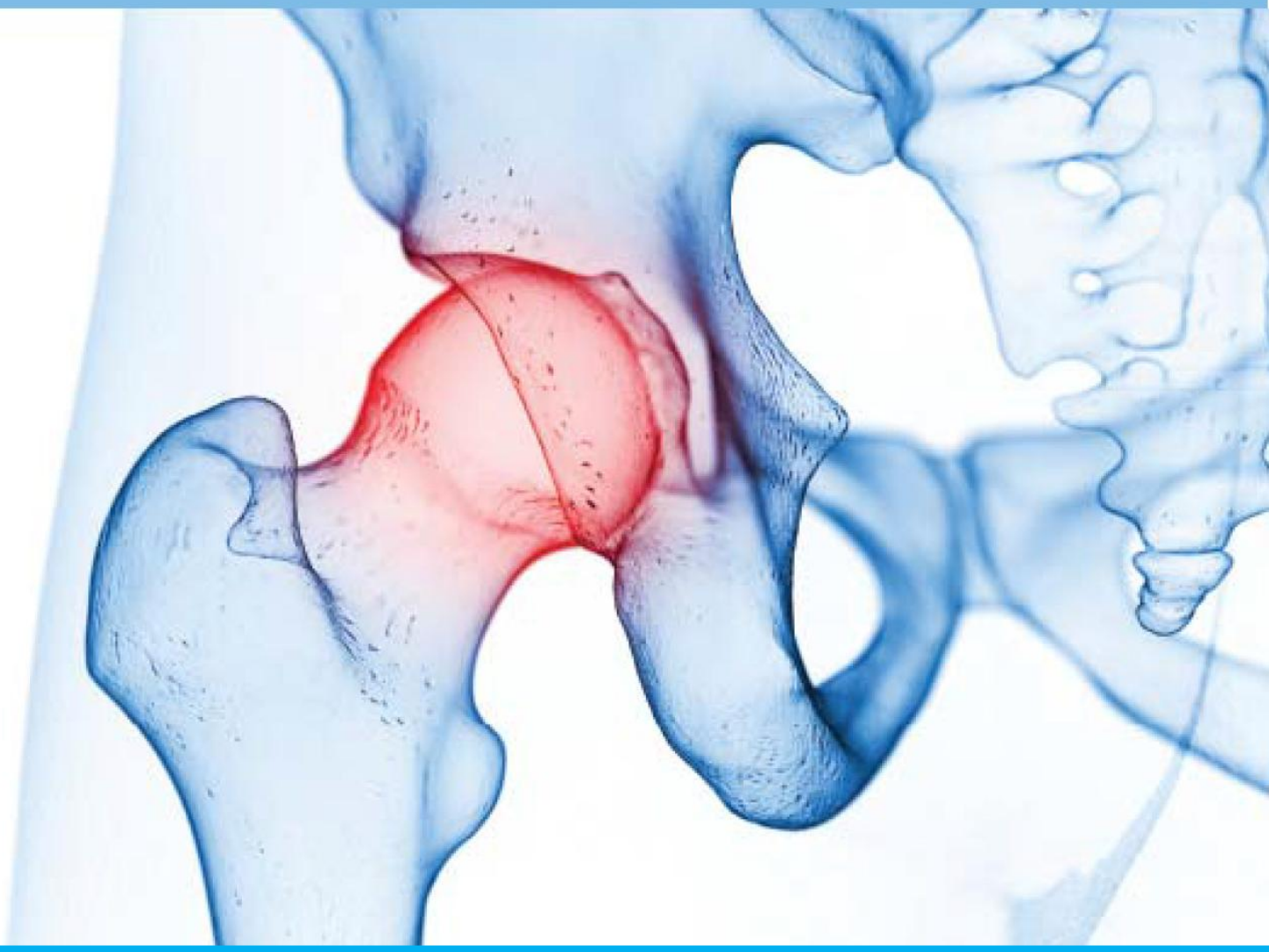
6th October university

Faculty of medicine

Internal Medicine
For Fourth Level Medical Students

Basics of Rheumatology



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Chapter One

Introduction To Rheumatology

Objectives: by the end of this chapter, the student will:

1) Understand the different methods for classifying rheumatologic diseases.
2) Understand the pathophysiology of various rheumatologic diseases.
3) Know examples for each class of rheumatologic diseases.
4) Understand the overall diagnostic tools used in investigating rheumatologic diseases.

- Rheumatic diseases are disorders of connective tissue in which general or localized inflammation frequently manifests as pain attributable to peripheral joints, the spine, or muscles.
- Systemic features such as stiffness, fever, or weight loss and a multitude of extra-musculoskeletal features, ranging from skin rashes to renal dysfunction, often accompany rheumatic diseases.
- Rheumatic diseases are often also termed connective tissue diseases, understandably, because connective tissue is the most abundant tissue in the body supporting and connecting other tissues and organs.
- Musculoskeletal disorders may manifest as acute, subacute, or chronic problems.

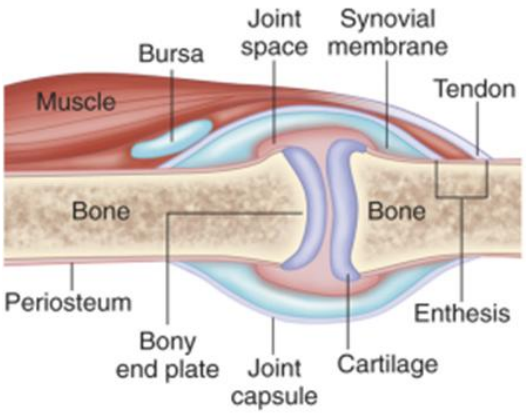
I) Classification Of Rheumatic Diseases

DEGENERATIVE DISEASES OF BONES AND JOINTS	SYSTEMIC AUTOIMMUNE DISEASES	SERONEGATIVE SPONDYLOARTHROPATHIES	VASCULAR RHEUMATIC DISEASES	AUTOINFLAMMATORY DISEASES	PAIN DISORDERS
Osteoarthritis	Rheumatoid arthritis	Ankylosing spondylitis	ANCA- associated vasculitis	Adult-onset Still's disease	Regional myofascial pain syndromes
DISH	Systemic lupus erythematosus	Psoriatic arthritis	Temporal artery vasculitis	Crystal diseases	Tendonitis/bursitis
Degenerative disc disease	Sjögren's syndrome	Reactive arthritis	Polymyalgia rheumatica	Pediatric periodic fever syndromes	Adhesive capsulitis
Spinal stenosis	Inflammatory myopathies (polymyositis, dermatomyositis)	Enteropathic arthritis	Behçet's disease		Reflex sympathetic dystrophy
Osteoporosis	Systemic sclerosis				Pain with hypermobility syndromes
					Fibromyalgia*

*The only pain disorder that has not been associated primarily with inflammation.
ANCA = antineutrophil cytoplasmic antibody; DISH = diffuse idiopathic sclerosing hyperostosis (also linked to metabolic factors, including elevated growth hormone).

II) Categories of Musculoskeletal Diseases

Category	Prototypes	Useful Tests
Synovitis	❖ Rheumatoid arthritis ❖ Autoimmune diseases	❖ Rheumatoid factor, ESR ❖ Antinuclear antibody test
Enthesopathy	Ankylosing spondylitis and spondyloarthropathies	Sacroiliac radiographs
Crystal-induced synovitis	Gout	Joint fluid crystal examination
Joint space disease	Septic arthritis	Joint fluid culture
Cartilage degeneration	Osteoarthritis	Radiographs of affected area
Inflammatory myopathy	Polymyositis	Muscle enzymes, electromyography, muscle biopsy
Local and regional conditions	Tendonitis or bursitis	Aspirate bursa if infection is suspected
General conditions	Polymyalgia rheumatica	Elevated ESR



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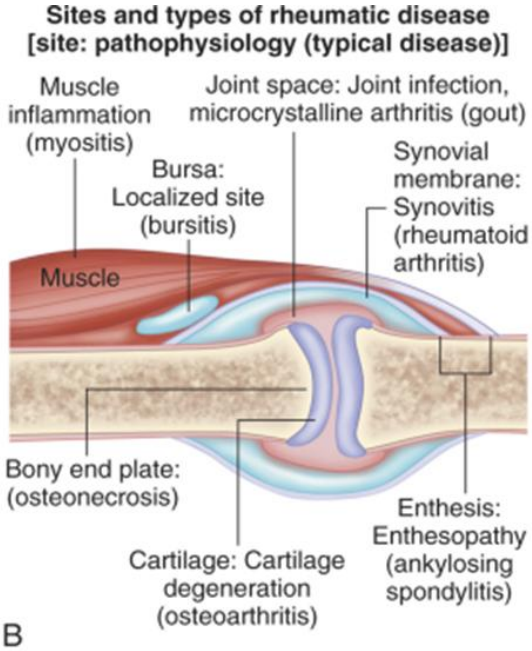

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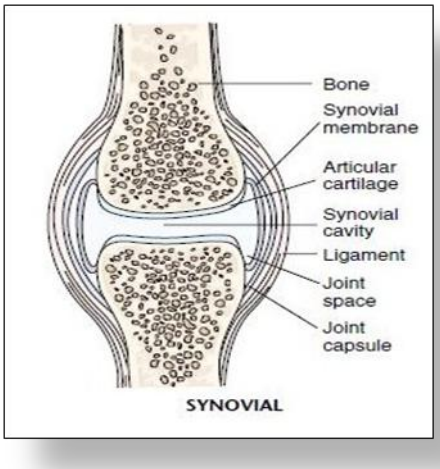
III) Basic Anatomy Of The Joints

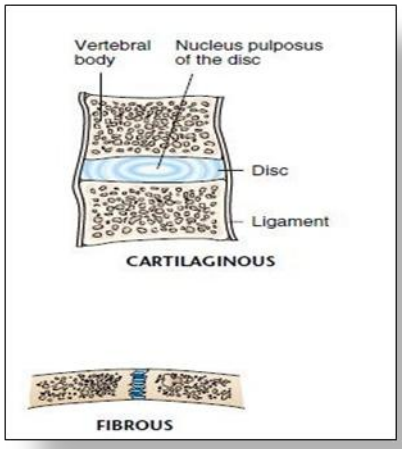
Types Of Joints

Type of Joint	Extent of Movement	Example
Synovial	Freely movable	Knee, shoulder
Cartilaginous	Slightly movable	Vertebral bodies of the spine
Fibrous	Immovable	Skull sutures

Synovial joints



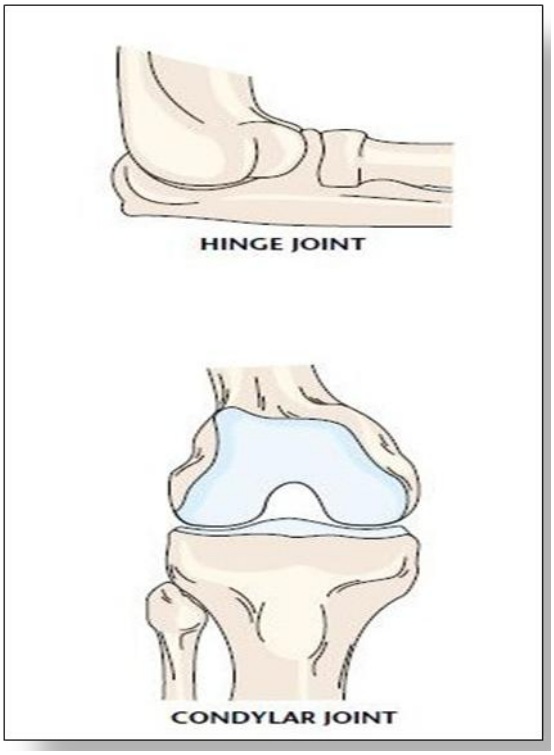
Cartilaginous and fibrous joints

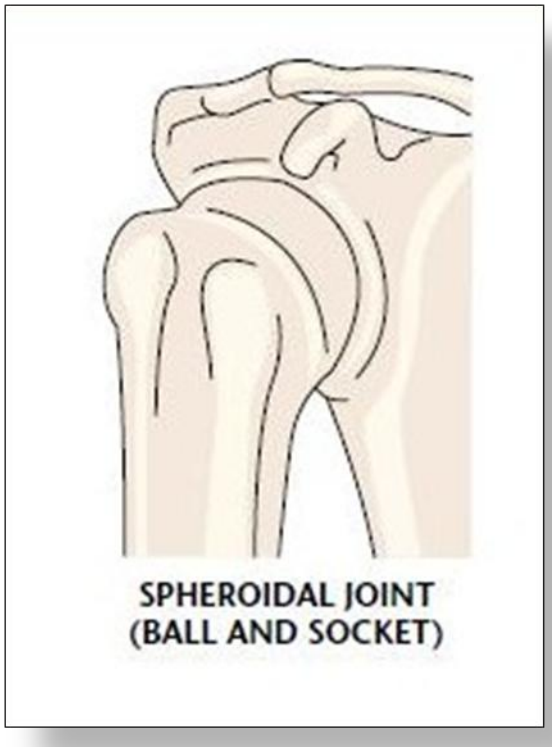


Types Of Synovial Joints

Synovial Joints			
Type of Joint	Articular Shape	Movement	Example
Spheroidal (ball and socket)	Convex surface in concave cavity	Wide-ranging flexion, extension, abduction, adduction, rotation, circumduction	Shoulder, hip
Hinge	Flat, planar	Motion in one plane; flexion, extension	Interphalangeal joints of hand and foot; elbow
Condylar	Convex or concave	Movement of two articulating surfaces not dissociable	Knee; temporo-mandibular joint

Synovial Joints





IV) Laboratory Testing In The Rheumatic Diseases

1) Markers of inflammation.
2) Rheumatoid factor.
3) Antibodies.
4) Joint fluid analysis.

Markers Of Inflammation

- The immediate response to inflammatory stimuli is termed the acute phase response and comprises a set of proteins produced primarily in the liver in response to cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α (TNF-A), and IL-1.
- These cytokines are produced by macrophages and dendritic cells after stimulation.
- Of the proteins stimulated during the acute phase response is C-reactive protein (CRP).
- Another simple laboratory text reflecting the acute phase response is the erythrocyte sedimentation rate (ESR).

- Patients with inflammation frequently have a leukocytosis or thrombocytosis, most likely reflecting the action of cytokines and other mediators, with chronic inflammation, anemia of chronic disease can also occur.

Rheumatoid Factor

- RF comprises a family of specificities that bind to the immunoglobulin G (IgG) molecule.
- RFS occur in approximately 80% of patients with RA and represent a criterion for the classification or diagnosis of this disease.
- Furthermore, high levels of RFS are often associated with a worse prognosis, the occurrence of erosion as measured by radiographs, and deformity.

Causes of positive rheumatoid factor:

DISEASES	FREQUENCY
Rheumatoid arthritis	50-90%
Systemic lupus erythematosus	15-35%
Sjögren's syndrome	75-95%
Systemic sclerosis	20-30%
Polymyositis/dermatomyositis	5-10%
Cryoglobulinemia	40-100%
Mixed connective tissue disease	50-60%
Aging (>70 yr)	10-25%
Infection	
Bacterial endocarditis	25-50%
Liver disease	15-40%
Tuberculosis	8%
Syphilis	Up to 13%
Parasitic diseases	20-90%
Leprosy	5-58%
Viral infection	15-65%
Pulmonary disease	
Sarcoidosis	3-33%
Interstitial pulmonary fibrosis	10-50%
Silicosis	30-50%
Asbestosis	30%
Miscellaneous diseases	
Primary biliary cirrhosis	45-70%
Malignancy	5-25%

Antinuclear Antibodies

- The expression of antibodies to components of the cell nucleus (antinuclear antibodies, or ANAS) is characteristic of CTD and is essentially invariable in patients with SLE.
- These antibodies target a host of nuclear macromolecules, including

DNA, RNA, and proteins as well as complexes of proteins with nucleic acid.

Selected Antinuclear Antibodies and Rheumatic Diseases:

Antibody	Disease Association
❖ Anti-histone	❖ Drug-induced lupus (>95%)
❖ Anti-double-stranded DNA	❖ SLE (50%)
❖ Anti-Sm	❖ SLE (30%)
❖ Anti-Ro (SS-A)	❖ SLE (30%); Sjögren's syndrome (70–80%)
❖ Anti-SCL-70	❖ PSS (40–70%); CREST (10–20%)
❖ Anticentromere	❖ CREST (80%); PSS (30%)
❖ Anti-Jo-1	❖ PM/DM (18–25%)

CREST = calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia; DM = dermatomyositis;; PM = polymyositis; PSS = progressive systemic sclerosis (diffuse scleroderma); SLE = systemic lupus erythematosus;.

Joint Fluid Analysis

- Analysis of joint fluid can provide decisive data in the evaluation of arthritis and, in some instances, a definitive diagnosis.
- This analysis is essential in the setting of acute monoarthritis to investigate the possibility of infection.

V) Imaging Studies In The Rheumatic Diseases

1) Radiography.
2) MRI.
3) Computed tomography.
4) Ultrasonography.

Radiography

- The radiograph is the initial imaging technique used for evaluation of arthropathy.
- Radiographs are affordable, easily available and render images of the joint with high spatial resolution.
- Frequently, a radiograph is all that is needed to assess joint damage.

Magnetic Resonance Imaging

- MRI has become the imaging modality of choice for evaluation of joint soft tissue, marrow structures, and muscle pathology, because it provides high-contrast resolution that enables the differentiation of various soft tissues from each other.
- With special regard to rheumatic disease, MRI demonstrates joint effusion, intra-articular masses, synovitis, damage to hyaline and fibrocartilage, tendons, ligaments, bone, muscle, and surrounding soft tissue.
- It is also used to evaluate the spine for disc disease, nerve root compression, and inflammatory changes.

Ultrasonography

- Ultrasound provides a less expensive, noninvasive method to visualize joint structures such as tendons, ligaments, fibrocartilage, muscles, and soft tissue masses without the use of ionizing radiation.
- Fluid within tendon sheaths can be easily assessed. fluid, blood, pus, and mucin in ganglia, cysts, bursae, hematomas, seromas, and abscesses can be differentiated from solid masses based on echogenicity.

Computed Tomography

- CT is useful for evaluating vertebral alignment and fractures as well as abnormalities of the flat bones and pelvis.
- Complex fractures are best evaluated with CT
- Joints that are difficult to assess on radiographs, including the sacroiliac, temporomandibular, wrist, and sternoclavicular joints, are well seen with CT
- Internal derangement of these joints is better appreciated on MRI, which provides better soft tissue contrast.

Chapter Two

Systemic Lupus Erythematosus

Objectives: by the end of this chapter the student will:

1) Understand the nature of systemic lupus as an immune disease.
2) Know the epidemiology of the disease.
3) Know the various clinical presentations of the disease.
4) Understand the various diagnostic methods for diagnosing the disease.
5) Understand the various treatment options of the disease.

Definition

- Systemic lupus erythematosus (SLE) is a multisystemic autoimmune disease that results from immune system–mediated tissue damage.

Epidemiology

- A notable feature of SLE is that it occurs much more frequently in females than in males.
- The female-to-male ratio is approximately 8:1 to 9:1 in adults, and most cases are diagnosed between the ages of 15 and 44 years.

CLINICAL MANIFESTATIONS

Symptoms And Signs

- *Constitutional Symptoms*

  Fatigue, headaches, weight loss, and fevers are common, along with generalized arthralgias, myalgias, and lymphadenopathy.

- *Cutaneous and Mucous Membranes*

  The erythematous facial rash with a butterfly distribution across the malar and nasal prominences and sparing of the nasolabial folds is the classic rash of SLE and is seen in 30 to 60% of patients.

  The butterfly rash is often triggered by sun exposure.

- *Musculoskeletal System*

  Arthralgias and nonerosive arthritis are among the most common clinical features of SLE.

  The proximal interphalangeal and metacarpophalangeal joints of the hand

are most commonly symptomatic, along with the knees and wrists.

- *Renal System*

  Kidney involvement in SLE is common, with 74% of patients being affected at some time in the course of disease and is a poor prognostic indicator.

  Renal pathology is generally attributed to the deposition of circulating immune complexes or in situ formation of these complexes in glomeruli and results in the activation of complement and subsequent recruitment of inflammatory cells.

- *Cardiovascular System*

  Pericarditis is the most common cardiac manifestation, but it is sometimes recognized only on imaging studies or at autopsy.

  Pericarditis is usually manifested as substernal chest pain that is improved by bending forward and can be exacerbated by inspiration or coughing. The symptoms and effusions associated with pericarditis are quite responsive to moderate-dose (20 to 30 mg/day of prednisone) corticosteroid treatment.

  Structural valve abnormalities in SLE range from the sterile nodules originally described by Libman and Sacks to nonspecific valve thickening.

  Premature and accelerated atherosclerosis is increasingly recognized as being prevalent in lupus patients, and preclinical atherosclerotic carotid plaque has been documented in 37% of SLE patients.

  Though not specific to SLE, **Raynaud's phenomenon**, characterized by episodic vasospasm and occlusion of the digital arteries in response to cold and emotional stress, is a feature in up to 60% of SLE patients and contributes to pain and sometimes necrosis of the distal ends of extremities.

- *Pulmonary System*

  Pleuritis is the most frequent manifestation of pulmonary involvement in SLE and occurs in about 30% of patients at some point in their disease course.

- *Neuropsychiatric Syndromes Observed In Systemic Lupus Erythematosus*

  Central Nervous System

  - Aseptic meningitis
  - Cerebrovascular disease
  - Demyelinating syndrome

- Movement disorder (chorea)
- Seizure disorders
- Psychosis

Peripheral Nervous System

- Mononeuropathy, single/multiplex
- Acute inflammatory demyelinating polyradiculoneuropathy (Guillain-Barré syndrome)
- Polyneuropathy

Cranial nerve and ocular involvement, most likely based on vasculopathy and focal ischemia, can sometimes affect vision. Ocular examination of the retina can reveal cotton-wool spots as a result of retinal ischemia or necrosis.

❖ *Gastrointestinal System*

Though uncommon, **vasculitis of the gastrointestinal** tract or mesentery can result in pain and bowel necrosis. Less common than pleuritis and pericarditis, peritonitis can be manifested as peritoneal effusion and abdominal pain.

❖ *Lymphadenopathy*

About a third of SLE patients demonstrate diffuse lymphadenopathy at some time during the course of their disease. The nodes are often nontender, and lymphoma is sometimes considered in the differential diagnosis.

❖ *Hematologic System*

In addition to autoantibody specificities that are fairly specific for SLE (anti-DNA, anti-Sm), antibodies that target each of the cellular blood elements are also common.

Anemia is present in about 50% of patients and is multifactorial. It can be associated with a positive Coombs test or microangiopathic hemolysis or reflect chronic disease (normochromic, normocytic).

Idiopathic thrombocytopenic purpura can be an early manifestation of SLE, and thrombocytopenia, induced by antiplatelet antibodies, can sometimes lead to a life-threatening risk for hemorrhage.

Antiphospholipid Antibody Syndrome

➢ Antiphospholipid antibodies represent a distinct class of autoantibodies that are seen in about a third of SLE patients but can also be present in individuals who do not carry a diagnosis of SLE.

➢ Whether in primary antiphospholipid syndrome or in SLE,

antiphospholipid antibodies have been associated with venous and arterial thromboses. In addition to vascular thromboses, clinical manifestations of antiphospholipid syndrome include thrombotic microangiopathic glomerular disease, cardiac valve lesions, livedo reticularis, thrombocytopenia, hemolytic anemia, and central nervous system disease.

Diagnosis

REVISED CRITERIA FOR CLASSIFICATION OF SYSTEMIC LUPUS ERYTHEMATOSUS

For the purpose of identifying patients in clinical studies, a person shall be said to have systemic lupus erythematosus if any 4 or more of the 11 criteria are present, serially or simultaneously, during any interval of observation.

Criterion	Definition
1. Malar rash	Fixed erythema, flat or raised, over the malar eminences, tending to spare the nasolabial folds
2. Discoid rash	Erythematous raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur in older lesions
3. Photosensitivity	Rash as a result of unusual reaction to sunlight, by patient history or physician observation
4. Oral ulcers	Oral or nasopharyngeal ulceration, usually painless, observed by a physician
5. Arthritis	Nonerosive arthritis involving two or more peripheral joints, characterized by tenderness, swelling, or effusion
6. Serositis	Pleuritis-convincing history of pleuritic pain or rub heard by a physician or evidence of pleural effusion *or*
	Pericarditis-documented by electrocardiogram or rub or evidence of pericardial effusion
7. Renal disorder	Persistent proteinuria >0.5 g/dL or >3+ if quantitation not performed *or*
	Cellular casts-may be red blood cell, hemoglobin, granular, tubular, or mixed
8. Neurologic disorder	Seizures-in the absence of offending drugs or known metabolic derangements, (e.g., uremia, ketoacidosis, or electrolyte imbalance) *or*
	Psychosis-in the absence of offending drugs or known metabolic derangements, (e.g., uremia, ketoacidosis, or electrolyte imbalance)
9. Hematologic disorder	Hemolytic anemia-with reticulocytosis, *or*
	Leukopenia-<4.0 × 10^9/L (4000/mm^3) total on two or more occasions *or*
	Lymphopenia-<1.5 × 10^9/L (1500/mm^3) on two or more occasions *or*
	Thrombocytopenia-<100 × 10^9/L (100 × 10^3/mm^3) in the absence of offending drugs
10. Immunologic disorder	Anti-DNA antibody to native DNA in abnormal titer *or*
	Anti-Sm-presence of antibody to Sm nuclear antigen *or*
	Positive finding of antiphospholipid antibodies based on (1) an abnormal serum level of IgG or IgM anticardiolipin antibodies, (2) a positive test for lupus anticoagulant using a standard test, or (3) a false-positive serologic test for syphilis known to be positive for at least 6 months and confirmed by negative *Treponema pallidum* immobilization or fluorescent treponemal antibody absorption test
11. Antinuclear antibody	An abnormal titer of antinuclear antibody by immunofluorescence or an equivalent assay at any point in time and in the absence of drugs known to be associated with drug-induced lupus syndrome

Laboratory Findings

- All cellular elements of blood can be affected in lupus, so the complete blood count is an essential test that aids in diagnosis and management. A prolonged partial thromboplastin time can indicate the presence of pathogenic antiphospholipid antibodies.
- Urinalysis with microscopic evaluation is another essential laboratory test in view of the frequency of proteinuria in lupus and the significant morbidity associated with GN.

In addition to protein, red blood cells, white blood cells, and cellular casts suggest active glomerular disease.

- The ANA test is positive in virtually all patients and does not need to be repeated once it has been documented to be positive
- Anti–double-stranded DNA antibodies are common in SLE
- Anti-Sm antibodies are highly specific for SLE and, along with anti-RNP antibodies
- Decreased levels of two of the more stable complement components, C3 and C4, are typically measured in serum, and decreased C3 and C4 levels are often indicators of enhanced consumption and increased disease activity.

Treatment

Medications

- NSAIDs usually control SLE-associated arthritis, arthralgias, fever, and serositis but not fatigue, malaise, or major organ system involvement
- Hydroxychloroquine may be effective in the treatment of rash, photosensitivity, arthralgias, arthritis, alopecia, and malaise associated with SLE and in the treatment of discoid and subacute cutaneous lupus erythematosus

- Glucocorticoid therapy
  - Indications for systemic glucocorticoids include **(a)** life-threatening manifestations of SLE, such as glomerulonephritis, CNS involvement, thrombocytopenia, and hemolytic anemia; and **(b)** debilitating manifestations of SLE (fatigue, rash) that are unresponsive to conservative therapy.
  - IV pulse therapy in the form of methylprednisolone, 500 mg IV q12h for 3-5 days, has been used in SLE in such life-threatening situations as rapidly progressive renal failure, active CNS disease, and severe thrombocytopenia.

- Immunosuppressive therapy
  - Indications for immunosuppressive therapy in SLE include (a) such life-threatening manifestations of SLE as glomerulonephritis, CNS involvement, thrombocytopenia, and hemolytic anemia; and (b) the inability to reduce corticosteroid dosage or severe corticosteroid side effects.

- Often, cyclophosphamide is used for life-threatening manifestations of SLE.
- Azathioprine and mycophenolate mofetil are used more often as steroid-sparing agents but may not be as effective as cyclophosphamide in treating nephritis.

Chapter Three

Rheumatoid Arthritis and Seronegative Spondyloarthropathies

Objectives: by the end of this chapter the student will:

1) Understand the nature of rheumatoid arthritis as an immune mediated disease.
2) Know the epidemiology of the disease.
3) Know the clinical features of the disease including extra-articular features.
4) Understand the principles of diagnosing the disease.
5) Know the various treatment options of the disease.
6) Understand the concept of seronegative spondyloarthropathies.
7) Know the various categories of spondyloarthropathies.
8) Know the various clinical features and treatment of spondyloarthropathies.

Definition

- Rheumatoid arthritis (RA) is a systemic, chronic inflammatory disease affecting multiple tissues but principally attacking the joints to produce a nonsuppurative proliferative synovitis that frequently progresses to destroy articular cartilage and underlying bone with resulting disabling arthritis.
- When extra-articular involvement develops-for example, of the skin, heart, blood vessels, muscles, and lungs-RA may resemble SLE or scleroderma.

Epidemiology

- The prevalence in women is two or three times greater than in men. RA can occur at any age, but onset before the age of 45 years in men is uncommon.
- The incidence of RA in women increases with age until approximately 45 years of age, then reaches a plateau.

Clinical Manifestations

Articular Manifestations

- RA can affect any of the synovial (diarthrodial) joints, Most commonly, the disease starts in the **MCP, PIP, and MTP joints, followed by the wrists, knees, elbows, ankles, hips, and shoulders in roughly that order.**
- RA may involve the **upper part of the cervical spine**, particularly the C1–C2 articulation.
  - **Hands**

    Typical early disease starts with swelling of the PIPs and MCPs. The DIP joints are rarely involved; significant involvement of the DIP joints should suggest the possibility of a different diagnosis.

    Ulnar deviation and swan-neck deformities (hyperextension of the PIP joints) that are commonly seen in late, more established disease**.** **Boutonniere (or buttonhole) deformities** also occur as a result of hyperextension of the MCP joints. If the clinical disease remains active, hand function deteriorates.
  - **Feet**

    Feet, particularly the MTP joints, are involved early in most patients with RA.
  - **Wrists**

    The wrist joints are involved in most patients with RA; radial deviation is the rule, and patients with severe involvement may progress to volar subluxation.

    Even early in the course of the disease, synovial proliferation in and around the wrists may compress the median nerve, causing carpal tunnel syndrome.
  - **Large Joints**
    - Involvement **of knees, ankles, elbows, hips, and shoulders** is common. Characteristically, the whole joint surface is involved in a symmetrical fashion. Therefore, RA is not only symmetrical from one side of the body to the other but also symmetrical within the individual joint.
    - In the case of the knee , the medial and lateral compartments are both severely narrowed in RA; in contrast, in patients with osteoarthritis , only one compartment of the knee may be involved.
    - Synovial cysts may occur around any of the joints (large or small),

and they occasionally manifest as soft, fluctuant masses that present diagnostic challenges. Synovial cysts from the knee are perhaps the best examples of this phenomenon. When the knee produces excess synovial fluid, it may accumulate in the popliteal space (popliteal or Baker's cyst).

- **Neck**

  Although most of the axial skeleton is spared in RA, the cervical spine and especially the C1–C2 articulation is commonly involved. Bony erosions and ligament damage can occur in this area and may lead to subluxation.

Extra-Articular Manifestations Of Rheumatoid Arthritis

- **Skin:** Nodules, fragility, vasculitis, pyoderma gangrenosum
- **Heart:** Pericarditis, premature atherosclerosis, vasculitis, valve disease, and valve ring nodules
- **Lung:** Pleural effusions, interstitial lung disease, bronchiolitis obliterans, rheumatoid nodules, vasculitis
- **Eye:** Keratoconjunctivitis sicca, episcleritis, scleritis, scleromalacia perforans, peripheral ulcerative keratopathy
- **Neurologic:** Entrapment neuropathy, cervical myelopathy, mononeuritis multiplex (vasculitis), peripheral neuropathy
- **Hematopoietic:** Anemia, thrombocytosis, lymphadenopathy, Felty's syndrome
- **Kidney:** Amyloidosis, vasculitis
- **Bone:** Osteopenia

Sjogren's syndrome, characterized by failure of exocrine glands, occurs in a subset of patients with RA, producing sicca symptoms (dry eyes and mouth), parotid gland enlargement, dental caries, and recurrent tracheobronchitis.

Diagnosis

CLASSIFICATION CRITERIA FOR RHEUMATOID ARTHRITIS

1. Morning stiffness (≥1 hr)
2. Swelling (soft tissue) of three or more joints
3. Swelling (soft tissue) of hand joints (PIP, MCP, or wrist)
4. Symmetrical swelling (soft tissue)
5. Subcutaneous nodules
6. Serum rheumatoid factor
7. Erosions and/or periarticular osteopenia in hand or wrist joints seen on radiograph

Criteria 1 through 4 must have been continuously present for 6 wk or longer, and criteria 2 through 5 must be observed by a physician. A classification as rheumatoid arthritis requires that four of the seven criteria be fulfilled.

Differential Diagnosis

- The distribution of involved joints is a critical clue to the underlying diagnosis. The joints that are involved in patients with RA at presentation are also variable; typically, the symptoms start in the small joints of the hands (the proximal interphalangeal [PIP] and metacarpophalangeal [MCP] joints) and in the toes (metatarsophalangeal [MTP] joints). Importantly, RA usually spares the distal interphalangeal (DIP) joints and the small joints of the toes.

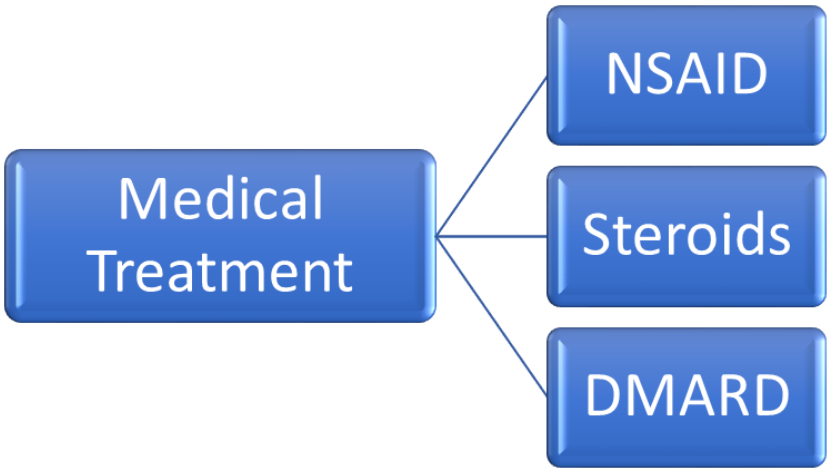
DIFFERENTIAL DIAGNOSIS OF RHEUMATOID ARTHRITIS

Viral arthritis (hepatitis B and C, parvovirus, rubella, others), Rheumatic fever, Sarcoidosis, Psoriatic arthritis, Systemic lupus erythematosus, and Osteoarthritis (erosive).

Laboratory Findings

- The most characteristic laboratory abnormality in RA is the presence of RF, which is found in approximately 80% of patients
- It is an antibody that recognizes immunoglobulin G as its antigen. The presence of RF is strongly associated with more severe articular disease as well as with essentially all of the extra-articular features
- Anti-CCP antibodies found in approximately 70% of patients with RA have a high specificity (93 to 98%), are often present before clinical disease is diagnosed, and are associated with aggressive erosive disease. RA is associated with many other autoantibodies, including antinuclear antibodies (approximately 30% of patients) and antineutrophil cytoplasmic antibodies, particularly of the perinuclear type (approximately 30% of patients).
- Most patients with RA have an anemia of chronic disease.

Treatment



- ***NSAIDs or selective COX-2 inhibitors*** are used as the initial therapy for RA and as an adjunct to immunomodulatory-immunosuppressive therapy. A longer-acting NSAID may facilitate patient compliance.
- ***Glucocorticoids*** are not curative and probably do not alter the natural history of RA; however, they are among the most potent anti-inflammatory drugs available.

  Indications for glucocorticoids include (a) symptomatic relief while waiting for a response to a slow-acting immunosuppressive or immunomodulatory agent, (b) persistent synovitis despite adequate trials of NSAIDs and immunosuppressive or immunomodulatory agents, and (c) severe constitutional symptoms (e.g., fever and weight loss) or extra-articular disease (vasculitis, episcleritis, or pleurisy).
- ***Immunomodulatory and immunosuppressive agents (including biologic therapy)*** appear to alter the natural history of RA by retarding the progression of bony erosions and cartilage loss. Because RA may lead to substantial long-term disability (and is associated with increased mortality), the current trend is to initiate therapy with such agents early in the course of RA.
  - Indications for the use of immunomodulatory or immunosuppressive agents include **(a)** active synovitis that does not respond to conservative management (e.g., NSAIDs); **(b)** rapidly progressive, erosive arthritis; and **(c)** dependence on steroids to control synovitis.
  - Methotrexate typically is the initial choice for moderate to severe RA. Hydroxychloroquine or sulfasalazine can be used as the initial choice in very mild RA. If response to the initial agent is unsatisfactory after an adequate trial (or if limiting toxicity supervenes), an alternate agent, such as leflunomide, a TNF α1 antagonist (Etanercept , Infliximab) or

IL-1 blocker, or azathioprine, can be used.

❖ ***Corrective surgical procedures***, including synovectomy, total joint replacement, and joint fusion, may be indicated in patients with RA to reduce pain and to improve function.

THE SPONDYLOARTHROPATHIES

❖ The spondyloarthropathies (SpAs) encompass a group of clinical syndromes that are linked in terms of disease manifestations and genetic susceptibility. The clinical subsets most commonly recognized are ankylosing spondylitis (AS), reactive arthritis (ReA), psoriatic arthritis (PsA), and enteropathic arthritis (EA).

Clinical Manifestations

- The SpAs have a strong predilection for the spine, in particular the sacroiliac joints.
- There is a predilection for sites of tendon insertion into bone (entheses), so enthesitis becomes one of the most specific clinical manifestations of the SpAs.
- Predilection for ocular inflammation, particularly acute anterior uveitis, is a common feature of all SpA subsets.

CONTRAST OF RHEUMATOID ARTHRITIS AND SPONDYLOARTHROPATHY

Feature	***Rheumatoid Arthritis***	***Ankylosing Spondylitis***	***Enteropathic Arthritis (with Crohn disease or Ulcerative colitis)***	***Psoriatic Arthritis***	***Reactive Arthritis (following GI or UT infections)***
Male-female ratio	1:3	3:1	1:1	1:1	10:1
Joint pattern	Symmetrical, peripheral	Axial	Axial and peripheral	Axial and asymmetrical peripheral	Axial and asymmetrical peripheral
Sacroiliac	0	Symmetrical	Symmetrical	Asymmetrical	Asymmetrical
Eye	Scleritis	Iritis	+/-	0	Iritis and conjunctivitis
Skin	Vasculitis	0	0	Psoriasis	Keratoderma
Rheumatoid factor	>80%	0	0	0	0

Medical Therapy

- *Nonsteroidal Anti-inflammatory Drugs*
- *Corticosteroids*

➢ The response to the commonly used intra-articular steroid injection of

peripheral joints in patients with SpA is often neither as dramatic nor as sustained as in patients with RA. Corticosteroid injection into the sacroiliac joints is usually performed under imaging guidance (fluoroscopy or computed tomography [CT]).

- Systemic corticosteroids (either orally or via an intravenous bolus protocol) have been used for severe symptomatic flares.
  - *Methotrexate*

    Concurrent with the widespread use of methotrexate (MTX) in patients with RA, there has been increasing use of MTX in patients with SpA. Generally, responses have been good, particularly for peripheral joint disease.
  - *Antibiotic Therapy*

    The current concept of the pathogenesis of RA postulates that a bacterial infection, usually gastrointestinal (GI) or genitourinary (GU), is the triggering event in an immunogenetically susceptible host.

Chapter Four

Scleroderma (Systemic Sclerosis)

Objectives: By the end of the chapter, the student will:

1) Understand the nature of systemic sclerosis.
2) Understand the clinical classification of systemic sclerosis.
3) Know the clinical presentation of the disease.
4) Understand the diagnostic methods of the disease.
5) Know the various treatment options of the disease.

Definition

- Scleroderma (systemic sclerosis) is a chronic systemic disease that targets the skin, lungs, heart, gastrointestinal tract, kidneys, and musculoskeletal system.
- The disorder is characterized pathologically by three features: ***(1) tissue fibrosis, (2) a proliferative and occlusive vasculopathy of small blood vessels, and (3) a specific autoimmune response associated with autoantibodies.***
- Because thickening of the skin is the most prominent clinical feature, scleroderma ("hard skin") has become the most popular name for this disease.
- The presence of disease-specific autoantibodies (e.g., anti-topoisomerase in the diffuse form, anticentromere in the limited form) and evidence of activated immune cells in tissues place scleroderma in the family of autoimmune disorders.

Classification

- Scleroderma is classified into two major subsets that are distinguished by the extent of skin thickening***: (1) limited and (2) diffuse cutaneous scleroderma.***

DIFFUSE SCLERODERMA

a) Widespread skin thickening involving distal and proximal body
b) Rapid onset (within 1 yr) of skin and other features after appearance of Raynaud's phenomenon
c) Significant visceral involvement including heart, lungs, gastrointestinal tract, or kidneys
d) High scores on disability and organ damage indices secondary to

extensive fibrosis of tissues

e) Poor prognostic signs include later age at onset, male gender, African American or Native American race, presence of large pericardial effusion, tendon friction rubs

f) Associated with antinuclear antibodies and the absence of anticentromere antibody

g) Highly variable disease course but overall poorer prognosis with 10-yr survival rate of 40–60%.

LIMITED SCLERODERMA

a) Limited to no skin thickening

b) Interval of several years or slow progression of disease from onset of Raynaud's phenomenon

c) Late visceral disease with unique features of isolated pulmonary hypertension and digital amputations secondary to severe ischemic vascular disease

d) CREST is a variant of limited scleroderma

e) Associated with primary biliary cirrhosis

f) Associated with anticentromere antibody

g) Relatively good prognosis with 10-yr survival rate of >70%.

CRITERIA AND CLASSIFICATION FOR SCLERODERMA (SYSTEMIC SCLEROSIS)

Classification	Criteria
a) Definite scleroderma	Scleroderma skin changes proximal to the MCP or MTP joints OR two of the following : (1) Sclerodactyly (scleroderma limited to the fingers); (2) digital pitting scars or loss of finger pad ؛ (3) bibasilar pulmonary fibrosis
b) Diffuse cutaneous scleroderma	Scleroderma skin changes above the elbows or knees and/or on the trunk (abdomen or chest)
c) Limited cutaneous scleroderma	Scleroderma skin changes distal to the elbows or knees and above the clavicles
d) CREST syndrome	Three or more of the following: subcutaneous calcinosis, Raynaud's phenomenon, esophageal dysfunction, sclerodactyly, and telangiectasia
e) Overlap syndromes	Diffuse or limited scleroderma plus typical features of one or more other CTD or autoimmune diseases
f) MCTD	Features of scleroderma, SLE, polymyositis, rheumatoid arthritis, and the presence of anti-U1snRNP
g) Undifferentiated CTD	Features of scleroderma but no clinical or laboratory findings sufficient to make a definite diagnosis
h) Systemic sclerosis sine scleroderma	Systemic features without skin involvement

CTD = connective tissue disease; MCP = metacarpophalangeal; MCTD = mixed connective tissue disease; MTP = metatarsophalangeal; SLE = systemic lupus erythematosus.

Incidence and Prevalence

- The average age at onset is between 35 and 50 years, and it is more common among women (3:1 to 7:1 female-to-male ratio). Although the disease is well described in the elderly, it is uncommon for it to manifest before 25 years of age, particularly the CREST variant.

Clinical Manifestations

- ***Raynaud's Phenomenon***
  a) In scleroderma, Raynaud's phenomenon and digital ischemia are the clinical manifestations of both fixed structural vascular disease and abnormal regulation of local blood flow.
  b) Digital pitting with loss of fingertip tissue and small, painful, superficial ulcerations are common and are usually secondary to disease in the small arteries and arterioles of the skin. Large, deep ulceration of the distal finger is a consequence of larger vessel (e.g., digital artery) occlusion associated with severe vasospasm.

