a few days (Fig. 10.9).
6. Antibacterial therapy is continued for a minimal period of 3 to 4 weeks, even if clinical improvement during the first few days has been satisfactory. (After 3 to 4 weeks, treatment is discontinued only when the sedimentation rate begins to approach a normal level.)

Prognosis
Four important factors determine the effectiveness of antibacterial treatment for acute hematogenous osteomyelitis and consequently its prognosis:

1. The time interval between the onset of infection and the institution of treatment. Treatment begun during the first 3 days of illness is ideal because at this stage the local area of osteomyelitis has not yet become
ischemic. Such early treatment, provided that the causative organism is sensitive to the drug chosen, usually controls the infection completely so that osteolysis, bone necrosis, and reactive new bone formation are prevented; under these circumstances, radiographic changes in the bone may not appear later (Fig. 10.10).

Treatment begun between 3 and 7 days usually attenuates the infection both systemically and locally, but is too late to prevent bone destruction (Fig. 10.11).

Treatment instituted after the first week of illness may control the septicemia and therefore still be lifesaving, but it has little effect on the relentless progression of the local pathological process within the bone (Fig. 10.8).

2. The effectiveness of the antibacterial drug against the specific causative bacteria. This depends on whether the bacteria is sensitive to the drug or resistant to it and emphasizes the importance of culture and sensitivity studies.

3. The dosage of the antibacterial drug. The local factor of compromised circulation within the area of bone infection necessitates much larger doses of antibacterial drugs for osteomyelitis than for soft tissue infections.

4. The duration of antibacterial therapy. Premature cessation of therapy, especially less than 3 to 4 weeks, frequently results in either chronic or recurrent osteomyelitis.

Complications of Acute Hematogenous Osteomyelitis
The early complications of acute hematogenous osteomyelitis include 1) death from the associated septicemia, 2) abscess formation, and 3) septic arthritis, especially in the hip joint.

The late complications include 1) chronic osteomyelitis, either persistent or recurrent; 2) pathological fracture through a weakened area of bone; 3) joint contracture; 4) local growth disturbance of the involved bone, either overgrowth from the stimulation of prolonged hy-
current chronic form of osteomyelitis are exceedingly difficult to eradicate.

Incidence
The continuing prevalence of chronic hematogenous osteomyelitis testifies to the frequent failure to diagnose acute osteomyelitis within the first few days of onset as well as the failure to provide effective antibacterial therapy and the failure to intervene surgically, when indicated, in the acute phase.

Pathogenesis and Pathology
The most significant pathological lesion in the chronic phase of hematogenous osteomyelitis, and the one that prevents its spontaneous resolution, is infected dead bone. Unlike a segment of sterile dead bone, which is gradually revascularized, resorbed, and replaced by living bone, infected dead bone always separates, or sequestrates, from the remaining living bone and thus becomes a sequestrum. Bacte-

Figure 10.11. Right tibia of a child with acute hematogenous osteomyelitis 5 weeks after the onset of infection. Treatment had been started 5 days after the onset of symptoms, and although the infection had been controlled systemically, the treatment was started too late to prevent bone destruction. Note evidence of destruction in the distal two thirds of the tibia and also the subperiosteal new bone formation.

Chronic Hematogenous Osteomyelitis
Inadequate treatment of the acute phase of hematogenous osteomyelitis allows the local pathological process either to persist and become chronic or to become relatively quiescent for a time, only to recur at a later date. Both the persistent chronic form and the re-

Figure 10.12. Local growth disturbance in the involved bone complicating osteomyelitis. A. Overgrowth of the right tibia in a 14-year-old girl with chronic osteomyelitis involving the distal end of the tibia. The infection has been chronic for 5 years. B. Premature cessation of growth in the left lower femoral epiphysis complicating osteomyelitis in early childhood. In this full-length radiograph, a severe leg length discrepancy is apparent.
ni are able to survive and continue to multiply within the tiny haversian canals and canaliculi of this island of avascular bone; the surrounding pond of pus prevents revascularization of the sequestrum and thereby protects its bacterial inhabitants not only from the living leukocytes of the defensive inflammatory reaction but also from the action of circulating antibacterial drugs. Furthermore, in the absence of revascularization, the living process of osteoclastic resorption of dead bone cannot reach the sequestrum. As a result, the sequestrum persists as a haven for bacteria and a source of either persistent or recurrent infection. Thus, the infection cannot be permanently eradicated until all sequestra have been eliminated, either by the natural process of spontaneous extrusion through an opening (cloaca) in the involucrum and thence through a sinus tract to the exterior (Fig 10.13), or by surgical removal (sequestrectomy). An area of persistent infection within cancellous bone may eventually become walled off from the surrounding bone by fibrous tissue to form a chronic abscess (Brodie's abscess).

