Joint Disorders

Kathleen S. Schrank

Disability for OA alone is estimated to run 2% of the gross national product. The most common cause of joint pain is OA, but the most crucial challenge is identification of a septic joint. Septic arthritis occurs in all age groups but is more common in children than in adults, and about 10% of patients with an acutely painful joint will be found to have infection.1

PATHOPHYSIOLOGY

The structure of diarthrodial joints (the most common type) includes the synovium, synovial fluid, articular cartilage, intraarticular ligaments, joint capsule, and juxtaarticular subchondral bone. The delicate synovium provides oxygen and nutrients to cartilage and produces lubricants. Articular cartilage deforms under mechanical load to minimize stress and provides a smooth surface for joint motion with minimal friction. Causes of joint disorders (Box 107.1) often overlap. Cumulative microdamage and remodeling occur with use and aging. Mechanical or metabolic disturbances may lead to a secondary inflammatory response, or an inflamed structure (e.g., a tendon) may rupture. Arthrosis is due to a mechanical insult, whereas arthritis is due to inflammation of the synovium. With inflammation comes white blood cell (WBC) infiltration, release of cytokines (e.g., tumor necrosis factor-α [TNF-α], interleukins) and other inflammatory mediators, and proliferation of cells or tissue. Edema collects around the joint, which causes stiffness. With prolonged inflammation, erosion of bone and destruction of the joint eventually occur and can produce deformity and chronic disability.

In addition to bone, muscles, and joints, “joint” pain may derive from nerves, skin, or periarticular structures (ligaments, tendons, bursae, bone). The enthesis is the structural insertion of tendon or ligament into bone. Inflammatory enthesitis is prominent in the spondyloarthropathies, such as reactive arthritis.

PRESENTING SIGNS AND SYMPTOMS

The clinical findings can narrow the potential cause of a patient’s symptoms (Table 107.1). Initial assessment must determine whether the anatomic site of the problem is the joint and then a general category of the disease, either inflammatory (septic versus aseptic) or noninflammatory (mechanical). A red, hot, swollen, painful joint is the classic finding with septic and other inflammatory arthritides. Arthritis patients may also have serious nonarticular complications of their disease or its treatment.
The onset and pattern of pain are important to determine (Box 107.2). Mechanical pain is worse with use, rapidly relieved with rest, and often least in the morning. If present, morning stiffness resolves quickly. Rapid onset over a period of minutes suggests trauma, internal derangement, or a loose fragment in the joint. Inflammatory pain is often worse with use as well, but not so quickly relieved with rest, and is commonly associated with morning stiffness (short duration with OA, prolonged with RA). “Gelling” (stiffness and immobility) after sitting in one position occurs with either type. Widespread pain with stiffness is typically due to inflammatory arthritis or fibromyalgia. Subjective pain without joint findings on examination is termed arthralgia. If the patient has tried medications without relief, the dosage should be determined because inadequate dosing is common.

Although pain is usually the main concern of patients with joint disease, it is important to determine whether other associated symptoms (e.g., fever, rash, eye symptoms) or findings are present that can aid in the diagnosis. For example, examination of the skin may reveal the malar or butterfly facial rash associated with lupus, the pustular lesions of gonococemia, or the subcutaneous nodules of RA and gout.

The musculoskeletal examination attempts to identify the exact site of the problem—joint versus bone, muscle, periarticular, or superficial skin pain. Particular joint involvement may aid in making the diagnosis (Box 107.3). True joint pain is usually diffuse on palpation and increases with active and passive motion. Periarticular inflammation (tendonitis, bursitis, cellulitis) is generally more focal, with pain reproduced only by certain movements—most often resisted active
contraction or passive stretching of the involved muscles or tendons and usually only toward one side.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Mechanical, inflammatory, or metabolic causes of arthritis may be present, and the whole picture must be considered in narrowing the lengthy differential diagnosis (Box 107.4). A new diagnosis of a specific type of inflammatory arthritis may not be possible in one visit, but recognition that the case is inflammatory is important for interim care. The severity of a patient’s discomfort will determine the urgency of analgesia, which may be initiated well before refining the list of possible diagnoses.

Early identification of septic arthritis is a top priority. An infected joint is especially likely to be present in patients with inflammatory, acute monarticular arthritis, with or without fever. The presence of another site of infection (skin, lungs, urine, heart) or a joint prosthesis increases the likelihood considerably. Moderate fever is typical with a septic joint, whereas low-grade fever may be present with any inflammatory arthritis. Treatment (antibiotics and drainage) must be initiated empirically while awaiting culture results. Infections in nearby sites (bursae, skin, periosteum) should be distinguishable by careful examination and radiologic studies; ultrasonography may be very helpful in looking for joint effusions. High fever, chills, and signs of sepsis should prompt fluid resuscitation as needed and studies to identify any additional site of infection.

Although patients usually have a history of trauma or very sudden monarticular pain, fractures may occasionally be surprises, especially in those with severe osteopenia or altered mentation (e.g., alcohol abuse, seizures), in whom trauma might not have been noticed.

Signs and symptoms may overlap, but crystals found on arthrocentesis are diagnostic of crystal-induced arthritis. The recent addition of a medication that can cause hyperuricemia may be a clue, but serum uric acid levels alone do not make or rule out the diagnosis of gout. Frequently, the patient has had multiple bouts of gout in the same joint, most commonly the great toe.

**DIAGNOSTIC STUDIES**

Blood tests are rarely diagnostic in patients with synovial disorders but may be ordered sparingly to assist in management decisions. A complete blood count (CBC) and basic chemistry profile will identify anemia, an elevated WBC count, and renal dysfunction (which affects selection of contraction or passive stretching of the involved muscles or tendons and usually only toward one side.

**BOX 107.2 Key Historical Points**

What is the source and type of pain?
- Muscle, nerve, skin, periarticular or articular structures, or joint?
- Acute, chronic, or chronic with acute complication?
- Monarticular, oligoarticular (2 or 3 joints) or polyarticular (>3 joints)?
- If polyarticular, is there a symmetric or migratory pattern?

What is the OPQRST (onset, palliation/aggravation, quality, radiation, severity, timing)?

Associated symptoms?
- Brief or prolonged morning stiffness?
- Evidence of inflammation?
- Fever, chills, fatigue, weight loss?
- Rash, eye complaints, mucosal lesions?

Personal history
- Previous diagnosis of arthritis or other diseases? How does this compare?
- Trauma?
- Recent infection?
- Joint surgery? Prosthesis?
- Joint instability or locking?
- Repetitive use?
- Intravenous drug use?

Family history of arthritis?

Medications, especially thiazides (can increase serum uric acid), isoniazid, procainamide, and hydralazine (can precipitate lupus)?

Current and previous treatments? Nontraditional remedies? Results?

**BOX 107.3 Articular Diseases Associated with Joint Location**

**Monarticular**
- Osteoarthritis (OA, often oligoarticular)
- Septic arthritis
- Gout
- Pseudogout
- Trauma
- Hemarthrosis

**Polyarticular**
- Rheumatoid arthritis (RA)
- Systemic lupus erythematosus
- Viral arthritis
- Rheumatic fever
- Reiter syndrome
- Lyme disease
- Serum sickness
- Drug induced

**Periarticular**
- Bursitis
- Tendinitis
- Cellulitis

**By Specific Joint or Joints**
- First metatarsophalangeal (MTP): Gout
- Knee: Septic arthritis, pseudogout, gout, OA
- Metacarpophalangeal, MTP, proximal interphalangeal, tarsometatarsal, and cervical spine: RA
- Distal and proximal interphalangeal, first carpometacarpal, knee, hip, cervical and lumbar sacral spine: OA
- Sternoclavicular: Injection drug abusers with septic joints
- Axial: Seronegative spondyloarthopathies
**BOX 107.4 Differential Diagnoses for Acute Arthritis**

<table>
<thead>
<tr>
<th>Monarticular Disorders</th>
<th>Noninflammatory Joint Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abscess</td>
<td>Avascular necrosis of the hip (including Legg-Calvé-Perthes disease*)</td>
</tr>
<tr>
<td>Bone tumor</td>
<td>Charcot (neuropathic) arthropathy</td>
</tr>
<tr>
<td>Bursitis</td>
<td>Congenital hip dysplasia</td>
</tr>
<tr>
<td>Cellulitis</td>
<td>Decompression sickness, bends</td>
</tr>
<tr>
<td>Compartment syndrome</td>
<td>Hemarthrosis, hemophilic arthropathy</td>
</tr>
<tr>
<td>Fibromyalgia, myofascial pain syndromes</td>
<td>Hemoglobinopathies</td>
</tr>
<tr>
<td>Inflammatory myopathies</td>
<td>Hypertrophic pulmonary osteoarthritis</td>
</tr>
<tr>
<td>Myalgia, myositis</td>
<td>Inherited storage diseases (e.g., Gaucher)</td>
</tr>
<tr>
<td>Neuropathic pain</td>
<td>Liquid lipid microsphere disease</td>
</tr>
<tr>
<td>Osteomyelitis</td>
<td></td>
</tr>
<tr>
<td>Polychondritis</td>
<td></td>
</tr>
<tr>
<td>Polymyalgia rheumatica</td>
<td></td>
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<tr>
<td>Reflex sympathetic dystrophy</td>
<td></td>
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<tr>
<td>Shoulder capsulitis</td>
<td></td>
</tr>
<tr>
<td>Temporal arteritis</td>
<td></td>
</tr>
<tr>
<td>Tendinitis, tendosynovitis</td>
<td></td>
</tr>
<tr>
<td>Trauma (ligament, tendon, muscle, bone)</td>
<td></td>
</tr>
</tbody>
</table>

| Malignancy                              | Malignancy                                                        |
| Osteoarthritis                          | Malignancy                                                        |
| Osteochondritis desiccans               | Malignancy                                                        |
| Osteocondroma                           | Malignancy                                                        |
| Osteonecrosis                           | Malignancy                                                        |
| Pigmented villonodular synovitis        | Malignancy                                                        |
| Slipped capital femoral epiphysis       | Malignancy                                                        |
| Trauma                                  | Malignancy                                                        |

