Radiology Overview of Arthritic Processes

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EDUCATION RESEARCH ADVOCACY

DISCLOSURES

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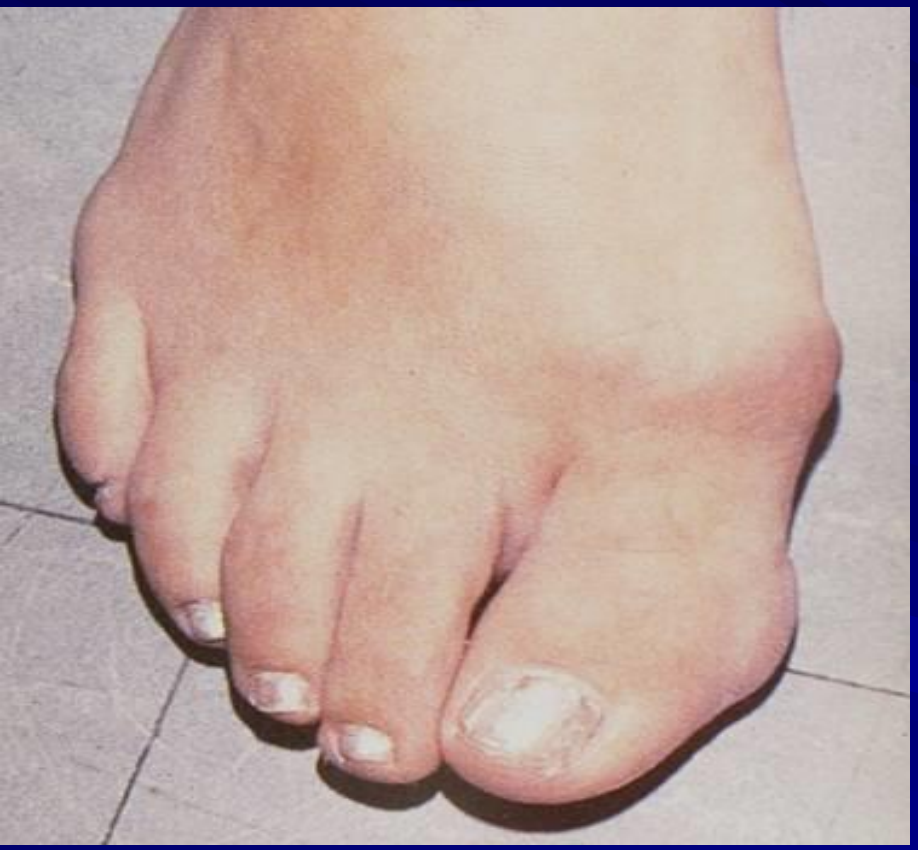
Classification of Arthritides

- **Non-Inflammatory**
  - Degenerative Joint Disease
- **Inflammatory**
  - **Seropositive Arthritide:**
    - Rheumatoid Arthritis
  - Erosive Osteoarthritis
  - **Seronegative Arthritides:**
    - Psoriatic Arthritis
    - Reactive Arthritis (Reiter's Disease)
    - Ankylosing Spondylitis
    - Enteropathic
- **Metabolic**
  - Gout
  - CPPD Disease
- **Neuropathic**
  - Neuropathic Joint Disease
- **Miscellaneous**
  - DISH **(Diffuse Idiopathic Skeletal Hyperostosis)**
  - PHO **(Pulmonary Hypertrophic Osteoarthropathy)**
  - PVNS **(Pigmented Villonodular Synovitis)**
- **Collagen Vascular Disorders**
  - SLE
  - Scleroderma

Degenerative Joint Disease

Degenerative Joint Disease

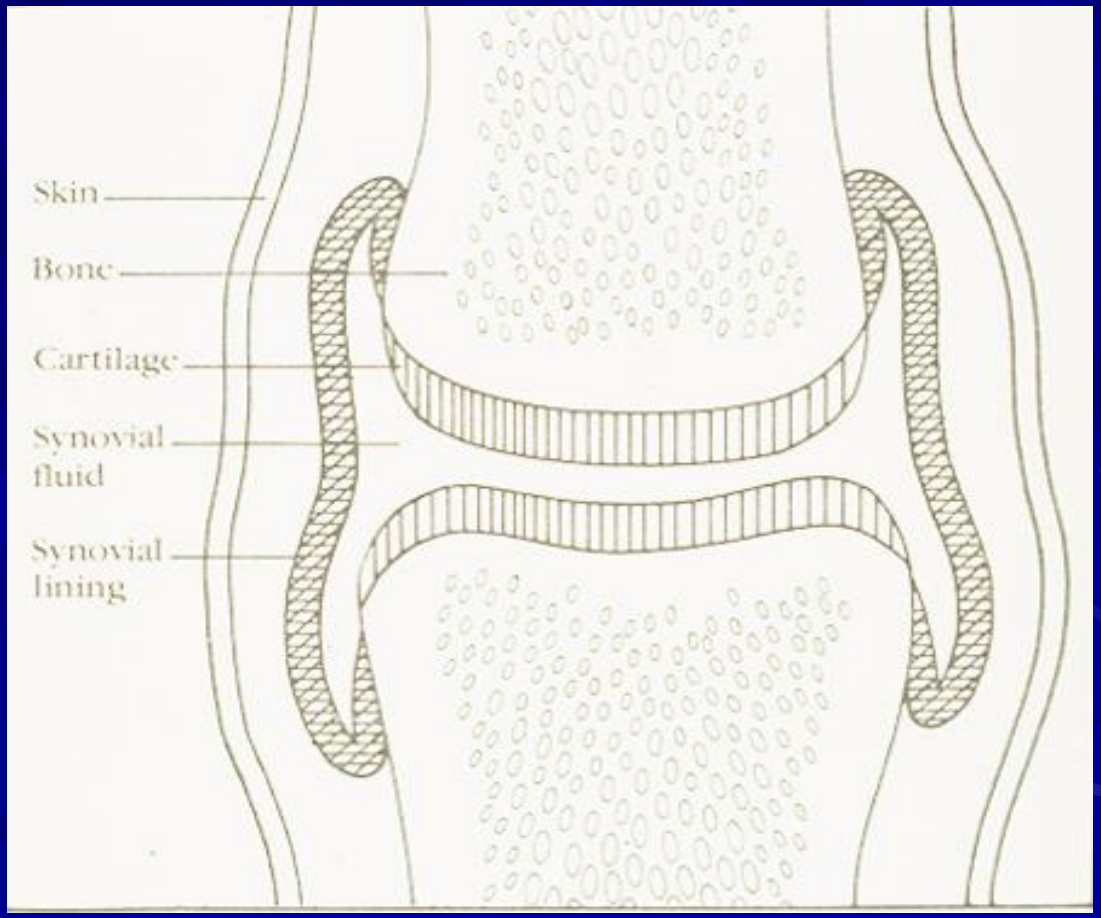


If we evaluate the x-ray before performing our physical exam, we would expect to find decreased ROM of the 1st MPJ left with possible crepitus & pain associated with dorsiflexion of the hallux. We would likely note a palpable exostosis/spur at the dorsal surface of the 1st MPJ as well as POP to that dorsal flag.

Degenerative Joint Disease

In a Normal Joint:

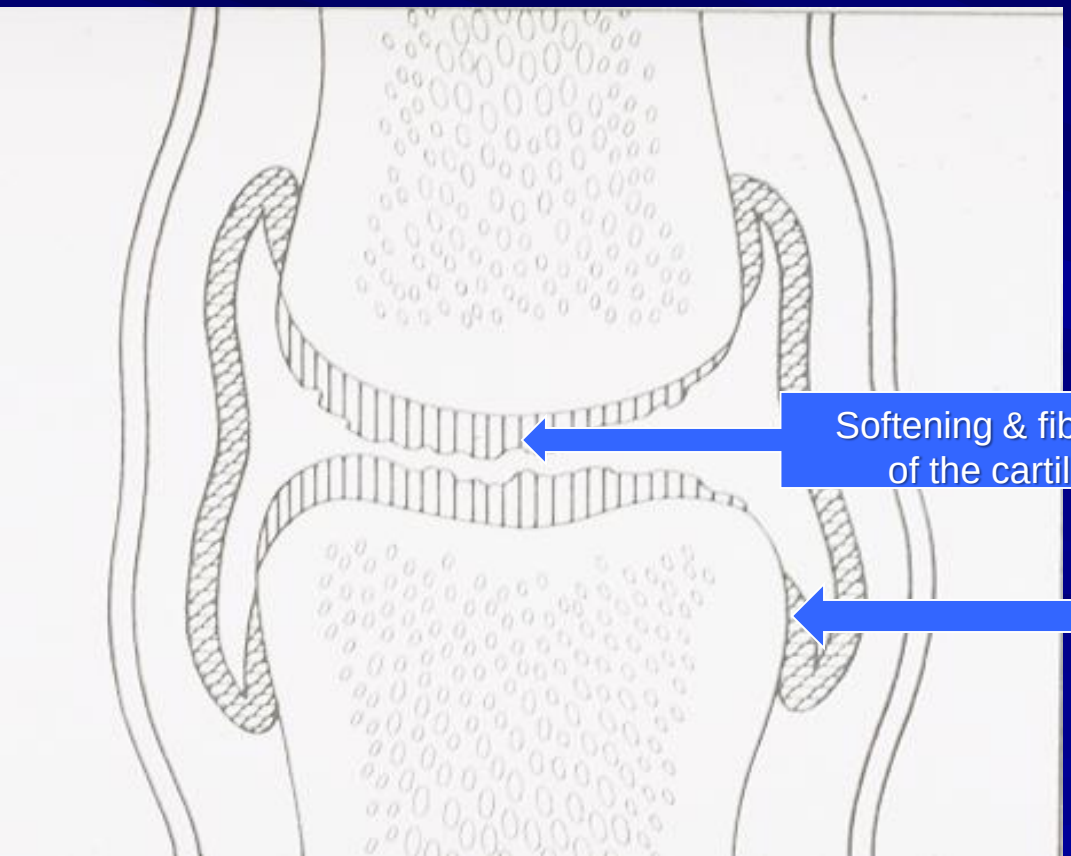
Chondrocytes regulate the extracellular matrix so that the synthesis and degradation of their structural components remain balanced.



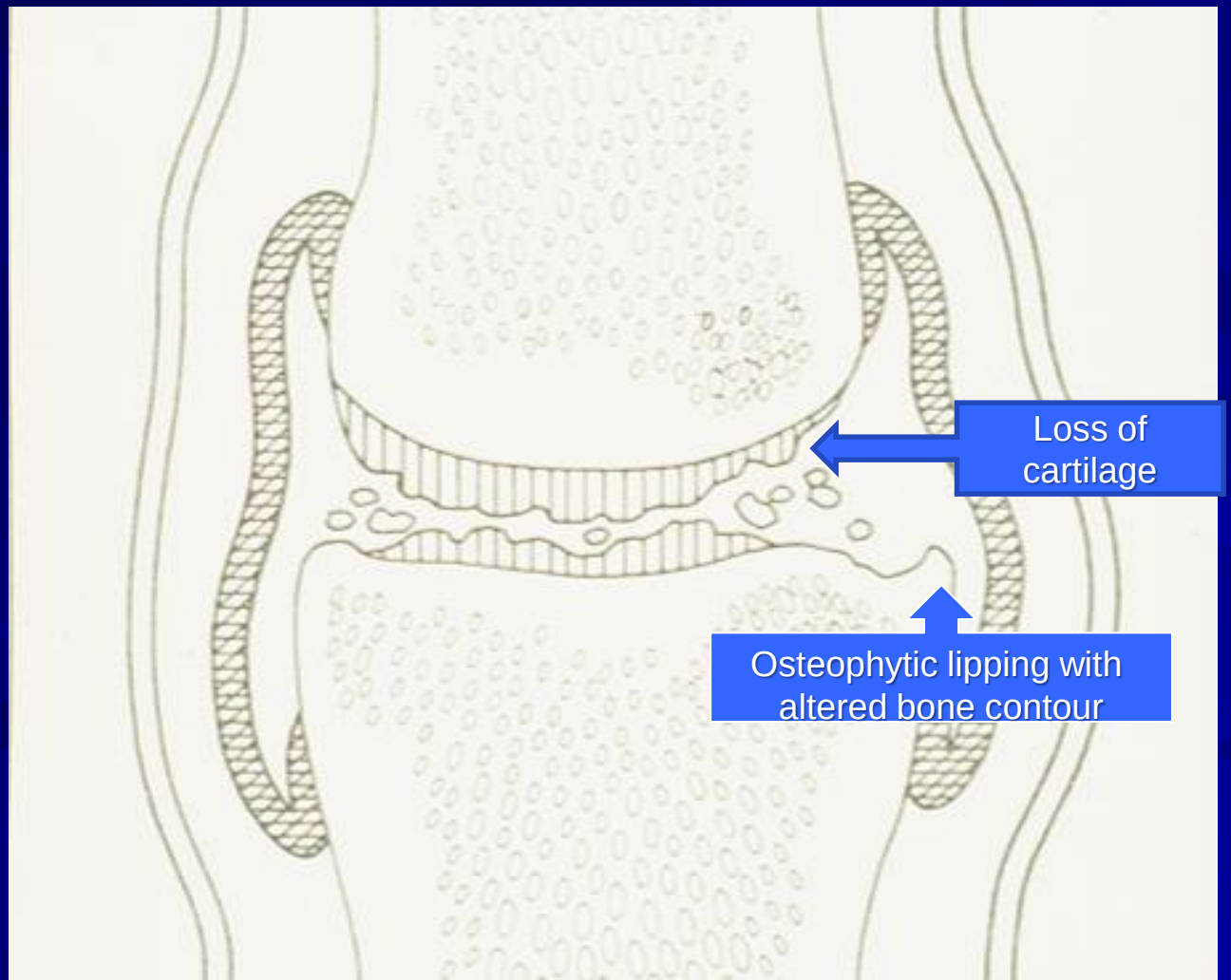
Degenerative Joint Disease

In Degenerative Arthritis:

The degradation and repair processes become unbalanced, with abnormal production of metalloproteases, collagenase, cytokines, and growth factors. This results in weakening & biochemical breakdown/damage of the cartilaginous structure.

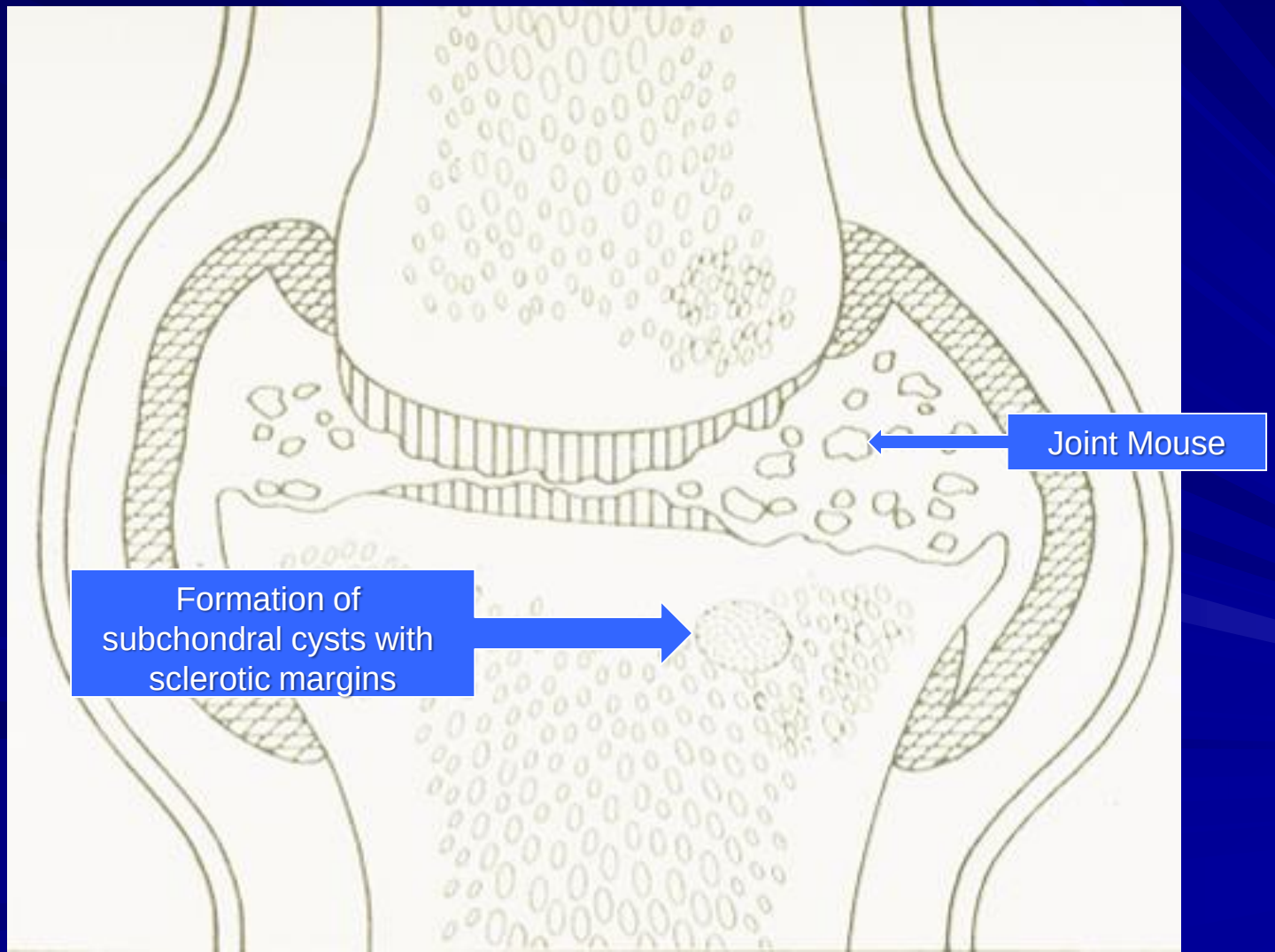


Degenerative Joint Disease

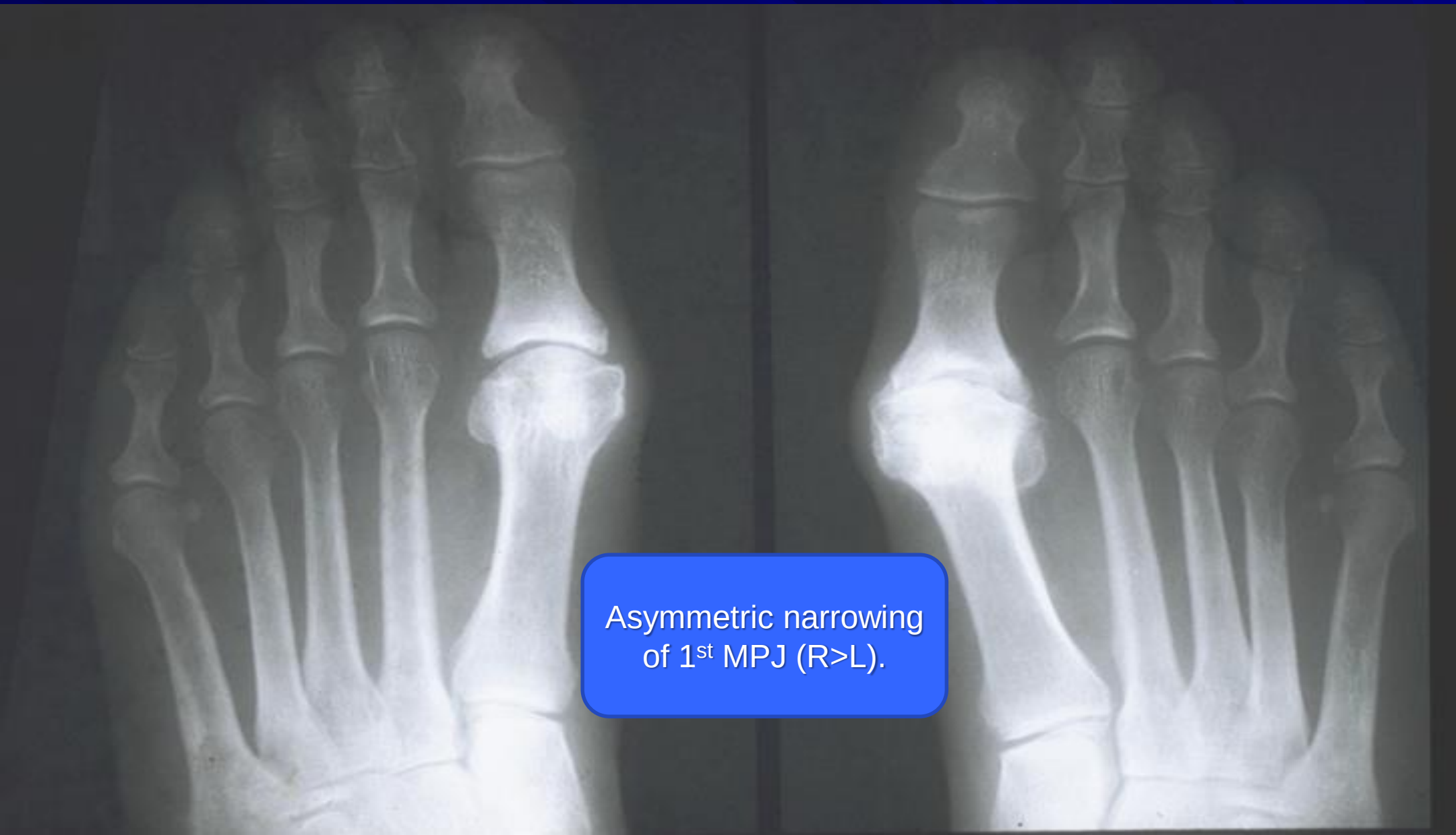


Changes in the proteoglycans make the cartilage less resistant to compressive forces in the joint & more susceptible to the effects of stress. The remaining fibers are placed under additional strain, eventually leading to mechanical failure.

