Rheumatoid Arthritis
I. Definition
A chronic, inflammatory, systemic disease that produces its most common and most prominent manifestations in the diarthrodial joints.

II. Epidemiology
Age Predilection: 20-60 years old (majority is between 35-40 y.o.)
Sex Predilection: F<M (3:1) (after 65 y.o., there is equal distribution)

III. Etiology
- Unknown
- Believed to be autoimmune because individuals with RA produces antibodies to their own immunoglobulin.
- Rheumatoid Factor (RF) – auto antibodies with specificity for the FC fragment of IgG are found in 70% of RA pts.

IV. Pathophysiology
- Synovial inflammation – venous distention, capillary obstruction, neutrophilic infiltration of arterial walls, areas of thrombosis and hemorrhage
- As a consequence of inflammation, the synovium becomes hypertrophic from proliferation of blood vessels and synovial fibroblasts and from multiplication and enlargement of synovial lining layers
- These leads to formation of granulation tissue called PANNUS
- Pannus
  - extends to the articular cartilage as it dissolves the collagen
  - actively invades and destroys the periarticular bone and the cartilage at the margin between synovium and bone
  - will eventually result in adhesions and fibrous or bony ankylosis of the joint
  - the destructive element of RA
  - responsible for most of the deformities in RA
  - the hallmark of RA

V. Clinical Manifestation

Extra-Articular
1. Rheumatoid Nodules
   - most common extra articular manifestation
   - all are RF positive
   - common in extensor surfaces of FA, olecranon, Achilles tendon, ischial area, over MTP joints, flexion or surface of fingers
2. Vasculitic Lesions
   - most common are leukocytoclastic vasculitis and palpable purpura
3. Peripheral neuropathies
   - usually found in elderly RA pt.
4. Ocular involvement
   - keratoconjunctivitis sicca in association with sjogren’s sx
   - Scleritis has worse prognosis
5. Hematologic
   - Anemia is most common
   - Felty’s syndrome characterized by splenomegaly, neutropenia, thrombocytopenia and anemia in long standing RA

Systemic Manifestation
1. malaise and low grade fever
2. anorexia
3. weight loss
4. fatigue

**Articular Manifestation**
- always BILATERAL SYMMETRICAL
- can affect any diarthrodial jt.
  1. Morning stiffness
     - universal feature of synovial inflammation
     - last for more than 2 hours
  2. Tenderness
  3. Structural Damage and Crepitus
     - caused by cartilage loss and erosion of periarticular bone
  4. Flexor contractures
  5. Finger deformities
     a. Swan neck deformity – more common
     b. Boutonniere deformity
  6. usually affects the hands, wrists, shoulder, knee, C1-C2 jt and midcervical jt.

VI. Diagnostic Criteria
1987 Revised Criteria for the Classification of RA:
1. morning stiffness
2. arthritis of 3 or more jts.
3. arthritis of the hand
4. symmetric arthritis
5. rheumatoid nodule
6. serum RF
7. radiographic changes
* RA = @ least 4 out of & criteria
*1-4 – must have been present for 2 least 6 weeks
*2-5 – observed by physician
*6&7- laboratory findings

VII. Dx

- Laboratory:
  a. ESR
  b. RF
  c. CBC- RBC -dec.; WBC-Normal
  d. Synovial fluid analysis
     Normal-transparent, yellowish, viscous with clots
     With inflammation-cloudy, less viscous, will clot

- Radiography
  o Marginal erosion with juxta-articular osteoporosis
  o Classification of progression of RA:
     Stage I-early
     Stage II- moderate
     Stage III- severe
     Stage IV- terminal

VIII. Prognosis

- individuals with RA may live less
- almost 50% will eventually have marked restrictions in ADL or will be incapacitated
- elderly onset, better functional outcome than those with early onset
*American College of Rheumatology Revised Criteria for Classification of Functional Status in RA:
  Class I  - independent
  Class II - able to perform with pain
  Class III - able to do some
  Class IV - unable to perform

IX. Medical/Surgical Mgt.
salicylates
NSAIDS-1st line of defense
Anti-malarial drugs
D-penicillainine
Steroids-exogenous glucocorticoids

X. PT Mgt.
- superficial heating IR and HMP
- deep heating modalities e.g. MWD, SWD, & US.

**Systemic Lupus Erythematosus**

I. Definition:
- a systemic immune-mediated d/o char. by presence of a number of antibodies to nuclear components.

II. Epidemiology:
- females (8:1), increase in women of child bearing age
- 15-45 y.o.
- 2-4 times greater in blacks and Hispanics than in whites
- may be hereditary or genetically determined, greater in identical twins.