**Inflammatory Joint Disorder**

Amyloidosis
Connective tissue diseases: systemic lupus erythematosus, scleroderma, Sjögren syndrome, mixed connective tissue disease
Crystal deposition: gout, pseudogout
Drug reaction (serum sickness)
Erythema nodosum
Familial Mediterranean fever
Foreign body reaction
Infection related
Juvenile idiopathic arthritis and subtypes*
Multicentric histiocytosis
Osteoarthritis, degenerative joint disease
Palindromic rheumatism
Polymyalgia rheumatica with joint involvement
Rheumatoid arthritis
Sarcoidosis
Seronegative spondyloarthropathies*
Vasculitides

*Additional information is provided in the online appendix to this chapter.

medications). The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are nonspecific but useful markers of inflammation. Coagulation studies are needed only if a patient is taking anticoagulants or a bleeding disorder is suspected. Creatine phosphokinase is helpful if muscle pain or weakness is detected. If clinical assessment raises concern for other autoimmune or systemic diseases, additional screening for multiorgan involvement with urinalysis, liver enzymes, electrocardiogram, and chest films may help. Serologic testing (e.g., rheumatoid factor [RF], antinuclear antibody, and Lyme serology, depending on the clinical impression) is generally done in follow-up settings.

Plain radiographs are ordered if a fracture, foreign body, septic joint, or tumor is suspected. If the initial films show no fracture but suspicion remains, films repeated in 1 to 2 weeks may show callus formation or abnormal alignment. Radiographic findings may also assist in diagnosing the type of arthritis (Box 107.5), although they may remain normal early in the course. The presence of degenerative changes in a painful joint supports the clinical suspicion of OA as the cause, but such changes also become common with age, even in asymptomatic joints; conversely, a normal film does not rule out OA. Similarly, calcified fibrocartilage is often found in patients with calcium pyrophosphate deposition (CPPD) disease but is common in asymptomatic patients as well. Ultrasonography is useful in confirming joint effusion, especially in joints that are difficult to assess, such as the hip. Other modalities are rarely indicated in the emergency department (ED). Although magnetic resonance imaging (MRI) can distinguish synovitis from effusion and identify rotator cuff tears or be used to evaluate ligament trauma, emergency MRI or computed tomography (CT) is indicated only if a severe joint complication is strongly suspected or if axial skeletal pain merits evaluation for stenosis or metastatic disease.

Arthrocentesis with synovial fluid analysis is an important diagnostic and therapeutic procedure for joint disease (see the Tips and Tricks box and Box 107.6). It is the only reliable means to rule out a septic joint, and it is essential in acute monarthritis to look for joint infection, crystals, or hemorrhrosis. Possible complications of arthrocentesis include introduction of infection into the joint space, hemorrhrosis, and adverse reactions to medications. Arthrocentesis of prosthetic joints is best done with orthopedic consultation.

Normal synovial fluid is clear and yellow in appearance. In degenerative joint disease the fluid itself is normal and thus remains clear. Bloody fluid suggests hemorrhrosis. Fat droplets may confirm a fracture. Turbid fluid is observed in
inflammatory conditions: gout, pseudogout, and septic, rheumatoid and seronegative arthritides (Table 107.2).

Crystal analysis is performed under compensated polarizing microscopy. In patients with acute gout, monosodium urate crystals are present inside neutrophils in fluid from the affected joint. The crystals are typically needle shaped and appear yellow when parallel to the compensator; this is negative birefringence. Sensitivity is at least 85%, and specificity for gout is 100%. In pseudogout, the crystals are positively birefringent (blue when parallel to the compensator), usually rhomboid shaped, and also phagocytized by neutrophils. Acute gouty arthritis may occasionally coexist with septic arthritis or pseudogout.

Glucose may be decreased relative to serum glucose in severe inflammatory disorders: down to less than 50% of the serum glucose level in septic arthritis and 50% to 75% in rheumatoid and seronegative arthritides. However, evidence suggests that chemistry studies on joint fluid should be discouraged because their results may be misleading or redundant.

Joint Fluid Collection

Identify and mark landmarks before infiltration with an anesthetic.

Preprocedure use of an ice pack will decrease pain.

Support the joint in a position of comfort during and after the procedure.

Contact the laboratory technicians before collecting the fluid to verify the following:

- Quantity of fluid required for the studies desired
- Correct tubes required and their availability (e.g., a liquid heparin tube is optimal for specimen for crystal analysis because inflamed fluid may clot)
- Where the fluid is to be sent (e.g., cell count to the hematology laboratory, Gram stain to the microbiology laboratory)

Use sonographic localization of joint fluid.

Prepare the area thoroughly with the antisepctic of choice. Use sterile gloves and equipment.

Use an 18- to 22-gauge needle depending on the size of the joint; smaller needles may not be sufficient to collect joint fluid.

Attachment of extension tubing between the needle hub and the syringe helps decrease movement of the needle in the joint space and makes changing syringes into large-volume arthrocentesis and injection of medications into the joint space easier. Tubing must be flushed when injecting corticosteroids so that the full dose actually enters the joint space.

Collect enough fluid for appropriate testing (this is not an easily repeated procedure).

Send fluid for a cell count and differential, Gram stain and culture, crystals, glucose, and viscosity.

Seeding the fluid into blood culture flasks immediately after aspiration may increase the yield.

Have the patient rest the joint for 12 to 24 hours after the injection of corticosteroids.

BOX 107.5 “Seconds” Mnemonic for Radiographic Evaluation of Arthritis

Soft tissue swelling—Nonspecific, often seen with acute arthritides such as gout, pseudogout, and septic arthritis, as well as with tuberculous arthritis; also present in trauma

Erosions—May be present in late rheumatoid arthritis as a result of the pannus eroding into articular cartilage and bone

Calcification—In late pseudogout, there may be linear calcification in cartilage

Osteoporosis—Sometimes present in late septic arthritis as a result of joint destruction (about 8 to 10 days of disease before changes are evident on plain films). Osteoporosis or periarticular bone may be seen with late rheumatoid arthritis but not with pseudogout or osteoarthritis

Narrowing of the joint space—Present in late septic arthritis; asymmetric narrowing is consistent with late pseudogout and osteoarthritis; symmetric narrowing is consistent with late rheumatoid arthritis. Joint space is typically preserved with tuberculous arthritis

Deformity—In late septic arthritis, subchondral bone destruction and periosteal new bone may be visualized; in late pseudogout and osteoarthritis, changes may include sclerosis, osteophyte formation, and subchondral cyst formation

Separation from fracture


BOX 107.6 Arthrocentesis

Indications

Suspected septic arthritis

Diagnosis of nontraumatic joint disease by synovial fluid analysis

Diagnosis of ligamentous or bony injury by confirmation of blood in the joint

Establishment of the existence of an intraarticular fracture by the presence of blood with fat globules in the joint

Relief of pain accompanying acute hemorrhage in a tense effusion

Local instillation of medications

Obtaining fluid for analysis (culture, cell count, crystal studies)

Contraindications (Relative)

Infection in tissue overlying the site to be punctured

Presence of bacteremia

Coagulopathy

Joint prosthesis (contact an orthopedic consultant)

Uncooperative patient

Viscosity can be measured grossly in the laboratory and ED. Inflammation decreases the hyaluronate portion of synovial fluid, and thus viscosity decreases. When dropped from a syringe, normal synovial fluid makes a 5- to 10-cm string of fluid before dropping. With inflammation, the string of fluid will be shorter or the fluid may simply form droplets.
begins in 1 to 2 days, peaks at about 1 week, and lasts for 1 week to a few months, during which time compliance with adjunctive measures helps prevent recurrence. Although minimal evidence supports the concern, repeated steroid use traditionally raises concern over cartilage damage, so use in the same joint is limited to every 3 to 4 months. Long-acting local anesthetic may be added to the injection for same-day short-term relief. If suspicion for an infected joint is high, intravenous antibiotics are administered after appropriate material is sent for culture (see discussion later under “Septic Arthritis”).

