

**MINISTRY OF HEALTHCARE OF UKRAINE
HSEEU "Ukrainian Medical Stomatological Academy"**

"Approved"
at the meeting of internal
medicine №1 department
Head of Department
Prof. Skrypnyk I.M.

Protocol № 4 from 13.10.2016

**GUIDELINES
FOR STUDENTS
INDEPENDENT WORK
IN THE PRACTICAL CLASSES PREPARING**

<i>Academic discipline</i>	Internal medicine
<i>Module</i>	Current practice of internal medicine
<i>Content module</i>	Management of the patients with main symptoms and syndromes in rheumatology clinic
<i>Study subject</i>	Management of the patients with joint syndrome
<i>Course</i>	VI
<i>Faculty</i>	of foreign students training

1. Topic actuality: Social significance of this pathology is rather large, because articular syndrome is one of the common diseases. Among the therapeutic profile patients this pathology is present in 30-50% of cases. The disease occurs in any age, but more often in young, predominantly in women 20 to 50 years, also suffering from articular syndrome and men, but more often it is the working person.

2. The aims of the training course:

To Know:

Differential diagnosis of joint's syndrome.

Plan of examination, additional laboratory and instrumental methods of examination, screening tests (X-ray, ultrasound, computed tomography, arthrography, synovial fluid analysis).

Tactic of patient's management.

Medical and surgical management.

Primary and secondary prevention.

Weather and performance.

To be able to:

- conduct surveys and examination of patients with joint's syndrome;
- draft examination of patients with joint's syndrome;
- diagnostic methods used in rheumatology, identify indications and contraindications for their conduction, possible complications;
- carry out differential diagnosis, justify and formulate diagnoses for diseases with joint's syndrome;
- prescribe treatment, determine prognosis, conduct primary and secondary prevention for diseases with joint's syndrome;
- demonstrate knowledge of moral principles.

3. Basic knowledge, skills necessary for study of the topic. (Interdisciplinary integration).

The names of previous courses	These skills
<p>Department of Anatomy</p> <p>Department of Physiology</p>	<p>Demonstrate the knowledge anatomic and physiological features of joints. Demonstrate ability to carry out the survey (collecting complaints), and life history of the disease, physical examination of patients, data describing physical examination. Identify the main syndromes and symptoms; identify changes on the ultrasound, computed tomography, arthrography, to apply the tools etiological, pathogenetic and symptomatic therapy.</p>

4. Tasks for independent work in preparation for classes.

4.1. The list of key terms, parameters, characteristics that students must learn in preparation for classes.

Term	Definition
Rheumatoid factor	Antibodies to the Fc-fragment modified immunological M or G.
Antinuclear factor	Antibodies to nuclei
Cytokines, interleukins.	Terms of immunologic importance
Chalk-stone	Deposition of uric acid crystals
Sacroileitis	Inflammation sacroiliac connections
Felty Syndrome	Option seropositive RA, progressing from high immunological activity, splenomegaly and neutropenia.
Steele syndrome in adults	Option seronegative RA, which is characterized by fever, exanthema, leukocytosis, pharyngitis, lymphadenopathy, splenomegaly.

The contents of topic:

Joint pain can be induced by diverse processes, including inflammation, cartilage degeneration, crystal deposition, infection, and trauma. The initial aim of the evaluation is to localize the source of the joint symptoms and to determine the type of pathophysiologic process responsible for their presence.

The differential diagnoses of joint pain are generated in large part from the history and physical examination. Screening laboratory test results serve primarily to confirm clinical impressions and can be misleading if used indiscriminately.

For patient education resources, see the Hand, Wrist, Elbow, and Shoulder Center, as well as Tennis Elbow.

Pathophysiology

Joint pain may arise from structures within or adjacent to the joint or may be referred from more distant sites. Sources of pain within the joint include the joint capsule, periosteum, ligaments, subchondral bone, and synovium, but not the articular cartilage, which lacks nerve endings. Determination of the anatomic part responsible for joint pain is often a difficult task, but it is critical, in that it guides the approach to diagnosis and therapy. Knowledge of the anatomy of complex joints (eg, the knee, shoulder, and ankle) aids in this assessment.

The evaluation of joint pain, both in terms of the history and the physical examination findings, is best achieved through an understanding of the basic pathophysiologic types of joint disease. These include synovitis, enthesopathy, crystal deposition, infection, and structural or mechanical derangements.

Synovitis

The synovial membrane is the principal site of inflammation in persons with rheumatoid arthritis (RA) and many other inflammatory arthritides. Synovitis is characterized pathologically by the following:

- Neovascularization
- Infiltration of the synovium with lymphocytes, plasma cells, and macrophages
- Synovial lining cell hyperplasia

These cause synovial proliferation, clinically manifested by warmth, tenderness, and a boggy consistency of the soft tissues overlying the involved joint. The inflamed synovium may infiltrate and erode intra-articular bone and cartilage.

Enthesitis

The enthesis is the transitional zone where collagenous structures such as tendons and ligaments are interwoven into bone. Other examples of entheses include the interface between cortical bone and the periosteum and that between vertebral bodies and the annulus fibrosus. The enthesis is the principal site of pathology in the seronegative spondyloarthropathies.

As a result of inflammation at these interfaces, the radially oriented collagen fibers undergo metaplasia, forming fibrous bone. These metaplastic transformations result in new bone formation (periostitis), gradual ossification of syndesmoses (eg, the sacroiliac joints), and syndesmophyte formation along the outer fibers of the vertebral discs. When enthesitis occurs in a diarthrodial joint, a secondary synovitis may develop.

Crystal deposition

The deposition of crystals in articular structures may lead to symptomatic joint disease. The responsible crystals include monosodium urate, calcium pyrophosphate dihydrate, basic calcium phosphate (including hydroxyapatite), and calcium oxalate.

Monosodium urate crystal deposition occurs on the surface of hyaline cartilage, within the synovium, and in periarticular structures, including tendon sheaths and bursae. As a result, inflammation related to urate crystal deposits may be localized to a bursa or tendon sheath adjacent to the joint or may be widespread, involving multiple joint structures. Clinically, an acute gouty joint is inflamed, with overlying erythema, warmth, or both. Prominent periarticular inflammation may resemble cellulitis.

Calcium pyrophosphate crystal deposition is confined to hyaline cartilage, fibrocartilage, and areas of chondroid metaplasia (ie, degenerated areas of tendons, ligaments, and the joint capsule) within the joint.^[5] Shedding of these crystals into the joint space may trigger an acute inflammatory arthritis, known as pseudogout.

Infectious arthritis

The synovium may become the seat of acute or chronic infections related to bacterial, fungal, or viral organisms. These infections almost always arise from blood-borne organisms and may be part of a systemic infection. The infection is based in the synovium.

The cardinal pathologic findings include intense infiltration by neutrophils with resultant necrosis of the synovium and subsequent formation of granulation and scar tissue. A dense mass of fibrin, infiltrated by neutrophils, forms over the surface of the synovium. Bacterial products released within the joint are capable of producing rapid cartilage destruction.

Structural or mechanical joint derangement

Degeneration of the articular cartilage is the principal pathologic feature of osteoarthritis. It occurs in response to both local and host factors. Local factors include the following:

- Previous joint trauma (eg, meniscal tears)
- Congenital or developmental joint alterations (eg, congenital hip dysplasia and slipped capital femoral epiphysis)
- Alterations of the subchondral bone (eg, osteopetrosis, avascular necrosis, and Paget disease)
- Alterations of supporting structures (eg, hypermobility)
- Cartilage derangements (eg, ochronosis and crystal deposition)

Host factors include the following:

- Genetic traits
- Obesity
- Occupation

Damage to the articular cartilage is associated with subchondral bone sclerosis and marginal osteophyte formation. Patients with osteoarthritis may have an associated synovitis, with the formation of bland synovial effusions.

Clinical Presentation

Patient history

A key initial step in the clinical evaluation of a patient with a painful joint is to determine whether the pain stems from the joint or an adjacent bursa, tendon, ligament, bone, or muscle or whether it is referred from a visceral organ or nerve root. This determination is generally more difficult when the pain is in proximal, larger joints. Thus, hip pain can arise from degenerative disc disease or stenosis of the lumbar spine, aortoiliac occlusive disease, hip arthritis, or trochanteric bursitis.