III. Etiology:
- unknown
- (+) of regulatory mechanisms in immune response

IV. Clinical Manifestations:
- the 11 criteria: SHIN ROAD MAP
  - Serositis- pericarditis & pleuritis
  - Hematologic- felty’s syndrome
  - Immunologic
  - Neurologic- psychosis & epilepsy
  - Renal- lupus nephritis
  - Oral- ulcers
  - Arthritis- non-erosive non-deforming (JACCOUD)
  - Discoid rash- ant. Neck & scalp area
  - Malar rash- aka butterfly rash
  - Anti-nuclear antibody (ANA)- lab. Hallmark of SLE
  - Photosensitivity- can’t tolerate light

V. Medical/Surgical Mgt.
- immunosuppressive drugs may be helpful
- salicylate
- corticosteroids

**Juvenile Rheumatoid Arthritis (Still’s dse)**

I. Definition
- an uncommon crippling dse of children associated with fever and enlargement of lymph nodes and spleen.

II. Epidemiology
- >boys=girls
- >most common in childhood
- >1st degree relatives and 40% of monozygotic twins may be affected

III. Clinical Manifestation
- growth retardation
- >abn speech
- >rapid loss of ROM & contractures
- small mandible (microagnathia)

IV. Types
  a. systemic onset
b. pauciarticular arthritis
- 30% of JRA pts.
- Most frequent type
- Girls > boys; early age of onset is 2-4 y.o
- Involvement of 1 or few jts. Common in knees & ankles
- Has mild arthritis; good prognosis
- May lead to cataract irritation, loss of vision and band keratopathy

c. polyarticular arthritis
- 25% of pts.
- Predominantly girls at younger age
- Has relatively good prognosis
- Onset is insidious
- Initial involvement of small jts.

V. Medical/Surgical Mgt.
- Salicylates- 1st drug of choice
- Gold salts be given if salicylates is not effective
- Antimalarials
- Systemic glucocorticosteroids

VI. PT Mgt & Assessment
- goal is relief of pain and maintenance of function
- splinting of jts.
- proper ex.
- adequate rest

Progressive Systemic Sclerosis
(scleroderma or systemic sclerosis)

I. Definition
An uncommon connective tissue dse with the most prominent feature which is THICKENING OR FIBROSIS of the skin. It is heterogenous, both involving the internal organs and joints.

II. Epidemiology
- Common in Females (3:1)
- Rare in children and in men under 30 y.o.
- Slightly more common in black women in childbearing years

III. Etiology
- Etiologic agent is obscure and no strong hypotheses exist to its nature

IV. Pathophysiology
- The abnormal deposition of collagen in the CT of the microvessels causing obliteration, vasomotor(vasospasm) and permeability changes (edema), platelet activation and perimuscular mononuclear cell infiltration leading to inflammation. The injury to endothelial cell lining of the vessels makes the organ damaged since there is the disturbance which activates the clotting system releasing vasoactive peptides. Thus smooth ms migrate in, proliferate and deposit CT to a proliferative vascular lesion of PSS.

V. Clinical Manifestation
- Raynaud’s Phenomenon
  - caused by a spastic blood vessel in the extremities especially in the digits
- presence of pain and numbness especially on toes and fingers of the involved extremity
- ulcerations
- webbing conditions
- compromised blood supply to the digits

-Skin
  - early disease – swollen fingers and hands, forearm, feet, lower legs and face are affected.

-GIT
  - esophageal hypomotility leads to dysphagia or heart burn.
  - gastric hypomotility leads to bloating & abdominal pain.

-Pulmonary
  - exertional dyspnea often accompanied by a non-productive cough

-cardiac
  - pericarditis with or without effusions, heart failure, and varying degrees of heart blocks or arrythmias.

-Renal
  - will manifest encephalopathy, severe h/a, retinopathy, seizures

VI. Complications
- pleuritis, interstitial fibrosis, pulmonary Htn, wt. loss, constipation, dysphagia
- may result to “crest syndrome”
  a. subcutaneous phenomenon
  b. raynaud’s phenomenon
  c. esophageal dysfunction
  d. sclerodactyly
  e. telangiectasia

VII. Medical/Surgical Mgt.
- drug therapy: e.g. penicillamine, antoplatelet, glucocorticoid
- for Raynaud’s phenomenon: reserpine, phenoxybenzamine

Dermatomyositis – Polymyositis (DM-PM)
I. Definition
  It is an inflammatory disease of muscle and skin often associated with profound weakness of skeletal muscle, including the heart, with or without the presence of rash.