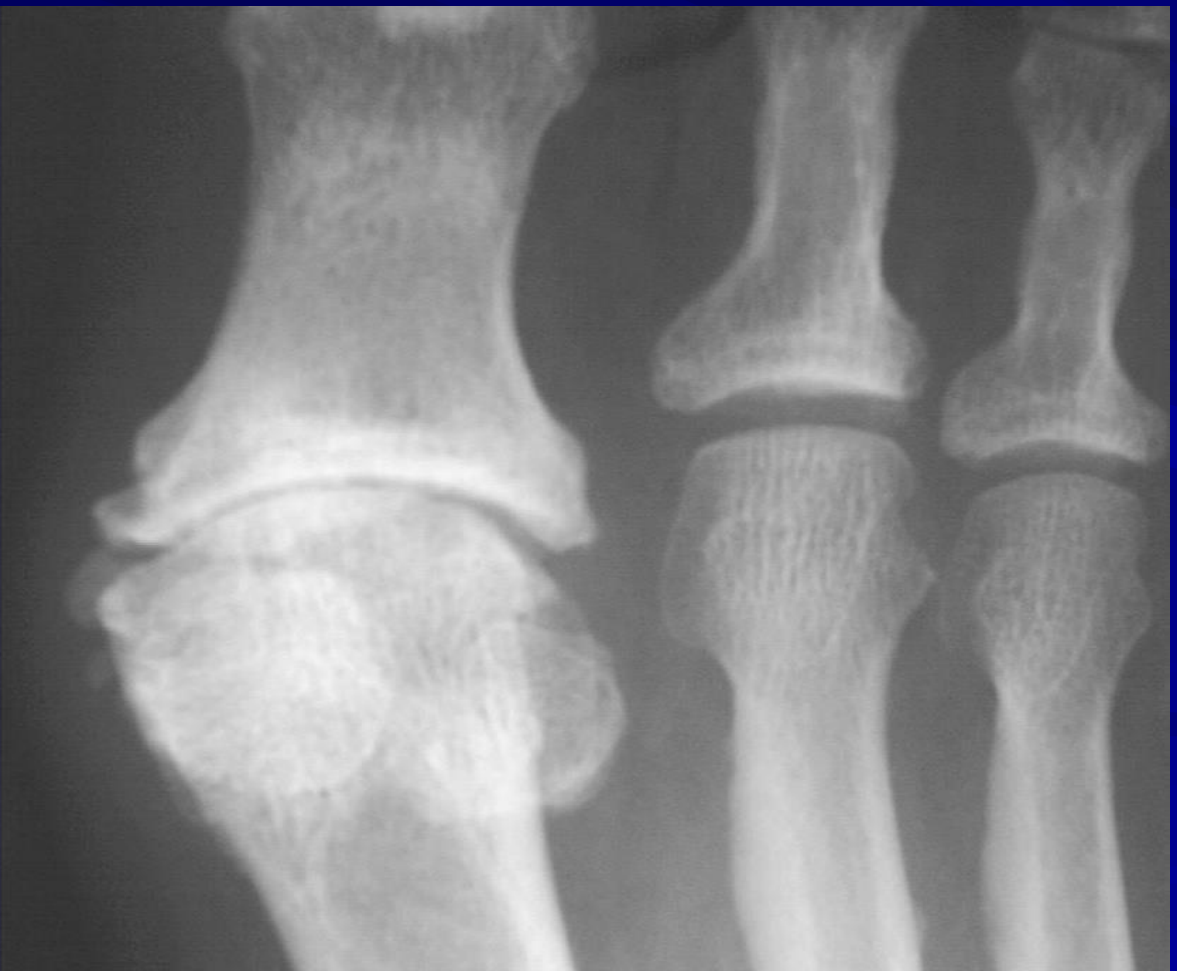
Degenerative Joint Disease

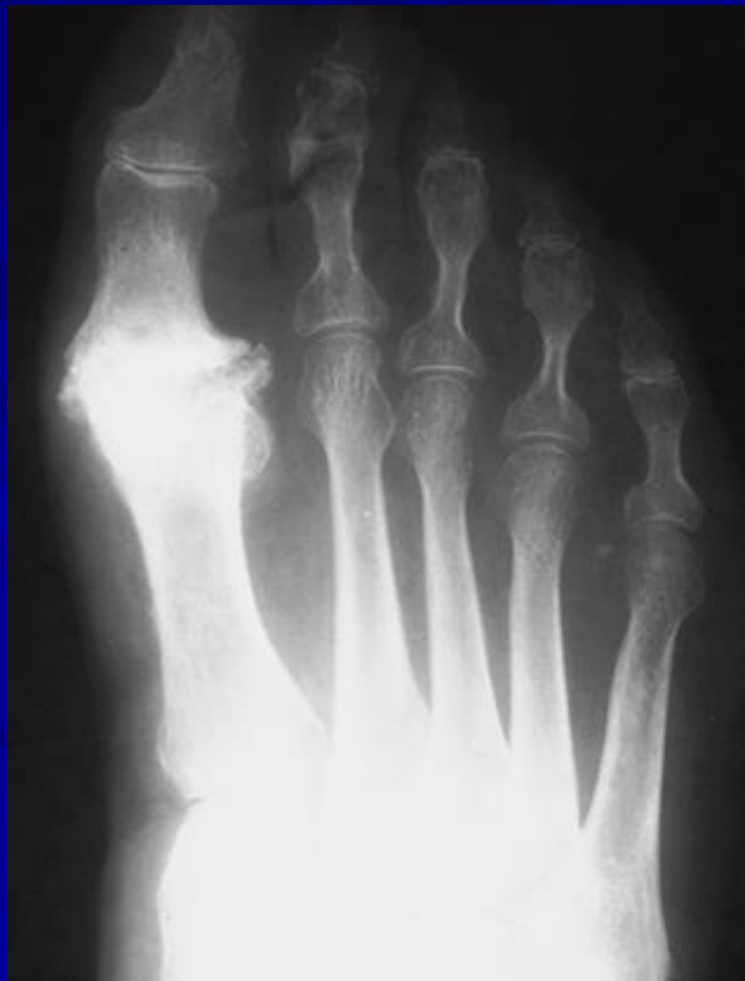


Degenerative Joint Disease



Degenerative Joint Disease



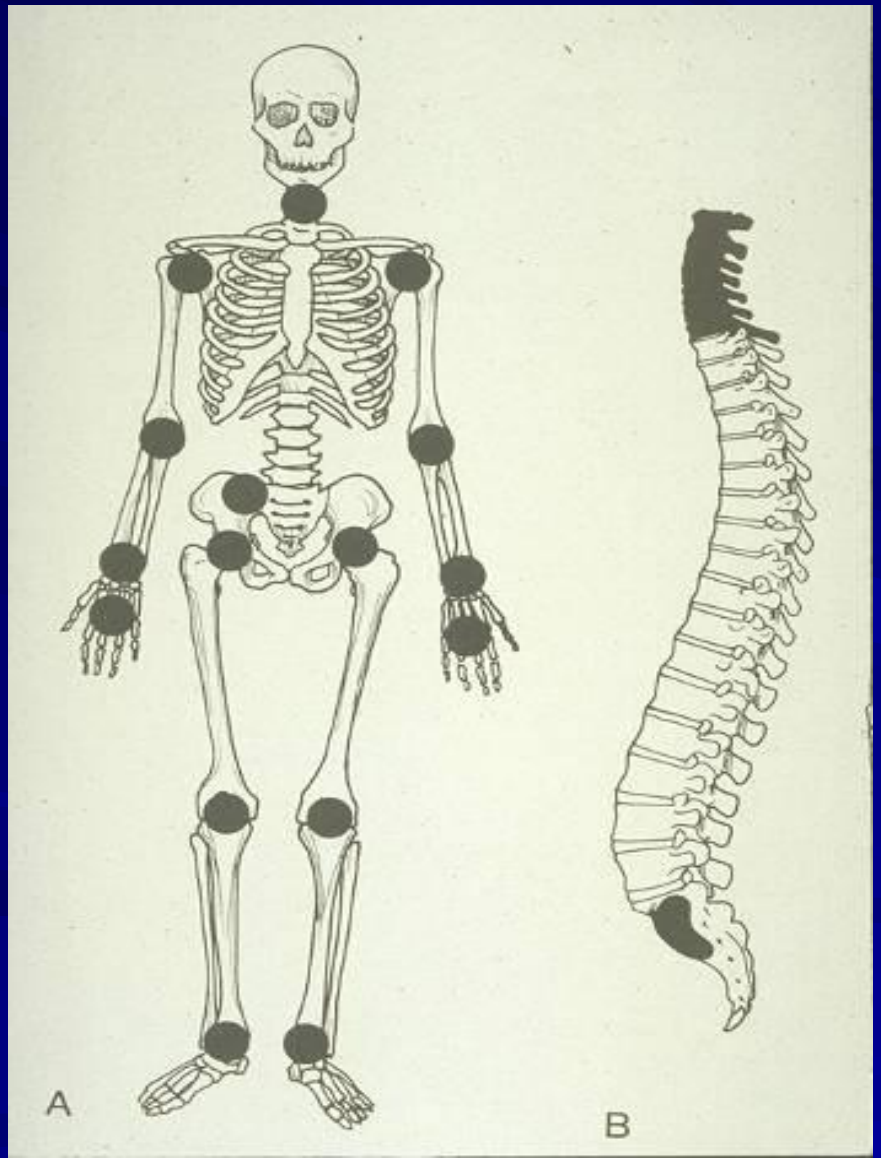


Rheumatoid Arthritis

Rheumatoid Arthritis

- A chronic, systemic inflammatory disorder that may affect many tissues and organs, but principally attacks synovial joints.
- Age of onset: 40 – 60 yoa
  - Peak Incidence: 40 – 50 yoa, males = females
  - Between 20 – 40 yoa, females 3:1 males
- Lab Findings:
  - Seropositive for rheumatoid factor antibodies: 70-90%
  - Positive for Anti-CCP (Cyclic Citrullinated Peptide)
- Distribution:
  - C-spine
  - Bilateral joint involvement
  - Symmetrical joint space narrowing

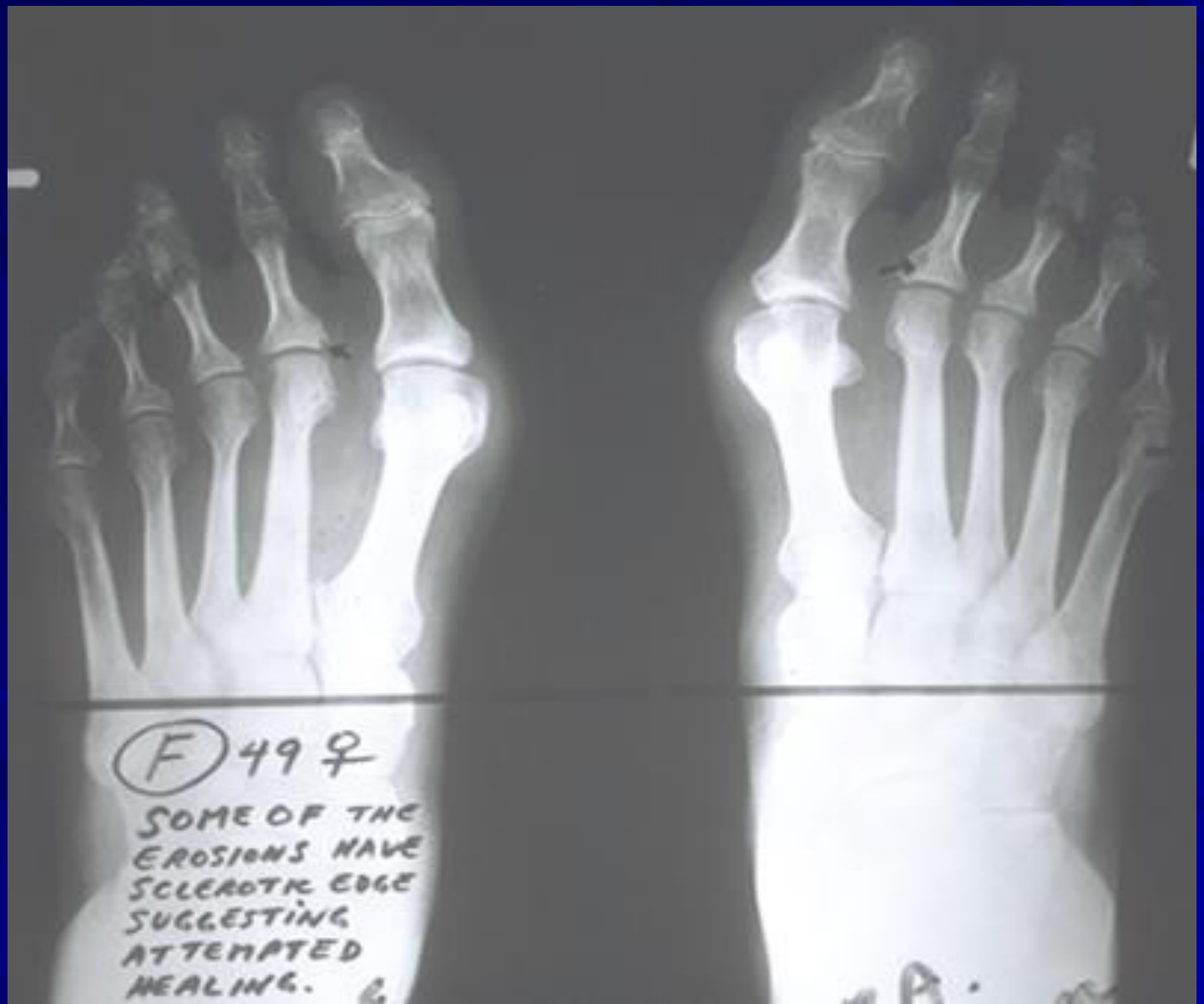
Rheumatoid Arthritis



Bilateral Joint Involvement

With Symmetrical Joint Space Narrowing

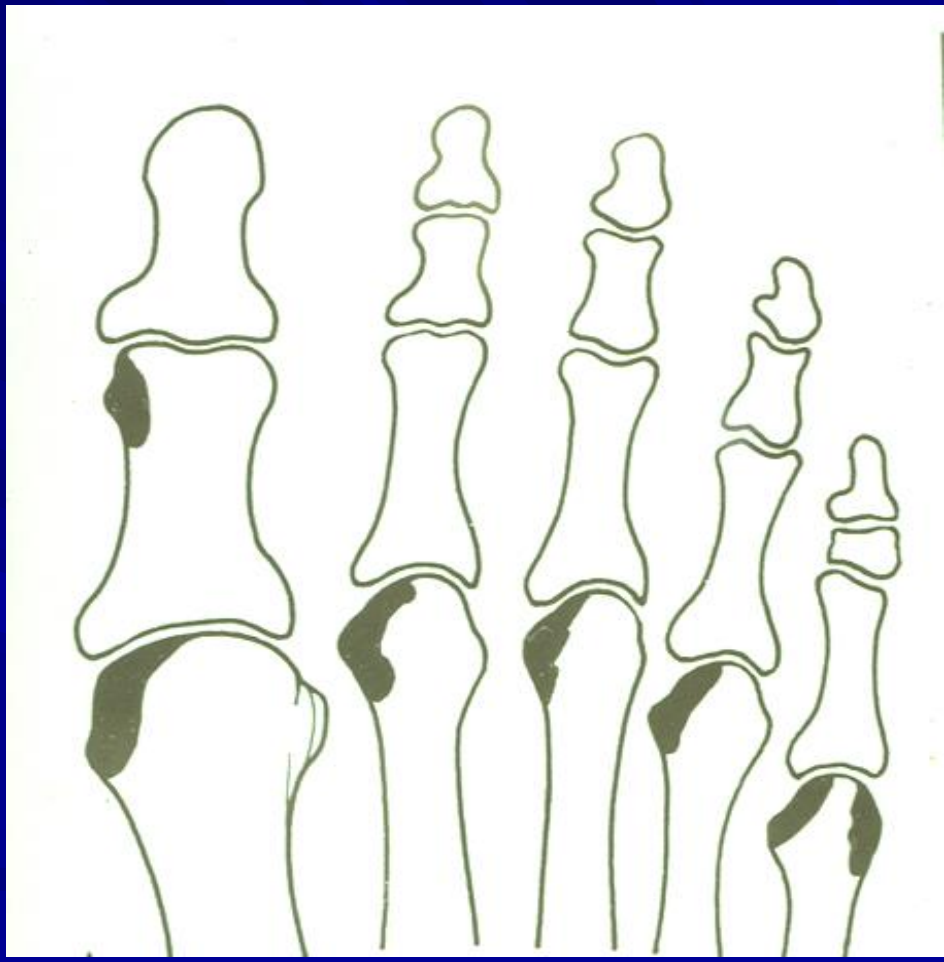
Rheumatoid Arthritis



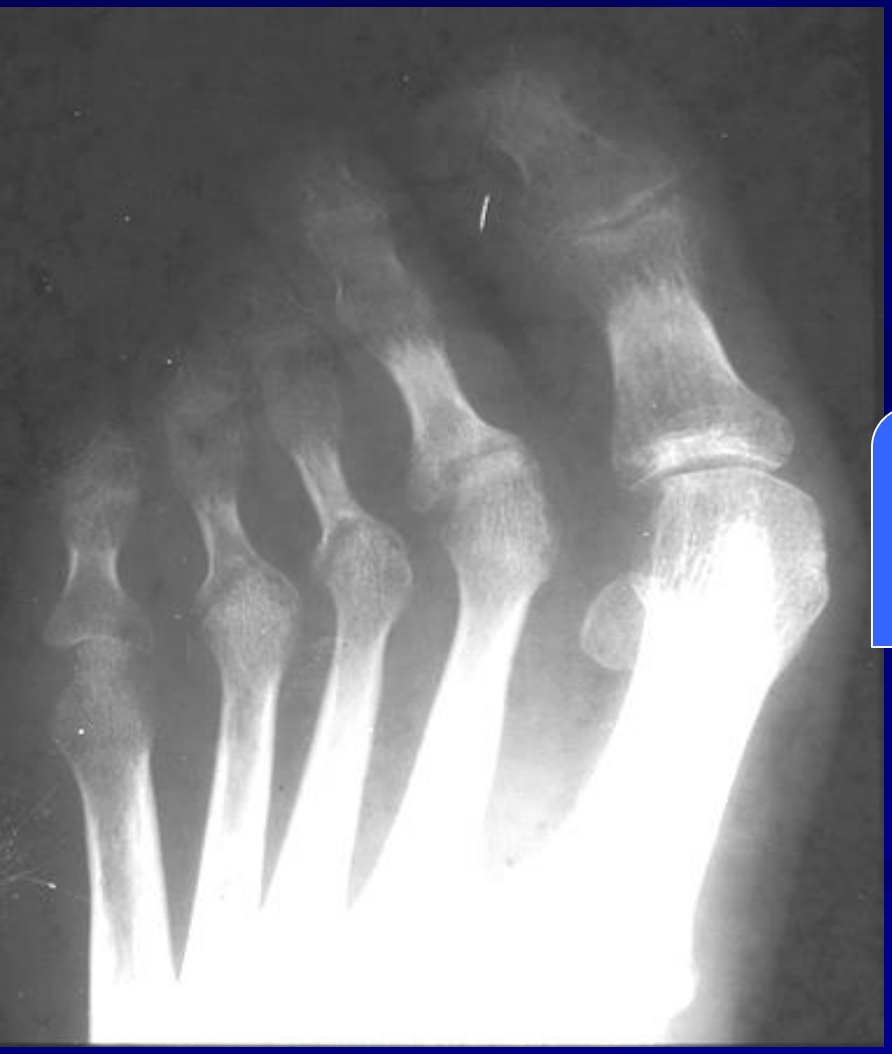
Earlier Stage of RA with Marginal Erosions

Rheumatoid Arthritis

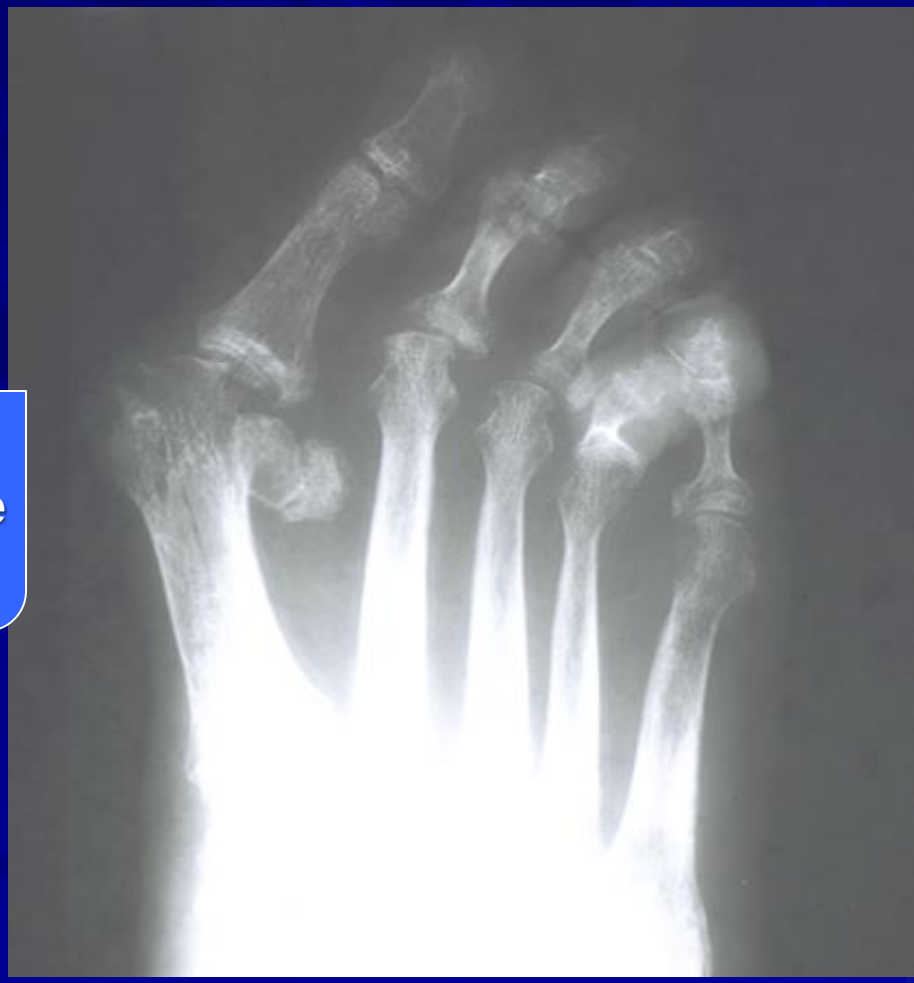
- **Forefoot** is the most common site for radiographic changes
  - Forefoot is the initial site of radiographic change in 15% of patients with RA
  - Of the FF joints, the 1st and 5th MPJs are most often affected first
- **Erosive processes** favor the medial-plantar aspect of the 1st-4th MPJs, medial aspect of the hallux IPJ, & medial/lateral aspects of the 5th MPJ



