INFECTIONS in EMERGENCY MEDICINE

VOLUME 2

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PREFACE

As emergency medicine continues to grow and shape itself into a true multidiscipline specialty, we find ourselves expanding into subspecialty fields. Like emergency medicine, infectious disease crosses the line into many other fields of study. Although traditionally managed by internists, pediatricians, and other primary care physicians, infections are frequently seen by the emergency physician. Although excellent infectious disease texts are available, none present the material from the vantage point of the emergency physician. Our goal was to prepare a text, consisting of two volumes, covering the subject in depth, but focusing on clinical issues, so that the two books would be useful to practitioners, as well as academicians, in our field. To this end, we believe we have achieved our goal.

Each chapter presents topics in a logical progression, beginning with the etiology and pathophysiology, followed by in-depth discussions of the clinical presentation, differential diagnosis, and emergency department management. Where applicable, attention is given to issues concerning subspecialty consultation, admission guidelines, transfer guidelines, and preventive measures. Inpatient management is discussed in sufficient detail to allow the emergency physician to assist in the decision-making process regarding therapy.

We would like to thank the contributing authors for providing scholarly, well-researched chapters, and extensive references. With their effort, and the persistence of the staff at Churchill Livingstone, it is with pleasure we offer you the second and final volume of *Infections in Emergency Medicine.*

David Schillinger, M.D.
Ann Harwood-Nuss, M.D.

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1 Infections of Bones and Joints

W. Bruce Watson
Robert C. Jorden

NONGONOCOCCAL SEPTIC ARTHRITIS

Septic arthritis is the inflammation of a joint caused by the invasion of the normally sterile synovium and synovial fluid by one or more pathogens. The clinical course may be fulminant or insidious, the diagnosis a "textbook classic" or an enigma, the treatment simple or complex, and the outcome excellent or tragic. It is not a single disease, but a family of diseases that affect different age groups and patient populations. No reliable data on the incidence are available. Most reviews report only the number of cases at a given institution or group of institutions over a period of time. Denominators, such as the populations at risk or even the number of patient admissions, are largely unavailable.

Etiology

Many infectious agents can cause septic arthritis (Table 1-1); however, just a few of these account for the majority of cases. Table 1-2 lists the common pathogens and their wide variation of incidence in the different age groups. This most likely reflects age-related differences in the etiologies of bacteremia.[1]

Staphylococcus aureus is the most common cause of septic arthritis.[2,3] In most age groups, it surpasses the number-two pathogen by a wide margin. Exceptions to this pattern occur with infants between 6 and 24 months old,

Table 1-1. Etiologies of Septic Arthritis

I.	Staphylococci
	A. Coagulase positive
	B. Coagulase negative
II.	Aerobic cocci
	A. Streptococci
	1. Alpha-hemolytic
	2. Beta-hemolytic
	3. Enterococcus
	B. Pneumococcus
III.	Neisseria
	A. Gonococcus
	B. Meningococcus
IV.	*Haemophilus influenzae*
V.	Gram-negative bacilli
	A. *Pseudomonas*
	B. *Proteus*
	C. *Enterobacter*
	D. *Escherichia*
	E. *Serratia*
	F. *Klebsiella*
VI.	*Salmonella*
VII.	Anaerobes
	A. *Bacteroides*
	B. *Clostridium*
	C. Anaerobic cocci
	1. *Peptococcus*
	2. *Peptostreptococcus*
VIII.	Granulomatous
	A. *Brucella*
	B. *Mycobacterium*
	1. Mammalian
	2. Nonmammalian
IX.	Fungal
	A. Candida
	B. Others

(Adapted from Kelly,[335] with permission.)

where *Haemophilus influenza* predominates, and in young, sexually active adults, where *Neisseria gonorrhoeae* is most often encountered. In both cases, *S. aureus* is the next most common organism. The majority of strains of *S. aureus* causing arthritis are resistant to penicillin.[4,5] Methicillin-resistant staphylococci have been documented in only one report, in which they accounted for 39 percent of the strains isolated from community-acquired infections, predominantly in intravenous drug abusers.[6] From these data one can infer that methicillin-resistant staphylococci cause septic arthritis more commonly than currently recognized.