II. Epidemiology
- most common in 40 – 60 years old
- children of ages 5 – 15 may acquire it
- males=females

III. Etiology
- unknown cause
- theories: due to viral infection and autoimmune disturbances

IV. Pathophysiology
- there is abnormal recognition of the self in which antibodies of the individual attacks its own self causing damage to the muscle and skin leading to weakness in skeletal and articular muscles

V. Clinical Manifestation
- profound weakness of skeletal muscle
- weakness of respiratory and swallowing muscles
- joint diseases are rare but bony erosions are common

VI. Types of DM-PM
  Type 1 – Primary, Idiopathic PM
insidious onset, mod-severe arthritis, Raynaud’s phenomenon is present
pelvic girdle > shoulder girdle and neck muscle > dysphagia and dysphonia

Type 2 – Primary, Idiopathic DM
- acute onset, proximal muscle weakness and heliotropic rash and grottron’s papules
- muscle tenderness, systemic-fever, malaise, wt. loss

Type 3 – DM-PM associated with malignancy
- more common in M>40 y.o., muscle weakness usually progressive
- dysphagia and respiratory weakness
- death due to pneumonia or respiratory failure

Type 4 – DM/PM associated with vasculitis
- disease of childhood
- rapid and progressive muscle weakness with dysphagia, dysphonia and respiratory weakness
- contracture and atrophy is high
- calcinosis is present

Type 5 – Associated with other collagen vascular diseases
- RA, SLE, PSS
- Functional problems associated with the individual collagen diseases often dominates the clinical picture

Type 6 – Inclusions body myositis

VII. Complications
- Aspiration pneumonia
- lung dysfunction

VIII. Diagnosis
- evaluation of serum muscle enzymes
- muscle biopsy
- EMG
- Steroids not effective

Sjogren’s Syndrome

I. Definition
A chronic, slowly progressive inflammatory autoimmune exocrinopathy which is characterized by dry eyes (keratoconjunctivitis sicca) and dry mouth (xerostomia); the second most common immune-mediated disorder

II. Epidemiology
- Most common in females, 50 yrs old, 9:1

III. Etiology
- unknown
- associated with other autoimmune diseases such as RA, SLE and PSS

IV. Pathophysiology
- two main autoimmune phenomena are: lymphocytic infiltration of exocrine glands and B-lymphocyte hyperactivity

V. Clinical Manifestation
- Keratoconjunctivitis sicca – dryness of the eyes
- xerostomia – dryness of the mouth
- arthritis
- dyspareunia – pain during sexual intercourse
- parotid gland enlargement
- raynaud’s phenomenon
- fever/fatigue
- lymphoma and Waldenstrom’s macroglobulinemia

VI. Complication
- dry nose, throat, and trachea
- esophageal mucosal atrophy
- atrophic gastritis
- fatigability
- renal involvement
- nephritis, vasculitis

VII. Tests
- Schirmer’s Test – test for keratoconjunctivitis sicca
- Rose Bengal Test
- Test for Xerostomia

VIII. Medical/Surgical Management
- Goal
  - aimed at symptomatic relief and limit damaging effects of chronic xerostomia and keratoconjunctivitis sicca
- Management
  - fluid replacement
  - avoid diuretics, antihypertensive and anti-depressant drugs
  - eye patching and boric acid ointments
  - glucocorticoids and immunosuppressive agents

SERONEGATIVE ARTHROPATHIES

Ankylosing Spondylitis
I. Definition:
- systemic, chronic, inflammatory disorder of the axial skeleton, affecting SI joints and spine
- aka von Bechterew’s Disease, Strumpell-Marie Disease, Rheumatoid Disease
- prototype of the Spondyloarthropathies

II. Epidemiology
- more common in males (3:1)
- 20 - 40 yrs. Old
- 90% with HLA-B27 positive (genetic predisposition)

III. Etiology
- unknown
- hereditary

IV. Pathophysiology
- no specific exogenous agents has been identified to trigger the disease
- implicate immunomediated mechanisms
- inflammatory processes tend to start or originate in ligametous and capsular sites of attachment to bones (enthesitis), juxta-articular ligamentous structures, and the synovium, articular cartilage and subchondral bones of involved joints

V. Clinical Manifestation
a. Sacroilitis
  - Hallmark of AS
  - 1st initial symptom
  - dull pain felt in the lower lumbar with back morning stiffness
b. Low Back Pain
c. Bony Tenderness
d. Enthesitis
   ➢ Inflammation of ligamentous tendinous insertions

e. Peripheral Arthritis
   ➢ usually in the shoulder and hip joints

f. Loss of Spinal Mobility
g. LOM in hip and shoulder joints
h. Extraskeletal manifestation

VI. Diagnosis
   Diagnostic criteria:
   ➢ hx of inflammatory back pain
   ➢ (+) sacroilitis
   ➢ LOM of lumbar spine
   ➢ Limited chest mobility

VII. Complications
   ➢ spinal fracture – most serious complication

VIII. Medical/Surgical Management
   Indications for surgery:
   ➢ hip pain and stiffness

Drugs
   ➢ indomethacin – most common
   ➢ NSAIDS
   ➢ Local corticosteroids

IX. PT Assessment
   Exercises
   ➢ early morning warm-ups – to facilitate ADLs
   ➢ neck and back exercises – McKenzie’s ex.
   ➢ ROM ex. Of cervical spine