Rheumatoid Arthritis



Fibular
Deviation of the Toes

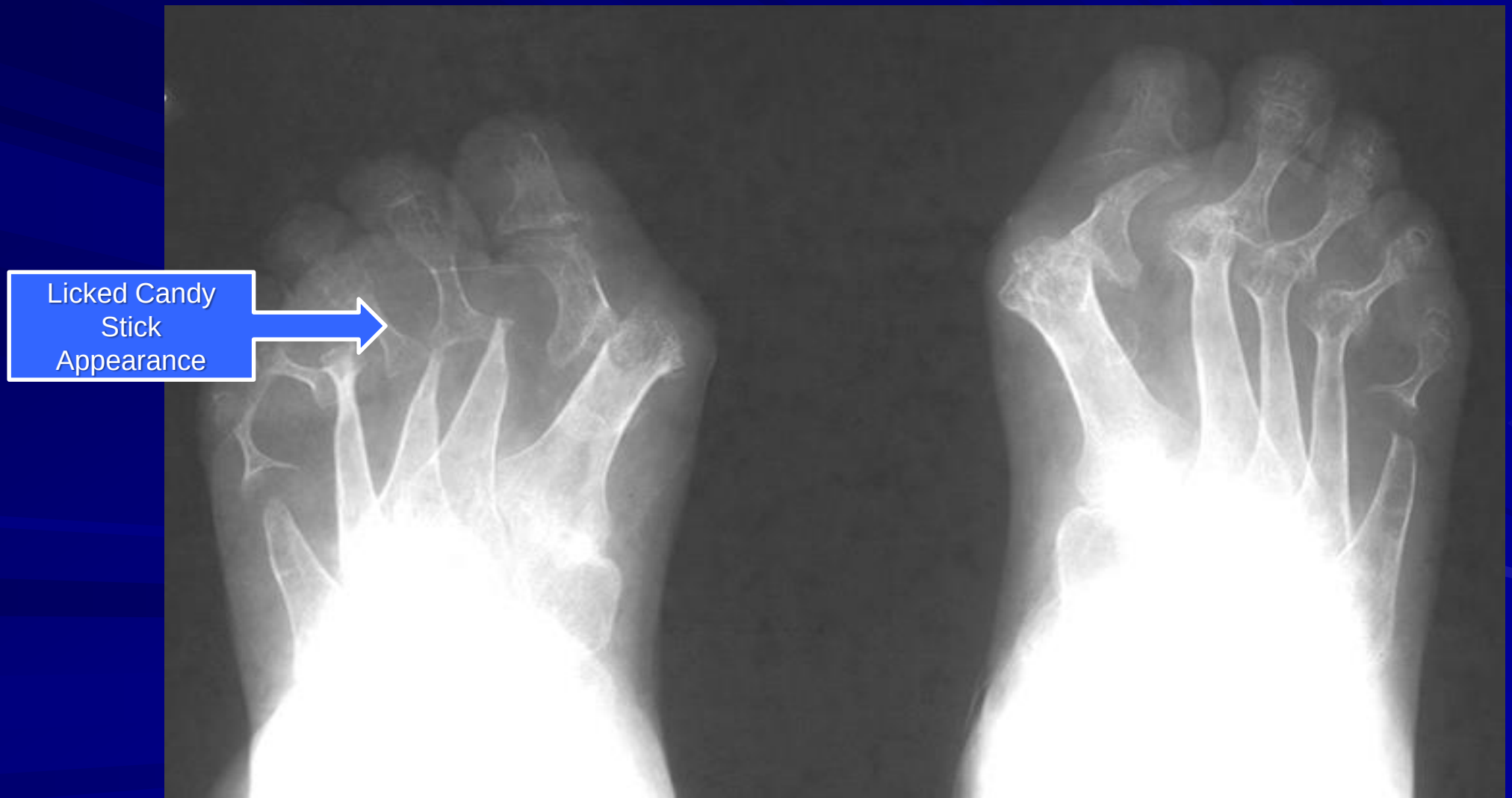


RA: Soft Tissue Manifestation

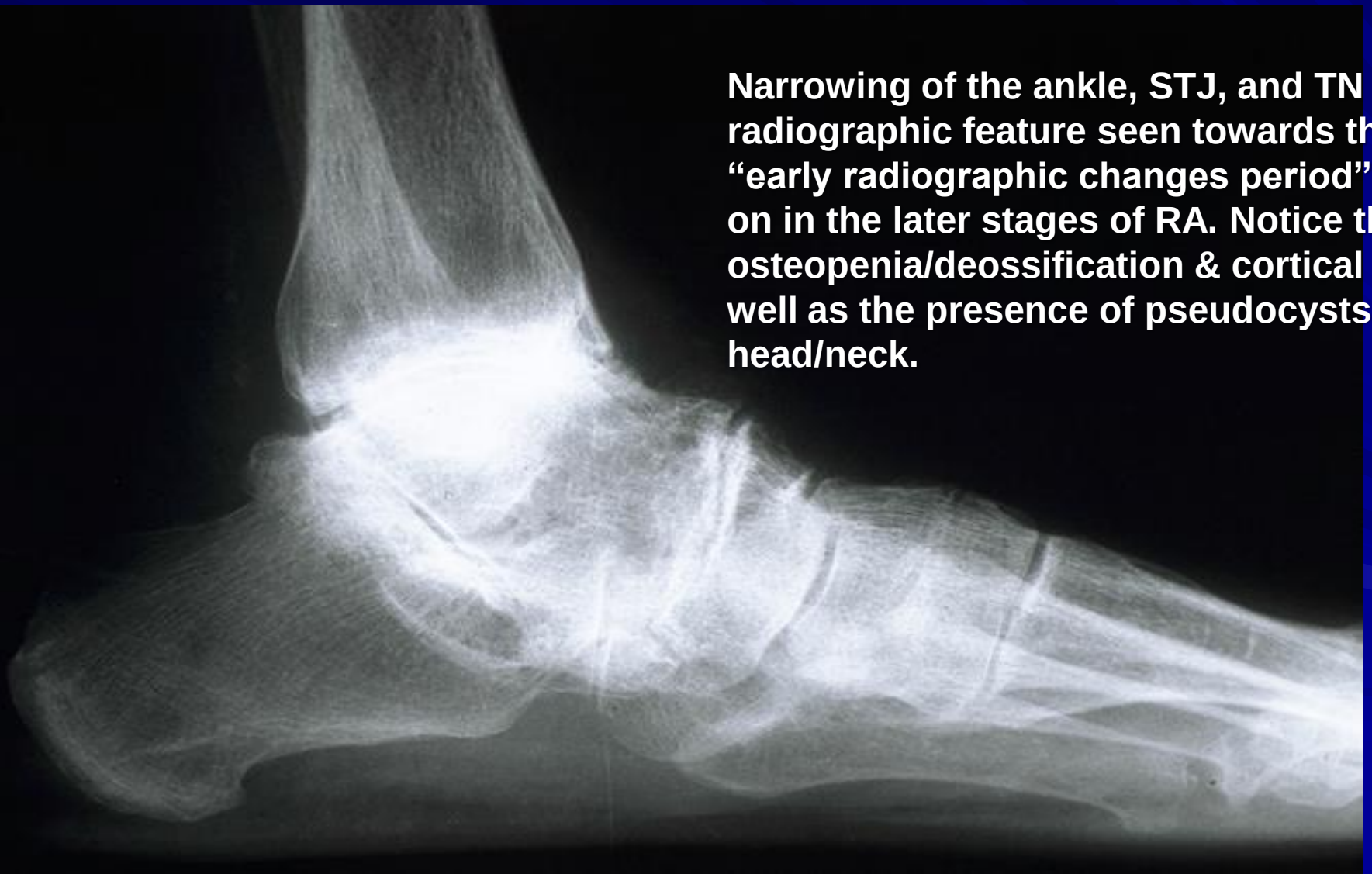
- **Rheumatoid Nodule:**
  - Extra-articular subcutaneous lesion/mass
  - 20%-30% of RA patients will develop nodules
  - Occur almost exclusively in patients who are rheumatoid factor positive
  - Usually located on extensor surfaces of the arms & elbows
  - Can develop at pressure points on the feet & knees

Rheumatoid Arthritis

Later Stage of RA with Fibular Deviation at the MPJs & Joint Destruction

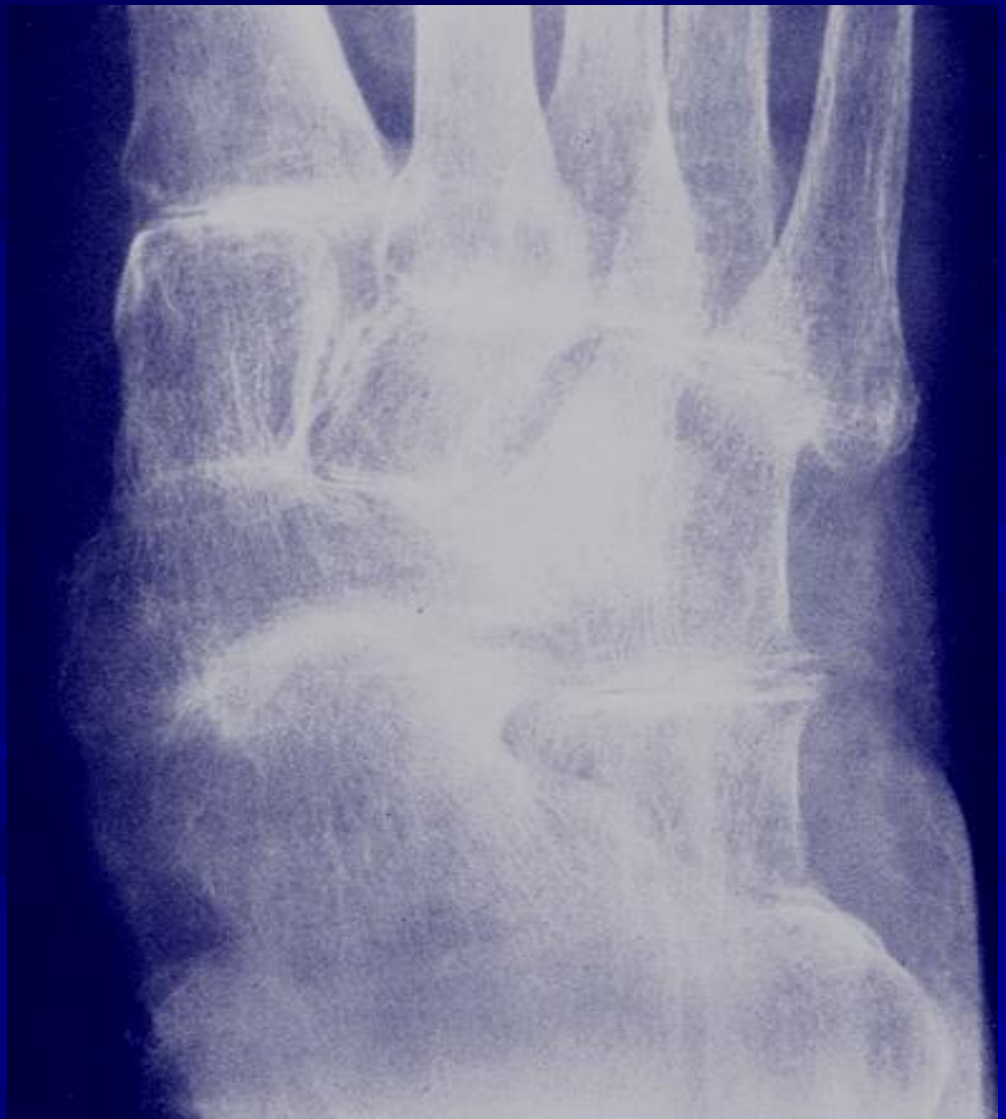


Rheumatoid Arthritis



Narrowing of the ankle, STJ, and TN joint is a radiographic feature seen towards the end of the "early radiographic changes period" and continues on in the later stages of RA. Notice the disuse osteopenia/deossification & cortical thinning, as well as the presence of pseudocysts at the talar head/neck.

Rheumatoid Arthritis



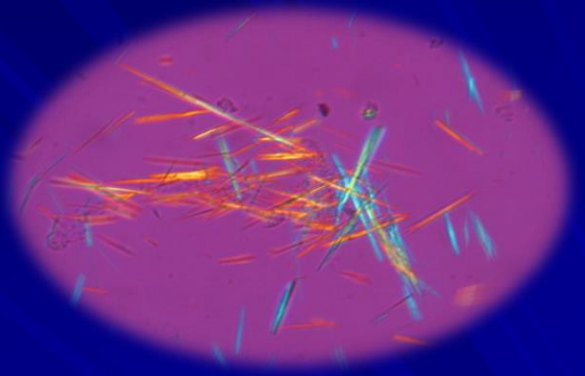
In this x-ray, we see absent joint spaces throughout the lesser tarsus (signifying ankylosis) with the exception of the narrowed CC joint.

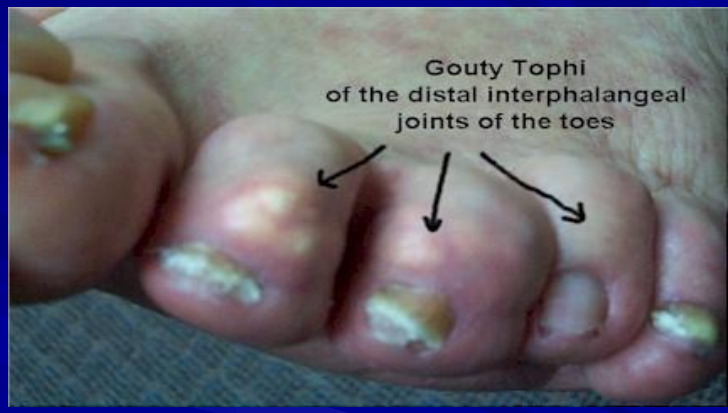
Metabolic Arthritides

Gouty Arthritis

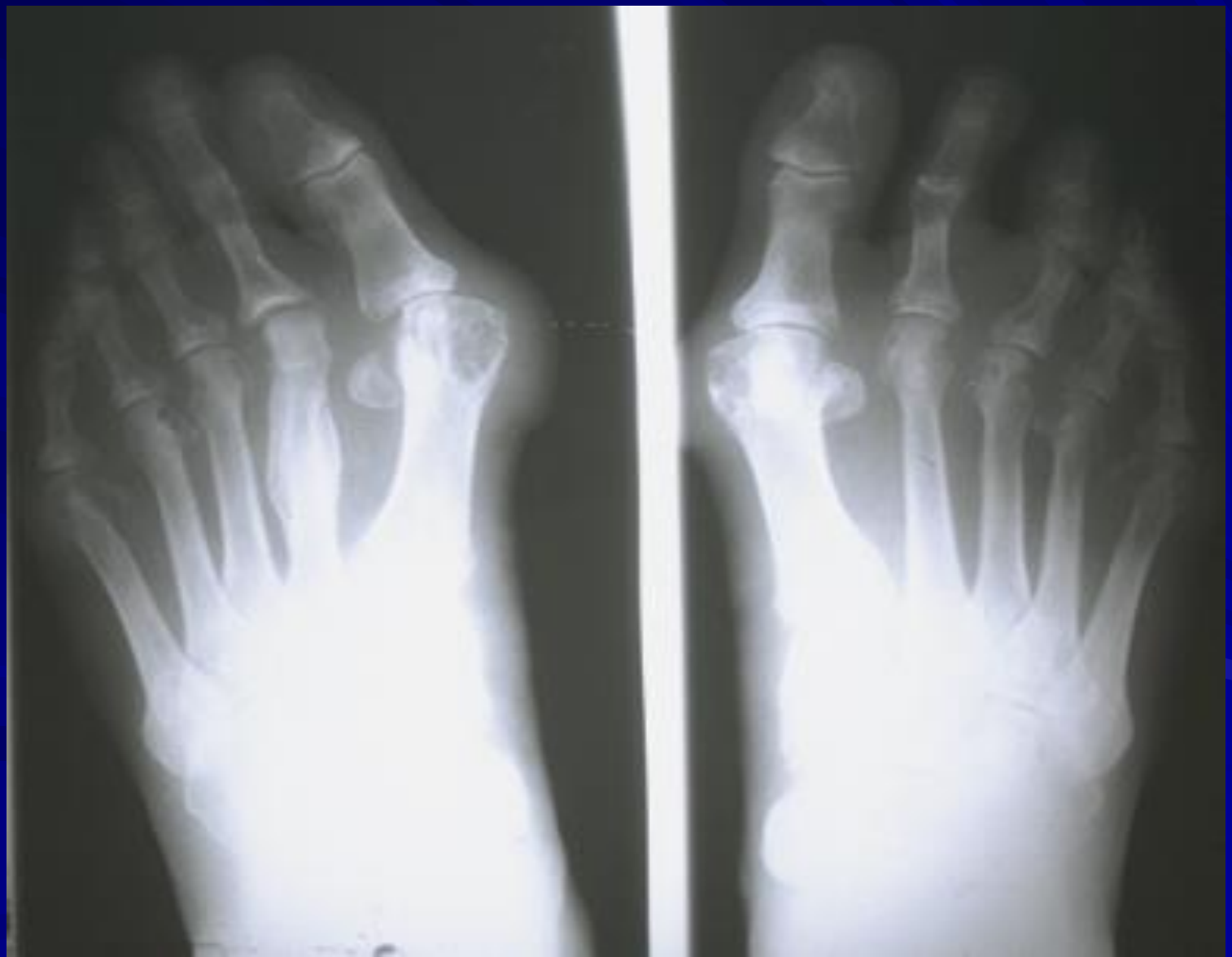
Gouty Arthritis

- **In-borne error of purine metabolism**
  - **Hyperuricemia**
- **Clinical features**
  - Age: 40 – 50 yoa
  - Gender: Male 20:1 Female
- **Lab findings**
  - Elevated serum uric acid
    - 10% uricosuric
  - Polarizing microscopy
    - **Negatively bi-refringent urate crystals**
    - Needle-shaped & blue in color when oriented perpendicular to the compensator

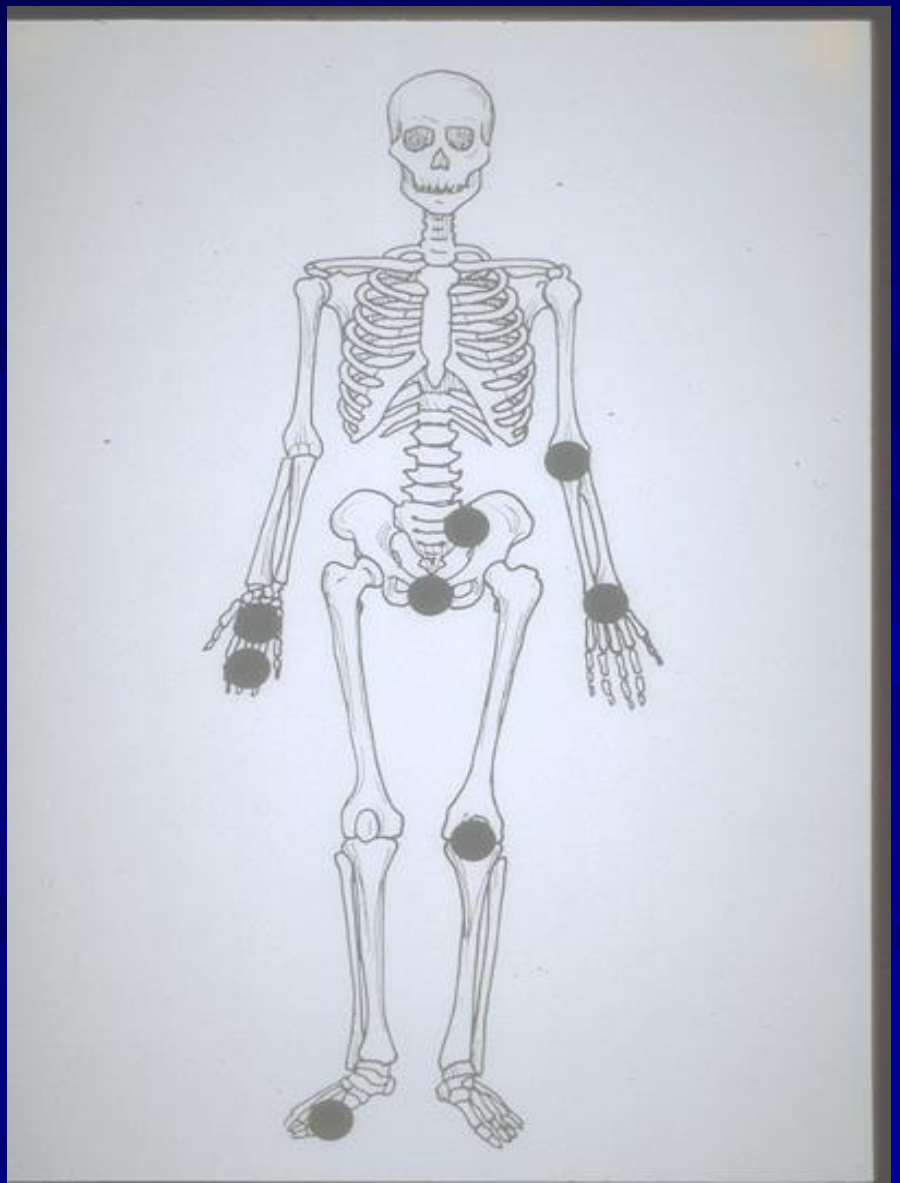




Gouty Arthritis



Gouty Arthritis



Asymmetric Joint Involvement

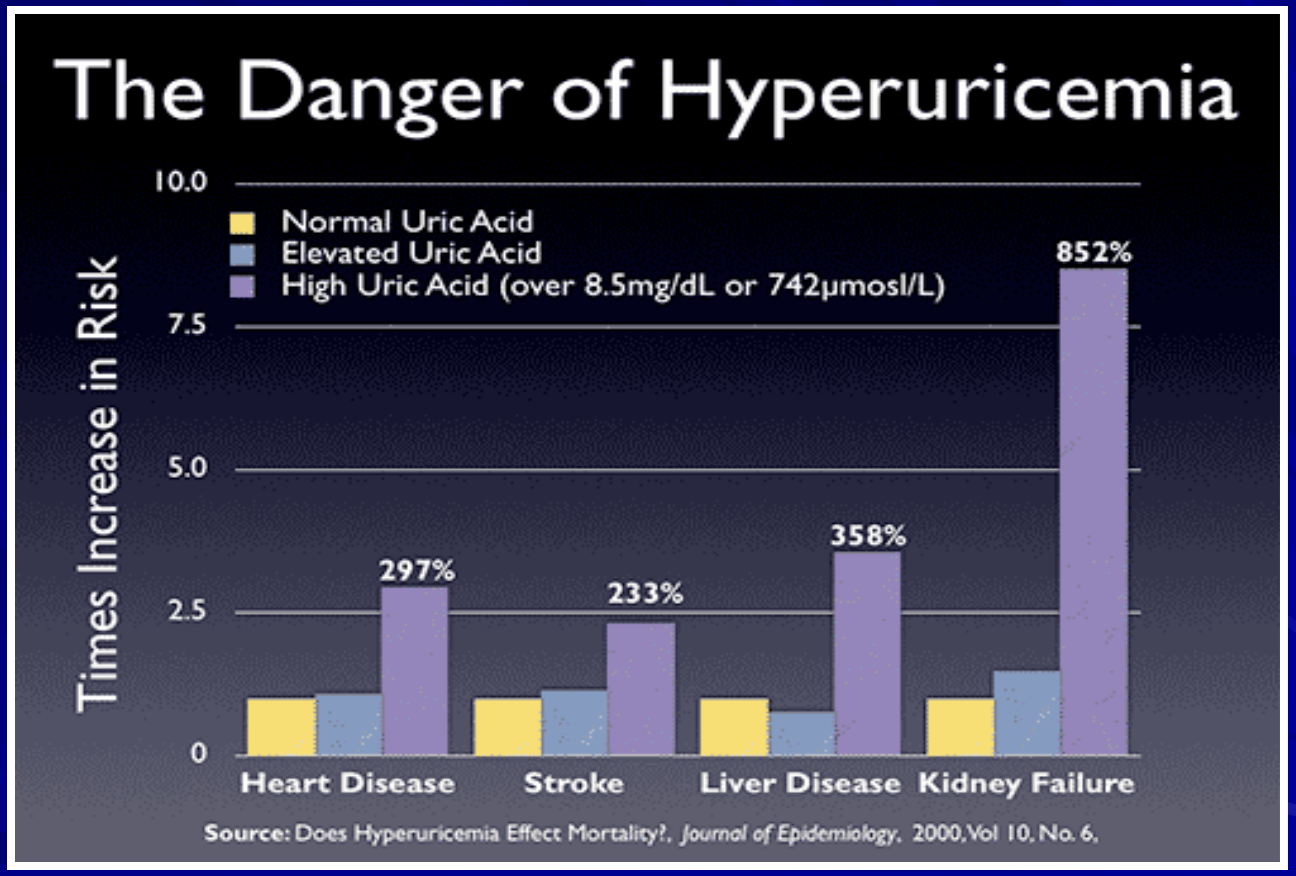
Gouty Arthritis

- **Primary gout** (hormonal/genetic factors cause metabolic abnormalities)
  - **Hyperuricemia** due to:
    - **Over-production of uric acid due to error in purine metabolism**
    - **Under-excretion of urates by kidneys**

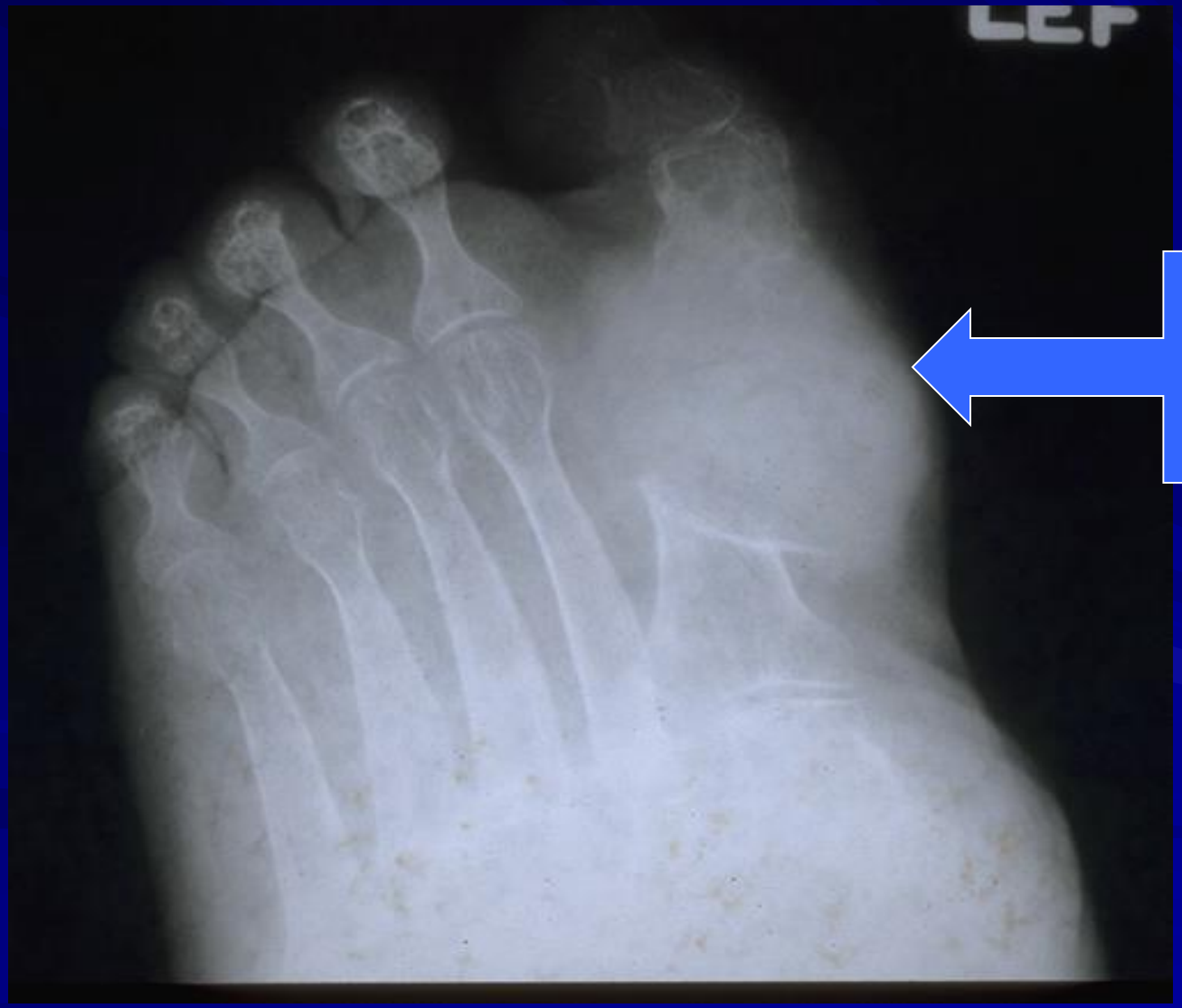
- **Secondary gout**
  - Hyperuricemia precipitated by drug therapy or a medical condition
    - Drug therapy: **SPEED**- Salicylates, Pyrazinamide, Ethambutol, Ethanol, Diuretics (ie. thiazide - decrease excretion of uric acid)
    - Hyperparathyroidism – increases serum Ca which impairs renal excretion of urates

Gouty Arthritis

- Asymptomatic Hyperurecemia:
  - Elevated urate levels in patients with no known hx. of gout or renal disease, no known disorders/drugs which elevate uric acid levels.