Staphylococcus epidermidis is cultured much less often than *S. aureus*, ranging from zero[7–9] to 17 percent of unselected nongonococcal cases.[2,10,11]

Table 1-2. Etiologies of Septic Arthritis by Age

	0–6 mo %	6–24 mo %	2–5 yr %	5–15 yr %	15–30 yr %	30–60 yr %	>60 yr %
N. gonorrhoeae	7	<1	4	6	80	28	<1
S. aureus	45	9	18	42	2	29	53
Streptococci	25	6	12	10	2	9	17
S. pneumoniae	1	6	3	<1	1	1	3
H. influenzae	3	43	12	<1	<1	<1	<1
Gram-negatives	10	4	6	7	3	16	14
No diagnosis	0–14	0–29	0–43	0–29	0–11	0–16	0–24
References	9,18,107,336	3,9,107,150	3,107,150	2,3,107,150	2,8,11	2,8	2,8,31,32

In patients with prosthetic joints, however, it is a much more important pathogen and accounts for 20 to 25 percent of isolates.[12–14] Resistance to multiple antibiotics, including methicillin, is common; however, the organism remains sensitive to vancomycin.[15]

N. gonorrhoeae is by far the most important pathogen among young, sexually active patients, with 70 to 90 percent of all cases occurring before 30 years of age.[2,8,11] In the 30- to 60-year-old age range, the incidence of gonococcal arthritis declines to less than 30 percent of documented cases. Beyond age 60, gonorrhea almost never causes arthritis.

The streptococci are consistent causes of septic arthritis, usually trailing *S. aureus* in incidence. All the major groups have been implicated, but their relative importance varies significantly with the clinical situation.

In most series of septic arthritis, group A beta-hemolytic streptococci *(S. pyogenes)* is the most commonly encountered species.[3,4,9,11] This is most likely due to the high incidence of group A pharyngitis and asymptomatic carriage in the population at large.[16,17]

Group B streptococcal septic arthritis is mainly a disease of neonates, causing one-fourth to one-half of cases before 1 month of age.[18,19] Other than meningitis, septic arthritis is the most common serious group B streptococcal infection.[20] Adults are rarely affected, with approximately 30 cases reported in the literature.[21–23] However, these tend to be severe infections in chronically ill patients and result in high morbidity and mortality rates. The incidence of such infection, as well as other group B infections, appears to be rising.[24] Group B streptococci remain uniformly sensitive to penicillin.[25]

Group D species, the enterococci, often cause septicemia, urinary tract infections (UTI), biliary tract infections, and endocarditis, but rarely septic arthritis.[26] Cases have occurred in patients with predisposing factors such as previous arthritis, prosthetic joints, alcohol abuse, and steroid therapy and commonly have poor outcomes. Because of antibiotic resistance, these organisms often require combination antimicrobial therapy, such as ampicillin plus an aminoglycoside.[27]

Group G streptococci are occasionally found as part of the normal flora of the skin and upper respiratory, gastrointestinal, and female genital tracts. Endocarditis and septic arthritis are the most common serious infections, but both are rare.[28] Arthritis, which can be polyarticular,[20] usually affects patients with some underlying illness such as rheumatoid arthritis (RA), malignant neoplasms, and prosthetic joints. Of particular interest is the appearance of cellulitis in nearly three-fourths of cases.[30] The organisms demonstrate in vitro sensitivity to most beta-lactam antibiotics, but cases of impaired killing are frequent.[28]

Septic arthritis due to *Streptococcus pneumoniae* is not common,[2,11] despite the ubiquity of other pneumococcal infections. It is found most often in the 6-month to 2-year age group, where it accounts for about 6 percent of cases and is often associated with osteomyelitis. The incidence declines rapidly after infancy and the pathogen is rare among young and middle-

aged adults[2,3]; however, in the elderly, the incidence again rises to approximately 3 percent.[31,32] Predisposing factors include sickle cell disease, other major underlying medical diseases, and pneumococcal infection elsewhere.[33] Most strains of *Pneumococcus* remain sensitive to penicillin. Multiple-resistant strains have been reported, but are rare and have not been demonstrated in septic arthritis.[34]

Viridans streptococci are part of the normal oral flora. They cause up to one-half of cases of bacterial endocarditis and can also cause suppurative infection elsewhere, including septic arthritis, where they account for up to 25 percent of streptococcal isolates.[4,35] In general these organisms are of low virulence and respond well to penicillin.