   Positioning
   ➢ use of a firm mattress
   ➢ use of a Jackson pillow which allows lateral cervical support in sidelying
   ➢ prone positioning of atleast 1 hr daily

Reiter’s Syndrome
I. Definition
   ➢ presents as a clinical triad of non-gonococcal urethritis, conjunctivitis and arthritis

II. Epidemiology
   ➢ Males are more commonly affected
   ➢ Almost 100% HLA-B27 positivity

III. Etiology
   ➢ Believed to be triggered by infection of the genitourinary tract caused by Chlamydia, Campylobacter, Salmonella, Shigella, and Yersinia

IV. Pathophysiology
   ➢ The triggering organisms invade host cells and survive intracellularly
   ➢ Antigens of Chlamydia, Yersinia and Salmonella persist in the synovium for long periods following the acute attack
   ➢ CD4+T cells that respond to antigens of the inciting organisms are typically found in inflamed synovium but not in peripheral blood
   ➢ It remains to be determined where the primary process is an autoimmune response against antigens of triggering organisms that have disseminated to the target tissue

V. Clinical Manifestation
   ➢ Constitutional symptoms: fatigue, fever, malaise and weight loss
   ➢ Musculoskeletal symptoms
Urethritis
Discharge is mucopurulent, prostatitis is common
Conjunctivitis and iritis
Arthritis which begins in the wt. bearing joints (knees, ankles, feet and wrist)
Arthritis of the hands and fingers may give a sausage digit appearance
Skin involvement may include
  - Keratoderma Blenorrhagica – inflammatory hyperkeratotic lesion of the toes, nails and soles of the feet resembling pustular psoriasis
  - Balanitis Circinata – shallow, painless ulcers in glans penis and urethral meatus

VI. Medical Management
  - NSAIDS
  - indomethacin
  - systemic glucocorticoids

VII. Surgical Management
  - synovectomy – for severe joint pain
  - excision arthroplasty for metatarsalgia
  - tenosynovectomy

Psoriatic Arthritis
I. Definition
  - a benign inflammatory skin disease with genetic predisposition
  - an arthropathy associated with combined features of both RA and seronegative spondyloarthropathies
  - a polyarthritis with psoriasis

II. Epidemiology
  - male = female
  - 1% prevalence
  - 30-50 yrs old onset

III. Etiology
  - skin lesions usually antedate the arthritis and exacerbation and remissions of psoriatic arthritis are poorly correlated with the course of skin lesions

IV. Pathophysiology
  - arthritis may affect one digit causing an inflammatory dactylitis
  - severe osteolysis at the opposing articular surfaces may occur in peripheral and in proximal joints
  - tendency to bony fusion may typically be seen and may manifest to patients with generalized psoriatic erythroderma
  - severe resorptive arthroplasty in which a loss of bonestock and joint surface is extensive that the skin overlying the fingers or wrists may fold upon itself – so called Main en Lorgenette syndrome

V. Clinical Manifestation
  - asymmetric oligoarthritis or monoarthritis
  - symmetric polyarthritis resembling RA
  - Auspitz sign – the phenomenon where bleeding occurs when the scaly psoriatic plaques are lifted from the skin
  - Nail findings include stippling and onycholysis
  - Severely deforming arthritis known as arthritis mutilans causes shortening of the fingers secondary to excessive bone resorption plaques
  - Enthesitis
  - Psoriatic lesions are seen
  - In radiographic features: pencil-in-cup deformity
VI. Medical Management
- Hydroxychloroquine – exacerbate psoriasis
- Gold therapy – beign reevaluated
- Immunosuppressive therapy (Methotrexate) – control the disease
- Steroids, Local skin treatment

Inflammatory Bowel Disease
I. Definition
- General term for a group of chronic inflammatory disorders of unknown cause involving the gastrointestinal tract
- 2 Major Groups of Chronic IBD
  1. Chronic non-specific ulcerative colitis
  2. Crohn’s Disease

II. Epidemiology
- Whites > blacks and orientals
- Males = females
- Peak age: 15 – 35 years old

III. Etiology
- Unknown cause
- An immune mechanism may be involved
- Psychological features suggested that patients with IBD have characteristic personality which renders susceptible emotional stresses which may precipitate their symptoms

IV. Pathophysiology
  Chronic Ulceritis
  - Inflammatory reaction in the colonic mucosa and extends proximal in a continuous fashion
  - Backwash ileitis on the entire colon
  - Inflammatory reaction with neutrophilic infiltration which may cause eventual destruction
  - Deeper layers of the bowel beneath the submucosa usually are not involved

  Crohn’s Disease
  - Characterized by chronic inflammation of the intestinal wall and its mesentery
  - Bowel wall is pliable during the early stage
  - As progresses, it appears greatly thickened and leathery with its lumen narrowed
  - Mesentery appears with fingerlike projections
  - Granulomas are usually present and is often discontinuous