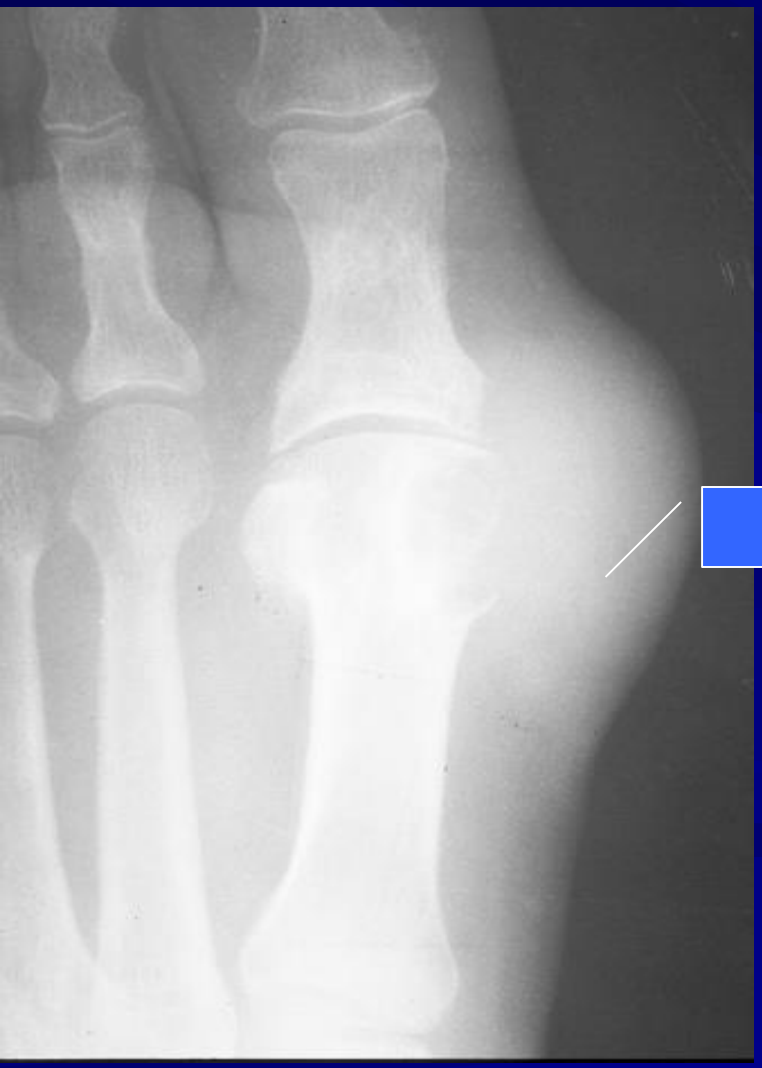


Gouty Arthritis

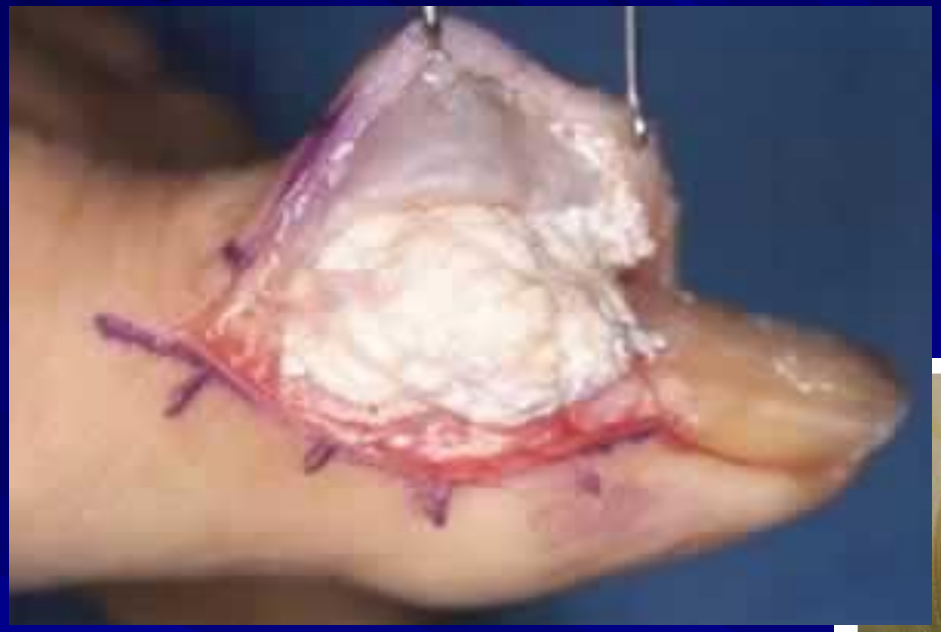


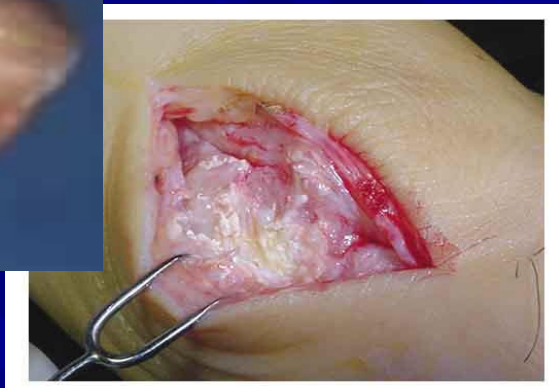
Tophi with Extensive Erosions & Joint Destruction seen with Chronic Gout

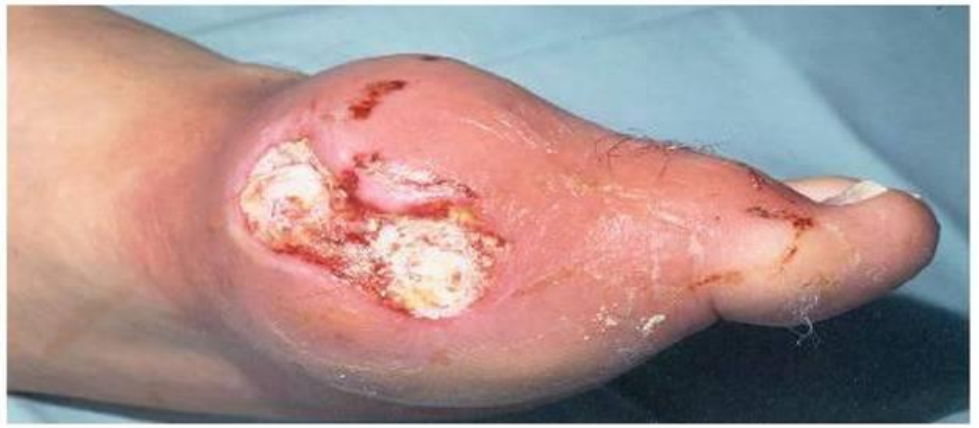
Gouty Arthritis



Tophi

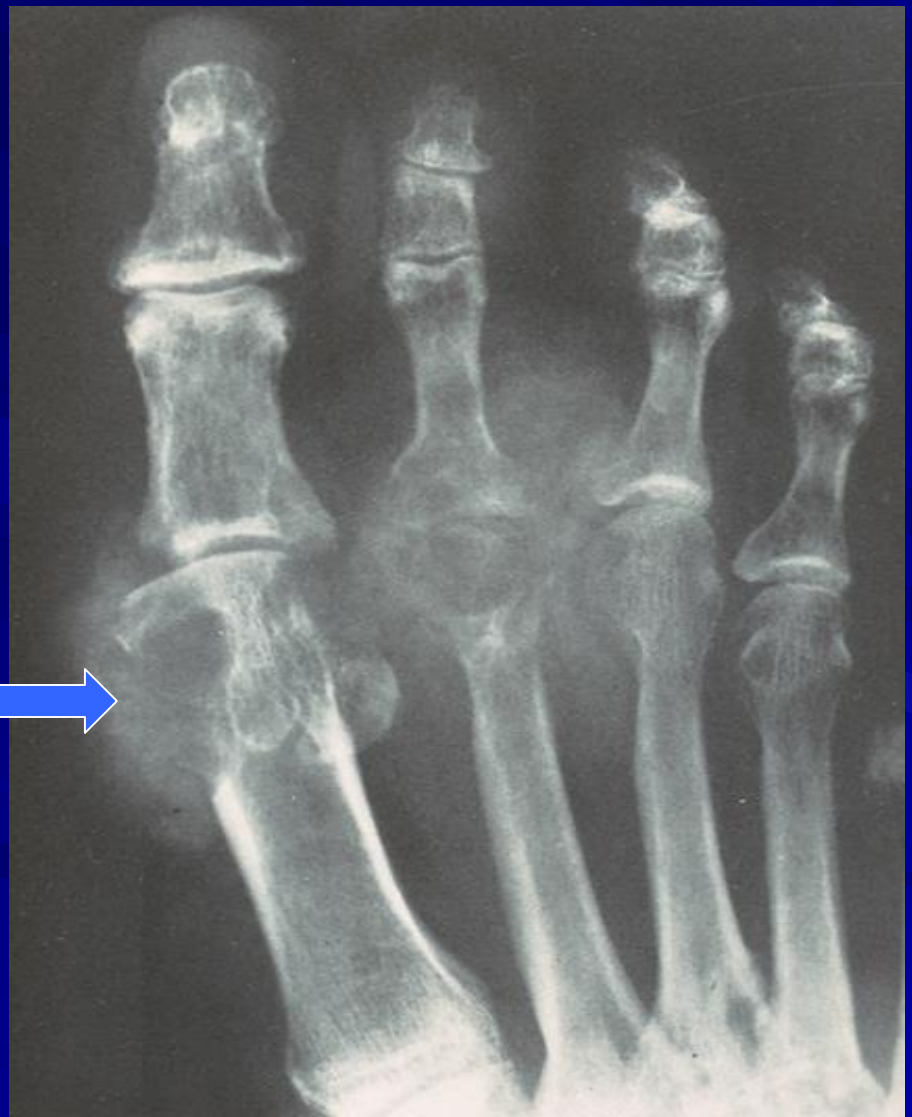




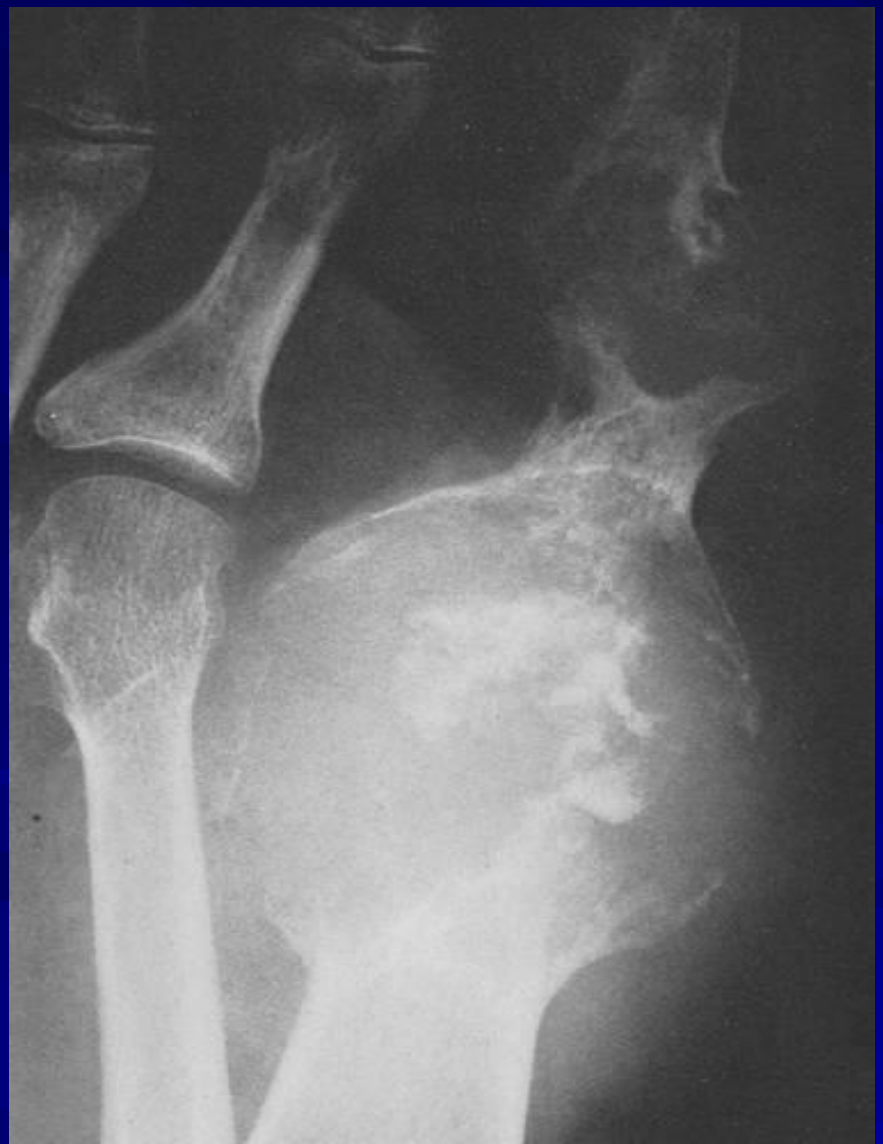


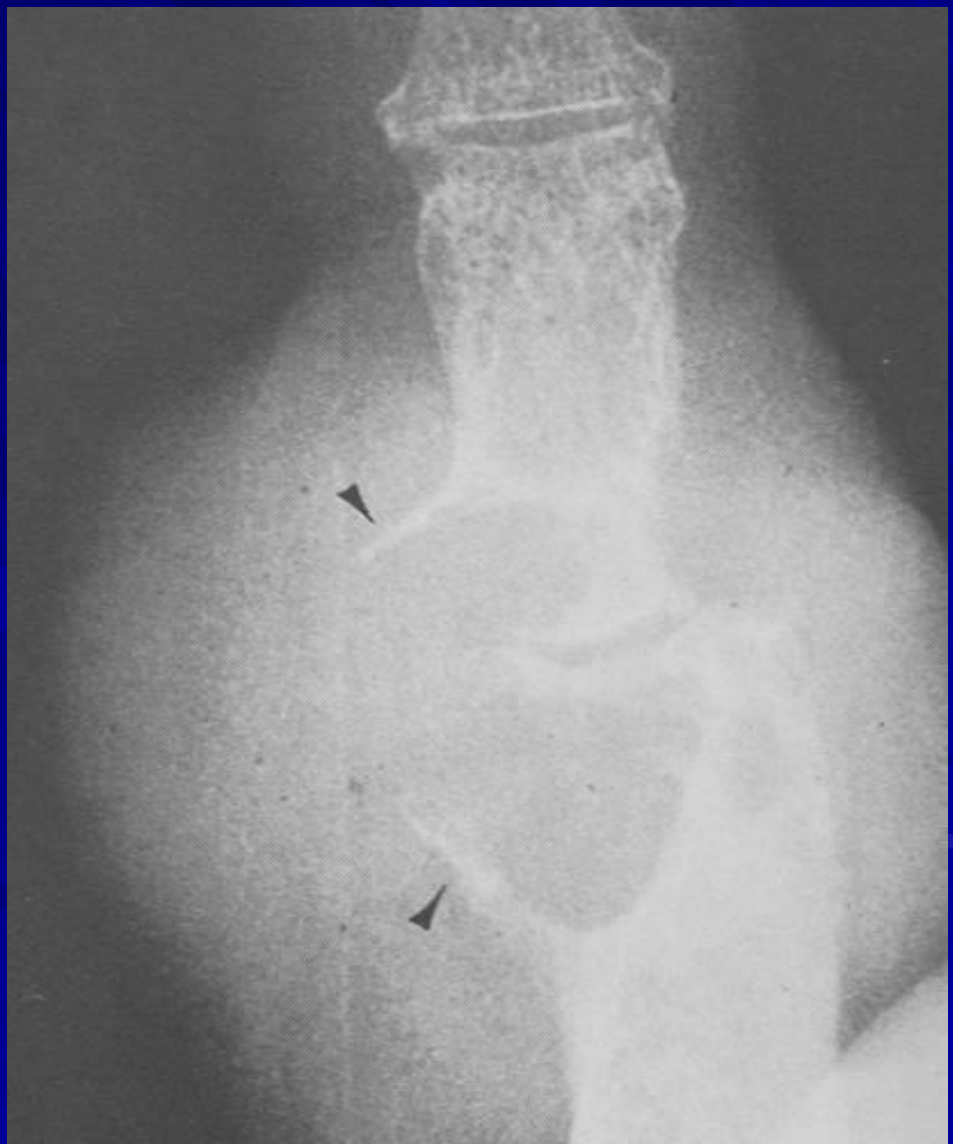
Gouty Arthritis

Tophi forming away from the 1st MPJ



Gouty Arthritis



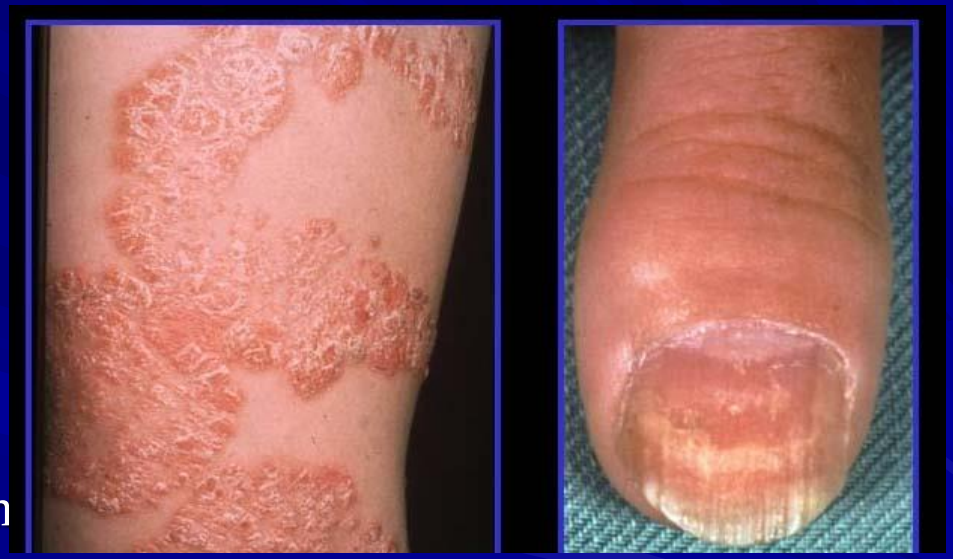


Inflammatory Seronegative Arthritides

Psoriatic Arthritis

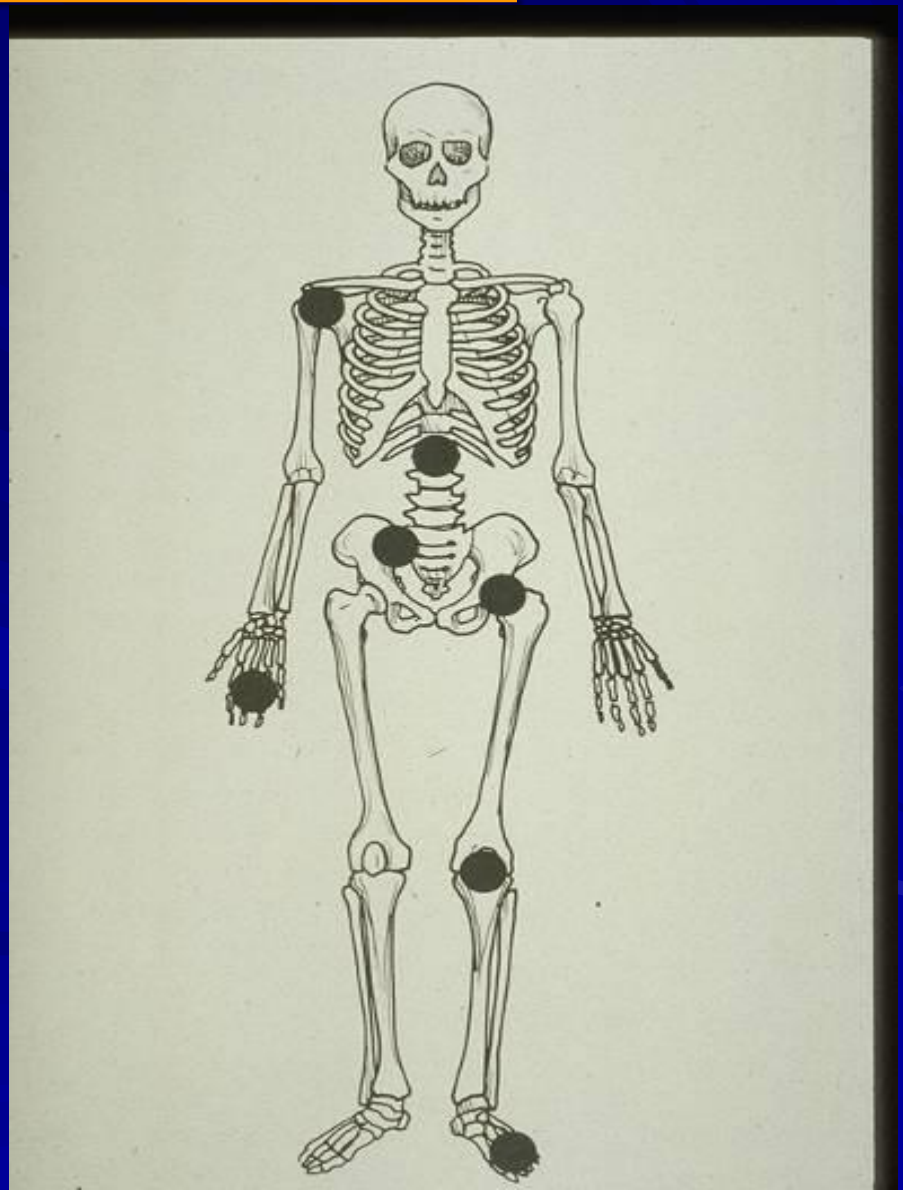
Psoriatic Arthritis

- **Incidence:**
  - 7 - 15% of patients with psoriasis will develop psoriatic arthritis
  - 80% in those exhibiting nail changes
- **Clinical features:**
  - Age: 20 – 50 yoa
  - No gender predilection
- **Lab findings:**
  - HLA-B27 antigen
    - 25%-75% SI involvement
    - 30% with peripheral joint involvement (han
- **Distribution:**
  - Asymmetrical joint involvement (typically DIPJs)
  - Unilateral joint involvement

