APPROACH TO ARTICULAR AND MUSCULOSKELETAL DISORDERS: INTRODUCTION

Musculoskeletal complaints account for >315 million outpatient visits per year. Recent surveys by the Centers for Disease Control and Prevention suggest that 33% (69.9 million) of the U.S. population is affected by arthritis or joint disorders. Many of these are self-limited conditions requiring minimal evaluation and only symptomatic therapy and reassurance. However, in some patients, specific musculoskeletal symptoms or their persistence may herald a more serious condition that requires further evaluation or laboratory testing to establish a diagnosis or document the extent and nature of the pathologic process. The goal of the musculoskeletal evaluation is to formulate a differential diagnosis that leads to an accurate diagnosis and timely therapy, while avoiding excessive diagnostic testing and unnecessary treatment (Table 325-1). There are several urgent conditions that must be diagnosed promptly to avoid significant morbid or mortal sequelae. These "red flag" diagnoses include septic arthritis, acute crystal-induced arthritis (e.g., gout), and fracture. Each may be suspected by its acute onset and monarticular or focal presentation (see below).

Table 325-1 Evaluation of Patients with Musculoskeletal Complaints

Goals
- Accurate diagnosis
- Timely provision of therapy
- Avoidance of unnecessary diagnostic testing

Approach
- Anatomic localization of complaint (articular vs. nonarticular)
- Determination of the nature of the pathologic process (inflamatory vs. noninflamatory)
- Determination of the extent of involvement (monarticular, polyarticular, focal, widespread)
- Determination of chronology (acute vs. chronic)
- Consider the most common disorders first
- Formulation of a differential diagnosis

Individuals with musculoskeletal complaints should be evaluated with a thorough history, a comprehensive physical examination, and, if appropriate, laboratory testing. The initial encounter should determine whether the musculoskeletal complaint is (1) articular or nonarticular in origin, (2) inflammatory or noninflammatory in nature, (3) acute or chronic in duration, and (4) localized or widespread (systemic) in distribution.

With such an approach and an understanding of the pathophysiologic processes that underlie musculoskeletal complaints, a diagnosis can be made in the vast majority of individuals. However, some patients will not fit immediately into an established diagnostic category. Many musculoskeletal disorders resemble each other at the outset, and some may take weeks or months to evolve into a readily
recognizable diagnostic entity. This consideration should temper the desire to establish a definitive diagnosis at the first encounter.

**ARTICULAR VERSUS NONARTICULAR**

The musculoskeletal evaluation must discriminate the anatomic origin(s) of the patient’s complaint. For example, ankle pain can result from a variety of pathologic conditions involving disparate anatomic structures, including gonococcal arthritis, calcaneal fracture, Achilles tendinitis, cellulitis, and peripheral neuropathy. Distinguishing between articular and nonarticular conditions requires a careful and detailed examination. Articular structures include the synovium, synovial fluid, articular cartilage, intraarticular ligaments, joint capsule, and juxtaarticular bone. Nonarticular (or periarticular) structures, such as supportive extraarticular ligaments, tendons, bursae, muscle, fascia, bone, nerve, and overlying skin, may be involved in the pathologic process. Although musculoskeletal complaints are often ascribed to the joints, nonarticular disorders (rather than articular) more frequently underlie such complaints. Distinguishing between these potential sources of pain may be challenging to the unskilled examiner. Articular disorders may be characterized by deep or diffuse pain, pain or limited range of motion on active and passive movement, and swelling (caused by synovial proliferation, effusion, or bony enlargement), crepitation, instability, "locking," or deformity. By contrast, nonarticular disorders tend to be painful on active, but not passive (or assisted), range of motion, demonstrate point or focal tenderness in regions adjacent to articular structures, and have physical findings remote from the joint capsule. Moreover, nonarticular disorders seldom demonstrate swelling, crepitus, instability, or deformity.

**INFLAMMATORY VERSUS NONINFLAMMATORY DISORDERS**

In the course of a musculoskeletal evaluation, the examiner should determine the nature of the underlying pathologic process and whether inflammatory or noninflammatory findings exist. Inflammatory disorders may be infectious (infection with *Neisseria gonorrhoea* or *Mycobacterium tuberculosis*), crystal-induced (gout, pseudogout), immune-related [rheumatoid arthritis (RA), systemic lupus erythematosus (SLE)], reactive (rheumatic fever, Reiter's syndrome), or idiopathic. Inflammatory disorders may be identified by any of the four cardinal signs of inflammation (erythema, warmth, pain, or swelling), systemic symptoms (fatigue, fever, rash, weight loss), or laboratory evidence of inflammation [elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP), thrombocytosis, anemia of chronic disease, or hypoalbuminemia]. Articular stiffness commonly accompanies chronic musculoskeletal disorders. However, the severity and duration of stiffness may be diagnostically important. Morning stiffness related to inflammatory disorders (such as RA or polymyalgia rheumatica) is precipitated by prolonged rest, is described as severe, lasts for hours, and may improve with activity and anti-inflammatory medications. By contrast, intermittent stiffness (also known as gel phenomenon), associated with noninflammatory conditions [such as osteoarthritis (OA)], is precipitated by brief periods of rest, usually lasts < 60 min, and is exacerbated by activity. Fatigue may accompany inflammation (as seen in RA and polymyalgia rheumatica) but may also be prominent in fibromyalgia (a noninflammatory disorder), anemia, cardiac failure, endocrinopathy, poor nutrition, poor sleep, or depression. Noninflammatory disorders may be related to trauma (rotator cuff tear), repetitive use (bursitis, tendinitis), degeneration or ineffective repair (OA), neoplasm (pigmented villonodular synovitis), or pain amplification (fibromyalgia). Noninflammatory disorders are often characterized by pain without synovial swelling or warmth, absence of inflammatory or systemic features, daytime gel phenomena rather than morning stiffness, and normal (for age) or negative laboratory investigations.

Identification of the nature of the underlying process and the site of the complaint will enable the examiner to narrow the diagnostic considerations and to assess the need for immediate diagnostic or therapeutic intervention, or for continued observation. Figure 325-1 presents a logical approach to the
evaluation of patients with musculoskeletal complaints.