V. Clinical Manifestation

<table>
<thead>
<tr>
<th></th>
<th>Ulcerative Colitis</th>
<th>Crohn’s Disease</th>
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</thead>
<tbody>
<tr>
<td>1. Diarrhea</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>2. Rectal bleeding</td>
<td>++</td>
<td>+</td>
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<td>3. Abdominal pain</td>
<td>+</td>
<td>++</td>
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<tr>
<td>4. Palpable mass</td>
<td>0</td>
<td>++</td>
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<tr>
<td>5. Fistulas</td>
<td>+/-</td>
<td>++</td>
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<tr>
<td>6. Small bowel mov’t</td>
<td>+/-</td>
<td>++</td>
</tr>
<tr>
<td>7. Rectal involvement</td>
<td>++ (95%)</td>
<td>++ (50%)</td>
</tr>
<tr>
<td>8. Toxic megacolon</td>
<td>+</td>
<td>+/-</td>
</tr>
<tr>
<td>9. Recurrence after colectomy</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>10. Malignancy</td>
<td>+</td>
<td>+/-</td>
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VI. Medical/Surgical Management
General Measures
- bed rest
proper diet

Drug Treatment
- salicylates – ASA is the drug of choice
- NSAIDs
- Steroids
- Gold salts
- Anti-malarials
- Methotrexate

Local treatment of Joints
- Heat
- Active exercises
- immobilization

Prevention of Deformity
- splinting
- exercise

VII. Surgical Management:
1. Synovectomy
   - performed in RA to relieve pain & inflammation associated with chronic swelling.
   - To alleviate or restore ROM in contracted jts.
2. Tenosynovectomy
   - most frequently in extensor-flexor tendons of the hand and TA.
   - Major C/I:
     a. very active polyarticular dse
     b. poor general medical condition
     c. poor motivation of the pt.
     d. satge 4 in jt. destruction
3. Arthrodesis
   - surgical fusion of bony surfaces of a jt.; usually done in cases of severe jt. pain & instability in which mobility of a jt. is a lesser concern.
   - Indications:
     a. relieve persistent pain
     b. provides stability where there is mechanical destruction of jt ant to halt progress of dse.
   - C/I:
     a. significant bilat. Jt. dse.
4. Tendon transfer
   - common in RA for the ff:
     a. ruptured tendons of the body
     b. tendon release
5. Osteotomy
   - help to correct valgus deformity in JRA
6. Jt. replacement
   - indications:
     a. persistent pain
     b. LOM
     c. Loss of function

Complications of Jt. replacement:
- Loosening
- Early or late infection
- Dislocation
- Fx of bone
- Wearing out of components
Nerve injury
- Pulmonary embolus

Pre-operative Rehab mgmt:
- teaching the pt. crutch walking
- wt. reduction in obese individual
- strengthening of quads before knee replacement
- strengthening of adductors after knee replacement

Post-Op Rehab Mgt:
- perform pain free ROM ex.
- Perform ms strengthening ex. In pain free range
- Encourage the use of proper assistive device

CRYSTAL-INDUCED ARTHROPATHIES.

Pseudogout Arthritis
I. Definition:
The deposition of calcium pyrophosphate dehydrate crystals in the joints characterized by acute inflammatory jt. disease. This is known as chondrocalcinosis.

II. Epidemiology:
- affects older individuals in their 4th-6th decade
- affects in both gender
- prevalence increases with age
- ratio of pseudogout with gout in incidence is 2:3

III. Etiology:
- unknown
- associated with metabolic d/o:
  a. hyperparathyroidism
  b. hypothyroidism
  c. hemochromarosis
  d. hypophosphatasia
  e. hypomagnecemia
  f. gout
  g. ochronosis
  h. wilson’s dse.

IV. Pathophysiology:
- 3 possible mechanisms:
  a. lowering of either Ca or pyrophosphate ions in the synovial fluid may loosen and shed crystals from cartilage into the synovial fluid.
  b. Crystal may enter synovial fluid secondary to mechanical destruction of cartilage resulting from microfractures of subchondral bone.
  c. Release of crystals from degradation of cartilage matrix by enzymes.

V. Clinical Manifestation:
- patterns of joint involvement:
  a. pseudogout
    - occurs 25% of cases
    - onset is rapid; peak in 12 to 36 hours
    - joints are edematous, swollen, warm & painful
    - usually confined in single joint
    - may provoke trauma, surgery, or medical illness
    - (+) radiographic evidence of chondrocalcinosis
  b. pseudorheumatoid dse.
    - occurs approx. 5% of cases
    - multiple jt. involvement
    - subacute attacks last to weeks or months
    - develops synovial proliferation, LOM, & flexor deformities.
c. chronic
  ➢ more in women
  ➢ progressive degeneration of multiple joints
  ➢ jt. involvement is usually symmetrical
  ➢ affects mostly in knees followed by wrist, MCP, hips, shoulders, elbows, and ankles
  ➢ may resemble neuropathic arthropathy

VI. Classifications
  ➢ hereditary type
  ➢ CPDD associated with metabolic dses.
  ➢ Idiopathic CPDD
  ➢ CPDD concomitant with OA

VII. Medical/Surgical Management
  ➢ phenylbutazone
  ➢ indomethacin
  ➢ NSAIDS
  ➢ Glucocorticoids
  ➢ Salicylates

Gouty Arthritis
I. Definition:
  ➢ a familial d/o of purine metabolism in which uric acid is involved.
  ➢ Charac. by hyperuricemia and deposition of Na urate in the jts.