Clinical Features and Diagnosis
The child, having recovered from the sepsisemia of the acute phase, is no longer acutely ill but has a residual painful lesion in the involved long bone associated with swelling, tenderness, and loss of function of the limb; there may be one or more draining sinuses (Fig. 10.13).

The radiographic diagnosis is usually apparent, particularly in the presence of obvious sequestra (Fig. 10.14). Nevertheless, the combination of local rarefaction, sclerosis, and periosteal new bone formation may mimic other bone lesions such as osteosarcoma, Ewing's sarcoma, and Langerhans cell histiocytosis (eosinophilic granuloma). The radiographic appearance of a Brodie's abscess is not unlike that of an osteolytic bone neoplasm (Fig. 10.14). In the presence of a draining sinus, a sinogram often helps locate the site of underlying infection (Fig. 10.15).

Persistent anemia and elevation of the sedimentation rate reflect the chronic infection.

Treatment
Chronic osteomyelitis can seldom be completely eradicated until all the infected dead bone has separated, or sequestrated, and has either been extruded spontaneously through a sinus track or been removed surgically (sequestrectomy). In the absence of clinical evidence of local and systemic infection, a small sequestrum may be resorbed.

Antibacterial therapy is required both systematically and locally. A residual abscess cavity within the bone usually necessitates an operation in which one surface of the tubular bone is removed to make it open like a saucer (sauzerization). Following either sequestrectomy or saucerization, antibacterial drugs in saline solution are instilled into the area by continu-

Figure 10.14. A and B. Residual chronic osteomyelitis with several small sequestra in the lower end of the femur of a 40-year-old woman who had acute hematogenous osteomyelitis in this site at 10 years of age. C. Brodie's abscess in the distal end of the ulna in a young adult. The osteolytic lesion is not unlike that of an osteolytic bone neoplasm.
Incidence
The incidence of septic arthritis parallels that of hematogenous osteomyelitis with which it is so frequently associated. Septic arthritis, therefore, is primarily a disease of childhood. Newborn infants are particularly susceptible, especially those who have an immunodeficiency, as suggested by Lloyd-Roberts. During childhood, the most common sites are those in which the metaphysis of the bone is entirely intracapsular, namely, the hip and the elbow (Fig. 10.3). In adult life, septic arthritis can develop in any joint because it is unrelated to osteomyelitis.

Etiology
The spread of pyogenic bacteria from hematogenous osteomyelitis in the metaphysis directly into the joint is the most common source of septic arthritis in children. Consequently, as in osteomyelitis, the most frequent causative organism is S. aureus. However, bacteria, particularly streptococci and pneumococci and less commonly Salmonella, may reach the joint by the bloodstream to produce hematogenous septic arthritis. In adults, staphylococci, pneumococci, and gonococci may also invade a synovial joint by the hematogenous route as a complication of systemic infection. Human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS), as well as intravenous drug use and prolonged adrenocorticosteroid therapy are risk factors for the development of septic arthritis.

Complications
The complications of persistent chronic osteomyelitis include 1) joint contracture, 2) pathological fracture, 3) amyloid disease, and 4) malignant changes in the epidermis (epidermoid carcinoma) of a sinus tract in which infection has been allowed to persist for many years.

Acute Septic Arthritis (Pyogenic Arthritis)
When pyogenic bacteria invade a synovial joint, the result is acute septic (pyogenic) arthritis, a rapidly progressive infection that, unless adequately treated, leads to severe destruction of the joint.

Pathogenesis and Pathology
Acute septic arthritis is an extremely serious infection because the purulent exudate—particularly that of staphylococci—rapidly digests articular cartilage. The mechanism of initial cartilage destruction involves enzymatic digestion of the matrix by lysosomal enzymes from both polymorphonuclear leukocytes and bacteria. As a result, the collagen fibers lose their support and the cartilage disintegrates. Granulation tissue may creep over the articular cartilage as a pannus, blocking its nutrition from synovial fluid and thereby leading to even further destruction. Since cartilage is virtually incapable of regeneration under ordi-
Clinical Features and Diagnosis
The clinical manifestations of acute septic arthritis in infants are significantly different from those in older children or adults and consequently are best considered separately.