If the pain is stemming from the joint, the following 3 broad categories of joint disease must be differentiated:

- Inflammatory arthritis
- Noninflammatory arthritis
- Arthralgia

Inflammatory arthritis is characterized by inflammation affecting joint structures, such as the synovium, synovial cavity, and entheses. Noninflammatory arthritis is joint disease resulting primarily from alterations in the structure or mechanics of the joint. The joint disease may occur as a result of either (1) cartilage or meniscal damage with or without concomitant alterations in the structure of the subchondral bone or (2) alterations in joint anatomy caused by congenital, developmental, metabolic, or past inflammatory diseases. Arthralgia is characterized by joint tenderness, but abnormalities of the joint cannot be identified. Such patients may have a syndrome of altered pain sensation (eg, fibromyalgia) or an early rheumatic syndrome whose clinical signs are not yet apparent or are too subtle for detection (eg, arthralgia of systemic lupus erythematosus [SLE]).

These types of joint disorders may occur together in the same joint. Inflammatory joint disorders often lead to structural derangement of the joint; similarly, structural joint problems (eg, traumatic arthritis or osteoarthritis) often have an associated, albeit minor, inflammatory component. Finally, reports of joint pain and tenderness in any type of joint disease are influenced by the patient's emotional state and pain threshold. Symptoms of joint disease include the following:

- Pain
- Stiffness
- Swelling
- Limitation of motion
- Weakness
- Fatigue

With inflammatory joint disease, pain is present both at rest and with motion. It is worse at the beginning of usage than at the end. With noninflammatory (ie, degenerative, traumatic, or mechanical) joint disease, the pain occurs mainly or only during motion and improves quickly with rest. Patients with advanced degenerative disease of the hips, spine, or knees may also have pain at rest and at night.

Pain that arises from small peripheral joints tends to be more accurately localized than pain arising from larger proximal joints.

Stiffness is a perceived sensation of tightness when attempts are made to move joints after a period of inactivity. It typically subsides over time. Its duration may serve to distinguish inflammatory forms of joint disease from noninflammatory forms. With inflammatory arthritis, the stiffness is present upon waking and typically lasts 30-60 minutes or longer. With noninflammatory arthritis, stiffness is experienced briefly (eg, for about 15 minutes) upon waking in the morning or after periods of inactivity.

With inflammatory arthritis, joint swelling is related to synovial hypertrophy, synovial effusion, or inflammation of periarticular structures. The degree of swelling often varies over time. With noninflammatory arthritis, the formation of osteophytes leads to bony swelling. Patients may report gnarled fingers or knobby knees. Mild degrees of soft tissue swelling do occur and are related to synovial cysts, thickening, or effusions.

Loss of joint motion may be due to structural damage, inflammation, or contracture of surrounding soft tissues. Patients may report restrictions on their activities of daily living, such as fastening a bra, cutting toenails, climbing stairs, or combing hair.

Muscle strength is often diminished around an arthritic joint as a result of disuse atrophy. Weakness with pain suggests a musculoskeletal cause (eg, arthritis or tendinitis) rather than a pure myopathic or neurogenic

cause. Manifestations include decreased grip strength, difficulty rising from a chair or climbing stairs, and the sensation that a leg is “giving way.”

Fatigue is usually synonymous with exhaustion and depletion of energy in patients with arthritis. With inflammatory polyarthritis, the fatigue is usually noted in the afternoon or early evening. With psychogenic disorders, the fatigue is often noted upon arising in the morning and is related to anxiety, muscle tension, and poor sleep.

Historical features important to the differential diagnosis include the following:

- Onset, duration, and temporal pattern of arthritis
- Number of involved joints
- Symmetry of joint involvement
- Distribution of affected joints
- Distinctive types of musculoskeletal involvement
- Extra-articular manifestations

The onset of symptoms can be abrupt or insidious. With an abrupt onset, joint symptoms develop over minutes to hours. This may occur in the setting of trauma, crystalline synovitis, or infection. With an insidious pattern, joint symptoms develop over weeks to months. This onset is typical of most forms of arthritis, including rheumatoid arthritis (RA) and osteoarthritis.

With respect to duration, symptoms are considered either acute or chronic. Acute symptoms are defined as those that have been present for less than 6 weeks; chronic symptoms are defined as those that have lasted for 6 weeks or longer.

The temporal patterns of joint involvement are (1) migratory, (2) additive or simultaneous, and (3) intermittent. With a migratory pattern, inflammation persists for only a few days in each joint (as in acute rheumatic fever or disseminated gonococcal infection). With an additive or simultaneous pattern, inflammation persists in involved joints as new ones become affected. With an intermittent pattern, episodic involvement occurs, with intervening periods free of joint symptoms (as in gout, pseudogout, or Lyme arthritis).

The involvement of only 1 joint is referred to as monoarthritis. Oligoarthritis is the involvement of 2-4 joints. Polyarthritis is the involvement of 5 or more joints.

Symmetric arthritis is characterized by involvement of the same joints on each side of the body. This symmetry is typical of RA and SLE. Asymmetric arthritis is characterized by involvement of different joints on the 2 sides. This is typical of psoriatic arthritis, reactive arthritis, and Lyme arthritis.

With regard to distribution, the distal interphalangeal joints of the fingers are usually involved in psoriatic arthritis, gout, or osteoarthritis but are usually spared in RA. Joints of the lumbar spine are typically involved in ankylosing spondylitis but are spared in RA.

Different diseases exhibit distinctive types of musculoskeletal involvement. Spondyloarthropathy involves entheses, leading to heel pain (inflammation at the insertions of the Achilles tendon or plantar fascia), dactylitis (sausage digits), tendinitis, and back pain (sacroiliitis and vertebral disc insertions). Gout commonly involves tendon sheaths and bursae, resulting in superficial inflammation.

Extra-articular manifestations also vary. Constitutional symptoms suggest an underlying systemic disorder and are not expected in patients with degenerative joint disease. These may include fatigue, malaise, and weight loss. Skin lesions may be present. Physical examination of the skin, but not the joints, may indicate the specific diagnosis of a number of rheumatic diseases. Examples include SLE, dermatomyositis, scleroderma, Lyme disease, psoriasis, Henoch-Schönlein purpura, and erythema nodosum.

Ocular symptoms or signs are also possible. Episcleritis and scleritis may be associated with RA or granulomatosis with polyangiitis (Wegener granulomatosis), anterior uveitis with ankylosing spondylitis, and iridocyclitis with juvenile idiopathic arthritis. Conjunctivitis may be caused by reactive arthritis.

Physical examination

The musculoskeletal examination helps distinguish joint inflammation (eg, RA) from joint damage (eg, degenerative joint disease). It can also help elucidate the site of musculoskeletal involvement (eg, synovitis, enthesitis, tenosynovitis, or bursitis) and the distribution of joint involvement.

Signs distinguishing joint inflammation from joint damage

Signs of inflammatory joint disease include the following:

- Synovial hypertrophy
- Joint effusions
- Pain with motion, particularly at the extremes of joint motion
- Erythema and warmth
- Limited range of motion
- Joint tenderness

Synovial hypertrophy is the most reliable sign of an inflammatory arthritis. The synovial membrane is normally too thin to palpate. In a person with chronic inflammatory arthritis, the synovial membrane has a doughy or boggy consistency, a feature best appreciated at the joint line or margin.

Joint effusions develop in response to synovial inflammation, trauma, anasarca, intra-articular hemorrhage (hemarthrosis), or an adjacent focus of acute inflammation (sympathetic effusion). These are detected by performing fluid ballottement or cross-fluctuation through the synovial cavity.

Pain throughout the whole range of motion is observed in a person with an acutely inflamed joint. Pain experienced as the joint is gently forced (ie, stressed) towards its limitation of range is suggestive of synovitis. Pain not present throughout the entire range of motion may indicate an extra-articular source, such as tendinitis.

Erythema of the joint is restricted to acute inflammatory forms of arthritis, such as gout, septic arthritis, or acute rheumatic fever. It is rare in persons with RA but may occasionally occur in those with psoriatic arthritis. Warmth of the joint is a sensitive sign of inflammatory arthritis and can be detected by passing the hand back and forth from the joint to a neutral area distal or proximal to the joint. Differences in warmth can also be detected by comparing the same joint on each side of the body.

In a person with inflammatory joint disease, limitation of motion results from the presence of a tense effusion, a markedly thickened synovium, adhesions, capsular fibrosis, or pain.