II. Epidemiology:
  ➢ occurs after age 30
  ➢ 90% of patients are male
  ➢ rare in blacks
  ➢ affects single jt.
  ➢ affects primarily the Big toe - podagra

III. Etiology:
  ➢ maybe due to:
    a. alcohol intake
    b. dietary excess of purine
    c. trauma
    d. drugs
    e. radiation therapy

IV. Pathophysiology:
  ➢ probable causes:
    a. sustained hyperuricemia leads to development of microphi (tophi are pathognomonic features of gout) into synovial lining cells.
    b. Accumulation of monosodium urate in the cartilage in proteoglycans that has higher affinity.
    c. Episodic release of urate crystals in the synovial fluid due to several mechanisms involving disruption of microrphi turn over of cartilage proteoglycans.
    d. Lower temp. in jt, space on an unequal distribution of water and urate in the synovial fluid may accelerate precipitation.

V. Clinical Manifestations:
  ➢ recurrent acute monoarticular pain (early stage)
  ➢ inflammation
  ➢ attacks precipitated by excessive protein intake, drugs, fasting, alcohol abuse, and trauma.
  ➢ Rapid onset
  ➢ Sx free between attacks
Most involved joints are foot (1st MTP), hand, wrist, knee, and elbow
(+ ) tophi

VI. Diagnosis:
   A. X-ray
      ➢ early stage: no joint changes
      ➢ later stage: typical small punch-out areas containing uratic deposits
        at the ends of the joints.
   B. Laboratory findings:
      ➢ elevated blood uric acid

VII. Medical/Surgical Management:
   ➢ colchicines
   ➢ phenylbutazone
   ➢ indomethacin
   ➢ ibuprofen
   ➢ other NSAIDS

*Prevention of tophaceous deposits:
   ➢ xanthine oxidase inhibitor
   ➢ allopurinol
   ➢ uricosuric agent
   ➢ increase fluid intake
   ➢ alkalinization of urine

PT EVALUATION & MANAGEMENT FOR SEROPOSITIVE / SERONEGATIVE
ARTHROPATHIES & CRYSTAL-INDUCED ARTHROPATHIES

PT evaluation & assessment:
1. Hx.
2. ROM
3. palpation
4. MMT
5. joint stability
6. Endurance
7. functional assessment-ADL's
8. Gait assessment-in LE affectation
9. psychological status

PT MGT.
1. Problems
   a. pain
   b. joint stiffness & LOM
   c. muscle atrophy
   d. deformities
2. Goals & Plan of Care
   Goal Plan of Care
   a. pain relief -heat; cold therapy; splints for immobility
   b. maintain joint mobility -PROM & AROM, stretching exercises
   c. muscle integrity -muscle strengthening exercises
   d. prevent deformities -pt. education; bracing; assistive device
3. Rationale of Tx
   a. decrease pain
   b. increase or maintain strength
   c. increase functional endurance
   d. maintain ROM
   e. promote independence
   f. increase joint stability
   g. improve gait patterns
DEGENERATIVE ARTHROPATHIES

OSTEOARTHRITIS
Osteoarthritis, Degenerative Joint Disease (DJD), Hypertrophic Arthritis, Degenerative Disc Disease (DDD, in the Spine), Generalized Osteoarthritis (Kellegren's Syndrome)

I. Definition:
- A slowly progressive musculoskeletal disorder
- Affects the joints of the hands (those involved with a pinch grip), spine and weight bearing joints (hip, knee) of LE
- The most common articular disorder

II. Epidemiology:
- Associated with increased age
- More common in women than men
- Radiographic evidence in > 50-80% of those 65 y/o.
- Estimated 2-3% of the audit population has symptomatic OA.