Serious situations may warrant admission (Box 107.7). Patients with joint infections require admission and early consultation with an orthopedic surgeon or rheumatologist. Most other patients will be discharged home with an analgesic and general treatment measures to relieve pain. Full-dose acetaminophen (650 to 1000 mg four times daily) may be adequate (especially if a history such as gastrointestinal [GI] bleeding, heart failure, or renal failure makes use of an NSAID risky), or an NSAID may be selected based on low cost and safety profile (e.g., celecoxib may be safer for the stomach). The NSAID is begun at a high dose (e.g., ibuprofen, 600 to 800 mg three times daily), continued for at least 2 to 3 days (or with inflammatory arthritis, for at least a few days after the pain stops), and then may be continued as needed. The patient is advised to take NSAIDs with food, especially with a history of stomach upset, and cotreatment with a stomach-protective drug (H2 blocker, proton pump inhibitor, or sucralfate [Carafate]) should be considered. Nonresponders begin in 1 to 2 days, peaks at about 1 week, and lasts for 1 week to a few months, during which time compliance with adjunctive measures helps prevent recurrence. Although minimal evidence supports the concern, repeated steroid use traditionally raises concern over cartilage damage, so use in the same joint is limited to every 3 to 4 months. Long-acting local anesthetic may be added to the injection for same-day short-term relief.

**Table 107.2 Joint Fluid Analysis of the Various Arthritides**

<table>
<thead>
<tr>
<th>DIAGNOSIS</th>
<th>APPEARANCE</th>
<th>WBCs/MM³</th>
<th>PMN LEUKOCYTES</th>
<th>GLUCOSE (% BLOOD LEVEL)</th>
<th>CRYSTALS UNDER POLARIZED LIGHT</th>
<th>CULTURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Clear</td>
<td>&lt;200</td>
<td>&lt;25%</td>
<td>95-100</td>
<td>None</td>
<td>Negative</td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>Clear</td>
<td>&lt;4000</td>
<td>&lt;25%</td>
<td>95-100</td>
<td>None</td>
<td>Negative</td>
</tr>
<tr>
<td>Traumatic</td>
<td>Straw colored, bloody, xanthochromic, occasionally with fat droplets</td>
<td>&lt;4000</td>
<td>&lt;25%</td>
<td>95-100</td>
<td>None</td>
<td>Negative</td>
</tr>
<tr>
<td>Acute gout</td>
<td>Turbid</td>
<td>2000-50,000</td>
<td>&gt;75%</td>
<td>80-100</td>
<td>Negative birefringence; needlelike</td>
<td>Negative</td>
</tr>
<tr>
<td>Pseudogout</td>
<td>Turbid</td>
<td>2000-50,000</td>
<td>&gt;75%</td>
<td>80-100</td>
<td>Positive birefringence; rhomboid</td>
<td>Negative</td>
</tr>
<tr>
<td>Septic arthritis</td>
<td>Turbid, purulent</td>
<td>5000 to &gt;50,000</td>
<td>&gt;75%</td>
<td>&lt;50</td>
<td>None</td>
<td>Usually positive</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>Turbid</td>
<td>2000-50,000</td>
<td>50-75%</td>
<td>&lt;75</td>
<td>None</td>
<td>Negative</td>
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PMN, Polymorphonuclear; WBCs, white blood cells.

Although a joint WBC count higher than 50,000/mm³ is generally said to be positive for infection, septic arthritis can occur with lower joint WBC counts, especially early in infection (36% of patients with septic arthritis had joint WBC counts lower than 50,000/mm³). In addition, patients with inflammatory arthritides such as RA, gout, and pseudogout may have very high joint WBC counts. Thus fluid must also be sent for Gram stain and culture. The yield is increased by immediate plating in the laboratory and perhaps by inoculating blood culture bottles with joint fluid in the ED. The serum WBC count, ESR, and joint WBC count are extremely variable in adults with septic arthritis.

In the absence of a positive Gram stain, the ED clinician must consider the whole picture when determining the probability of septic arthritis.

**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION**

ED care focuses on early relief of pain, typically with nonsteroidal antiinflammatory drugs (NSAIDs) such as ibuprofen (800 mg orally) or acetaminophen (1 g orally) (or both), ice, and limb support in a position of comfort (usually partial flexion). If the pain is severe or unrelieved by initial analgesia, tramadol or narcotic analgesics are used, and the joint may be immobilized with an elastic bandage, splint, or brace. “Buddy taping” to the adjacent digit helps relieve the pain in finger joints.

Removal of fluid from a joint effusion provides considerable relief. Intraarticular corticosteroids (e.g., triamcinolone hexacetonide, ranging from 5 mg in a finger joint to 40 mg in a large joint, or methylprednisolone, 2 to 5 mg in small joints and 10 to 25 mg in large joints) are recommended for effusions unless infection is suspected. The patient should be informed that the pain relief with corticosteroids typically

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PMN, Polymorphonuclear; WBCs, white blood cells.
General treatment measures should be recommended, including ice packs or heat (or both), temporary support (elastic wrap; brace, cane, or walker) and joint rest, but unnecessary or prolonged immobilization should be avoided. Patient education should include simple measures to avoid repetitive injury (e.g., patients with shoulder pain often ignore the obvious trigger of carrying a heavy shoulder bag). Simple splinting or ergonomic advice may assist patients with pain related to repetitive motions when the activity is unavoidable.

Appropriate lifestyle recommendations for patients with chronic arthritis are to stay as active as possible with daily activities and reasonable exercise programs. Physical and occupational therapy may contribute greatly to improved quality of life and ability to maintain independence in self-care but are usually arranged by the continuity physician. Initial range-of-motion (ROM) and later strengthening and aerobic exercise regimens are recommended; swimming pool exercise programs are quite helpful. Obese patients with lower extremity arthritis should be educated about the importance of weight reduction.

Follow-up care includes referral to a primary care physician for most patients. Rheumatology referral is recommended for patients with clinical suspicion of new inflammatory or autoimmune arthritides such as RA or for patients not improving despite adequate general care. Chronic severe pain with significant disability merits orthopedic referral. Patient education is vital for achieving an optimal outcome (see the Patient Teaching Tips box).

### RHEUMATOID ARTHRITIS

#### EPIDEMIOLOGY

RA is the most common inflammatory arthritis, with about 0.8% of the world’s population afflicted, and it has a 3:1 female preponderance. It begins most commonly in the 40s. Overall, life expectancy is only modestly reduced, but quality of life may be significantly impaired. The majority of patients experience chronic remitting but overall progressive disease, including about 10% with an aggressive, severely destructive pattern and 15% to 30% with intermittent remissions lasting up to 1 year; about 15% experience a long-lasting remission with excellent function.

#### PATHOPHYSIOLOGY

RA is a systemic inflammatory autoimmune disease that primarily targets the synovium and transforms it into hyperplastic inflamed and thickened tissue that proliferates into a pannus. The pannus is unique to RA, where it grows over the articular cartilage, erodes into bone, and causes destruction and deformity. Synovial fluid contains abundant neutrophils, cytokines, proteolytic enzymes, prostaglandins, and leukotrienes. The etiology of RA remains unknown, but it is probably caused by a combination of environmental factors and genetic susceptibility, with several contributing genes, particularly the class II major histocompatibility complex. An ongoing and uncontrolled immune response is elicited, perhaps against an autoantigen. RFs are IgM or IgG antibodies synthesized in the synovium that form immune complexes with IgG in the blood or joints.

#### PRESENTING SIGNS AND SYMPTOMS

RA is characterized by symmetric polyarthritis persisting for more than 6 weeks, prolonged morning stiffness (>30 minutes),
and systemic symptoms of fatigue, malaise, and weight loss. Diagnostic criteria are listed in Table 107.3. Arthritis typically starts in the small joints (metacarpophalangeal [MCP], metatarsophalangeal [MTP], and proximal interphalangeal [PIP] joints of the hands and feet but not the distal interpha-
langeal [DIP] joints) and later affects larger extremity joints. Migratory polyarthritis occurs, and the symptoms may wax and wane. The onset of RA is typically insidious but can be abrupt. Cervical spine involvement is prevalent, although the rest of the spine is usually spared. RA increases the risk for a septic joint or tendon rupture, and temporomandibular joint (TMJ) problems are common.

Examination in the early stages usually finds tenderness, swelling, and limited ROM in at least three joints, especially in the hands and feet. Warmth and erythema are uncommon. Palpation may reveal loss of the normal contour across joints (especially the MCP joints) because of pannus. Rheumatoid nodules are found in 20% of patients and can appear anywhere but especially over bony prominences, pressure points, and tendon sheaths. These nodules may be fixed or mobile with a rubbery or granular texture and are sometimes indistinguishable from gouty tophi. They are not a serious problem unless they occur in the vocal cords or cardiac conduction tissue. Typical later joint deformities are radial deviation at the wrist (usually the earliest deformity), ulnar deviation at the MCP joints (the most characteristic deformity of RA), swan neck or boutonnière deformities of the fingers, cock-up toes, loss of arches, and hallux valgus.

Extraarticular manifestations are common. Acutely life-threatening complications are rare but disastrous. Patients may also have serious complications of chronic treatment, particularly with infections from immunosuppression.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Identification of septic arthritis is the top priority given its risk for rapid joint destruction or systemic infection. Patients with known RA are at significantly increased risk, and clinical findings may be more subtle because of their drug regimens. Joint fluid analysis is essential. Systemic infections may seed a joint or induce an immunologic arthritis; mild systemic symptoms may be difficult to distinguish from those of exacerbation of RA.