**Figure 325-1**
Musculoskeletal Complaint

Initial rheumatic history and physical exam to determine
1. Is it articular?
2. Is it acute or chronic?
3. Is inflammation present?
4. How many/which joints are involved?

Nonarticular condition
Consider
• Trauma/fracture
• Fibromyalgia
• Polymyalgia rheumatica
• Bursitis
• Tendinitis

Is it articular?

Yes

Is complaint > 6 wk?

No

Acute

Chronic

Is inflammation present?
1. Is there prolonged morning stiffness?
2. Is there soft tissue swelling?
3. Are there systemic symptoms?
4. Is the ESR or CRP elevated?

No

Chronic noninflammatory arthritis

Yes

Chronic inflammatory arthritis

Are DIP, CMC1, hip or knee joints involved?

No

Unlikely to be osteoarthritis
Consider
• Osteonecrosis
• Charcot arthritis

Osteoarthritis

Yes

Chronic inflammatory mono/oligoarthritis
Consider
• Indolent infection
• Psoriatic arthritis
• Reactive arthritis
• Pauciarticular JA

How many joints involved?

1–3

>3

Are PIP, MCP, or MTP joints involved?

No

Rheumatoid arthritis

Yes

Unlikely to be rheumatoid arthritis
Consider
• SLE
• Scleroderma
• Polymyositis

Algorithm for the diagnosis of musculoskeletal complaints. An approach to formulating a differential diagnosis (shown in italics). (ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; DIP, distal interphalangeal; CMC, carpometacarpal; PIP, proximal interphalangeal; MCP, metacarpophalangeal; MTP, metatarsophalangeal; PMR, polymyalgia rheumatica; SLE, systemic lupus erythematosus; JA, juvenile arthritis.)

In the formulation of a differential diagnosis, the examiner should be mindful of the most common causes of musculoskeletal complaints (Fig. 325-2). Thus, the prevalence of these disorders in the general population may facilitate an early diagnosis. As trauma, fracture, and fibromyalgia are among the most common causes of presentation, these should be considered during the initial encounter. The frequency of these disorders is best clarified by dividing patients according to their age. Hence, those <60 years are commonly affected by repetitive use/strain disorders, gout (men only), RA, spondyloarthritis, and infectious arthritis. Patients >60 years are frequently affected by OA, crystal (gout and pseudogout) arthritis, polymyalgia rheumatica, osteoporotic fracture, and septic arthritis.

**Figure 325-2**

![MOST COMMON MUSCULOSKELETAL CONDITIONS](image)


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Algorithm for consideration of the most common musculoskeletal conditions. (IBD, inflammatory bowel disease; GC, gonococcal.)

**Clinical History**

Additional historic features may reveal important clues to the diagnosis. Aspects of the patient profile,
complaint chronology, extent of joint involvement, and precipitating factors can provide important information. Certain diagnoses are more frequent in different age groups (Fig. 325-2). SLE and reactive arthritis occur more frequently in the young, whereas fibromyalgia and RA are frequent in middle age and OA and polymyalgia rheumatica are more prevalent among the elderly. Diagnostic clustering is also evident when sex and race are considered. Gout and the spondyloarthropathies (e.g., ankylosing spondylitis) are more common in men, whereas RA, fibromyalgia, and lupus are more frequent in women. Racial predilections may be influential. Thus, polymyalgia rheumatica, giant cell arteritis, and Wegener's granulomatosis commonly affect whites, whereas sarcoidosis and SLE more commonly affect African Americans. Familial aggregation may be seen in disorders such as ankylosing spondylitis, gout, and Heberden's nodes of OA.

The chronology of the complaint is an important diagnostic feature and can be divided into the onset, evolution, and duration. The onset of disorders such as septic arthritis or gout tends to be abrupt, whereas OA, RA, and fibromyalgia may have more indolent presentations. The patients' complaints may evolve differently and be classified as chronic (OA), intermittent (crystal or Lyme arthritis), migratory (rheumatic fever, gonococcal or viral arthritis), or additive (RA, psoriatic arthritis). Musculoskeletal disorders are typically classified as acute or chronic based upon a symptom duration that is either less than or greater than 6 weeks, respectively. Acute arthropathies tend to be infectious, crystal-induced, or reactive. Chronic conditions include noninflammatory or immunologic arthritides (e.g., OA, RA) and nonarticular disorders (e.g., fibromyalgia).

The extent of articular involvement is often diagnostic. Articular disorders are classified based on the number of joints involved, as either monarticular (one joint), oligoarticular or pauciarticular (two or three joints), or polyarticular (more than three joints). Although crystal and infectious arthritis are often mono- or oligoarticular, OA and RA are polyarticular disorders. Nonarticular disorders may be classified as either focal or widespread. Complaints secondary to tendinitis or carpal tunnel syndrome are typically focal, whereas weakness and myalgia, due to polymyositis or fibromyalgia, are more diffuse in their presentation. Joint involvement in RA tends to be symmetric, whereas the spondyloarthropathies and gout are often asymmetric and oligoarticular. The upper extremities are frequently involved in RA and OA, whereas lower extremity arthritis is characteristic of reactive arthritis and gout at their onset. Involvement of the axial skeleton is common in OA and ankylosing spondylitis but is infrequent in RA, with the notable exception of the cervical spine.

The clinical history should also identify precipitating events, such as trauma, drug administration (Table 325-2), or antecedent or intercurrent illnesses, that may have contributed to the patient's complaint. Certain comorbidities may predispose to musculoskeletal consequences. This is especially so for diabetes mellitus (carpal tunnel syndrome), renal insufficiency (gout), psoriasis (psoriatic arthritis), myeloma (low back pain), cancer (myositis), and osteoporosis (fracture) or when using certain drugs such as glucocorticoids (osteonecrosis, septic arthritis) and diuretics or chemotherapy (gout). Lastly, a thorough rheumatic review of systems may disclose useful diagnostic information. A variety of musculoskeletal disorders may be associated with systemic features such as fever (SLE, infection), rash (SLE, psoriatic arthritis), nail abnormalities (psoriatic or reactive arthritis), myalgias (fibromyalgia, myopathy), or weakness (polymyositis, neuropathy). In addition, some conditions are associated with involvement of other organ systems including the eyes (Behçet's disease, sarcoidosis, spondyloarthritis), gastrointestinal tract (scleroderma, inflammatory bowel disease), genitourinary tract (reactive arthritis, gonococcemia), or the nervous system (Lyme disease, vasculitis).

| Table 325-2 Drug-Induced Musculoskeletal Conditions |
Lastly, the examiner should assess the level of pain and physical limitation that accompanies the complaint. The intensity of the patient's pain, stiffness, or weakness can be quantified (0-10) verbally or with the use of a 10-cm visual analogue scale (0 = no pain and 10 = the worst possible pain). Functional limitation and disability should be identified and recorded for future comparisons. There are several validated functional measures that are easily incorporated into the musculoskeletal evaluation, such as the modified Health Assessment Questionnaire (Fig. 325-3).