III. Risk Factors for OA:
- Obesity
- Heredity (esp. OA of the DIP joints)
- Age
- Previous Joint Trauma
- Abnormal Joint Mechanics (Excessive knee varus or valgus)
- Smoking (may contribute to degenerative joint disease)

IV. Pathologic Features of OA
A. EARLY:
- Swelling
- Loosening of collagen framework structure restraint
- Chondrocytes increase proteoglycan synthesis but also release more degradative enzymes.
- Increase Cartilage Water Content

B. LATER
- Degradative Enzymes break down proteoglycans faster than it can be produced by chondrocytes, resulting in diminished proteoglycan content in the cartilage.
- Articular Cartilage thins and softens (joint-space narrowing will be seen eventually)
- Fissuring and cracking of cartilage. Repair attempted but inadequate
- Underlying bone is exposed, allowing synovial fluid to be forced by the presence of wt into the bone. This shows up as cyst or geodes on radiographs
- Remodelling and hypertrophy of the subchondral sclerosis and osteophytes ("spurs") formation

V. CLASSIFICATION OF OSTEOARTHRITIS:
A. PRIMARY OR INDIOPATHIC OA:
1. LOCALIZED:
   - Hands (Heberden's and Bouchard's, First CMC)
   - Hands (Erosive, Inflammatory)
   - Feet (first MTP)
   - Hip
   - Knee
   - Spine
2. GENERALIZED (KELLENGREN'S SYNDROME)
B. SECONDARY OA:
   1. Pain in involved joints
   2. Pain worse activity, better with rest
   3. Morning stiffness (if present) < 30 mins
4. Stiffness after a period of immobility (gelling)  
5. Joint enlargement  
6. Joint Instability  
7. Limitation of joint mobility  
8. Periarticular Mm atrophy  
9. Crepitus  

VI. JOINTS TYPICALLY INVOLVED IN PRIMARY (IDIOPATHIC) OA:  
1. DIP jts of hands  
2. Pip jts hands  
3. First CMC jts of thumb  
4. Acromioclavicular jt  
5. Hip  
6. Knee  
7. First NTP jts of the feet  

VII. RADIOGRAPHIC FEATURES:  
A- No ankylosis  
Alignement may be abnormal  
B- Bone Mineralization  
Bony Subchondral sclerosis  
Bony Spurs (Osteophytes)  
C- No Calcification in cartilage  
Cartilage space narrowing which is non-uniform (occurs in area of maximal stress in wt bearing jts.)  
D- Deformities of Heberden’s Bouchard’s Nodes  
E- No erosions  
"Gull wing" Sign in Erosive Arthritis  
S- Slowly progression over years  
No specific nail in degenerative Disc Disease (a collection of nitrogen in a degered disc space)  

VIII. Laboratory Findings:  
- ESR normal  
- RF Negative  
- ANA not present  
- Synovial Fluid  
  High Viscosity with good string sign  
  Color is yellow and clear  
  WBC counts typically < 1000-2000/ mm3  
  No crystals and negative cultures  

IX. Differential Dx of OA and RA  
<table>
<thead>
<tr>
<th>OA</th>
<th>RA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. non systemic</td>
<td>systemic</td>
</tr>
<tr>
<td>2. non-inflammatory</td>
<td>assoc. with cutaneous and</td>
</tr>
<tr>
<td></td>
<td>inflammatory changes</td>
</tr>
<tr>
<td>3. affects wt. bearing jts</td>
<td>small jts.</td>
</tr>
<tr>
<td>4. (-) RF</td>
<td>(+) RF but not all</td>
</tr>
<tr>
<td>5. (-) subcutaneous</td>
<td>(+) subcutaneous nodes</td>
</tr>
<tr>
<td>6. Normal ESR and Serologic</td>
<td>inc. ESR; Leukocytosis with</td>
</tr>
<tr>
<td>test</td>
<td>eosinophilia</td>
</tr>
<tr>
<td>7. clear synovial fluid; high viscosity</td>
<td>synovial fluid is turbid; low viscosity</td>
</tr>
<tr>
<td></td>
<td>and few cells with many</td>
</tr>
<tr>
<td></td>
<td>polymorphonuclear cells</td>
</tr>
<tr>
<td>8. (+) osteophytes</td>
<td>(-) osteophytes</td>
</tr>
<tr>
<td>9. DIP affectation</td>
<td>terminal jts not usually affected</td>
</tr>
<tr>
<td></td>
<td>( ex. DIP)</td>
</tr>
<tr>
<td>10. involve fewer jts</td>
<td>involves many jts at particular time</td>
</tr>
</tbody>
</table>

- OA is sometimes difficult to differentiate with RA because sometimes the two
may co exist.  
➢ OA maybe stipulated by gouty, neuropathic or tuberculous jt dse.  
CLASS:  
a. Primary OA  
- affects DIP, PIP, 1st CMC, hip, knee, MTP, cervical and lumbar jt.  
b. Secondary OA  
- See ETIOLOGY  
X. MEDICAL MANAGEMENT  
General Measures:  
a. reassurance  
b. rest/ modification of activity  
DRUGS:  
a. Aspirin-drug of choice  
b. NSAIDS  
c. Corticosteroid  
LOCAL TREATMENT:  
a. Splints/ braces  
b. Massage  
c. Exercise  
XI. SURGERY - last resort  
Indications:  
a. Severe pain  
b. Loss of function  
c. Progression of deformity  
SOFT TISSUE PROCEDURES  
a. Synovectomy  
b. Soft tissue release  
c. Tendon transfer  
BONE AND JOINT PROCEDURES  
a. Arthrodesis  
- to relieve pain, result to a very stable joint but sacrifices freedom of motion  
b. Osteotomy  
- improve jt alignment  
c. Arthroplasty  
- jt replacement to relieve pain and restore fxn  