H. influenzae is the most common organism causing septic arthritis in the age group 6 months to 2 years, accounting for more than 50 percent of cases[3,9]; it is also the major pathogen in bacterial meningitis, otitis media, and epiglottitis in the same age group.[36] Thirty percent of *H. influenzae* septic joints are associated with meningitis[37] and may represent either hematogenous spread or an antigen-induced reactive process.[38] An additional 22 percent are associated with osteomyelitis.[37] Most isolates are type b, but nontypable species have been documented as well.[39] This organism rarely causes arthritis in adults, with only 25 cases having been reported. When affected, these adults tend to have underlying joint pathology and major underlying diseases. The arthritis is polyarticular in almost half of the cases.[40] Ampicillin is the drug most commonly used in treating *H. influenzae* infections, although resistance occurs in up to 25 percent of cases.[36] This has been infrequently documented in septic arthritis but must be considered possible in areas where resistance is endemic.[41] Chloramphenicol and most of the third-generation cephalosporins are reasonable alternatives.[27,36]

Gram-negative bacilli represent a small but persistent group of pathogens in septic arthritis. They are most commonly isolated at the extremes of age, in neonates and the elderly, as well as in patients with significant underlying medical diseases or IV drug abuse.[3,4,18,31,32,42] They are part of the normal gastrointestinal flora and frequently cause intra-abdominal and genitourinary infections, as well as generalized sepsis.[43] *Escherichia coli, Proteus, Klebsiella,* and *Pseudomonas aeruginosa* are encountered most often, although virtually all Gram-negative aerobic bacilli have been reported to cause arthritis.[42,44-47] Empiric antibiotics must include a broad-spectrum beta-lactam plus an aminoglycoside[48] or a third-generation cephalosporin.[27]

P. aeruginosa displays a sufficiently unusual clinical pattern to merit separate consideration. *Pseudomonas* causes septic arthritis in three clinical settings: IV drug abusers, puncture wounds of the feet, and nosocomial sepsis. In IV drug abusers, it is the dominant Gram-negative organism and displays an unusual predilection for fibrocartilaginous joints, such as the symphysis pubis, sacroiliac, sternoclavicular, and acromioclavicular joints.[49,50] The organism has been cultured in the rubber soles of sneakers[51]

and is a frequent cause of late infections following puncture wounds of the feet.[52] Among hospitalized patients, *Pseudomonas* causes 10 to 20 percent of nosocomial infections and Gram-negative sepsis. It is particularly dangerous when it infects patients already receiving antibiotics for another infection.[53] Therapy consists of an antipseudomonal penicillin, such as ticarcillin, and an aminoglycoside. Of the third-generation cephalosporins, ceftazidime and cefoperazone demonstrate good antipseudomonal activity.[54]

Anaerobic bacteria are rarely documented in septic arthritis. This is partially the result of inadequate laboratory techniques for recovering anaerobes and failure to consider them as a potential etiology in septic arthritis.[55] They are part of the normal flora of the skin, oropharynx, and gastrointestinal tract and primarily cause opportunistic infections.[56] Anaerobic septic arthritis occurs in three clinical settings: prosthetic joints, traumatic injuries, and debilitating diseases.[57] In the first two situations the bacteria isolated are Gram-positive cocci, which were introduced during surgery or by the initial injury. In the last situation, Gram-negative organisms arrive at the joint hematogenously.[57] Forty percent of cases yield pure anaerobic cultures; the remainder demonstrate multiple anaerobic and/or aerobic isolates.[57] Pure anaerobic infections are associated with a better outcome than are the mixed infections. Penicillin, clindamycin, metronidazole, and cefoxitin demonstrate good anaerobic activity.[27]