**Table 325-3**

### Arthralgias
Quinidine, cimetidine, quinolones, chronic acyclovir, interferon, IL-2, nicardipine, vaccines, rifabutin, aromatase and HIV protease inhibitors

### Myalgias/myopathy
Glucocorticoids, penicillamine, hydroxychloroquine, AZT, lovastatin, simvastatin, pravastatin, clofibrate, interferon, IL-2, alcohol, cocaine, taxol, docetaxel, colchicine, quinolones, cyclosporine

### Tendon rupture
Quinolones, glucocorticoids

### Gout
Diuretics, aspirin, cytotoxics, cyclosporine, alcohol, moonshine, ethambutol

### Drug-induced lupus
Hydralazine, procainamide, quinidine, phenytoin, carbemazepine, methyldopa, isoniazid, chlorpromazine, lithium, penicillamine, tetracyclines, TNF inhibitors, ACE inhibitors, ticlopidine

### Osteonecrosis
Glucocorticoids, alcohol, radiation, bisphosphonates

### Osteopenia
Glucocorticoids, chronic heparin, phenytoin, methotrexate

### Scleroderma
Vinyl chloride, bleomycin, pentazocine, organic solvents, carbidopa, tryptophan, rapeseed oil

### Vasculitis
Allopurinol, amphetamines, cocaine, thiazides, penicillamine, propylthiouracil, montelukast, TNF inhibitors, hepatitis B vaccine, trimethoprim/sulfamethoxazole

**Note:** IL-2, interleukin 2; TNF, tumor necrosis factor; ACE, angiotensin-converting enzyme.

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RHEUMATOLOGIC EVALUATION OF THE ELDERLY

The incidence of rheumatic diseases rises with age, such that 58% of those >65 years will have joint complaints. Musculoskeletal disorders in elderly patients are often not diagnosed because the signs and symptoms may be insidious, overlooked, or overshadowed by comorbidities. These difficulties are compounded by the diminished reliability of laboratory testing in the elderly, who often manifest nonpathologic abnormal results. For example, the ESR may be misleadingly elevated, and low-titer positive tests for rheumatoid factor and antinuclear antibodies (ANAs) may be seen in up to 15% of elderly patients. Although nearly all rheumatic disorders afflict the elderly, certain diseases and drug-induced disorders (Table 325-2) are more common in this age group. The elderly should be approached in the same manner as other patients with musculoskeletal complaints, but with an emphasis on identifying the potential rheumatic consequences of medical comorbidities and therapies. OA, osteoporosis, gout, pseudogout, polymyalgia rheumatica, vasculitis, drug-induced SLE, and chronic salicylate toxicity are all more common in the elderly than in other individuals. The physical examination should identify the nature of the musculoskeletal complaint as well as coexisting diseases that may influence diagnosis and choice of treatment.

PHYSICAL EXAMINATION

The goal of the physical examination is to ascertain the structures involved, the nature of the underlying pathology, the functional consequences of the process, and the presence of systemic or extraarticular manifestations. A knowledge of topographic anatomy is necessary to identify the primary site(s) of involvement and differentiate articular from nonarticular disorders. The musculoskeletal examination depends largely on careful inspection, palpation, and a variety of specific physical maneuvers to elicit diagnostic signs (Table 325-3). Although most articulations of the appendicular skeleton can be examined in this manner, adequate inspection and palpation are not possible for many axial (e.g.,

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### Modified Health Assessment Questionnaire

<table>
<thead>
<tr>
<th>Today are you able to (check box)</th>
<th>No difficulty</th>
<th>Some difficulty</th>
<th>Much difficulty</th>
<th>Cannot do</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dress yourself; including laces &amp; buttons?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Get in and out of bed?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lift a full cup or glass to your mouth?</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Walk outdoors on flat ground?</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Wash and dry your entire body?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bend down &amp; pick up clothing from floor?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Turn regular faucets on and off?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Get in and out of a car?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


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Modified Health Assessment Questionnaire. *(From TPincus et al: Arthritis Rheum 26:1346, 1983; with permission.)*
zygapophyseal) and inaccessible (e.g., sacroiliac or hip) joints. For such joints, there is a greater reliance upon specific maneuvers and imaging for assessment.

Table 325-3 Glossary of Musculoskeletal Terms

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crepitus</td>
<td>A palpable (less commonly audible) vibratory or crackling sensation elicited with joint motion; fine joint crepitus is common and often insignificant in large joints; coarse joint crepitus indicates advanced cartilaginous and degenerative changes (as in osteoarthritis)</td>
</tr>
<tr>
<td>Subluxation</td>
<td>Alteration of joint alignment such that articulating surfaces incompletely approximate each other</td>
</tr>
<tr>
<td>Dislocation</td>
<td>Abnormal displacement of articulating surfaces such that the surfaces are not in contact</td>
</tr>
<tr>
<td>Range of motion</td>
<td>For diarthrodial joints, the arc of measurable movement through which the joint moves in a single plane</td>
</tr>
<tr>
<td>Contracture</td>
<td>Loss of full movement resulting from a fixed resistance caused either by tonic spasm of muscle (reversible) or to fibrosis of periarticular structures (permanent)</td>
</tr>
<tr>
<td>Deformity</td>
<td>Abnormal shape or size of a structure; may result from bony hypertrophy, malalignment of articulating structures, or damage to periarticular supportive structures</td>
</tr>
<tr>
<td>Enthesitis</td>
<td>Inflammation of the entheses (tendinous or ligamentous insertions on bone)</td>
</tr>
<tr>
<td>Epicondylitis</td>
<td>Infection or inflammation involving an epicondyle</td>
</tr>
</tbody>
</table>