CERVICAL SPONDYLOSIS  
I. DEFINITION:  
Spondylosis is described as the degenerative changes which occur to the intervertebral disc and vertebral bodies.  
II. EPIDEMIOLOGY:  
➢ Common in advancing age (esp. in the cervical spine)  
➢ Less than 40 y.o (asymptomatic), 25% have DJD, 4% have foraminal stenosis.  
➢ More than 70 yo: 70% have degenerative spine changes.  
III. ETIOLOGY  
■ No specific cause  
■ Factors contributing to degenerative changes of the spine:  
a. Aging  
b. Trauma  
c. Work activities  
d. Genetics  
IV. PATHOPHYSIOLOGY  
1. IV disc loose hydration with age, leading to cracks and fissures.  
2. Disc subsequently collapses owing to biomechanical incompetence causing annulus to bulge outwards
3. Surrounding ligaments also lose their elastic properties and develop traction spurs.
4. Uncovertebral spurring occurs as a result of the degenerative process in which the facet joints lose cartilage, become sclerotic and develop osteophytes.
5. Stenosis due to spur formation, disc protrusion, ligamentum hypertrophy.

V. CLINICAL MANIFESTATION
a. Morning neck pain
b. Stiffness
c. Neck fatigue late in the day
d. Loss of neck ROM
e. Pain at the extremes of ROM extension ROM is affected first
f. Sometimes crystallization

VI. COMPLICATIONS
a. Neurological deficits
b. Vertebral artery injury- (due to facet osteophyte formation)
c. Myelopathy- (if arthritis is combined with disc degeneration or post disc herniation)
d. Cervical spinal stenosis

VII. DIAGNOSIS:
1. Plain films- later radiograph
2. CT scan- (to R/O fx)
3. MRI- Most sensitive

VIII. MEDICAL MANAGEMENT
1. Long hot shower for morning stiffness
2. Soft cervical collar
3. NSAIDS
4. Acetaminophen- NSAIDS posses unacceptable medical risk for complication.

PT EVALUATION AND MANAGEMENT OF OSTEOARTHRITIS AND CERVICAL SPONDYLOSIS

PT EVALUATION
A. Objectives of Rehabilitation
   a. To improve function
   b. To prevent/remedy musculoskeletal impairment
B. Assessment
   1. HPI
   2. ROM
   3. Strength
   4. Endurance
   5. Joint stability
      a. Ligamentous laxity
      b. Ligamentous instability
   6. Functional Assessment – ATDEP
   7. Functional mobility and gait analysis

PT MANAGEMENT
A. Gen Guidelines:
   1. Problems
      a. Joint stiffness
      b. Pain due to stress/excessive activity
      c. LOM due to progression of condition
      d. If present, pain at rest
      e. Deformities
   2. Goals and Plans of Care
      GOAL
      a. dec. joint stiffness  
      Plan of Care
      PROM progressing to AROM; joint play techniques; Pt education
b. dec. pain from mechanical strengthening ex., modification of stresses activities with intermittent rest pd. Stretching exercise
c. Inc. ROM grade 1 & 2 oscillation and modalities d. prevent deformities braces, pt education

**Specific Jt. Problems**

1. Hip pain- usually felt around greater trochanter - may radiate to the groin but often experienced in L3 dermatone
   Mx: use of assistive devices to decrease mechanical stresses in ambulation
2. Tredelenburg Gait- due to abductor weakness
   Mx: Isometric exercise to gluteus medius; use o assistive devices
3. Limitation of hip extension- leads to backache due to attempted extension
   Mx: maintenance of Hip ext by lying prone position ( McKenzie #1) for 30-40 min. bid.
4. Inc. Leg length of the affected side- associated with unilateral hip ossification
   Mx: Shoe modification
5. Knee pain- causes LOM
   Mx: Modalities with rest, elastic wrap or splint; grade 1 oscillation.
6. Knee jt effusion inhibits voluntary contraction of the squads
   Mx: ROM exercise
7. Restricted ROM due to contractures of the knee jt capsules and hamstrings
   Mx: Stretching exercise
8. Genu Varum
   Mx: shoe modification
9. Hallux Valgus with Bunions
10. Hallux Rigidus
11. Abrasions at soles/ dorsum of toes
12. Metatarsal head calluses
    Mx: Use of proper footwear/ shoe modification
13. CMC Jt pain
    Mx. use a functional thumb post splint to relieve pain and allow functional activities
14. Inc tension in the Spine
    Mx. Relaxation techniques; traction to inc. IV foramen diameter