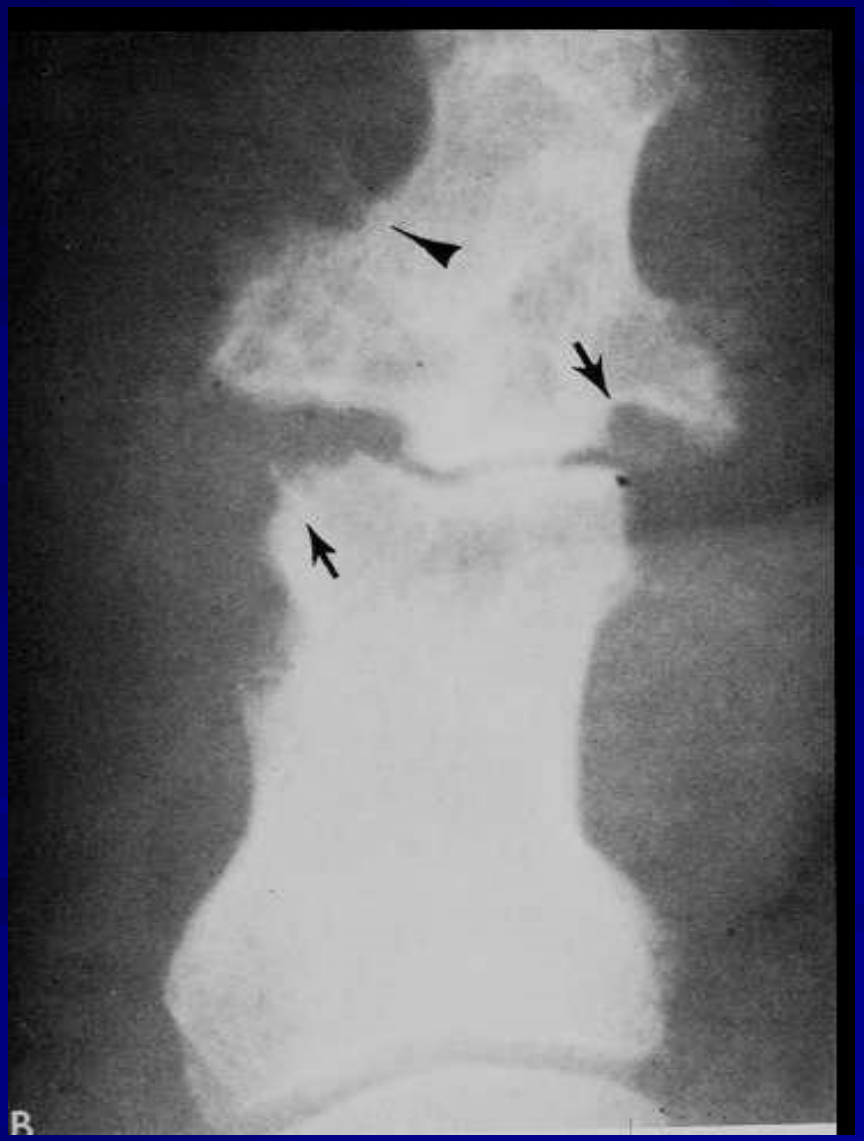


Psoriatic Arthritis

Asymmetrical & Unilateral Joint Involvement



Psoriatic Arthritis

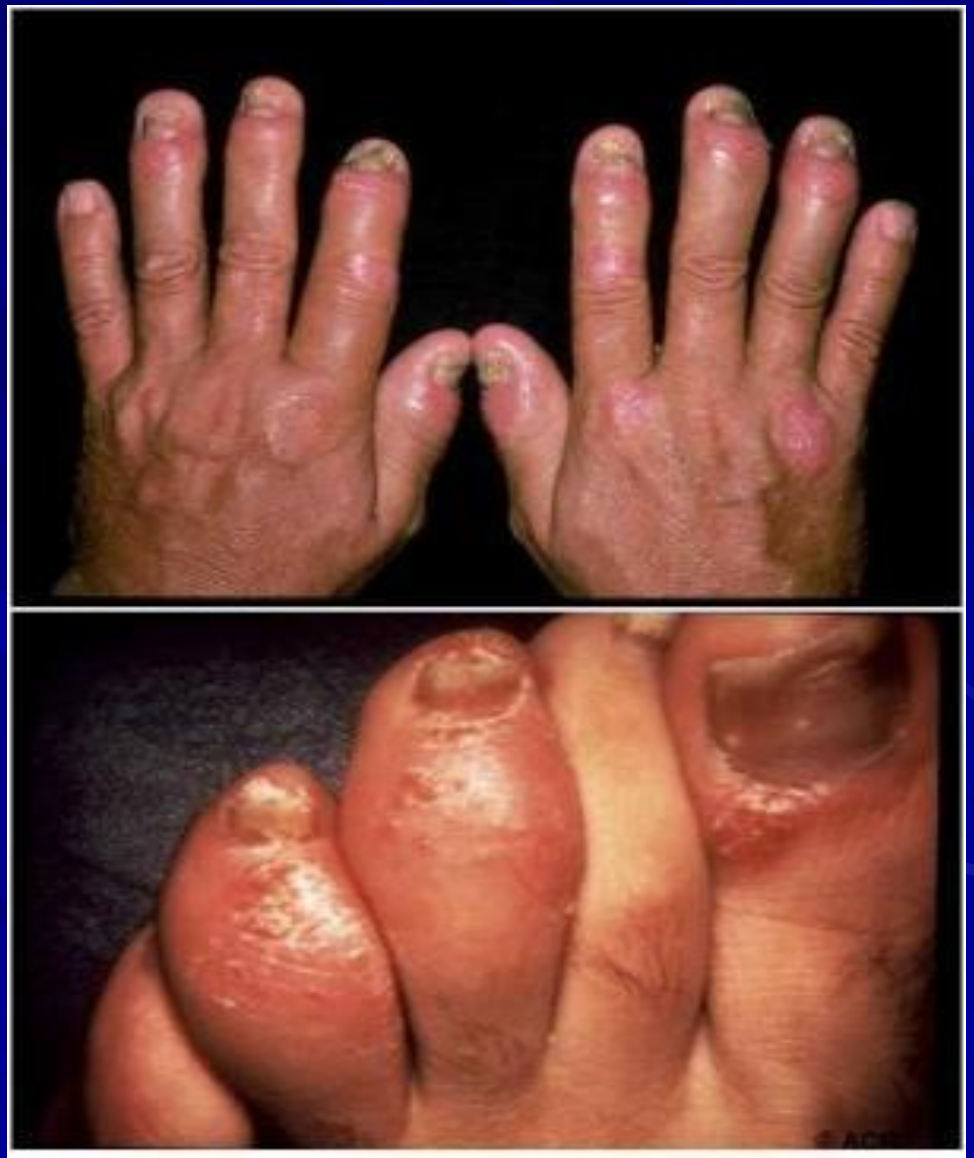


Take Home Point:

Erosions & Bone Formation

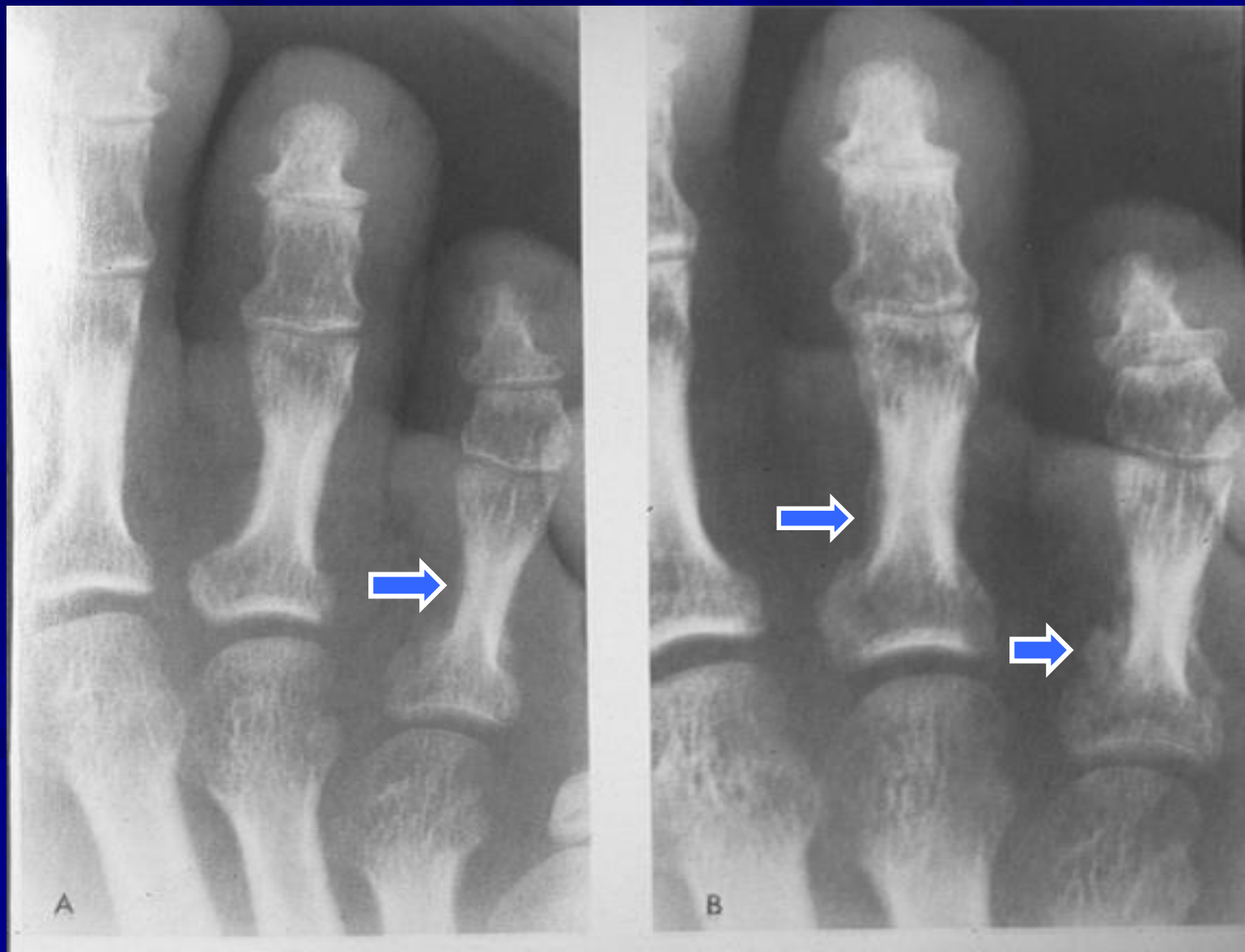
Psoriatic Arthritis

- **Soft-tissue changes**
  - Inflammatory synovitis leads to symmetrical soft-tissue edema around involved joint, similar to RA
  - However, in PA the edema extends beyond joint creating a **sausage-like appearance of the digit**



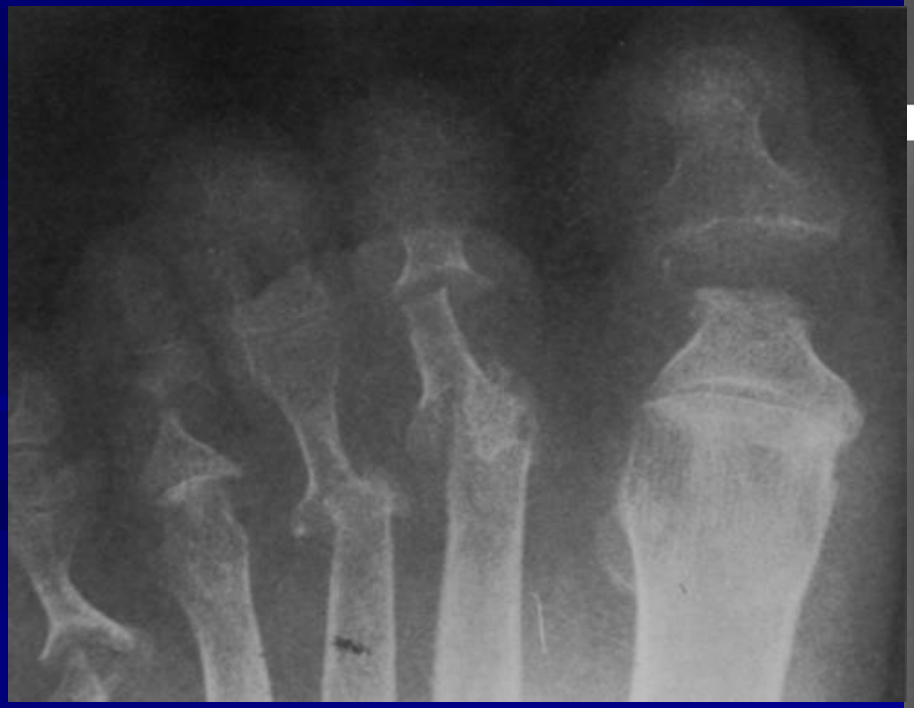
Psoriatic Arthritis

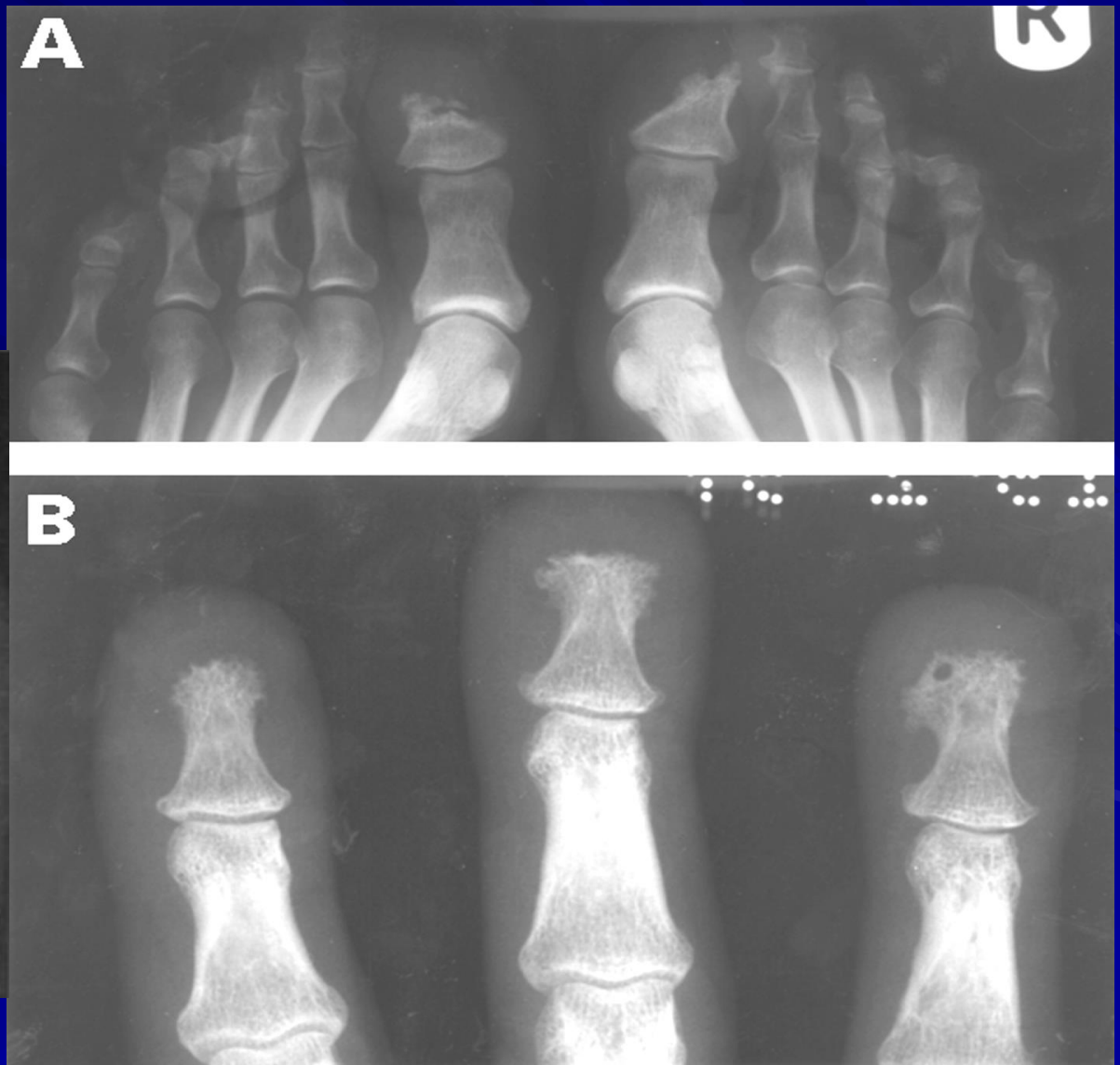
Periostitis



Psoriatic Arthritis

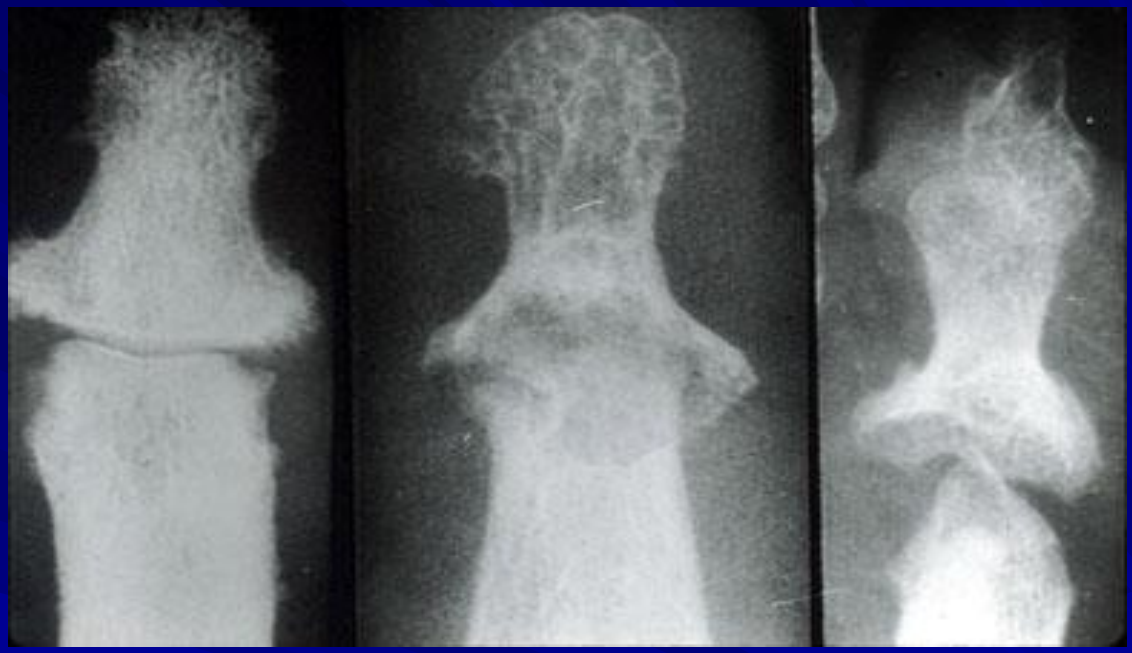
Acro-osteolysis

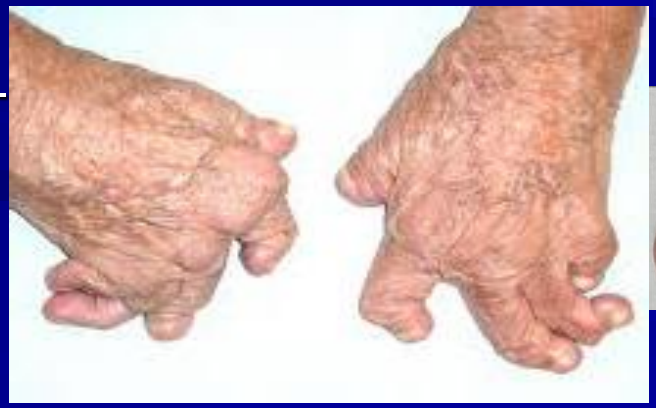


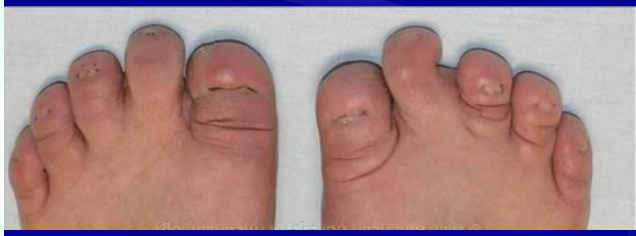


Psoriatic Arthritis

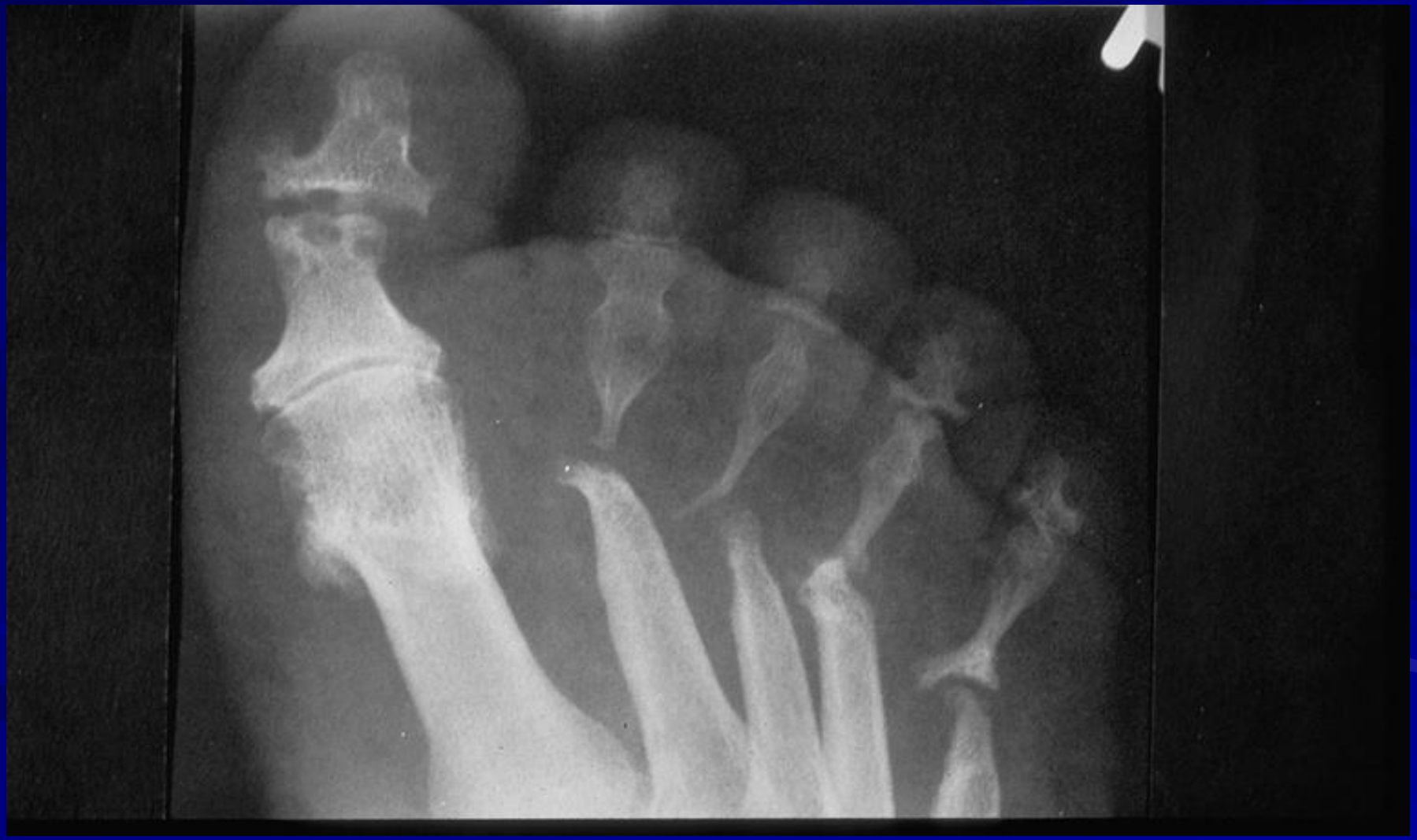
- **Erosions** (continued)
  - Typically occur simultaneously at medial/lateral margins & progress across entire articular surface
  - With progression, bony ends can become tapered like a "pencil point"
    - Cup and saucer
    - Pencil in cup
    - Mortar and pestle
    - Whittling
  - Telescoping appearance - "Main-en-lorgnette"
    - Seen in severe PA/arthritis mutilans