- ***Skin Involvement***
  a) The most overt clinical manifestation of scleroderma, particularly in patients with diffuse disease, is cutaneous fibrosis.
  b) Cutaneous involvement in scleroderma begins with an edematous phase that is associated with an active inflammatory process. This phase persists for several weeks to months and is characterized by nonpitting edema of the affected limbs, erythema of the skin, and intense pruritus.
  c) The edematous phase eventually gives way to a fibrotic stage, which may last months or years. Excessive collagen and other extracellular material in the dermis thickens the skin, making it inflexible.
  d) Patients with diffuse cutaneous scleroderma develop masked facies, small oral apertures, and vertical furrowing of the perioral skin.
  e) Telangiectasias of the skin appear as erythematous spots that blanch on pressure and are a manifestation of abnormal dilated capillaries. Telangiectasias on the face, fingers, palms, and mucous membranes are prominent in the CREST syndrome.

❖ ***Gastrointestinal Involvement***

a) Almost every patient with scleroderma has symptoms or signs of gastrointestinal disease.
b) Approximately 90% of patients have symptoms of esophageal disease. Heartburn, regurgitation, or dysphagia for pills and solids (more than liquids) is caused by the loss of normal smooth muscle function and dysmotility of the lower two thirds of the esophagus.
c) Bloating, abdominal distention, diarrhea, and constipation are common complaints caused by dysmotility of the small and large bowel. Sluggish or atonic bowel function allows bacterial overgrowth to result in serious diarrhea with malabsorption, weakness, and progressive loss of weight.

❖ ***Pulmonary Involvement***

a) The most common symptom of scleroderma lung disease is dyspnea in the absence of chest pain.
b) Lung injury in scleroderma results from one of two processes: (1) fibrosing alveolitis (leading to restrictive lung disease) or (2) obliterative vasculopathy of medium and small pulmonary vessels (associated in some cases with PHTN).
c) Both interstitial fibrosis and pulmonary vascular disease are present to some degree in most patients. However, interstitial lung disease is more characteristic of diffuse scleroderma.

❖ ***Cardiac Involvement***

Cardiac disease is a major problem in scleroderma. There are diverse clinical presentations, including pericardial involvement, conduction abnormalities and arrhythmias, and myocardial disease with heart failure.

❖ ***Renal Involvement***

Mild proteinuria without loss of renal function or evidence of glomerular disease is the most common sign of renal disease in scleroderma.

❖ ***Musculoskeletal Involvement***

The most common symptoms are pain, stiffness, and diffuse muscular discomfort that mimics a flulike syndrome. The pain is more intense around joints, including the fingers, wrists, elbows, shoulders, knees, and ankles, yet inflammatory signs of synovitis are infrequent.

❖ ***Other Symptoms and Signs***

Dry eyes (keratoconjunctivitis sicca) and/or dry mucous membranes (xerostomia) occur in 25% of patients.

Diagnosis

- The early symptoms of scleroderma, unexplained fatigue, arthralgia, myalgia, and the new onset of Raynaud's phenomenon are nonspecific and mimic other rheumatic diseases such as systemic lupus erythematosus, polymyositis, rheumatoid arthritis, and Sjögren's syndrome.
- The diagnosis of Raynaud's phenomenon is based on clinical criteria. The patient must give a history of excessive cold sensitivity and recurrent events of sharply demarcated pallor and/or cyanosis of the skin of the digits.
- Distinguishing primary Raynaud's phenomenon from that associated with an underlying disorder is frequently challenging.
- Primary Raynaud's phenomenon occurs when no disease process is associated with the events. Young age at onset (less than 30 years), symmetrical manifestation of symptoms, mild to moderate severity and no association with tissue gangrene, normal nail-fold capillary examination, and a negative antinuclear antibody (ANA) titer are all indicative of primary Raynaud's phenomenon.
- The presence of intense Raynaud's attacks, especially when accompanied by skin gangrene or ulceration, warrants a thorough diagnostic evaluation for secondary causes.

CONDITIONS ASSOCIATED WITH RAYNAUD'S PHENOMENON
RHEUMATIC DISEASES ▪ Scleroderma ▪ Systemic lupus erythematosus ▪ Dermatomyositis and polymyositis
DRUGS/CHEMICALS ▪ Ergotamines ▪ Nonselective β-blockers
TRAUMA OR INJURY TO DIGITAL HAND VESSELS ▪ Repetitive occupational stress
CONDITIONS CAUSING ARTERIAL DISEASE ▪ Vasculitis ▪ Thromboangiitis obliterans
OTHER ▪ Hypothyroidism ▪ Carpal tunnel syndrome ▪ Cold agglutinins

Laboratory Features

- Anemia is frequent in patients with SSc and may have different etiologies. Most common is a mild normocytic or microcytic anemia due to chronic inflammation. Serum iron is low or normal, whereas ferritin levels are elevated. Iron deficiency anemia may indicate gastrointestinal bleeding caused by watermelon stomach or chronic esophagitis. Macrocytic anemia, indicating a maturation disorder, may be caused by folate and vitamin B12 deficiency due to small bowel bacterial overgrowth and malabsorption.
- Antinuclear autoantibodies can be detected in almost all patients with SSc and are present at disease onset. Some of these autoantibodies, such as those directed against topoisomerase-I (Scl-70) and centromere, are very highly specific for SSc and are mutually exclusive.

Treatment

a) *Skin and periarticular changes*. No therapeutic agent is clearly effective for these cutaneous manifestations, although penicillamine or methotrexate is sometimes used. Physical therapy is important to retard and reduce joint contractures.

b) *Reflux esophagitis* generally responds to standard therapy (e.g., H2-receptor antagonists, proton pump inhibitors, and promotility agents.)

c) *Renal involvement*. Aggressive blood pressure control with ACE inhibitors may delay or prevent the onset of uremia.

d) Raynaud's Phenomenon:

- Calcium channel antagonists (e.g., nifedipine) are the preferred initial agents, although they may exacerbate gastroesophageal reflux and constipation in these patients
- Daily low-dose aspirin therapy is often prescribed for its antiplatelet effects.

Chapter Five

Systemic Vasculitides (Vasculitis Syndromes)

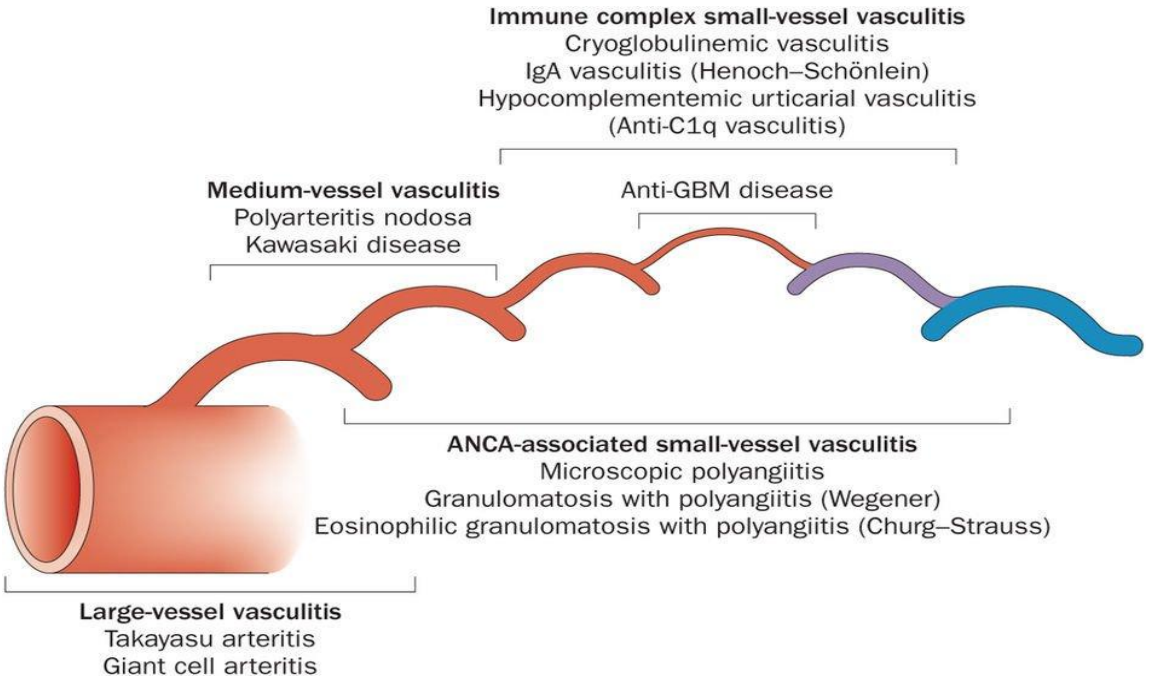
Objectives: by the end of the chapter, the student will:

1) Understand the concept of vasculitis.
2) Understand the principles of classifying various types of vasculitis.
3) Know the clinical presentations of various forms of vasculitis.
4) Understand the principles and tools for diagnosing various forms of vasculitis.
5) Know the treatment options for various forms of vasculitis.

- The Vasculitides are a heterogeneous group of disorders linked by the common finding of destructive inflammation within blood vessel walls.
- The vessel lumen is usually compromised, and this is associated with ischemia of the tissues supplied by the involved vessel.

CLASSIFICATION SCHEME OF VASCULITIDES ACCORDING TO SIZE OF PREDOMINANT BLOOD VESSELS INVOLVED

Predominantly large vessel Vasculitides ▪ Takayasu's arteritis ▪ Behcet's disease ▪ Giant cell arteritis
Predominantly medium vessel Vasculitides ▪ Polyarteritis nodosa ▪ Kawasaki disease ▪ Berger's disease ▪ Rheumatoid vasculitis
Predominantly small vessel Vasculitides ▪ Henoch-Schoenlein purpura ▪ Wegener's granulomatosis ▪ Churg-Strauss syndrome



Role of ANCAs (ANCA = antineutrophil cytoplasmic antibody)

The three types of vasculitis often associated with ANCAs are Wegener's granulomatosis, microscopic polyangiitis, and Churg-Strauss syndrome.

CLINICAL MANIFESTATIONS

(I) TAKAYASU'S ARTERITIS

- Takayasu's arteritis has a predilection for the aorta and its major branches.
- The most commonly involved arteries are the subclavian and innominate arteries. Because of its ability to obliterate peripheral pulses (particularly in the upper extremities), Takayasu's arteritis has been termed “pulseless disease.
- The pulmonary circulation is involved in approximately 50% of cases of Takayasu's arteritis.
- The cornerstone of treatment of Takayasu's arteritis is corticosteroids.

(II) BEHÇET'S DISEASE

- Behcet’s disease may affect small, medium, and large vessels, in either the venous or the arterial circulation.
- The triad of recurrent mouth ulcers, genital ulcers, and eye inflammation is the classic presentation.
- Large-vessel complications of Behcet’s disease may include aneurysms in the pulmonary and systemic arterial systems. Venous complications

include deep venous, vena caval, portohepatic vein, and cerebral sinus thrombosis.

- Colchicine (up to 1.5 to 2 mg/day)is sometimes effective for the treatment of mild oral and genital ulcers in Behcet's disease. Thalidomide (100 mg/day) is also useful for mucocutaneous disease manifestations. Severe disease in any organ system almost always requires high doses of prednisone (e.g., 1 mg/kg/day). Cyclophosphamide (2 mg/kg/day) or chlorambucil (0.2 mg/kg/day, increased as tolerated) is indicated for the most severe forms of uveitis or meningoencephalitis.

(III) POLYARTERITIS NODOSA

- Classic polyarteritis nodosa ai necrotizing inflammation of medium-sized or small arteries that spares the smallest blood vessels (arterioles, venules, and capillaries).
- Polyarteritis nodosa has a striking predilection for certain organs, particularly the skin, peripheral nerves, gastrointestinal tract, and kidneys.
- The classic gastrointestinal manifestation of polyarteritis nodosa is "intestinal angina," the occurrence of postprandial abdominal pain. Polyarteritis nodosa can also affect individual gastrointestinal tract organs, such as the gallbladder or appendix, presenting as cholecystitis or appendicitis.
- The typical renal manifestation of polyarteritis nodosa is vasculitis involvement of the medium-sized intrarenal arteries, leading to renin-mediated hypertension and renal infarctions.
- The diagnosis of polyarteritis nodosa requires either a tissue biopsy or an angiogram that demonstrates microaneurysms.
- Approximately half of patients with polyarteritis nodosa achieve remissions or cures with high doses of corticosteroids alone.

(IV) BUERGER'S DISEASE

- Berger's disease, also known as thromboangiitis obliterans, has a remarkably strong yet poorly understood association with cigarette smoking.
- The vessels affected by Berger's disease are the distal, medium-sized arteries and veins, particularly vessels at the levels of the ankles and wrists.
- Berger's disease tends to be segmental in nature, involving 5- to 10-cm lengths of blood vessels.

- Vascular occlusion in Berger's disease often leads to the loss of digits and, if smoking persists, to loss of larger amounts of tissue (e.g., hands or feet). Despite the intense involvement of the extremities in Berger's disease, internal organ disease almost never occurs.
- Complete abstinence from tobacco is essential to the treatment of Berger's disease.

(V) KAWASAKI'S DISEASE

- Kawasaki's disease occurs exclusively in young children. Because of its striking mucocutaneous findings and lymphadenopathy, Kawasaki's disease is also known as mucocutaneous lymph node syndrome.
- Features of Kawasaki's disease include high fevers, cervical adenopathy, conjunctival congestion, buccal erythema, prominence of the tongue papillae ("strawberry tongue"), polymorphous truncal rash, erythema of the palms and soles, and desquamation of skin from the fingertips occurring days to weeks into the illness.
- The recommended therapeutic regimen in Kawasaki's disease is the combination of intravenous immune globulin (IVIG; 400 mg/kg/day on 4 consecutive days) and acetylsalicylic acid (100 mg/kg/day, lowered to 3 to 5 mg/kg/day after resolution of the fever).

(VI) WEGENER'S GRANULOMATOSIS

- Classic Wegener's granulomatosis involves the upper respiratory tract, the lungs, and the kidneys.
- The three pathologic hallmarks of Wegener's granulomatosis are (1) granulomatous inflammation in the upper or lower respiratory tract, (2) necrotizing vasculitis affecting arteries or veins, and (3) segmental glomerulonephritis, associated with necrosis and thrombosis of capillary loops, with or without granulomatous lesions.
- Approximately 90% of patients with Wegener's granulomatosis have nasal involvement, including crusting, bleeding, and obstruction.
- Renal involvement is the most ominous clinical manifestation of Wegener's granulomatosis. The clinical presentation of renal disease in Wegener's granulomatosis is that of rapidly progressive glomerulonephritis: hematuria, red blood cell casts, and proteinuria (usually non-nephrotic). Without appropriate therapy, end-stage renal disease may ensue within days to weeks.
- Sixty percent of patients with Wegener's granulomatosis have

musculoskeletal symptoms during their disease courses. The presenting complaint is frequently arthralgias or arthritis. Splinter hemorrhages, digital ischemia, and digital gangrene may occur in Wegener's granulomatosis.

- Wegener's granulomatosis is the prototype of conditions associated with ANCA.
- Manifestations of Wegener's granulomatosis that constitute immediate threats either to the function of a vital organ or to the patient's life require urgent treatment with both a cytotoxic agent (usually cyclophosphamide, 2 mg/kg orally daily) and high doses of corticosteroids (1 mg/kg orally daily, tapered during 6 to 12 months).

(VII) HENOCH-SCHÖNLEIN PURPURA

- Henoch-Schoenlein purpura (HSP) is characterized by non-thrombocytopenic purpura, arthritis, abdominal pain, and glomerulonephritis.
- HSP can develop at any age, but 80 to 90% of the cases occur in children.
- The classic HSP patient presents with the acute onset of fever, palpable purpura on the lower extremities and buttocks, abdominal pain, arthritis, and hematuria.
- Most patients with HSP, especially children, have a self-limited disease that lasts an average of 4 weeks. Corticosteroids ameliorate the joint and gastrointestinal symptoms in most cases and improve the cutaneous manifestations in some.

(VIII) CHURG-STRAUSS SYNDROME

- Churg-Strauss syndrome is an eosinophil-rich form of granulomatous inflammation that involves the respiratory tract and other organs.
- Two hallmarks of Churg-Strauss syndrome are asthma and eosinophilia.
- Patients with mild disease may be treated with prednisone. Those with evidence of neurologic, cardiac, renal, or gastrointestinal involvement should be treated with cyclophosphamide in addition to corticosteroids.

MAJOR DISEASE CATEGORIES IN THE DIFFERENTIAL DIAGNOSIS OF VASCULITIDES

Infections
Bacterial, viral, mycobacterial
Occlusive processes
Hypercoagulable states Atheroembolic disease
Connective tissue disorders
Systemic lupus erythematosus, mixed connective tissue disease Systemic sclerosis Rheumatoid arthritis

Ideally, the diagnosis of vasculitis is established through biopsy of an involved organ.

Chapter Six

Sjogren's Syndrome

Objectives: by the end of this chapter, the student will.

1) Understand the definition of Sjogren's syndrome.
2) Know the clinical presentations of various forms of Sjogren's Syndrome.
3) Know the diagnostic tools for Sjogren's Syndrome.
4) Know the treatment options for Sjogren's Syndrome.

- Sjogren's syndrome (SS) is an autoimmune disease characterized by immune-mediated destruction of exocrine glands, particularly salivary and lacrimal glands, with secondary development of keratoconjunctivitis and xerostomia.
- *Primary and secondary forms of SS have been defined*.
- Primary SS occurs in the absence of a connective tissue disease; secondary SS occurs most commonly in association with a systemic connective tissue disorder, particularly rheumatoid arthritis but also systemic sclerosis, systemic lupus erythematosus, and polymyositis.

Clinical Manifestations

- Presentation is most common in women in their 30s to 50s. Dry mouth leads to difficulty in swallowing, recurrent dental infections, pain on eating salty or spicy foods, and difficulty in talking
- In many patients, impairment of fluid secretion within the nasal and throat passage predisposes to oral thrush.
- Other upper and lower respiratory symptoms also are secondary to dryness and include a nonproductive cough and occasionally tracheobronchitis.

SYMPTOMS OF SJÖGREN'S SYNDROME SECONDARY TO EXOCRINE GLAND DYSFUNCTION

EYE (XEROPHTHALMIA) ▪ Burning ▪ Blurring ▪ Foreign body sensation ▪ Photosensitivity
ORAL (XEROSTOMIA) ▪ Difficulty chewing ▪ Increased dental caries ▪ Sialadenitis ▪ Nocturnal awakening to drink water
NOSE, THROAT ▪ Difficulty swallowing (especially dry foods) Epistaxis, dry nares Hoarseness
CARDIOPULMONARY ▪ Tracheobronchitis, including a dry cough
SKIN ▪ Xerosis

Extra glandular manifestations of primary Sjogren's syndrome

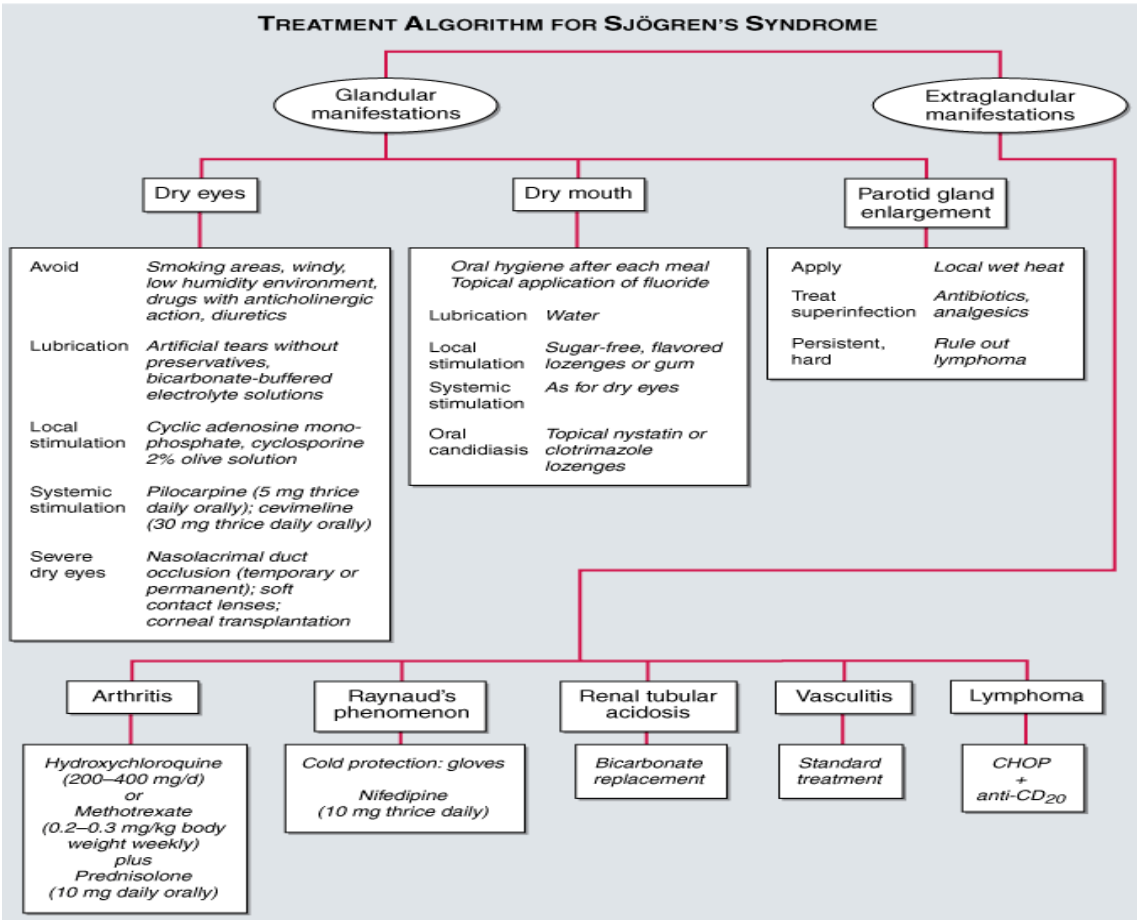
CONSTITUTIONAL SYMPTOMS ▪ Fatigue ▪ Low-grade fever	NEUROLOGIC ▪ Peripheral motor sensory neuropathy. ▪ Multiple sclerosis–like focal lesions. ▪ Transient ischemic attacks, including cerebrovascular accident.
SKIN AND VASCULAR ▪ Small vessel vasculitis ▪ Raynaud's phenomenon ▪ Photosensitivity reactions similar to subacute cutaneous systemic lupus erythematosus	GASTROINTESTINAL Hepatomegaly Primary biliary cirrhosis
UPPER AND LOWER AIRWAYS ▪ Pyogenic sialadenitis or parotitis Interstitial pneumonitis or fibrosis Chronic bronchitis Bronchiectasis	ENDOCRINE Hashimoto's thyroiditis with possible hypothyroidism
MUSCULOSKELETAL ▪ Polyarthralgia, polyarthritis Myopathy, polymyositis	NEOPLASIA Lymphadenopathy, lymphoma

DIFFERENTIAL DIAGNOSIS OF DRY EYES AND MOUTH

(SICCA SYNDROME)

Xerostomia	Dry Eye	Bilateral Parotid Gland Enlargement
• Viral infections • Drugs *Psychotherapeutic* *Parasympatholytic* • Irradiation • Diabetes mellitus • Sjögren's syndrome	• Inflammation *Stevens-Johnson syndrome* *Chronic conjunctivitis* *Chronic blepharitis* • Sjögren's syndrome • Neurologic conditions *Impaired eyelid function* *Parkinson's disease* • Miscellaneous *Hypovitaminosis A* *Lid scarring*	• Viral infections *Mumps* *Epstein-Barr* *HIV* • Sarcoidosis • Sjögren's syndrome • Diabetes mellitus • Hepatic cirrhosis • Hyperlipoproteinemias • Chronic pancreatitis

TREATMENT



Chapter Seven

Polymyositis And Dermatomyositis

Polymyositis And Dermatomyositis

Objectives: by the end of this chapter, the student will.

1) Understand the definition and the difference between polymyositis and dermatomyositis.
2) Know the clinical features of both diseases.
3) Know the diagnostic tests for both diseases.
4) Know the treatment options for both diseases.

Definition

- Polymyositis and dermatomyositis are the most common forms of a family of acquired, systemic, connective tissue diseases, known as the idiopathic inflammatory myopathies, characterized by the clinical and pathologic effects of chronic muscle inflammation of unknown cause.

CRITERIA FOR THE DIAGNOSIS OF POLYMYOSITIS AND DERMATOMYOSITIS

❖ **Symmetric, often progressive, proximal muscle weakness**
❖ **Characteristic electromyographic findings**
❖ **Elevations of serum activities of myositis-associated enzymes** 1) Creatine kinase 2) Aldolase 3) Lactate dehydrogenase
❖ **Evidence of chronic inflammation in muscle biopsy specimen**
❖ **Characteristic rashes of dermatomyositis** 1) Scaly erythematous palpable eruptions over the metacarpophalangeal or interphalangeal joints 2) Periorbital purplish discoloration (heliotrope rash)

Clinical Manifestations

Polymyositis and dermatomyositis can occur in three forms: (a) alone, (b) in association with any of the other autoimmune diseases, or (c) with a variety of neoplasms.

a) Symmetric, proximal muscle weakness is present in most cases and results in difficulty arising from chairs, getting out of cars, reaching overhead, or combing hair.

b) Many patients also report generalized fatigue or muscle pain.

c) Patients with dermatomyositis can present with a variety of photosensitive rashes over the face, chest, and hands.

d) Patients with dermatomyositis can present with a variety of photosensitive rashes over the face, chest, and hands. Gottron's papules are raised, often scaly, palpable lesions overlying an erythematous base on extensor surfaces such as the metacarpophalangeal and proximal interphalangeal joints, elbows, and knees; these are virtually pathognomonic for dermatomyositis.

e) On occasion, patients may develop classic dermatomyositis rashes without apparent muscle weakness or elevated muscle enzymes in a syndrome called dermatomyositis sine myositis. The idiopathic inflammatory myopathies are systemic diseases with frequent extra muscular manifestations that can become the most troubling aspects of the patient's disease.

EXTRAMUSCULAR MANIFESTATIONS OF POLYMYOSITIS AND DERMATOMYOSITIS

General ▪ Fatigue ▪ Weight loss ▪ Raynaud's syndrome	**Gastrointestinal** ▪ Esophageal dysphagia with occasional nasal regurgitation or aspiration ▪ Delayed gastric emptying and reflux ▪ Vasculitis with infarcts and necrosis of the bowel
Musculoskeletal ▪ Arthralgias, usually symmetrical and involving the hand joints ▪ Nonerosive polyarthritis, usually symmetrical and involving the hand joints	**Pulmonary** ▪ Ventilatory insufficiency due to respiratory muscle weakness ▪ Interstitial lung disease (pulmonary fibrosis) ▪ Pulmonary hypertension
Cutaneous ▪ Characteristic dermatomyositis rashes ▪ Photosensitivity ▪ Vasculitis with infarcts and digital ulcers ▪ Subcutaneous inflammation (panniculitis)	**Cardiac** ▪ Myocarditis with arrhythmias and congestive failure ▪ Cardiomyopathy

Diagnosis

- Muscle pain and weakness are frequent patient complaints arising from many causes. These can be the result of intrinsic muscle diseases (myopathies), disorders of the nerves innervating the muscle (neuropathies), or the effects of drugs, toxins, or infectious agents.

DIFFERENTIAL DIAGNOSIS OF IDIOPATHIC INFLAMMATORY MYOPATHY

Drug- and toxin-induced myopathies ▪ Corticosteroids ▪ Ethanol	**Other immunologic or connective tissue diseases** ▪ Polyarteritis nodosa and other forms of vasculitis ▪ Rheumatoid arthritis ▪ Systemic lupus erythematosus
Endocrine diseases ▪ Hypothyroidism ▪ Diabetes mellitus	**Acquired metabolic abnormalities** ▪ Hypokalemia ▪ Hypercalcemia
Neurologic disorders ▪ Amyotrophic lateral sclerosis ▪ Myasthenia gravis ▪ Guillain-Barré syndrome	**Infectious myopathies** ▪ Parasitic (trichinosis, toxoplasmosis, sarcosporidiosis, trypanosomiasis) ▪ Viral (influenza, adenovirus, coxsackievirus

Laboratory Studies

Elevated muscle enzyme levels (creatine kinase, aldolase, AST). Certain subsets of disease are associated with myositis-specific antibodies such as Jo-1

Treatment

a) Prednisone When polymyositis and dermatomyositis occur without associated disease, it usually responds well to prednisone, 1-2 mg/kg PO daily.

b) IV infusion of immunoglobulin may hasten improvement of severe dysphagia.

c) Patients who do not respond or cannot tolerate the side effects of glucocorticoids may respond to methotrexate or azathioprine.

Chapter Eight

Gout And Hyperuricemia

Objectives: by the end of the chapter the student will.

1) Understand the concept of crystal deposition arthritis.
2) Understand the causes of hyperuricemia.
3) Know the clinical features of gout.
4) Understand the diagnostic tools for gout.
5) Know the treatment options for gout.

Definition

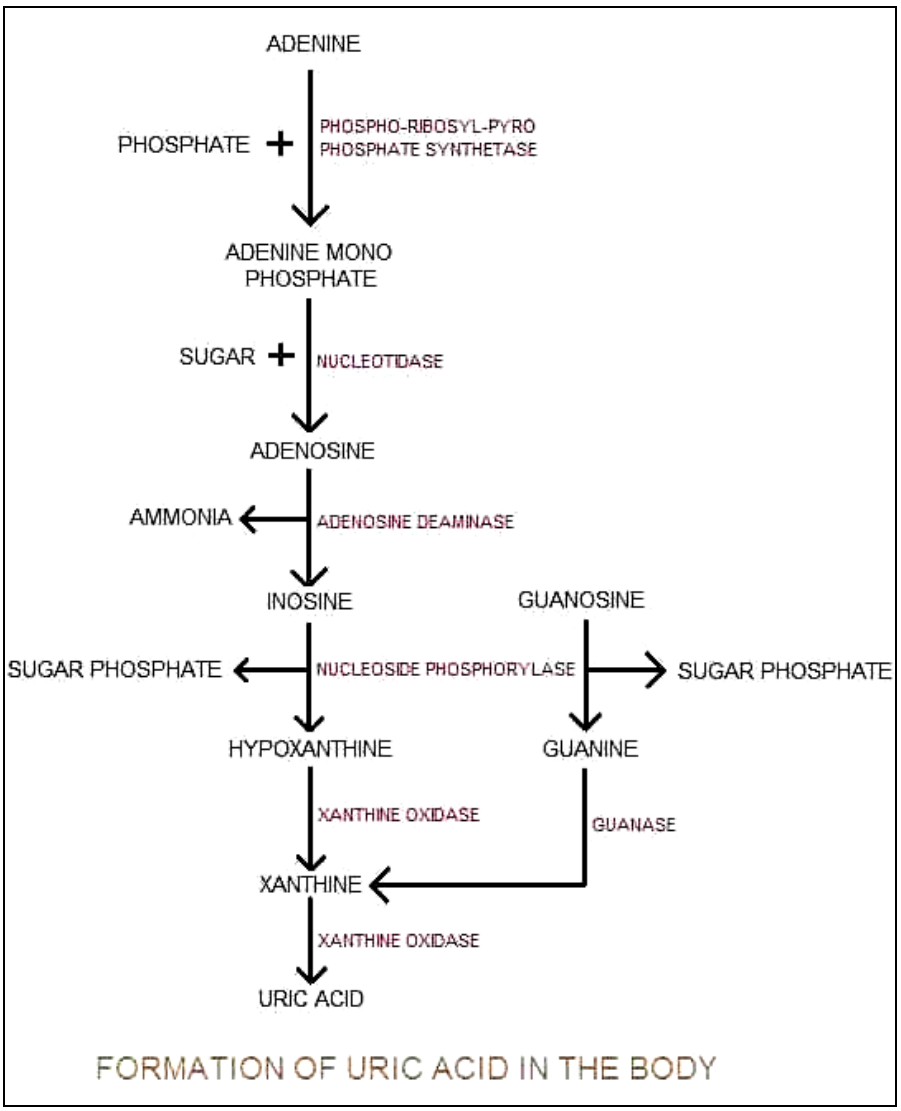
- The term gout refers to heterogeneous disorders resulting from tissue deposition of monosodium urate crystals or crystallization of uric acid in the urinary tract.
- Fundamental to the development of gout is a substantial increase in total body uric acid stores, as reflected in the metabolic disorder hyperuricemia.
- Typically, hyperuricemia is defined as a serum uric acid level greater than 7.0 mg/dL in adult men and 6.0 mg/dL in premenopausal women.

Epidemiology

- Gout is predominantly an idiopathic or multifactorial disease of adult men, with a peak incidence in the fifth decade.
- Gout rarely occurs in men before adolescence or in women before menopause.

Pathobiology

a) Uric acid is normally produced as an end product of the degradation of purine compounds.



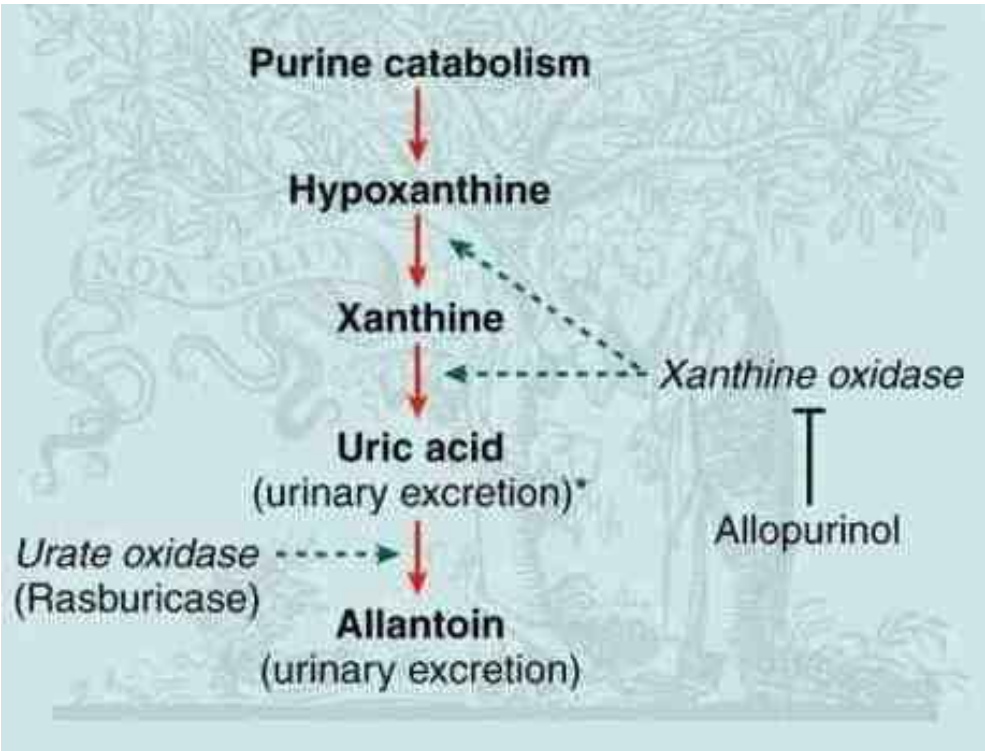
FORMATION OF URIC ACID IN THE BODY

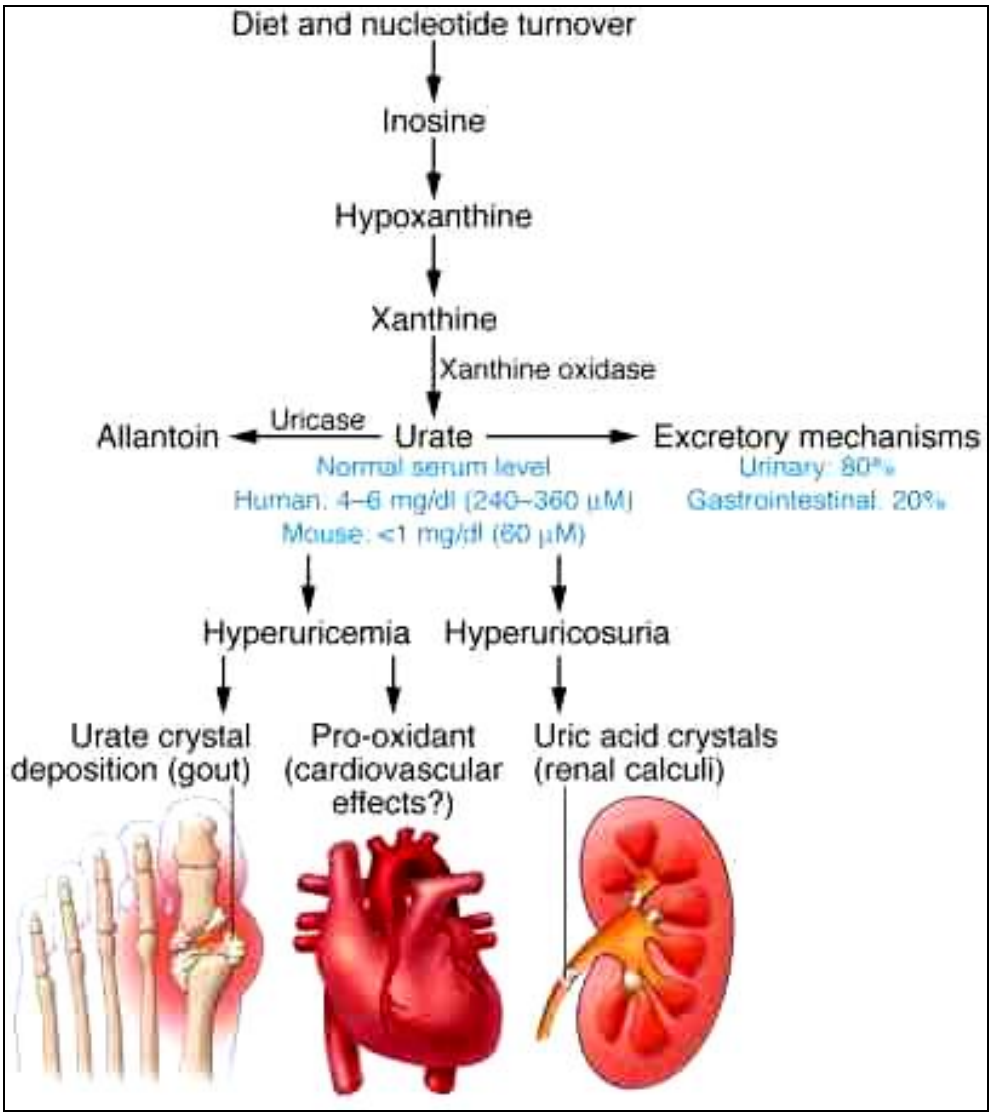
b) However, the solubility of the principal physiologic salt of uric acid, monosodium urate, in connective tissues is normally close to 7 mg/dL at 37° C, and urate solubility declines progressively at cooler temperatures such as those in distal peripheral joints.

c) The predilection for marked urate crystal deposition in the first metatarsophalangeal (MTP) joint may also relate to repetitive minor trauma at that site.

d) Abrupt rises and declines in serum urate levels, as stimulated by diuretic and alcohol use and initiation of therapy with antihyperuricemic drugs, may promote the release of urate crystals from tophi via changes in packing of crystals in tophaceous deposits.

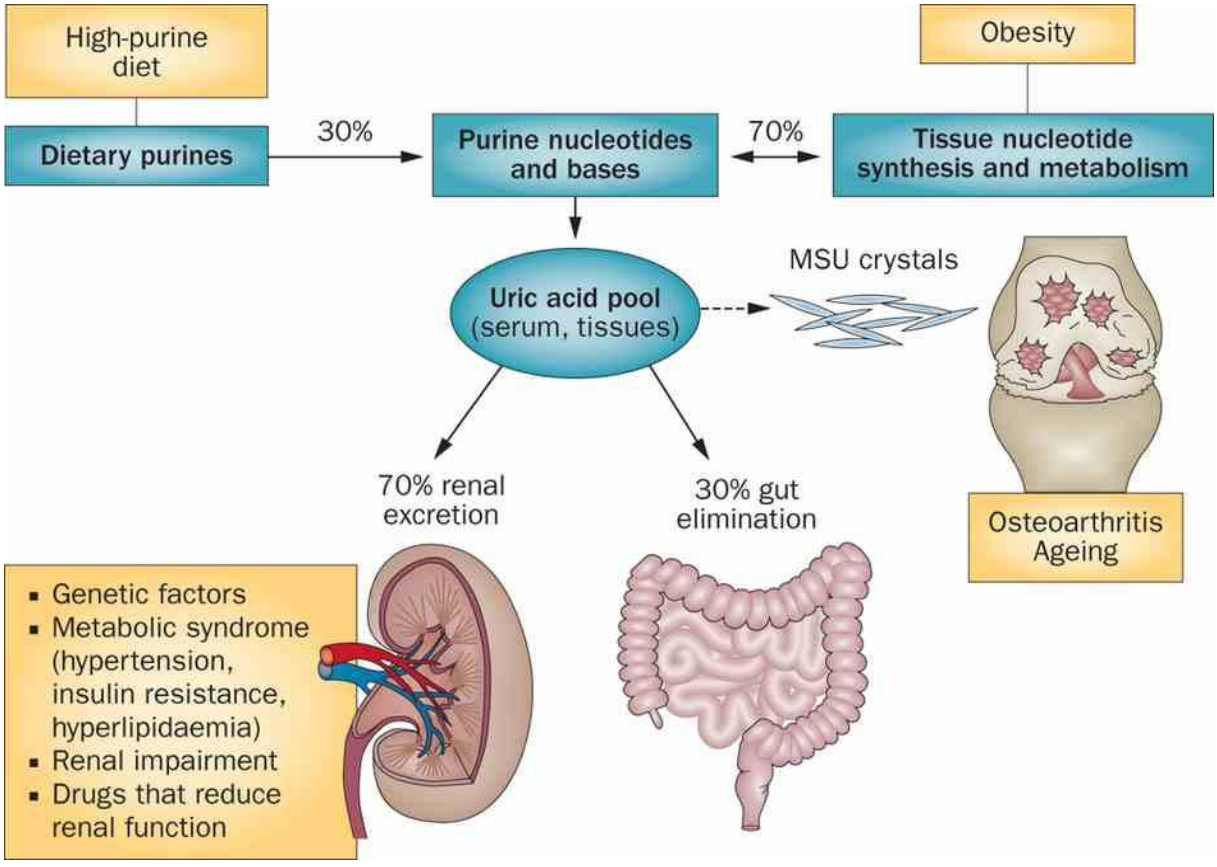
e) Free urate crystals have considerable pro-inflammatory potential via the ability to activate synovial lining cells and leukocytes, induce activation of the membrane attack complex of complement, and trigger certain other inflammatory cascades.