Septic Arthritis in Infants
During infancy, particularly in the newborn period, acute septic arthritis may develop with few clinical manifestations other than irritability and the infant's reluctance to move the affected joint, with resultant "pseudoparalysis." Local examination reveals tenderness over the joint and obviously painful restriction of joint motion (Fig. 10.16). Fever and elevation of the white blood cell count are misleadingly slight in this age group, and unless the major joints of the limbs are examined daily during any febrile illness, the diagnosis of septic arthritis may not be made sufficiently early to prevent avascular necrosis of the femoral head and irreparable damage to the joint. Clinical suspicion of acute septic arthritis is an urgent indication for immediate needle aspiration of the joint as a valuable diagnostic procedure and as a means of obtaining fluid from the joint for a Gram stain and culture.

Radiographic examination and also ultrasonography during the first week may reveal evidence of soft tissue swelling, but not until the second week is there evidence of a pathological dislocation (Fig. 10.17). Equally delayed are the radiographic changes of osteomyelitis in the intracapsular part of the metaphysis (Fig. 10.18).

Septic Arthritis in Older Children and Adults
Unlike the uncommunicative infant, the older child or adult with septic arthritis is able to tell you of severe pain in the region of the involved joint and, furthermore, that the pain is made much worse by even the slightest movement in the joint. Clinical signs include protective spasm in the muscles controlling the joint, marked tenderness and, when the involved joint is superficial, an obvious effusion. The systemic manifestations of infection and elevation of temperature, white blood cell count, and sedimentation rate are more
Radiographic findings in the older age group are comparable to those seen in infants, although pathological subluxation is more common than dislocation. Only after considerable destruction of articular cartilage is there evidence of a narrowed cartilage space (Fig. 10.19).

**Treatment**

Acute septic arthritis represents a surgical emergency that demands early and vigorous treatment to preserve normal joint function. The general plan of treatment, including antibacterial drugs, is similar to that described, in a previous section of this chapter, for acute hematogenous osteomyelitis, with the addition of specific local treatment for the joint itself. Although needle aspiration of an infected joint is of the utmost importance in establishing the diagnosis and obtaining the causative organism, the therapeutic regimen of repeated aspiration and instillation of antibacterial drugs is seldom sufficient to control septic arthritis; after the first few days, the pus has become too thick to be completely removed even through a large-bore needle. Nevertheless, arthroscopic lavage is effective for the knee joint.

For more effective treatment for other joints (especially the hip joint) is the operation of opening and exploring the joint (arthrotomy) with complete removal of the pus and thorough irrigation of the joint. The wound may be closed, but continuous local infusion of saline with an appropriate antibacterial drug

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**Figure 10.18.** Late metaphyseal changes in the neck of the femur associated with septic arthritis of the hip. A. One month after the onset of septic arthritis of the left hip in an infant. Note the pathological dislocation of the left hip and marked metaphyseal changes in the neck of the femur. B. Sequelae of acute septic arthritis of the hip in a 14-year-old girl. Note the marked destruction of the upper end of the left femur that has resulted from acute septic arthritis of the hip in infancy. This girl's hip, which is also severely subluxated, is seriously damaged and will require reconstructive operations.

**Figure 10.19.** Septic arthritis of the left hip in a 13-year-old girl. A. This radiograph, taken 1 month after the onset of symptoms, shows that the cartilage space is narrowed and the hip has subluxated slightly. Note also the rarefaction in the neck of the femur. B. The same hip 2 months later shows further changes in the neck of the femur and radiographic evidence of avascular necrosis of the femoral head. This girl's hip is irreparably damaged.
should be combined with drainage for at least a few days, until the fluid being drained from the joint is sterile.

When septic arthritis of the hip in an infant is complicated by a pathological dislocation, the dislocated hip should be reduced and the hip immobilized in a stable position. In the absence of a pathological dislocation and in all other sites of septic arthritis, the infected joint should be allowed to move in an attempt to prevent complications such as intra-articular adhesions and progressive destruction of cartilage. Indeed, in an experimental model of acute septic arthritis of the knee in rabbits, we have found that continuous passive motion (CPM) has a protective effect on articular cartilage (Salter et al.).