OA is the most common consideration in the differential diagnosis, but its pattern of joint involvement (especially in the hands) and minimal to absent systemic symptoms distinguish it from RA. Self-limited arthritic syndromes (e.g., viral infections, Lyme disease) can be difficult to distinguish clinically from the initial findings in patients with RA, and thus the RA criteria require persistence of the joint symptoms for longer than 6 weeks. The usual time from onset to diagnosis of RA is 6 to 12 months, but joint damage occurs early, with 30% of patients exhibiting bone erosion at time of diagnosis.

Studies may assist in the differential diagnosis. RA typically produces a mild normochromic normocytic anemia and thrombocytosis but normal WBC count (unless infected or Felty syndrome is present), an ESR of 30 to 60 mm/hr, and an elevated CRP level. On plain films, RA is characterizedly associated with joint space narrowing (especially in the MCP, PIP, and wrist joints), marginal bony erosions, periarticular osteopenia, and soft tissue swelling (Fig. 107.1). Joint destruction (e.g., femoral head protruding through the acetabulum) occurs late.

Serologic testing (RF IgM or anti–citrullinated protein antibody [ACPA]) for a new diagnosis is generally best done in
Table 107.3 2010 American College of Rheumatology Revised Criteria for the Classification of Rheumatoid Arthritis*

<table>
<thead>
<tr>
<th>FINDING</th>
<th>POINTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Joint Involvement</strong></td>
<td></td>
</tr>
<tr>
<td>1 large joint</td>
<td>0</td>
</tr>
<tr>
<td>2-10 large joints</td>
<td>1</td>
</tr>
<tr>
<td>1-3 small joints (with or without the involvement of large joints)</td>
<td>2</td>
</tr>
<tr>
<td>4-10 small joints (with or without the involvement of large joints)</td>
<td>3</td>
</tr>
<tr>
<td>&gt;10 joints (at least 1 small joint)</td>
<td>5</td>
</tr>
<tr>
<td><strong>B. Serology (at Least 1 Test Result Is Needed for Classification)</strong></td>
<td></td>
</tr>
<tr>
<td>Negative RF and negative ACPA</td>
<td>0</td>
</tr>
<tr>
<td>Low positive RF or low positive ACPA</td>
<td>2</td>
</tr>
<tr>
<td>High positive RF or high positive ACPA</td>
<td>3</td>
</tr>
<tr>
<td><strong>C. Acute Phase Reactants (at Least 1 Test Result Is Needed for Classification)</strong></td>
<td></td>
</tr>
<tr>
<td>Normal CRP and normal ESR</td>
<td>0</td>
</tr>
<tr>
<td>Abnormal CRP or abnormal ESR</td>
<td>1</td>
</tr>
<tr>
<td><strong>D. Duration of Symptoms</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;6 wk</td>
<td>0</td>
</tr>
<tr>
<td>≥6 wk</td>
<td>1</td>
</tr>
</tbody>
</table>


ACPA, Anti–citrullinated protein antibody; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; RF, rheumatoid factor IgM.

*Score-based algorithm: Add the scores of categories A to D; a score of 6 or higher of a total of 10 is needed for to classify a patient as having definite RA.
The majority of RA patients will be discharged (see Box 107.7). Immunosuppressed patients warrant high suspicion for infection and a low threshold for admission. Follow-up visits with either a primary care physician or rheumatologist (if a new diagnosis or not responding to treatment) are essential. Chronic severe joint dysfunction merits orthopedic referral for potential surgical intervention (joint replacement, arthrodesis, synovectomy).

Disease-modifying antirheumatic drugs (DMARDS) are essential for all RA patients to prevent joint damage and are now initiated as early as possible after diagnosis (but not in the ED). The mainstay remains methotrexate, alone or in combination. Leflunomide (an immunomodulatory drug) and sulfasalazine are alternatives. Exciting new and effective biologic agents include anticytokine therapies such as etanercept, anakinra, abatacept, and rituximab.

OSTEOARTHRITIS

EPIDEMIOLOGY

OA is the most common cause of joint pain and frequently leads to chronic pain and disability. In the United States, symptomatic knee OA occurs in 6% of persons older than 30 years and hip OA in 3%. About one third of adults aged 25 to 74 years have radiographic evidence of OA in at least one joint group, most commonly the hands and then the feet and knees. Prevalence increases considerably with age, and OA is a major cause of disability, lost work time, and early retirement. Before the age of 50, prevalence in most joints is higher in men, but at older ages women are more often affected in the hands, feet, and knees.

Other risk factors include obesity, trauma, family history, and occupations involving repetitive knee or hip bending and lifting (e.g., farmers, dockworkers). The role of aggressive exercise remains unclear, although moderate running appears to be low risk. High-intensity contact sports and those involving repetitive joint impact and twisting are higher-risk categories.

PATHOPHYSIOLOGY

OA is a disease of articular cartilage and subchondral bone that is characterized by patchy loss of cartilage, overgrowth of bone at the joint margins (osteoophytes are hallmarks of OA), hypertrophy of subchondral bone, fibrosis in the joint capsule, loss of joint space, and mild inflammation of the synovium. Loss of cartilage allows the underlying bones to rub together, which produces pain, swelling, and limited ROM. The primary process is mechanical, not inflammatory, but past views of OA as being entirely mechanical (hence names used in the past such as degenerative joint disease and osteoarthrosis) are inaccurate because recent evidence shows considerably more synovial inflammation.
than was previously considered. Its pathogenesis involves chronic mechanical microdamage, disturbed chondrocyte regulation of the synthesis and degradation of cartilage matrix, genetic factors, and inflammatory pathways. Local mechanical factors (malalignment, laxity, proprioception) contribute in specific joints.

Most cases are classified as idiopathic or primary. Secondary osteoarthritis may result from congenital or developmental diseases, trauma, deposition diseases (calcium, hemochromatosis), neuropathic arthropathy, or endocrinopathies (e.g., acromegaly, hyperparathyroidism).

**PRESENTING SIGNS AND SYMPTOMS**

OA most commonly affects the knees, hips, spine, fingers (especially the DIP joints and first carpometacarpal joint), and toes (especially the MTP joints). ED visits are usually prompted by significant pain in a large joint (knees or hips) that is often associated with an acute but minor injury. Neck or back pain is also common. Patients typically report a gradual onset of pain and stiffness in one or a few joints with limited ROM. Locking or instability of the knee is common, as is joint effusion. Baseline pain is mild to moderate, worse with use, and rapidly better with rest, and the symptoms are worse in damp, cool weather.

On examination, disease is found to be limited to the symptomatic joints. Joint tenderness, bone enlargement, and crepitus on joint motion are common findings. Heberden nodes (hard nodules on the dorsal aspect of the DIP joints) are commonly seen in older women with OA. Malalignment is found in about half of knees with OA, typically with a varus (bowleg) deformity and often with instability on excess ROM. Joints may be mildly warm, especially if an effusion is present, but not dramatically inflamed. Late in the disease course, significant joint disability is evident (Fig. 107.2).

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Although OA is a polyarthritis, it most commonly affects just a few joints in each patient. Prominent general systemic symptoms point to another diagnosis, such as RA or fibromyalgia, whereas active involvement of other organs suggests other autoimmune arthritides. Patients are prone to coexisting pseudogout in the knee, or gout may develop. A red, hot swollen joint is probably infected or acute crystal-induced arthritis.

If ordered, laboratory results are generally normal. Joint fluid is usually noninflammatory, with a WBC count lower than 2000/mm³; inflammatory fluid may occur in OA but more often suggests gout or infection. Radiologic results must be correlated with the clinical picture. In asymptomatic patients older than 40 years, radiographic studies often show degenerative joint changes, but a label of OA would be incorrect. The classic changes with OA are degenerative with marginal osteophyte formation, subchondral bony sclerosis, and asymmetric joint space narrowing. Later, subchondral cysts with sclerotic walls form, and bone remodeling distorts the ends of bones. Bone demineralization and marginal erosions suggest an inflammatory arthritis such as RA.

**TREATMENT**

Initial attempts at pain relief in the ED include analgesics, ice, and support in a position of comfort. Acetaminophen or ibuprofen may be adequate. If a patient has already adequately tried and failed to obtain relief with these medications, tramadol or narcotic analgesics should be considered. If a significant effusion is causing pain and disability, removal of fluid provides considerable relief. Intraarticular corticosteroids are also effective, especially in the knee or MCP joints, with pain relief lasting weeks to months. Intraarticular hyaluronic acid injections have been used in the knee, but their efficacy is limited.

As with other arthritides, the vast majority of patients with OA are discharged home with recommendations for primary care follow-up, whereas those with known or strongly suspected joint infection are treated and admitted to the hospital. Several options are available for discharge analgesia.

Patients with mild symptoms may need only general care measures. Acetaminophen and NSAIDs are the first-line choices. In studies both have been shown to be effective in reducing pain, although NSAIDs or celecoxib is modestly better. However, acetaminophen has less risk for side effects and thus remains a good first choice. Effective additions or alternatives include tramadol or short-term use of narcotics.
Topical NSAIDs (not salicylates) are considered core treatment of OA of the knee or hands, and topical capsaicin is considered adjunctive to core treatment of these joints; strong evidence supports their benefits. Glucosamine sulfate (1500 mg/day) and chondroitin sulfate (1200 mg/day) may shift cartilage metabolism toward a positive balance and are widely popular among patients, but the overall evidence to date shows limited efficacy; these over-the-counter preparations may vary considerably in composition.