Purine catabolism
Hypoxanthine
Xanthine
Xanthine oxidase
Uric acid
(urinary excretion)*
Allopurinol
Urate oxidase
(Rasburicase)
Allantoin
(urinary excretion)


Diet and nucleotide turnover
Inosine
Hypoxanthine
Xanthine
Xanthine oxidase
Allantoin
Uricase
Urate
Excretory mechanisms
Normal serum level
Human: 4–6 mg/dl (240–360 μM)
Mouse: <1 mg/dl (60 μM)
Urinary: 80%
Gastrointestinal: 20%
Hyperuricemia
Hyperuricosuria
Urate crystal deposition (gout)
Pro-oxidant (cardiovascular effects?)
Uric acid crystals (renal calculi)

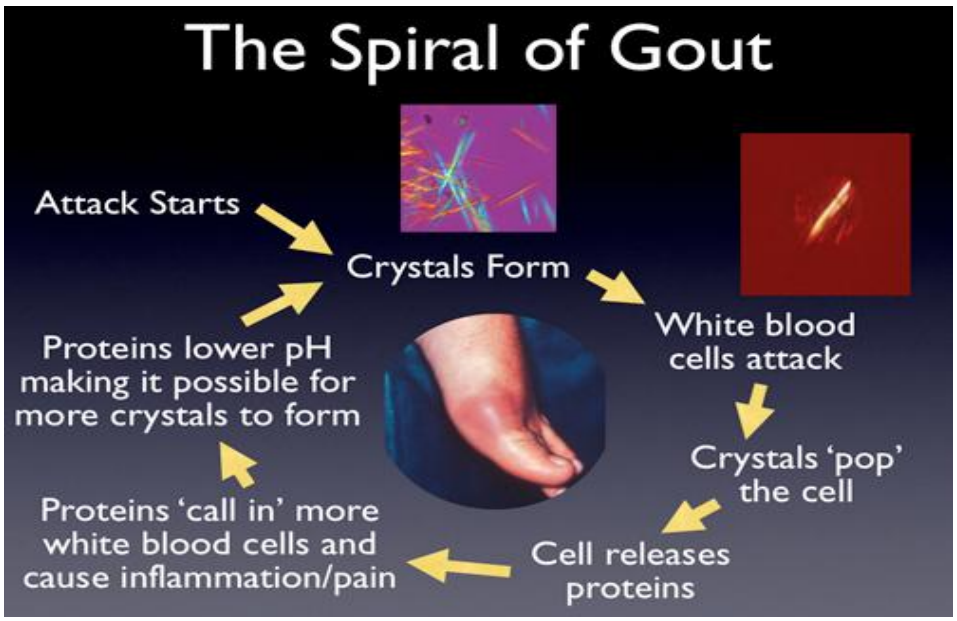
HYPERURICEMIA: CAUSES AND CLASSIFICATION

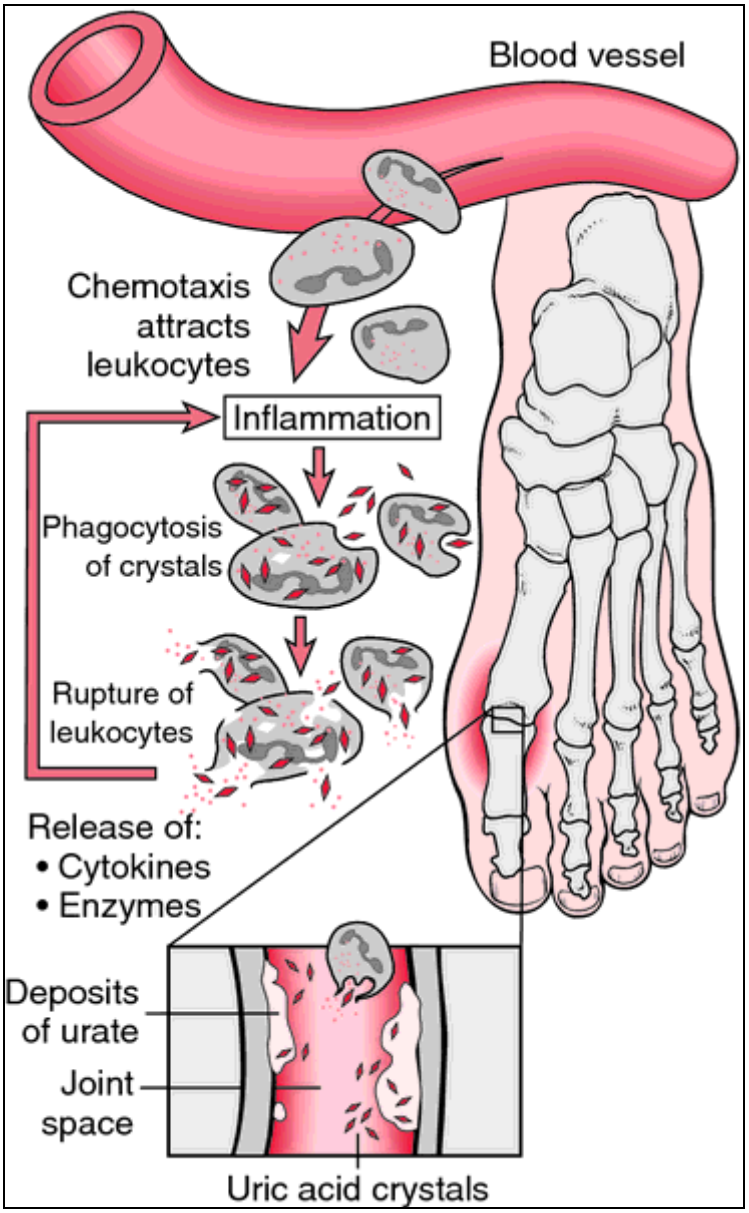
Uric acid overproduction
Primary hyperuricemia ❖ Idiopathic *Secondary hyperuricemia* ❖ Excessive dietary purine intake ❖ Increased nucleotide turnover (e.g., myeloproliferative and lymphoproliferative disorders, hemolytic diseases
Uric acid underexcretion
Primary hyperuricemia ❖ Idiopathic *Secondary hyperuricemia* ❖ Diminished glomerular filtration rate ❖ Inhibition of tubular urate secretion



Clinical Manifestations

- Gout is classically manifested as recurrent attacks of *acute arthritis* characterized by often excruciatingly painful articular and periarticular inflammation, including erythema and edema of the skin that can mimic bacterial cellulitis.
- *Acute gouty arthritis* is typically monoarticular or oligoarticular.


The Spiral of Gout
Attack Starts
Crystals Form
White blood cells attack
Crystals 'pop' the cell
Cell releases proteins
Proteins 'call in' more white blood cells and cause inflammation/pain
Proteins lower pH making it possible for more crystals to form


Blood vessel
Chemotaxis attracts leukocytes
Inflammation
Phagocytosis of crystals
Rupture of leukocytes
Release of:
• Cytokines
• Enzymes
Deposits of urate
Joint space
Uric acid crystals

- *Acute polyarticular gout* can also occur, particularly in the elderly and in transplant patients taking cyclosporine.
- *Chronic gouty inflammation* and proliferative, erosive arthritis in gout can also mimic rheumatoid arthritis.
- *Gouty tophi* can involve not only the synovium and cartilage of joints but also subchondral bone and soft tissues, including the olecranon bursa, the first MTP joint bursa, and the helix of the ear.
- *Uric acid urolithiasis* is a common manifestation of gout, particularly in acid urine. Excessive excretion of uric acid in the urinary tract also promotes calcium oxalate urolithiasis.

Diagnosis

1) A definitive diagnosis of gout or pseudogout is made by finding intracellular crystals in joint fluid examined with a compensated polarized light microscope. Urate crystals, which are diagnostic of gout, are needle shaped and strongly negatively birefringent.
2) The serum uric acid level may fluctuate and is often normal at the time of an acute gout attack.
3) 24-hour uric acid excretion and the use of radiographs to detect classic tophaceous changes of articular gout.
4) Measurement of 24-hour urinary uric acid in subjects with intact renal function and no clear cause for hyperuricemia is valuable to identify subjects with uric acid overproduction.

Treatment

Acute gout

a) NSAIDs are the treatment of choice due to ease of administration and low toxicity. Clinical response may require12-24 hours, and initial doses should be high, followed by rapid tapering over 2-8 days.

b) Glucocorticoids are useful when NSAIDs are contraindicated. An intra-articular injection of glucocorticoids produces rapid dramatic relief.

c) Colchicine is most effective if given in the first 12-24 hours of an acute attack and usually brings relief in 6-12 hours. In view of the efficacy and tolerability of a short course of NSAIDs, colchicine is not commonly used to treat gout but is useful when NSAIDs or glucocorticoids are contraindicated or not tolerated.

Chronic gouty arthritis

1) Allopurinol, a xanthine oxidase inhibitor, is effective therapy for hyperuricemia in most patients.
2) The drug allopurinol, which is used to treat gout, is oxidized by xanthine oxidase to oxypurinol, which is a potent inhibitor of xanthine oxidase. When xanthine oxidase is inhibited, hypoxanthine and xanthine accumulate, and the concentration of uric acid is reduced. Hypoxanthine and xanthine are more water-soluble than uric acid، thereby facilitating the urinary excretion of purine degradation products and reducing the likelihood of urate crystal deposition.
3) Uricosuric drugs lower serum uric acid levels by blocking renal tubular reabsorption of uric acid. A 24-hour measurement of creatinine clearance and urine uric acid should be obtained before therapy is started, as these drugs are ineffective with glomerular filtration rates of <50 mL/min

Chapter Nine

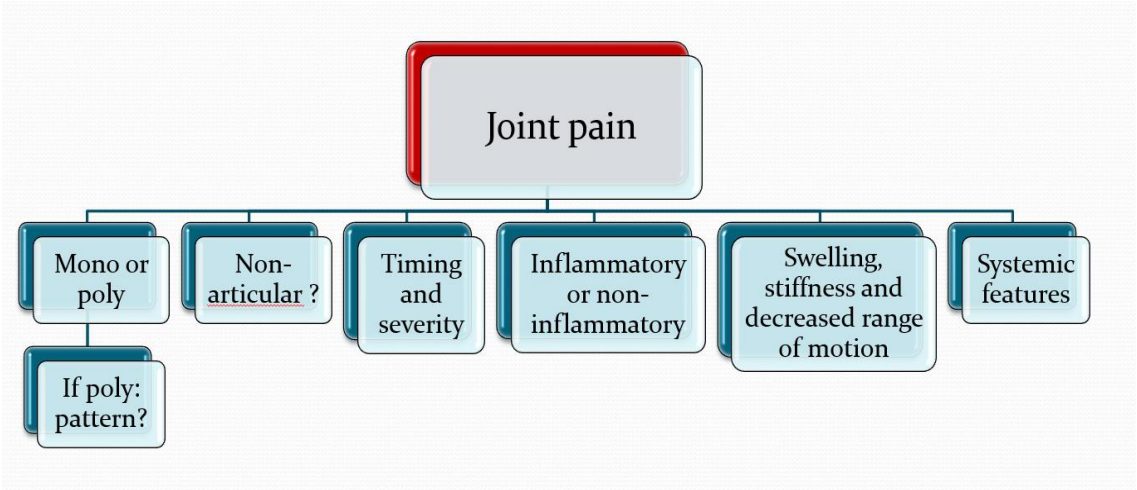
Clinical Examination of The Musculoskeletal System

THE HEALTH HISTORY

Common or Concerning Symptoms

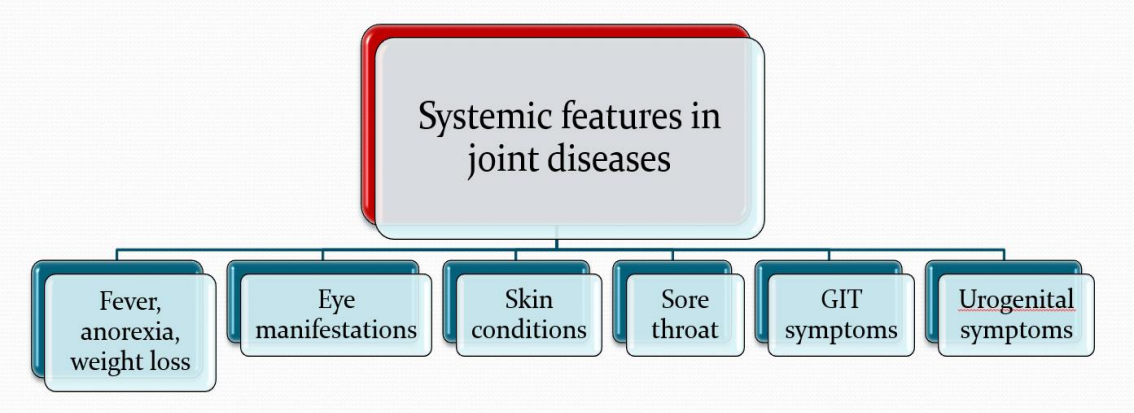
- Low back pain
- Neck pain
- Monoarticular or polyarticular joint pain
- Inflammatory or infectious joint pain
- Joint pain with systemic features such as fever, chills, rash, anorexia, weight loss, weakness
- Joint pain with symptoms from other organ systems

- Joint pain is a common complaint of patients seeking health care.
- Backache is the most common and widespread disorder of the musculoskeletal system, establish whether the pain is on midline, in the area of the vertebrae, or off the midline. If the pain radiates into the legs, ask about any associated numbness, tingling, or weakness.
- Neck pain is also common, especially after trauma. Approach it in the same manner. For both neck and back pain, be especially alert for symptoms such as weakness, loss of sensation, or loss of bladder or bowel function.



Assessing musculoskeletal pain

Site
Onset
Character
Radiation
Associated features
Exacerbating factors
Timing (frequency, duration, periodicity)
Severity



Patterns of presentation of arthritis

Monoarthritis	Oligoarthritis (≤5 joints)	Polyarthritis (>5 joints involved)	
Septic arthritis	Crystal arthritis	*Symmetrical:*	*Asymmetrical:*
Crystal arthritis (gout, CPPD)	Psoriatic arthritis	Rheumatoid arthritis	Reactive arthritis
Osteoarthritis	Reactive arthritis, eg *Yersinia, Salmonella, Campylobacter*	Osteoarthritis	Psoriatic arthritis
Trauma, eg haemarthrosis	Ankylosing spondylitis	Viruses (eg hepatitis A, B & C; mumps)	
	Osteoarthritis	Systemic conditions[1] (can be either)	

- Note that joint pain may also be nonarticular, involving bones, muscles, and tissues around the joint such as the tendons, bursae, or even overlying skin. Generalized "aches and pains" are called myalgias if in muscles and arthralgias if there is pain but no evidence of arthritis.

Causes of muscle pain (myalgia)
Infective • Viral Coxsackie, CMV, ECHO • Bacterial Streptococcus pneumoniae, Mycoplasma • Parasitic Schistosomiasis, toxoplasmosis **Inflammatory** Polymyalgia rheumatica, myositis, dermatomyositis, temporal (giant cell) arteritis
Traumatic: Tears, hematoma, rhabdomyolysis
Drugs e.g. alcohol, statins, zidovudine
Neuropathic

- Try to determine if the problem is inflammatory or noninflammatory. Is there tenderness, warmth, or redness? These features are best assessed on examination, but patients can sometimes guide you to points of tenderness. Ask about systemic symptoms such as fever or chills.
- To assess limitations of motion, ask about changes in level of activity because of problems with the involved joint. When relevant, inquire specifically about the patient's ability to walk, stand, lean over, sit, sit up, rise from a sitting position, climb, pinch, grasp, turn a page, open a door handle or jar, and care for bodily needs such as combing hair, brushing teeth, eating, dressing, and bathing.

Arthritis and other manifestations out with the musculoskeletal system	
Felty's syndrome	Rheumatoid arthritis with splenomegaly, lymphadenopathy, and neutropenia
Sjögren's syndrome	Arthritis with 'dry eyes' (keratoconjunctivitis sicca), xerostomia (reduced or absent saliva production), salivary gland enlargement and Raynaud's phenomenon
Enteropathic arthritis	Associated with inflammatory bowel disease - ulcerative colitis and Crohn's disease
Psoriatic arthritis	With skin and nail features of psoriasis
Haemophilia	Associated with (especially knee) arthropathy because of recurrent haemarthroses

Sickle cell disease	Associated with osteonecrosis of the hip due to bone infarction
Still's disease	Juvenile idiopathic arthritis
Reiter's syndrome	Urethritis, conjunctivitis, and inflammatory oligoarthropathy about 1-3 weeks after sexually transmitted chlamydial infection or infective gastroenteritis

Association of arthropathy with extra-articular features

Type of arthropathy	Symmetry	Condition	Extra-articular features
Monoarthropathy		Septic arthritis Gout	Fever, malaise, source of sepsis, e.g. skin, throat, gut. Tophi, signs of renal failure.
Oligoarthropathy	Asymmetrical	Reiter's syndrome Ankylosing Spondylitis Psoriatic arthritis	Urethritis, mouth and/or genital ulcers, conjunctivitis, iritis, enthesopathy, e.g. Achilles tendinopathy/plantar fasciitis, skin rash (keratoderma blenorrhagica) Enthesopathy, iritis. Psoriasis, nail pitting
Polyarthropathy	Symmetrical	Rheumatoid arthritis	Raynaud's phenomenon, subcutaneous rheumatoid nodules, episcleritis, dry eyes, pleurisy
		Systemic lupus	Raynaud's phenomenon, photosensitive rash especially on face, alopecia, fever, episcleritis

Drug history

Many drugs have side-effects that may either worsen or precipitate musculoskeletal conditions

Drugs associated with adverse musculoskeletal effects

Drug	Possible adverse musculoskeletal effects
Corticosteroids	Osteoporosis, myopathy, avascular necrosis, infections
Statins	Myalgia, myositis, myopathy
Angiotensin-converting enzyme inhibitors	Myalgia, arthralgia, positive antinuclear antibody
Antiepileptics	Osteomalacia, arthralgia
Immunosuppresant/cytotoxic	Infections
Quinolones	Tendonopathy, tendon rupture

TECHNIQUES OF EXAMINATION

Important Areas of Examination for Each of the Major Joints

- Inspection for joint symmetry, alignment, bony deformities
- Inspection and palpation of surrounding tissues for skin changes, nodules, muscle atrophy, crepitus
- Range of motion and maneuvers to test joint function and stability, integrity of ligaments, tendons, bursae, especially if pain or trauma
- Assessment of inflammation or arthritis, especially swelling, warmth, tenderness, redness

As you examine the musculoskeletal system, direct your attention to function as well as structure.

Your examination should be systematic. It should include inspection, palpation of bony landmarks as well as related joint and soft-tissue structures, assessment of range of motion, and special maneuvers to test specific movements.

Remember the following clues to guide your examination.

- During inspection, it is especially important to note symmetry of involvement. Is there a symmetric change in joints on both sides of the body, or is the change only in one or two joints?
- Use inspection and palpation to assess the surrounding tissues, noting skin changes, subcutaneous nodules, and muscle atrophy. Note any crepitus, an audible and/or palpable crunching during movement of tendons or ligaments over bone.
- Testing range of motion and maneuvers (described for each joint) may demonstrate limitations in range of motion or increased mobility and joint instability from excess mobility of joint ligaments, called ligamentous laxity.
- Finally, testing muscle strength may aid in the assessment of joint function (check this particular point in examination of the motor system in neurology).

Be especially alert to signs of inflammation and arthritis;

- Swelling. Palpable swelling may involve: (1) the synovial membrane, which can feel boggy or doughy; (2) effusion from excess synovial fluid within the joint space; or (3) soft-tissue structures such as bursae, tendons, and tendon sheaths.
- Warmth. Use the backs of your fingers to compare the involved joint with its unaffected contralateral joint, or with nearby tissues if both joints are involved.

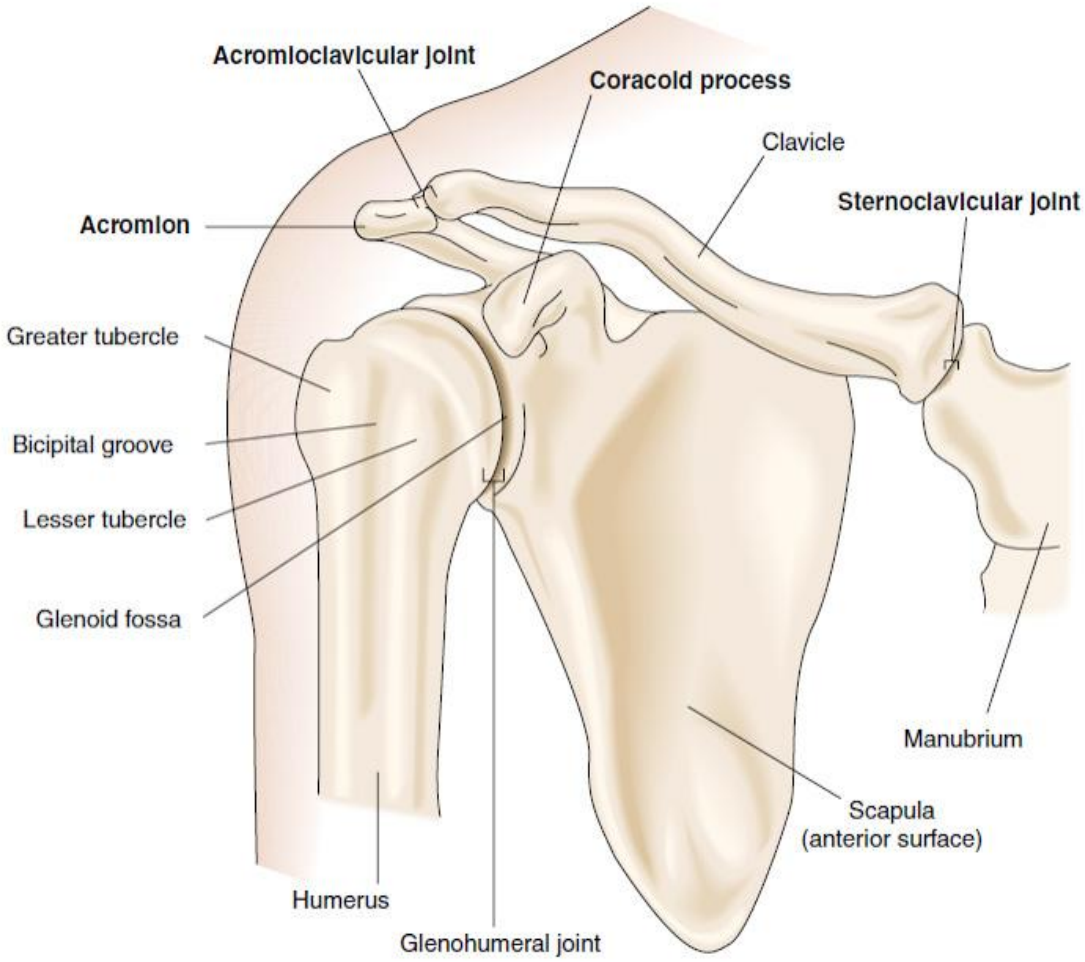
- Tenderness. Try to identify the specific anatomic structure that is tender. Trauma may also cause tenderness.
- Redness. Redness of the overlying skin is the least common sign of inflammation near the joints.

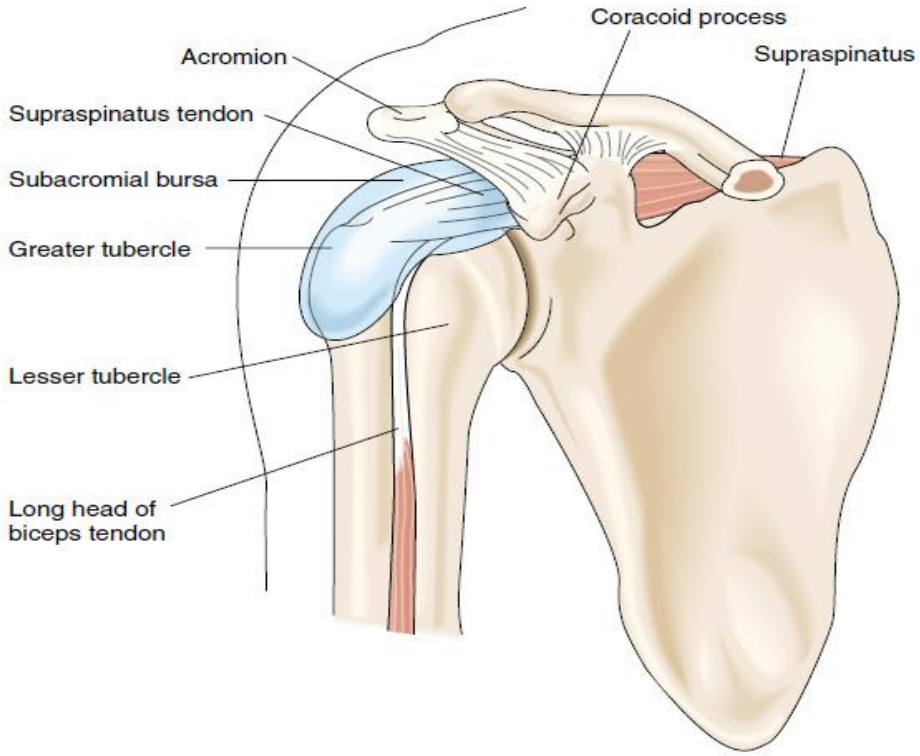
The Shoulder Joint

- The shoulder is distinguished by wide-ranging movement in all directions.
- The shoulder derives its mobility from a complex interconnected structure of four joints, three large bones, and three principal muscle groups, often referred to as the shoulder girdle.

Bony Structures.

- The bony structures of the shoulder include the humerus, the clavicle, and the scapula. The scapula is anchored to the axial skeleton only by the sternoclavicular joint and inserting muscles.





Joints.

Three different joints articulate at the shoulder:

- *The glenohumeral joint*. In this joint, the head of the humerus articulates with the shallow glenoid fossa of the scapula. This joint is deeply situated and not normally palpable. It is a ball-and-socket joint, allowing the arm its wide arc of movement—flexion, extension, abduction (movement away from the trunk), adduction (movement toward the trunk), rotation, and circumduction.
- *The sternoclavicular joint*. The convex medial end of the clavicle articulates with the concave hollow in the upper sternum.
- *The acromioclavicular joint*. The lateral end of the clavicle articulates with the acromion process of the scapula.

Muscle Groups.

Three groups of muscles attach at the shoulder:

1) **The Scapulohumeral Group.**

   This group extends from the scapula to the humerus and includes the muscles inserting directly on the humerus, known as "SITS muscles" of the rotator cuff:

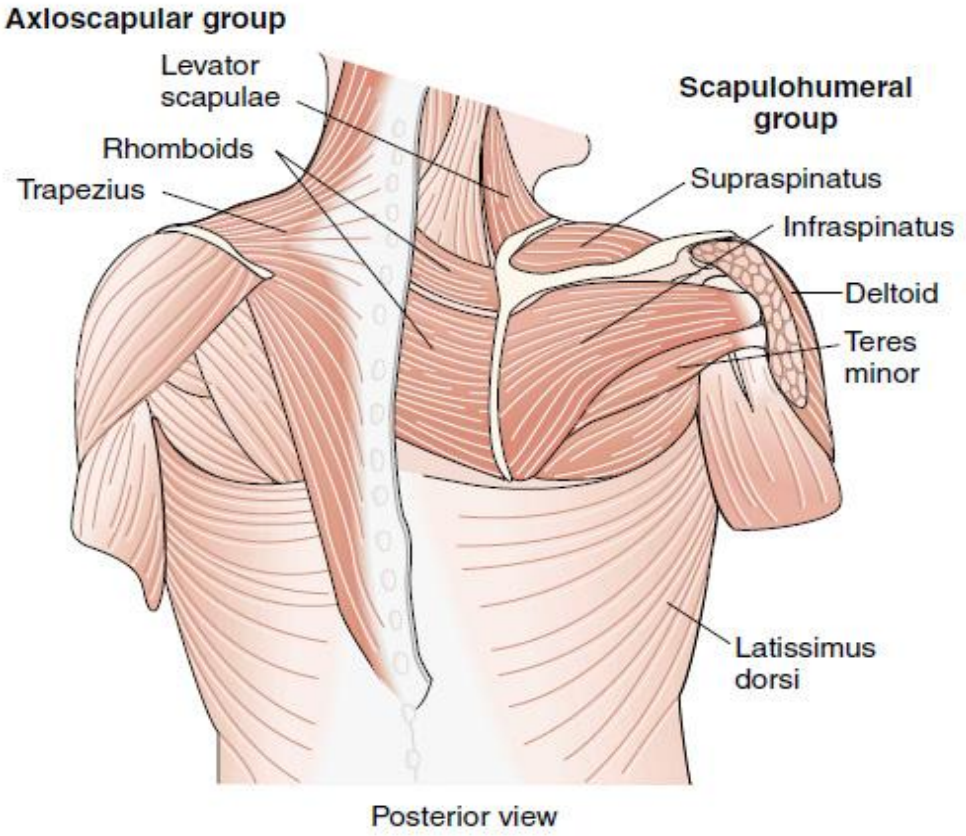
   Supraspinatus—runs above the glenohumeral joint; inserts on the greater tubercle

   Infraspinatus and teres minor— cross the glenohumeral joint posteriorly;

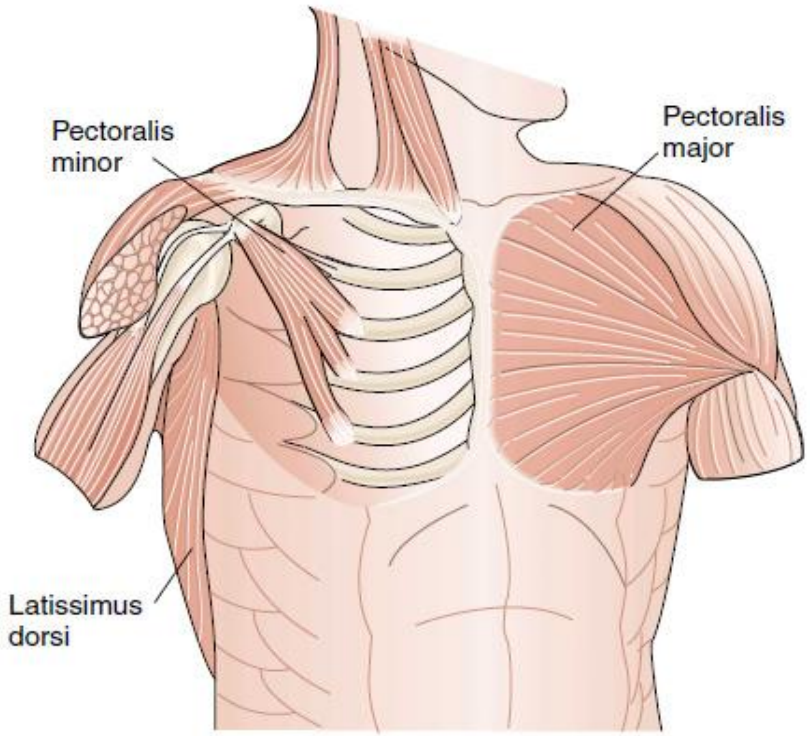
insert on the greater tubercle

Subscapularis (not illustrated)— originates on the anterior surface of the scapula and crosses the joint anteriorly; inserts on the lesser tubercle.

The scapulohumeral group rotates the shoulder laterally (the rotator cuff) and depresses and rotates the head of the humerus



Axioscapular group (pulls shoulder backward)
Scapulohumeral group (rotates shoulder laterally; includes rotator cuff)



Anterior view

Axiohumeral group (rotates shoulder internally)

2) **The Axioscapular Group.**

This group attaches the trunk to the scapula and includes the trapezius, rhomboids, serratus anterior, and levator scapulae. These muscles rotate the scapula

3) **The Axiohumeral Group.**

This group attaches the trunk to the humerus and includes the pectoralis major and minor and the latissimus dorsi. These muscles produce internal rotation of the shoulder.

The biceps and triceps, which connect the scapula to the bones of the forearm, are also involved in shoulder movement, particularly abduction.

Technique Of Examination

INSPECTION

- Observe the shoulder and shoulder girdle anteriorly and inspect the scapulae and related muscles posteriorly. Note any swelling, deformity, or muscle atrophy or fasciculations.
- Look for swelling of the joint capsule anteriorly or a bulge in the subacromial bursa under the deltoid muscle.

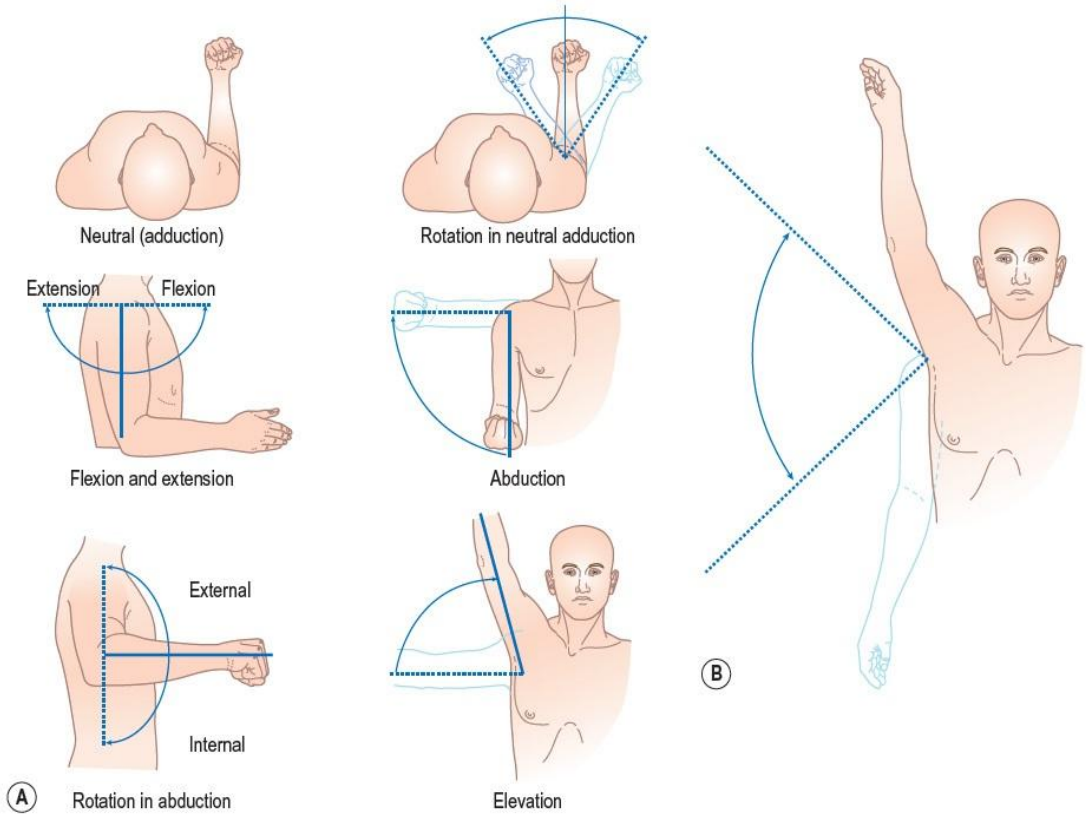
PALPATION

If there is a history of shoulder pain, ask the patient to point to the painful area. The location of the pain may provide clues as to its origin:

- Top of the shoulder, radiating toward the neck—acromioclavicular joint
- Lateral aspect of the shoulder, radiating toward the deltoid insertion—rotator cuff
- Anterior shoulder—bicipital tendon.

RANGE OF MOTION AND MANEUVERS

- The six motions of the shoulder girdle are flexion, extension, abduction, adduction, and internal and external rotation.
- Watch for smooth, fluid movement as you stand in front of the patient and ask the patient to (1) raise (abduct) the arms to shoulder level (90°) with palms facing down (tests pure glenohumeral motion); (2) raise the arms to a vertical position above the head with the palms facing each other (tests scapulothoracic motion for 60°, and combined glenohumeral and scapulothoracic motion during adduction for the final 30°; (3) place both hands behind the neck, with elbows out to the side (tests external rotation and abduction); and (4) place both hands behind the small of the back (tests internal rotation and adduction).
- Placing your hand on the shoulder during these movements allows you to detect any crepitus.

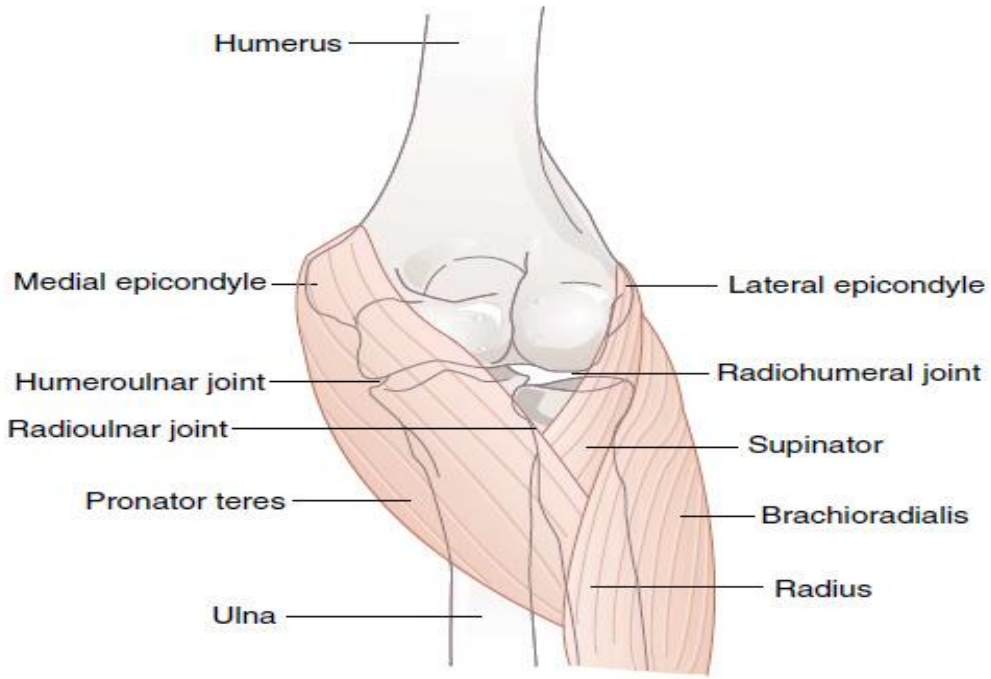


The Elbow Joint

- The elbow joint is formed by the humerus and the two bones of the forearm, the radius and the ulna.
- These bones have three articulations:
  The humeroulnar joint, the radiohumeral joint, and the radioulnar joint. All three share a large common articular cavity and an extensive synovial lining.

Muscle Groups and Additional Structures.

Muscles traversing the elbow include the biceps and brachioradialis (flexion), the triceps (extension), the pronator teres (pronation), and the supinator (supination).



LEFT ELBOW—ANTERIOR VIEW

Technique Of Examination

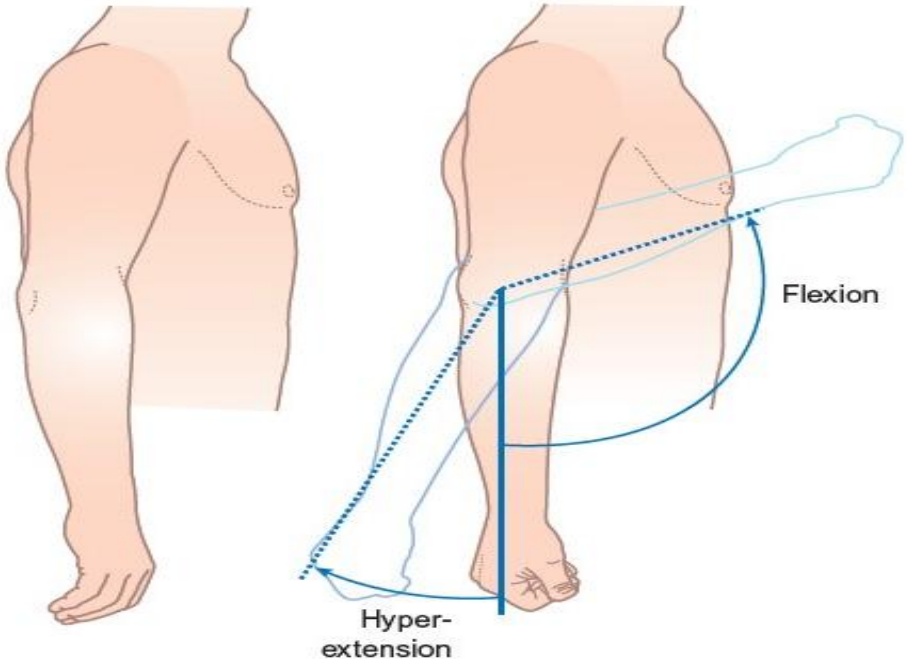
INSPECTION AND PALPATION

- Support the patient's forearm with your opposite hand so the elbow is flexed to about 70°. Identify the medial and lateral epicondyles and the olecranon process of the ulna. Inspect the contours of the elbow, including the extensor surface of the ulna and the olecranon process. Note any nodules or swelling.
- Palpate the olecranon process and press on the epicondyles for tenderness.

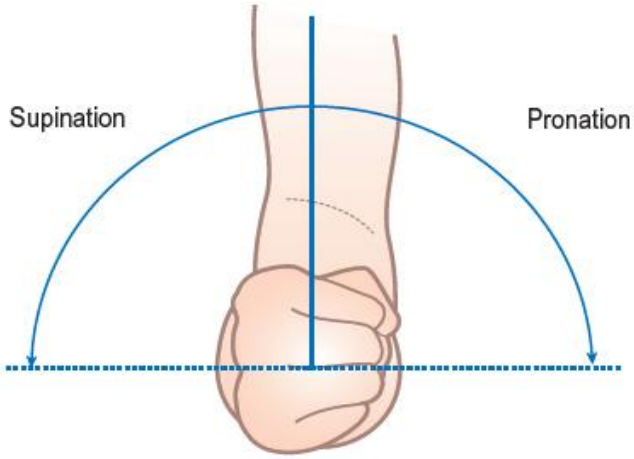
Note any displacement of the olecranon.

RANGE OF MOTION AND MANEUVERS

- Range of motion includes flexion and extension at the elbow and pronation and supination of the forearm. To test flexion and extension, ask the patient to bend and straighten the elbow.
- With the patient's arms at the sides and elbows flexed to minimize shoulder movement, ask the patient supinate, or turn up the palms, and to pronate, or turn down the palms.



Movements of the elbow.

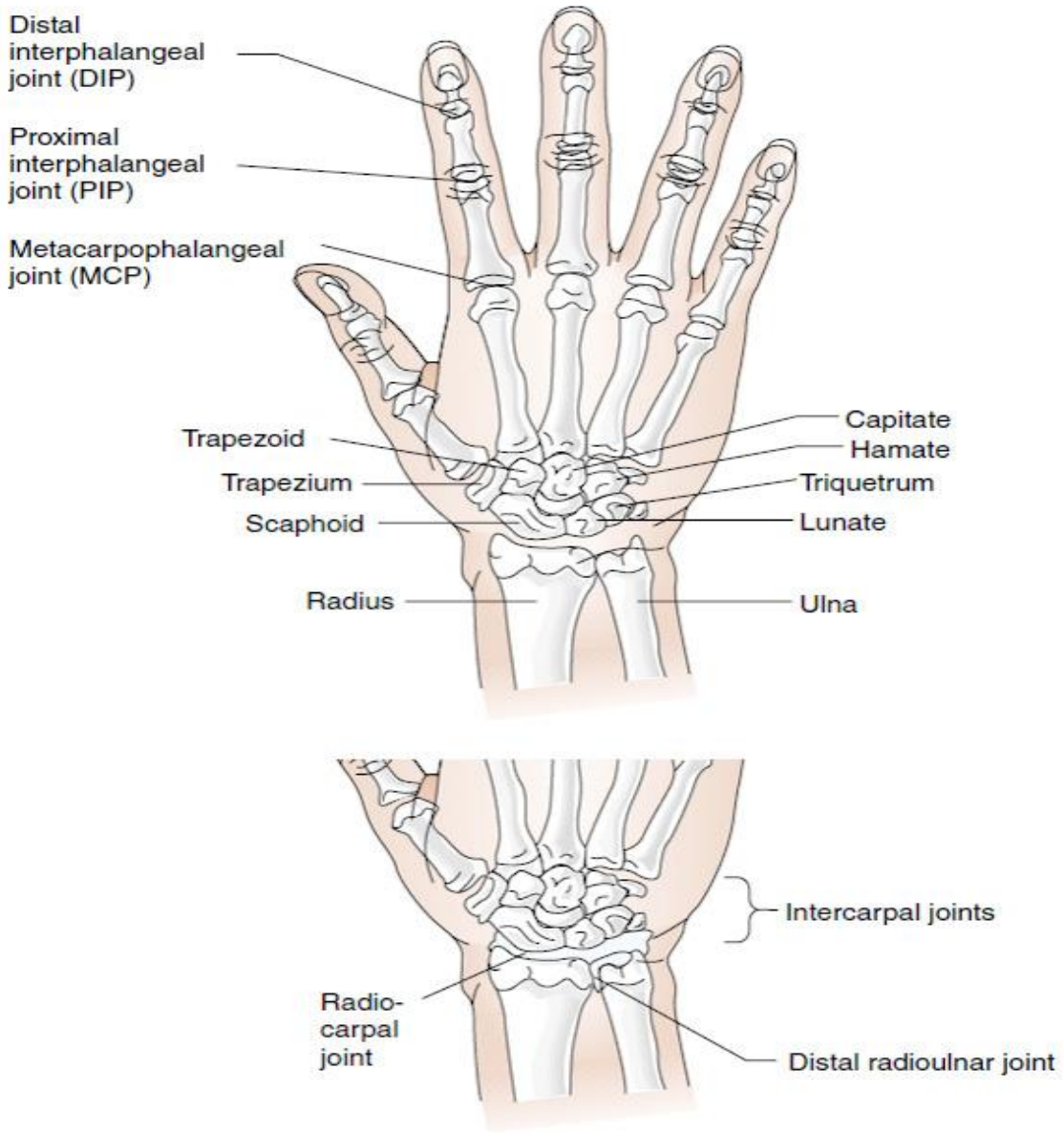


Supination and pronation

The Wrist and Hands

Bony Structures.