Psoriatic Arthritis

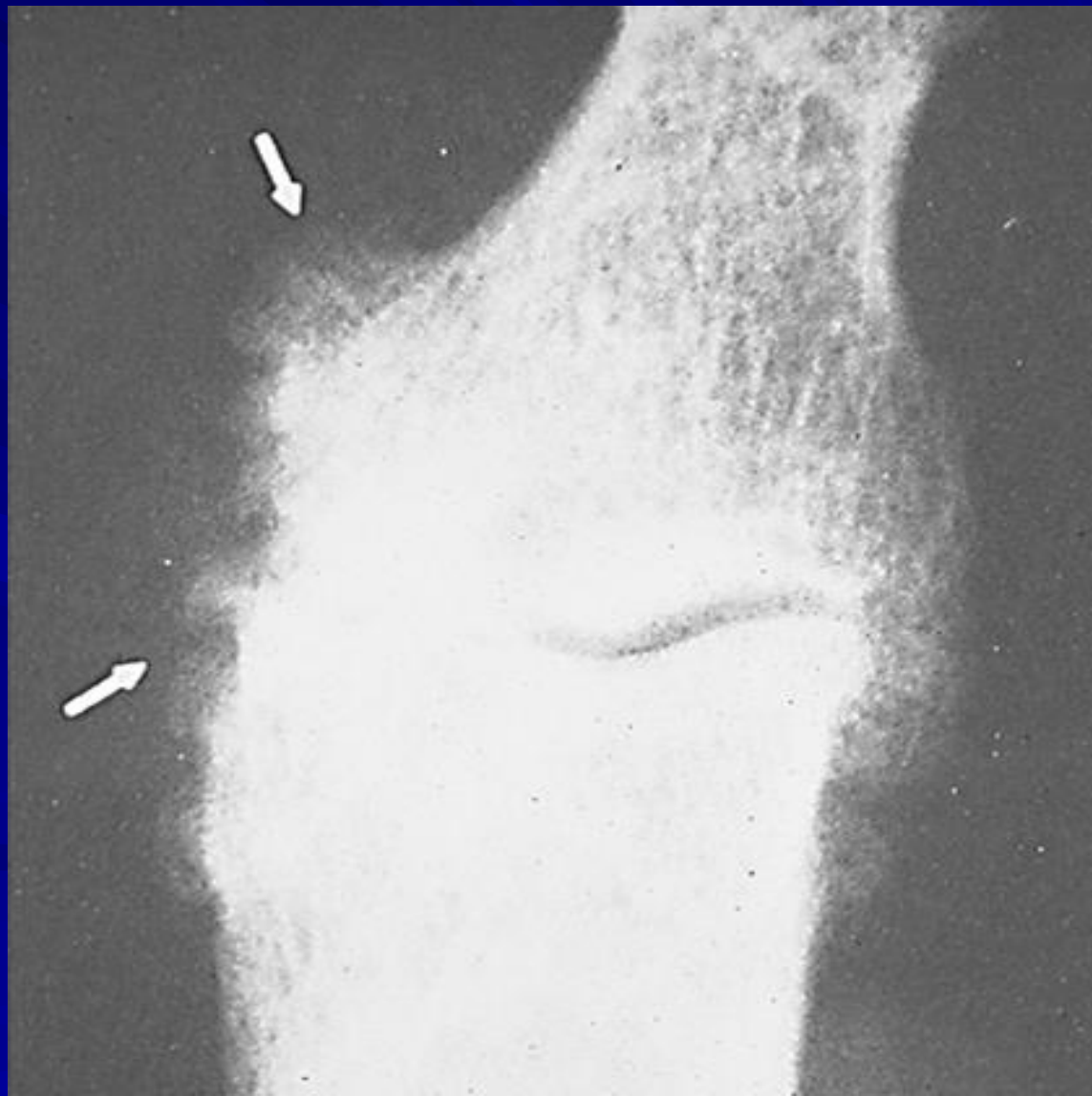


“Pencil Point” Metatarsals

Psoriatic Arthritis

IPJ Whiskering:

- Resembles stubble of new beard growth
- Spiculated appearance that radiates away from the bone margin
- Ill-defined sclerosis often accompanies whiskering



Reactive Arthritis (Reiter's Disease)

Hans Conrad Julius ReiterFrom Wikipedia:

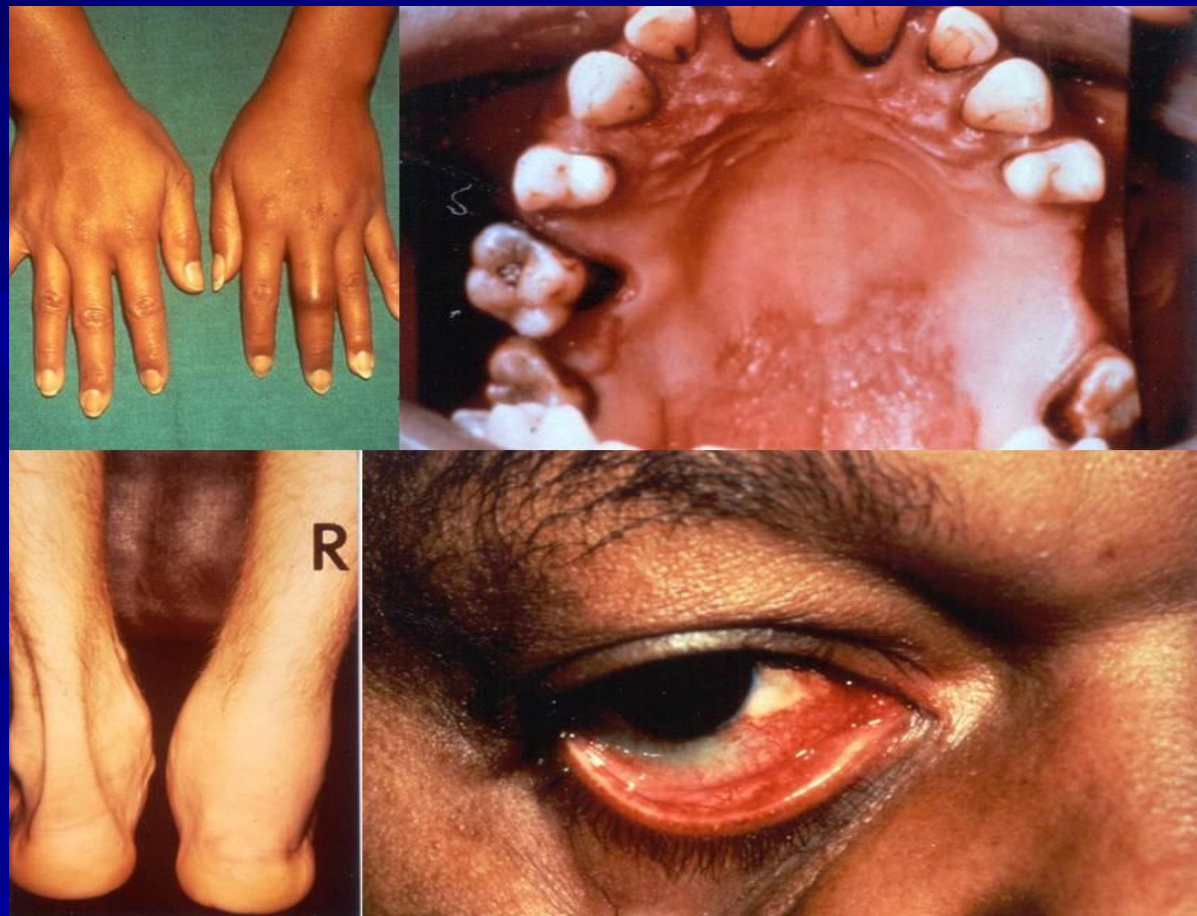
*Hans Reiter.**Hans Conrad Julius Reiter (February 26, 1881 – November 25, 1969) was an infamous German physician who was convicted of war crimes for his medical experiments at the Buchenwald concentration camp. He wrote a book on "racial hygiene" called Deutsches Gold, Gesundes Leben - Frohes Schaffen.Reiter was born in Reudnitz, near Leipzig in the German Empire. He studied medicine at Leipzig and Breslau (now Wrocław), and received a doctorate from Tübingen on the subject of tuberculosis. After receiving his doctorate, he went on to study at the hygiene institute in Berlin, the Pasteur Institute in Paris and St. Mary's Hospital in London, where he worked with Sir Almroth Wright for two years.[1] Reiter was also known for implementing strict anti-smoking laws in Nazi Germany.***

Hans Conrad Julius Reiter

Reiter was a member of the *Schutzstaffel during World War II and participated in medical experiments performed by the Nazis. After the Nazis were defeated, he was arrested by the Red Army in Soviet Union-occupied Germany and tried at Nuremberg. During his detention, he admitted to knowledge of involuntary sterilization, euthanasia, and the murder of mental hospital patients in his function as the gatherer of statistics and acting as "quality control" officer, and to helping design and implement an explicitly criminal undertaking at Buchenwald concentration camp, in which internees were inoculated with an "experimental" typhus vaccine, resulting in over 200 deaths. He gained an early release from his internment, possibly because he assisted the Allies with his knowledge of germ warfare.*

Reactive Arthritis

- An aseptic, peripheral, idiopathic disease complex preceded by a history of diarrhea or sexual contact followed by:
  - Conjunctivitis
  - Urethritis
  - Polyarthiritis
  - Mucocutaneous lesions



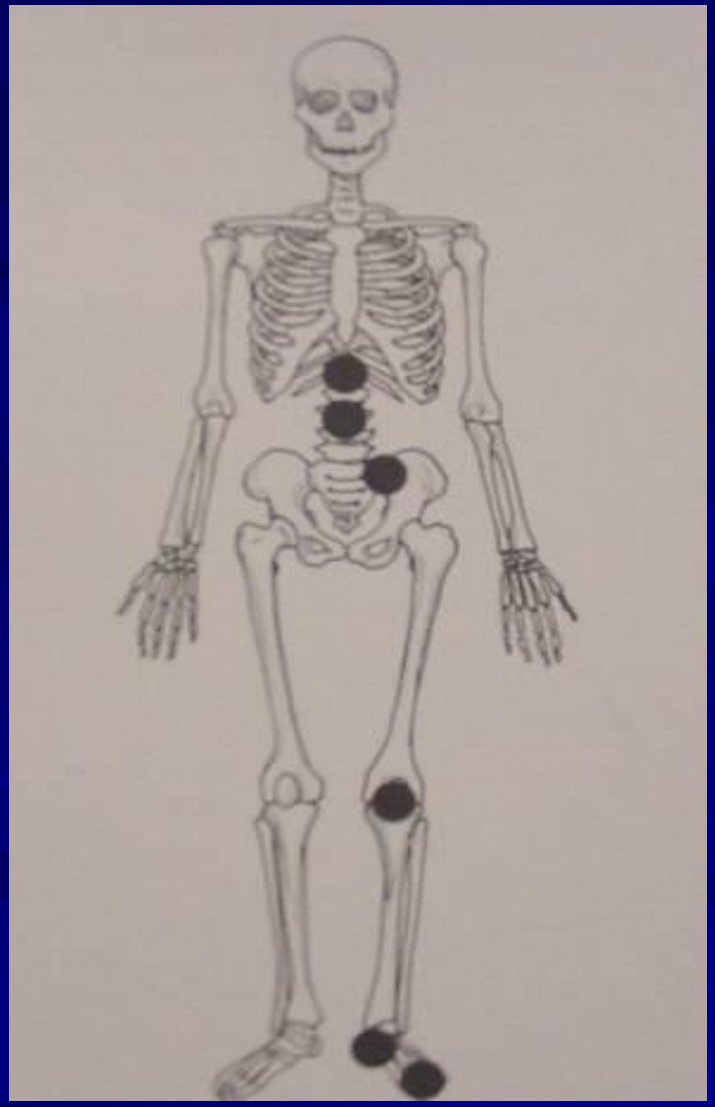
Reactive Arthritis

Saying:

"Can't See, Can't Pee, Can't Climb a Tree"

- **Feet are affected in 84-93% of patients suffering from Reactive Arthritis/Reiter's Disease**
- **At presentation the knee & ankle are most commonly affected**

Reactive Arthritis



Asymmetrical

&

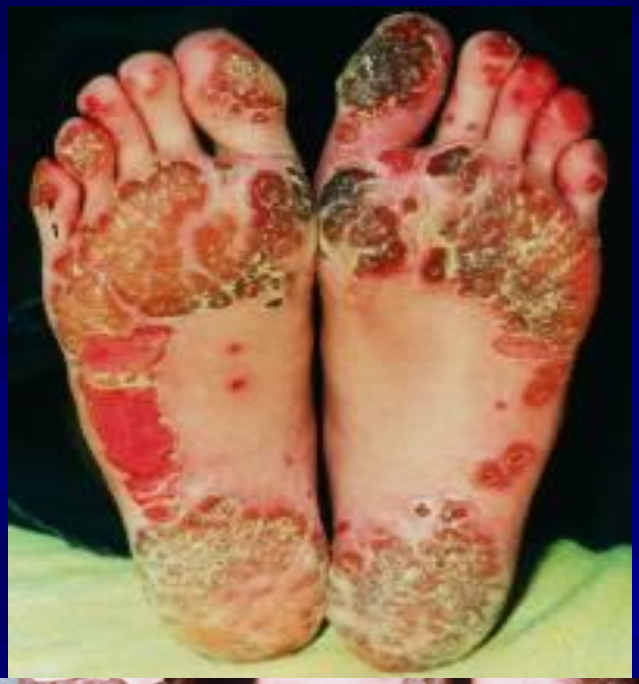
Unilateral Joint Involvement with Lower Extremity Predilection

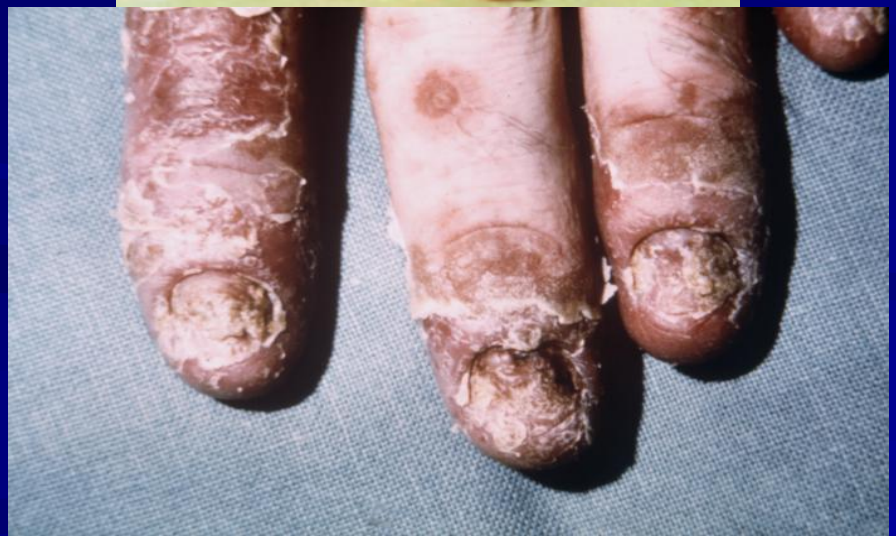
Reactive Arthritis

- **Clinical features:**
  - Symptoms are mild and spontaneously regress
  - Etiology unknown but attributed to gonococcal infection or chlamydia
  - Clinical diagnosis

  - **Two types:**
    - **Endemic** – venereal/genitourinary. (Males 20-30)
    - **Epidemic** – post-dysenteric -Salmonella infection/food poisoning/drinking contaminated water.
    - (Women/Children)

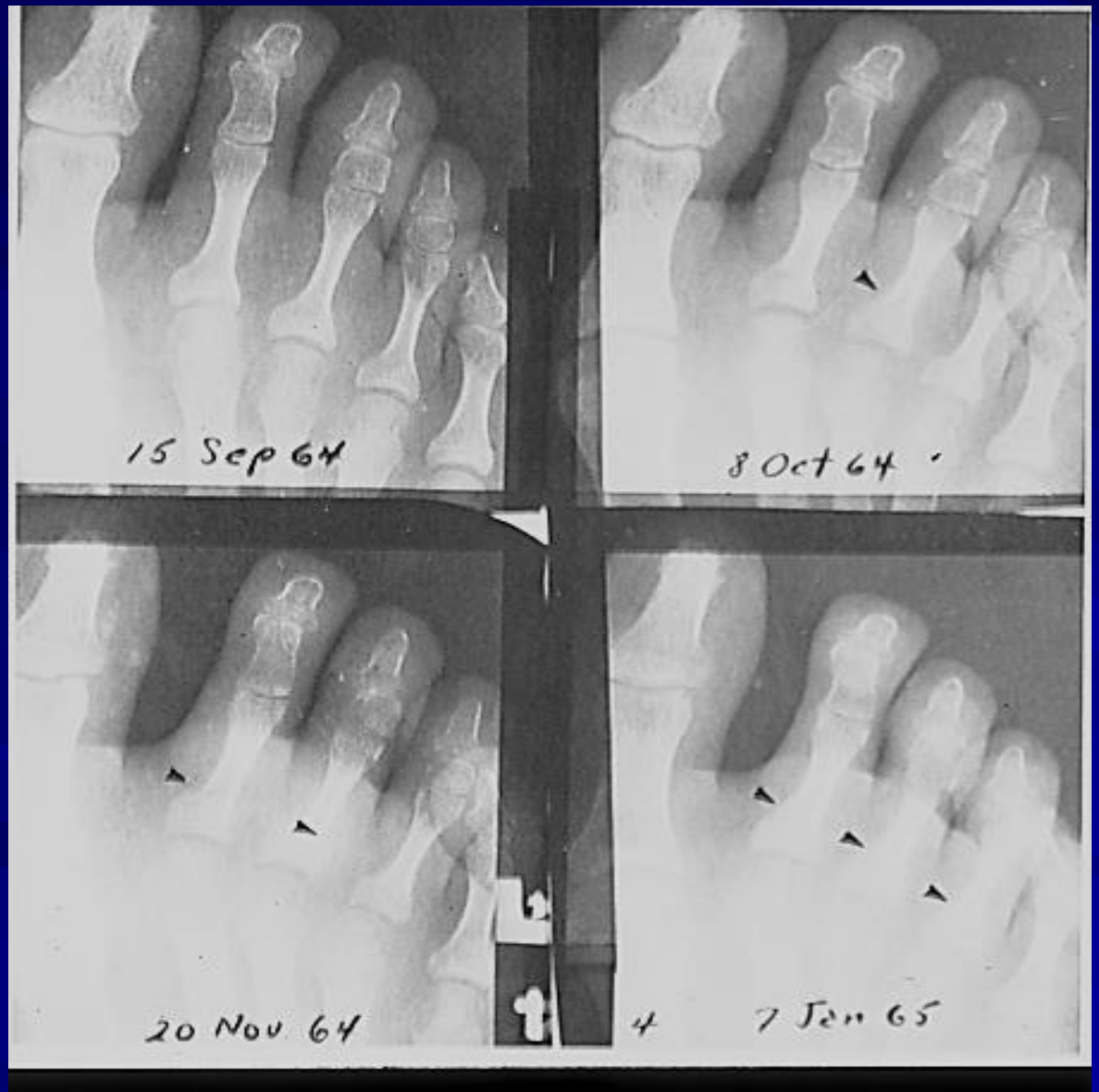
Reactive Arthritis





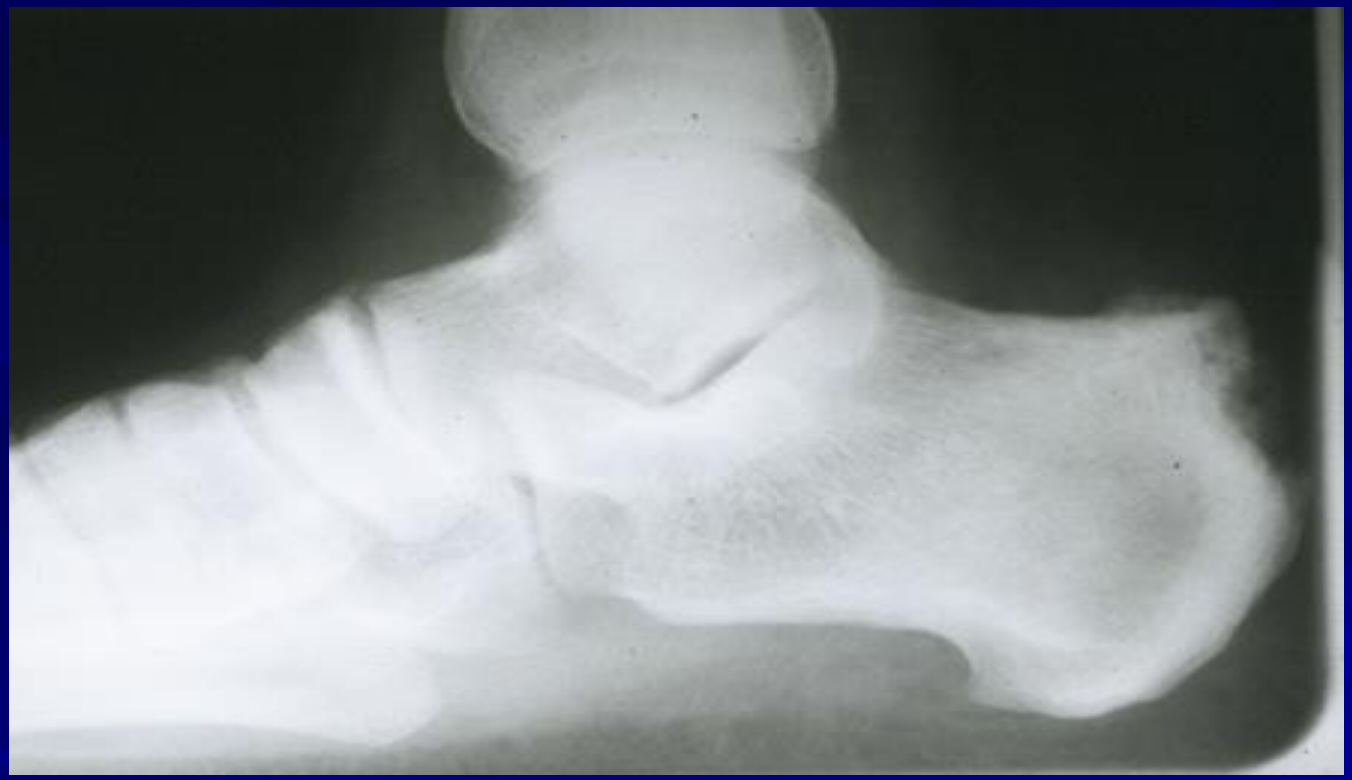
- Clinical features (continued):
  - Prior history of diarrhea or sexual exposure 3-11 days prior to onset of classic triad:
    - Conjunctivitis
    - Urethritis
    - Polyarthritis
      - Develops within a month of initial infection
    - Balanitis and keratoderma blennorhagica are also consistent findings
  - Triad can occur in any sequence and not all may be manifested