General care measures and patient education are also important (see the Patient Teaching Tips box presented earlier and Box 107.8). Evidence supports the benefit of exercise regimens and weight loss in patients with knee arthritis. Correction of knee malalignment with a neoprene sleeve, valgus brace, orthotics, or a combination of such devices is beneficial. Evidence on the benefit of acupuncture is mixed. Surgical interventions are useful in selected situations; knee arthroscopy is beneficial if cartilage flaps, loose bodies, or meniscal disruption is causing mechanical locking or instability. Total joint replacement for knee or hip OA often dramatically improves severe refractory pain and disability, particularly if the patient has a relatively low body mass index. Chondrocyte transplantation is an exciting intervention for future care.

**REACTIVE ARTHRITIS**

**EPIDEMIOLOGY**

Reactive arthritides are seronegative spondyloarthropathies that are much less common than OA and RA; however, identification is important to guide management. Most are self-limited illnesses, but persistent and severe disease develops in a minority of patients (particularly those with acquired immunodeficiency syndrome). Prevalence parallels that of the human leukocyte antigen (HLA) B27 genes in different populations. In the United States, 6% to 14% of Caucasians have HLA-B27, as do 2% to 3% of African Americans. The peak onset is during the third decade of life. Reactive arthritis has a 5:1 to 6:1 male preponderance, but cases in women may be underdiagnosed—women tend to have milder symptoms, and their genitourinary (GU) manifestations may be occult. The incidence of reactive arthritis is estimated to be 3 to 6 cases per year per 100,000 males younger than 50 years.

**PATHOPHYSIOLOGY**

In reactive arthritides, a sterile inflammatory arthropathy arises, commonly after a primary infection elsewhere in the body. Onset typically occurs 1 to 4 weeks after a diarrheal or GU infection, once the triggering infectious illness is over. Enteric pathogens include *Shigella*, *Yersinia*, *Salmonella*, *Campylobacter*, and *Clostridium difficile*. Sexually transmitted pathogens include *Chlamydia* (mainly *Chlamydia trachomatis*), *Ureaplasma urealyticum*, and human immunodeficiency virus (HIV). Presumably, microbial material or products are disseminated to the joints and extraarticular structures and trigger an inflammatory response consisting of mononuclear infiltration into the joints and entheses, synovial effusions, and inflammatory mediators. Most patients carry HLA-B27 genes. In many, the inciting infection is not identifiable.

Inflammation of the entheses, eyes, and mucosal surfaces is a distinctive feature of reactive arthritis. The illness tends to be self-limited over several months, but relapses occur in about one third of patients. HIV-associated disease is often severe and disabling.

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**BOX 107.8 General Treatment Measures for Chronic Arthritis**

<table>
<thead>
<tr>
<th><strong>Patient Education</strong></th>
</tr>
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<tbody>
<tr>
<td>Nature and usual course of the disease</td>
</tr>
<tr>
<td>Exacerbating and relieving factors</td>
</tr>
<tr>
<td>Avoidance of repetitive injuries, impacts</td>
</tr>
<tr>
<td>Arthritis self-help course available from the Arthritis Foundation (<a href="http://www.arthritis.org">www.arthritis.org</a>)</td>
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<table>
<thead>
<tr>
<th><strong>Painful Joint</strong></th>
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<tbody>
<tr>
<td>Analgesics (take a bedtime dose if early morning pain), oral and/or topical</td>
</tr>
<tr>
<td>Acute exacerbations:</td>
</tr>
<tr>
<td>- Rest, ice, compression, elevation</td>
</tr>
<tr>
<td>- Temporary limitation of range of motion and forceful use</td>
</tr>
<tr>
<td>Correction of misalignment—joint sleeve or brace, orthotics</td>
</tr>
<tr>
<td>Chronic pain—trials with ice, heat, in-water therapy</td>
</tr>
<tr>
<td>Unloading joint stress with a cane or crutch (contralateral to the affected leg)</td>
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</tbody>
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<table>
<thead>
<tr>
<th><strong>Physical Therapy</strong></th>
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<tbody>
<tr>
<td>Relief of pain and muscle spasm (massage, heat, ultrasound, electrical stimulation therapy, physical maneuvers)</td>
</tr>
<tr>
<td>Improvement in and preservation of range of motion</td>
</tr>
<tr>
<td>Strengthening (general or surrounding a specific joint)</td>
</tr>
<tr>
<td>Progressive individualized exercise regimen</td>
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<tr>
<td>General conditioning</td>
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<thead>
<tr>
<th><strong>Occupational Therapy</strong></th>
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<tbody>
<tr>
<td>Improved activities of daily living</td>
</tr>
<tr>
<td>Assist devices</td>
</tr>
<tr>
<td>Temporary splinting</td>
</tr>
<tr>
<td>Protective techniques</td>
</tr>
<tr>
<td>Energy conservation skills</td>
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</table>

<table>
<thead>
<tr>
<th><strong>Lifestyle</strong></th>
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<tbody>
<tr>
<td>Weight loss program</td>
</tr>
<tr>
<td>Adequate nutrition and calcium intake</td>
</tr>
<tr>
<td>Range-of-motion preservation</td>
</tr>
<tr>
<td>Exercise program with low-impact aerobic conditioning</td>
</tr>
<tr>
<td>Evaluation of the home for fall prevention, improved functionality</td>
</tr>
<tr>
<td>Well-cushioned shoes or orthotics</td>
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<table>
<thead>
<tr>
<th><strong>Acupuncture</strong></th>
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</thead>
<tbody>
<tr>
<td>Role unclear; efficacy not well supported by available evidence; appears beneficial for knee pain</td>
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</table>

<table>
<thead>
<tr>
<th><strong>Glucosamine and Chondroitin</strong></th>
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<tbody>
<tr>
<td>Role unclear; efficacy not well supported by available evidence</td>
</tr>
</tbody>
</table>
BOX 107.9 Classic Signs of Reactive Arthritis (Formerly Known as Reiter Syndrome)

- Enthesitis—periarticular, classically Achilles or plantar tendinitis
- Peripheral arthritis pattern—may be a hot erythematous joint for which active infection must be ruled out; often asymmetric oligoarthritis
- Dactylitis—“sausage digits”
- Conjunctivitis—bilateral or unilateral, usually painful
- Urethritis, cervicitis
- Circinate balanitis on the shaft or glans of the penis; vulvitis in women—ranging from vesicles to ulcerations
- Keratodema blenorrhagicum—painless papulosquamous rash on the palms and soles, similar to pustular psoriasis
- Oral ulcerations—painless

PRESENTING SIGNS AND SYMPTOMS

Cases of reactive arthritis seen in the ED are likely to be a new diagnosis. Typically, the patient reports one or a few sites of acute joint pain, often asymmetric and with sequential onset. Common sites include large joints (one or both ankles, wrists, knees) and small joints in the feet; the upper extremities may be involved later. Fever (up to 102.2°F [39°C]), constitutional symptoms (fatigue, malaise, weight loss), and mucosal problems are common findings (Box 107.9). Low back pain, back stiffness, or sacroiliitis occurs in half the patients. The involved joints are often inflamed. The classic triad that was formerly named Reiter syndrome includes acute peripheral arthritis (asymmetric, oligoarticular, additive), conjunctivitis (mild, usually several days before the appearance of joint pain), and nongonococcal urethritis or cervicitis (generally mild, precedes the joint pain).

Most patients do not manifest the full triad. Some may not volunteer information about other symptoms before their joint pain or about recent diarrheal or GU infections.

Later complications can include ankylosing spondylitis, uveitis, and cardiac involvement (in about 10% of patients) with conduction blocks, nonspecific ST-segment changes, Q waves, or aortic regurgitation.

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

With an acute hot joint, infection (especially gonococcal) must be ruled out. Active infection elsewhere (especially the GI and GU tracts) must also be considered. Reactive arthritis should be distinguished from seronegative RA, crystal-induced arthritis, sarcoidosis, acute rheumatic fever, psoriatic arthritis, and erythema nodosum. A carefully constructed clinical picture will usually distinguish reactive arthritis, but this may not easily be done in the ED.

Given the frequent finding of fever, acute arthritis, and involvement of mucosal surfaces in patients with reactive arthritis, laboratory studies are appropriate (CBC, chemistries, liver enzymes, urinalysis, ESR, arthrocentesis, joint fluid cultures, and usually blood cultures). In reactive arthritis, a modest increase in WBCs, platelets, and ESR is expected, and mild anemia is common. Active urethral or GI infection should be considered, and tests for Chlamydia and gonorrhea are appropriate if the patient has had any recent GU symptoms. If the precipitating infection was dysenteric, stool should be sent for culture. Serologic and HLA studies are not done in the ED.

TREATMENT

Full-dose NSAIDs are the mainstay of therapy for reactive arthritis—a good response is typical. General care measures are also appropriate (see earlier), especially encouragement of continuing exercise. Systemic corticosteroid therapy is not generally indicated, but intraarticular glucocorticoids may help in alleviating persistently problematic joints after ruling out infection. Second-line medications for nonresponders include sulfasalazine or methotrexate. Experience with anti-TNF agents is limited but promising.