The wrist includes the distal radius and ulna and eight small carpal bones.
The carpal bones lie distal to the wrist joint within each hand.



Joints.

The numerous joints of the wrist and hand lend unusual dexterity to the hands.

Wrist joints.

- The wrist joints include the radiocarpal or wrist joint, the distal radioulnar joint, and the intercarpal joints.
- The joint capsule, articular disc, and synovial membrane of the wrist join the radius to the ulna and to the proximal carpal bones. On the dorsum of the wrist, locate the groove of the radiocarpal joint.

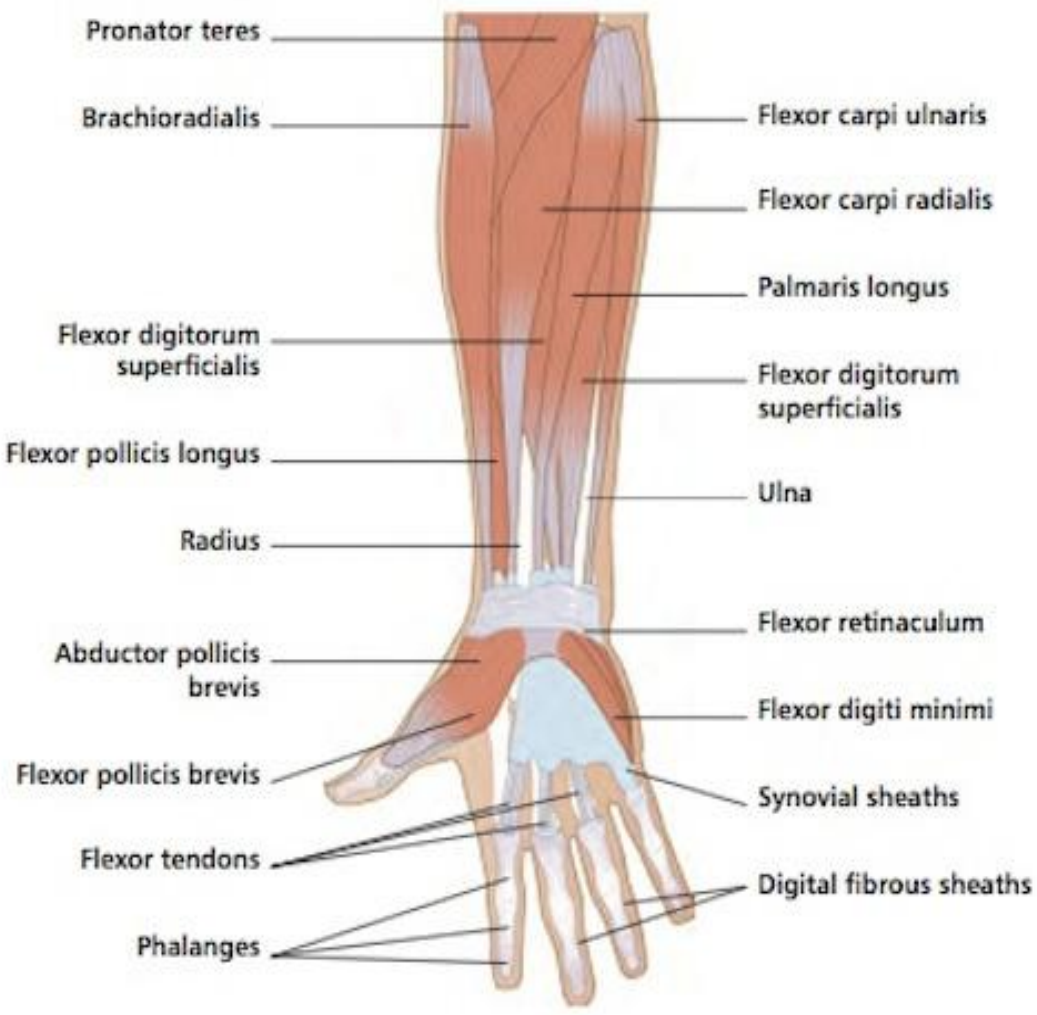
Hand joints.

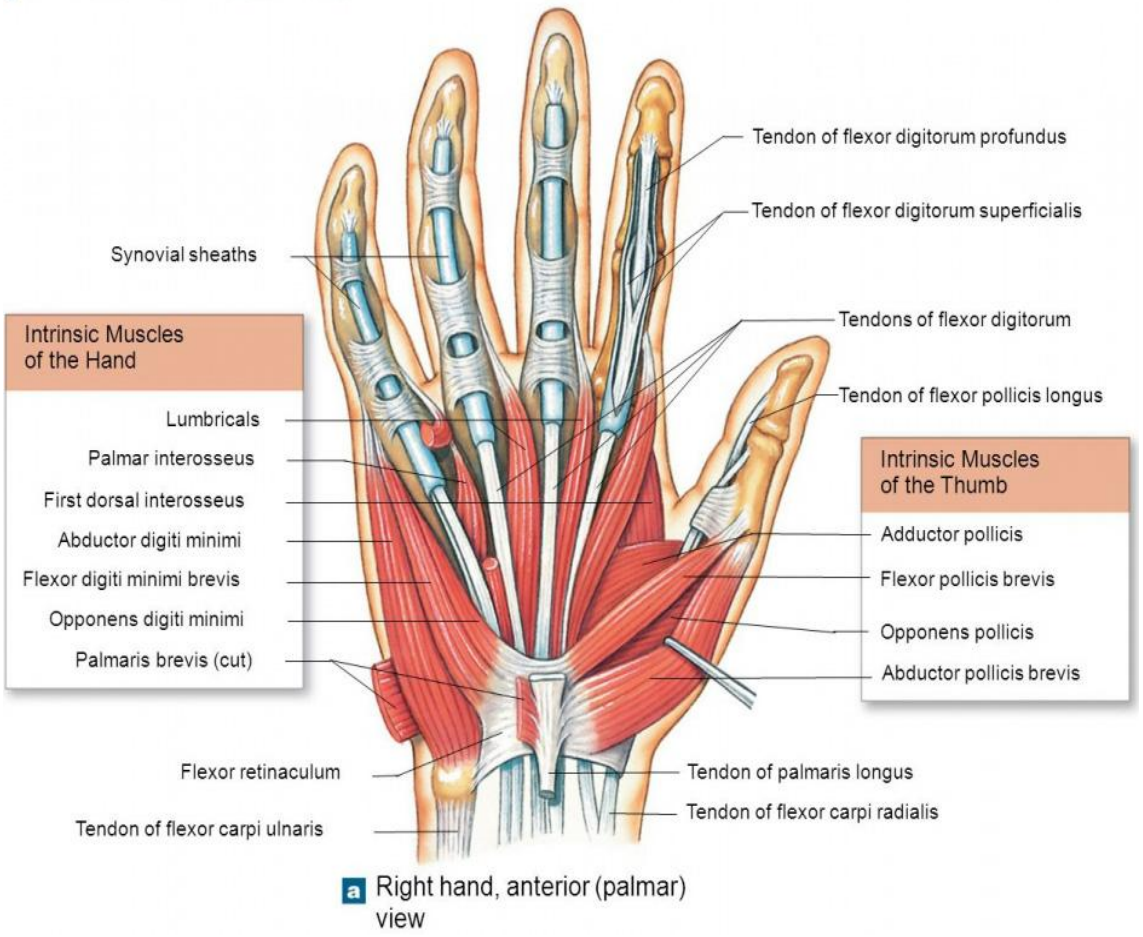
The joints of the hand include the metacarpophalangeal joints (MCPs), the proximal interphalangeal joints (PIPs), and the distal interphalangeal joints (DIPs).

Muscle Groups.

- Wrist flexion arises from the two carpal muscles, located on the radial and ulnar surfaces. Two radial and one ulnar muscle provide wrist extension. Supination and pronation result from muscle contraction in the forearm.
- The thumb is powered by three muscles that form the thenar eminence and provide flexion, abduction, and opposition.
- The intrinsic muscles of the hand attaching to the metacarpal bones are involved in flexion (lumbricals), abduction (dorsal interossei), and adduction (palmar interossei) of the fingers.

FLEXOR MUSCLES OF THE RIGHT FOREARM AND WRIST, SEEN FROM THE FRONT.





Additional Structures.

- Soft-tissue structures, especially tendons and tendon sheaths, are extremely important in the wrist and hand. Six extensor tendons and two flexor tendons pass across the wrist and hand to insert on the fingers.
- Through much of their course these tendons travel in tunnel like sheaths, generally palpable only when swollen or inflamed.
- Holding the tendons and tendon sheath in place is a transverse ligament, the flexor retinaculum. The median nerve lies between the flexor retinaculum and the tendon sheath. It provides sensation to the palm and the palmar surface of most of the thumb, the second and third digits, and half of the fourth digit. It also innervates the thumb muscles of flexion, abduction, and opposition.

Technique Of Examination

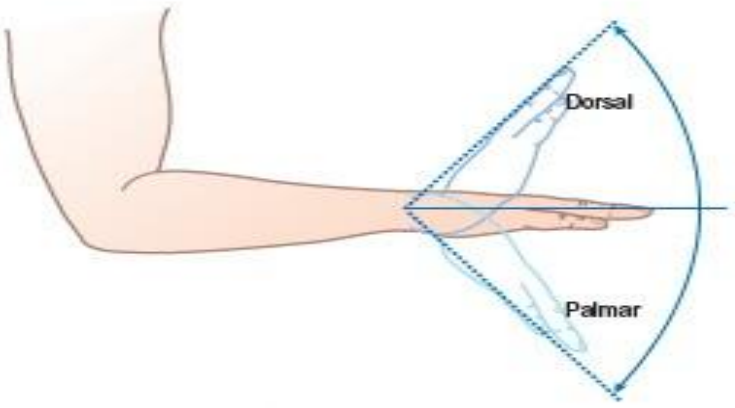
INSPECTION

- Observe the position of the hands in motion to see if movements are smooth and natural. At rest the fingers should be slightly flexed and aligned almost in parallel.

- Inspect the palmar and dorsal surfaces of the wrist and hand carefully for swelling over the joints.
- Note any deformities of the wrist, hand, or finger bones, as well as any angulation from radial or ulnar deviation.
- Observe the contours of the palm, namely the thenar and hypothenar eminences.
- Note any thickening of the flexor tendons or flexion contractures in the fingers.

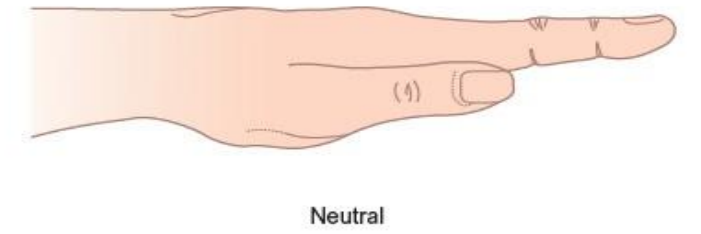
Deviation

Neutral

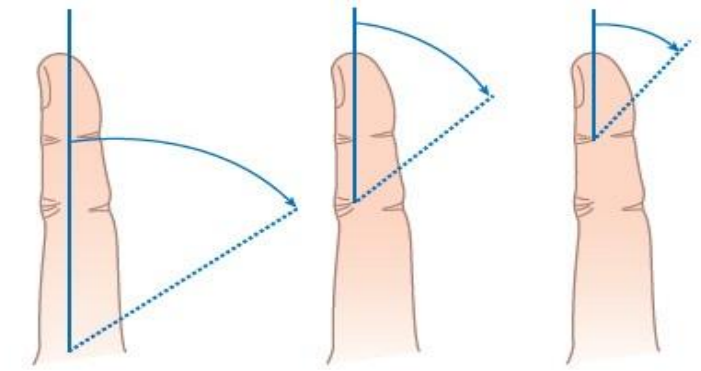


Flexion of the wrist

Movements of the wrist.



Neutral



Movements of the fingers.

PALPATION

- At the wrist, palpate the distal radius and ulna on the lateral and medial surfaces. Palpate the groove of each wrist joint with your thumbs on the dorsum of the wrist, your fingers beneath it. Note any swelling, bogginess, or tenderness.
- Palpate the eight carpal bones lying distal to the wrist joint, and then each of the five metacarpals and the proximal, middle, and distal phalanges.
- Compress the MCP joints by squeezing the hand from each side between the thumb and fingers. Alternatively, use your thumb to palpate each MCP joint just distal to and on each side of the knuckle as your index finger feels the head of the metacarpal in the palm. Note any swelling, bogginess, or tenderness.
- Now examine the fingers. Palpate the medial and lateral aspects of each PIP joint between your thumb and index finger, again checking for swelling, bogginess, bony enlargement, or tenderness.
- Using the same techniques, examine the DIP joints.

RANGE OF MOTION AND MANEUVERS

Now assess range of motion for the wrists, fingers, and thumbs. At the wrist, test flexion, extension, and ulnar and radial deviation.

Flexion.

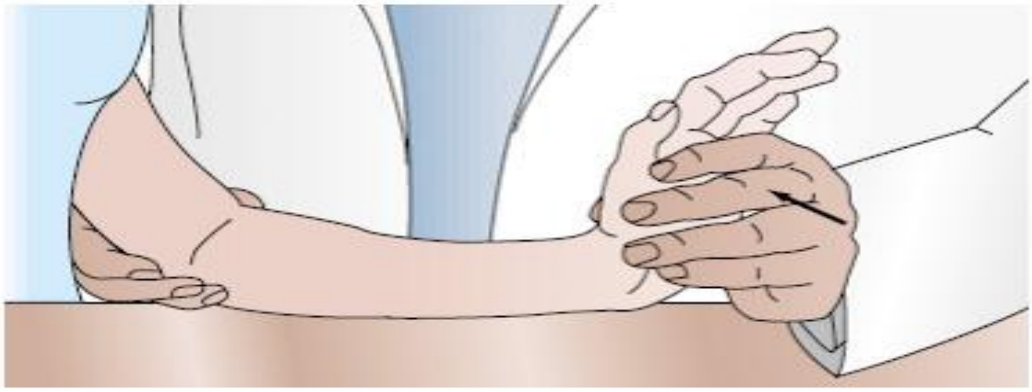
With the patient's forearm stabilized, place the wrist in extension and place your fingertips in the patient's palm. Ask the patient to flex the wrist against gravity, then against graded resistance.

Extension.

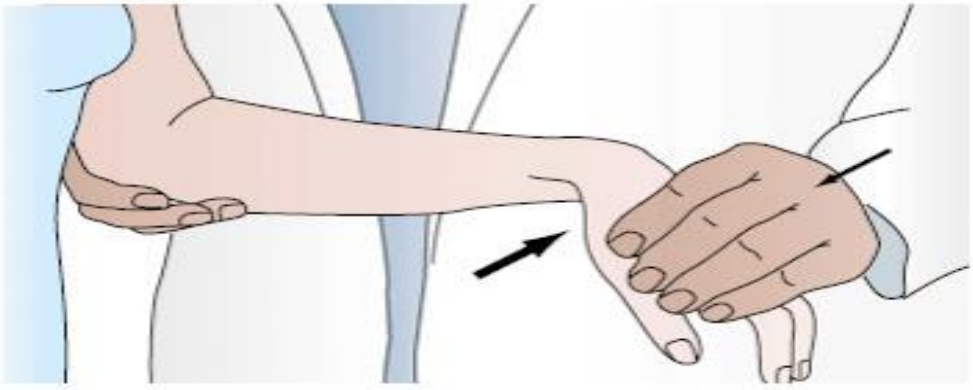
With the patient's forearm stabilized, place the wrist in flexion and put your hand on the patient's dorsal metacarpals. Ask the patient to extend the wrist against gravity, then against graded resistance.

Ulnar and radial deviation.

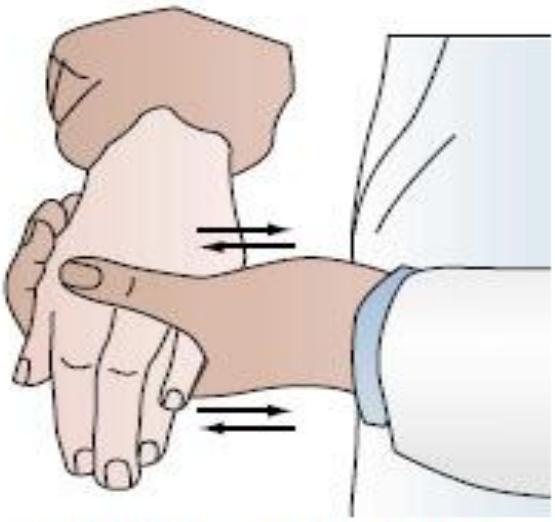
With palms down, ask the patient to move the wrists laterally and medially.



FLEXION



EXTENSION



ULNAR AND RADIAL DEVIATION

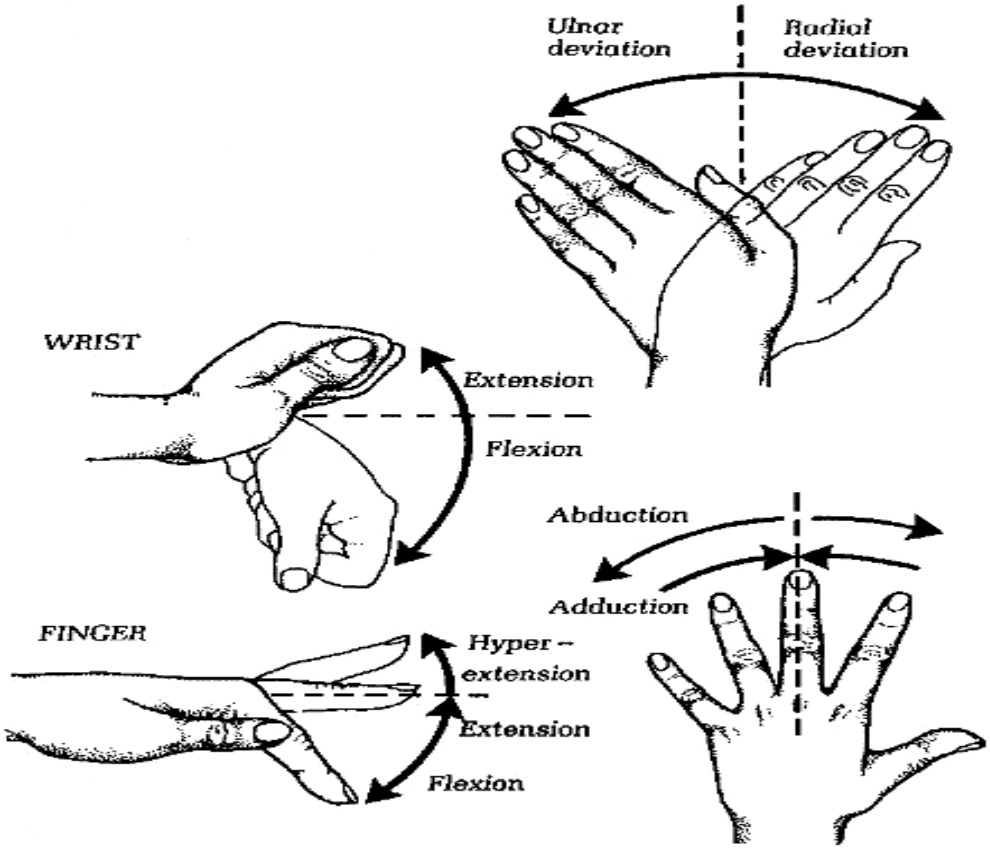
Test flexion, extension, abduction, and adduction of the fingers:

Flexion and extension.

Ask the patient to make a tight fist with each hand, thumb across the knuckles, and then extend and spread the fingers. The fingers should close and open smoothly and easily. At the MCPs, the fingers may extend beyond the neutral position. Also test flexion and extension at the PIP and DIP joints.

Abduction and adduction.

- Ask the patient to spread the fingers apart (abduction) and back together (adduction). Check for smooth, coordinated movement.
- At the thumb, assess flexion, extension, abduction, adduction, and opposition. Ask the patient to move the thumb across the palm and touch the base of the 5th finger to test flexion, and then to move the thumb back across the palm and away from the fingers to test extension.
- Next, ask the patient to place the fingers and thumb in the neutral position with the palm up, then have the patient move the thumb anteriorly away from the palm to assess abduction and back down for adduction. To test opposition, or movements of the thumb across the palm, ask the patient to touch the thumb to each of the other fingertips.



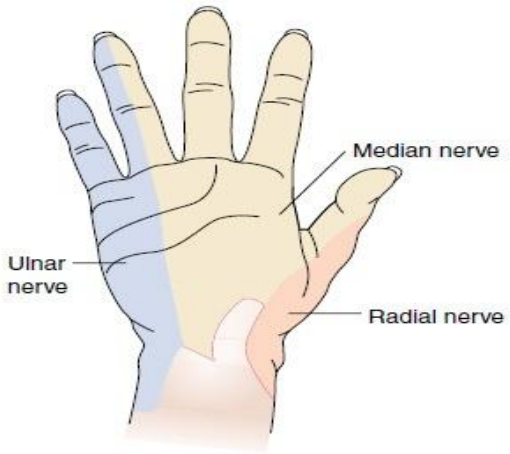
Neutral

Extension Opposition

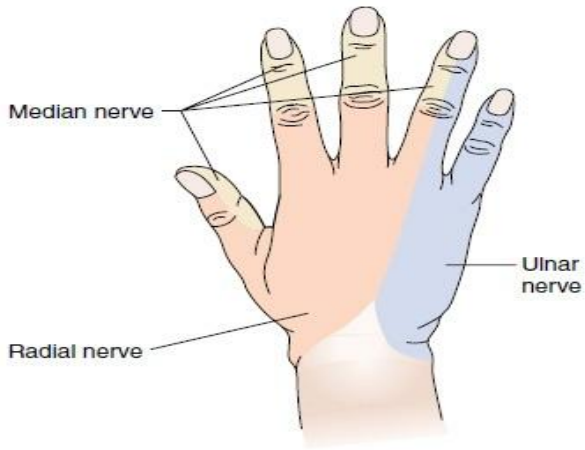
Movements of the thumb.

Test sensation in the fingers only along the lateral and medial surfaces to isolate any alterations in the digital nerves. Test median, ulnar, and radial nerve function by checking sensation as follows:

- Pulp of the index finger—median nerve
- Pulp of the 5th finger—ulnar nerve
- Dorsal web space of the thumb and index finger—radial nerve.



VOLAR SURFACE

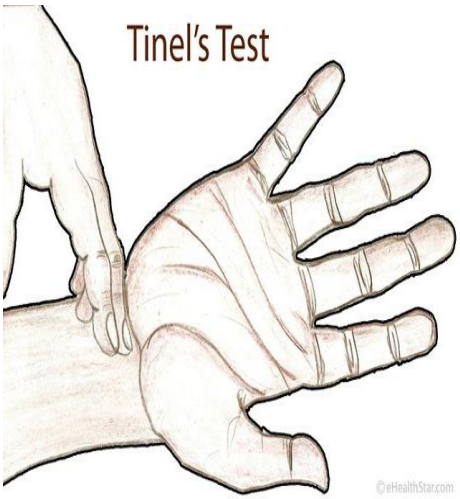


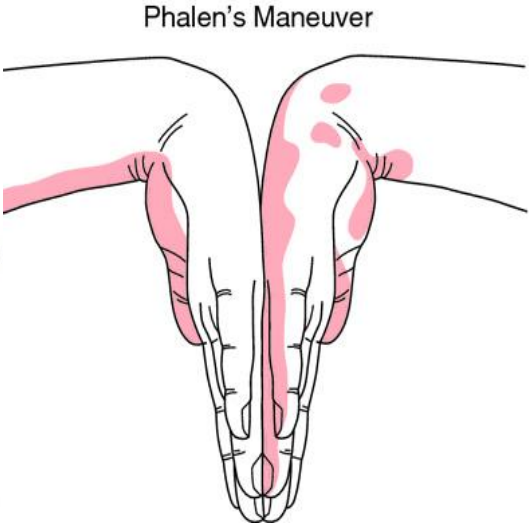
DORSAL SURFACE

SPECIAL TECHNIQUES

For the Carpal Tunnel Syndrome

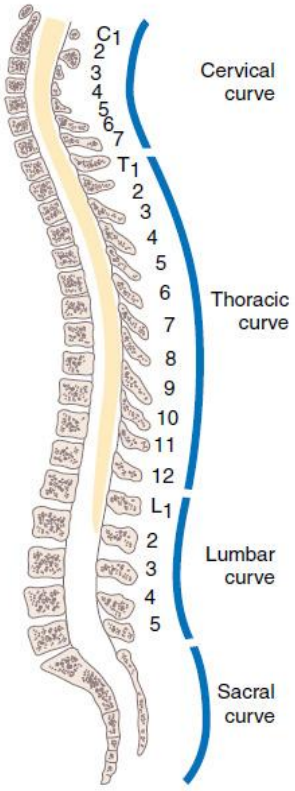
- Pain and numbness on the ventral surface of the first three digits of the hand (but not in the palm), especially at night, suggest median nerve compression in the carpal tunnel, which lies between the carpal bones dorsally and a ventral band of more superficial fascia, the flexor retinaculum.
- **Tinel's Sign**. With your finger, percuss lightly over the course of the median nerve in the carpal tunnel.
- **Phalen's Test**. Hold the patient's wrists in acute flexion for 60 seconds. Alternatively, ask the patient to press the backs of both hands together to form right angles. These maneuvers compress the median nerve.





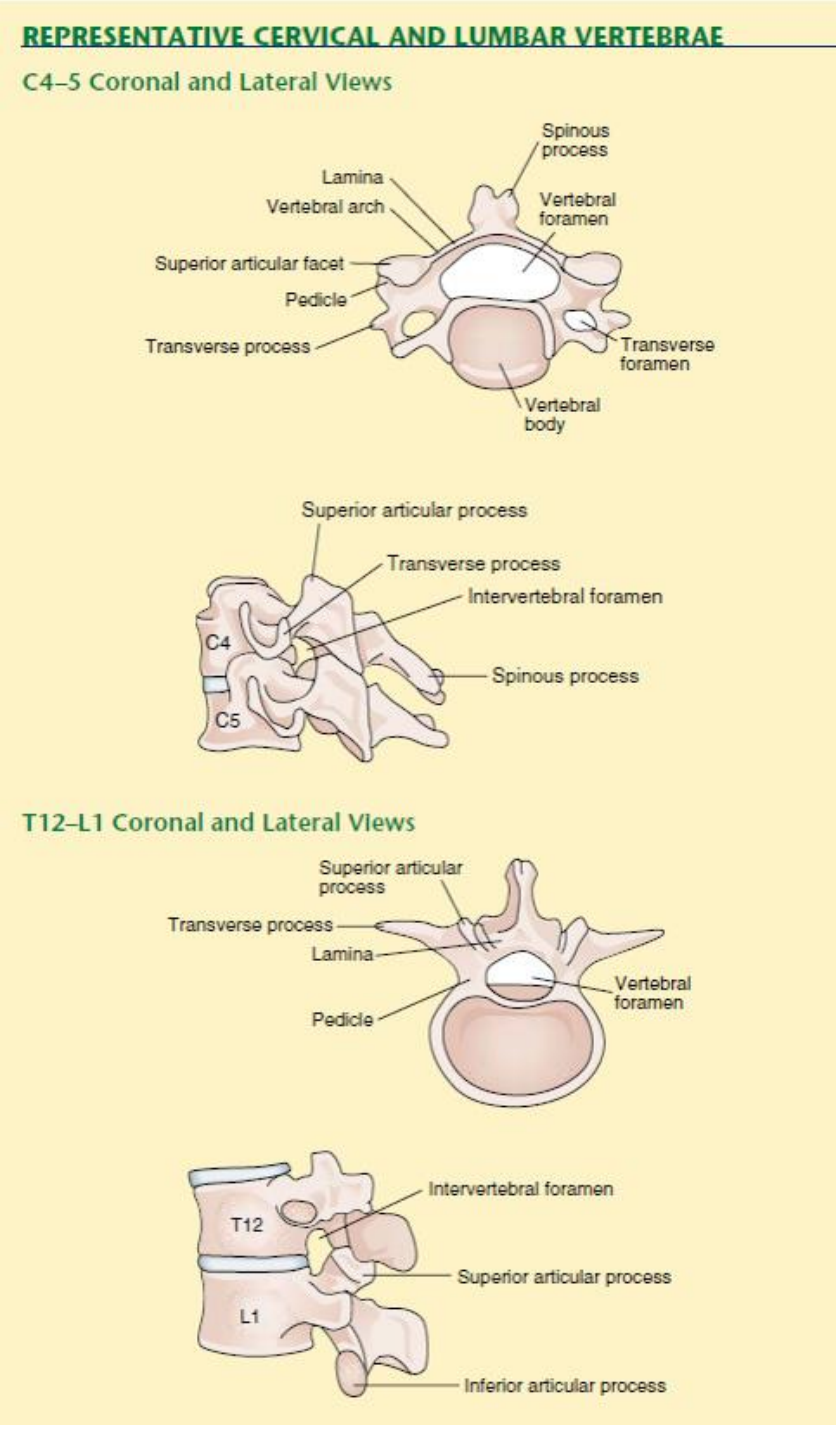
The Spine

- The vertebral column, or spine, is the central supporting structure of the trunk and back. Note the concave curves of the cervical and lumbar spine and the convex curves of the thoracic and sacrococcygeal spine. These curves help distribute upper body weight to the pelvis and lower extremities and cushion the concussive impact of walking or running.
- Spinous processes, usually more prominent at C7 and T1 and more evident on forward flexion.
- A line drawn above the posterior iliac crests crosses the spinous process of L4.



Bony Structures.

- The vertebral column contains 24 vertebrae stacked on the sacrum and coccyx. A typical vertebra contains sites for joint articulations, weight bearing, and muscle attachments, as well as foramina for the spinal nerve roots and peripheral nerves. Anteriorly, the vertebral body supports weight bearing. The posterior vertebral arch encloses the spinal cord.
- The proximity of the spinal cord and spinal nerve roots to their bony vertebral casing and the intervertebral discs makes them especially vulnerable to disc herniation, impingement from degenerative changes in the vertebrae, and trauma.

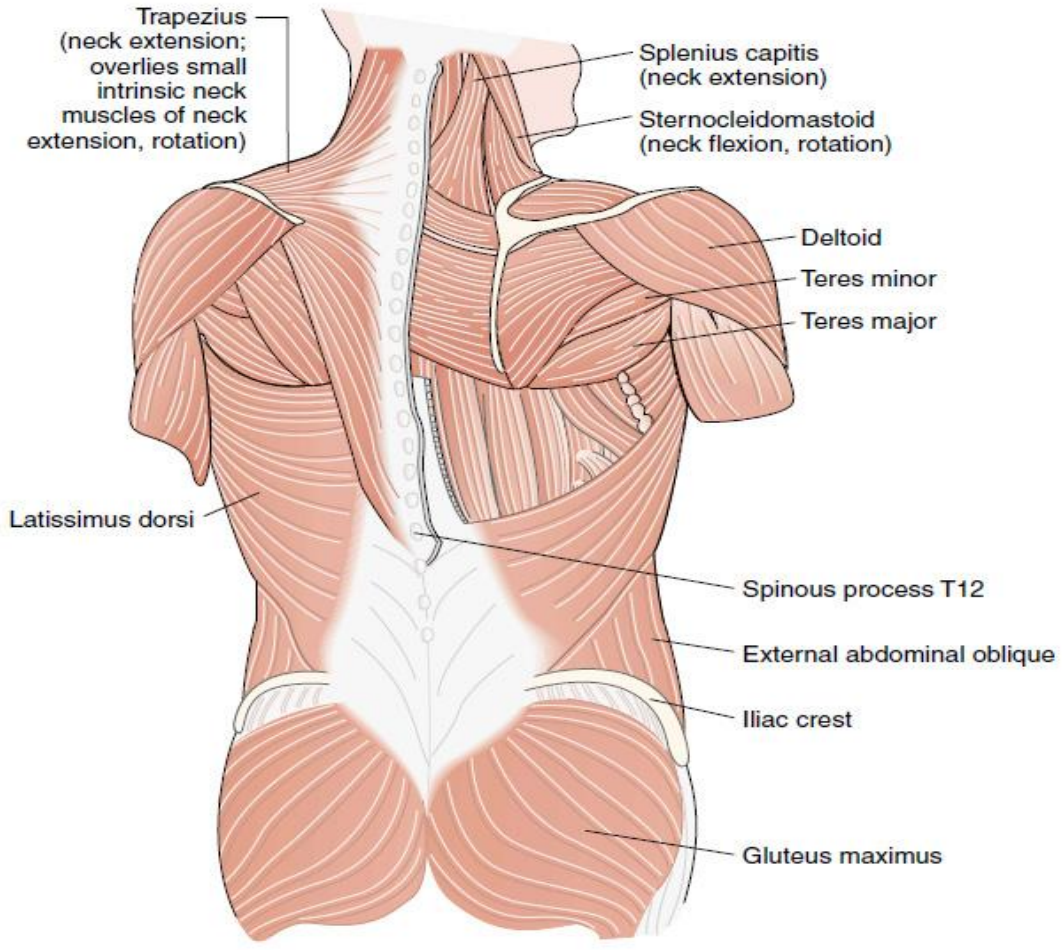


Joints.

- The spine has slightly movable cartilaginous joints between the vertebral bodies and between the articular facets. Between the vertebral bodies are the intervertebral discs, each consisting of a soft mucoid central core, the nucleus pulposus, rimmed by the tough fibrous tissue of the annulus fibrosis.
- The vertebral column angles sharply posterior at the lumbosacral junction and becomes immovable. The mechanical stress at this angulation contributes to the risk of disc herniation and subluxation, or slippage, of L5 on S1.

Muscle Groups.

- The trapezius and latissimus dorsi form the large outer layer of muscles attaching to each side of the spine. They overlie two deeper muscle layers—a layer attaching to the head, neck, and spinous processes (splenius capitis, splenius cervicis, and sacrospinalis) and a layer of smaller intrinsic muscles between vertebrae.
- Muscles attaching to the anterior surface of the vertebrae, including the psoas muscle and muscles of the abdominal wall, assist with flexion.



Movement	Principal Muscle Group
Cervical Spine (neck)	
Flexion	Sternocleidomastoid, scalene, and prevertebral muscles
Extension	Splenius, trapezius, small intrinsic neck muscles
Rotation	Sternocleidomastoid, small intrinsic neck muscles
Lateral bending	Scalene and small intrinsic neck muscles
Lumbar Spine	
Flexion	Psoas major, psoas minor, quadratus lumborum; abdominal muscles such as the internal and external obliques and rectus abdominis, attaching to the anterior vertebrae
Extension	Intrinsic muscles of the back, sacrospinalis
Rotation	Abdominal muscles, intrinsic muscles of the back
Lateral bending	Abdominal muscles, intrinsic muscles of the back

Technique Of Examination

INSPECTION

- Begin by observing the patient's posture, including the position of both neck and trunk, when entering the room.
- Inspect the patient from the side. Evaluate the spinal curvatures.

PALPATION

- From a sitting or standing position, palpate the spinous processes of each vertebra with your thumb.
- In the neck, also palpate the facet joints that lie between the cervical vertebrae about 1 inch lateral to the spinous processes of C2–C7.
- Palpate over the sacroiliac joint, often identified by the dimple overlying the posterior superior iliac spine.
- Inspect and palpate the paravertebral muscles for tenderness and spasm.

RANGE OF MOTION AND MANEUVERS

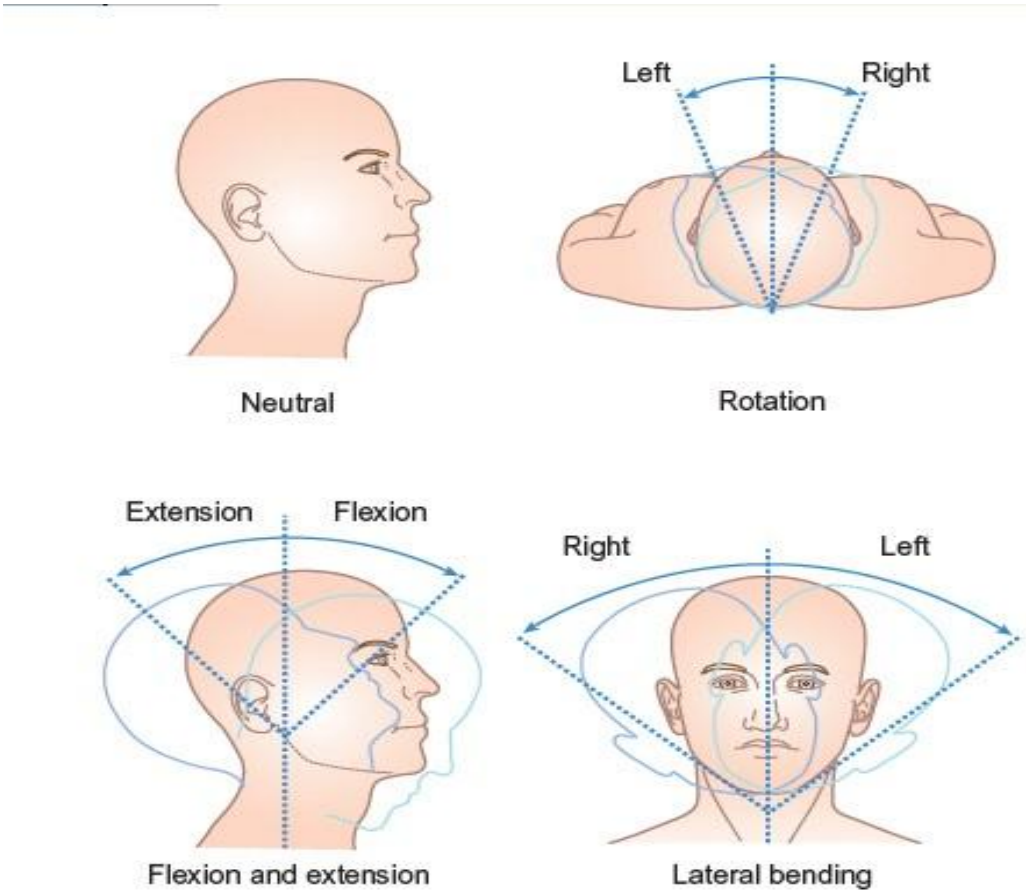
The neck is the most mobile portion of the spine.

Flexion and extension occur primarily between the skull and C1 (the atlas), rotation at C1–C2 (the axis), and lateral bending at C2–C7.

Ask the patient to perform the following maneuvers, and check for smooth, coordinated motion:

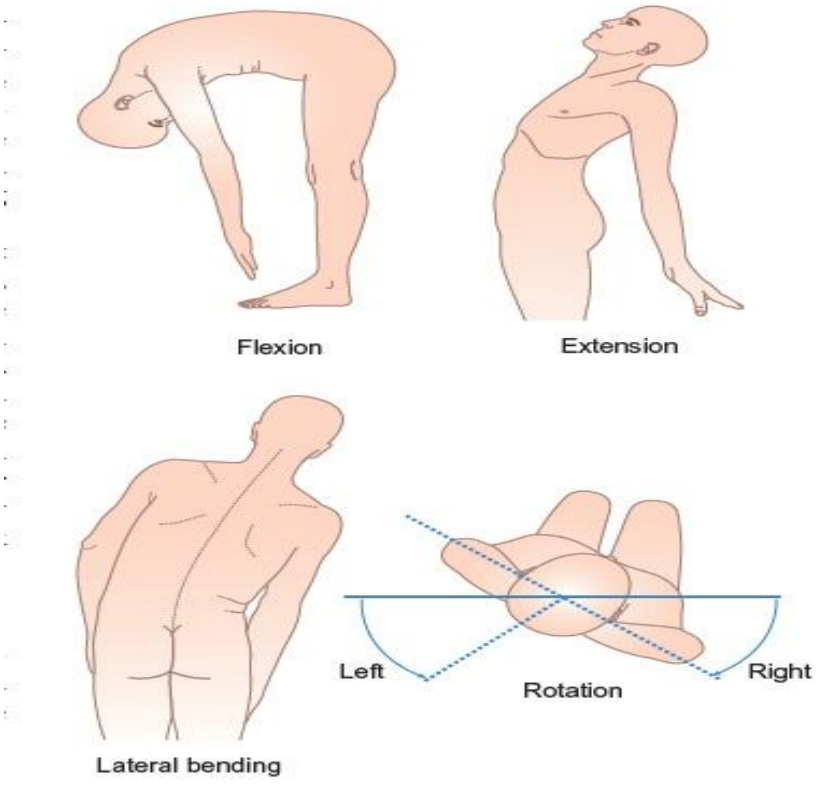
- Flexion. Touch the chin to the chest.
- Extension. Look up at the ceiling.
- Rotation. Turn the head to each side, looking directly over the shoulder.
- Lateral bending. Tilt the head, touching each ear to the corresponding

shoulder.



Movements of the neck.

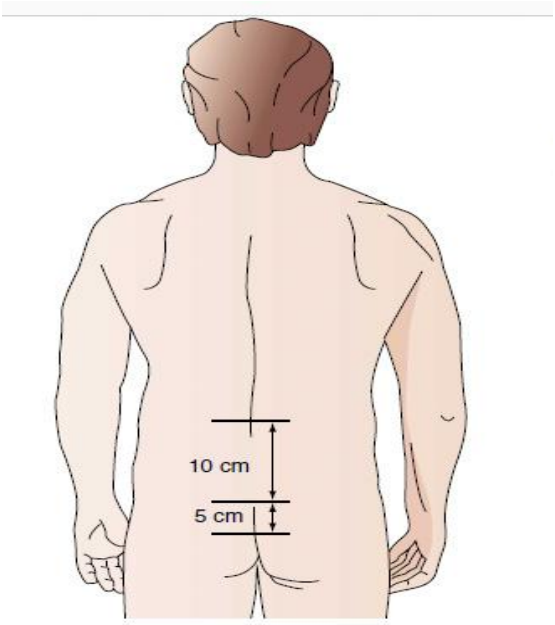
NOW ASSESS RANGE OF MOTION IN THE SPINAL COLUMN.

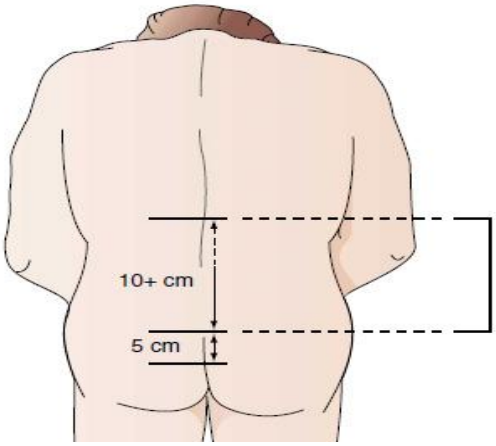


Movements of the lumbar and dorsal spine.

Flexion.

- Ask the patient to bend forward to touch the toes (flexion). Note the smoothness and symmetry of movement, the range of motion, and the curve in the lumbar area. As flexion proceeds, the lumbar concavity should flatten out.
- You may wish to measure the degree of flexion of the spine with the patient standing and bending forward. Mark the spine at the lumbosacral junction, then 10 cm above and 5 cm below this point. A 4-cm increase between the two upper marks is normally seen The distance between the lower two marks should be unchanged.