Joint tenderness is a sensitive sign of joint disease, but it is not specific for inflammatory arthritides. In an acutely inflamed joint, tenderness can be elicited over the entire synovial reflection. Focal tenderness may indicate a focus of inflammation outside the joint (eg, tendinitis, osteomyelitis, or fracture). The presence of joint tenderness in the absence of other joint abnormalities must be interpreted in the context of the patient's emotional state.

Signs of degenerative or mechanical joint disease include the following:

- Bony overgrowth of the joints (osteophytes)
- Limited range of motion
- Crepitus during active or passive range of motion
- Joint deformity

Osteophytes located at the distal interphalangeal joints are called Heberden nodes, whereas those located at the proximal interphalangeal joints are called Bouchard nodes.

In persons with degenerative or traumatic joint disease, the limitation of motion results from intra-articular loose bodies, osteophyte formation, or subluxation.

A palpable or audible grating sensation is typically produced during motion of the joint. Soft, fine crepitus may be felt (or heard with a stethoscope) in a rheumatoid joint when the cartilage surface is no longer smooth. Coarse crepitus or grating may be felt in joints severely damaged by long-standing RA or degenerative arthritis.

Three main types of joint deformity must be distinguished. The first type is restriction of the normal range of motion (eg, a lack of full joint extension that results in a flexion deformity). The second is malalignment of the articulating bones (eg, ulnar deviation of the fingers or valgus deformity of the knee). The third is an alteration in the relation of the 2 articulating surfaces, such as subluxation (ie, some contact between the articulating surfaces) or dislocation (ie, complete loss of contact between the articulating surfaces).

General techniques of musculoskeletal examination

Techniques used in the musculoskeletal examination include the following:

- Inspection
- Palpation
- Assessment of range of motion

On inspection, each joint has a characteristic or normal appearance, and each assumes a characteristic resting position.

Compare one side of the body with the other in order to detect joint abnormalities, including swelling, deformity, overlying erythema, or wasting of the periarticular musculature. With a sagittal view of the patient, take note of joint deformities that result from the lack of full extension of a joint (eg, flexion deformities). With a coronal view of the patient, take note of joint malalignment, which may result in valgus or varus deformities.

Palpation of the joints is used to assess for signs of inflammation (eg, warmth, synovial hypertrophy, joint effusion, and tenderness) and signs of joint damage (eg, bony swelling and crepitus). The examiner should palpate with enough pressure to blanch his or her thumbnail. This ensures that the assessment of joint tenderness is uniform. Application of this amount of force during palpation should not cause pain in a normal joint.

Assess limitation of passive motion by comparing it with the expected range of motion observed in healthy individuals and with the range of motion in the contralateral joint. Assessment of active range of motion can be used to determine the presence of pathology in juxta-articular structures (eg, tendons and bursae). Pain occurring during only a portion of the range of motion may be related to an extra-articular structure. Assess pain with joint motion; observe the patient's face for wincing.

Assess crepitus by palpating the joint with one hand while moving the joint passively with the other. In the lower extremities, crepitus of the hip or knee can sometimes be heard as the patient arises from a chair, climbs a step, or pivots on the affected joint.

Assess instability or abnormal mobility by applying forces to the relaxed joint in planes of motion normally associated with little or no motion. Instability of a lower-extremity joint (eg, a knee or ankle) should also be assessed by observing the joint during weight-bearing and walking. Instability of the joint may be due to laxity of ligaments or to destruction of the articular surface.

Differential Diagnosis

Processes associated with different arthritis types

Arthritis may be acute or chronic, may involve a single joint (monoarthritis) or multiple joints (polyarthritis), and may be related to either inflammatory or noninflammatory processes.

Inflammatory processes associated with acute monoarthritis include the following :

- Septic Arthritis
- Gout and Pseudogout
- Systemic rheumatic disease manifesting as monoarticular involvement

Noninflammatory processes associated with acute monoarthritis include the following:

- Juxta-articular fracture
- Trauma
- Hemarthrosis
- Osteonecrosis

Inflammatory processes associated with chronic monoarthritis include the following:

- Chronic infectious arthritis
- Lyme Disease
- Crystalline synovitis
- Pauciarticular juvenile idiopathic arthritis (Rheumatoid Arthritis)
- Systemic rheumatic disease presenting with monoarticular involvement

Noninflammatory processes associated with chronic monoarthritis include the following:

- Osteoarthritis
- Ischemic necrosis
- Hemarthrosis
- Paget disease involving the joint
- Stress Fracture
- Osteomyelitis
- Osteosarcoma
- Metastatic tumor
- Synovial osteochondromatosis

Processes associated with acute polyarthritis include the following:

- Rheumatic fever
- Gonococcal Arthritis
- Polyarticular gout
- Polyarticular pseudogout
- Viral arthritis
- Bacterial endocarditis
- Rheumatoid Arthritis
- Still disease (systemic-onset juvenile idiopathic arthritis)
- Systemic Lupus Erythematosus
- Reactive Arthritis
- Acute sarcoid arthritis
- Mediterranean Fever, Familial
- Enteropathic Arthropathies

Inflammatory processes associated with chronic polyarthritis include the following:

- Rheumatoid Arthritis
- Systemic Lupus Erythematosus
- Viral arthritis
- Psoriatic Arthritis
- Reactive Arthritis
- Enteropathic Arthropathies
- Behçet Disease
- Ankylosing Spondylitis and Undifferentiated Spondyloarthropathy

Noninflammatory processes associated with chronic polyarthritis include the following:

- Osteoarthritis
- Traumatic osteoarthritis
- Hemochromatosis
- Ochronosis
- Hypertrophic pulmonary osteoarthropathy
- Amyloidosis
- Acromegaly

Differential diagnoses for regional musculoskeletal pain

Shoulder

Referred pain may derive from cervical disorders, Pancoast tumor of the lung, subphrenic pathology, or entrapment neuropathies and brachial neuritis. Rotator cuff tendinitis is inflammation of the rotator cuff

tendons, arising acutely as a result of a recognizable injury (throwing) or insidiously as a result of repeated impingement on the overlying acromion, coracoacromial ligament, acromioclavicular joint, or coracoid. The principal symptom of rotator cuff tendinitis is pain in the deltoid region of the shoulder, aggravated by an overhead motion of the arm. The patient may also describe shoulder pain when sleeping on the affected side. Examination findings include the following:

- Tenderness in the subacromial region, between the greater tubercle of the humerus and acromial process
- Pain in the middle of the arc of active abduction, usually between 60° and 120°
- Reproduction of pain when midarc abduction and external rotation are resisted isometrically
- Range of passive shoulder abduction exceeding that of active abduction

Treatment includes avoidance of overhead reaching, administration of nonsteroidal anti-inflammatory drugs (NSAIDs) for 2-3 weeks, and physical therapy with stretching and strengthening exercises. Subacromial corticosteroid injections may be used if symptoms do not improve.

Rotator cuff tears (see Rotator Cuff Injuries) are transverse or longitudinal tears of the supraspinatus or infraspinatus tendons. They occur at the musculotendinous juncture, approximately 1 cm from their insertion on the humerus. They may arise as a result of an acute injury (eg, a fall on an outstretched arm, hyperabduction, or a fall onto the side of the shoulder) or gradual attrition in the setting of chronic rotator cuff tendonitis.

With acute injury, symptoms include sharp shoulder pain followed by weakness of abduction. In the setting of chronic rotator cuff tendinitis, a tear is signaled by weakness of abduction or loss of smooth motion during abduction. Examination findings include the following:

- Weakness and pain in the midarc of abduction and external rotation
- Loss of smooth overhead reaching (partial tears) or inability to reach overhead (complete tears)

Initial management is conservative. Young patients with acute tears should be evaluated by an orthopedic surgeon.

Bicipital tendinitis is inflammation of the long head of the biceps as it passes through the bicipital groove of the anterior humerus. It usually arises as a result of overuse with activities that require repetitive lifting. The primary symptom is pain in the anterior aspect of the shoulder (over the humeral head), which is aggravated by lifting or overhead pushing or pulling. Examination findings include the following:

- Tenderness of the bicipital groove
- Pain aggravated by isometric resistance to elbow flexion or supination of the arm flexed to 90°

Treatment includes elimination of lifting, avoidance of over-the-shoulder reaching, and 3-4 weeks of NSAID therapy. Corticosteroids may be injected into the bicipital groove if symptoms persist.

Subacromial bursitis is the accumulation of fluid within the subacromial bursa, arising as a result of rotator cuff tendonitis. Significant fluid may be detected during a physical examination. Treatment is similar to that of rotator cuff tendinitis. For a significant effusion, drainage is indicated, followed by corticosteroid instillation.