Antibiotic treatment of Chlamydia is appropriate if the initial infection was untreated or is found on testing. Empiric administration of an antibiotic to patients with a previous history of GI infection is not useful unless current stool cultures show persistence of a pathogenic trigger.

FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION

Most patients with reactive arthritis can be discharged home with primary care follow-up, but admission is indicated if the patient is febrile or if active joint infection is likely. Referral to ophthalmology is advised if uveitis is suspected.

SEPTIC ARTHRITIS

EPIDEMIOLOGY

The incidence (expressed per 100,000 per year) of septic arthritis varies between 2 and 5 in the general population, 5 and 12 in children, 28 and 38 in patients with RA, and 40 and 68 in patients with joint prostheses. Males are usually affected more commonly than females, although with underlying RA, females are affected more often. About 10% of patients with acutely painful joints will have septic arthritis.

The organisms causing bacterial arthritis depend on the epidemiologic circumstances (Table 107.4). For example, monarthritus of a prosthetic joint is probably due to Staphylococcus species, whereas a migratory arthritis in a sexually active woman is probably due to disseminated gonococcal infection. Rarely, the cause may be fungal, protozoal, or mycobacterial, particularly in immunosuppressed patients. Viral joint infections are not considered part of the “septic” category.

Major risk factors for septic arthritis in adults include age older than 80 years, diabetes mellitus, RA, prosthetic joint or recent joint surgery, skin infection or ulceration, alcoholism,
intravenous drug use, and prior intraarticular corticosteroid injection. Previous joint damage from any cause also appears to increase risk.

**PATHOPHYSIOLOGY**

Bacteria can infect the joint via hematogenous spread, direct inoculation (arthritis, trauma, surgery), or contiguous contact (cellulitis, bursitis, tenosynovitis). Any microorganisms, including bacteria, fungi, and protozoa, may invade joints; however, the overwhelming majority of cases (90%) are caused by the pyogenic bacteria *Staphylococcus aureus* and *Streptococcus*. Once the pathogen penetrates the joint space, it initiates a series of inflammatory reactions that may lead to joint destruction and permanent damage. Microorganisms or their products (or both) activate the release of proinflammatory cytokines, such as TNF-α and interleukin-1, and proteolytic enzymes, such as metalloproteinases and other collagen-degrading enzymes. These proteins induce synovial membrane proliferation, granulation tissue, neovascularization, and infiltration by polymorphonuclear cells and may result, if untreated, in cartilage and bone destruction. The articular damage may progress even after eradication of microorganisms because persistence of bacterial antigens and metalloproteinases within the joint will continue to promote an inflammatory response.

**PRESENTING SIGNS AND SYMPTOMS**

Septic arthritis is generally manifested acutely as a “hot joint”—with joint pain, swelling, erythema, limited and painful ROM, and tenderness. Septic arthritis is usually monarticular, but about 10% of patients have polyarticular involvement (of those, >50% have underlying RA). The knee is the most common site, followed by the hip, wrist, ankle, and shoulder. Fever is common, though often absent in the elderly, and patients frequently have another site of recent infection.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

The differential diagnosis for septic arthritis is focused on other inflammatory diagnoses. Although low-grade fever is common with many types of inflammatory arthritis, higher fevers are more commonly associated with a septic joint, and the physician must rule out a septic joint with joint fluid analysis (see Table 107.2).

A history of gout makes a recurrent gout attack more likely. However, patients with gout are more susceptible to septic arthritis. Gouty arthritis is more likely than septic arthritis to be manifested in a polyarticular fashion. In general, a septic joint will be redder and warmer, whereas a gouty joint will have more fluid.

Unfortunately, laboratory tests, including synovial fluid analysis, do not diagnose or rule out septic arthritis with accuracy. Therefore, if suspicion for septic arthritis is high in the setting of negative testing, the emergency physician should not hesitate to treat for infection while awaiting the results of bacterial culture. Imaging studies are indicated when trauma, bone infection, malignancy, or a foreign body is suspected and are recommended before arthrocentesis. Ultrasonography may assist in identification of joint effusion.

**TREATMENT**

Pain management should be initiated early. After appropriate material for culture is obtained, parenteral antibiotics should be selected to treat the most likely pathogens (see Table 107.4). Another reasonable approach is to treat according to Gram stain results (for gram-positive cocci, start vancomycin; for gram-negative organisms, start ceftriaxone or cefotaxime). If the Gram stain is negative, vancomycin is reasonable for an immunocompetent host and vancomycin plus ceftriaxone (or cefotaxime) for an immunosuppressed individual, injection drug user, or traumatic bacterial arthritis.

**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION**

Hospital admission is needed, along with orthopedic consultation and repeated arthrocentesis if fluid reaccumulates. The patient should be advised about the probable diagnosis and plan of care.
GOUT AND PSEUDOGOUT

EPIDEMIOLOGY

The incidence of gout ranges from 1% to 15% in the general population and increases with age and with elevations in serum urate. In adults, serum urate levels correlate strongly with serum creatinine levels, body weight, height, age, blood pressure, and alcohol intake. Acute gout is much more common in men (peak age at onset between the fourth and sixth decades) but tends to occur at older ages in women (sixth to eighth decades). Radiography and autopsy studies have found the incidence of pseudogout to be 15% at age 65 and 50% at age 85.

PATHOPHYSIOLOGY

Gout and pseudogout are characterized by crystal deposition in joints with recurring attacks of acute inflammatory arthritis, as well as chronic arthropathy.

Although acute gout never develops in most hyperuricosuric individuals, all patients with gouty arthritis have hyperuricemia at some point. Microscopic tophaceous deposits of urate crystals develop in synovial membranes, but deposits alone are asymptomatic. Abrupt increases and decreases in serum urate levels may promote the release of free urate crystals from deposits, which have considerable proinflammatory potential because of their ability to activate synovial epithelial cells and promote the ingress of leukocytes into the joint, which triggers multiple inflammatory cascades. Precipitants include initiation of diuretics and other drugs that inhibit the excretion of uric acid (including aspirin), alcohol use, initiation of urate-lowering drugs, starvation, and tumor lysis. Repetitive joint microtrauma may produce locally increased urate concentrations, which perhaps explains the predilection for the first MTP joint.

An acute gouty attack is spontaneously self-limited (usually lasting 7 to 10 days) and probably mediated by an altered balance between proinflammatory and antiinflammatory mediators in the joint. Low-grade synovitis may persist in affected joints. Inflammation, especially with untreated disease, can lead to chronic synovial proliferation, cartilage loss, and bone erosion. Tophi commonly develop in osteoarthritic interphalangeal joints, thus suggesting a role of connective tissue matrix structure and turnover in urate crystal deposition.

Calcium-containing crystals in the pericellular matrix of cartilage are often deposited in the form of CPPD and therefore lead to a disorder termed chondrocalcinosis, pyrophosphate arthropathy, CPPD crystal deposition disease, or when associated with acute arthritis, pseudogout. Precipitation of CPPD crystals in connective tissue is most often asymptomatic. Acute attacks are generally self-limited and often triggered by trauma, surgery, or severe medical illness.

PRESENTING SIGNS AND SYMPTOMS

Acute inflammatory joint pain, swelling, erythema, and tenderness with painful limited ROM are typical. Inflammatory signs often extend beyond the involved joint and resemble cellulitis. Classically, acute gout affects the first MTP joint, but more than 50% of pseudogout attacks affect the knee. Eighty percent of first gouty attacks are monarticular. Low-grade fever is common in both diagnoses, and a previous history of the joint disease is common.

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

A known history of gout, typical precipitants, or location in the great toe suggests gout. Chondrocalcinosis (radiographic evidence of calcification in hyaline cartilage, fibrocartilage, or both) is common in pseudogout; though highly suggestive of the diagnosis, it is neither absolutely specific nor universal. Leukocytosis with a left shift and an elevated ESR may be present but are more common in pseudogout than gout. An elevated uric acid level may or may not be found in acute gout and is nondiagnostic by itself. Normal to low levels are reported in 12% to 43% of patients with acute gout attacks. The pattern of symptoms in chronic pseudogout is often similar to OA and may sometimes mimic RA. A significant minority of patients have coexisting arthritides.

Evaluation for septic arthritis is the diagnostic priority in the ED. Joint aspiration with evaluation of synovial fluid for crystals or evidence of infection is necessary, and fluid must be sent for Gram stain and culture. Urate crystals inside neutrophils diagnose gout; phagocytosed CPPD crystals are seen in pseudogout. Some patients have concurrent gout and pseudogout, with both types of crystals being found. Coexistence of crystalline and infectious arthritis in the same joint is well reported. If joint fluid analysis cannot be done, a clinical diagnosis may be made from historical and clinical data, but specificity is reduced (Table 107.5).