Most of the pathogenic fungi including *Candida*,[58,59] *Coccidioides*,[60] *Sporothrix*,[61,62] *Blastomyces*,[63] *Cryptococcus*,[64] and *Histoplasma*[65] can cause septic arthritis, although the incidence is low. These infections usually reflect disseminated disease, with hematogenous spread to the joint, and are often difficult to diagnose because they are not considered and often present atypically. Such difficulties can produce delays that result in unnecessary joint damage. Once diagnosed, most fungal arthritides are easily treated with drainage and antifungal agents.[66]

Candidal septic arthritis is probably the most common fungal arthritis; it occurs in neonates and debilitated patients, particularly immunosuppressed individuals and patients with indwelling urinary and IV hyperalimentation catheters. Two-thirds of cases present acutely, with the remainder displaying a more indolent course. Associated osteomyelitis is common. Treatment is with ketoconazole or amphotericin B. Morbidity and mortality are significant, probably related to the underlying diseases.[58,66]

Coccidioidomycosis primarily affects healthy individuals in endemic areas. The arthritis is usually acute and monoarticular but chronic forms occur. Other signs of coccidioidal infection are uncommon. Diagnosis usually rests on characteristic histopathologic findings of the synovial fluid or synovial biopsy. Complement fixation tests are positive in 90 percent of cases. Morbidity is high but mortality is low. Treatment consists of amphotericin B, with or without surgical drainage.[60,66]

Most of the pathogenic mycobacteria, including *M. tuberculosis, M. kansasii, M. marinum, M. intracellulare,* and *M. leprae* can cause infectious arthritis.[66,67] Of these, tuberculous arthritis is the most common, but is rare

as the overall incidence of tuberculosis has declined. Tuberculosis reaches the joint hematogenously or by spread from an adjacent osteomyelitis. It causes a chronic synovitis, which can result in significant joint damage if untreated. Half of the cases involve the spine (Pott's tumor); the majority of the remaining cases affect weight-bearing joints. Treatment is initiated with a combination of isoniazid and rifampin.[66]

Atypical mycobacteria more often cause tenosynovitis than frank arthritis, and they, too, are indolent in nature.[67] Leprosy can produce an acute polyarthritis of the small joints of the hands, knees, and ankles.[66] Since the atypical mycobacteria are such a diverse group and so often demonstrate resistance to standard antituberculous regimens, the treatment must depend on sensitivity studies.[66]

Regardless of etiology, laboratory investigations fail to pinpoint a pathogen in a certain number of septic arthritis cases.[2,3] Nade[68] blames this on prior use of antibiotics, inadequate anaerobic cultures, the standard of microbiologic laboratories, the changing patterns of the organism involved and their culture characteristics, and failure to culture blood and other sources of infection or to perform arthrocentesis frequently enough. In these instances, the diagnosis must be presumptive and therapy pursued with empiric antibiotics appropriate to the clinical situation.[27]

Pathophysiology

Bacterial pathogens can reach the joint via three routes: hematogenous seeding, spread from an adjacent focus of infection, or direct inoculation—either by surgery or trauma. Of these, the hematogenous route is by far the most common in children, with the others becoming more important in adults. Considering the relative frequency of bactermia and the infrequency of septic arthritis, the synovium is normally quite resistant to invasion.[69]

The synovial membrane is a highly vascular structure that creates and contains the synovial fluid and forms both an active and passive barrier to infection. To gain access to the joint, organisms must cross two barriers: the capillary wall and the synovial matrix. The lining cells also have a prominent phagocytic function, which, together with normal mononuclear cells within the synovial fluid, can remove debris and bacteria.[69] To create an infection, pathogens must escape this phagocytosis.[70]