Frozen shoulder (adhesive capsulitis) is a term for conditions in which the range of motion of the glenohumeral joint is significantly reduced as a result of pathology within the joint capsule. Associated medical conditions include diabetes mellitus, recent myocardial infarction, stroke, a recent neurosurgical procedure, Parkinson disease, and hypothyroidism.

The primary symptoms of frozen shoulder are pain and gradual loss of shoulder motion without any known injury. Examination findings include a reduced range of motion during both active and passive motion. Pain is present particularly at the extreme ranges of motion. Radiographic images do not show evidence of glenohumeral arthritis.

The initial treatment regimen includes NSAIDs, nonnarcotic analgesics, and physical therapy. Occasionally, a 2- to 4-week course of oral corticosteroids combined with aggressive physical therapy may result in decreased pain and increased shoulder motion.

In acromioclavicular syndrome (see Acromioclavicular Joint Injury), pain arises from the acromioclavicular joint as a result of arthritis or injury to the acromioclavicular ligaments. Osteoarthritis of the acromioclavicular joint with inferior osteophytes can lead to rotator cuff impingement and associated tendinitis. This injury may be acute or chronic, and patients may report a history of trauma (eg, fall during a contact sport).

Examination findings include the following:

- Tenderness
- Swelling of the acromioclavicular joint

Deformity of the joint may result from subluxation. Pain in the joint is aggravated by downward traction of the ipsilateral arm or forced passive adduction. An acute acromioclavicular injury is treated with a shoulder immobilizer.

Elbow, wrist, and hand

Lateral epicondylitis (tennis elbow) is the most common cause of elbow pain. Pain is felt along the lateral aspect of the elbow. Tenderness is present over the lateral epicondyle at the attachment of the extensor tendons of the forearm. Resisting wrist dorsiflexion with the elbow in extension produces increased pain. Elbow extension is normal. Treatment includes rest, NSAIDs, and local steroid injections.

Medial epicondylitis (golfer elbow) is less common than lateral epicondylitis. Resisted wrist flexion with the elbow in extension produces pain. Tenderness may occur at the insertion of the common flexor tendon at the medial epicondyle.

In olecranon bursitis, the anatomically superficial position of the bursa predisposes it to injury and inflammation. The patient reports pain when leaning on the elbow and during flexion. Examination findings include tenderness at the tip of the olecranon process and an occasional friction rub. Visible swelling of the bursa may be evident. In acute cases, warmth and erythema are present. Patients with acute bursitis must undergo aspiration for culture and crystal examination.

De Quervain tenosynovitis is a stenosing tenosynovitis of the abductor pollicis longus and extensor pollicis brevis tendons, resulting from repetitive motion or overuse. Pain is noted along the radial aspect of the wrist and thumb during pinching, grasping, and similar movements. Ulnar deviation of the wrist, with the thumb held in abduction by the flexed fingers of the same hand (Finkelstein test), reproduces the pain. Crepitus of the tendons may be evident.

Treatment of de Quervain tenosynovitis includes use of a thumb spica splint, avoidance of repetitive thumb flexion or abduction, and NSAIDs.

Trigger finger and trigger thumb (see Trigger Finger) are also known as stenosing digital tenosynovitis, snapping finger, and snapping thumb. Injury is the result of overuse. Examination findings include the following:

- Pain and tenderness
- Snapping, triggering, or catching during movement of the finger or thumb

A nodule is felt in the palm on the flexor tendon just proximal to the digital-palmar crease.

Hip

Pain in the posterior aspect of the hip is often referred from the lumbar spine. Sacroiliac disorders can also cause buttock pain. Pain from arthritis of the thoracolumbar junction may be referred pain to the area of the greater trochanters and may mimic trochanteric bursitis. Radiculopathies of the L2-L4 nerve roots may produce pain in the inguinal area and the anterior thigh; this may mimic hip disease. Iliopsoas abscesses, retroperitoneal appendicitis, tuberculous abscesses, or pelvic inflammatory disease can cause pain in the hip region.

Thrombosis or aneurysm formation in the branches of the aorta or iliac vessels may produce buttock, thigh, or leg pain that may be confused with hip pain. True intra-articular hip pain is most often felt in the groin and anterior thigh. Occasionally, hip disease can manifest with isolated knee pain.

Trochanteric bursitis is the most common cause of pain in the hip region (felt over the lateral aspect of the hip). Pain increases with activities such as walking, squatting, and climbing stairs; pain typically decreases at rest. Patients note increased pain when lying on their ipsilateral side. The pain may be associated with a limp. The area over the greater trochanter may be tender and boggy. Resisted abduction of the hip reproduces the pain. Local corticosteroids with anesthetics may help.

Iliopsoas bursitis can occur in patients with osteoarthritis, RA, pigmented villonodular synovitis, osteonecrosis, and septic arthritis. Most patients are asymptomatic or present with a painful inguinal mass. Computed tomography (CT) is the best diagnostic test. Instillation of corticosteroids is effective therapy. Ischiogluteal bursitis occurs most commonly in patients with occupations that favor repeated friction of the ischial bursa. Patients note pain over the ischial tuberosities; the pain is aggravated by sitting and lying down. Local tenderness of the ischial tuberosities is found upon palpation. Symptoms may be alleviated through avoidance of pressure or friction on the ischial tuberosities (ie, by using doughnut-shaped cushions) and local instillation of corticosteroids.

Adductor tendinitis occurs in patients engaged in sports activities that involve straddling (eg, horseback riding, gymnastics, or dancing). Pain is typically felt in the groin and the inner aspect of the thigh.

Tenderness can be elicited by local palpation of the adductor muscles, especially near their insertion on the front of the pelvis. Pain is increased by passive abduction of the thighs and active adduction against resistance.

Treatment of adductor tendinitis consists of rest and ice packs during the acute phase. NSAIDs, ultrasonography, and progressive stretching exercises are used in the subacute phase. Local corticosteroid injections are reserved for patients resistant to these conservative modalities.

Knee and ankle

Prepatellar bursitis (housemaid knee) is related to recurrent trauma and usually occurs in persons who spend significant time kneeling. Etiologies include trauma, gout, and infection. In chronic cases, a well-circumscribed area of fluctuance is present over the prepatellar area. In acute cases, warmth, edema, and erythema are noted over the anterior knee. Fluctuance may be subtler. Tenderness is maximal over the prepatellar bursa. Knee flexion increases the pain, whereas knee extension does not. A joint effusion, if present, is small.

Aspiration of acute bursitis is necessary to assess for the presence of an infection or crystals. Traumatic bursitis improves with rest and avoidance of kneeling.

In anserine bursitis, pain is noted over the medial aspect of the knee, is made worse by climbing stairs, and is often present at night. It is most common in overweight women with osteoarthritis of the knees. Patellar tendinitis (jumper's knee) most commonly affects young athletes who are engaged in sports that require repetitive running, kicking, and jumping. Pain is noted at the inferior pole of the patella during activities such as climbing stairs, running, and jumping. Treatment consists of rest, NSAIDs, knee bracing, and an exercise regimen to stretch and strengthen the quadriceps and hamstring muscles.

Achilles tendinitis is characterized by pain, swelling, tenderness, and crepitus over the tendon near its insertion. This form of tendinitis is usually caused by repetitive trauma and microscopic tears from excessive use of the calf muscles in ballet dancing, distance running, basketball, jumping, and other athletic activities. Faulty footwear with a rigid shoe counter also may produce Achilles tendonitis.

Examination findings include thickening and irregularity of the tissues surrounding the tendon and palpable nodule or nodules within the tendon (occasionally representing xanthomata, tophi, or rheumatoid nodules). Passive dorsiflexion of the ankle intensifies the pain. Abnormalities of the tendon and peritendinous tissues can be demonstrated on images from ultrasonography and magnetic resonance imaging (MRI).

Treatment of Achilles tendinitis consists of rest, avoidance of the provocative occupational or athletic activity, shoe modification, a heel lift to reduce tendon stretching during walking, and NSAID therapy. Physical therapy includes local heat application, gentle stretching exercises, and a temporary splint with slight plantar flexion.

Retrocalcaneal bursitis is inflammation of the retrocalcaneal bursa, resulting in pain and tenderness at the back of the heel. The area anterior to the Achilles tendon and posterior to the calcaneus is tender; passive dorsiflexion of the ankle produces pain. Bursal distention is palpable and produces bulging on both sides of the tendon.