Extension.

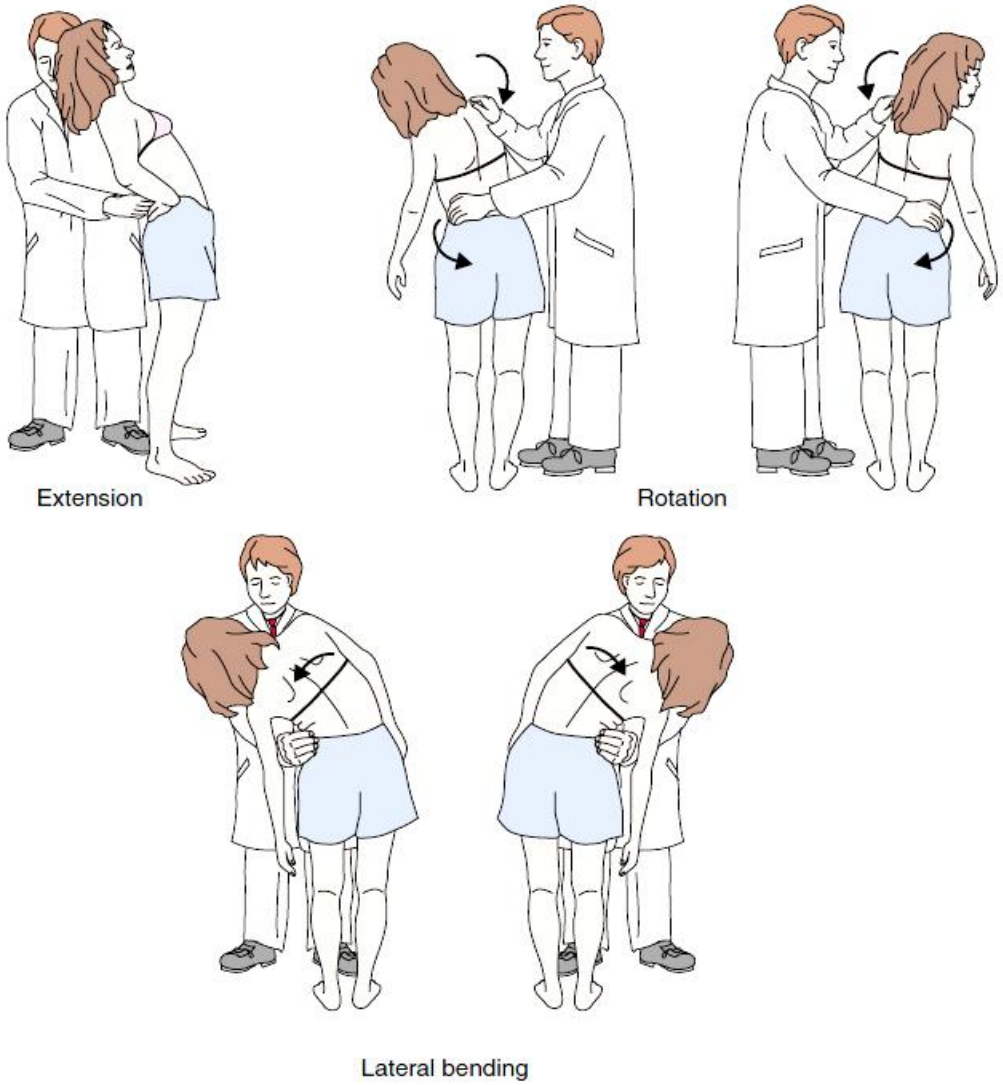
Place your hand on the posterior superior iliac spine, with your fingers pointing toward the midline, and ask the patient to bend backward as far as possible.

Rotation.

Stabilize the pelvis by placing one hand on the patient's hip and the other on the opposite shoulder. Then rotate the trunk by pulling the shoulder and then the hip posteriorly. Repeat these maneuvers for the opposite side.

Lateral bending.

Again, stabilize the pelvis by placing your hand on the patient's hip. Ask the patient to lean to both sides as far as possible.

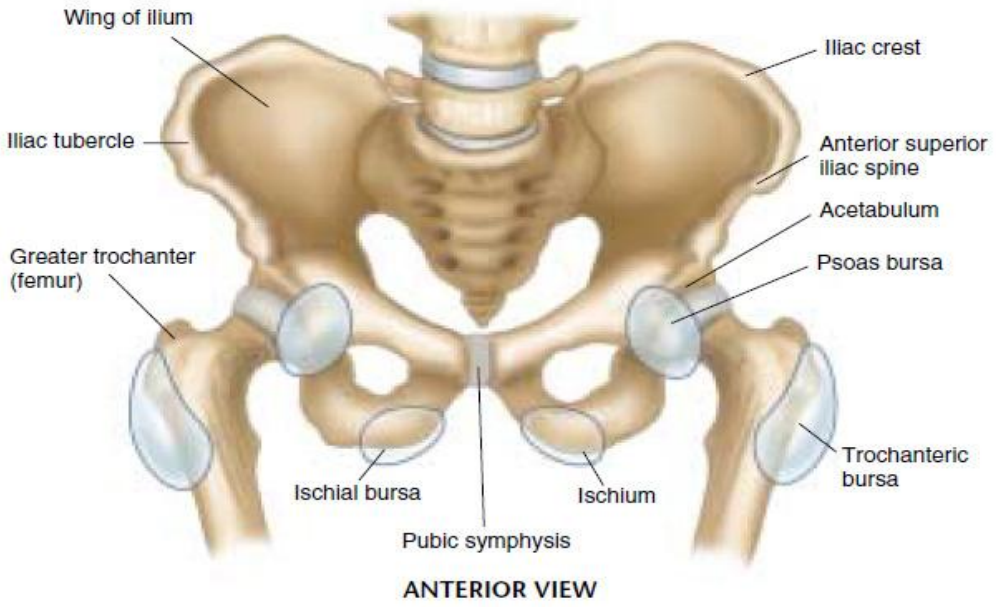


The Hip Joint

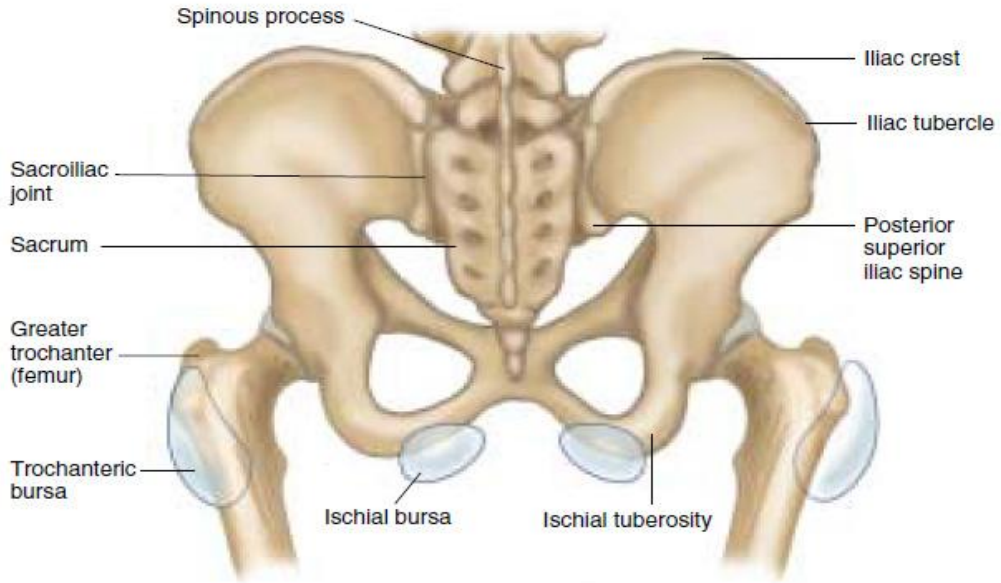
- The hip joint is deeply embedded in the pelvis, and is notable for its strength, stability, and wide range of motion.
- The stability of the hip joint, so essential for weight bearing, arises from the deep fit of the head of the femur into the acetabulum, its strong fibrous

articular capsule, and the powerful muscles crossing the joint and inserting below the femoral head, providing leverage for movement of the femur.

- It is a ball-and-socket joint— note how the rounded head of the femur articulates with the cuplike cavity of the acetabulum. Because of its overlying muscles and depth, it is not readily palpable.



ANTERIOR VIEW



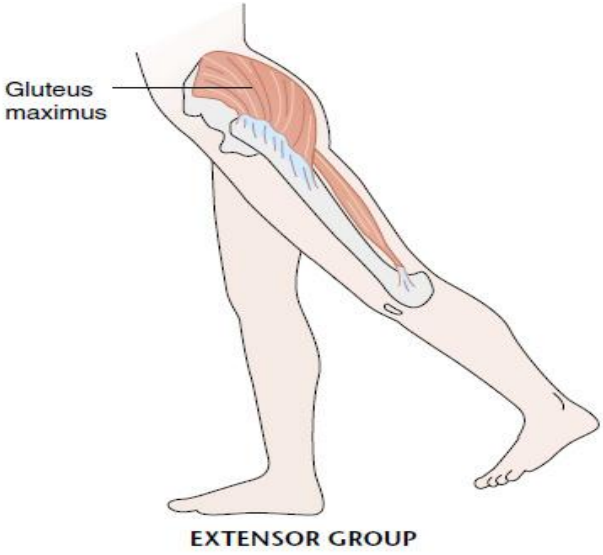
POSTERIOR VIEW

Muscle Groups.

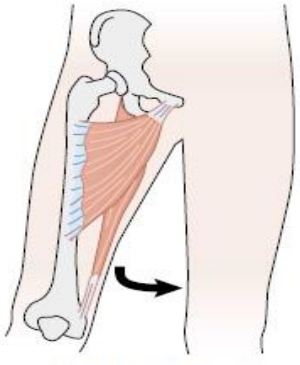
- Four powerful muscle groups move the hip. The flexor group lies anteriorly and flexes the thigh. The primary hip flexor is the iliopsoas, extending from above the iliac crest to the lesser trochanter.

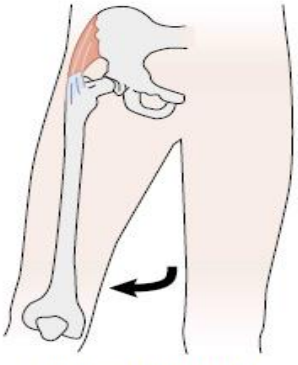
- The extensor group lies posteriorly and extends the thigh. The gluteus maximus is the primary extensor of the hip. It forms a band crossing from its origin along the medial pelvis to its insertion below the trochanter. The adductor group is medial and swings the thigh toward the body.
- The muscles in this group arise from the rami of the pubis and ischium and insert on the posteromedial aspect of the femur. The abductor group is lateral, extending from the iliac crest to the head of the femur, and moves the thigh away from the body. This group includes the gluteus medius and minimus. These muscles help stabilize the pelvis during the stance phase of gait.

FLEXOR GROUP



EXTENSOR GROUP


ADDUCTOR GROUP


ABDUCTOR GROUP

Technique Of Examination

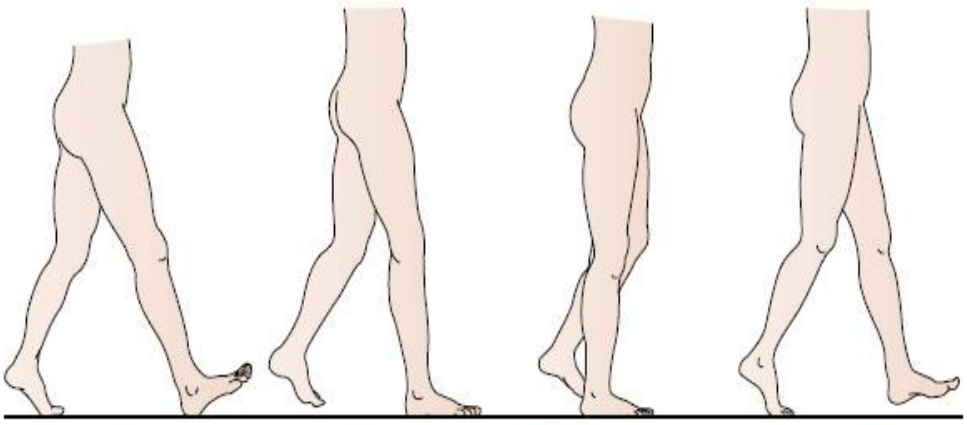
INSPECTION

Inspection of the hip begins with careful observation of the patient's gait on entering the room. Observe the two phases of gait;

- Stance—when the foot is on the ground and bears weight (60% of the

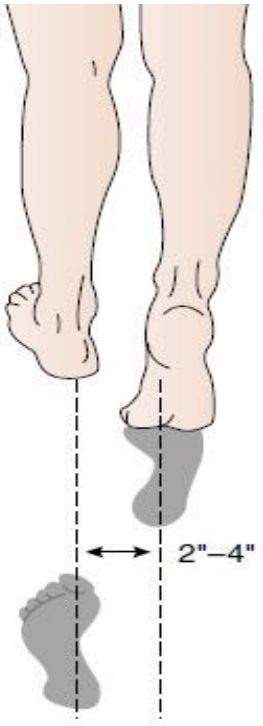
walking cycle).

- Swing—when the foot moves forward and does not bear weight (40% of the cycle).



THE STANCE PHASE OF GAIT

- Observe the gait for the width of the base, the shift of the pelvis, and flexion of the knee. The width of the base should be 2 to 4 inches from heel to heel. Normal gait has a smooth, continuous rhythm, achieved in part by contraction of the abductors of the weight-bearing limb.
- Abductor contraction stabilizes the pelvis and helps maintain balance, raising the opposite hip. The knee should be flexed throughout the stance phase, except when the heel strikes the ground to counteract motion at the ankle.



Observe the lumbar portion of the spine for slight lordosis and, with the patient supine, assess the length of the legs for symmetry.

PALPATION

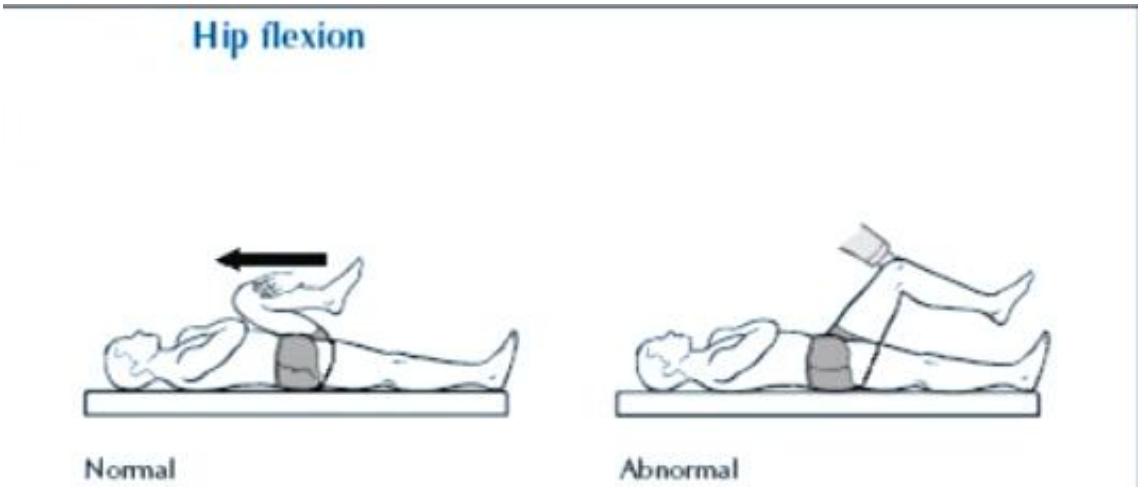
- With the patient supine, ask the patient to place the heel of the leg being examined on the opposite knee. Then palpate along the inguinal ligament, which extends from the anterior superior iliac spine to the pubic tubercle.
- The femoral nerve, artery, and vein bisect the overlying inguinal ligament; lymph nodes lie medially. The mnemonic NAVEL may help you remember the lateral-to-medial sequence of Nerve—Artery—Vein—Empty space—Lymph node. If the hip is painful, palpate the iliopectineal (iliopsoas) bursa, below the inguinal ligament but on a deeper plane.

RANGE OF MOTION AND MANEUVERS

Motions at the hip include flexion, extension, abduction, adduction, and rotation. Note that the hip can flex farther when the knee is also flexed. Rotation at the hip while the knee is flexed may be confusing at first: when the lower leg swings laterally, the femur rotates internally. It is the motion of the femur at the hip joint that identifies these movements.

Flexion.

- With the patient supine, place your hand under the patient's lumbar spine. Ask the patient to bend each knee in turn up to the chest and pull it firmly against the abdomen. Note when the back touches your hand, indicating normal flattening of the lumbar lordosis—further flexion must arise from the hip joint itself.
- As the thigh is held against the abdomen, observe the degree of flexion at the hip and knee. Normally the anterior portion of the thigh can almost touch the chest wall. Note whether the opposite thigh remains fully extended, resting on the table.

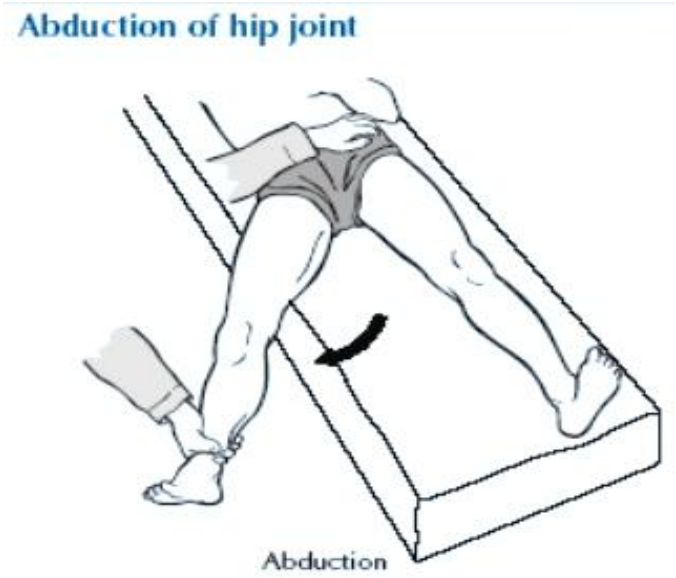


Extension.

With the patient lying face down, extend the thigh toward you in a posterior direction.

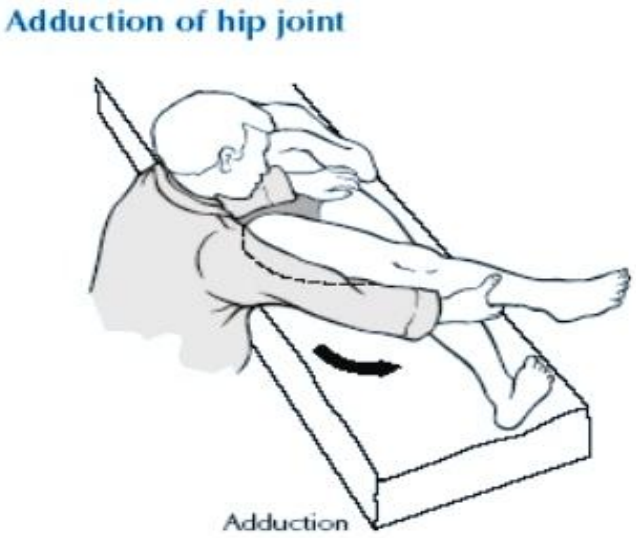
Abduction.

- Stabilize the pelvis by pressing down on the opposite anterior superior iliac spine with one hand. With the other hand, grasp the ankle and abduct the extended leg until you feel the iliac spine move. This movement marks the limit of hip abduction.



Adduction.

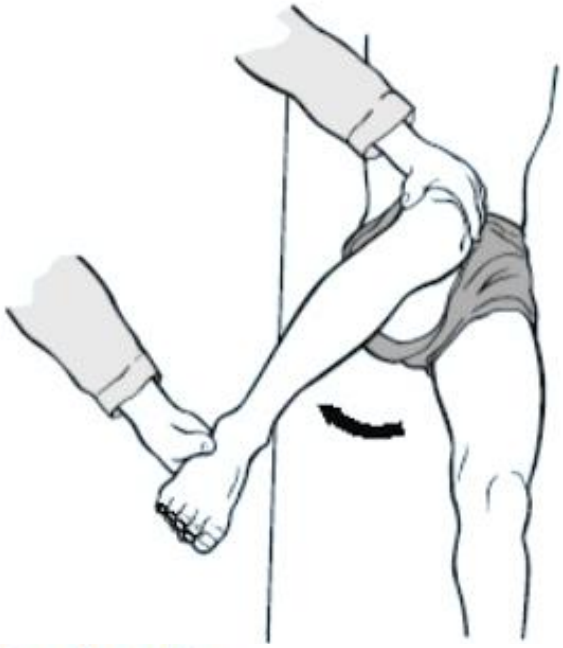
With the patient supine, stabilize the pelvis, hold one ankle, and move the leg medially across the body and over the opposite extremity.



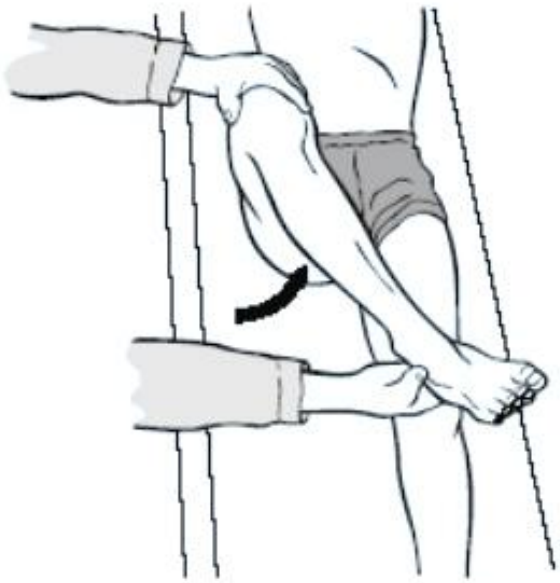
Rotation.

Flex the leg to 90° at hip and knee, stabilize the thigh with one hand, grasp the ankle with the other, and swing the lower leg—medially for external rotation at the hip and laterally for internal rotation.

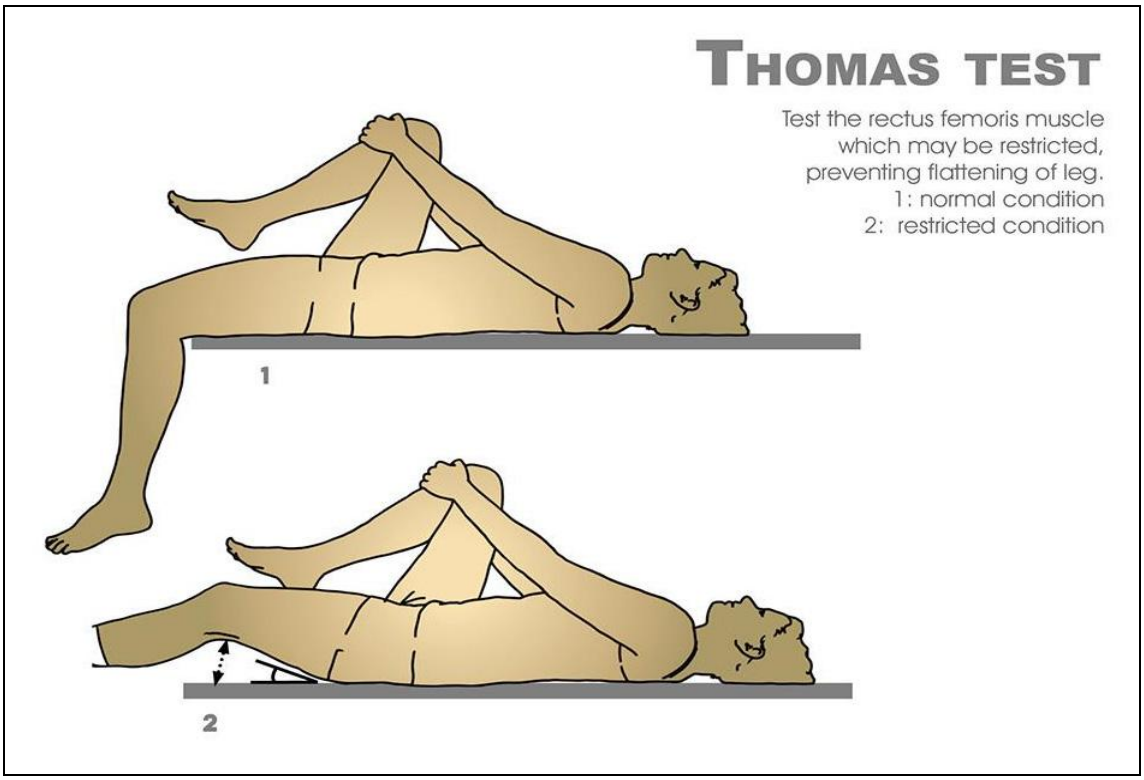
Internal rotation

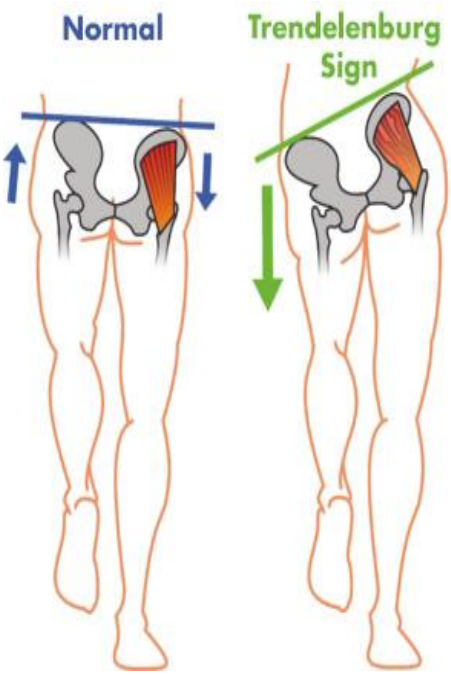


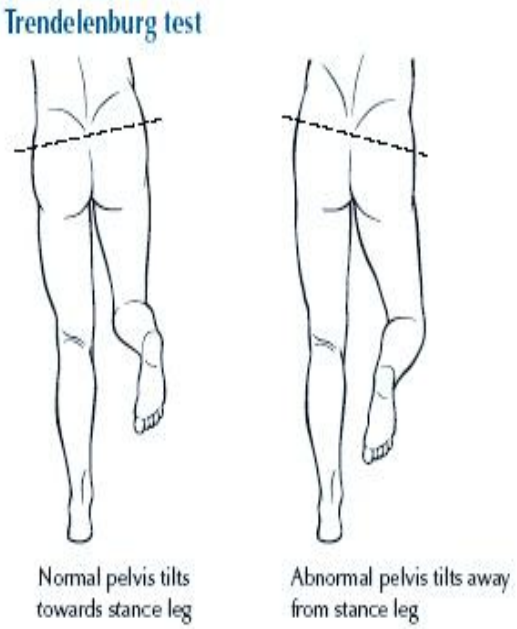
External rotation



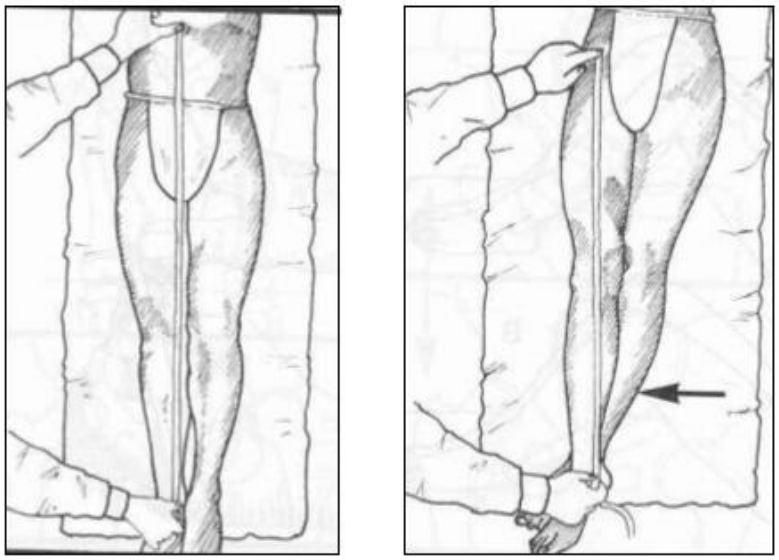
SPECIAL TECHNIQUES







Special Tests - Leg length assessment

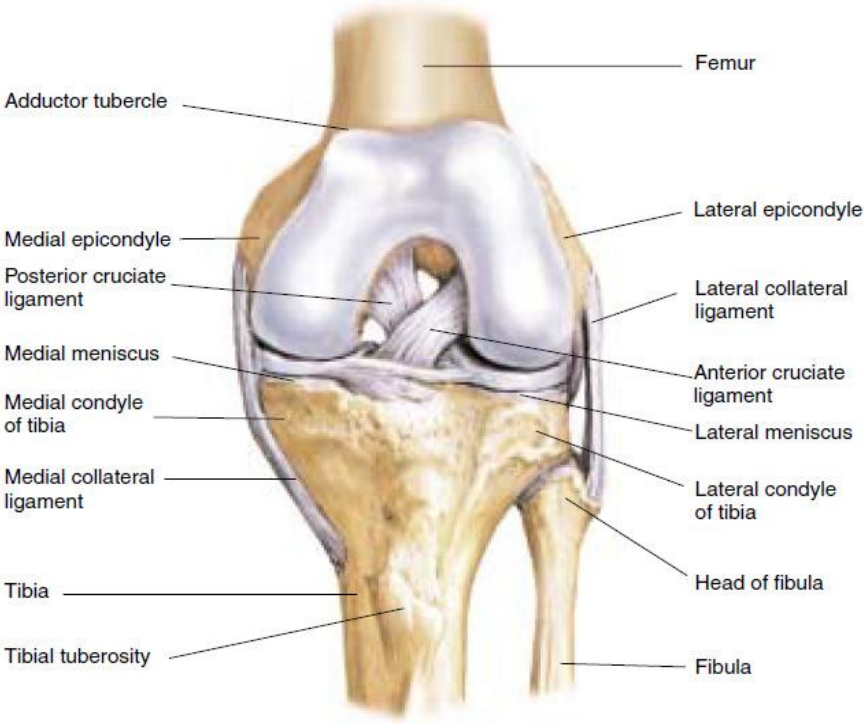


Apparent Length
Midpoint to Medial Malleolus

True Length
ASIS to Medial Malleolus

The Knee Joint

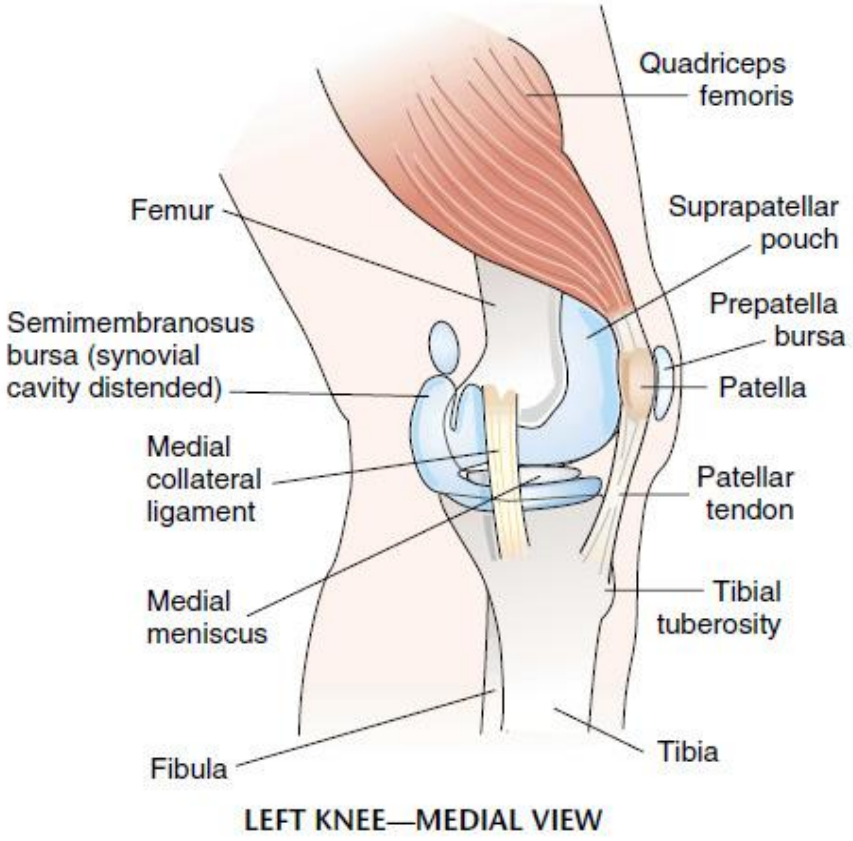
- The knee joint is the largest joint in the body. It involves three bones: the femur, the tibia, and the patella (or knee cap), with three articular surfaces, two between the femur and the tibia and one between the femur and the patella.



ANTERIOR ASPECT OF THE KNEE

Joints.

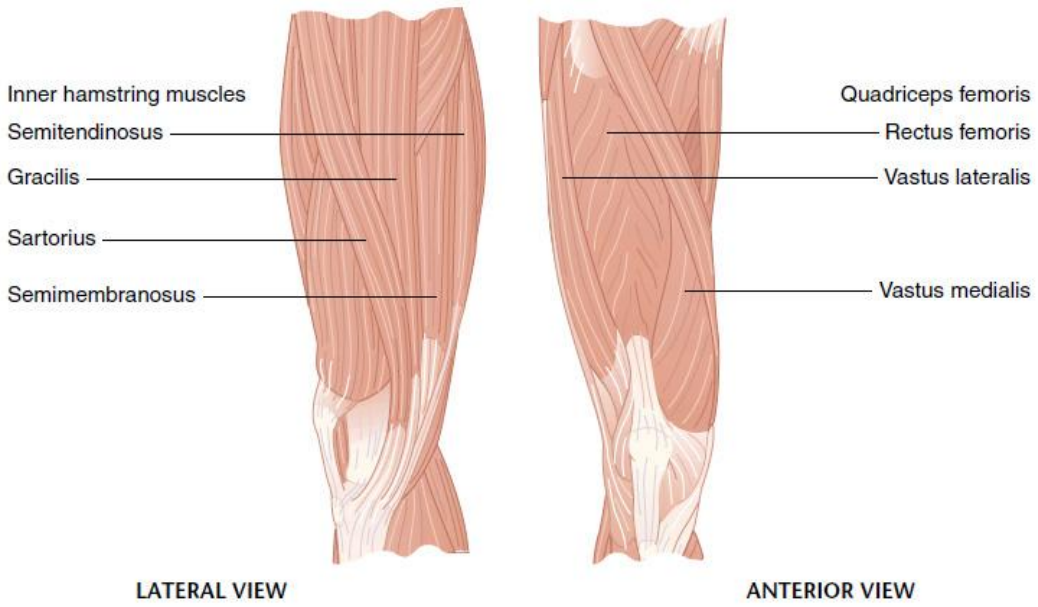
- Two condylar tibiofemoral joints are formed by the convex curves of the medial and lateral condyles of the femur as they articulate with the concave condyles of the tibia. The third articular surface is the patellofemoral joint. The patella slides in a groove on the anterior aspect of the distal femur, called the trochlear groove, during flexion and extension of the knee.



LEFT KNEE—MEDIAL VIEW

Muscle Groups.

- Powerful muscles move and support the knee. The quadriceps femoris extends the leg, covering the anterior, medial, and lateral aspects of the thigh. The hamstring muscles lie on the posterior aspect of the thigh and flex the knee.



Additional Structures.

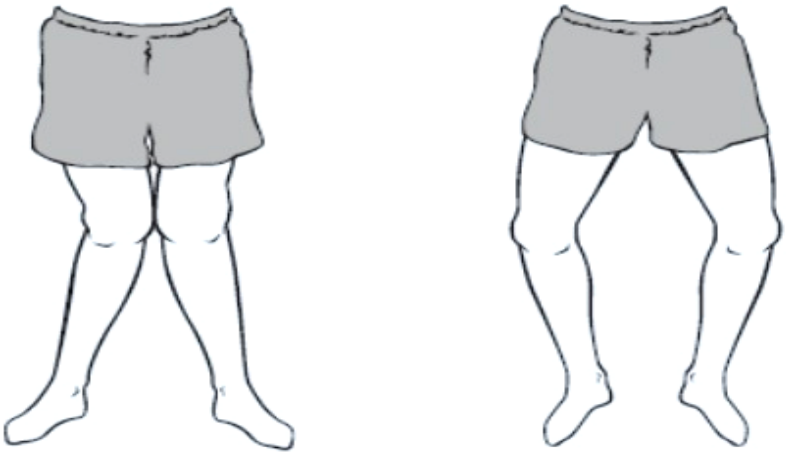
Two important pairs of ligaments, the collateral ligaments and the cruciate ligaments, and the menisci provide stability to the knee.

Technique Of Examination

INSPECTION

- Observe the gait for a smooth, rhythmic flow as the patient enters the room. The knee should be extended at heel strike and flexed at all other phases of swing and stance.
- Check the alignment and contours of the knees. Observe any atrophy of the quadriceps muscles.
- Look for loss of the normal hollows around the patella, a sign of swelling in the knee joint and suprapatellar pouch; note any other swelling in or around the knee.

Valgus and varus angulation



PALPATION

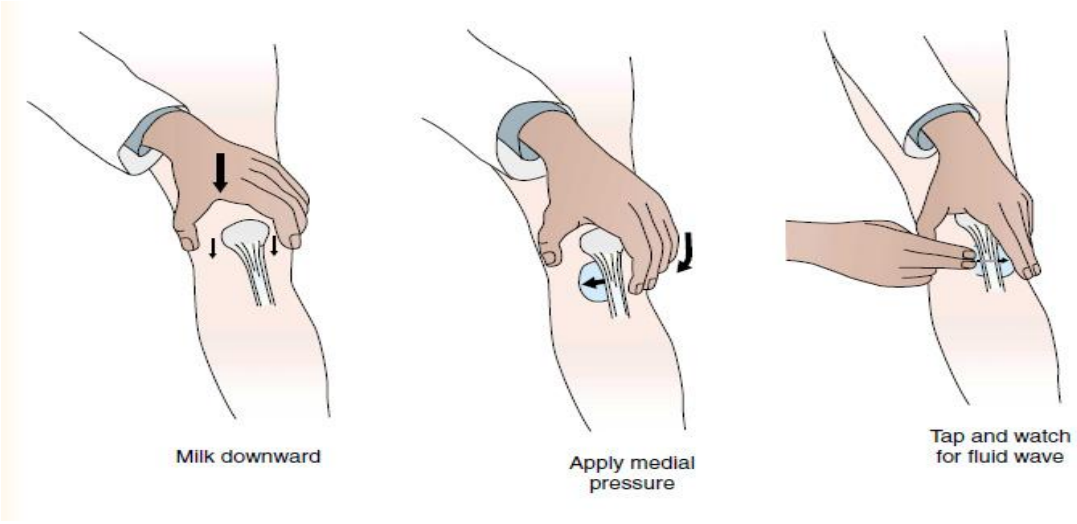
- First review the important bony landmarks of the knee. Facing the knee, place your thumbs in the soft-tissue depressions on either side of the patellar tendon. On the medial aspect, move your thumb upward and then downward and identify the medial femoral condyle and the upper margin of the medial tibial plateau. Trace the patellar tendon distally to the tibial tubercle. The adductor tubercle is posterior to the medial femoral condyle.
- Lateral to the patellar tendon, identify the lateral femoral condyle and the lateral tibial plateau. The medial and lateral femoral epicondyles are lateral to the condyles with the knee in flexion. Locate the patella.
- With the patient supine and the knee extended, push the patella against the underlying femur. Ask the patient to tighten the quadriceps as the patella moves distally in the trochlear groove. Check for a smooth sliding motion (the patellofemoral grinding test).
- Try to feel any thickening or swelling in the suprapatellar pouch and along the sides of the patella. Start 10 centimeters above the superior border of the patella (well above the pouch) and feel the soft tissues between your thumb and fingers. Move your hand distally in progressive steps, trying to identify the pouch. Continue your palpation along the sides of the patella. Note any tenderness or warmth greater than in the surrounding tissues.

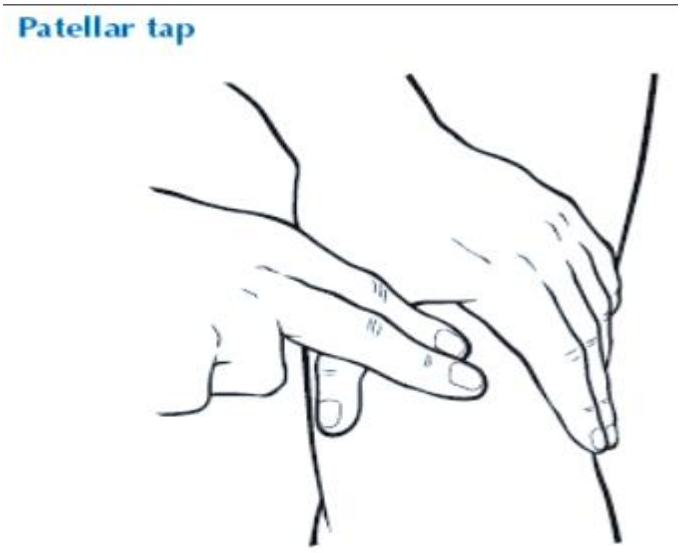
Three further tests will help you detect fluid in the knee joint.

1) **The Bulge Sign** (for minor effusions). With the knee extended, place the left hand above the knee and apply pressure on the suprapatellar pouch, displacing or “milking” fluid downward. Stroke downward on the medial

aspect of the knee and apply pressure to force fluid into the lateral area. Tap the knee just behind the lateral margin of the patella with the right hand.

2) **The Balloon Sign** (for major effusions). Place the thumb and index finger of your right hand on each side of the patella; with the left hand, compress the suprapatellar pouch against the femur. Feel for fluid entering (or ballooning into) the spaces next to the patella under your right thumb and index finger.
3) **Ballotting the patella**. To assess large effusions, you can also compress the suprapatellar pouch and "ballotte" or push the patella sharply against the femur. Watch for fluid returning to the suprapatellar pouch.



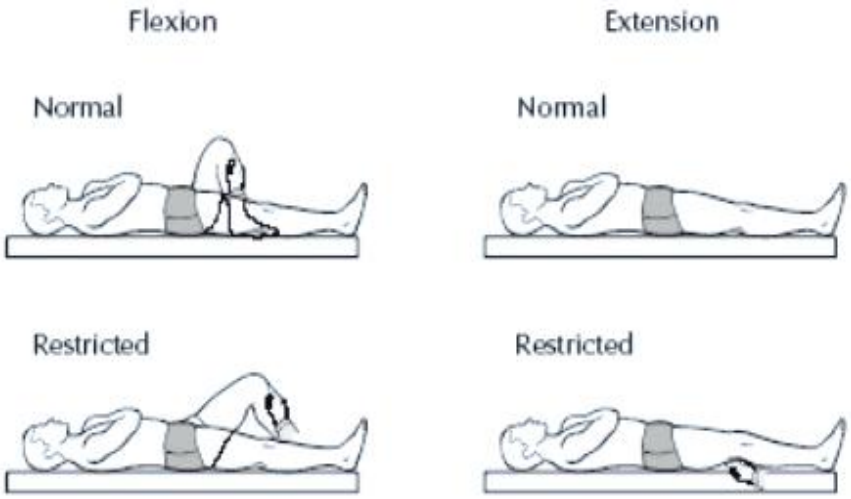


RANGE OF MOTION AND MANEUVERS

- The principal movements of the knee are flexion, extension, and internal and external rotation. Ask the patient to flex and extend the knee while sitting. To check internal and external rotation, instruct the patient to

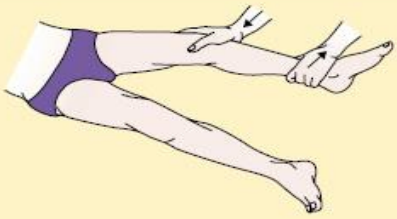
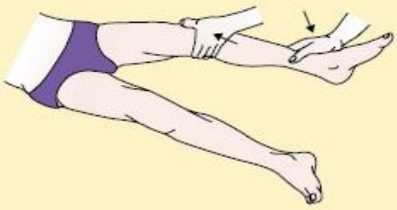
rotate the foot medially and laterally. Knee flexion and extension can also be assessed by asking the patient to squat and stand up—provide support if needed to maintain balance.