Synovial fluid is a dialysate of plasma that lubricates and nourishes the avascular cartilage. It includes water, electrolytes, and small serum molecules, such as glucose and albumin, in concentrations approaching those of the blood. Larger molecules, such as the clotting factors, complement and many of the globulins are excluded. Albumin accounts for 60 to 75 percent of the total protein content, which is approximately one-third that of serum. To this is added hyaluronic acid, a large mucopolysaccharide. There are normally approximately 60 white blood cells (WBCs) per milliliter of synovial fluid, most of which are monocytes. Polymorphonuclear leukocytes (PMNs) rarely account for more than 2 percent of the cells.[69,71,72]

When an infection establishes itself within a joint, a number of pathologic changes occur. First, there is an increase in the volume of the synovial fluid. A purulent exudate forms, containing PMNs, plasma proteins, fibrinogen, and fibrin.[69,70] Lysosomal enzymes released by the WBCs and synovial cells damage the cartilage, first by removing proteoglycan and collagen and later by killing the chondrocytes.[69,73,74] Fibrin clots form, which adhere to the cartilage and block the influx of nutrients and the outflow of metabolic by-products and provide a sequestrum for the bacteria.[69,73] There can be direct bacterial damage as well, depending on the organism involved.[73] Articular destruction can continue after the synovial fluid has been sterilized if all the products of infection have not been removed.[75,76]

There are a number of predisposing factors for septic arthritis (Table 1-3). In neonates, prematurity and instrumentation, such as umbilical vein catheterization, are risk factors.[18] An additional mechanism is extension from an adjacent osteomyelitis through the transphyseal blood vessels.[77,78] In adults, drug abuse, alcoholism, previous joint pathology, immunosuppression, diabetes mellitus, prosthetic joint, trauma, malignancy, and renal disease all increase the likelihood of joint sepsis.[5,8,10,11,32] Infections elsewhere, such as cellulitis, pneumonia, endocarditis, meningitis, and otitis media, can also lead to bacteremias.[9,32]

Clinical Presentation

Nongonoccal septic arthritis typically presents as an acute onset of a hot, swollen, and tender monarticular arthritis associated with fever and chills.[79,80] Polyarticular involvement develops in 20 percent of cases.[81] The infection preferentially strikes weight-bearing joints, such as the knee and hip, but can include any diarthrodial or cartilaginous joint.[2,4,10] On physical examination, the involved joints are erythematous and have a decreased and painful range of motion. Careful searching often uncovers other foci of infection, such as the nasopharynx, lungs, central nervous system (CNS), skin, or urinary tract.[82] Atypical presentations are frequent in neonates and adults with significant underlying medical diseases. As a result, diagnosis is

Table 1-3. Conditions Predisposing to Nongonococcal Septic Arthritis

Systemic Factors	Local Factors
Diabetes mellitus	Trauma
Malignancy	Preexisting arthritis
Immunosuppression	Rheumatoid
Alcoholism	Crystal
IV drug abuse	Degenerative
Sexually transmitted diseases	Neuropathic
Renal failure	Joint prosthesis
Steroid therapy	Intra-articular steroid injection

often delayed in these patients. Neonates demonstrate the usual paucity of symptoms with their infections. Systemic signs such as fever and chills are infrequent, but tachycardia and hypotension can be seen. Symptoms are often limited to irritability, poor feeding, or a dislike of handling. The hip is the usual target, but it is difficult to examine. Suggestive signs and symptoms include tenderness to palpation and motion; lack of active motion; an abnormal posture; or subtle swelling of the buttock, leg, or genitalia.[68,83]

Adults present in atypical fashion based on local or systemic predisposing factors (Table 1-3). Preexisting joint pathology, such as RA, crystal arthropathies, neuropathic joints, prosthetic joints, and degenerative joint disease, can confuse the clinical picture.[84] New complaints can be lost among a welter of old complaints. Systemic diseases, such as diabetes, cancer, cirrhosis, or chronic renal failure, often alter the inflammatory response and prolong the clinical course.[5,12,32]

Septic arthritis of the axial skeleton is less common and more difficult to diagnose.[85] Sites of infection include the vertebral discs,[86,87] sacroiliac,[88,89] manubriosternal,[90,91] sternoclavicular,[92,93] and acromioclavicular joints, and the symphysis pubis.[94,95] Populations at risk include children, the elderly, and IV drug abusers. The symptoms are often indolent, the physical examination misleading, and standard laboratory and radiography unrewarding. A high index of suspicion and sophisticated diagnostic techniques are needed to establish the diagnosis.