TREATMENT

Although acute attacks will resolve spontaneously, antiinflammatory medications are essential for more rapid relief of pain. Even though colchicine has traditionally been used for the acute treatment of gout, rheumatologists prefer NSAIDs (other than aspirin), oral prednisone (30 to 50 mg/day for 5 to 7 days), or intraarticular corticosteroids. Low-dose oral colchicine (1.2 mg and then 0.6 mg in 1 hour or 0.5 mg every 8 hours for 1 day) is safe and effective and avoids the diarrhea and vomiting that were common with higher-dose regimens. Pseudogout is treated with NSAIDs or intraarticular cortisone (or both); effective prevention is often achieved with low-dose colchicines or NSAIDs.

FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION

Drugs that lower uric acid production (e.g., allopurinol, febuxostat) or enhance excretion (e.g., probenecid) may be
### Table 107.5  Clinical Diagnostic Rule for Acute Monarticular Gout (Without Synovial Fluid Analysis)

<table>
<thead>
<tr>
<th>Factor</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>2</td>
</tr>
<tr>
<td>Previous patient-reported arthritis attack</td>
<td>2</td>
</tr>
<tr>
<td>Onset within 1 day</td>
<td>0.5</td>
</tr>
<tr>
<td>Joint redness</td>
<td>1</td>
</tr>
<tr>
<td>First metatarsophalangeal joint involvement</td>
<td>2.5</td>
</tr>
<tr>
<td>Hypertension or at least 1 cardiovascular disease</td>
<td>1.5</td>
</tr>
<tr>
<td>Serum uric acid level &gt; 5.88 mg/dL</td>
<td>3.5</td>
</tr>
</tbody>
</table>

**Interpretation of total:**

- High probability of gout: ≥8 points
- Intermediate probability: 4-8 points
- Low probability: <4 points

Arthritis
Presence or absence of the following:
- Cardinal signs of inflammation (pain, tenderness, heat, erythema, swelling)
- Functional impairment and ability to bear weight
- Constitutional and systemic symptoms
- Traumatic injury
- Risk factors for joint space infection
- Risk factors for adverse effects of the planned treatment regimen

Clearly identify the specific anatomic sites of tenderness, swelling, and erythema

Assess and document the following:
- Characteristics of pain (PQRST mnemonic)
- Full set of vital signs
- Approximate range of motion of the affected joints; level of activity
- Witnessed functional impairment, weight bearing, and gait
- Distal neurovascular status
- Patient consent, procedure note, results of a tap if arthrocentesis is performed

Document specific discharge instructions:
- Medications and potential serious side effects and drug interactions
- Adjunctive measures as indicated (splinting, rest, ice, elevation)
- Follow-up plan for any pending test results
- Recommended follow-up visits
- Need to bring all medications to follow-up visits
- Potential reasons to return to the ED for reassessment

SUGGESTED READINGS

REFERENCES
References can be found on Expert Consult @ www.expertconsult.com.
PSORIATIC ARTHRITIS

EPIDEMIOLOGY
Psoriatic arthritis (PsA) is another autoimmune seronegative spondyloarthropathy with joint, periarticular, skin, and eye involvement and is second in prevalence to RA as a progressive inflammatory arthritis. PsA occurs in 10% to 40% of patients with psoriasis; it does not have a significant gender predominance but often occurs in familial clusters. Onset typically takes place in the fourth or fifth decades of life, long after the skin disease has begun, but there is a juvenile-onset variant that usually appears before psoriasis lesions do (frequently with a positive family history).

PATHOPHYSIOLOGY
The trigger and exact pathogenesis of PsA remain unknown, but inflammation and autoimmunity both play a role, with complex interactions between infection, trauma, host genetics, and abnormal responses from several cell lines. Its histopathology resembles that of RA, but PsA is associated with less synovial hyperplasia, fewer macrophages, and greater angiogenesis, along with higher levels of cytokines. Activated CD4+ T cells are found throughout the skin lesions and synovium; CD8+ T cells also play a major role. Onset is usually insidious, though sometimes abrupt. Trauma to a joint may precipitate a flare at that site. Several patterns of joint involvement occur, most commonly an asymmetric oligoarthritis similar to reactive arthritis or a symmetric polyarthritis similar to RA. Spondylitis may be prominent. Most patients with PsA do very well, with severe and progressive disease developing in less than 25%. A rare subtype includes pustular acne and osteomyelitis. HIV-infected patients manifest a more severe and destructive form of PsA.44

PRESENTING SIGNS AND SYMPTOMS
A combination of arthritis, enthesitis, tenosynovitis, and dactylytis should be sought in patients with psoriasis or a family history of it. Common forms of PsA affect the fingers (scattered DIP, PIP, and MCP joint involvement), feet (MTP joints), and less commonly the larger peripheral joints, sternoclavicular joint, or TMJ. Spinal pain, stiffness, and sacroiliitis are common, especially in men, but often begin later in the course. Systemic symptoms are uncommon.

The scalp, umbilicus, or gluteal folds should be examined for psoriasis if skin lesions are not obvious. Nail lesions (onycholysis, ridging, pitting) are common clues. Conjunctivitis and iritis may occur. Rarely, aortic regurgitation, conduction blocks, and atlantoaxial subluxation have been reported.

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING
Although PsA may have several patterns of joint involvement, its skin lesions and nail changes assist in diagnosis. Psoriasis must be distinguished from eczema and seborrheic dermatitis. The keratoderma blennorrhagica of reactive arthritis may be difficult to distinguish from pustular psoriasis, and both diseases include conjunctivitis. The finger involvement of OA often includes Heberden and Bouchard nodes, whereas PsA does not. Vasculitis, rheumatoid nodules, and joint crystals are not a part of PsA.

Laboratory studies are generally unnecessary unless the patient is febrile and should have normal results in those with PsA. RF is negative. Joint fluid is inflammatory, with a preponderance of polymorphonuclear neutrophils. Radiographs show differences from RA. In PsA, proliferative bone formation is often seen around marginal erosions, joint fusion and periostitis may be present, and bone density is usually preserved. Patients with PsA may have erosion of the terminal phalangeal tufts (acroosteolysis), bony ankylosis, or “pencil-in-cup” deformities in the digits (whittling of the distal end and cupping of the proximal end of phalanges). The spine often shows asymmetric sacroilitis, large asymmetric syndesmophytes, and asymmetric paravertebral ossification.

TREATMENT AND FOLLOW-UP
Similar to other arthritides, most patients are managed as outpatients with NSAIDs, general measures, and primary care follow-up, unless infected. Occasionally, one NSAID may aggravate the skin lesions; if so, a different NSAID family should be tried. Flare-up in a single joint may be treated intraarticularly with a corticosteroid. Severe progressive disease warrants rheumatologic and possibly orthopedic referral. Dermatologic referral helps with skin lesions, which are treated separately with topical agents, retinoic acid derivatives, or psoralen with ultraviolet light. Poor responders (skin, joint, or both) may benefit from methotrexate or etanercept, and recent studies with anti-TNF agents show great promise. Other DMARDs and immune modulators may be useful. All immunosuppressive agents are risky in HIV-infected patients.

JUVENILE IDIOPATHIC (RHEUMATOID) ARTHRITIS

EPIDEMIOLOGY
Juvenile rheumatoid arthritis (JRA), the most common childhood connective tissue disease, occurs in patients up to 16 years of age. JRA is actually a collection of distinct inflammatory illnesses linked by synovitis, patient age, and limited knowledge of pathogenesis. Juvenile idiopathic arthritis (JIA) is the new name that has evolved for this group and includes more subtypes than found in the traditional JRA classification. Although most children do well in the long term, these arthritides frequently do result in chronic pain and significant functional and emotional disability. Prevalence in the United States is generally estimated to be 1 to 2 per 1000 children. The typical age at onset varies with the subtype, but more than half develop by the age of 5. Systemic JIA has no gender preference, but females predominate in other subtypes. In the
United States, whites and blacks appear to be at equal risk, but its incidence in Native Americans is higher.

**PATHOPHYSIOLOGY**

Causes remain unclear, but the pathogenesis includes an abnormal immune response to infection, stress, or environmental factors that is influenced by genetic background. Viral triggers have been postulated but not identified. Immuno-pathogenesis is complex. T cells respond to antigens in the synovium, especially in the setting of specific HLA types. Cellular infiltration initiates a cascade of events consisting of release of various cytokines, B-cell activation, and complement fixation. Synovitis with villous hypertrophy, vascular hyperplasia, and edema results, sometimes with pannus formation and its erosion of bone and cartilage.

**PRESENTING SIGNS AND SYMPTOMS**

JRA and JIA may be especially difficult to identify in pre-verbal age children, in whom changes in motor activities (crawling, walking, running, holding a cup or spoon) are important clues. Joint complaints in children should be taken seriously, with a close look for objective findings. Pain is usually mild and may be quantifiable with pediatric pain scales. Ask about systemic symptoms (fever, weakness, fatigue), other organ symptoms (GI, eyes, skin), growth and development, and recent injuries, illnesses, or exposures. Consider trauma (and abuse), attendant bruising, and progressive swelling during the visit. Carefully check all joints, not just the most symptomatic. Examine the child for evidence of infection or other involvement, such as rashes, uveitis, carditis, or organomegaly. Major subtypes of JIA include systemic arthritis, oligoarthritis, polyarthritis, enthesitis-related arthritis, and PsA.