Flexion, extension

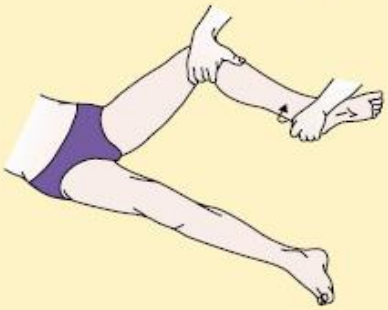


Collateral ligament test

Techniques for Examining the Knee

Structure		Maneuver
Medial collateral ligament (MCL)		*Abduction Stress Test.* With the patient supine and the knee slightly flexed, move the thigh about 30° laterally to the side of the table. Place one hand against the lateral knee to stabilize the femur and the other hand around the medial ankle. Push medially against the knee and pull laterally at the ankle to open the knee joint on the medial side (*valgus stress*).
Lateral collateral ligament (LCL)		*Adduction Stress Test.* Now, with the thigh and knee in the same position, change your position so you can place one hand against the medial surface of the knee and the other around the lateral ankle. Push medially against the knee and pull laterally at the ankle to open the knee joint on the lateral side (*varus stress*).
Anterior cruciate ligament (ACL)		*Anterior Drawer Sign.* With the patient supine, hips flexed and knees flexed to 90° and feet flat on the table, cup your hands around the knee with the thumbs on the medial and lateral joint line and the fingers on the medial and lateral insertions of the hamstrings. Draw the tibia forward and observe if it slides forward (like a drawer) from under the femur. Compare the degree of forward movement with that of the opposite knee.

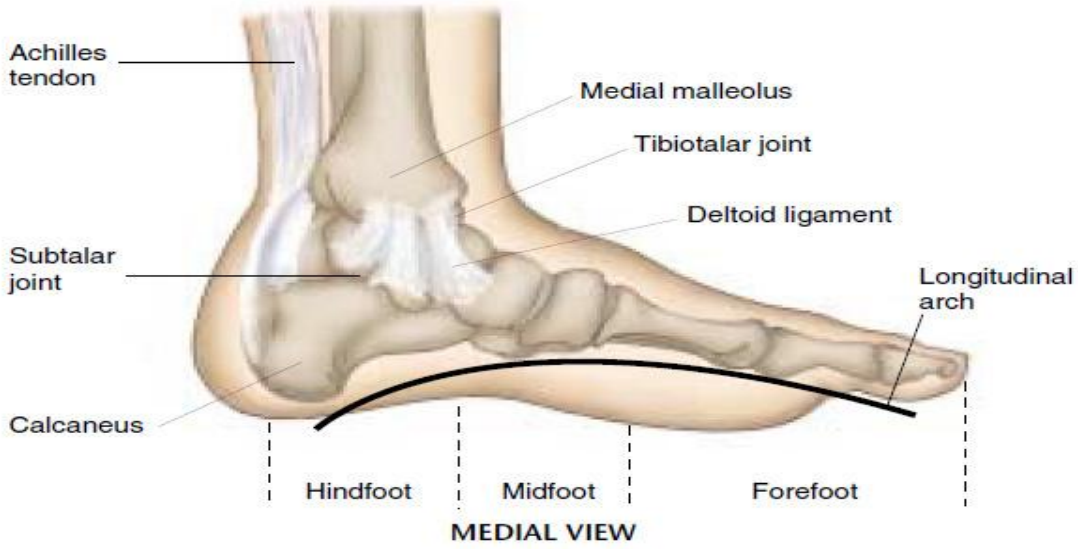
Techniques for Examining the Knee (Continued)

Structure		Maneuver
		*Lachman Test.* Place the knee in 15° of flexion and external rotation. Grasp the distal femur with one hand and the upper tibia with the other. With the thumb of the tibial hand on the joint line, simultaneously move the tibia forward and the femur back. Estimate the degree of forward excursion.
Posterior cruciate ligament (PCL)		*Posterior Drawer Sign.* Position the patient and place your hands in the positions described for the anterior drawer test. Push the tibia posteriorly and observe the degree of backward movement in the femur.
Medial meniscus and lateral meniscus		*McMurray Test.* If a click is felt or heard at the joint line during flexion and extension of the knee, or if tenderness is noted along the joint line, further assess the meniscus for a posterior tear. With the patient supine, grasp the heel and flex the knee. Cup your other hand over the knee joint with fingers and thumb along the medial and lateral joint line. From the heel, rotate the lower leg internally and externally. Then push on the lateral side to apply a valgus stress on the medial side of the joint. At the same time, rotate the leg externally and slowly extend it.

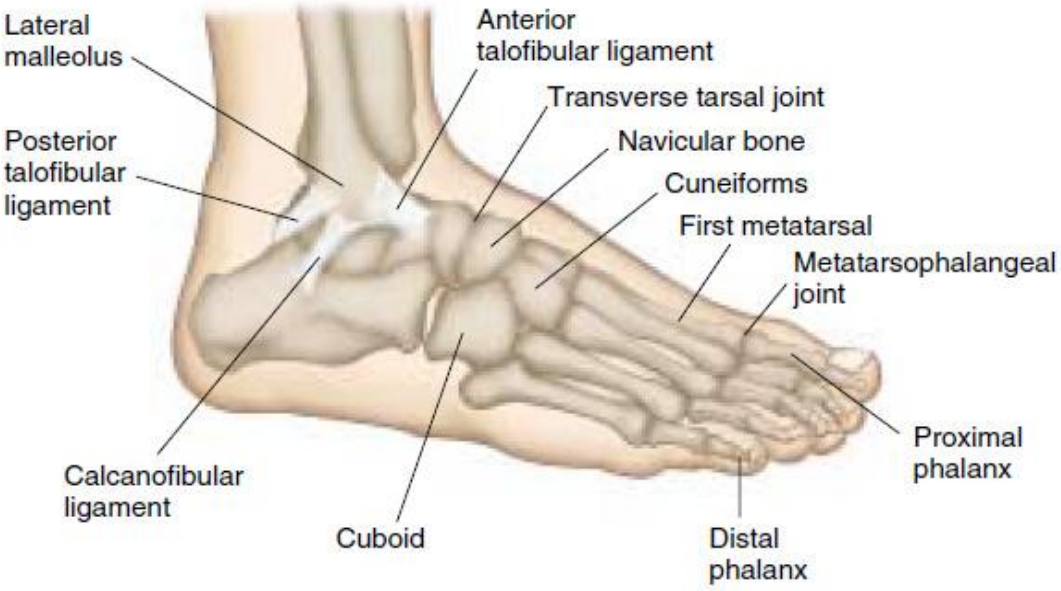
THE ANKLE AND FOOT

- The total weight of the body is transmitted through the ankle to the foot. The ankle and foot must balance the body and absorb the impact of the heel strike and gait.
- The ankle is a hinge joint formed by the tibia, the fibula, and the talus.
- The principal joints of the ankle are the tibiotalar joint, between the tibia and the talus, and the subtalar (talocalcaneal) joint.
- An imaginary line, the longitudinal arch, spans the foot, extending from

the calcaneus of the hind foot along the tarsal bones of the midfoot to the forefoot metatarsals and toes.



MEDIAL VIEW



LATERAL VIEW

Muscle Groups and Additional Structures.

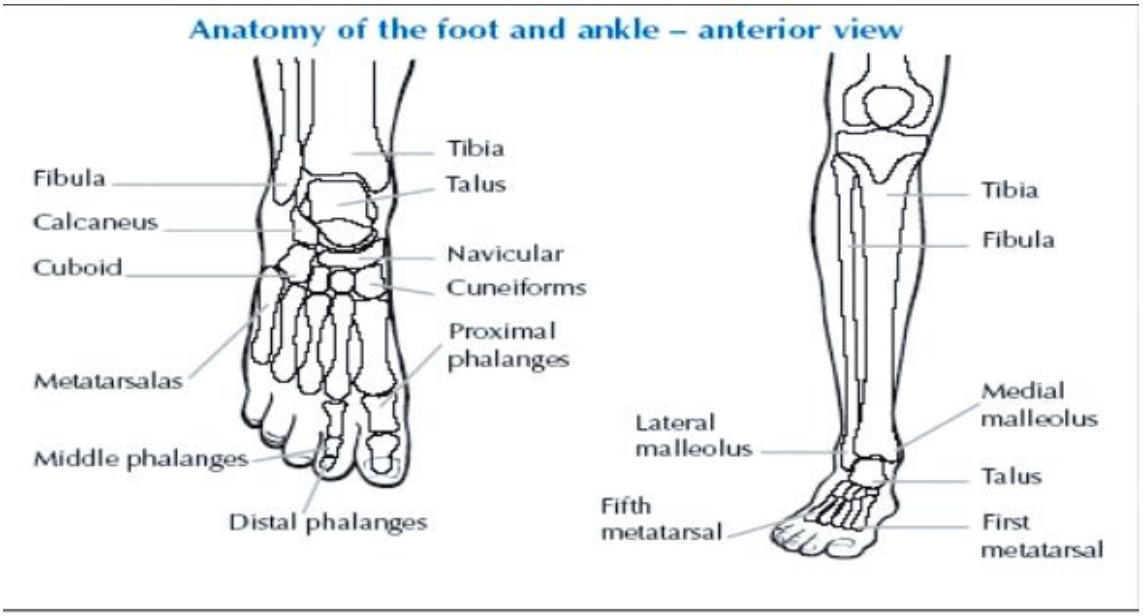
- Movement at the ankle joint is limited to dorsiflexion and plantar flexion. Plantar flexion is powered by the gastrocnemius, the posterior tibial muscle, and the toe flexors. Their tendons run behind the malleoli. The dorsiflexors include the anterior tibial muscle and the toe extensors. They lie prominently on the anterior surface, or dorsum, of the ankle, anterior to the malleoli.

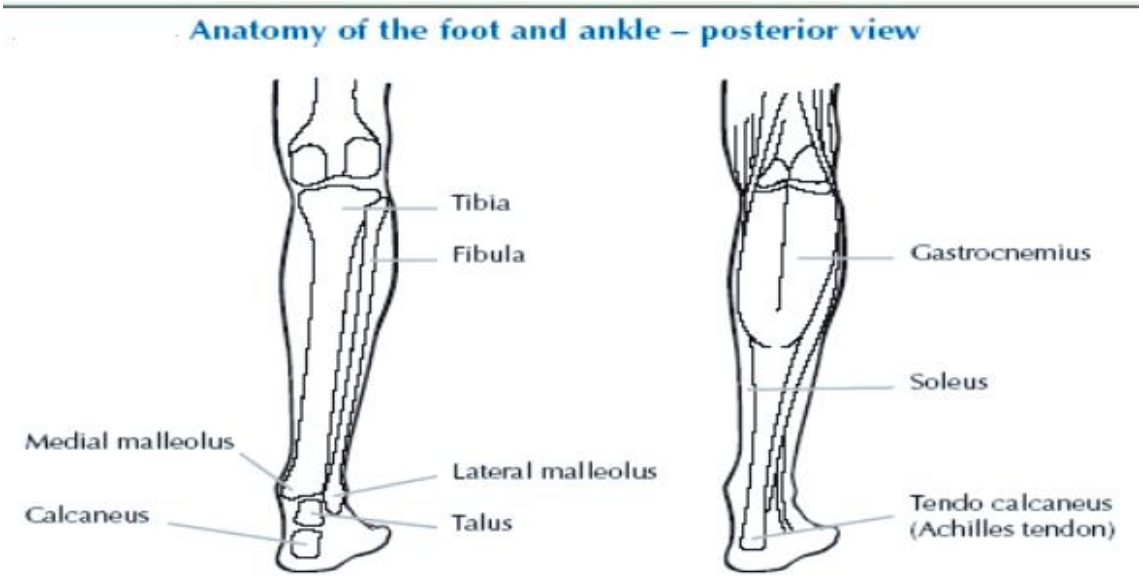
- Ligaments extend from each malleolus onto the foot. Medially, the triangle-shaped deltoid ligament fans out from the inferior surface of the medial malleolus to the talus and proximal tarsal bones, protecting against stress from eversion.

Technique Of Examination

INSPECTION

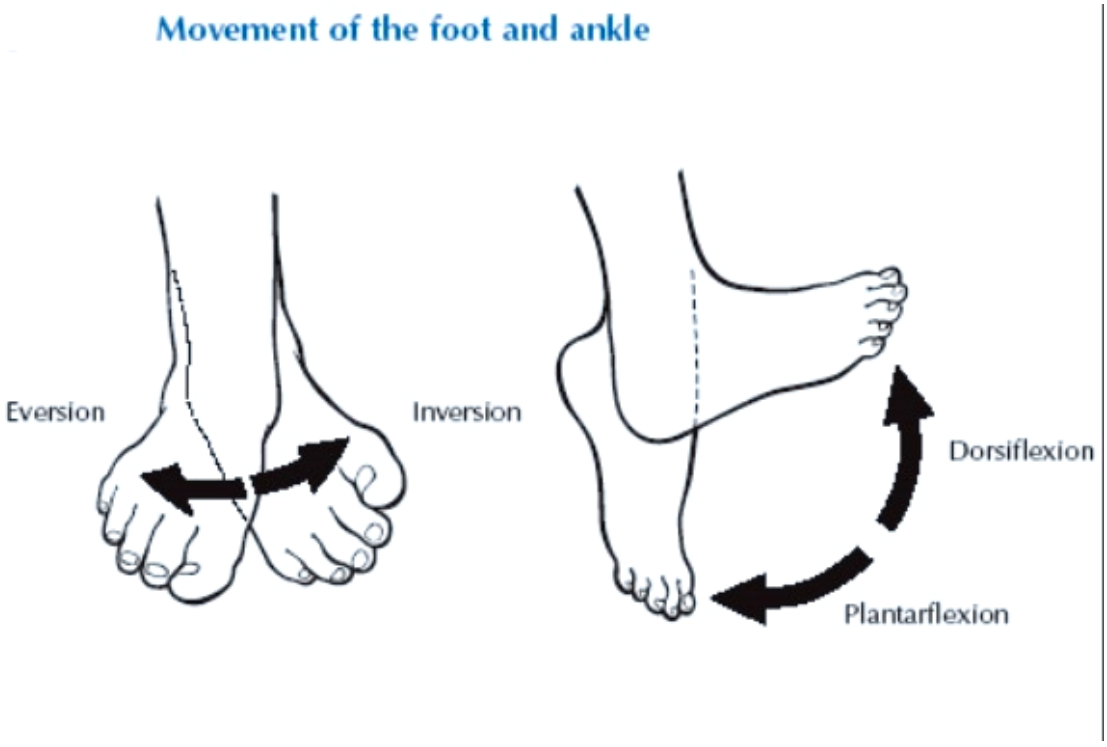
Observe all surfaces of the ankles and feet, noting any deformities, nodules, or swellings, and any calluses or corns.





PALPATION

- With your thumbs, palpate the anterior aspect of each ankle joint, noting any bogginess, swelling, or tenderness.
- Feel along the Achilles tendon for nodules and tenderness.
- Palpate the heel, especially the posterior and inferior calcaneus, and the plantar fascia for tenderness.
- Palpate the metatarsophalangeal joints for tenderness. Compress the forefoot between the thumb and fingers. Exert pressure just proximal to the heads of the 1st and 5th metatarsals.
- Palpate the heads of the five metatarsals and the grooves between them with your thumb and index finger. Place your thumb on the dorsum of the foot and your index finger on the plantar surface.

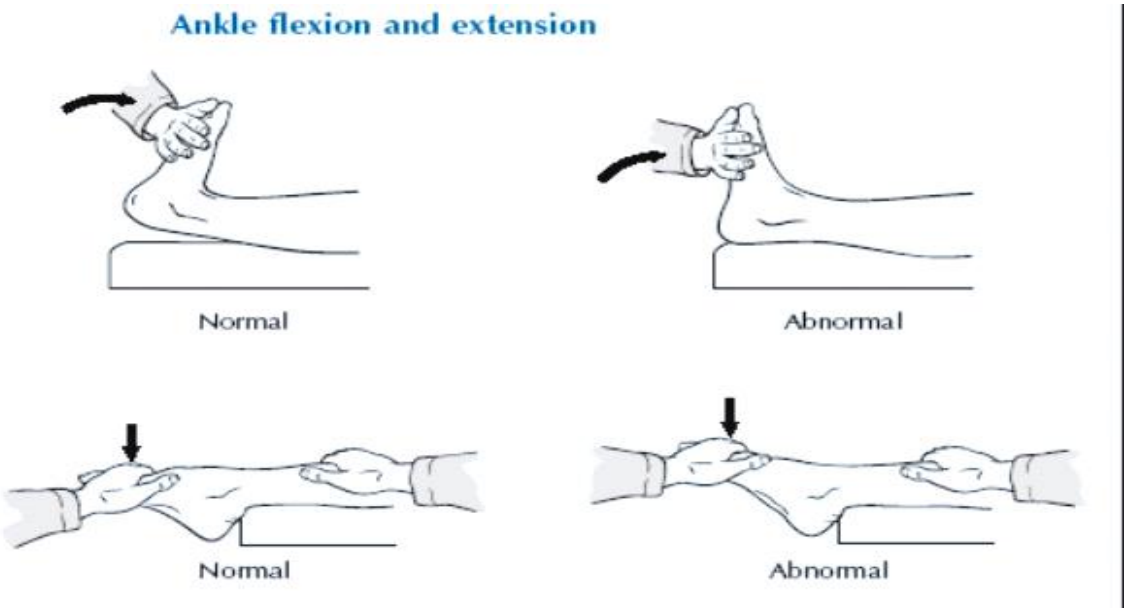


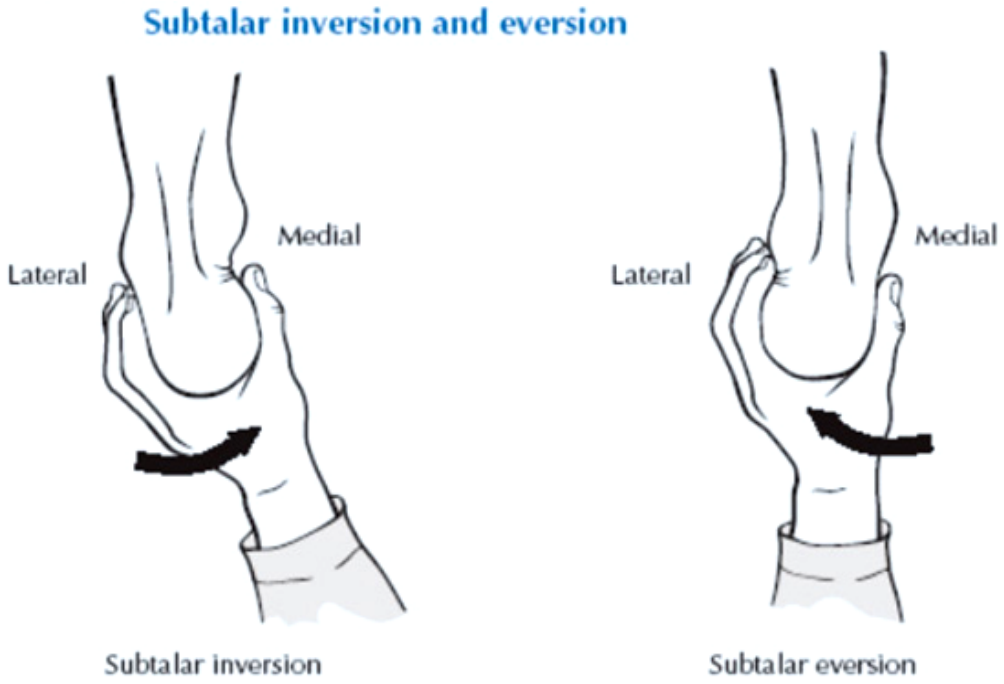
RANGE OF MOTION AND MANEUVERS

- Range of motion at the ankle includes flexion and extension at the ankle (tibiotalar) joint and, in the foot, inversion and eversion at the subtalar and transverse tarsal joints.
- The Ankle (Tibiotalar) Joint. Dorsiflex and plantar flex the foot at the ankle.
- The Subtalar (Talocalcaneal) Joint. Stabilize the ankle with one hand, grasp the heel with the other, and invert and evert the foot.
- The Transverse Tarsal Joint. Stabilize the heel and invert and evert the

forefoot.

- For the Metatarsophalangeal joints, flex the toes in relation to the feet.





Palpation of the second metatarsolphalanceal joint

Squeezing the MTPs

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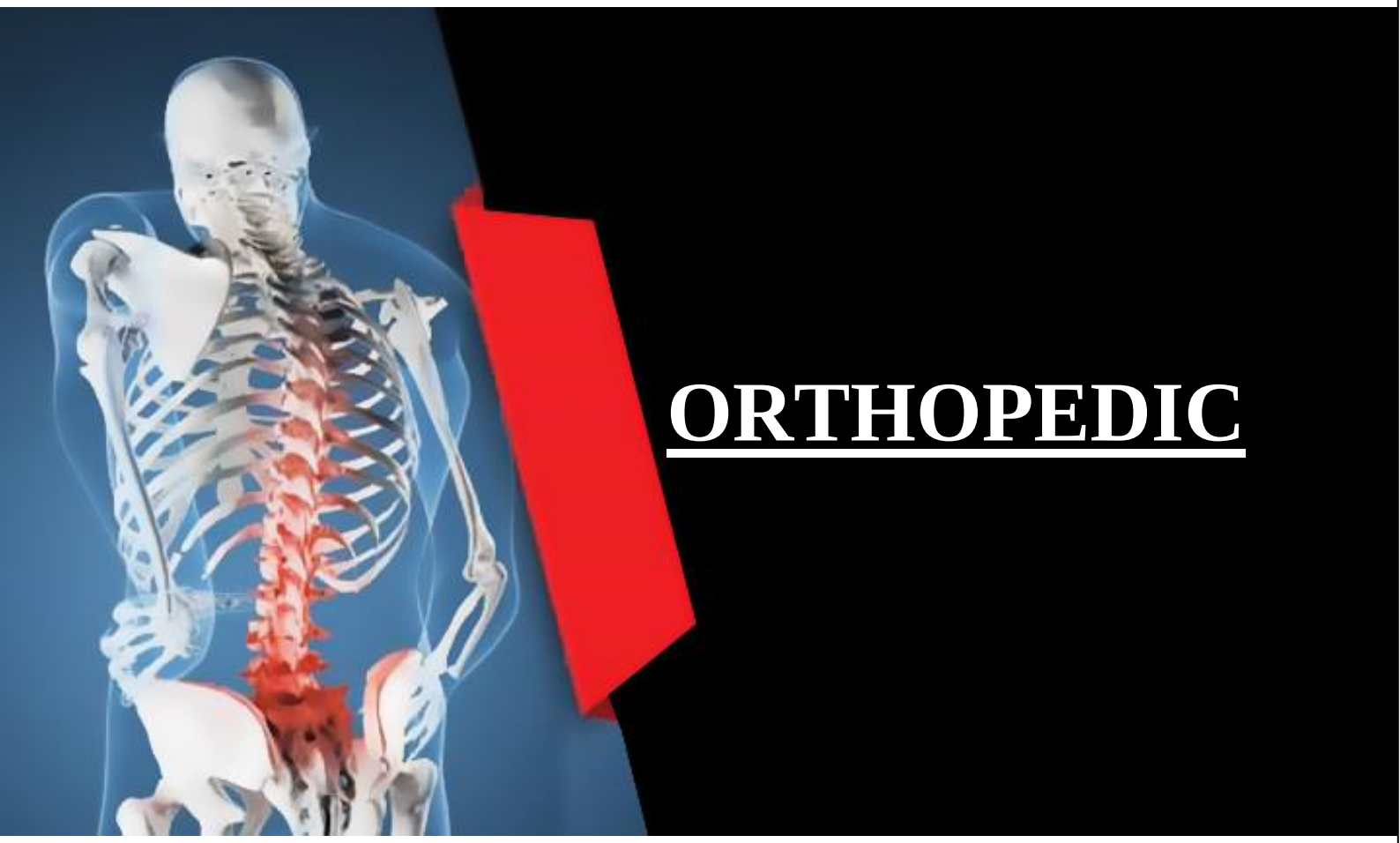


ORTHOPEDIC

Orthopedic Objectives:

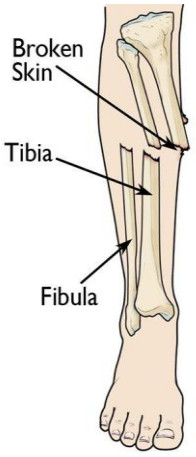
In the orthopedic part we will teach the students

- How to deal with a case of open fracture or compartment syndrome
- How to deal with a poly trauma case and/or spine fracture
- How to diagnose fractures of upper and lower limbs
- How to diagnose common nerve entrapment
- How to manage arthritis, osteomyelitis and different types of bursitis cases
- How to diagnose bone tumors

Clinical part of this orthopedic module will be about

- Reading X rays

Open Fractures



- Open fractures are fractures with direct communication to the external environment.
- Diagnosis is made clinically by assessing the size and nature of the external wound.
- Treatment depends on location of fracture but generally requires immediate IV antibiotics and urgent irrigation and debridement followed by surgical fixation as needed.
- **Atiology:**

  High energy trauma
- **Clinical Features:**

  Patients will present with pain, swelling, and deformity, with an overlying wound or punctum (in severe cases, the bone end may be visible protruding from the wound).

  On examination, ensure to check neurovascular status and overlying skin for any skin or tissue loss
- **Gustilo Classification:**
  - Type I
    - wound ≤1 cm

- Type II
  - wound 1-10 cm

- Type IIIA
  - wound usually >10 cm, high energy, extensive soft-tissue damage, contaminated
  - adequate tissue for flap coverage

- Type IIIB
  - extensive periosteal stripping, wound requires soft tissue coverage.

- Type IIIC
  - vascular injury requiring vascular repair

Imaging:

- X-ray of affected limb including joint above and below fracture
- CT for peri-articular injuries

Treatment:

Nonoperative:

-urgent IV antibiotics, tetanus prophylaxis, and extremity stabilization and dressing

Antibiotics (best results if administrated within 3 hours)

- Gustillo Grade I and II
  - 1st generation cephalosporin
- Gustillo Grade III
  - 1st generation cephalosporin + aminoglycoside

Operative:

- Irrigation and debridement , temporary fracture stabilization , local antibiotic administration and soft tissue coverage

- definitive reconstruction and fracture fixation BY external or internal fixation

Polytrauma management

- Trauma is a major public health problem with high disability, death, and societal cost
- Three peak times of death after trauma
  - 50% within the first minutes of sustaining the injury
    - caused by massive blood loss or neurologic injury
  - 30% within the first few days
    - most commonly from shock, hypoxia, or neurologic injury
  - 20% within days to weeks following injury
    - multi system organ failure and infection are leading causes
- Golden Hour
  - period of time when life threating and limb threatening injuries should be treated in order to decrease mortality
  - estimated 60% of preventable deaths can occur during this time ranging from minutes to hours
- Use of an airbag in a head-on collision significantly decreases the rate of
  - closed head injuries

- facial fractures
- thoracoabdominal injuries
- need for extraction

- Evaluation
  - **Primary survey**
    - treat greatest threats to life first
      - pelvic fractures can be life threatening if not intervened on by orthopedic surgeons
    - brief history
    - ABCDE's
      - Airway
        - includes cervical spine control
      - Breathing and ventilation
      - Circulation
        - includes hemorrhage control and resuscitation (below)
      - Disability
      - Exposure
  - **Secondary survey**
    - physical hair to toe examination and updated history
    - obtain indicated imaging studies
    - spine examination:-
  - examine face and scalp for evidence of direct trauma
  - inspect for angular or rotational deformities in the holding position of the patient's head
  - palpate posterior cervical spine looking for tenderness along the midline or paraspinal tissues
  - log roll patient to inspect and palpate entire spinal axis
  - perform careful neurologic exam
  - **Tertiary survey**
    - repeat physical examination and additional imaging as indicated when mental status has stabilized
    - formal tertiary survey decreases chances of missed orthopedic injury

Compartment Syndrome

Compartment Syndrome is a devastating extremity condition where the osseofascial compartment pressure rises to a level that decreases perfusion and may lead to irreversible muscle and neurovascular damage.

Diagnosis is made with the presence of severe and progressive pain that worsens with passive ankle motion. Firmness and decreased compressibility of the compartments is often present. Needle compartment pressures are diagnostic in cases of inconclusive physical exam findings and in sedated patients.

Treatment is usually emergent fasciotomies of all compartments.

Anatomic location

compartment syndrome may occur anywhere that skeletal muscle is surrounded by fascia, but most commonly in leg forearm hand foot thigh and buttock

Etiology

a- trauma, fractures , crush injuries, contusions, gunshot wounds, tight casts, dressings, or external wrappings

b- extravasation of IV infusion

c-burns

d-bleeding disorders

e-arterial injury

Hand compartments

- hypothenar
- thenar
- adductor pollicis
- dorsal interosseous (x4)
- volar (palmar) interosseous (x3)

Compartments of the leg

Anterior, lateral, deep posterior and superficial posterior compartment

Cascade of events includes

a- local trauma and soft tissue destruction

b- bleeding and edema

c- increased interstitial pressure

d- vascular occlusion (decreased venous outflow relative to arterial inflow)

e- myoneural ischemia

Symptoms

a- pain out of proportion to the clinical situation is usually the first symptom.

b- may be absent in cases of nerve damage.

c- pain is difficult to assess in a polytrauma patient and impossible to assess in a sedated patient.

d- difficult to assess in children (unable to verbalize).

Physical exam

1- pain w/ passive stretch

Is most sensitive finding prior to onset of ischemia

2- paresthesia and hypoesthesia

indicative of nerve ischemia in affected compartment

3- paralysis

late finding

full recovery is rare in this case

4- palpable swelling

5- peripheral pulses absent

late finding

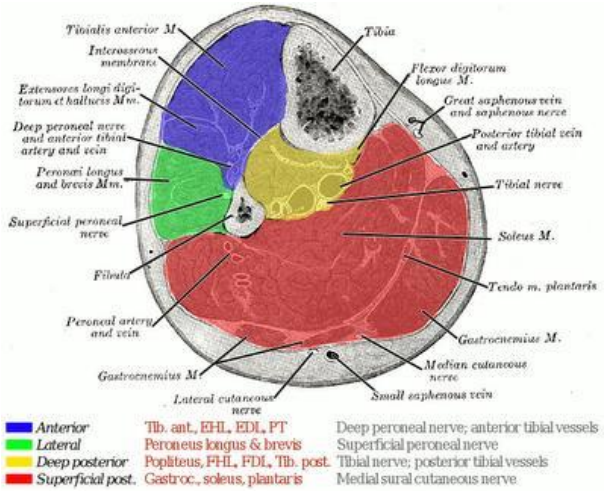
amputation usually inevitable in this case

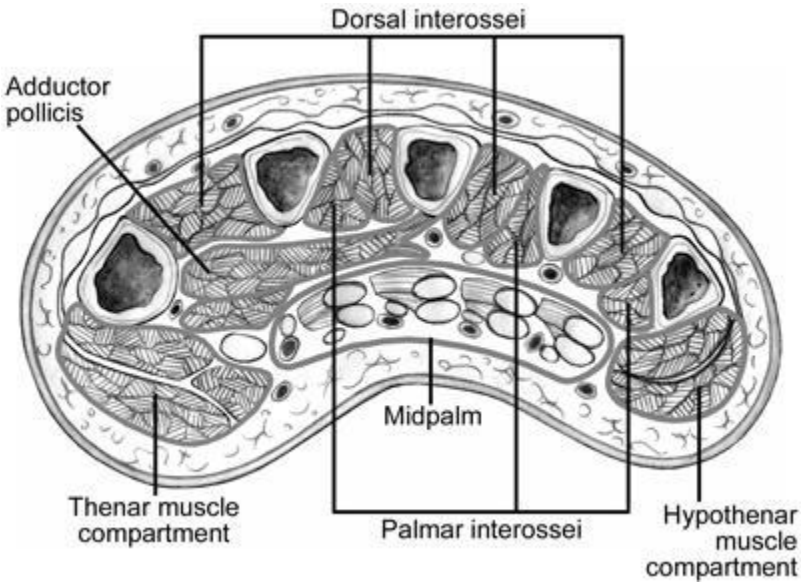
Diagnosis

is mainly Clinical based primarily on physical exam in patient with intact mental status

Treatment

Emergent fasciotomy of all four compartments





Clavicle Fractures

- Midshaft clavicle fractures are common traumatic injuries seen in young adults that occur in the middle third of the clavicle.
- Diagnosis can be made radiographically with shoulder radiographs.
- Treatment is controversial but may be nonoperative or operative based on patient activity demands and the degree of radiographic displacement and shortening

Atiology:

fall on an outstretched arm or direct trauma to the shoulder

Anatomy

Clinical picture:

pain

swelling

deformity

important to perform careful neurovascular examination

Classification

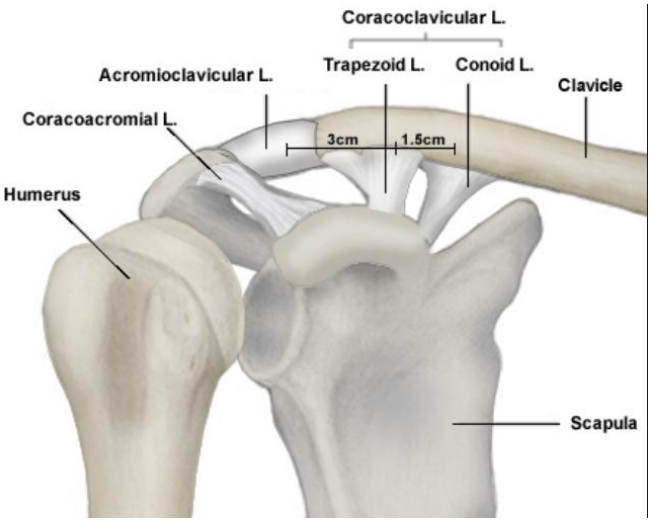
- **Mid shaft fractures**: 75-80% of all clavicle fractures will occur in the middle third segment
  - the junction of the outer and middle thirds is the thinnest part of the bone and is the only area not protected by or reinforced with muscle and ligamentous attachments

- it is therefore prone to fracture, particularly with axial loading
  - medial fragment: sternocleidomastoid muscle pulls the medial fragment posterosuperiorly
  - lateral fragment: pectoralis and weight of arm pull the lateral fragment inferomedially

Distal fractures

Can affect AC Joint stability which stabilized by the following

- acromioclavicular (AC) ligament provides anterior/posterior stability
- coracoclavicular (CC) ligaments (trapezoid and conoid) provides superior/inferior stability
- deltoid
- trapezius



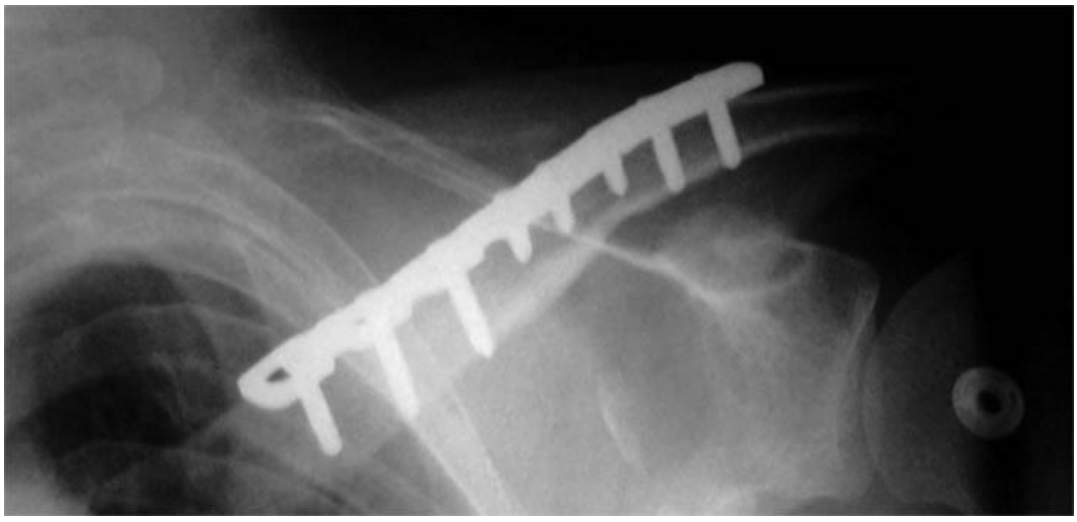
Neer Classification		
Nondisplaced	< 100% displacement	Nonoperative
Displaced	> 100% displacement	Operative

Imaging:

X-ray AP of the shoulder

Treatment

- According to :
- fracture location (e.g. extra-articular vs. intra-articular)
- fracture pattern (e.g. simple vs. comminuted)
- integrity of the coracoclavicular ligments
  - conoid (medial) and trapezoid (lateral) provide primary resistance to superior displacement of the lateral clavicle
- age (e.g. pediatric patients usually have an intact periosteal sleeve)



Nonoperative

- sling immobilization with gentle ROM exercises at 2-4 weeks

Operative

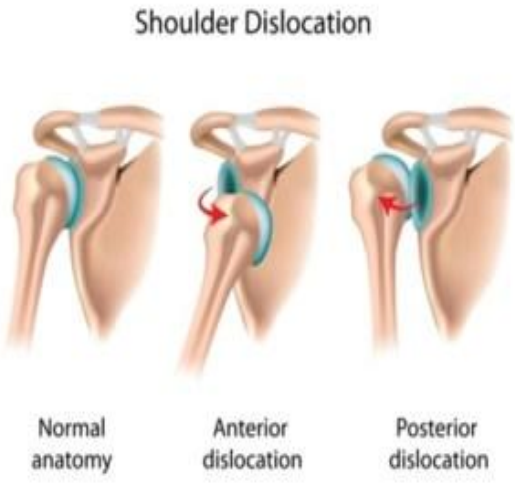
- closed reduction and intramedullary fixation vs. open reduction internal fixation

Shoulder dislocation:

- Traumatic Anterior shoulder instability, also referred to as TUBS

(Traumatic Unilateral dislocations with a Bankart lesion requiring Surgery), are traumatic shoulder injuries that generally occur as a result of an anterior force to the shoulder while its abduced and externally rotated and may lead to recurrent anterior shoulder instability.

- Posterior shoulder instability and dislocations are less common than anterior shoulder instability and dislocations and are commonly missed.
- Diagnosis is made clinically with presence of positive anterior instability provocative tests and confirmed with MRI studies that may reveal labrum and/or bony injuries of the glenoid and proximal humerus (Hill sachs).
- Treatment may be nonoperative or operative depending on chronicity of symptoms, recurrence of instability, and the severity of labrum and/or glenoid defects.



Types of shoulder dislocation:

1-Anterior dislocation (most common type)

2-Posterior dislocation (commonly seen after convulsions and electrical shock)

3-Inferior dislocation (rare)

Anatomy:

- Glenohumeral stability
  - Static restraints
    - glenohumeral ligaments
    - glenoid labrum
    - articular congruity
    - negative intraarticular pressure
  - Dynamic restraints
    - rotator cuff muscles

- the primary biomechanical role of the rotator cuff is stabilizing the glenohumeral joint by compressing the humeral head against the glenoid _
- biceps long head
- periscapular muscles

Mechanism of injury:

- anteriorly directed force on the arm when the shoulder is abducted and externally rotated in anterior type
- posteriorly directed force on the arm when the shoulder is adducted and internally rotated arm in posterior type

Clinical picture:

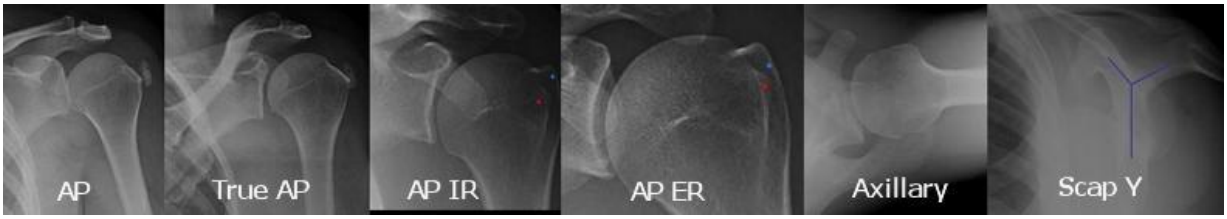
- traumatic event causing dislocation
- shoulder pain complaints
  - caused by subluxation and excessive translation of the humeral head on the glenoid
- limitation of movement

  limited external rotation for acute posterior dislocation

  shoulder locked in an internally rotated position common in undiagnosed posterior dislocations

Imaging:

- X-ray true AP, scapular Y and axillary views



- CT scan helpful for evaluation of bony injuries (hill sachs or glenoid defect)
- MRI best for visualization of labral tear (bankart lesion)

Treatment:

Nonoperative

- **acute reduction, immobilization, followed by physiotherapy**
- reduction
  - simple traction-countertraction is most commonly used
  - relaxation of patient with sedation is essential

Operative

Open or **Arthroscopic Bankart repair** for chronic recurrent cases

Humeral Shaft Fractures

- Proximal humerus fractures are common fractures often seen in older patients with osteoporotic bone following a ground-level fall on an outstretched arm.
- Humeral shaft fractures are common fractures of the diaphysis of the humerus, which may be associated with radial nerve injury.
- Diagnosis is made with orthogonal radiographs of the humerus.
- Treatment can be nonoperative or operative depending on location of fracture, fracture morphology, and association with other ipsilateral injuries.