Retrocalcaneal bursitis may occur as a result of repetitive trauma or as a manifestation of gout or a systemic inflammatory arthritis. The diagnosis can be confirmed by means of radiography (showing obliteration of the retrocalcaneal recess), ultrasonography, or MRI.

For most patients with retrocalcaneal bursitis, rest, activity modification, moist heat application, slight heel elevation using a felt heel pad, and NSAIDs constitute sufficient therapy. A walking cast or cautious corticosteroid injection into the bursa is sometimes required.

Laboratory Studies

The most useful diagnostic tests for specific rheumatic diseases are as follows :

- Septic arthritis – Gram stain and culture of synovial fluid
- Gout or pseudogout – Compensated polarized light microscopy to examine a drop of synovial fluid for intracellular urate crystals (gout) or calcium pyrophosphate dihydrate crystals (pseudogout)
- Ankylosing spondylitis – Sacroiliac joint radiography to demonstrate bilateral sacroiliitis
- Osteoarthritis – Radiography of the affected joint
- Systemic lupus erythematosus (SLE) – Antinuclear antibody (ANA) test; if results are positive, test for Smith (Sm) and double-stranded DNA antibodies, which are more specific for SLE but are present in only 30% and 60% of SLE patients, respectively

Screening tests for all types of inflammatory arthritis include the following :

- Erythrocyte sedimentation rate (ESR)
- C-reactive protein (CRP)
- Rheumatoid factor (RF) and cyclic citrullinated peptide (CCP)
- ANAs

In the setting of joint pain and equivocal joint examination findings, an elevated ESR supports the presence of an inflammatory arthritis.

The CRP level is a nonspecific measure of inflammation and is obtained as an alternative to obtaining the ESR. In contrast to the ESR, the CRP level (1) can be measured on frozen serum, (2) is not influenced by the presence of anemia or hyperglobulinemia, (3) rises more rapidly in response to an inflammatory stimulus, and (4) may require more time for the laboratory result to be available (ie, more than 24 hours, as opposed to 1 hour for the ESR).

An RF test should be obtained when rheumatoid arthritis (RA) is considered at least moderately possible. Results may be positive in as many as 20% of healthy elderly persons and in persons with other rheumatic diseases (eg, SLE, Sjögren syndrome, and vasculitis), chronic infections (eg, subacute bacterial endocarditis and hepatitis C), chronic liver disease, or chronic lung disease.

CCP antibody testing has higher specificity than the RF test but lower sensitivity. The CCP antibody test is particularly useful in the evaluation of patients with joint pain in whom RF titers are low and findings on joint examination are not definitive for synovitis.

ANA tests are commonly obtained in patients with arthralgias or arthritis as a screening test for SLE or another connective-tissue disorder. More than 95% of patients with SLE have ANAs; thus, a negative ANA result is a strong indicator that SLE is not present. However, a positive ANA result lacks specificity and may occur in persons with other connective-tissue diseases or certain medical illnesses, as well as in 5-10% of otherwise healthy individuals.

The diagnostic yield of the ANA test is increased substantially when the patient has features that suggest a diagnosis of SLE or another autoimmune disease in addition to joint pain. These include a photosensitive skin rash, pleuritis, pericarditis, Raynaud phenomenon, constitutional symptoms (eg, fever), leukopenia, thrombocytopenia, sicca symptoms, and proteinuria.

Screening tests for acute polyarthritis include the following:

- Blood cultures
- Antistreptolysin O titer
- Parvovirus B-19 immunoglobulin G (IgG) and immunoglobulin M (IgM) levels
- Hepatitis B serology
- ANAs
- Others – Additional tests that may be considered are an HIV test, a rubella titer, an angiotensin-converting enzyme (ACE) level, chest radiography, and an antineutrophil cytoplasmic antibody (ANCA) test

Screening tests for chronic polyarthritis include the following:

- Complete blood count (CBC)
- ESR and CRP level
- ANAs
- RF and CCP antibody
- Chemistry profile, including liver function tests (LFTs) and a serum creatinine level
- Serum uric acid level
- Urinalysis
- Others – Additional tests that may be considered are a thyroid-stimulating hormone (TSH) level, a serum ferritin level, and iron saturation of serum transferrin

Screening tests for diffuse arthralgias and myalgias include the following:

- ESR and CRP level to exclude inflammatory disease (eg, polymyalgia rheumatica)
- Creatine kinase and aldolase level to exclude myositis
- Thyroid testing
- Chemistry profile (ie, calcium, phosphorus, electrolyte, glucose, and total protein) to exclude metabolic or endocrine disorders
- Others – Additional tests that may be considered are a 25-hydroxy vitamin D level (in elderly housebound individuals, to exclude osteomalacia), sacroiliac joint radiography (to exclude ankylosing spondylitis, especially in woman younger than 45 years with neck, chest wall, and low back pain), HLA-B27 (to support a diagnosis of reactive arthritis), hepatitis B and C serology testing, serum and urine protein electrophoresis (to exclude multiple myeloma), and ANA and RF (if clinical features suggest RA, SLE, or another connective-tissue disease)

Plain Radiography

Plain radiography is the least expensive imaging modality and is most useful for clarifying the nature of joint abnormalities already noted during the physical examination, such as swelling (bony vs soft tissue), loss of motion (bony vs soft tissue), instability (ligamentous damage vs destruction of articular surface), and focal bony tenderness (fracture vs osteomyelitis).

The appearance of joints on plain radiographs is often distinctive for various forms of arthritis (see below), though these characteristic changes may not be apparent early in the disease course. Plain radiographs are also useful for monitoring the progression of chronic arthritides (eg, osteoarthritis and rheumatoid arthritis [RA]).

Rheumatoid arthritis

Early radiographic changes in RA include soft tissue swelling and periarticular demineralization. Later changes include uniform loss of joint space (indicative of diffuse cartilage loss) and bony erosions (initially along joint margins where intra-articular bone is not covered by cartilage). Advanced changes include diffuse bony erosions, joint subluxation, and foreshortening of digits. Ankylosis of joints is rare.

Psoriatic arthritis

Early radiographic changes in psoriatic arthritis include soft tissue swelling, occasionally involving the entire digit (ie, sausage digit), and an absence of periarticular demineralization. Later changes include erosions coupled with reactive new bone formation, initially at joint margins and later within the center of the joint. Other late changes are uniform joint space narrowing and ankylosis of involved joints. Advanced changes are joint-space widening in interphalangeal (IP) joints caused by severe destruction of marginal and subchondral bone, resorption of tufts of distal phalanges of fingers and toes, arthritis mutilans (ie, severe joint destruction with extensive bone resorption), and the pencil-in-cup deformity. Distinctive features are involvement of the distal IP joints, a tendency for early ankylosis, asymmetric joint involvement, and abnormalities of phalangeal tufts.

Reactive arthritis

The radiographic features of reactive arthritis are similar to psoriatic arthritis, but they are often less severe and have a predilection for lower-extremity joints. Distinctive features include a predilection for the lower extremities, a tendency for unilateral or asymmetric sacroiliitis, paravertebral ossification, and calcaneal erosions or periostitis at sites of Achilles tendon and plantar fascia insertion.

Gout

On plain radiography, acute gouty arthritis is indicated by soft tissue swelling. Degenerative changes of the involved joint are common. Intercritical gout does not manifest radiographic abnormalities, apart from possible degenerative changes in the joint.

Chronic tophaceous gout is indicated by soft tissue swelling, often asymmetric or outlining an eccentric nodular subcutaneous mass. The joint space may be preserved despite extensive erosions, a finding not expected in RA. Bone erosions are contiguous with tophi and are characterized by overhanging and sclerotic margins. Osteolytic bone lesions occur near joints. Periarticular demineralization is absent or mild, except late in the disease course.

Calcium pyrophosphate dihydrate crystal deposition disease

Radiographic evidence of calcium crystal deposition in articular structures is seen most often in the knee, symphysis pubis, wrist, elbow, and hip. The prevalence of calcium crystal deposition increases with age, and it is often an incidental finding that tends not to be associated with joint symptoms.

Hyaline cartilage calcification is fine and linear, and it follows the contour of the underlying subchondral bone. Fibrocartilage calcification is coarse and irregular, and it is often seen in knee menisci, triangular fibrocartilage and the meniscus of the wrist, and the symphysis pubis. Synovial calcification is amorphous and usually occurs at sites of synovial reflection. Capsular calcification consists of linear deposits bridging the peripheral joint margins. Extra-articular calcification occurs in tendons, ligaments, and para-articular soft tissues.