Examination of involved and uninvolved joints will determine whether pain, warmth, erythema, or swelling is present. The locale and level of pain elicited by palpation or movement should be quantified. One example would be to count the number of tender joints on palpation of 28 easily examined joints [proximal interphalangeals (PIPs), metacarpophalangeals (MCPs), wrists, elbows, shoulders, and knees] (with a range of 0–28). Similarly, the number of swollen joints (0–28) can be counted and recorded. Careful examination should distinguish between true articular swelling (caused by synovial effusion or synovial proliferation) and nonarticular (or periarticular) involvement, which usually extends beyond the normal joint margins. Synovial effusion can be distinguished from synovial hypertrophy or bony hypertrophy by palpation or specific maneuvers. For example, small to moderate knee effusions may be identified by the "bulge sign" or "ballottement of the patellae." Bursal effusions (e.g., effusions of the olecranon or prepatellar bursa) are often focal, periarticular, overlie bony prominences, and are fluctuant with sharply defined borders. Joint stability can be assessed by palpation and by the application of manual stress. Subluxation or dislocation, which may be secondary to traumatic, mechanical, or inflammatory causes, can be assessed by inspection and palpation. Joint swelling or volume can be assessed by palpation. Distention of the articular capsule usually causes pain and evident swelling. The patient will attempt to minimize the pain by maintaining the joint in the position of least intraarticular pressure and greatest volume, usually partial flexion. For this reason, inflammatory effusions may give rise to flexion contractures. Clinically, this may be detected as fluctuant or "squishy" swelling, voluntary or fixed flexion deformities, or diminished range of motion—especially on extension, when joint volumes
are decreased. Active and passive range of motion should be assessed in all planes, with contralateral comparison. Serial evaluations of the joints should record the number of tender and swollen joints and the range of motion, using a goniometer to quantify the arc of movement. Each joint should be passively manipulated through its full range of motion (including, as appropriate, flexion, extension, rotation, abduction, adduction, lateral bending, inversion, eversion, supination, pronation, medial/lateral deviation, plantar- or dorsiflexion). Limitation of motion is frequently caused by effusion, pain, deformity, or contracture. If passive motion exceeds active motion, a periarticular process (e.g., tendon rupture or myopathy) should be considered. Contractures may reflect antecedent synovial inflammation or trauma. Joint crepitus may be felt during palpation or maneuvers and may be especially coarse in OA. Joint deformity usually indicates a long-standing or aggressive pathologic process. Deformities may result from ligamentous destruction, soft tissue contracture, bony enlargement, ankylosis, erosive disease, or subluxation. Examination of the musculature will document strength, atrophy, pain, or spasm. Appendicular muscle weakness should be characterized as proximal or distal. Muscle strength should be assessed by observing the patient's performance (e.g., walking, rising from a chair, grasping, writing). Strength may also be graded on a 5-point scale: 0 for no movement; 1 for trace movement or twitch; 2 for movement with gravity eliminated; 3 for movement against gravity only; 4 for movement against gravity and resistance; and 5 for normal strength. The examiner should assess for often-overlooked nonarticular or periarticular involvement, especially when articular complaints are not supported by objective findings referable to the joint capsule. The identification of soft tissue/nonarticular pain will prevent unwarranted and often expensive additional evaluations. Specific maneuvers may reveal common nonarticular abnormalities, such as a carpal tunnel syndrome (which can be identified by Tinel's or Phalen's sign). Other examples of soft tissue abnormalities include olecranon bursitis, epicondylitis (e.g., tennis elbow), enthesitis (e.g., Achilles tendinitis), and trigger points associated with fibromyalgia.

**APPROACH TO REGIONAL RHEUMATIC COMPLAINTS**

Although all patients should be evaluated in a logical and thorough manner, many cases with focal musculoskeletal complaints are caused by commonly encountered disorders that exhibit a predictable pattern of onset, evolution, and localization; they can often be diagnosed immediately on the basis of limited historic information and selected maneuvers or tests. Although nearly every joint could be approached in this manner, the evaluation of four common involved anatomic regions—the hand, shoulder, hip, and knee—are reviewed here.

**Hand Pain**

Focal or unilateral hand pain may result from trauma, overuse, infection, or a reactive or crystal-induced arthritis. By contrast, bilateral hand complaints commonly suggest a degenerative (e.g., OA), systemic, or inflammatory/immune (e.g., RA) etiology. The distribution or pattern of joint involvement is highly suggestive of certain disorders (Fig. 325-4). Thus, OA (or degenerative arthritis) may manifest as distal interphalangeal (DIP) and PIP joint pain with bony hypertrophy sufficient to produce Heberden's and Bouchard's nodes, respectively. Pain, with or without bony swelling, involving the base of the thumb (first carpometacarpal joint) is also highly suggestive of OA. By contrast, RA tends to involve the PIP, MCP, intercarpal, and carpometacarpal joints (wrist) with pain, prolonged stiffness, and palpable synovial tissue hypertrophy. Psoriatic arthritis may mimic the pattern of joint involvement seen in OA (DIP and PIP joints), but can be distinguished by the presence of inflammatory signs (erythema, warmth, synovial swelling), with or without carpal involvement, nail pitting or onycholysis. Hemochromatosis should be considered when degenerative changes (bony hypertrophy) are seen at the second and third MCP joints with associated chondrocalcinosis or episodic, inflammatory wrist arthritis.

**Figure 325-4**
Soft tissue swelling over the dorsum of the hand and wrist may suggest an inflammatory extensor tendon tenosynovitis possibly caused by gonococcal infection, gout, or inflammatory arthritis (e.g., RA). The diagnosis is suggested by local warmth, swelling, or pitting edema and may be confirmed when pain is induced by maintaining the wrist in a fixed, neutral position and flexing the digits distal to the MCP joints to stretch the extensor tendon sheaths.