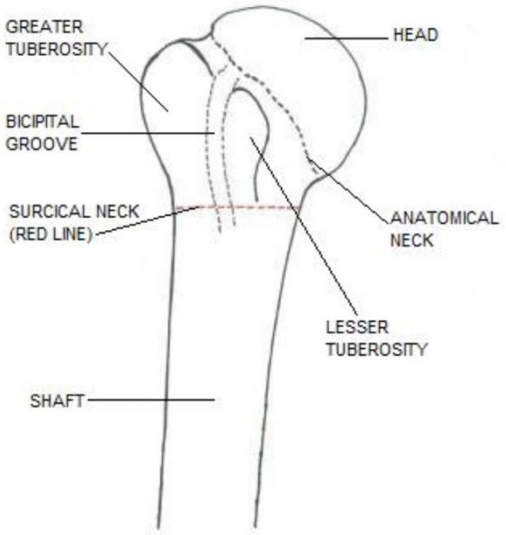
Atiology:

- young patients with high-energy trauma
- elderly patients with low-energy trauma

anatomy

- anatomic neck
  - represents the old epiphyseal plate
- surgical neck
  - represents the weakened area below head
  - more often involved in fractures than anatomic neck
- average neck-shaft angle is 135 degrees
- humeral shaft is cylindrical
- distally humerus becomes triangular
- trochlea
  - articulates with sigmoid notch
  - allows for flexion and extension
- capitellum
  - articulates with proximal radius
  - allows for forearm rotation
- axillary nerve injury may be injured with proximal fractures
  - determine function of deltoid muscle and lateral shoulder sensation
- radial nerve courses along spiral groove and may be injured with mid shaft fractures causing finger and wrist drop

- ulnar nerve
  - resides in the cubital tunnel in a subcutaneous position below the medial condyle



PARTS OF UPPER END OF HUMERUS

Classification

Proximal

- greater tuberosity
- lesser tuberosity
- articular surface
- shaft
- Fracture dislocation

Mid shaft

Distal:- supracondylar, lateral, medial condyle, trochlear , capitellar and or epicondylar fractures

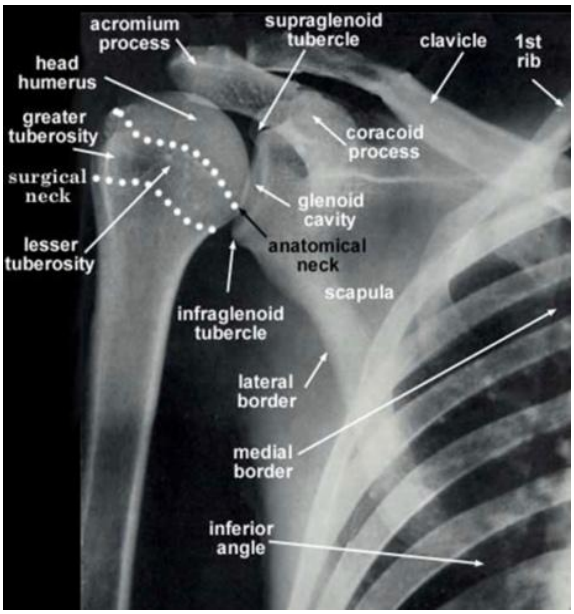
Clinical picture:

pain

swelling

deformity

important to perform careful neurovascular examination

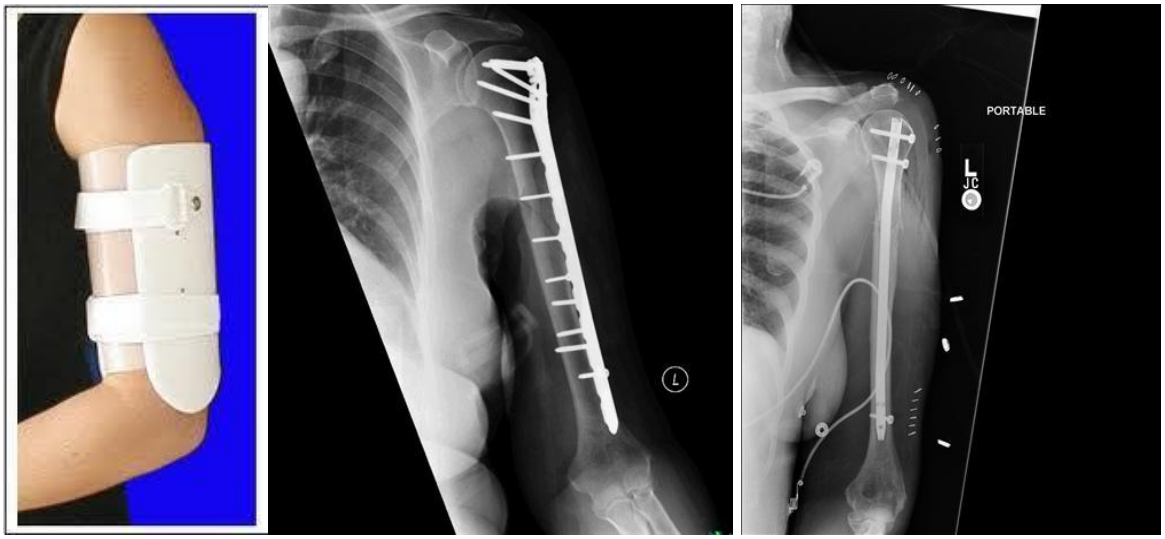


Imaging:

X-ray AP and lateral views

Ct scan for articular fractures

Treatment:



- Nonoperative: coaptation splint & Functional Bracing for non displaced fractures
- Operative: ORIF plate and screws or intramedullary nailing for open fracture, vascular injury requiring repair, brachial plexus injury, pathologic fractures, segmental fractures, ipsilateral forearm fracture (floating elbow) , polytrauma or compartment syndrome

Supracondylar Fracture – Pediatric

- Supracondylar Fractures are one of the most common traumatic fractures seen in children and most commonly occur in children 5-7 years of age from a fall on an outstretched hand.
- Diagnosis can be made with plain radiographs.
- Treatment is usually closed reduction and percutanous pinning (CRPP)

Atiology:

Fall on an outstretched hand.

Associated injuries

- anterior interosseous nerve (AIN) neurapraxia (branch of median n.)
- vascular compromise (5-17%)
- rich collateral circulation can maintain circulation despite vascular injury

Anatomy

Ossification centers of elbow

- age of ossification/appearance and age of fusion are two independent events that must be differentiated

Ossification Centers of the Elbow

Ossification Center	***Years at ossification*** *(appear on xray)*	***Years at fusion*** *(appear on xray)*	
Capitellum	1	12	
Radial Head	4	15	
Medial epicondyle	6	17	
Trochlea	8	12	
Olecranon	10	15	
Lateral epicondyle	12	12	

Clinical picture:

Pain

swelling

limitation of movement

gross deformity

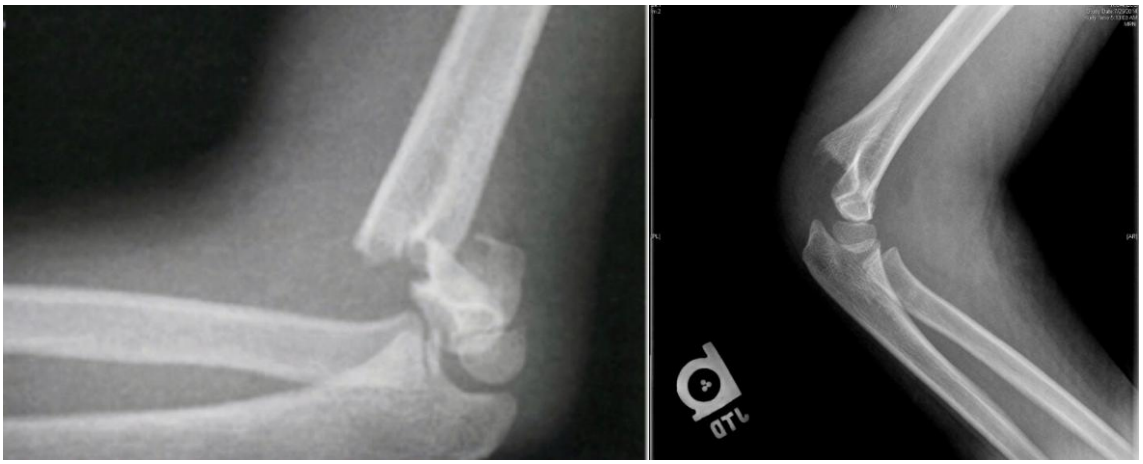
- neurovascular exam must be done before any reduction maneuver to be certain nerve or vascular injury :- Evaluate for
  - AIN neurapraxia
    - unable to flex the interphalangeal joint of the thumb and the distal interphalangeal joint of the index finger (can't make A-OK sign)
  - median nerve injury
    - loss of sensation over volar index finger
  - radial nerve neurapraxia
    - inability to extend wrist, MCP joints, thumb IP joint

- PIP and DIP can still be extended via intrinsic function (ulnar n.)

Classification

- extension type most common
- flexion type less common

Imaging:



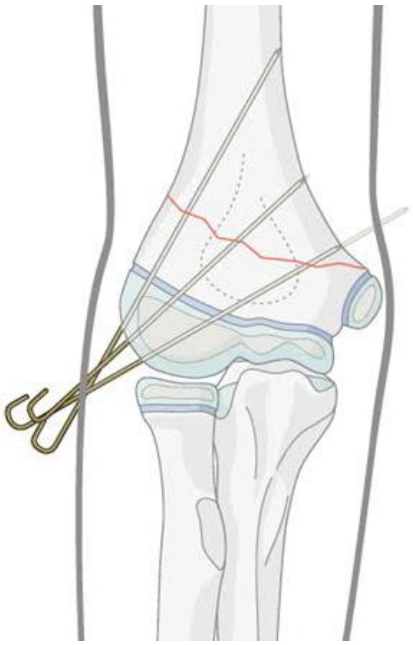
AP and lateral x-ray of the elbow

Treatment:

-Nonoperative: long arm casting if non displaced

-Operative:

- closed reduction and percutanous pinning (k wire fixation)



- open reduction, percutaneous pinning if failed closed reduction

Radius and Ulnar Shaft Fractures

- Radius and ulnar shaft fractures, also known as adult both bone forearm fractures, are common fractures of the forearm caused by either direct trauma or indirect trauma (fall).
- A Monteggia fracture is defined as a proximal 1/3 ulna fracture with an associated radial head dislocation
- Diagnosis is made by physical exam and plain orthogonal radiographs.
- Treatment is generally surgical open reduction and internal fixation with compression plating of both the ulna and radius fractures.

Atiology:

Radius and ulnar shaft fractures, also known as adult both bone forearm fractures, are common fractures of the forearm caused by either direct trauma or indirect trauma (fall).

Clinical picture:

pain and swelling

loss of forearm and hand function

gross deformity

neurovascular exam: assess radial and ulnar pulses

Imaging:

X-ray AP and lateral views of the forearm

Treatment:

- **cast immobilization**

  completely nondisplaced fractures in patients who are not surgical candidates
- Treatment is generally surgical open reduction and internal fixation with compression plating of both the ulna and radius fractures.

- Nonoperative: cast or brace immobilization
- Operative: ORIF plate and screws

  external fixation in severe soft tissue injury

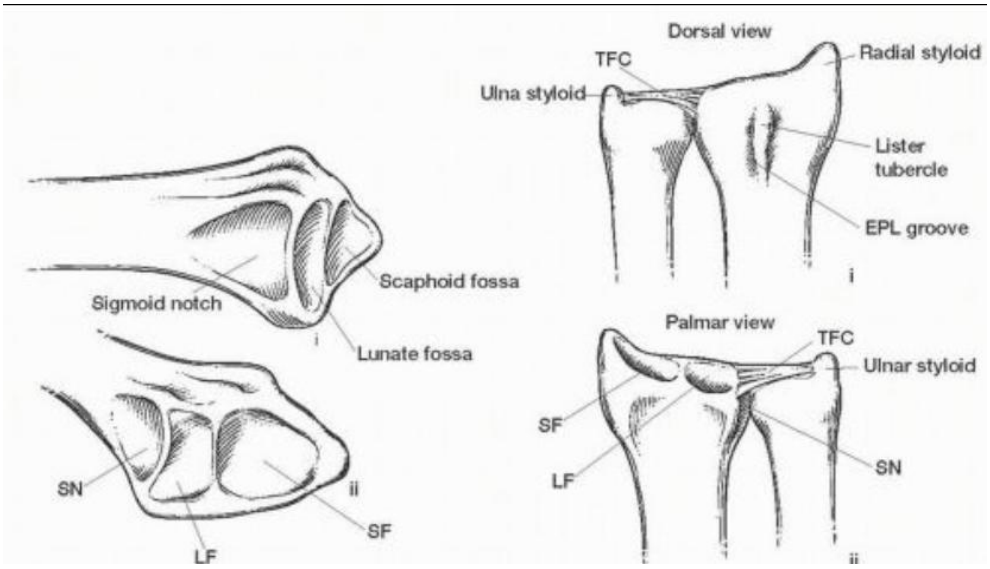
Distal Radius Fractures

Introduction

o Distal radius fractures are the most common orthopaedic injury and generally result from fall on an outstretched hand.

o Diagnosis is made clinically and radiographically with orthogonal radiographs of the wrist

o Treatment can be nonoperative or operative depending on fracture stability and fracture displacement as well as patient age and activity demands



Atiology:

fall on outstretched hand is most common in older population

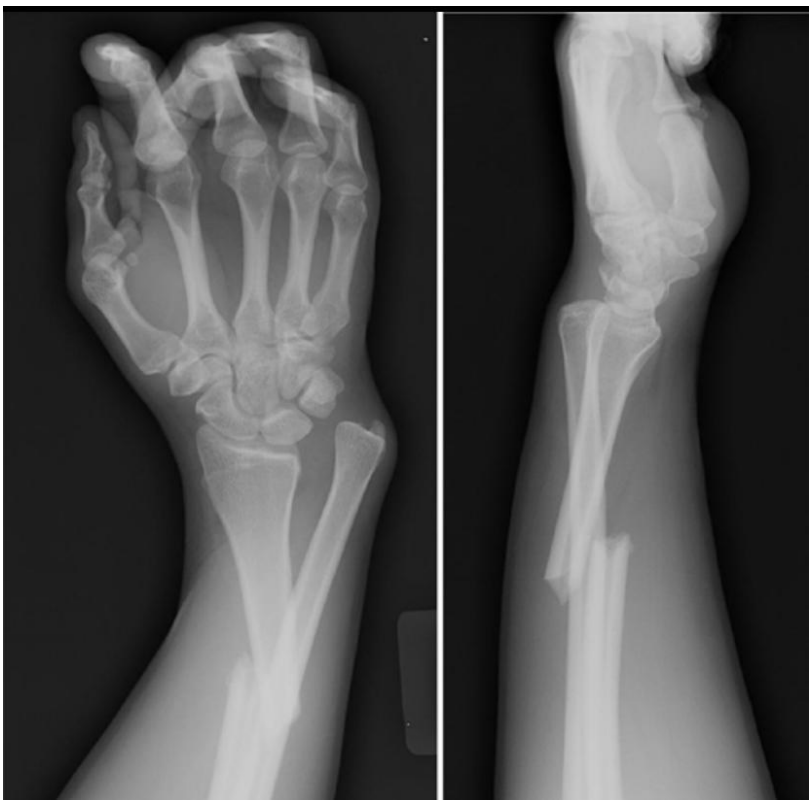
higher energy mechanism more common in younger patients

Classification:-

Can be classified into intra articular and extra articular fractures

Eponyms	
Barton's fx	Fracture-dislocation of radiocarpal joint with intra-articular fx involving the volar or dorsal lip (volar Barton or dorsal Barton fx)
Chauffer's fx	Radial styloid fx
Colles' fx	Low energy, dorsally displaced, extra-articular fx
Smith's fx	Low energy, volarly displaced, extra-articular fx

A galeazzi fracture is a distal 1/3 radial shaft fracture with an associated distal radioulnar joint (DRUJ) injury

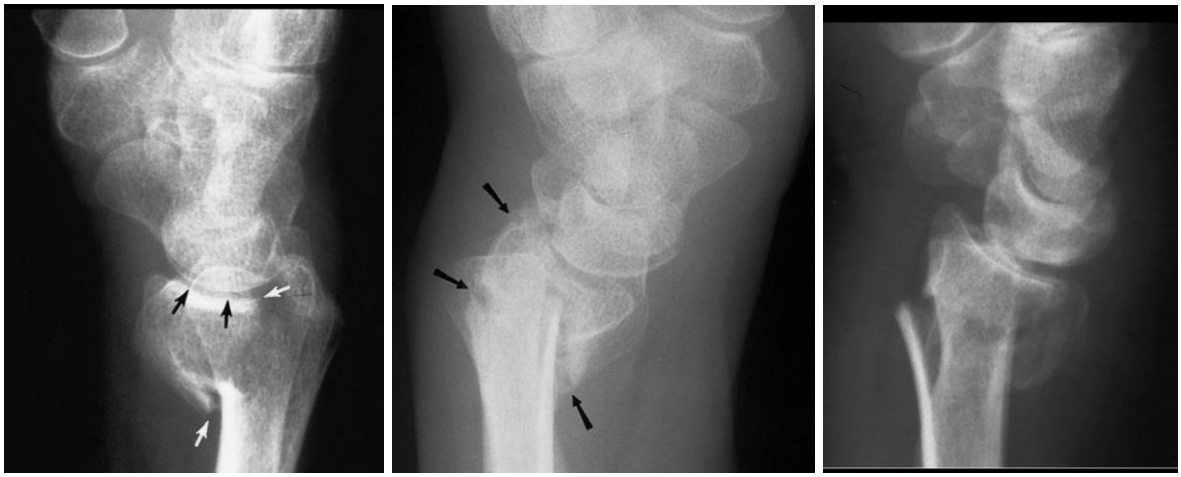


Clinical picture:

- wrist pain
- wrist swelling
- wrist deformity
- diffuse tenderness
- limitation of movement by pain

Imaging:

- X-ray AP,lateral &oblique views
- CT to evaluate intra-articular involvement



Treatment:

Treatment can be nonoperative or operative depending on fracture

stability and fracture displacement

- Nonoperative:

  closed reduction and splint/cast immobilization

- operative:

  - CRPP
  - ORIF

Nonoperative _

▪closed reduction and splint/cast immobilization

- indications
- extra-articular
- < 5mm radial shortening
- dorsal angulation < 5°
- Operative

▪CRPP

- ▪indications
- ▪extra-articular fractures

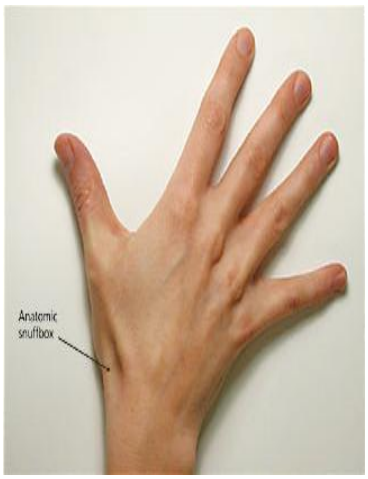
▪ORIF

- indications

  Unaccepted closed reduction

- displaced intra-articular fractures > 2mm
- comminuted fractures


Scaphoid Fracture




- Scaphoid Fractures are the most common carpal bone fracture, often occurring after a fall onto an outstretched hand.
- Diagnosis can generally be made by dedicated radiographs but CT or MRI may be needed for confirmation.
- Treatment may require a prolonged period of cast immobilization, percutaneous surgical fixation, or open reduction and internal fixation.

Anatomy: Blood supply

- major blood supply is dorsal carpal branch (branch of the radial artery)
  - enters scaphoid in a nonarticular ridge on the dorsal surface and supplies proximal 80% of scaphoid via retrograde blood flow
- minor blood supply from superficial palmar arch (branch of volar radial artery)

- enters distal tubercle and supplies distal 20% of scaphoid

Atiology: fall onto outstretched hand by axial load across a hyper-dorsiflexed, pronated wrist

Classification:- anatomic location

- waist -65%
- proximal third - 25%
- distal third - 10%

Clinical picture:

- pain with resisted pronation
- Swelling
- Limitation of movement
- anatomic snuffbox tenderness dorsally
- scaphoid tubercle tenderness volarly

Imaging:

- X-ray PA,lateral &scaphoid views (30 degree wrist extension, 20 degree ulnar deviation)

  if radiographs are negative (27%) and there is a high clinical suspicion then repeat radiographs in 14-21 days
- CT scan
- MRI for immediate identification of fractures and assessment of vascular status of bone

Treatment:

- **Nonoperative:**

  cast immobilization in stable non-displaced fracture (majority of waist fractures)
- **Operative:**
  - percutaneous screw fixation
  - open reduction internal fixation

Pelvic and acetabular fractures

Pelvic ring fractures are high energy (road traffic accidents or pedestrian crossing)fractures of the pelvic ring

Acetabulum fractures are pelvis fractures that involve the articular surface of the hip joint and may involve one or two columns, one or two walls, or the roof within the pelvis.

Diagnosis is made radiographically with pelvic radiographs and further characterized with CT scan.

Treatment is typically operative fixation depending on degree of pelvis instability, fracture displacement and patient activity demands.

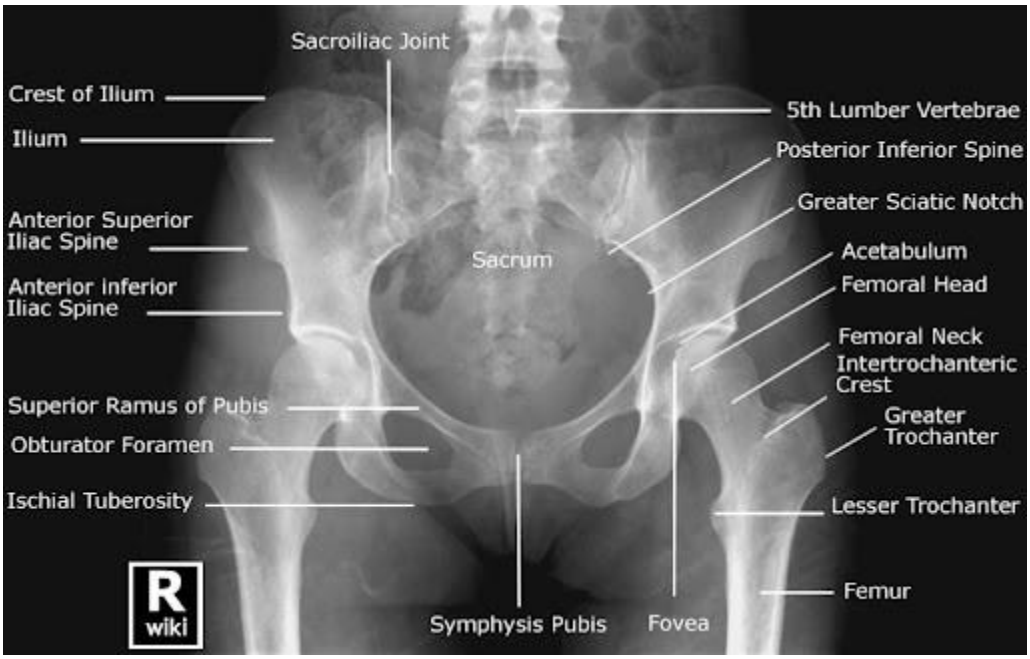
Associated injuries

- orthopaedics
  - chest injury in up to 63%
  - long bone fractures in 50%
  - spine fractures in 25%

- non-orthopaedic:

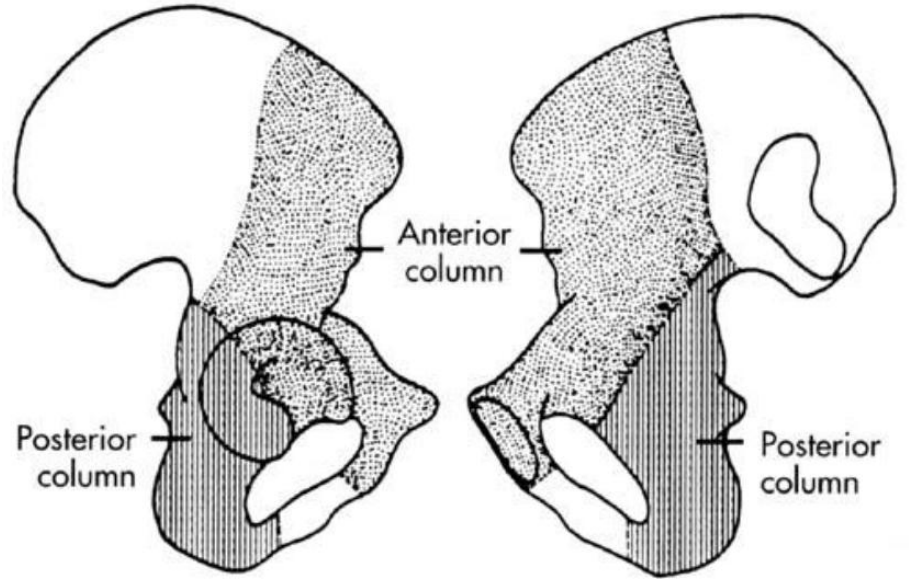
- urogenital
- sexual dysfunction up to 50%
- head and abdominal injury in 40%

Anatomy:

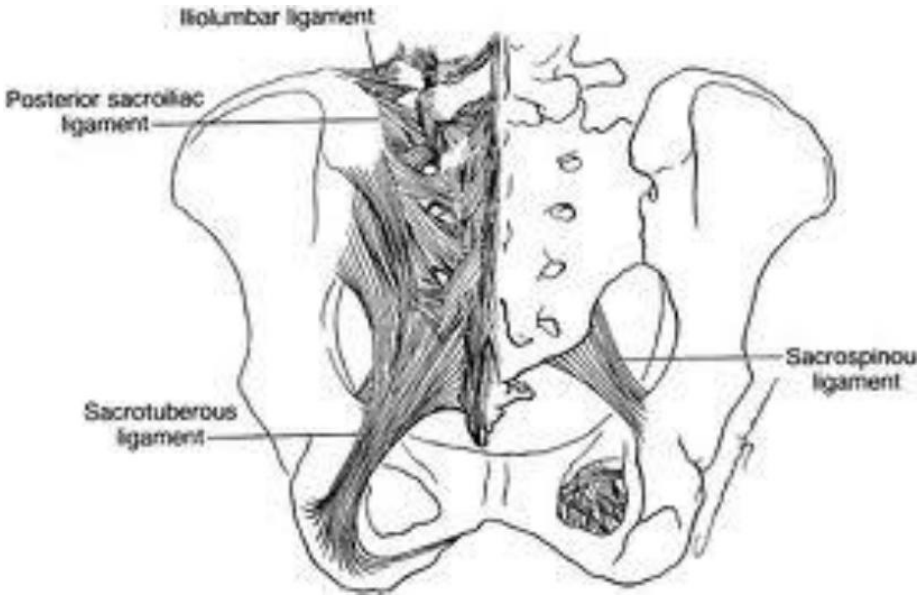


Pelvic ring structure made up of the sacrum and two innominate bones

acetabulum is supported by two columns of bone (anterior and posterior column) and has two walls



Ligaments



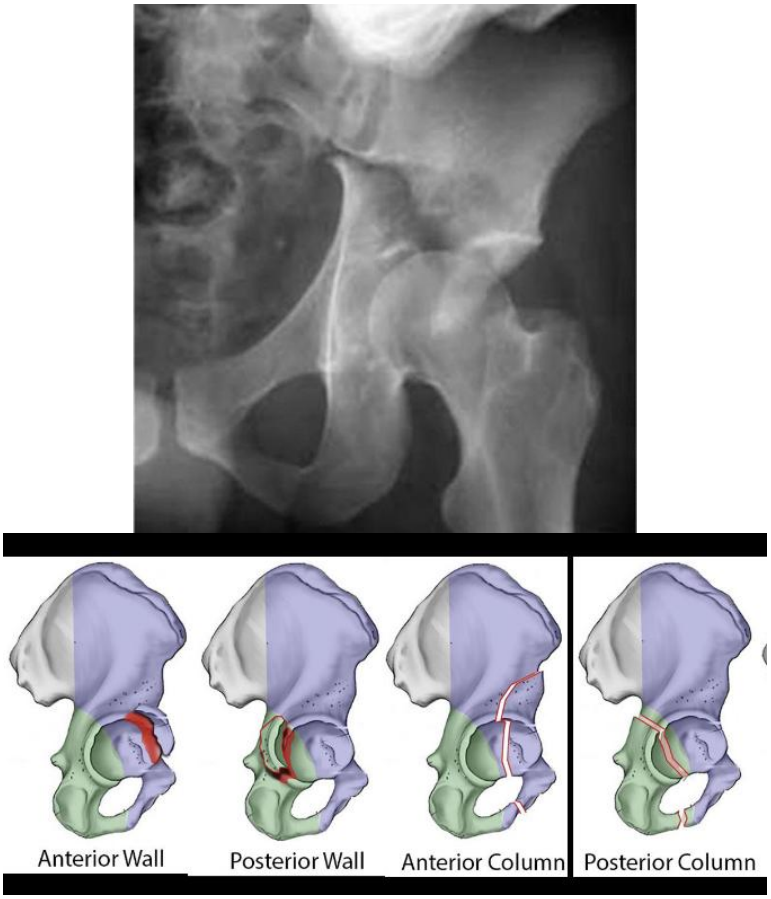
- Ligaments
  - anterior
    - symphyseal ligaments
  - pelvic floor
    - sacrospinous ligaments
    - sacrotuberous ligaments
  - posterior sacroiliac complex (posterior tension band)
    - strongest ligaments in the body

- more important than anterior structures for pelvic ring stability
- anterior sacroiliac ligaments
- interosseous sacroiliac
- posterior sacroiliac

Clinical picture:

- Pain
- Deformity: external rotation of the affected limb
- Inability to bear weight
- skin
  - scrotal, labial or perineal hematoma, swelling or ecchymosis
  - flank hematoma
  - lacerations of perineum
- neurological examination
  - rule out lumbosacral plexus injuries (L5 and S1 are most common)
- urogenital examination :
  - as it is common to have bladder injury or urethral injury

Imaging:



X-ray pelvis :

Initial imaging for ATLS survey

If suspected or confirmed fracture of the pelvic ring or acetabulum then proceed with CT pelvis for better visualization of the fracture

Management:

Most of the patients presenting with ring pelvis fracture or acetabulum have associated injures due to the high energy trauma mostly causing this type of fracture

We start doing the ATLS protocol :

Airway : maintain patent airway

Breathing: confirm by equal respiratory chest movement

Circulation: wide bore cannula and IV fluids until availability of fresh blood

Disability :

Exposure of the patient

Pelvic binder is done if the pelvic xray shows displaced fracture of the pelvis

External fixator : in case of unstable open fracture pelvis with ongoing blood loss

After stabilization definitive treatment is done:

Conservative treatment with Weight bearing after 6-8 weeks:

Stable fracture pelvic with no blood loss

Fracture ilium

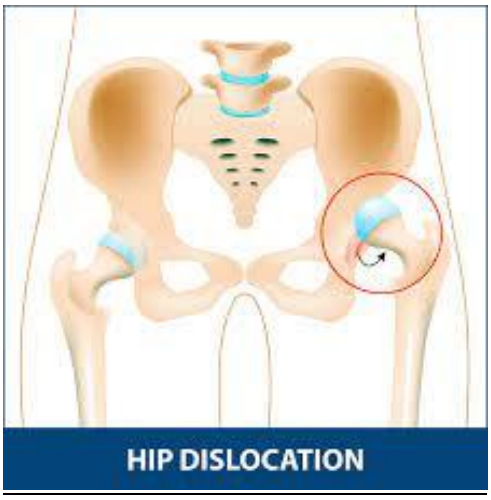
Acetabular fracture with displacement less than 2 mm on CT pelvis

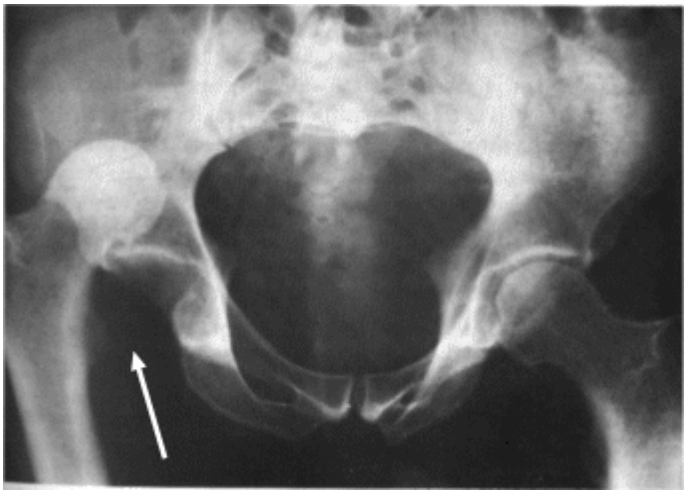
Open reduction internal fixation with plate and screws : in case of unstable fracture pelvis or fracture with more than 2 cm displacement

Acetabular fracture with more than 2 mm displacement on CT pelvis

Hip dislocation

- Hip dislocations are traumatic hip injuries that result in femoral head dislocation from the acetabular socket.
- Diagnosis can be made with hip radiographs to determine the direction of dislocation and CT scan studies to assess for associated injuries.
- Treatment is urgent reduction to minimize risk of avascular necrosis followed by CT scan to assess for associated injuries that may require surgical treatment (loose bodies, femoral head fractures, acetabular fractures).





Etiology :

High energy trauma in Road traffic accidents in young and elderly population (dash board injury)

Anatomy:

- Hip joint inherently stable due to
  - bony anatomy
  - soft tissue constraints including:

- labrum
- capsule
- ligamentum teres

Types of disclocations: Posterior dislocation (90%)

Anterior dislocation (10%)

Clinical picture

:

pain in hip region

swelling

deformity (mostly the lower limb is in internal rotation, flexion and adduction)

inability to bear weight

check Neurovascular status of the limb (mostly siatic n.)

Imaging :

X-ray of the Hip AP and lateral view for dislocation and associated fractures (neck femur or acetabulum)

CT of the hip for assessment of direction of dislocation and after reduction

Treatment:

- **Nonoperative:**
  - **emergent closed reduction within 12 hours**
    - acute anterior and posterior dislocations
- **Operative**
  - **open reduction and/or removal of incarcerated fragments**
    - irreducible dislocation

- radiographic evidence of incarcerated fragment
- delayed presentation
- should be performed on urgent basis

- **ORIF**
  - indications

    associated fractures of acetabulum, femoral head or femoral neck

Fracture neck femur (intracapsular fracture)

- Femoral neck fractures are common injuries to the proximal femur associated with increased risk of avascular necrosis, and high levels of patient morbidity and mortality.
- Diagnosis is generally made radiographically with orthogonal radiographs of the hip.
- Treatment is generally operative with open reduction and internal fixation versus arthroplasty depending on the age of the patient, activity demands and pre-injury mobility.

Etiology :

High energy trauma in Road traffic accidents in young population

Low energy trauma in old patients (fall on hip)

Anatomy:

The femoral neck is supplied by:

- major contributor is medial femoral circumflex (lateral epiphyseal artery)
- some contribution to anterior and inferior head from lateral femoral circumflex
- some contribution from inferior gluteal artery

- small and insignificant supply from artery of ligamentum teres
- displacement of femoral neck fracture will disrupt the blood supply and cause an intracapsular hematoma
- disruption of the blood supply will lead to avascular necrosis of the femoral head and leading to early onset of arthritis of the hip joint

Clinical picture:

pain in hip region

swelling

deformity (mostly the lower limb is in External rotation)

inability to bear weight

check Neurovascular status of the limb

Imaging :

X-ray of the Hip AP and lateral view

CT of the hip for assessment of degree of displacement

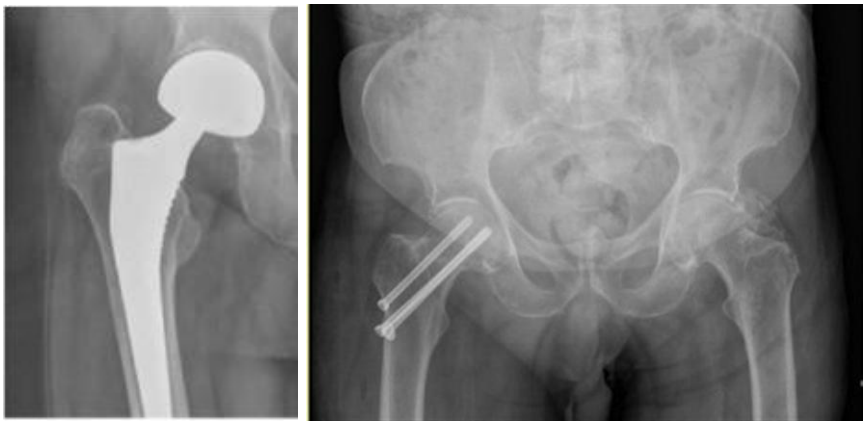
Treatment:

Observation : only in older patient with contraindications to surgery.

ORIF: in most patients

Cannulated screws for young patients

Total hip or hemiarthroplasty for older patients



Intertrochanteric fracture femur

- Intertrochanteric femur fractures are extracapsular fractures of the proximal femur at the level of the greater and lesser trochanter, most commonly seen following ground level falls in the elderly population.
- Diagnosis is made with orthogonal radiographs of the hip in patients that present with inability to bear weight.
- Treatment is operative with sliding hip screw versus cephalomedullary nail depending on fracture stability and fracture orientation

Etiology :

High energy trauma in Road traffic accidents in young population

Low energy trauma in old patients (fall on hip)

Anatomy:

Intertrochanteric area exists between greater and lesser trochanters

Clinical picture:

pain in hip region

swelling

deformity (mostly the lower limb is in External rotation)

inability to bear weight

check Neurovascular status of the limb

Imaging :

X-ray of the Hip AP and lateral view

CT of the hip for assessment of degree of displacement

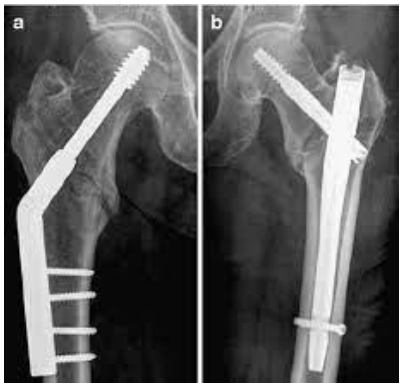
Treatment:

Observation : only in older patient with contraindications to surgery.

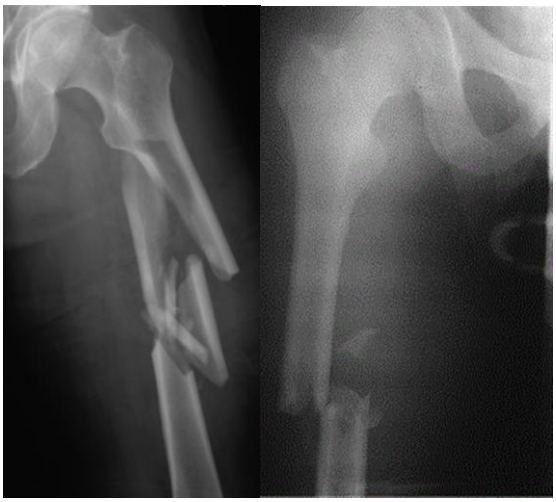
ORIF: in most patients

Dynamic hip Screw or Proximal femoral nail : mostly in young patients

Total hip replacement or hemiarthroplasty for elderly patients



Fracture shaft femur



Etiology :

High energy trauma in Road traffic accidents in young population

Low energy trauma in old patients

Clinical picture:

pain in thigh region

swelling

deformity (mostly the lower limb is in External rotation)

inability to bear weight

check Neurovascular status of the limb

Imaging :

X-ray of the femur AP and lateral view (showing joint above and below)

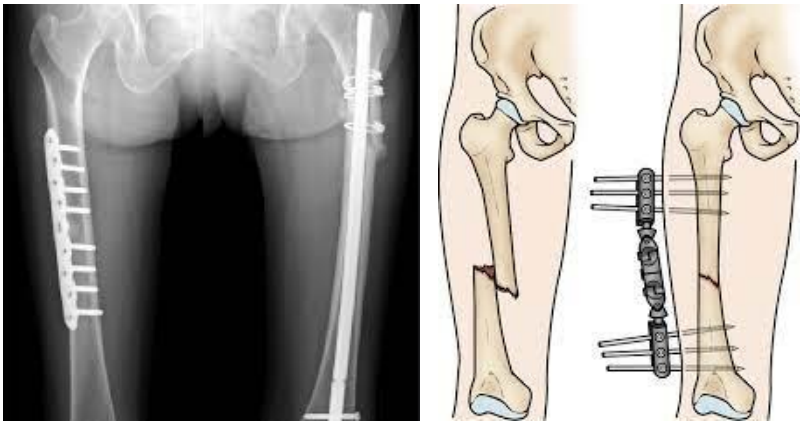
Treatment:

Observation : only in older patient with contraindications to surgery.

ORIF: in most patients

Plate and screws or intramedullary nail

External fixator in case of open fractures



Anterior Cruciate Ligament injury

is a very common athletic injury

Etiology : Twisting knee usually during football tackle

Anatomy:

- ACL is originated from the postero-medial aspect of the lateral femoral condyle
- And is inserted into the antero-medial aspect of the intercondylar area of the proximal tibia

Clinical picture:

pain in knee region

swelling and ecchymosis

inability to bear weight

check Neurovascular status of the limb

instability of the knee

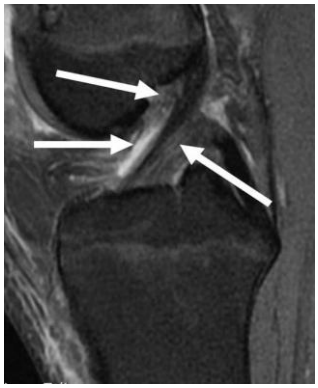
Provocative Tests:

- Anterior drawer test for assessment of degree of tibial translation
- Pivot test
- Lachman test

Imaging :

X-ray of the knee AP, lateral view : often normal

MRI of the knee joint : gold standard for diagnosis of the ACL tear or sprain.



Normal ACL

Torn ACL

Treatment:

- Nonoperative:
  - **physical therapy, lifestyle modifications**

    low demand patients with decreased laxity
    - partial ACL tear

- Operative
  - **Arthroscopic ACL reconstruction**
    - younger, more active patients with complete tear or failure of medical and physical therapy after 6-8 weeks and patient is complaining of recurrent knee instability

Meniscal injury

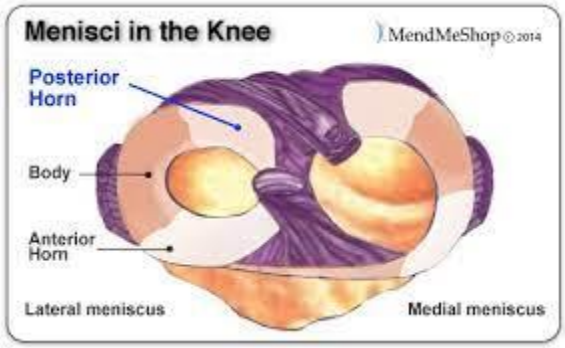
is a very common athletic injury

Etiology :

Twisting knee

Anatomy:

- meniscus consist of : anterior horn and body and posterior horn



- medial meniscal tears
  - more common than lateral tears
  - degenerative tears in older patients usually occur in the posterior horn medial meniscus

Clinical picture:

pain in knee region

swelling

inability to bear weight

instability and locking of the knee

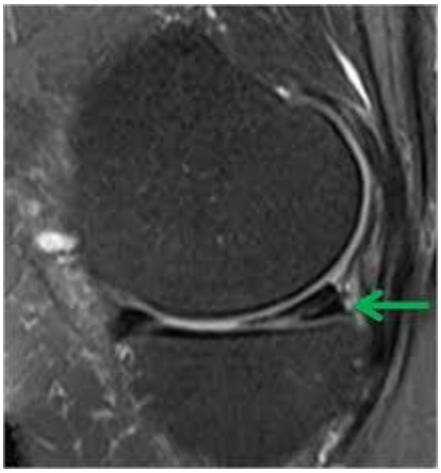
Provocative Tests:

- Mc-Murray test: for assessment of the meniscal injury:
  - ✓ flex the knee and place a hand on medial side of knee, externally rotate the leg and bring the knee into extension.
  - ✓ a palpable pop / click + pain is a positive test and can correlate with a medial meniscus tear.

Imaging :

X-ray of the knee AP, lateral view : often normal

MRI of the knee joint : gold standard for diagnosis of the meniscal tear or degeneration



Meniscal tear

Treatment:

- Nonoperative:

  rest, NSAIDS, rehabilitation

  - indicated as first line treatment for degenerative tears

Operative

- arthroscopic repair or partial meniscectomy : for most of the meniscal tears

Tibial plateau fracture

Etiology :

High energy trauma in Road traffic accidents in young population

Low energy trauma in old patients (fall on knee)

Clinical picture:

Pain and swelling in knee region

Deformity (mostly the lower limb is in External rotation)

Inability to bear weight

check Neurovascular status of the limb

Imaging :

X-ray of the knee AP and lateral view

CT of the knee for further assessment of the fracture type and degree of displacement

Treatment:

Observation : only in older patient with contraindications to surgery.

ORIF: in most patients

Plate and screws with or without bone grafting

Tibial shaft fracture

Etiology :

High energy trauma in Road traffic accidents in young population

Low energy trauma in old patients

Clinical picture:

pain in leg region

swelling

deformity (mostly the lower limb is in External rotation)

inability to bear weight

check Neurovascular status of the limb

Imaging :

X-ray of the tibia AP and lateral view

Treatment:

Observation : only in older patient with contraindications to surgery.

ORIF: in most patients

Plate and screws or intramedullary nail

External fixator in case of open fractures

Ankle fractures

Ankle fractures are very common injuries to the ankle which generally occur due to a twisting mechanism

Etiology : Twisting ankle

Anatomy:

ankle joint consist of :

Medial malleolus which is made from the distal part of the tibia

Lateral malleolus which is made from the distal part of the fibula

Talus bone which articulates with them

Motion: mainly Plantar-flexion and Dorsi-flexion

Clinical picture:

pain in ankle region

swelling

deformity

inability to bear weight

check Neurovascular status of the limb

Imaging :

X-ray of the ankle AP, lateral

Treatment:

Observation with medical treatment and casting of the leg : in non-displaced fractures

ORIF: in displaced fractures

Plate and screws

Sprain ankle

Ankle Sprains are very common twisting injuries to the ankle that are the most common reason for missed athletic participation.