Reactive Arthritis



Diaphyseal Periosteal Reaction of Phalanges

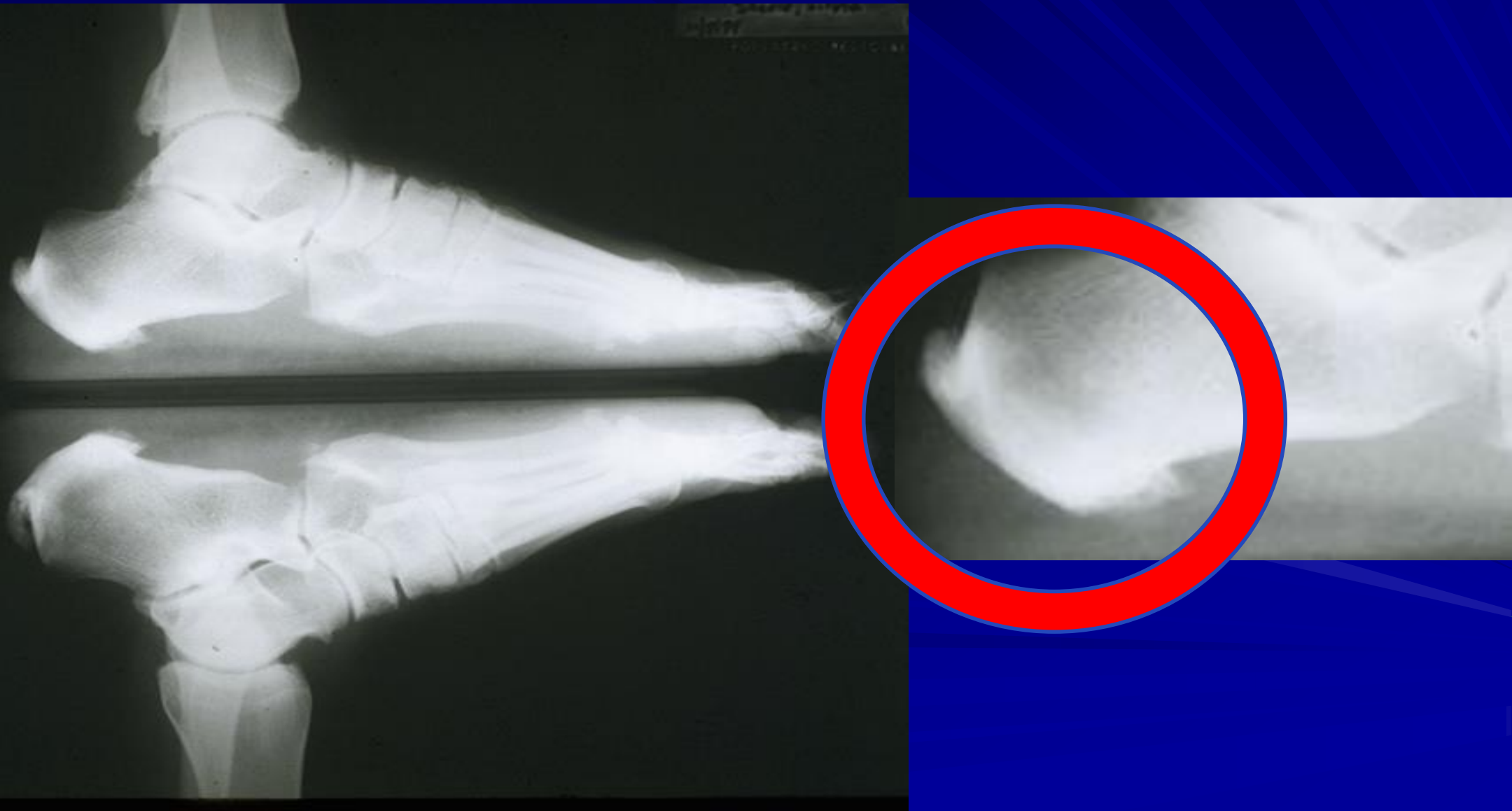
Reactive Arthritis



Retrocalcaneal Bursitis leading to bone erosion

Calcaneal Periostitis

Reactive Arthritis

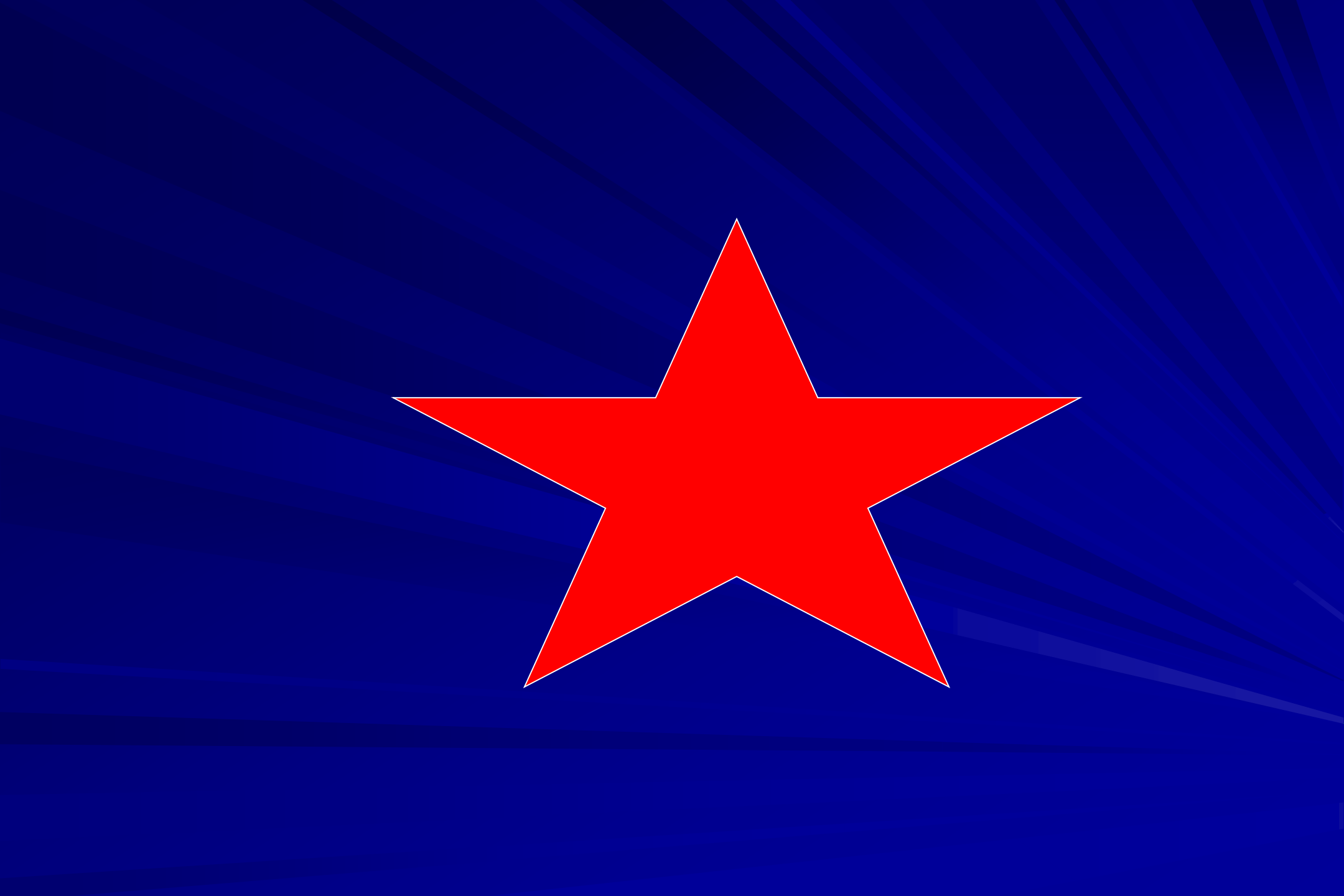


CONCLUSION

- There are many different forms of Arthritis.
- The foot and ankle are commonly affected by these disorders.
- Many of these Arthritic processes lead to marked pain and destruction of bone.
- Some may be associated with other disease processes.
- Radiographs are a crucial component in the diagnosis of these arthritic diseases

THANK YOU!

Daniel P. Evans, DPM, ACFAOM
Professor, Scholl College of Podiatric Medicine



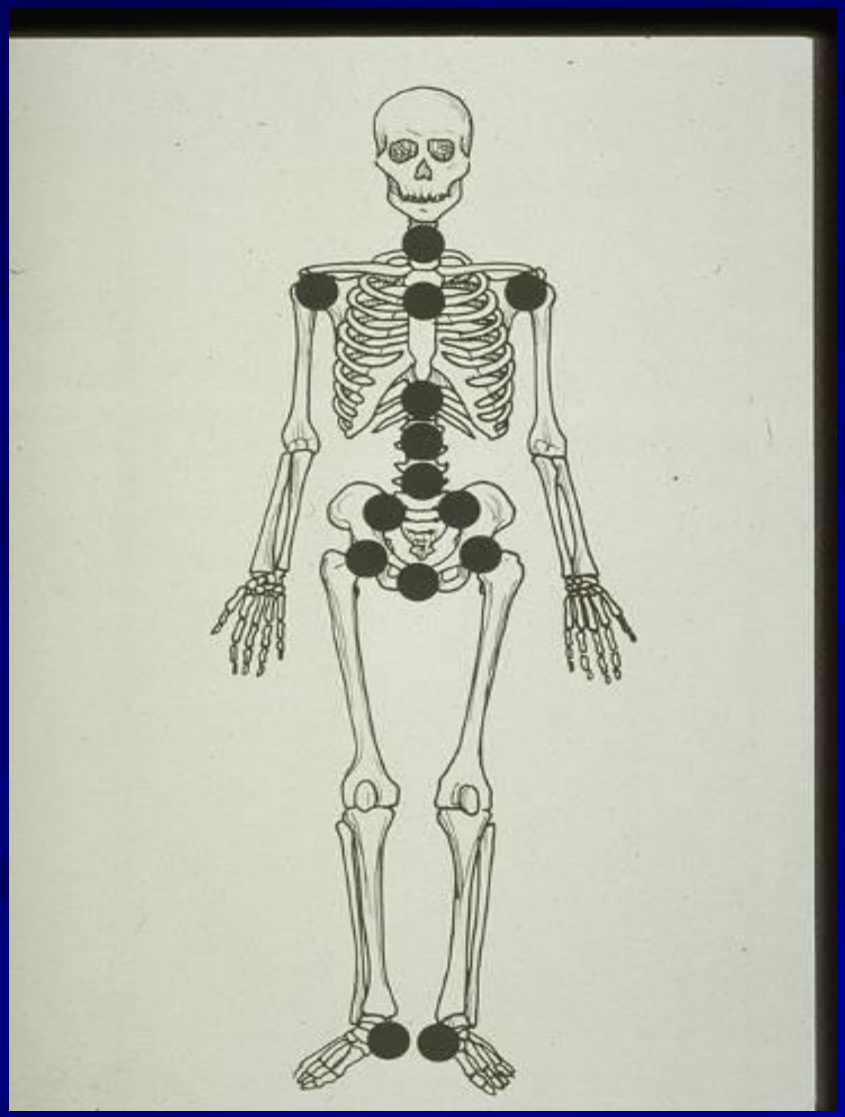
Radiographic Evaluation of the Arthritides

Ankylosing Spondylitis

Ankylosing Spondylitis

- Affects articulations, ligaments and tendons of pelvis and spine
- **Clinical features**
  - Age: 15 – 35 yoa
  - Gender: Male 10:1 female
  - Often seen in conjunction with Irritable Bowel Disease
- **Lab findings**
  - HLA-B27 – 90%
- **Distribution**
  - Low back, pain may be uni- or bi-lateral
  - Thoraco-lumbar spine initially

Ankylosing Spondylitis



Bilateral, Symmetrical Joint Involvement Originating at the:

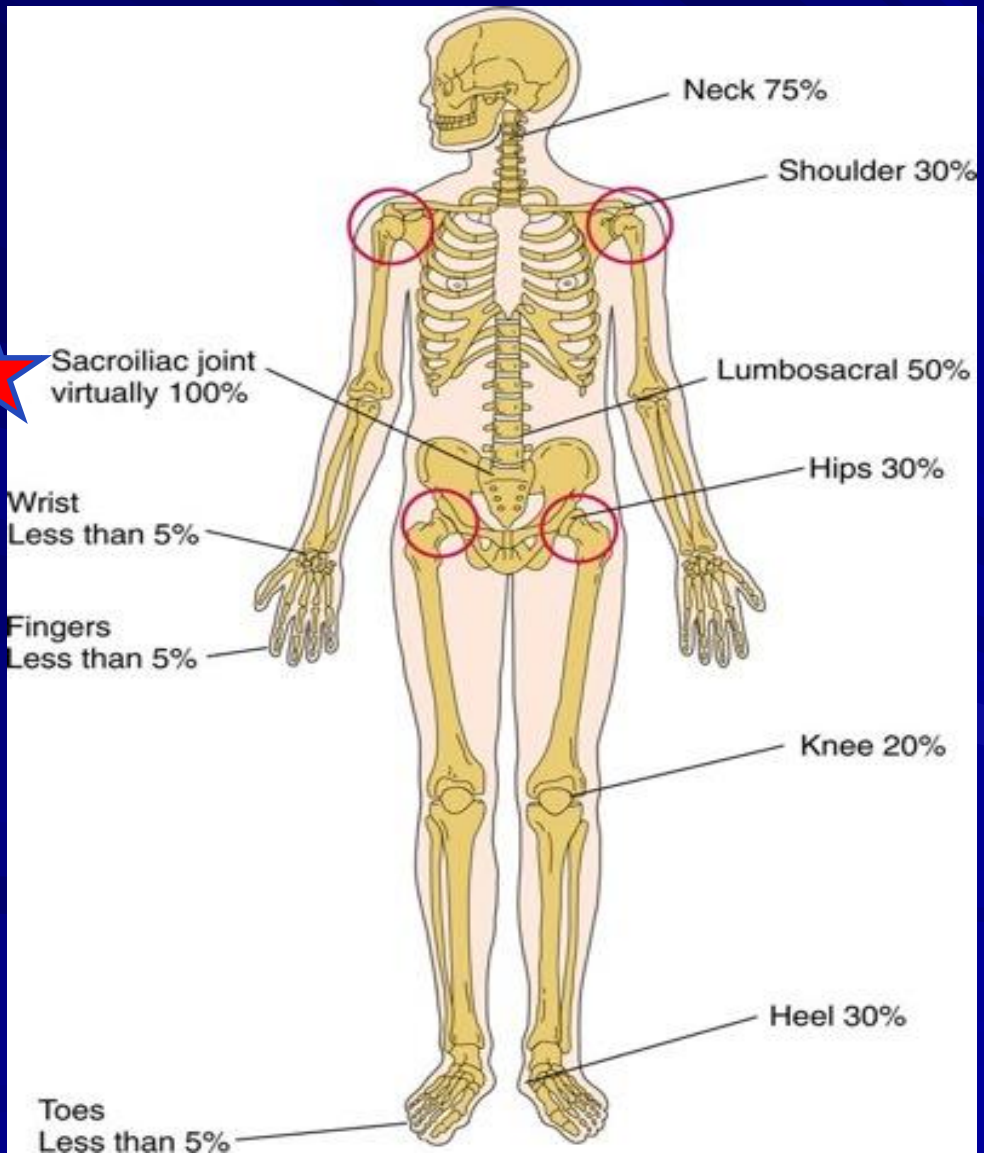
Axial Skeleton

(SI Joint & Spine)

Appendicular Skeleton

(Hips, Shoulders, Knees, Hands & Feet)

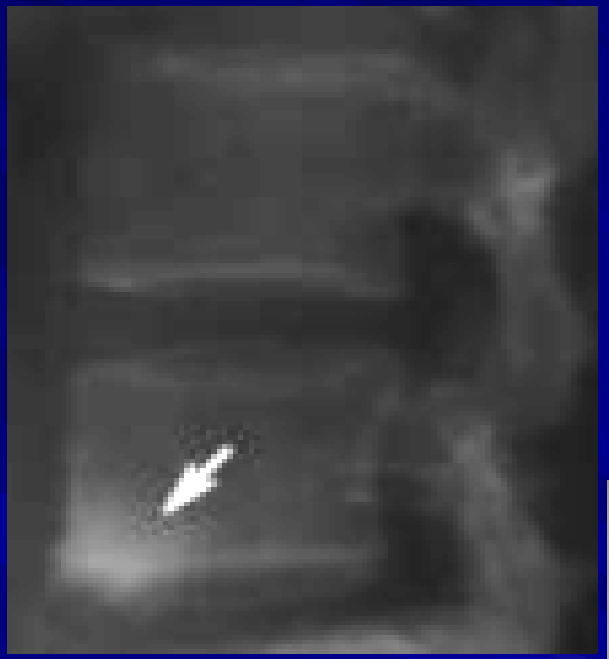
Ankylosing Spondylitis

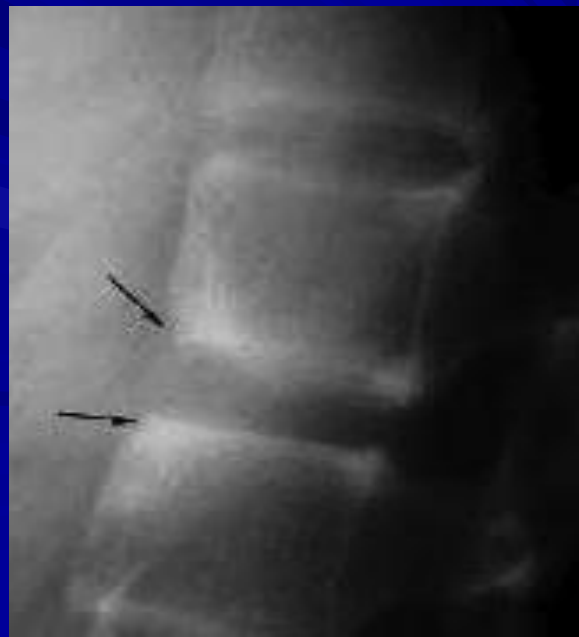


Ankylosing Spondylitis

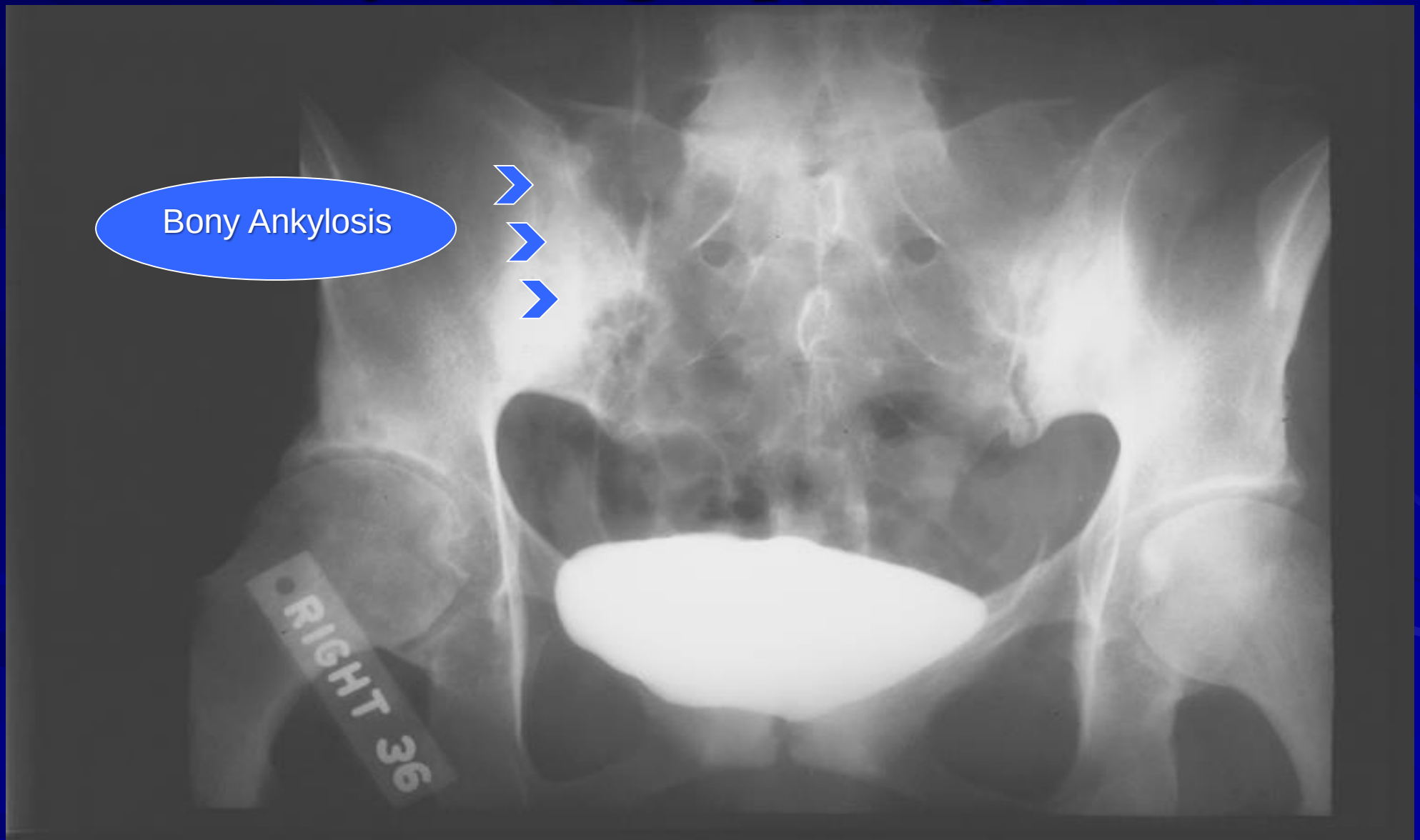
Shiny Corner Sign:

- Also known as a **Romanus Lesion**
- Early spinal finding in AS
- Represents small erosions at the superior and inferior endplates (corners on lateral radiograph) of the vertebral bodies, with surrounding reactive sclerosis
- Eventually squaring of the anterior margin of the vertebral body occurs