Focal wrist pain localized to the radial aspect may be caused by De Quervain's tenosynovitis resulting from inflammation of the tendon sheath(s) involving the abductor pollicis longus or extensor pollicis brevis (Fig. 325-4). This commonly results from overuse or follows pregnancy and may be diagnosed with Finkelstein's test. A positive result is present when radial wrist pain is induced after the thumb is flexed and placed inside a clenched fist and the patient actively deviates the hand downward with ulnar deviation at the wrist. Carpal tunnel syndrome is another common disorder of the upper extremity and results from compression of the median nerve within the carpal tunnel. Manifestations include paresthesia in the thumb, second and third fingers, and radial half of the fourth finger and, at times, atrophy of thenar musculature. Carpal tunnel syndrome is commonly associated with pregnancy, edema, trauma, OA, inflammatory arthritis, and infiltrative disorders (e.g., amyloidosis). The diagnosis is suggested by a positive Tinel’s or Phalen’s sign. With each test, paresthesia in a median nerve...
distribution is induced or increased by either "thumping" the volar aspect of the wrist (Tinel's sign) or pressing the extensor surfaces of both flexed wrists against each other (Phalen's sign).

**Shoulder Pain**

During the evaluation of shoulder disorders, the examiner should carefully note any history of trauma, fibromyalgia, infection, inflammatory disease, occupational hazards, or previous cervical disease. In addition, the patient should be questioned as to the activities or movement(s) that elicit shoulder pain. Shoulder pain is referred frequently from the cervical spine but may also be referred from intrathoracic lesions (e.g., a Pancoast tumor) or from gall bladder, hepatic, or diaphragmatic disease. Fibromyalgia should be suspected when glenohumeral pain is accompanied by diffuse periarticular (i.e., subacromial, bicipital) pain and tender points (i.e., trapezius or supraspinatus). The shoulder should be put through its full range of motion both actively and passively (with examiner assistance): forward flexion, extension, abduction, adduction, and rotation. Manual inspection of the periarticular structures will often provide important diagnostic information. The examiner should apply direct manual pressure over the subacromial bursa that lies lateral to and immediately beneath the acromion. Subacromial bursitis is a frequent cause of shoulder pain. Anterior to the subacromial bursa, the bicipital tendon traverses the bicipital groove. This tendon is best identified by palpating it in its groove as the patient rotates the humerus internally and externally. Direct pressure over the tendon may reveal pain indicative of bicipital tendinitis. Palpation of the acromioclavicular joint may disclose local pain, bony hypertrophy, or, uncommonly, synovial swelling. Whereas OA and RA commonly affect the acromioclavicular joint, OA seldom involves the glenohumeral joint, unless there is a traumatic or occupational cause. The glenohumeral joint is best palpated anteriorly by placing the thumb over the humeral head (just medial and inferior to the coracoid process) and having the patient rotate the humerus internally and externally. Pain localized to this region is indicative of glenohumeral pathology. Synovial effusion or tissue is seldom palpable but, if present, may suggest infection, RA, or an acute tear of the rotator cuff.

Rotator cuff tendinitis or tear is a very common cause of shoulder pain. The rotator cuff is formed by the tendons of the supraspinatus, infraspinatus, teres minor, and subscapularis muscles. Rotator cuff tendinitis is suggested by pain on active abduction (but not passive abduction), pain over the lateral deltoid muscle, night pain, and evidence of the impingement sign. This maneuver is performed by the examiner raising the patient’s arm into forced flexion while stabilizing and preventing rotation of the scapula. A positive sign is present if pain develops before 180° of forward flexion. A complete tear of the rotator cuff is more common in the elderly and often results from trauma; it may manifest in the same manner as tendinitis but is less common. The diagnosis is also suggested by the drop arm test in which the patient is unable to maintain his or her arm outstretched once it is passively abducted. If the patient is unable to hold the arm up once 90° of abduction is reached, the test is positive. Tendinitis or tear of the rotator cuff can be confirmed by MRI or ultrasound.

**Knee Pain**

A careful history should delineate the chronology of the knee complaint and whether there are predisposing conditions, trauma, or medications that might underlie the complaint. For example, patellofemoral disease (e.g., OA) may cause anterior knee pain that worsens with climbing stairs. Observation of the patient’s gait is also important. The knee should be carefully inspected in the upright (weight-bearing) and prone positions for swelling, erythema, contusion, laceration, or malalignment. The most common form of malalignment in the knee is genu varum (bowlegs) or genu valgum (knock knees). Bony swelling of the knee joint commonly results from hypertrophic osseous changes seen with disorders such as OA and neuropathic arthropathy. Swelling caused by hypertrophy of the synovium or synovial effusion may manifest as a fluctuant, ballotable, or soft tissue enlargement in the suprapatellar
pouch (suprapatellar reflection of the synovial cavity) or regions lateral and medial to the patella.

Synovial effusions may also be detected by balloting the patella downward toward the femoral groove or by eliciting a “bulge sign.” With the knee extended the examiner should manually compress, or “milk,” synovial fluid down from the suprapatellar pouch and lateral to the patellae. The application of manual pressure lateral to the patella may cause an observable shift in synovial fluid (bulge) to the medial aspect. The examiner should note that this maneuver is only effective in detecting small to moderate effusions (< 100 mL). Inflammatory disorders such as RA, gout, pseudogout, and reactive arthritis may involve the knee joint and produce significant pain, stiffness, swelling, or warmth. A popliteal or Baker's cyst is best palpated with the knee partially flexed and is best viewed posteriorly with the patient standing and knees fully extended to visualize popliteal swelling or fullness.

Anserine bursitis is an often missed periarticular cause of knee pain in adults. The pes anserine bursa underlies the semimembranosus tendon and may become inflamed and painful following trauma, overuse, or inflammation. It is often tender in patients with fibromyalgia. Anserine bursitis manifests primarily as point tenderness inferior and medial to the patella and overlies the medial tibial plateau. Swelling and erythema may not be present. Other forms of bursitis may also present as knee pain. The prepatellar bursa is superficial and is located over the inferior portion of the patella. The infrapatellar bursa is deeper and lies beneath the patellar ligament before its insertion on the tibial tubercle.