Etiology :

Twisting ankle

Anatomy:

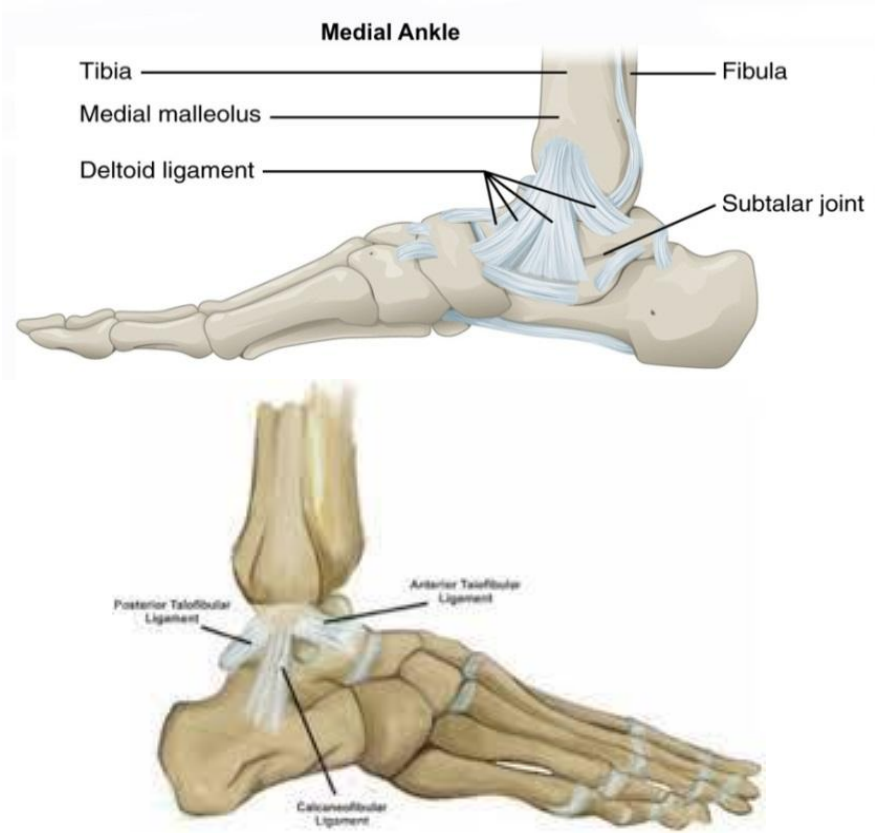
Ligaments of the Lateral side :

- anterior talo-fibular ligament
- posterior talo-fibular ligament

- calcaneo-fibular ligament

ligaments of the Medial side:

Deltoid ligament



Clinical picture:

pain in ankle region

swelling and ecchymosis

deformity

inability to bear weight

Imaging : (usually required to exclude fractures)

X-ray of the ankle AP, lateral and mortise view

MRI of the ankle joint : if medical treatment failed after 6-8 weeks

Treatment:

Observation with medical treatment and casting of the leg : in most sprains

Ligament reconstruction: for chronic cases

Spinal injuries

Anatomy

Nervous system

- central nervous system

    - includes the
        - brain
        - spinal cord
            - spinal cord ends at L3 at birth, and L1 at maturity

- peripheral nervous system
    - contains the
        - cranial nerves
        - peripheral nerves
- autonomic nervous system

  sympathetic system
        - a total of 22 ganglia (3 cervical, 11 thoracic, 4 lumbar, 4 sacral)

  parasympathetic nervous system
    - hypogastric plexus
        - formed by S2, S3, S4 parasympathetic fibers and lumbar sympathetic fibers (splanchnic nerves)

Spinal cord

Spinal cord extends from brainstem to inferior border of L1

- conus medullaris
    - is termination of spinal cord
- filum terminale
    - is residual fragment of spinal cord that extends from conus medullaris to sacrum.
- thecal sac
    - the dural surrounded sac that extends from the spinal cord and contains CSF, nerve roots and the cauda equina
- cauda equina
    - nerve roots and filum terminale surrounded by dura that extend from the spinal cord

osteology

The spine contains 7 cervical 12 thoracic and 5 lumbar vertebral bodies

- C1 (atlas)
    - intrinsic ligaments are located within the spinal canal, provide most of the ligamentous stability. They include transverse ligament, paired alar ligaments and apical ligament

- C2 (axis) axis has odontoid process (dens) and body
- C3-L5

  1. Anterior vertebral body
  2. Posterior arch formed by pedicles project posteriorly from posterolateral corners of vertebral bodies
  3. Lamina project posteromedially from pedicles, join in the midline
  4. spinous process
  5. transverse process

Articulations

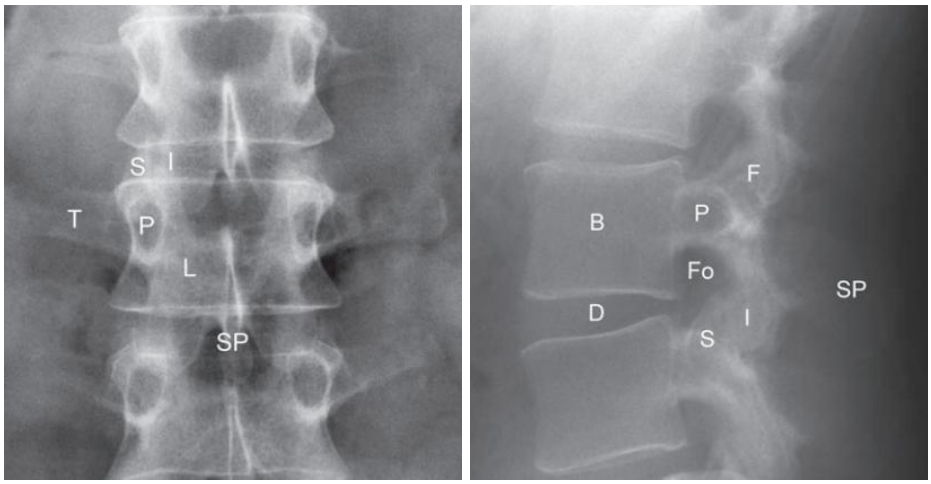
occipital-cervical junction and atlantoaxial junction are coupled

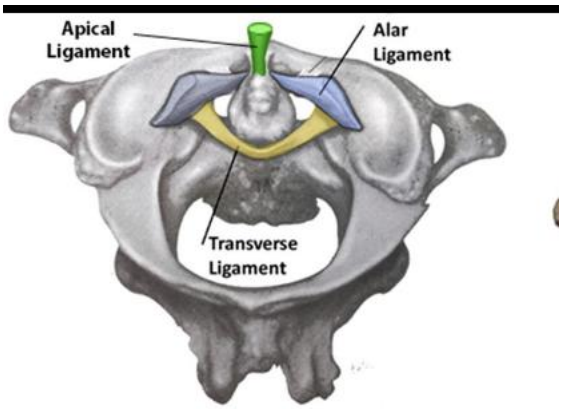
Intervertebral disc act as an articulation above and below

Facet joint formed by superior and inferior articular processes that project from junction of pedicle and lamina

Nerve roots

there is no C8 vertebral body although there is a C8 nerve root





Injuries of spinal cord

- tetraplegia
  - injury to the cervical spinal cord leading to impairment of function in the arms, trunk, legs, and pelvic organs
- paraplegia
  - injury to the thoracic, lumbar or sacral segments leading to impairment of function in the trunk, legs, and pelvic organs depending on the level of injury. Arm function is preserved
- complete injury
  - an injury with no spared motor or sensory function below the affected level.
- incomplete injury
  - an injury with some preserved motor or sensory function below the injury level

Spine fractures

Presentation

- History
  - high-energy mechanism
    - axial-loading and flexion mechanisms
      - fall from height (e.g. fall from deer hunting stand, fall from ladder, etc.)
      - high-speed motor vehicle collision
- Symptoms
  - Neck pain
  - Dysphagia
  - severe back pain
  - radicular pain
  - parasthesias
- **Physical exam** :- assessment as per Advanced Trauma and Life Support(ATLS) protocol

  Primary survey
    - Airway
    - Breathing

- Cardiovascular support
- Disability
  - spinal precautions with cervical spine immobilization and log-roll procedures should be performed
  - spine board should be used to prevent flexion of the cervical spine
  - evaluate neurologic response using the Glasgow Coma Scale
- Exposure

Secondary survey

- trauma specific history
  - mechanism of injury, last meal,, Allergies, medications
- full neurological examination
  - motor and sensory examination by myotome and dermatome, respectively
  - rectal and genital examination
  - bulbocavernosus reflex, when appropriate
- physical examination
  - inspection and palpation of the entire spine and paraspinous region
  - note step-offs, crepitus, bruising, pain, or open injuries
  - head-to-toe assessment for associated injuries

Classification

- compression fracture
- burst fraction
- flexion-distraction injury
- facet dislocation
- facet fracture

cervical clearance

Removal of cervical collar if radiologically free

Or WITHOUT radiographic studies allowed only if

patient is awake, alert, and not intoxicated AND

has no neck pain, tenderness, or neurologic deficits AND

has no distracting injuries

Radiographs

- recommended views
  - AP/lateral cervical, thoracic, lumbar spine

Finding

- malalignment
- fracture
- dislocation

CT scan

- indications
  - fracture on plain film
  - neurologic deficit in extremity
  - poly-trauma and high energy injuries
  - high clinical suspicion of spine injury and altered mental status

MRI

- indications
  - whenever neurological deficits

useful to evaluate for

- spinal cord or thecal sac compression by disk or osseous material
- cord edema or hematoma
- injury of the ligamentous complex

Treatment

- **pain control and activity as tolerated for** stable fracture patterns
- cervical collar immobilization for stable odontoid fractures
- thoracolumbosacral braces for compression fractures (<50% anterior height loss)
- **surgical stabilization of cervical spine for** instability
- **surgical stabilization of thoracolumbar spine for** unstable burst fracture and spinal cord compression

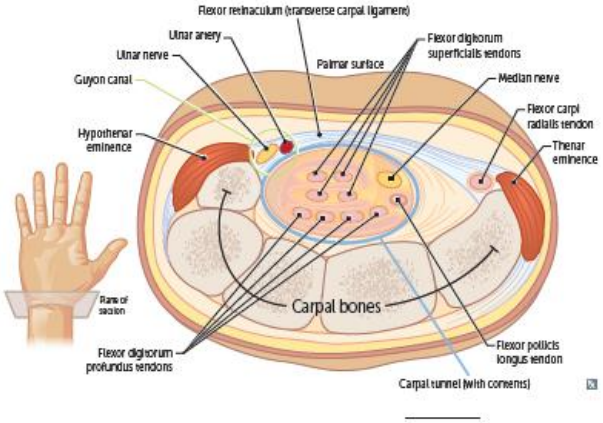
Carpal Tunnel Syndrome

Carpal Tunnel Syndrome is a compressive neuropathy of the median nerve at the level of the wrist.

Diagnosis is made by clinical signs and symptoms (night pain, hand weakness/clumsiness, numbness in median nerve distribution) and positive

provocative tests (i.e Tinel's, Durkan's).

Treatment is generally conservative with night splints and injections with carpal tunnel release reserved for refractory cases.



Carpal tunnel borders

transverse scaphoid tubercle and trapezium radially

hook of hamate and pisiform ulnarly

carpal ligament palmarly (roof)

proximal carpal row dorsally (floor)

Carpal tunnel contents

four flexor digitorum superficialis (FDS) tendons

four flexor digitorum profundus (FDP) tendons

flexor pollicis longus (FPL)

most radial structure

median nerve

Symptoms

numbness and tingling in radial 3-1/2 digits

clumsiness

pain and paresthesias that awaken patient at night

Physical exam

A- **thenar atrophy**

B- **carpal tunnel compression test (Durkan's test)**

is the most sensitive test to diagnose carpal tunnels syndrome

performed by pressing thumbs over the carpal tunnel and holding pressure for 30 seconds.

onset of pain or paresthesia in the median nerve distribution within 30 seconds is

a positive result.

C- **Phalen test**

wrist volar flexion against gravity for ~60 sec produces symptoms

less sensitive than Durkin compression test

D- **Tinel's test**

provocative tests performed by tapping the median nerve over the volar carpal tunnel

Diagnostic criteria

A- numbness and tingling in the median nerve distribution

B- nocturnal numbness

C- weakness and/or atrophy of the thenar musculature

D- positive Tinel sign

E- positive Phalen test

F- loss of two-point discrimination

EMG and NCV

provides objective evidence of a compressive neuropathy

Treatment

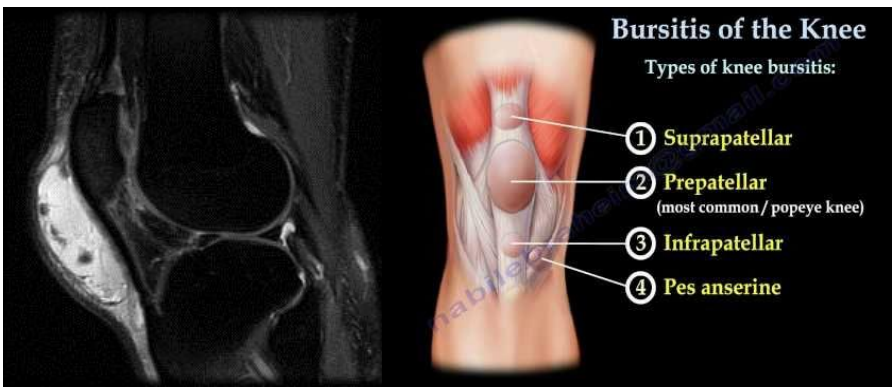
NSAIDS, night splints, activity modifications

steroid injections

carpal tunnel release

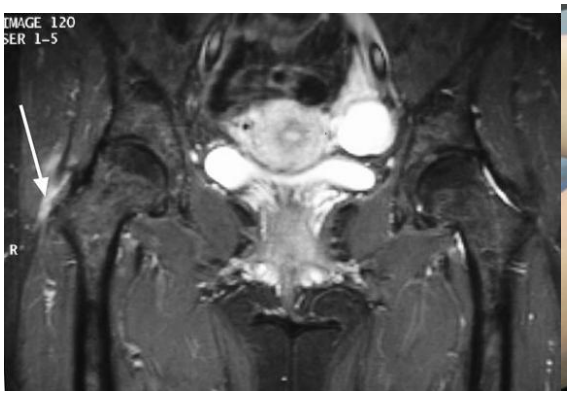
Prepatellar Bursitis (Housemaid's Knee)

- Prepatellar bursitis is the swelling and inflammation of the anterior knee bursa associated with pain with kneeling.
- Diagnosis is made clinically with mild swelling and tenderness over the anterior knee overlying the patella.
- Treatment is nonoperative with compressive wraps and NSAIDs. Occasionally, in the setting of septic bursitis, antibiotics may be indicated.

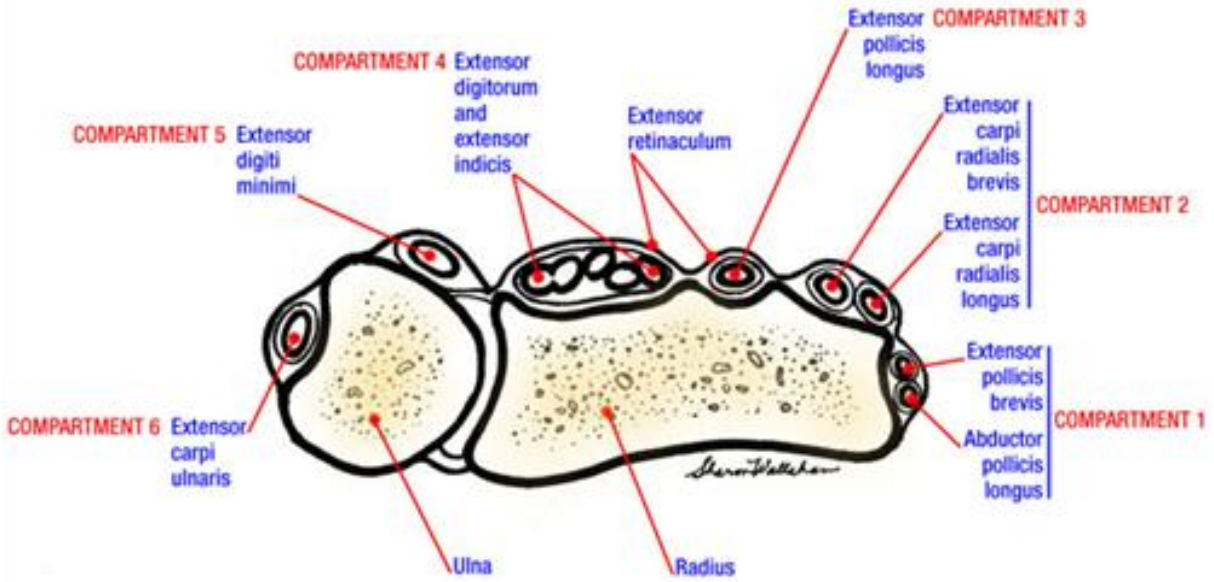


Trochanteric bursitis

- Trochanteric bursitis is a very common source of lateral hip pain caused by repetitive trauma secondary to iliotibial band tracking over the trochanteric bursa.
- Diagnosis is made clinically with point tenderness over the greater trochanter.
- Treatment is conservative with NSAIDs, stretching, physical therapy and corticosteroid injections.



De Quervain's Tenosynovitis



De Quervain's Tenosynovitis is a stenosing tenosynovial inflammation of the 1st dorsal compartment.

- Diagnosis is made clinically with radial sided wrist pain made worse with the Finkelstein maneuver.
- Treatment is generally conservative with thumb spica braces, injections and in refractory cases, 1st dorsal compartment surgical release.

Lateral Epicondylitis (Tennis Elbow)

- Lateral Epicondylitis (also know as Tennis Elbow) is an overuse injury caused by eccentric overload at the origin of the common extensor tendon, leading to tendinosis and inflammation of the ECRB.
- Diagnosis is made clinically with tenderness over the lateral epicondyle made worse with resisted wrist extension.
- Treatment is primarily nonoperative with NSAIDs, activity modification and bracing. Rarely, operative management is indicated for patients with persistent symptoms who fail nonoperative management.

Septic Arthritis

- Septic Arthritis is the inflammation of the joints secondary to an infectious etiology, most commonly affecting the knee, hip, and shoulder.
- Diagnosis is made with an aspiration of joint fluid with a WBC count> 50,000 being considered diagnostic for septic arthritis. Lower counts may still indicate infection in the presence of positive gram stains or cultures results.
- Treatment is usually urgent surgical irrigation and debridement followed by culture directed IV antibiotics.

etiologies of bacterial seeding of joint

1. bacteremia

2. direct inoculation
    - from trauma or surgery
3. contiguous spread
    - from adjacent osteomyelitis
    - most common pathogens is staphylococcus aureus (accounts for >50% of cases)

risk factors

neonates, IV drug users, elderly and immunocompromised patients with diabetes

Symptoms

1. pain in affected joint
2. fevers (only present in 60% of cases)
3. may appear toxic

Physical exam

1- inspection

- erythema, effusion, extremity tends to be in position of maximum joint volume
- hip would be in FABER position (flexed, abducted, externally rotated)

2- palpation

warmth and tender

3- motion

inability to bear weight

inability to tolerate PROM

Ultrasound

- may help in confirming joint effusion in large joint such as hip
- can be used in guiding aspirations

MRI

- detects joint effusion, and may detect adjacent bone involvement such as osteomyelitis

Studies

- WBC >10K
- ESR >30
- CRP >5 most helpful
    - **Joint fluid aspirate**

- joint fluid appears cloudy or purulent
- cell count with WBC > 50,000 is considered diagnostic for septic arthritis

treatment

IV abx, operative irrigation and drainage of the joint

Knee Osteoarthritis

- Knee osteoarthritis is degenerative disease of the knee joint that causes progressive loss of articular cartilage.
- Diagnosis can be made with plain radiographs of the knee.
- Treatment is observation, NSAIDs, tramadol and corticosteroids for minimally symptomatic patients. Knee arthroplasty is indicated for progressive symptoms with severe degenerative disease

Risk factors

1- **modifiable**

- articular trauma
- occupation, repetitive knee bending
- muscle weakness
- large body mass

2- **non-modifiable**

- gender

  females >males
- increased age
- genetics

pathoanatomy

1- **articular cartilage**

- increased water content

synovium and capsule

- early phase of OA
  - mild inflammatory changes in synovium
- middle phase of OA
  - moderate inflammatory changes of synovium
  - synovium becomes hypervascular
- late phases of OA
  - synovium becomes increasingly thick and vascular

2- **bone**

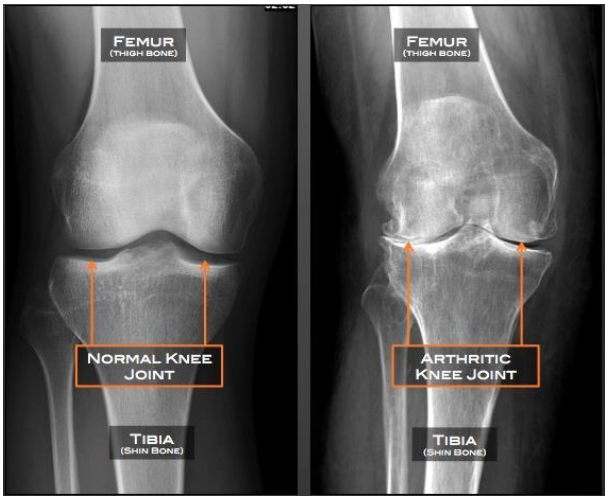
- subchondral bone attempts to remodel forming lytic lesion with sclerotic edges
- bone cysts form in late stages
- osteophytes formation

Symptoms

- function-limiting knee pain
  effect on walking distances
- pain at night or rest
  range of motion
- lack of full extension (>5 degrees flexion contracture)
- lack of full flexion

imaging

  - weight-bearing views of affected joint
- joint space narrowing
- osteophytes
- subchondral sclerosis
- subchondral cysts



Treatment is observation, NSAIDs, tramadol and corticosteroids for minimally symptomatic patients. Knee arthroplasty is indicated for progressive symptoms with severe degenerative disease.

Osteomylitis

- Osteomyelitis is the infection of bone characterized by progressive inflammatory destruction and apposition of new bone.
- Diagnosis requires careful assessment of radiographs, MRI and determining the organism via biopsy and cultures.
- Treatment is often a combination of culture-directed antibiotics and surgical debridement of nonviable tissue

Risk factors

- recent trauma or surgery

- immunocompromised patients
- illicit IV drug use
- poor vascular supply
- systemic conditions such as diabetes and sickle cell

mechanism of spread

- hematogenous
- contiguous-spread
  - associated with previous surgery, trauma, wounds, or poor vascularity
- direct-inoculation
  - penetrating injuries
  - open fractures

Organism

- S. aureus is most common in adults

Timing classification

- acute
  - within 2 weeks
- subacute
  - within one to several months
- chronic
  - after several months

PRESENTATION

- **History**

  duration

  prior treatments

- **Symptoms**

  pain

  fever

  more common in acute osteomyelitis

- **Physical exam**

  vital signs

  fever, tachycardia, and hypotension suggest sepsis

  inspection

  erythema, tenderness, and edema are commonly seen

draining sinus tract

more common in chronic osteomyelitis

- **motion**

  limp and/or pain inhibition with weight-bearing or motion may be present

  assess the joints above and below the area of concern

- **neurovascular**

  assessment of vascular insufficiency locally or systemically

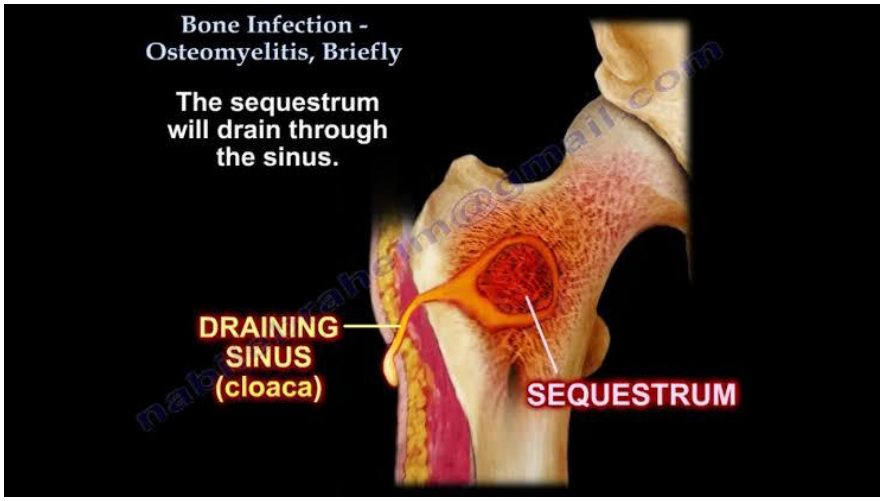
IMAGING

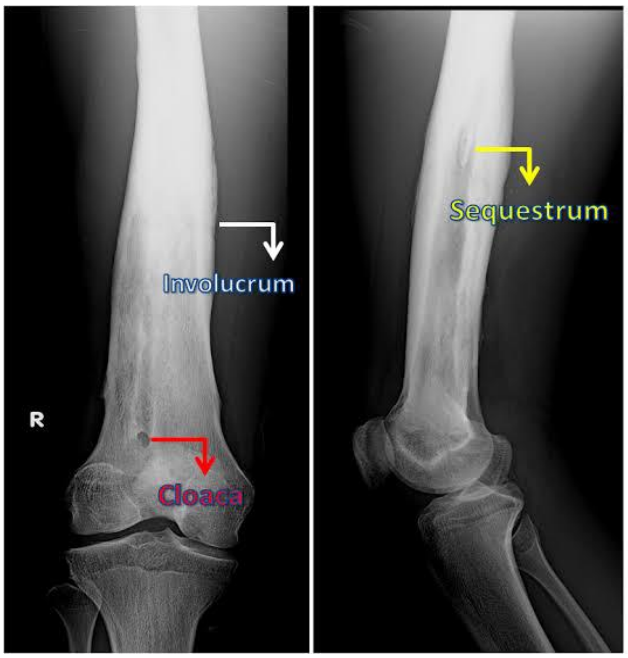
PXR

bone lucency, sclerotic rim, osteopenia, periosteal reaction, and lysis around hardware

sequestrum: devitalized bone that serves as a nidus for infection

involucrum: formation of new bone around an area of bony necrosis





CT

assist in diagnosis and surgical planning by identifying necrotic bone

MRI

assists in the diagnosis and surgical planning

best test for diagnosing early osteomyelitis and localizing infection

technetium bone scan

when radiographs are normal and MRI is not an option

highly sensitive but not specific

if negative rules out osteomyelitis

lab studies

- eukocyte count (WBC)

only elevated in 1/3 of acute osteomyelitis

erythrocyte sedimentation rate (ESR)

usually elevated in both acute and chronic osteomyelitis (90%)

a decrease in ESR after treatment is a favorable prognostic indicator

- C-reactive protein

most sensitive test with elevation in 97% of cases

decreases faster than ESR in successfully treated patients

- blood cultures

often negative, but may be used to guide therapy for hematogenous osteomyelitis

- **sinus tract cultures**

treatment

Goals

success in the treatment is dependent on various factors

- patient factors

  immunocompetence of patient

  nutritional status

- injury factors

  the severity of the injury as demonstrated by segmental bone loss

- infection location

  metaphyseal infections heal better than mid-diaphyseal infections

- other factors affecting prognosis and treatment include:

  residual foreign materials and/or ischemic and necrotic tissues

inappropriate antibiotic coverage

lack of patient cooperation or desire

Nonoperative Treatment

suppressive antibiotics

when operative intervention is not feasible

hyperbaric oxygen therapy

can be used as adjunct in refractory osteomyelitis

Operative treatment

- **irrigation and debridement followed by organism specific antibiotics**

indications

acute osteomyelitis that fails to improve on IV antibiotics

subacute osteomyelitis

abscess formation

chronic osteomyelitis

draining sinus

- **amputation**

indications

chronic infection with pervasive wound or bone damage that is unable to be salvaged

Bone tumors

Classification

- **Benign**
- **Malignant**
  - **Primary**
  - **Secondary**

Benign lesions are classified using numbers (1,2,3)

1 = latent lesion

e.g. non-ossifying fibroma

enchondroma

2 = active lesion

e.g. ABC, UBC

chondromyxoid fibroma

chondroblastoma

3 = aggressive lesion

e.g. giant cell tumor of bone

malignant lesions are classified in the table below

Stage	Grade	Site (1)	Metastasis
IA	Low Grade	T1 - intracompartmental	M0 (none)
IB	Low Grade	T2 - extracompartmental	M0 (none)
IIA	High Grade	T1 - intracompartmental	M0 (none)
IIB	High Grade	T2 - extracompartmental	M0 (none)
III	Metastatic	T1 - intracompartmental	M1 (regional or distant)
III	Metastatic	T2 - extracompartmental	M1 (regional or distant)

Diagnosis

can be reached by

History (thyroid breast prostate)

Clinical examination : Thyroid Breasts Chest Liver Kidney Rectal (prostate

& rectal tumours)

PXR CT MRI : affected bone

Angiography -> tumour blood supply and relationship to major vessels

CT : chest abd pelvis

Bone scan

Laboratory: CBC, ESR, LFT, C PH ALP, Tumor markers(AFP PSA), urine analysis

Electrophoresis

Biopsy

TREATMENTS

❑ **Non surgical treatment:**

- Observation
- Chemotherapy
- Radiotherapy
- Hormonal therapy
- Bisphosphonates

SURGICAL EXCISION

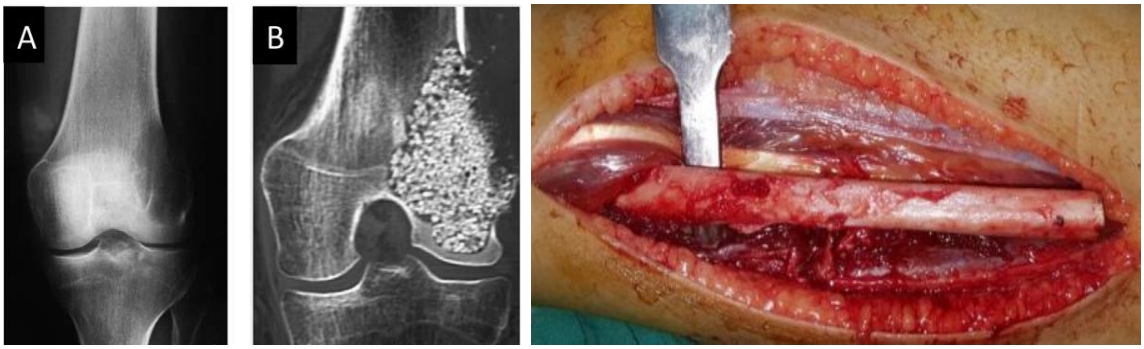
- *Intra-lesional*
  - through the tumour
- *Marginal*
  - through pseudo-capsule of tumour
- *Wide*
  - around reactive zone, take a cuff of normal tissue
- *Radical*
  - removal of entire compartment or compartments

Reconstructive options

➢ biological methods including:-

- Autograft or allograft (Vascularized or non)
- Tumor devitalized autograft or recycling (involves re-implantation of tumor-bearing bone after devitalized treatment (extracorporeal irradiation, heat treatment and freezing)

➢ Non biological methods, using custom-made implants



Benign Bone tumors

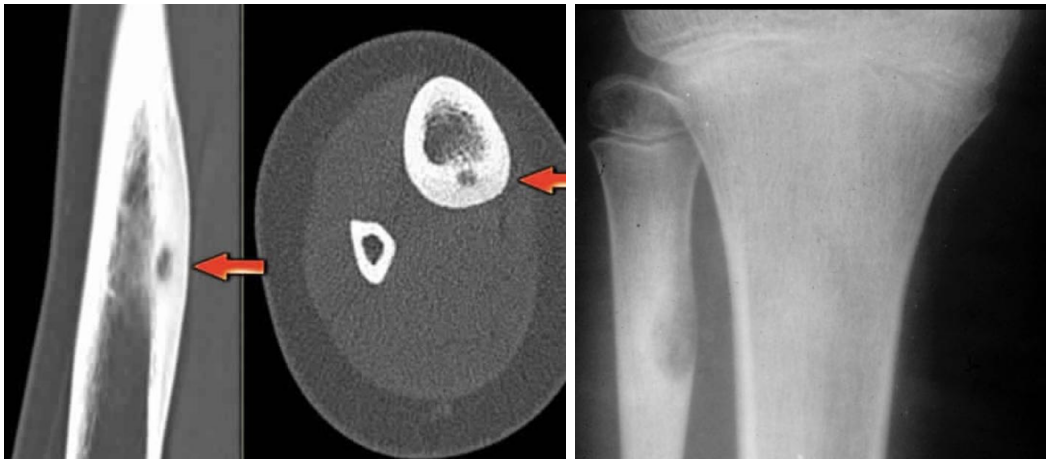
	DEFINITION	Age	SYMPTOMS	IMAGING	TREATMENT
SBC	serous fluid-filled bone lesion	Young patients arises in the metaphysis	most asymptomatic unless fractured	central, lytic, well-demarcated metaphyseal lesion "fallen leaf" sign	Immobilization Curettage and bone grafting
ABC	Benign bone lesion with multiple blood-filled cavitie	May be primary or secondary	pain and swelling pathologic fracture	PXR:- expansile, eccentric metaphyseal and lytic lesion with bony septae ("bubbly appearance") MRI or CT scan will show multiple fluid	curettage and bone grafting
NOF	a benign fibrogenic lesion	As patient approaches skeletal maturity, lesions become sclerotic	found incidentally pathologic fracture	metaphyseal eccentric lytic lesion surrounded by sclerotic rim	observation curettage and bone grafting
Osteoid osteoma	A small, painful, benign cortical bone lesion	nidus central nodule of woven bone and osteoid reactive zone area of thickened bone	pain constant and progressive worse at night and with drinking Alcohol relieved by NSAIDS	sclerotic bone around radiolucent nidus CT identify the location and size of nidus	NSAIDS radiofrequency ablation resection
enchondroma	A benign chondrogenic tumor composed of hyaline cartilage	the most common bone tumor in the hand is the enchondroma May be solitary enchondroma or multiple	Ollier's disease and Maffucci's syndrome (multiple enchondromatosis)	well defined, lucent, central medullary lesions that calcify over time pop-corn" stippling or may have purely lytic appearance (especially in hand)	Observation resection
osteochondroma	benign chondrogenic lesion derived from cartilage from the perichondral ring Occur on the surface of the bone	The most common benign bone tumor	Asymptomatic, painless mass *continue to grow away from joints until skeletal maturity*	sessile (broad base) or pedunculated (narrow stalk) lesions found on the surface of bones Cartilage cap is usually radiolucent (looks smaller than felt)	Observation Resection if symptomatic
GCT	benign aggressive tumor typically found in the epiphyseometaphyseal region	Can be malignant and metastasize to lung	pain of the involved extremity palpable mass	eccentric lytic epiphyseal/metaphyseal lesion with soap and bubble appearance	extensive curettage, adjuvant treatment and reconstruction

SBC

ABC

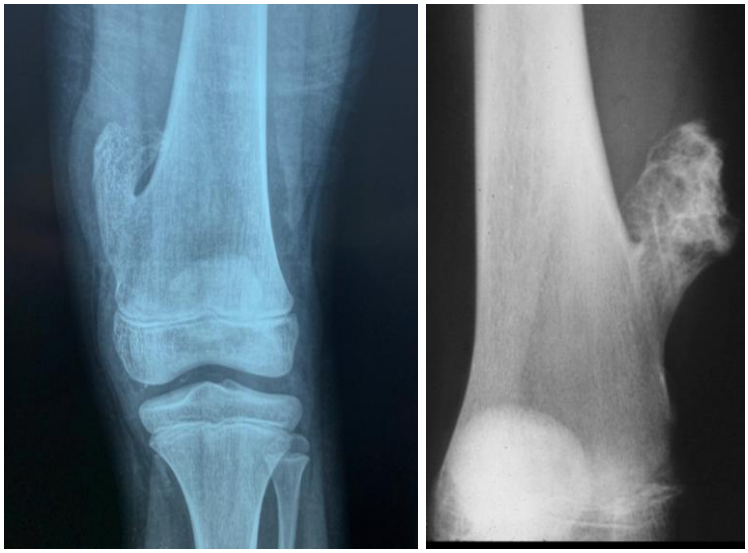
NOF

OSTEOID OSTEOMA

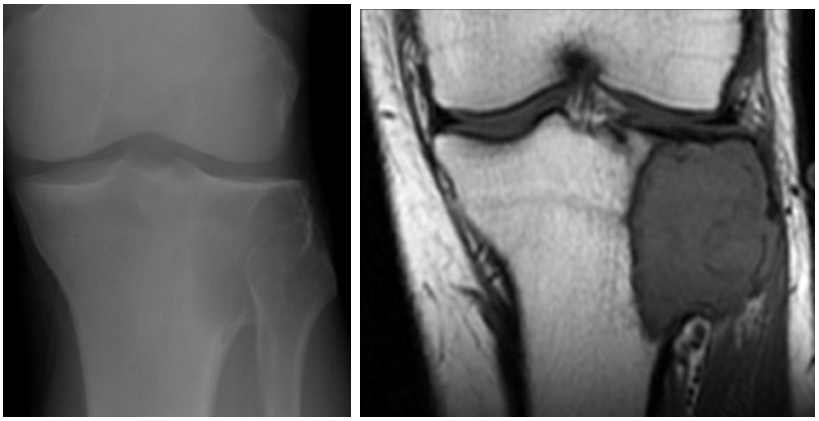


ENCHONDROMA

OSTEOCHONDROMA



GIANT CELL TUMOR



Osteosarcoma

Is a bone-producing sarcoma ; produce immature woven bone

It is the most common primary cancer of bone in children and adolescents

Primary Osteosarcoma: arises from the bone in the absence of a precursor lesion

Secondary Osteosarcoma: arises from a precursor lesion or one that is metastatic from a primary osteosarcoma

Age/Signs/Symptoms

- 15-25 years: Most common age is in childhood and adolescence
- Mild Pain for weeks-months
- Pain gradually becomes more severe & accompanied by swelling and limitation of motion
- Weight loss

Anatomic sites

- Long Bone metaphysis (Most common)
- Distal Femur: most common site

- Skip metastases: metastases within the same bone as the primary osteosarcoma
- may dictate that the entire bone be surgically removed

Gross Pathology:

- **Osteosarcomas are composed of ossified or non-ossified tissue**
- **Ossified tissue is yellow-white and hard**
- **Less ossified tissue is soft**
- **osteosarcomas penetrate the cortex and form a large extraosseous soft tissue mass**
- **Foci of hemorrhage and necrosis are common**
- **Periosteal reactions such as the Codman's triangle are apparent at periphery of soft tissue mass**

Radiology

- **X-Ray**:

  characteristic blastic and destructive lesion

  sun-burst or hair on end pattern of matrix mineralization

  periosteal reaction (Codman's triangle)

  large soft tissue mass with maintenance of bone cortices

➢ **MRI Best for:**

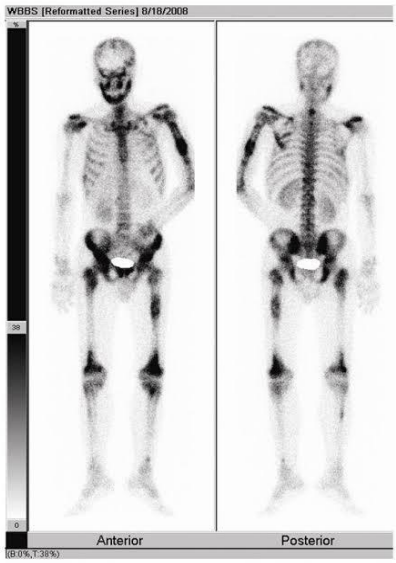
- determining intraosseous extent (size) of the tumor and **size** of the **soft tissue** component
- determining relationship of tumor to **neurovascular** structures
- detecting **skip** metastases

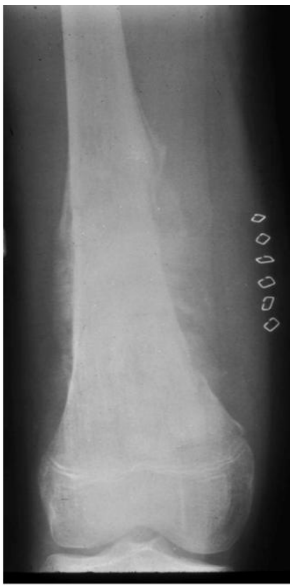
- Entire bone and adjacent joint should be visualized

➢ **CT Scan of the Chest**: Used to detect pulmonary metastases

➢ **Whole Body Bone Scan**: Used to detect bone metastases, extent of bony involvement by the tumor and presence of skip metastases

➢ **Biopsy: Confirm diagnosis**





TREATMENT

- osteosarcomas treated with a combination of chemotherapy and wide excision.
- usually, chemotherapy is administered before and after surgery
- chemotherapy before surgery = preoperative = neoadjuvant chemotherapy
- chemotherapy after surgery = adjuvant chemotherapy

Ewing sarcoma

- Ewing's Sarcoma is a malignant, distinctive small round cell sarcoma associated with a t(11:22) translocation and most commonly occurs in the diaphysis of long bones.
- Patients typically present at age < 25 with insidious onset of regional pain, swelling, and fevers.
- Diagnosis is made with a biopsy showing sheets of monotonous small round blue cells with prominent nuclei and minimal cytoplasm. Immunostaining will show positive CD99.
- Treatment is usually neo-adjuvant chemotherapy, limb salvage surgical resection, followed by adjuvant chemotherapy with or without radiation.
- small round cell sarcoma
- 5-25 years of age
- Found in the diaphysis of long bones
- Genetics t(11:22) translocation found in 95% of cases
- **Presentation**
    - May present with pain, fever, weight loss, leukocytosis and increased erythrocyte sedimentation rate mimicking osteomyelitis

❑ **Imaging**

- **PXR**
    - ➢ large destructive lesion in the diaphysis or metaphysis
    - ➢ lesion may be purely lytic or have variable amounts of reactive new bone formation
    - ➢ periosteal reaction may give an "onion skin" or "sunburst" appearance

MRI shows a large soft tissue component

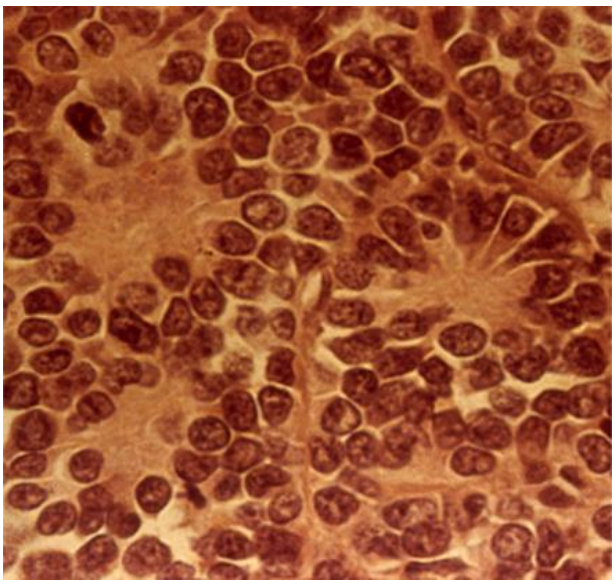
- **Bone scan** will show very "hot" lesion

CT chest required as initial staging workup to look for pulmonary metastasis

HISTOLOGY

Characteristic findings

- sheets of monotonous small round blue cells
- prominent nuclei and minimal cytoplasm
- may have pseudo-rosettes (circle of cells with necrosis in center)



❑ **Treatment**

- chemotherapy + radiation therapy for non-resectable tumors
- **chemotherapy + limb salvage resection ± adjuvant radiation**