Pyrophosphate arthropathy is a distinctive arthropathy that may occur in patients with calcium pyrophosphate dihydrate crystal deposition disease. Radiographic findings are the same as those for osteoarthritis. Distinctive features include the following:

- Involvement of joints not usually affected by osteoarthritis (eg, metacarpophalangeal (MCP) joint, wrist, elbow, ankle, and shoulder)
- Involvement of specific joint compartments (eg, the radiocarpal and trapezioscapoid joints of the wrists, the patellofemoral joint of the knee, and the talocalcaneonavicular joint of the midfoot)
- Prominent subchondral cysts
- Occasional articular destruction (resembling a neuropathic joint) with subchondral bone collapse and fragmentation and formation of intra-articular loose bodies

Infectious arthritis

Early radiographic changes of infectious arthritis include symmetric soft tissue swelling, an absence of periarticular demineralization in an acute pyogenic arthritis, and joint-space loss (although joint-space widening may be seen initially because of fluid accumulation in a small joint space).

Later changes include marginal bone erosions. In addition, the continuous white cortical line that normally defines the margin of articulating bone is lost; these changes are expected on both sides of the joint. A periosteal reaction occurs. Finally, gas formation within the joint and adjacent soft tissues can be seen with infections related to *Escherichia coli*, *Enterobacter liquefaciens*, and *Clostridium perfringens*. Advanced changes include destruction of subchondral bone, intra-articular bony ankylosis, and subluxation or dislocation.

Osteoarthritis

Early radiographic changes in osteoarthritis include small osteophytes at joint margins, focal narrowing of joint spaces (more uniform joint-space loss is noted in the IP and MCP joints of the hands and sacroiliac joints), subchondral bony sclerosis in the segment affected by joint-space loss, and an absence of periarticular demineralization.

Later changes include large and more extensive osteophytes at joint margins or at ligamentous attachments (eg, tibial spines), more pronounced focal joint-space narrowing, subchondral bone cysts with sclerotic margins in the areas of joints affected by joint-space loss, and the formation of bony ossicles (round or oval fragments of bone) in soft tissues adjacent to the joint or within the joint cavity. Advanced changes include extensive joint-space loss and joint deformity.

Other Imaging Studies

Ultrasonography

Musculoskeletal ultrasonography uses ultrasonic waves to image soft tissues, including tendons, bursae, ligaments, and components of the joint. It is performed by a specifically trained rheumatologist or radiologist and involves an examination with multiple views and positionings of the joint. It is safe and does not involve any exposure to radiation. Joint aspirations and injections are greatly facilitated if performed with ultrasound guidance, because this ensures correct positioning of the needle.

Computed tomography

Computed tomography (CT) obtains cross-sectional images of skeletal structures. In a patient with a painful joint, CT is most useful for the following applications:

- Assessing trauma of the spine and pelvis
- Evaluating arthritis in axial joints (eg, sacroiliac, atlantoaxial, and sternoclavicular)
- Evaluating pain in complex joints in which overlying structures obscure plain radiography views (eg, ankle, wrist, and temporomandibular joints)
- Evaluating degenerative disc disease of the spine and possible disc herniations

Magnetic resonance imaging

Magnetic resonance imaging (MRI) is the best modality for assessing soft tissue and spinal cord elements.^[26] It is of greatest use for assessing rotator cuff tears, spinal stenosis, ligamentous or meniscal abnormalities of the knee and wrist joints, osteonecrosis (ie, avascular necrosis of bone), stress fractures, osteomyelitis, and subchondral bone injury in osteoarthritis or meniscal tears.

Arthrography

Arthrography is most useful for defining abnormal communication between the synovial space and adjacent bursae and soft tissue (ie, popliteal cysts or rupture of the rotator cuff with communication between the glenohumeral joint space and the subacromial bursa).

Radionuclide bone scanning

Radionuclide bone scanning is widely available, and its cost is comparable to that of CT scanning. It is most useful for assessing osteomyelitis, stress fractures, and bony metastasis. It may be used to exclude skeletal disease in patients with diffuse musculoskeletal pain.

Synovial Fluid Analysis

Synovial fluid analysis is used to broadly characterize the type of arthritis, to identify crystals, and to establish the diagnosis of septic arthritis and crystal-induced synovitis.

Synovial fluid types are classified into 5 categories as follows:

- Normal – Characteristics include clear to pale yellow color, transparent clarity, white blood cell (WBC) count lower than 200/ μ L with less than 25% polymorphonuclear (PMN) leukocytes, and very high viscosity
- Noninflammatory (group I) – Characteristics include pale yellow color, transparent clarity, WBC count of 200-2000/ μ L with less than 25% PMN leukocytes, and high viscosity; this category typifies osteoarthritis, traumatic arthritis, and an early or resolving stage of an inflammatory arthritis
- Inflammatory (group II) – Characteristics include yellow-to-white color, translucent-to-opaque clarity, WBC count of 2000-50,000/ μ L with more than 70% PMN leukocytes, and low viscosity; this category typifies rheumatoid arthritis (RA) and other chronic inflammatory arthritides

- Septic (group III) – Characteristics include a white-to-cream color, opaque clarity, WBC count higher than 50,000/μL with more than 90% PMN leukocytes, and very low viscosity; this category typifies bacterial arthritis, but the fluid type also may occasionally be seen in crystalline arthritis and flares of RA
- Hemorrhagic (group IV) – Characteristics include a hemorrhagic color and opaque clarity; fat globules should be sought in hemorrhagic fluids by centrifuging the synovial fluid (a supernatant of fat is indicative of a juxta-articular fracture)

A joint may be affected by more than a single process; thus, septic arthritis and gout or pseudogout may coexist in the same joint. The synovial fluid WBC count may be lower in patients who are early in the course of septic arthritis or in patients with disseminated gonococcal infection.

Crystal analysis requires compensated polarized light microscopy, which is available in most diagnostic or pathologic laboratories.^[28] It is performed on a wet smear preparation of synovial fluid. Intracellular crystals in synovial fluid are required to establish a diagnosis of acute gout or pseudogout.

Urate crystals are needle-shaped with strong negative birefringence. Calcium pyrophosphate dihydrate crystals are rhomboid-shaped with weak positive birefringence. Urate crystals appear yellow and calcium pyrophosphate dihydrate crystals blue when their long axes are aligned parallel to that of the red compensator filter.

General principles of Treatment & Management

Most patients who present with a new musculoskeletal complaint have a problem restricted to one anatomic region or joint (eg, back or shoulder pain). These problems are usually recognized as self-limited and as not posing a major health hazard. Patients are treated symptomatically and advised about the optimal balance of activity and rest, the benign nature of the problem, and the expectation of healing in 2-6 weeks. Extensive testing is inappropriate; it may reveal irrelevant abnormalities that may prompt further, often unnecessary, investigations.

Clinical situations in which acute musculoskeletal symptoms must be evaluated promptly and thoroughly include the following:

- A severe condition involving one joint or, at most, a few joints
- A patient who is febrile, is systemically ill, or is showing signs of multiple organ involvement
- A problem associated with significant trauma
- A condition in which an associated neurologic problem exists, such as carpal tunnel syndrome, sciatica, or cervical radicular symptoms

In patients with arthritis, the goals of treatment include relief of pain, restoration or maintenance of joint function, and prevention of joint damage. These goals are achieved with both pharmacologic and nonpharmacologic therapeutic modalities.

Whereas some modalities are common to the treatment of all forms of arthritis, others are specific to certain forms of arthritis. Thus, proper treatment begins with an accurate diagnosis. One of the challenges in treating arthritis is determining the expected prognosis and instituting appropriate therapy in a timely fashion, thereby avoiding the development of irreversible joint dysfunction. The initial patient assessment should allow classification of the joint problem into one of the categories detailed below.

Medical management of acute monoarthritis

Hospitalize any patient with possible septic arthritis. Aspiration of joint fluid is a critical step in diagnosis.

The finding of noninflammatory joint fluid in an acutely inflamed joint should prompt consideration of juxta-articular osseous pathology (eg, stress fracture, osteomyelitis, or avascular necrosis), acute inflammation of periarticular structures (eg, gouty inflammation of tendon sheaths or bursae or septic bursitis), subcutaneous inflammation (eg, arthritis of ankles in erythema nodosum or pancreatic fat necrosis), or cellulitis.