Internal derangement of the knee may result from trauma or degenerative processes. Damage to the meniscal cartilage (medial or lateral) frequently presents as chronic or intermittent knee pain. Such an injury should be suspected when there is a history of trauma or athletic activity and when the patient relates symptoms of "locking," clicking, or "giving way" of the joint. Pain may be elicited during palpation over the ipsilateral joint line or when the knee is stressed laterally or medially. A positive McMurray test may indicate a meniscal tear. To perform this test, the knee is first flexed at 90°, and the leg is then extended while the lower extremity is simultaneously torqued medially or laterally. A painful click during inward rotation may indicate a lateral meniscus tear, and pain during outward rotation may indicate a tear in the medial meniscus. Lastly, damage to the cruciate ligaments should be suspected with acute onset of pain, possibly with swelling, a history of trauma, or a synovial fluid aspirate that is grossly bloody. Examination of the cruciate ligaments is best accomplished by eliciting a drawer sign. With the patient recumbent, the knee should be partially flexed and the foot stabilized on the examining surface. The examiner should manually attempt to displace the tibia anteriorly or posteriorly with respect to the femur. If anterior movement is detected, then anterior cruciate ligament damage is likely. Conversely, significant posterior movement may indicate posterior cruciate damage. Contralateral comparison will assist the examiner in detecting significant anterior or posterior movement.

**Hip Pain**

The hip is best evaluated by observing the patient’s gait and assessing range of motion. The vast majority of patients reporting "hip pain" localize their pain unilaterally to the posterior or gluteal musculature (Fig. 325-5). Such pain may or may not be associated with low back pain and tends to radiate down the posterolateral aspect of the thigh. This presentation frequently results from degenerative arthritis of the lumbosacral spine and commonly follows a dermatomal distribution with involvement of nerve roots between L5 and S1. Some individuals instead localize their "hip pain" laterally to the area overlying the trochanteric bursa. Because of the depth of this bursa, swelling and warmth are usually absent. Diagnosis of trochanteric bursitis can be confirmed by inducing point tenderness over the trochanteric bursa. Gluteal and trochanteric pain may also indicate underlying fibromyalgia. Range of movement may be limited by pain. Pain in the hip joint is less common and tends to be located anteriorly, over the inguinal ligament; it may radiate medially to the groin or along the anteromedial
thigh. Uncommonly, iliopsoas bursitis may mimic true hip joint pain. Diagnosis of iliopsoas bursitis may be suggested by a history of trauma or inflammatory arthritis. Pain associated with iliopsoas bursitis is localized to the groin or anterior thigh and tends to worsen with hyperextension of the hip; many patients prefer to flex and externally rotate the hip to reduce the pain from a distended bursa.

**Figure 325-5**

![Diagram of the lower body with anatomic labels](Image)

**Laboratory Investigations**

The vast majority of musculoskeletal disorders can be easily diagnosed by a complete history and physical examination. An additional objective of the initial encounter is to determine whether additional investigations or immediate therapy are required. A number of features indicate the need for additional evaluation. Monarticular conditions require additional evaluation, as do traumatic or inflammatory conditions and conditions accompanied by neurologic changes or systemic manifestations of serious disease. Finally, individuals with chronic symptoms (>6 weeks), especially when there has been a lack of response to symptomatic measures, are candidates for additional evaluation. The extent and nature of the additional investigation should be dictated by the clinical features and suspected pathologic process. Laboratory tests should be used to confirm a specific clinical diagnosis and not be used to screen or evaluate patients with vague rheumatic complaints. Indiscriminate use of broad batteries of diagnostic tests and radiographic procedures is rarely a useful or cost-effective means to establish a diagnosis.

Besides a complete blood count, including a white blood cell (WBC) and differential count, the routine evaluation should include a determination of an acute-phase reactant such as the ESR or CRP, which can be useful in discriminating inflammatory from noninflammatory disorders. Both are inexpensive and
easily obtained and may be elevated with infection, inflammation, autoimmune disorders, neoplasia, pregnancy, renal insufficiency, and advanced age.

Serum uric acid determinations are useful only when gout has been diagnosed and therapy contemplated. Uric acid, the end product of purine metabolism, is primarily excreted in the urine. Serum values range from 238–516 μmol/L (4.0–8.6 mg/dL) in men; the lower values [178–351 μmol/L (3.0–5.9 mg/dL)] seen in women are caused by the uricosuric effects of estrogen. Urinary uric acid levels are normally <750 mg per 24 h. Although hyperuricemia [especially levels >535 μmol/L (9 mg/dL)] is associated with an increased incidence of gout and nephrolithiasis, levels do not correlate with the severity of disease. Uric acid levels (and the risk of gout) may be increased by inborn errors of metabolism (Lesch-Nyhan syndrome), disease states (renal insufficiency, myeloproliferative disease, psoriasis), or drugs (alcohol, cytotoxic therapy, thiazides). Although nearly all patients with gout will demonstrate hyperuricemia at some time during their illness, up to 40% of patients with an acute gouty attack will have normal serum uric acid levels. Monitoring serum uric acid may be useful in assessing the response to hypouricemic therapy or chemotherapy.

Serologic tests for rheumatoid factor, cyclic citrullinated peptide (CCP) antibodies, ANAs, complement levels, Lyme and antineutrophil cytoplasmic antibodies (ANCA), or antistreptolysin O (ASO) titer should be carried out only when there is clinical evidence to suggest an associated diagnosis, as these have poor predictive value when used for screening, especially when the pretest probability is low. Although 4–5% of a healthy population will have positive tests for rheumatoid factor and ANAs, only 1% and <0.4% of the population will have RA or SLE, respectively. IgM rheumatoid factor (autoantibodies against the Fc portion of IgG) is found in 80% of patients with RA and may also be seen in low titers in patients with chronic infections (tuberculosis, leprosy); other autoimmune diseases (SLE, Sjögren's syndrome); and chronic pulmonary, hepatic, or renal diseases. When considering RA, anti-CCP antibodies are comparably sensitive but more specific than rheumatoid factor. In RA, the presence of anti-CCP and rheumatoid factor antibodies may indicate a greater risk for more severe, erosive polyarthritis. ANAs are found in nearly all patients with SLE and may also be seen in patients with other autoimmune diseases (polymyositis, scleroderma, antiphospholipid syndrome), drug-induced lupus (resulting from hydralazine, procainamide, quinidine, tetracyclines, tumor necrosis factor inhibitors), chronic liver or renal disorders, and advanced age. Positive ANAs are found in 5% of adults and in up to 14% of elderly or chronically ill individuals. The ANA test is very sensitive but poorly specific for lupus, as <5% of all positive results will be caused by lupus alone. The interpretation of a positive ANA test may depend on the magnitude of the titer and the pattern observed by immunofluorescence microscopy (Table 325-4). Diffuse and speckled patterns are least specific, whereas a peripheral, or rim, pattern [related to autoantibodies against double-stranded (native) DNA] is highly specific and suggestive of lupus. Centromeric patterns are seen in patients with limited scleroderma (CREST syndrome) or primary biliary sclerosis, and nucleolar patterns may be seen in patients with diffuse systemic sclerosis or inflammatory myositis.