Most gonococcal arthritis in adults is resistant to penicillin and requires a parenteral β-lactamase-resistant cephalosporin. Treatment of the late sequelae of septic arthritis involves various types of reconstructive operations. Often the residual damage of inadequately treated septic arthritis is so severe that surgical fusion (arthrodesis) of the joint is necessary to relieve pain, provide stability, and correct deformity—but at the cost of permanent loss of joint motion (Fig. 10.20).

**Prognosis**

The four important factors that determine the effectiveness of treatment for acute septic arthritis are the same four factors outlined for acute hematogenous osteomyelitis in a previous section of this chapter.

You will appreciate, however, that inadequately treated septic arthritis of a major joint, especially the hip, leads to an even more significant and more permanent disability for the patient than does inadequately treated osteomyelitis.

**Complications of Acute Septic Arthritis**

The early complications of acute septic arthritis include 1) *death* from the associated sepsisemia, 2) *destruction of joint cartilage*, 3) *pathological dislocation* of the joint (especially in infants), and 4) *avascular necrosis of the epiphysis*, particularly in the hip.

The late complications are the sequelae of a destroyed joint and include 1) *degenerative joint disease*, 2) *permanent dislocation with a false joint*, 3) *fibrous ankylosis*, and 4) *bony ankylosis*.

**Hematogenous Osteomyelitis of the Spine**

Acute hematogenous osteomyelitis of the spine differs sufficiently from osteomyelitis of the long bones that it merits separate consideration.

The vertebrae may become involved by acute osteomyelitis at any age, but young children are afflicted more often than are older children or adults. In young children, the condition is sometimes referred to by the somewhat misleading term *benign osteitis of the spine* because the systemic manifestations of the disease are relatively mild and there is little suppuration. Another, more descriptive term is *spondylarthritis*, which signifies that in addition to the bone of the vertebral bodies, the adjacent intervertebral disc is invariably involved and partially destroyed.

The most common sites are the vertebrae of the lower thoracic and upper lumbar spine, which raises the suspicion that the route of infection may be via Batson’s plexus of paravertebral veins. *Staphylococcus aureus* and *Escherichia coli* are the most frequent causative organisms.
Systemic manifestations include irritability and loss of appetite, but fever is usually mild. The white blood cell count is frequently normal, but the sedimentation rate is always elevated.

Radiographic examination of the spine within the first 2 weeks of illness fails to reveal any bony abnormality, but during this period a bone scan may be helpful (as discussed in Chapter 5). Subsequently, narrowing of the adjacent intervertebral disc space and osteolysis of the involved vertebrae become obvious (Fig. 10.22).

The most important differential diagnosis is spinal tuberculosis, which can be excluded if the tuberculin skin test result is negative. Vertebral punch biopsy (under anesthesia and with radiographic control) may be necessary.

**Clinical Features and Diagnosis**

In childhood, the first symptom is poorly localized back pain accompanied by the physical signs of protective muscle spasm in the back and local deep tenderness. There may be signs of meningeal irritation (painful limitation of neck flexion and straight-leg raising). The child is frequently reluctant to sit up or stand and is always reluctant to bend forward (Fig. 10.21).

Figure 10.21. A boy with hematogenous osteomyelitis of the spine. On attempting to pick something up from the floor he keeps his spine perfectly straight because of pain and muscle spasm in the lumbar region, the site of osteomyelitis.

Figure 10.22. Hematogenous osteomyelitis of the lumbar spine in a 7-year-old child. Note the marked narrowing of the involved intervertebral disc space and the osteolytic lesions in the adjacent vertebral bodies.
Figure 10.23. Osteomyelitis of the thoracic spine in a 41-year-old adult. Note the marked destruction of the intervertebral disc space and the destruction of the adjacent portions of the involved vertebral bodies.

to confirm the diagnosis of osteomyelitis, but it is safe in the lumbar region only.

In adults afflicted with osteomyelitis of the spine, severe back pain is a prominent feature. The physical signs are similar to those seen in children, but the systemic reaction to the infection is usually more marked. As with children, the radiographic findings of osteolysis of the vertebral body and narrowing of the intervertebral disc space become obvious only after the first 2 weeks of illness (Fig. 10.23).