If the possibility of septic arthritis cannot be excluded with reasonable certainty after the initial clinical and laboratory evaluation, begin intravenous (IV) antibiotic therapy. Provide for adequate drainage of the joint. This can be achieved via repeated percutaneous aspiration of the joint with a large-bore needle or via arthroscopic drainage if a large joint (eg, hip, knee, shoulder, ankle, or elbow) is involved. Obtain an orthopedic consultation so as to devise the best strategy for joint drainage.

For treatment of acute crystalline synovitis, nonsteroidal anti-inflammatory drugs (NSAIDs) initially should be given at their maximum recommended dosage until symptoms improve, then tapered gradually over several days. Indomethacin is highly effective, but adverse effects in some patients limit its utility. Other NSAIDs with short half-lives (eg, ibuprofen and diclofenac) can also be used.

Colchicine has a narrow therapeutic window, which limits its effectiveness. A low-dose regimen can be as effective as the higher-dose regimens advocated in the past, but it must be started at the first signs of an

attack: 1.2 mg followed by 0.6 mg 12 hours later. Colchicine must be used cautiously in the setting of renal insufficiency.

Corticosteroids are an effective alternative to NSAIDs and colchicine for patients in whom these drugs may be contraindicated or hazardous (eg, patients with advanced age, renal insufficiency, congestive heart failure, inability to take oral medications). Regimens include the following:

- Intramuscular (IM) injection of a long-acting crystalline preparation (eg, triamcinolone acetonide 60-80 mg), with an option to repeat once after 24-48 hours
- Prednisone 20-30 mg/day with a progressive taper over 7-10 days
- Intra-articular corticosteroid therapy

Medical management of acute polyarthritis

Hospitalize the patient in the presence of any of the following :

- Significant, concomitant internal organ involvement
- Signs of bacteremia, including vesiculopustular skin lesions, Roth spots, shaking chills, or splinter hemorrhages
- Systemic vasculitis
- Severe pain
- Severe constitutional symptoms
- Purulent (group III) synovial fluid in 1 or more joints
- Immunosuppression

An infectious etiology should receive first consideration. Obtain appropriate cultures (eg, blood, joint, cervix, urethra, or pharynx). Begin empiric antibiotic therapy if bacteremia or sepsis cannot be readily excluded. Extra-articular manifestations, such as a rash, hematologic abnormalities, or heart murmur, should be sought as important indicators of the diagnosis. Repeated examinations of the patient are required to detect diagnostic physical findings that may be absent at presentation.

Antibiotic therapy is indicated for septic polyarthritis or bacteremia with joint involvement (eg, disseminated gonococcemia). Systemic antibiotics are used after appropriate cultures are taken. Prolonged treatment of *Chlamydia* -induced reactive arthritis with antibiotics may be of benefit; this is not true for other forms of reactive arthritis.

Analgesics without anti-inflammatory properties may be appropriate as the initial treatment in patients with milder forms of acute rheumatic fever, viral arthritis (eg, parvovirus arthritis), or acute leukocytoclastic vasculitis. They also may be appropriate for those with polyarticular crystalline synovitis in whom significant concomitant medical problems preclude the use of NSAID or corticosteroid therapy. This therapy allows complete expression of the clinical manifestations of the disease, thereby aiding in diagnosis.

High-dose aspirin therapy can be used for acute rheumatic fever, with the goal of achieving a salicylate level of 20-30 mg/dL. High-dose nonsalicylate NSAID therapy is used to treat crystalline synovitis, acute viral arthritis, and polyarthritis related to rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), or other connective-tissue disorders.

Corticosteroids are used in persons with polyarthritis alone in whom high-dose NSAID therapy has failed or who cannot be treated safely with NSAIDs because of renal insufficiency, active gastrointestinal (GI) bleeding, or other conditions. Prednisone 15-20 mg/day (or the equivalent) is usually sufficient for acute polyarticular flares of RA.

High doses of prednisone (0.5-1 mg/kg/day) are used in the setting of severe constitutional symptoms, concomitant major organ involvement, or signs of systemic vasculitis. Examples include acute SLE, systemic-onset juvenile idiopathic arthritis, or acute rheumatic fever that fails to respond to NSAID therapy.

Medical management of chronic inflammatory monoarthritis

Diagnoses other than osteoarthritis should be considered if the patient has a synovial fluid white blood cell (WBC) count higher than 1000/ μ L, hemorrhagic synovial fluid, no significant radiographic changes associated with osteoarthritis, synovial proliferation, significant pain, or constitutional symptoms.

The initial diagnostic focus in a patient with a chronic inflammatory monoarthritis is always on a potential infectious etiology. Lyme arthritis can manifest as a subacute or chronic inflammatory monoarthritis; its diagnosis is based on the results of serologic testing. Antibiotic treatment is indicated.

Perform a synovial biopsy and culture if the initial evaluation (including synovial fluid cultures) fails to establish a specific diagnosis. Consider aseptic necrosis in a joint with noninflammatory joint fluid.

Therapy for chronic gout requires allopurinol or febuxostat to correct hyperuricemia. Suppress chronic inflammation with NSAIDs, colchicine (eg, 0.6 mg twice daily), or both. Intra-articular corticosteroid therapy may also be appropriate. Other crystalline arthropathies (eg, involving calcium pyrophosphate or

hydroxyapatite) are also treated by suppressing chronic inflammation with NSAIDs, colchicine, or both. Intra-articular corticosteroid therapy may also be appropriate for these conditions.

A monoarticular presentation of a systemic rheumatic disease is treated with systemic therapies appropriate to the rheumatic disease, particularly if intra-articular corticosteroids are contraindicated or ineffective for long-term suppression of the monoarticular disease.

Medical management of chronic inflammatory polyarthritis

Certain diagnoses should be sought during the initial patient evaluation because specific (and potentially curative) therapies are needed. These include chronic polyarticular gout, subacute bacterial endocarditis, and hepatitis C–related syndromes (eg, cryoglobulinemia and arthritis). However, treatment with NSAIDs is often initiated before a firm diagnosis is established.

Disease-modifying antirheumatic drugs (DMARDs) such as methotrexate, leflunomide, and tumor necrosis factor-alpha (TNF- α) antagonists should be started relatively early in the course of rheumatoid or psoriatic arthritis so as to prevent joint damage. Consultation with a rheumatologist is prudent to confirm these diagnoses and to allow initiation of appropriate DMARD therapy.

Corticosteroids in low doses (10 mg or less) may serve as a valuable adjunct to the treatment of chronic inflammatory arthritides, though attention must be paid to the adverse effects of long-term steroid use (eg, osteoporosis).

The choice of an NSAID is guided by the patient's comorbidities and past response to these drugs; it is also guided by cost and dosing frequency. Maximal doses of NSAIDs are generally required for effective management of chronic polyarthritides. However, lower doses may be used if the disease is being adequately suppressed with DMARDs.

DMARDs are used to suppress synovitis and thereby prevent or at least retard the development of joint damage or deformity. The choice of a DMARD regimen depends on a number of factors, including the underlying disease, comorbidities, and prior treatment responses. Guidelines for the use of DMARDs in various polyarthritides are presented in the specific articles describing these conditions (eg, Rheumatoid Arthritis).

Medical management of osteoarthritis

Management is most effective when it includes physical measures to reduce joint loading, an appropriate exercise regimen, medications, and, occasionally, surgery. Patient education is vital.

The natural history of osteoarthritis is punctuated by episodes of more intense joint pain, followed by long periods of relative quiescence. More persistent, chronic pain is a feature of advanced disease. Dosing of anti-inflammatory and analgesic medications should be calibrated to the severity of the joint pain. Acute episodes may require enforced joint rest for relief; use of crutches, a cane, splints, or other orthotic devices; and strict avoidance of certain activities. Prevention of symptomatic flares is key to proper management.

With regard to nonpharmacologic management, instruct the patient to attempt to achieve or maintain ideal body weight. Teach the patient joint preservation techniques. Recommend a physical therapy regimen that includes range-of-motion and flexibility, conditioning, and aerobic cardiovascular exercises. Prescribe orthotic devices (eg, a cane, walker, splint, or wedged insole) to rest or unload a joint. Recommend the use of devices to assist activities of daily living (eg, a tub seat, elevated toilet, dressing stick, or long-handled shoehorn).