Systemic arthritis (10% to 15% of cases of JIA) is the most dramatic finding, with systemic symptoms and onset usually occurring before the age of 4. Diagnostic criteria include high spiking fevers (>39°C) with daily return to normal (or below) for at least 2 weeks, arthritis that may follow the fever, and systemic involvement as shown by one or more of the following:

- Rash—evanescent, salmon-colored macules, often seen with a daily fever spike
- Generalized lymphadenopathy
- Serositis—pericarditis in more than one third, but tamponade is rare
- Hepatomegaly or splenomegaly

Systemic symptoms usually resolve within 1 year, but half will evolve into chronic polyarthritis and 25% will be result in erosive joint disease. The 15-year survival rate has been reported to be only 86%.

Oligoarthritis (35%) involves fewer than five joints in the first 6 months of disease, often large joints such as the knee or ankle but not the hip. Affected children are mostly girls (3:1) aged 1 to 4. Uveitis develops in about 15%. Although only about 25% of cases remit within 5 years, bone or cartilage destruction does not develop in most children.

Polyarthritis (30% to 40%) involves five or more joints, large and small, and resembles adult RA. Systemic symptoms are mild and fever is low grade. Rheumatoid nodules may be found. Micrognathia and cervical spine involvement occur, and there is a risk for atlantoaxial subluxation. At least two thirds are RF positive, which portends a greater risk for the development of severe erosive disease.

Additional subtypes include enthesitis-related arthritis (4:1 male preponderance, usually age >8, often HLA-B27 positive, spinal involvement) and PsA (pauciarticular, skin psoriasis or family history, nail changes, dactylitis), which often precedes any skin lesions by years. Other children demonstrate overlap patterns.45

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Unfortunately, the diagnosis of JIA can be difficult and requires a course of the disease over time, and the differential is long, so a single ED visit is highly unlikely to result in a specific diagnosis. Diagnosis of JIA requires true arthritis (swelling or at least two of the following: heat, limited ROM, tenderness, pain on motion), at least a 6-week duration, age younger than 16, and absence of another known cause. The fever pattern and rash of systemic JRA are distinctive but not conclusive.

Of course, the major concern is identifying active bacterial infection. Infectious arthritides such as Lyme disease (exposure, recent classic rash, neurologic involvement), parvovirus, Kawasaki disease (mucosal involvement), or tuberculosis should be considered. Rheumatic fever is often accompanied by exquisite joint tenderness, high spiking fevers, and migratory polyarthritis, along with chorea or carditis.

Other rheumatologic possibilities include systemic lupus erythematosus, juvenile dermatomyositis, autoimmune hepatitis, sarcoidosis, and drug reactions. Joint pain occurs with hemoglobinopathies, hemophilies, and leukemias. Localized pain may be traumatic. Diffuse musculoskeletal pain and systemic symptoms (not fever) occur with childhood fibromyalgia.46

No diagnostic studies prove the presence of JIA. CBC, urinalysis, and ESR or CRP are appropriate in the ED. In systemic JIA, severe nonhemolytic anemia, WBC count lower than 20,000/mm³, thrombocytosis, and an ESR higher than 100 mm/hr are common. In other forms, abnormalities are modest. Normal or low platelet counts suggest another diagnosis or a serious complication—rarely, macrophage activation syndrome with disseminated intravascular coagulation, severe anemia, leukopenia, and liver dysfunction develop in children with systemic JIA. Typical synovial fluid WBC counts are approximately 10,000 with JRA, but a range from 600 to 100,000 is reported. Gram stain and culture are obviously critical in evaluating infection. A febrile child requires more extensive evaluation to search for the source of the fever, particularly with a fever of unknown origin.

Radiographic studies help eliminate trauma, tumors, avascular necrosis, and infection. Abnormalities with JRA may include osteoporosis, periostitis, loss of joint space, erosions, and nonspecific soft tissue swelling. Late-stage disease shows joint destruction and fusion.
TREATMENT AND FOLLOW-UP

Initial treatment decisions in the ED usually focus on concern for differential diagnoses (trauma, infection) and the need for analgesia. If a fracture is suspected, a splint, ice, and elevation are used until trauma is ruled out. Decisions regarding antibiotic administration must be based on a thorough clinical evaluation plus the results of synovial fluid analysis.

NSAIDs are first-line medications for pain in all forms of JIA. Try ibuprofen (30 to 40 mg/kg/day divided into three doses) or naproxen (10 to 20 mg/kg/day divided into two doses). Recognize that adverse effects are somewhat different from those in adults. When the need for long-term use becomes apparent, long-acting preparations (naproietone, etoladac once a day) are available. Acetaminophen or tramadol are also effective for pain, and temporary joint immobilization or support and ice packs help. Oral opioids may be needed for severe acute situations (oxycodone, 0.05 to 2.0 mg/kg/day divided into 4-hour dosing) or severe chronic disease (methadone often used).

About half of JIA patients experience adequate improvement with NSAIDs and the multidisciplinary measures that are essential for pediatric patients with prolonged painful illnesses. Education, physical therapy, and occupational therapy are critical, especially daily ROM exercises. General health maintenance, child and family counseling, cognitive behavioral therapy to manage pain, physical conditioning, and sleep hygiene are all part of a team approach to long-term management. Children who fail to improve will require DMARDs, but these drugs are not appropriate for ED initiation. Methotrexate has been the most frequently used agent, but etanercept and infliximab are highly promising new options. Adjunctive medications include sulfasalazine and low-dose prednisone.

Disposition decisions may be more difficult than in adults, especially with febrile children. Patients should be admitted for severe pain that persists despite ED treatment, as well as for diagnostic confusion, toxic appearance, suspicion of joint or systemic infection, or fever (unless it has an obvious, easily treated source). Children who are active and comfortable in the ED are generally able to go home with close follow-up arranged.

PATHOPHYSIOLOGY

The cause of LCP disease and its inciting factors remain unknown. Disruption of vascular flow (perhaps by a cartilage disorder) to the capital femoral epiphysis causes small episodes of infarction that lead to osteochondrosis and potential collapse of the epiphysis. Later revascularization allows resorption and replacement with new bone and cartilage, usually with complete healing over a period of 3 to 4 years. Residual deformity may result in disability or severe OA years later. The process is generally unilateral, but 10% have bilateral involvement, and effusions may be present.

CLINICAL PRESENTATION

Most children have a limp and minimal or intermittent pain in the hip, groin, thigh, or knee. Stiffness, anterior tenderness, or muscle spasm may be present. Symptoms often worsen with activity and improve with rest. The onset of symptoms is usually slow and stuttering. Because the child is not usually bothered by the symptoms, LCP disease is often characterized by a “painless limp” caused by limited abduction and limited internal rotation. The child’s ROM should be examined and gait observed, although the findings are nonspecific. The usual long-term result is a variable amount of restricted motion, along with slight shortening of the limb and an insignificant limp.

DIFFERENTIAL DIAGNOSIS

Several other entities may cause hip problems and limping, and distinction may not be possible in a single ED visit. Constitutional symptoms, fever, and associated disorders are not part of LCP disease. Radiographs are often tremendously helpful in making the diagnosis, as is nuclear scintigraphy. Septic arthritis or osteomyelitis must always be considered, especially with systemic symptoms or another site of infection. Severe pain, acute onset, ESR higher than 20 mm/hr, or temperature above 37.5°C suggests a septic hip. Transient synovitis of the hip is the most common cause of hip pain, with age at peak onset similar to that for LCP disease, but it usually resolves within a month; about half are initially seen in an acute stage, unlike LCP disease. Persistent synovitis may follow a course consistent with LCP disease. Other inflammatory diseases (JRA, rheumatic fever) usually have other joint involvement or systemic findings (or both). Slipped capital femoral epiphysis (SCFE) tends to occur at older ages (12 to 16 in boys, 10 to 14 in girls). Those with stable SCFE have an intermittent limp and pain with a chronic onset, whereas unstable SCFE is often manifested acutely after a twisting injury. The diagnosis of SCFE is usually made with plain films, although CT or MRI is sometimes needed.

DIAGNOSTIC STUDIES

Laboratory studies are not useful except to look for other diseases. Anteroposterior and frog-leg lateral pelvic

LEGG-CALVÉ-PERTHES DISEASE

SCOPE

Legg-Calvé-Perthes (LCP) disease is an idiopathic pediatric form of avascular necrosis of the femoral head that may result in some flattening of the femoral head (coxa plana). It is a self-limited disease, but permanent deformity and restricted motion may result. LCP disease affects boys much more often than girls (4:1 to 5:1) and usually becomes evident between the ages of 5 and 7 (range, 2 years to teens). It is more common in shorter children with delayed bone age. A family history is positive in about 10%. The incidence of LCP disease also varies with ethnicity, being higher in Asians and central Europeans. Its relationship to trauma is unclear.
radiographs allow diagnosis and staging of LCP disease, with some help in prognosis.

**TREATMENT AND DISPOSITION**

Mild analgesics may be used as needed. Further treatment of LCP disease is done in the outpatient setting, with orthopedic consultation. “Containment” refers to reducing the pressure force on the hip by varus repositioning, with the goals of keeping the femoral head contained in the acetabulum, assisting blood flow, and molding its shape on remodeling. This is usually achieved by braces, but operative intervention may be required. Occasionally, temporary periods of rest are needed.\(^{50}\)
REFERENCES