**Treatment and Prognosis**

The general plan of treatment for acute hematogenous osteomyelitis of the spine is similar to that described for osteomyelitis of the long bones in a previous section of this chapter.

Bed rest for the patient is supplemented by local rest for the spine, which is provided by a body cast. Operative drainage of the vertebra and disc space is indicated only if nonoperative treatment fails to control the infection; it is seldom necessary.

In children, the involved disc space remains permanently narrow but seldom fuses spontaneously, whereas in adults, spontaneous fusion is more frequent. Occasionally, persistent or recurrent back pain arising from the abnormal segment necessitates local spinal fusion.

**Osteomyelitis and Septic Arthritis Secondary to Wounds**

Bone and joint infection secondary to wounds, whether accidental or surgical, is caused by pathogenic bacteria that have gained access to the skeletal tissues directly from the outside environment. This exogenous type of infection, in contradistinction to the hematogenous or endogenous type, can develop in any site and at any age. Patients affected by HIV and AIDS are particularly susceptible to exogenous bone and joint infections.

Pathogenic bacteria may reach a bone or joint through a variety of wounds, such as a penetrating wound produced by a high-velocity missile or even a small puncture wound produced by a sharp object (Fig. 10.24). Furthermore, all open (compound) fractures and joint injuries are obviously contaminated by exogenous bacteria and consequently carry the risk of serious infection. Likewise, closed (simple) fractures and joint injuries that are treated by operation (open reduction and internal fixation) may become infected. Indeed, any operation carries this risk, but it is particularly significant in the musculoskeletal system because the sequelae of bone and joint infection are so serious.

Synovial joints are particularly susceptible to infection and therefore even simple needle aspiration of a joint demands rigid aseptic pre-
Chronic Recurrent Multifocal Osteomyelitis

Although it resembles bacterial osteomyelitis in some ways, chronic recurrent multifocal osteomyelitis is distinctly different from either acute hematogenous osteomyelitis or subacute osteomyelitis. It is characterized by a series of recurrences and remissions of multifocal areas of bone pain in different sites at different times. The underlying bone lesions are somewhat similar radiographically to those of bacterial osteomyelitis. The striking difference, however, is that in chronic recurrent multifocal osteomyelitis (CRMO) no bacteria can be isolated from the lesions. Consequently, antibiotics are not indicated, but nonsteroidal anti-inflammatory drugs (NSAIDs) are helpful in relieving the pain of inflammation. It is probable that CRMO overlaps the seronegative spondyloarthropathies such as psoriatic arthritis. Fortunately, it is a self-limited disorder, although it may extend over a period of a few months to a few years before subsiding permanently with no significant sequelae.

Pyogenic Infections in the Hand

The soft tissues of the hand are frequently infected by pyogenic bacteria because of the high incidence of minor hand injuries such as lacerations and puncture wounds. Such infections are not only common but also potentially serious because they may spread to the bones, joints, or tendon sheaths.

Soft tissue infections in the hand include the following three groups: 1) those involving the nail fold (paronychia) (Fig. 10.25A); 2) those involving potential spaces in the hand—the pulp space ( felon) (Fig. 10.25B), the thenar space (Fig. 10.26A), and the midpalmar space (Fig. 10.26B); and 3) those involving a tendon sheath (pyogenic tenosynovitis). Of these, pyogenic tenosynovitis is the most serious and deserves special mention.

Pyogenic Tenosynovitis

Etiology

Laceration and puncture wounds provide the portal of entry to the tendon sheath for patho-
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Figure 10.25. A. Paronychia. B. Pulp space infection (Felon).

Figure 10.26. A. Thenar space infection. B. Mid-palmar space infection. The swelling rapidly extends to the dorsum of the hand where the areolar planes are loosely arranged.

Figure 10.27. Pyogenic tenosynovitis of the ring finger. Note that the involved finger is swollen and tends to assume a flexed position because of the tension in the inflamed synovial sheath.

Pyogenic bacteria, the most common of which is Staphylococcus aureus.