<table>
<thead>
<tr>
<th>ANA Pattern</th>
<th>Antigen Identified</th>
<th>Clinical Correlate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diffuse</td>
<td>Deoxyribonucleoprotein</td>
<td>Nonspecific</td>
</tr>
<tr>
<td></td>
<td>Histones</td>
<td>Drug-induced lupus, lupus</td>
</tr>
<tr>
<td>Peripheral (rim)</td>
<td>ds-DNA</td>
<td>50% of SLE (specific)</td>
</tr>
<tr>
<td>Speckled</td>
<td>U1-RNP</td>
<td>&gt;90% of MCTD</td>
</tr>
<tr>
<td></td>
<td>Sm</td>
<td>30% of SLE (specific)</td>
</tr>
</tbody>
</table>
Aspiration and analysis of synovial fluid are always indicated in acute monarthritis or when an infectious or crystal-induced arthropathy is suspected. Synovial fluid may distinguish between noninflammatory and inflammatory processes by analysis of the appearance, viscosity, and cell count. Tests for synovial fluid glucose, protein, lactate dehydrogenase, lactic acid, or autoantibodies are not recommended as they have no diagnostic value. Normal synovial fluid is clear or a pale straw color and is viscous, primarily because of the high levels of hyaluronate. Noninflammatory synovial fluid is clear, viscous, and amber-colored, with a white blood cell count of $<2000/\mu L$ and a predominance of mononuclear cells. The viscosity of synovial fluid is assessed by expressing fluid from the syringe one drop at a time. Normally, there is a stringing effect, with a long tail behind each synovial drop. Effusions caused by OA or trauma will have normal viscosity. Inflammatory fluid is turbid and yellow, with an increased white cell count (2000–50,000/\mu L) and a polymorphonuclear leukocyte predominance. Inflammatory fluid has reduced viscosity, diminished hyaluronate, and little or no tail following each drop of synovial fluid. Such effusions are found in RA, gout, and other inflammatory arthritides. Septic fluid is opaque and purulent, with a WBC count usually $>50,000/\mu L$, a predominance of polymorphonuclear leukocytes (>75%), and low viscosity. Such effusions are typical of septic arthritis but may occur with RA or gout. In addition, hemorrhagic synovial fluid may be seen with trauma, hemarthrosis, or neuropathic arthritis. An algorithm for synovial fluid aspiration and analysis is shown in Fig. 325-6. Synovial fluid should be analyzed immediately for appearance, viscosity, and cell count. Monosodium urate crystals (observed in gout) are seen by polarized microscopy and are long, needle-shaped, negatively birefringent, and usually intracellular. In chondrocalcinosis and pseudogout, calcium pyrophosphate dihydrate crystals are usually short, rhomboid-shaped, and positively birefringent. Whenever infection is suspected, synovial fluid should be Gram-stained and cultured appropriately. If gonococcal arthritis is suspected, immediate plating of the fluid on appropriate culture medium is indicated. Synovial fluid from patients with chronic monarthritis should also be cultured for *M. tuberculosis* and fungi. Last, it should be noted that crystal-induced and septic arthritis occasionally occur together in the same joint.

**Figure 325-6**
INTERPRETATION OF SYNOVIAL FLUID ASPIRATION

Strongly consider synovial fluid aspiration and analysis if there is
- Monarthritis (acute or chronic)
- Trauma with joint effusion
- Monarthritis in a patient with chronic polyarthritis
- Suspicion of joint infection, crystal-induced arthritis, or hemorrhosis

Analyze fluid for
- Appearance, viscosity
- WBC count, differential
- Gram stain, culture, and sensitivity (if indicated)
- Crystal identification by polarized microscopy

Is the effusion hemorrhagic?

No

Inflammatory or noninflammatory articular condition

Is the WBC > 2000/µL?

No

Consider noninflammatory articular conditions
- Osteoarthritis
- Trauma
- Other

Yes

Consider inflammatory or septic arthritis

Is the % PMNs > 75%?

No

Are crystals present?

No

Consider other inflammatory or septic arthritides
- Gram stain, culture mandatory

Yes

Crystal identification for specific diagnosis
- Gout
- Pseudogout

Is the WBC > 50,000/µL?

No

Possible septic arthritis

Probable inflammatory arthritis

Yes

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CONVENTIONAL RADIOGRAPHY has been a valuable tool in the diagnosis and staging of articular disorders. Plain x-rays are most appropriate when there is a history of trauma, suspected chronic infection, progressive disability, or monarticular involvement; when therapeutic alterations are considered; or when a baseline assessment is desired for what appears to be a chronic process. However, in acute inflammatory arthritis, early radiography is rarely helpful in establishing a diagnosis and may only reveal soft tissue swelling or juxtaarticular demineralization. As the disease progresses, calcification (of soft tissues, cartilage, or bone), joint space narrowing, erosions, bony ankylosis, new bone formation (sclerosis, osteophytes, or periostitis), or subchondral cysts may develop and suggest specific clinical entities. Consultation with a radiologist will help define the optimal imaging modality, technique, or positioning and prevent the need for further studies.