Differential Diagnosis

Trauma is the most common cause of joint pain seen in the emergency department. Fortunately, it is rarely difficult to discern. The history is the most important diagnostic point, and the patient usually relates some specific injury to the affected joint. However, in one review of proven septic arthritis in children, 20 percent of patients reported some sort of antecedent trauma,[9] and 32 percent of adults in another review did so.[10] After the injury, there is often an immediate onset of pain soon followed by swelling. However, the swelling can be delayed up to 24 hours in some instances. Fever should not occur with simple joint trauma. On examination, the joint is swollen and tender to palpation and active and passive motion. Although frequently warmer than the surrounding skin, a traumatized joint is rarely hot and erythematous. Arthrocentesis is not often indicated, but if performed demonstrates either a hemarthrosis or a grade I inflammatory picture (Table 1-4).

A slipped capital femoral epiphysis can cause hip pain in adolescents. It can be distinguished from a septic arthritis by the lack of constitutional complaints and the characteristic radiographic appearance. Avascular necrosis of the femoral head (Legg-Calvé-Perthes disease) can be somewhat more difficult to differentiate from a septic process. Early on, before the distinctive radiologic findings are present, the only complaint is pain.

Table 1-4. Synovial Fluid Analysis

Synovial Fluid Category	WBCs/mm^3	Percent PMNs
Normal	<200	<25
Noninfectious		
Type 1: No inflammation	1–2,000	<30
Type 2: Mild inflammation	3–5,000	<30
Type 3: Severe inflammation	5–50,000	50–90
Infectious		
Type 4	>50,000	>90

(Adapted from Cohen & Goldenberg,[72] with permission.)

Again, constitutional complaints are absent and the WBC count and erythrocyte sedimentation rate (ESR) are normal.

Loosening of an articular prosthesis can cause pain with motion and weight bearing. Technetium bone scans are usually positive. Plain radiographs can show abnormal lucencies in the bone-cement interface greater than 2 mm, changes in the position of prosthetic components, periosteal reactions, cement fractures, or motion on stress views. Arthrograms can show fistulas or defects at the bone-cement interface. Peripheral WBC counts and the ESR are frequently normal. Only arthrocentesis or arthrotomy with cultures reliably differentiates between loosening and infection of the prosthesis. Indeed, infection is a common cause of loosening.[13]

RA, a chronic systemic inflammatory disease, occasionally requires differentiation from septic arthritis in two ways: One must decide whether a particular joint is affected by infection or a flare-up of the chronic disease; patients present de novo and require establishment of the diagnosis. The clinical picture of RA, with its characteristic joint deformities and skin changes, is distinctive. However, complicating the issue is the fact that rheumatoid disease is both a risk factor for septic arthritis and an alternative in the differential diagnosis. Thirteen to 59 percent of patients in recent reviews of septic arthritis had RA, although these patients constitute only 0.3 to 3 percent of the general population.[84,96] The one study that determined an actual frequency found that 0.5 percent of all admissions for RA involved septic arthritis.[97]

Most patients with newly diagnosed cases of RA present with an insidious complex of malaise, fatigue, and generalized musculoskeletal pain preceding the arthritis by weeks or months. These cases bear little resemblance to septic arthritis. However, 20 percent of patients present acutely over a period of several days; in these cases RA may be confused with septic arthritis. RA is polyarticular three-fourths of the time,[98] a finding present in fewer than 20 percent of septic arthritis cases.[81] It also appears less erythematous and hot. Despite these differences, no dependable historical or clinical markers distinguish between the two disorders, so the physician must rely on the arthrocentesis. Most cases of RA produce type 3 inflamma-