Pathogenesis and Pathology
The synovial lining of the tendon sheath is comparable to the synovial lining of a joint and responds in the same manner to pyogenic infection, namely, by edema, hypertrophy, and production of a synovial effusion. The inflamed synovial sheath becomes progressively distended by pus under pressure, which explains the semiflexed position of the digit, a position in which the synovial sheath can accept the greatest volume of fluid. The blood supply to the tendon may be compromised, with resultant tendon necrosis. In the later stages of untreated tenosynovitis, fibrous adhesions between the tendon and its enveloping sheath lead to permanent loss of motion in the involved digit.

Clinical Features and Diagnosis
The symptom of severe local pain and the signs of local swelling, tenderness, and severe pain with any passive movement of the digit are readily understood on the basis of the underlying pathological process (Fig. 10.27). Elevation of temperature, white blood cell count, and sedimentation rate indicate the systemic reaction to infection.

Treatment
Pyogenic tenosynovitis requires the same plan of systemic and local treatment as that described for acute hematogenous osteomyelitis in a previous section of this chapter. Early operative treatment (through an incision along one side of the digit) is as important for tenosynovitis as for septic (pyogenic) arthritis; pus is evacuated and, in addition, continuous drainage and instillation of antibacterial drugs
are instituted in an attempt to preserve the tendon as well as the motion between it and its sheath.

**Necrotizing Fasciitis**

Necrotizing fasciitis, a potentially lethal soft tissue infection, is caused by a particularly virulent strain of group A β-hemolytic streptococcus. Initially involving the deep fascia and subcutaneous fat, the infection spreads at an alarming rate, causing extensive necrosis and even gangrene with associated toxic shock and end organ failure. Understandably, the lay term for necrotizing fasciitis is *flesh-eating disease.* Vigorous antibiotic therapy combined with radical surgical débridement and, when necessary, amputation of an involved limb, along with treatment of shock, are required as lifesaving measures. Even with such aggressive treatment, however, the mortality rate is greater than 30%.

**Meningococcal Septicemia**

A meningococcal infection may progress inexorably to an overwhelming and potentially fatal meningococcal septicemia despite antibiotic therapy. A serious complication is a rapidly developing peripheral vascular occlusion that initially leads to distal areas of soft tissue necrosis and sometimes even to extensive gangrene of one or more limbs that require lifesaving amputation.

**VIRAL INFECTIONS**

**Human Immunodeficiency Virus and Acquired Immunodeficiency Syndrome**

During the last two decades of the twentieth century, the incidence of HIV infection and the resultant fatal disorder of AIDS have reached epidemic proportions. Since the normal human immune system helps to ward off conditions such as infections and neoplasms and because AIDS ravages an individual’s immune system, it is understandable that these severely immunocompromised patients are at high risk for the development of a wide variety of infections, including the exogenous type of bacterial osteomyelitis and septic arthritis as well as tuberculous osteomyelitis, tuberculous arthritis, and various rheumatic diseases. These are the disorders of the musculoskeletal system that you are most likely to see in patients who are HIV-positive and especially those who have full-blown AIDS. You will learn much about HIV and AIDS in other parts of your curriculum, including their relevance to other body systems and the “universal precautions” that must be taken by health care workers who are exposed to the hazards of penetrating injuries from needles and sharp surgical instruments while attending such patients.

**GRANULOMATOUS BACTERIAL INFECTIONS**

The terms *granulomatous* or *granuloma-producing* infections refer to a group of chronic inflammatory conditions, some of which are caused by *bacteria,* such as tuberculosis and syphilis, and others by *fungi,* such as actinomycosis.

The inflammatory reaction incited by these granulomatous infections is chronic from the onset because the *productive* element of inflammation exceeds the *exudative* element. Characteristic of this type of chronic inflammation is the reaction of the local tissue cells (histiocytes, including epithelioid cells), which collect to produce small discrete lesions about the size of a *granule* (1 to 2 mm); hence the terms *granulomatous* or *granuloma-producing* infections. As the inflammatory reaction progresses, more granules are produced, and these subsequently coalesce to form progressively larger lesions. Of the granulomatous infections involving the musculoskeletal system, the most important is tuberculosis.

**Tuberculous Infections: General Features**

Improved public health measures concerning prevention and early detection of tuberculosis and the development of effective antituberculous drugs have both been important factors in the striking reduction of mortality and morbidity of tuberculous infection. However, the incidence of this potentially serious infection has actually increased significantly in recent years, even in well-developed countries; indeed, in some of the developing countries of the world, tuberculosis continues to be a com-