Additional imaging techniques may possess greater diagnostic sensitivity and facilitate early diagnosis in a limited number of articular disorders and in selected circumstances and are indicated when conventional radiography is inadequate or nondiagnostic (Table 325-5). Ultrasonography is useful in the detection of soft tissue abnormalities that cannot be fully appreciated by clinical examination. Although inexpensive, it is seldom the preferred method of evaluation. The foremost application of ultrasound is in the diagnosis of synovial (Baker’s) cysts, although rotator cuff tears and various tendon injuries may be evaluated with ultrasound by an experienced operator. Radionuclide scintigraphy provides useful information regarding the metabolic status of bone and, along with radiography, is well suited for total-body assessment of the extent and distribution of skeletal involvement. Radionuclide imaging is a very sensitive, but poorly specific, means of detecting inflammatory or metabolic alterations in bone or periartricular soft tissue structures. The limited tissue contrast resolution of scintigraphy may obscure the distinction between a bony or periartricular process and may necessitate the additional use of MRI. Scintigraphy, using $^{99m}$Tc, $^{67}$Ga, or $^{111}$In-labeled WBCs has been applied to a variety of articular disorders with variable success (Table 325-5). Although $^{99m}$Tc pertechnate or diphosphate scintigraphy (Fig. 325-7) may be useful in identifying osseous infection, neoplasia, inflammation, increased blood flow, bone remodeling, heterotopic bone formation, or avascular necrosis, MRI is preferred in most instances. The poor specificity of $^{99m}$Tc scanning has largely limited its use to surveys for bone metastases and Paget’s disease of bone. Gallium scanning utilizes $^{67}$Ga, which binds serum and cellular transferrin and lactoferrin, and is preferentially taken up by neutrophils, macrophages, bacteria, and tumor tissue (e.g., lymphoma). As such, it is primarily used in the identification of occult infection or malignancy. Scanning with $^{111}$In-labeled WBCs has been used to detect osteomyelitis and infectious and inflammatory arthritis. Nevertheless, the use of $^{111}$In-labeled WBC or $^{67}$Ga scanning has largely been replaced by MRI, except when there is a suspicion of prosthetic joint infections.

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### Table 325-5 Diagnostic Imaging Techniques for Musculoskeletal Disorders

<table>
<thead>
<tr>
<th>Method</th>
<th>Imaging Time, h</th>
<th>Cost</th>
<th>Current Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ultrasound$^b$</td>
<td>$&lt; 1$</td>
<td>+</td>
<td>Synovial cysts, Rotator cuff tears, Tendon injury</td>
</tr>
<tr>
<td>Radionuclide scintigraphy</td>
<td>$1-4$</td>
<td>++</td>
<td>Metastatic bone survey</td>
</tr>
</tbody>
</table>

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[71x192] Table 325-5 Diagnostic Imaging Techniques for Musculoskeletal Disorders
Evaluation of Paget’s disease
Acute and chronic osteomyelitis
Acute infection
Prosthetic infection
Acute osteomyelitis

$^{111}$In-WBC

| 24 | +++ |

$^{67}$Ga

| 24–48 | ++++ |

Acute and chronic osteomyelitis
Acute osteomyelitis

Computed tomography

| < 1 | +++ |

Herniated intervertebral disk
Sacroiliitis
Spinal stenosis
Spinal trauma
Osteoid osteoma
Stress fracture

Magnetic resonance imaging

| 1/2–2 | ++++ |

Avascular necrosis
Intraarticular derangement and soft tissue injury
Derangements of axial skeleton and spinal cord
Herniated intervertebral disk
Pigmented villonodular synovitis
Inflammatory and metabolic muscle pathology

$^a$Relative cost for imaging study.

$^b$Results depend on operator.

**Figure 325-7**
CT provides detailed visualization of the axial skeleton. Articulations previously considered difficult to visualize by radiography (e.g., zygapophyseal, sacroiliac, sternoclavicular, hip joints) can be effectively evaluated using CT. CT has been demonstrated to be useful in the diagnosis of low back pain syndromes (e.g., spinal stenosis vs. herniated disc), sacroiliitis, osteoid osteoma, and stress fractures. Helical or spiral CT (with or without contrast angiography) is a novel technique that is rapid, cost effective, and sensitive in diagnosing pulmonary embolism or obscure fractures, often in the setting of initially equivocal findings. High-resolution CT can be advocated in the evaluation of suspected or established infiltrative lung disease (e.g., scleroderma or rheumatoid lung). The recent use of hybrid [positron emission tomography (PET)/CT or single photon emission CT (SPECT/CT)] scans in metastatic evaluations have incorporated CT to provide better anatomic localization of scintigraphic abnormalities.

MRI has significantly advanced the ability to image musculoskeletal structures. MRI has the advantages of providing multiplanar images with fine anatomic detail and contrast resolution (Fig. 325-8) that allows for the superior ability to visualize bone marrow and soft tissue periarticular structures. Although more costly with a longer procedural time than CT, the MRI has become the preferred technique when evaluating complex musculoskeletal disorders.

**Figure 325-8**
MRI can image fascia, vessels, nerve, muscle, cartilage, ligaments, tendons, pannus, synovial effusions, and bone marrow. Visualization of particular structures can be enhanced by altering the pulse sequence to produce either T1-weighted or T2-weighted spin echo, gradient echo, or inversion recovery [including short tau inversion recovery (STIR)] images. Because of its sensitivity to changes in marrow fat, MRI is a sensitive but nonspecific means of detecting osteonecrosis, osteomyelitis, and marrow inflammation indicating overlying synovitis or osteitis (Fig. 325–8). Because of its enhanced soft tissue resolution, MRI is more sensitive than arthrography or CT in the diagnosis of soft tissue injuries (e.g., meniscal and rotator cuff tears); intraarticular derangements; marrow abnormalities (osteonecrosis, myeloma); and spinal cord or nerve root damage or synovitis.

Superior sensitivity of MRI in the diagnosis of osteonecrosis of the femoral head. A 45-year-old woman receiving high-dose glucocorticoids developed right hip pain. Conventional x-rays (top) demonstrated only mild sclerosis of the right femoral head. T1-weighted MRI (bottom) demonstrated low-density signal in the right femoral head, diagnostic of osteonecrosis.

FURTHER READINGS