As for pharmacologic management, mild disease can be treated with acetaminophen (up to 1 g 4 times daily), tramadol (50-100 mg 4 times daily), over-the-counter NSAIDs (eg, naproxen or ibuprofen) in analgesic doses, glucosamine (500 mg 3 times daily); or topical analgesics containing capsaicin, methylsalicylate, or an NSAID. Moderate disease is treated with NSAIDs.

Persistent symptoms that are not relieved by mild therapy often require NSAID administration for prolonged periods in anti-inflammatory doses. In this setting, give careful consideration to potential NSAID toxicities, including induction of GI ulcers and exacerbation of hypertension, renal impairment, and heart failure. Use the lowest effective dose. Monitor blood pressure and kidney function. Coadminister a gastroprotective agent (eg, a proton pump inhibitor [PPI]) with a conventional generic NSAID in patients at high risk for an adverse GI event.

Intra-articular hyaluronan may provide relief of symptomatic knee osteoarthritis for periods as long as 1 year. Intra-articular corticosteroids are beneficial for patients with symptomatic effusions. Use is limited to 1 injection per joint every 3 months.

Severe disease is the presence of intractable pain or significant incapacity, and this is an indication for surgical intervention (see below). Opiate analgesics may be used for intractable pain, but first thoroughly consider the risks associated with their long-term use.

Medical management of soft tissue rheumatic pain disorder

Regional musculoskeletal pain syndromes

In patients with regional musculoskeletal pain syndromes (eg, tendonitis, bursitis, acute soft tissue injuries, and regional myofascial pain syndromes), allow the soft tissue injury to heal with a short period of enforced rest. This can be achieved with immobilization or avoidance of activities that require the use of the involved part.

Provide pain relief using both nonpharmacologic modalities (eg, local heat or cold, electrical stimulation, massage) and pharmacologic agents (eg, oral analgesics, NSAIDs, muscle relaxants, corticosteroid injections, and topical formulations).

Prescribe an exercise program to be performed at home or under the guidance of a physical therapist. The goals should include stretching, muscle strengthening, and education about proper body mechanics. Identify and eliminate factors that have aggravated or precipitated soft tissue pain (eg, posture, repetitive trauma, or poor body mechanics).

Generalized noninflammatory soft tissue rheumatic pain syndromes

In patients with generalized noninflammatory soft tissue rheumatic pain syndromes (eg, fibromyalgia and hypermobility syndrome), screen for coexistent depression, and treat it if present. Screen for a sleep disorder, and treat it if present. Emphasize the primary role of low-level aerobic exercise in treatment. Treat pain using agents that are acceptable for prolonged use and that do not promote physical dependence.

Surgical management

Surgical management of arthritis may be indicated in patients with the following:

- Uncertain diagnosis
- Acute septic arthritis
- RA
- Osteoarthritis

When the diagnosis is uncertain, perform a synovial or bone biopsy.

In patients with acute septic arthritis, drain hip and shoulder joints. Use arthroscopic surgical techniques to drain joints that are not responding to repeated percutaneous needle drainage. If the prosthesis is infected, the hardware may have to be removed or the polyethylene components exchanged to prevent recurrence of the infection.

In patients with RA, perform total arthroplasty on large joints (eg, hips, knees, and shoulders). The primary indication is relief of pain that has not been relieved by medical therapy. Improvement of function and motion are secondary goals that are not always attainable.

Perform wrist synovectomy and dorsal hand tenosynovectomy for persistent synovitis of the dorsum of the wrist and hand that threatens tendon integrity. Correct atlantoaxial or subaxial subluxation in the cervical spine. Perform reconstructive surgery of the hands and feet.

In patients with osteoarthritis, use arthroscopic surgery to correct internal derangements (eg, meniscal tears) and to remove loose bodies that are causing mechanical symptoms. Total arthroplasty is indicated to relieve the pain of advanced joint disease; improvement of function and motion are secondary goals. Joint fusion is indicated for joints such as the ankle (eg, triple arthrodesis) and the carpometacarpal joints to relieve pain and instability.

Materials for self-control:

A. Objectives for the self.

1. Reasons for RA, OA, AS, gout, psoriatic jet, viral arthritis.
2. Criteria for diagnosis of diseases of the joint syndrome.
3. Criteria for differential diagnosis of rheumatoid arthritis, gout, RA, OA, RS reactive arthritis.

B. Objectives for the self.

1. A 21 y.o. man complains of having morning pains in his back for the last three months. The pain can be relieved during the day and after physical exercises. Physical examination revealed reduced mobility in the lumbar part of his spine, increase of muscle tonus in the lumbar area and sluch during moving. X-ray pattern of spine revealed bilateral sclerotic changes in the sacrolumbal part. What test will be the most necessary for confirming a diagnosis?

- A. ESR
- B. HLA-B27
- C. Rheumatoid factor
- D. Uric acid in blood plasma
- E. Antinuclear antibodies

2. A 30 y.o. female with rheumatoid arthritis of five years duration complains of pain in the frist three

fingers of her right hand over past 6 weeks. The pain seems especially severe at night often awakening her from sleep. The most likely cause is?

- A. Sensory peripheral neuropathy
- B. Atlanto-axial subluxation of cervical spine
- C. Carpal tunnel syndrome
- D. Rheumatoid vasculitis
- E. Rheumatoid arthritis without complication

3. A 47-year-old obese man complained of periodic attacks of acute arthritis in the 1st left tarsophalangeal joint. Lab exam revealed increased serum rate of uric acid. What is the diagnosis?

- A. Reiter's disease
- B. Gout arthritis
- C. Rheumatoid arthritis
- D. Rheumatic arthritis
- E. Osteoarthritis

4. A 30 y.o. man complains of intense pain, skin reddening in the region of ankle joint, temperature rise up to 39°C. He fell ill suddenly. In the past there were such onsets that lasted for 5-6 days and didn't cause any residual changes of the joint. The skin over the joint is hyperemic, without distinct outlines and infiltrative bank at the periphery. What is the most probable diagnosis?

- A. Rheumatoid arthritis
- B. Infectious arthritis
- C. Gout
- D. Erysipelatous inflammation
- E. Osteoarthrosis

5. A 17 y.o. patient complains of acute pain in the knee joint and to 38°C. He was ill with angina 3 weeks ago. Objectively: deformation and swelling of the knee joints with skin hyperemia. Small movement causes an acute pain in the joints. Which diagnose is the most correct?

- A. Systemic lupus erythematosus
- B. Rheumatism, polyarthritis
- C. Reactive polyarthritis
- D. Infectious-allergic polyarthritis
- E. Rheumatoid arthritis

6. A 31 y.o. woman has complained for 3 years of pain and swelling of radiocarpal and metacarpophalangeal articulations, morning stiffness that lasts up to 1,5 hours. Two weeks ago she felt pain, swelling and reddening of knee joints, body temperature raised up to 37,5°C. Examination of her internal organs revealed no pathologic changes. Her diagnosis was rheumatoid arthritis. What changes in X-ray pictures of her joints are the most probable?

- A. Multiple marginal osteophytes
- B. Constriction of joint space, subchondral osteosclerosis
- C. Cysts in subchondral bone
- D. Constriction of joint space, usura
- E. Epiphysis osteolysis

7. A 60 y.o. patient complains of pain in interphalangeal joints of hand that gets worse during working. Objectively: distal and proximal joints of the II-IV fingers are deformed, with Heberden's and Bouchard's nodes, painful, stiff. X-ray picture of joints: joint spaces are constricted, there are marginal osteophytes, subchondral sclerosis. What is the most probable diagnosis?

- A. Osteoarthritis
- B. Reiter's disease
- C. Bechterew's disease
- D. Rheumatic arthritis
- E. Psoriatic arthritis

Answers:

- | | | | |
|------|------|------|------|
| 1. b | 3. b | 5. b | 7. a |
| 2. c | 4. c | 6. d | |

Recommended literature:

A. Main:

1. "Harrison's principles of internal medicine", Editors: Anthony S. Fauci, Dennis L. Kasper, Stephen L. Hauser, Dan L. Longo, Joseph Loscalzo, McGraw-Hill Education / Medical; 19 edition (April 8, 2015), 1-2 volumes, 3000 p.
2. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)
3. CURRENT Diagnosis and Treatment Emergency Medicine, Seventh Edition (LANGE CURRENT Series) by C. Keith Stone (May 23, 2011)
4. Goldman's Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer.—24th ed. Elsevier Sanders. Rev. ed. of: Cecil medicine. 23rd ed. — 2012. p.

Composed by Prikhodko N.P.