Ankylosing Spondylitis

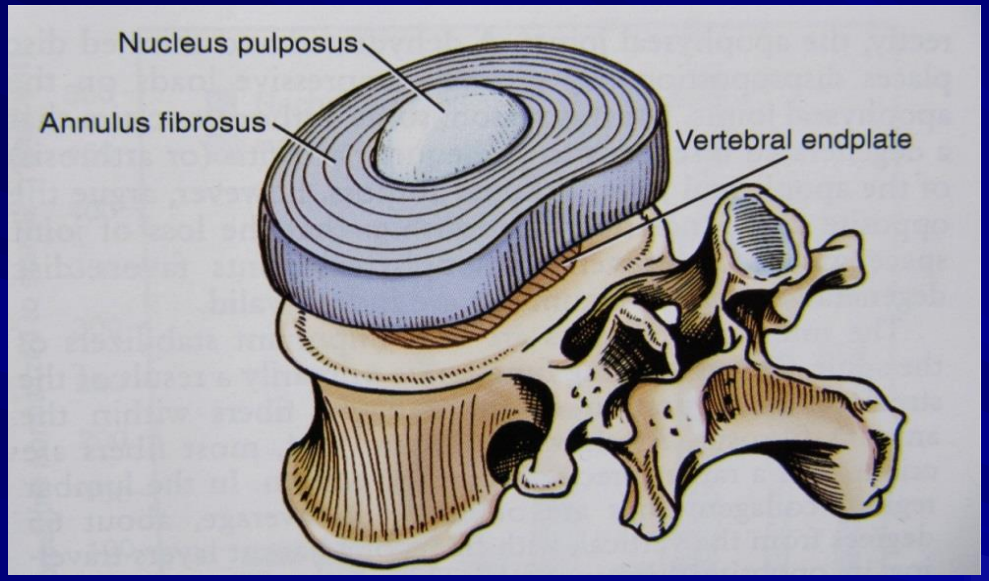


Ankylosing Spondylitis

- **Radiographic Features:**
  - Non-descript changes in LE
  - SI joint – enthesopathy
  - Spine – syndesmophytes (bamboo spine)
  - Osteopenia
  - Erosions
  - Reactive sclerosis
  - Bony ankylosis

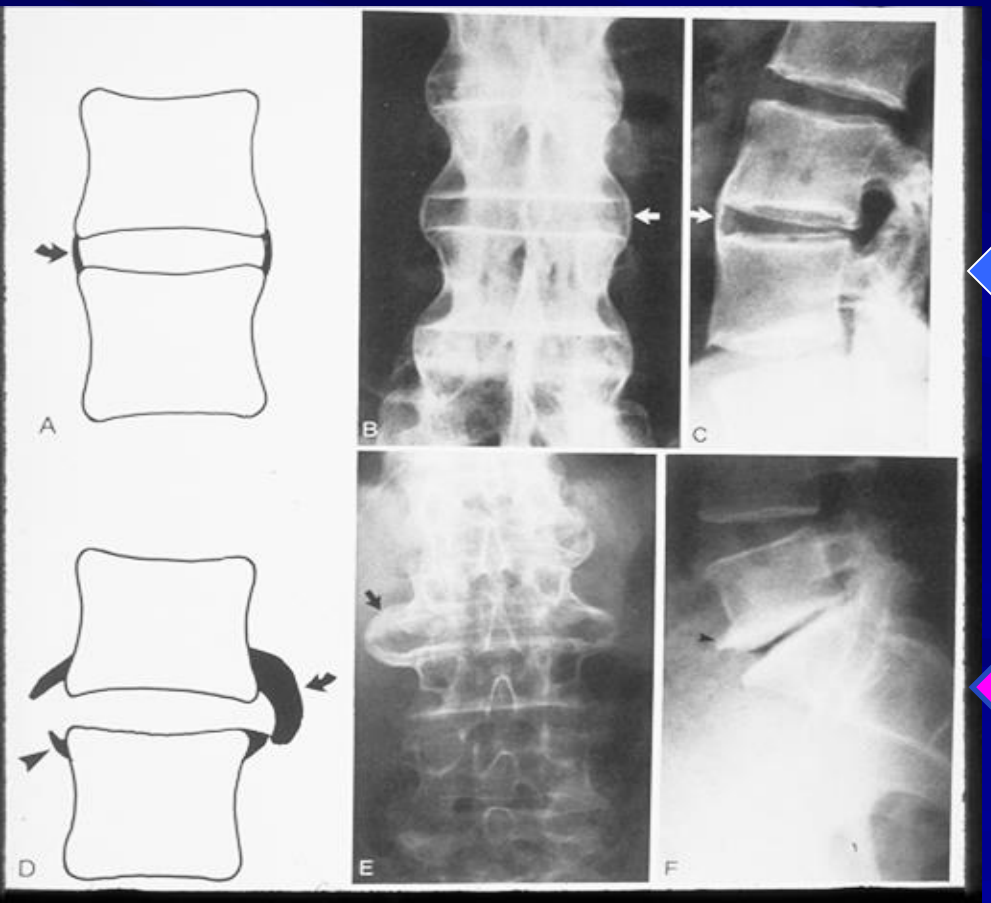
Ankylosing Spondylitis

Annulus fibrosus + Ossification = Syndesmophyte



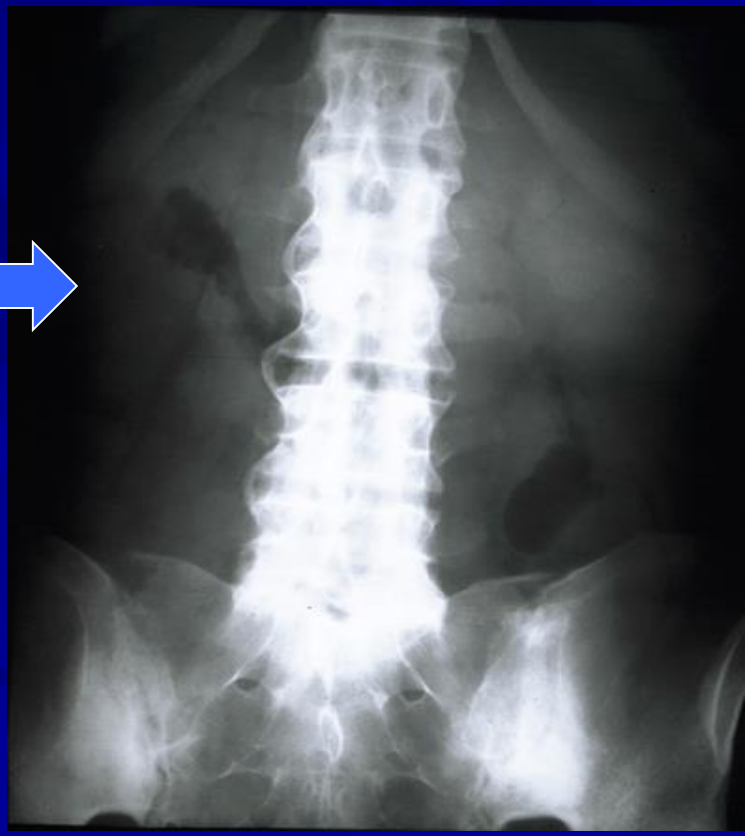
- **Annulus Fibrosus: ring of ligament fibers that surround/protect the inner core of the disc and connect the vertebrae to one another.**
- **Sharpey's fibers: outer portion of the annulus fibrosus.**
- **Syndesmophytes: ossification of Sharpey's fibers, which radiographically appear as thin vertical outgrowth of bone that extends across the margin of the intervertebral disc. Typically form at the anterior and lateral aspects of the spine, particularly near the thoracolumbar junction.**

Ankylosing Spondylitis



Bamboo Spine Syndesmophyte

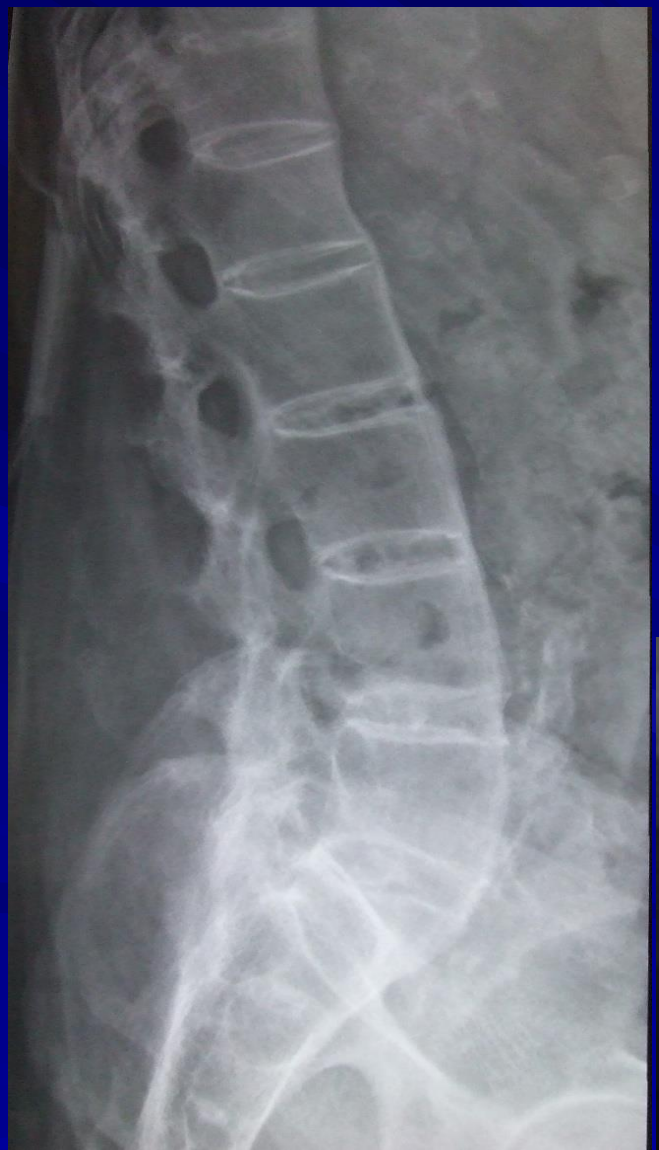
Osteophyte

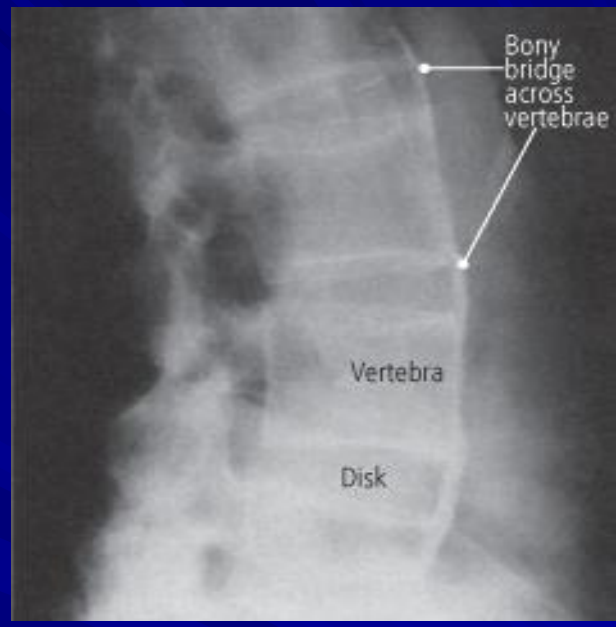


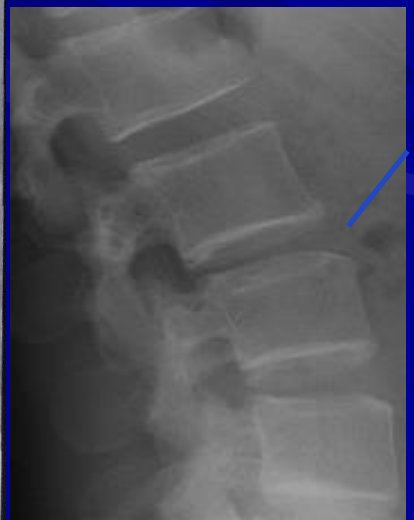
Ankylosing Spondylitis

Ossification of the Longitudinal Ligaments Connecting Adjacent Vertebrae Are Called:

SYNDESMOPHYTES







Normal

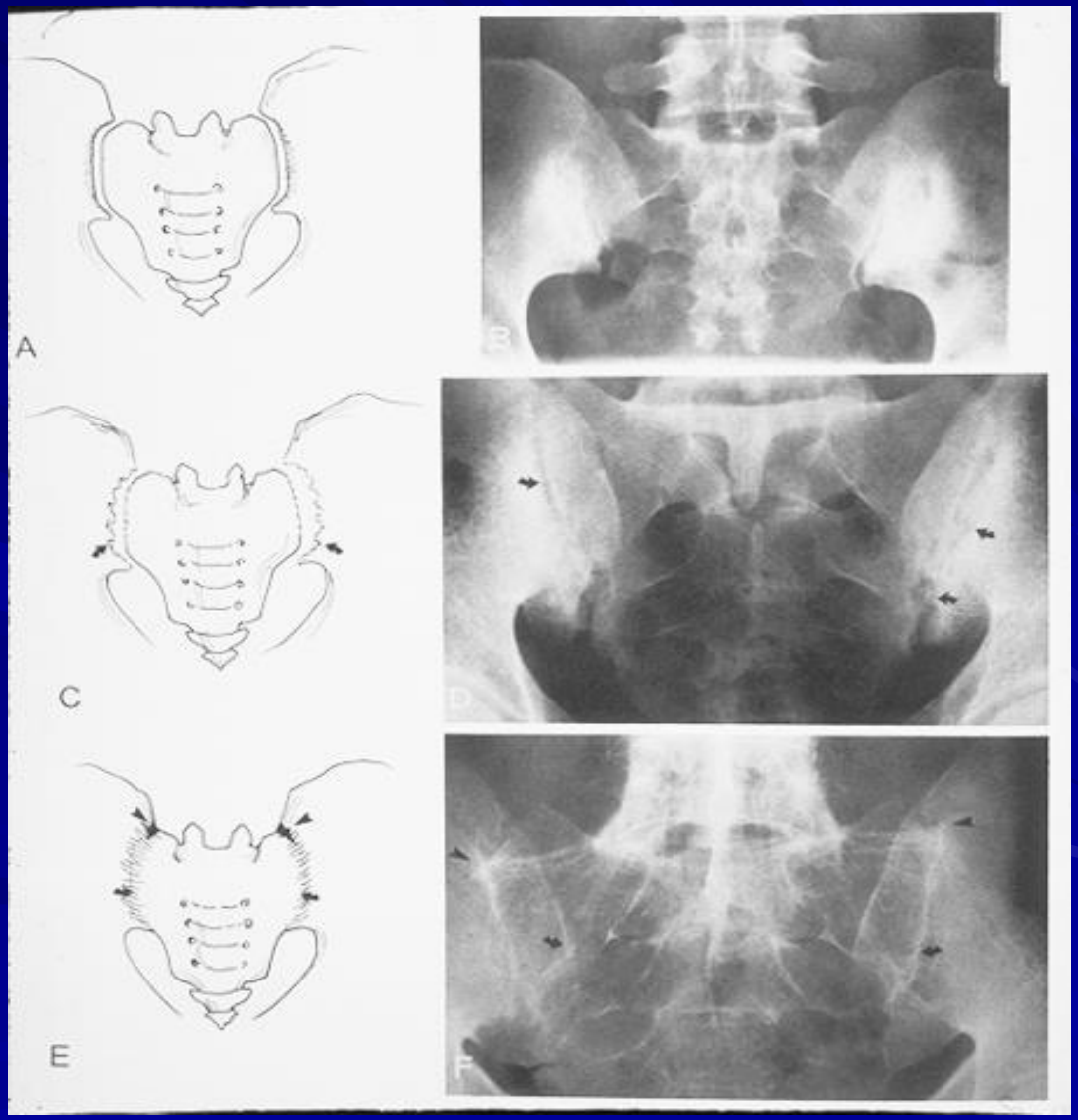
Ankylosing Spondylitis

Normal SI Joint

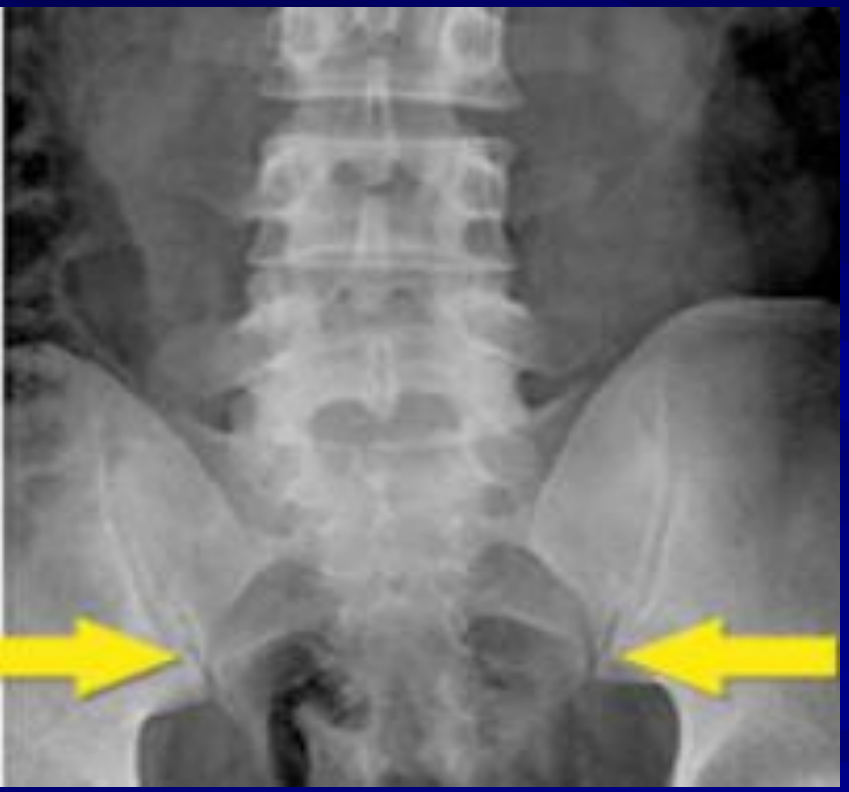
Serrated "Postage Stamp" Erosions

(typically iliac side first due to thinner cartilage)

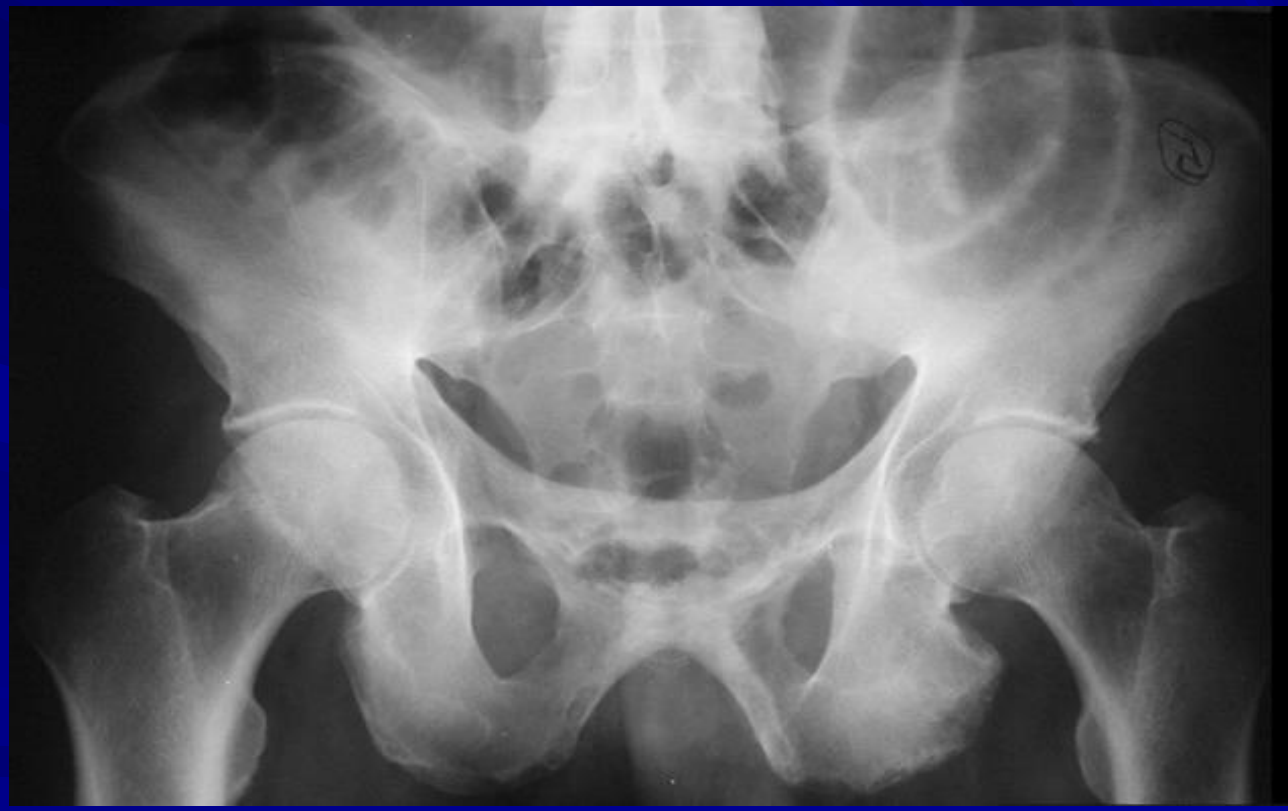
Ankylosis of SI Joint with Osteopenia



Ankylosing Spondylitis

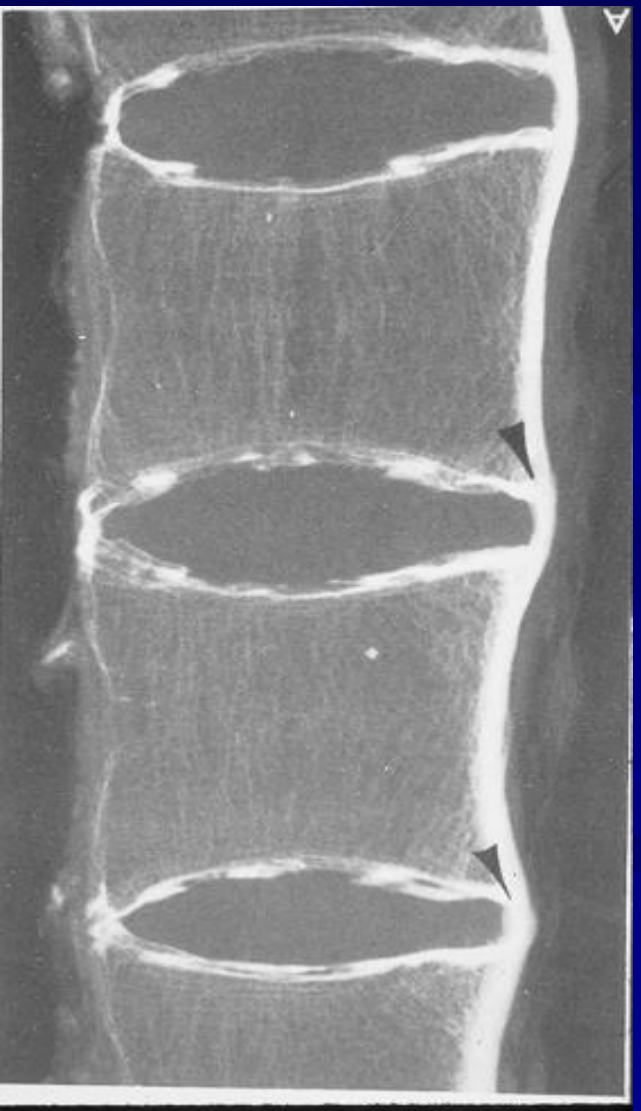


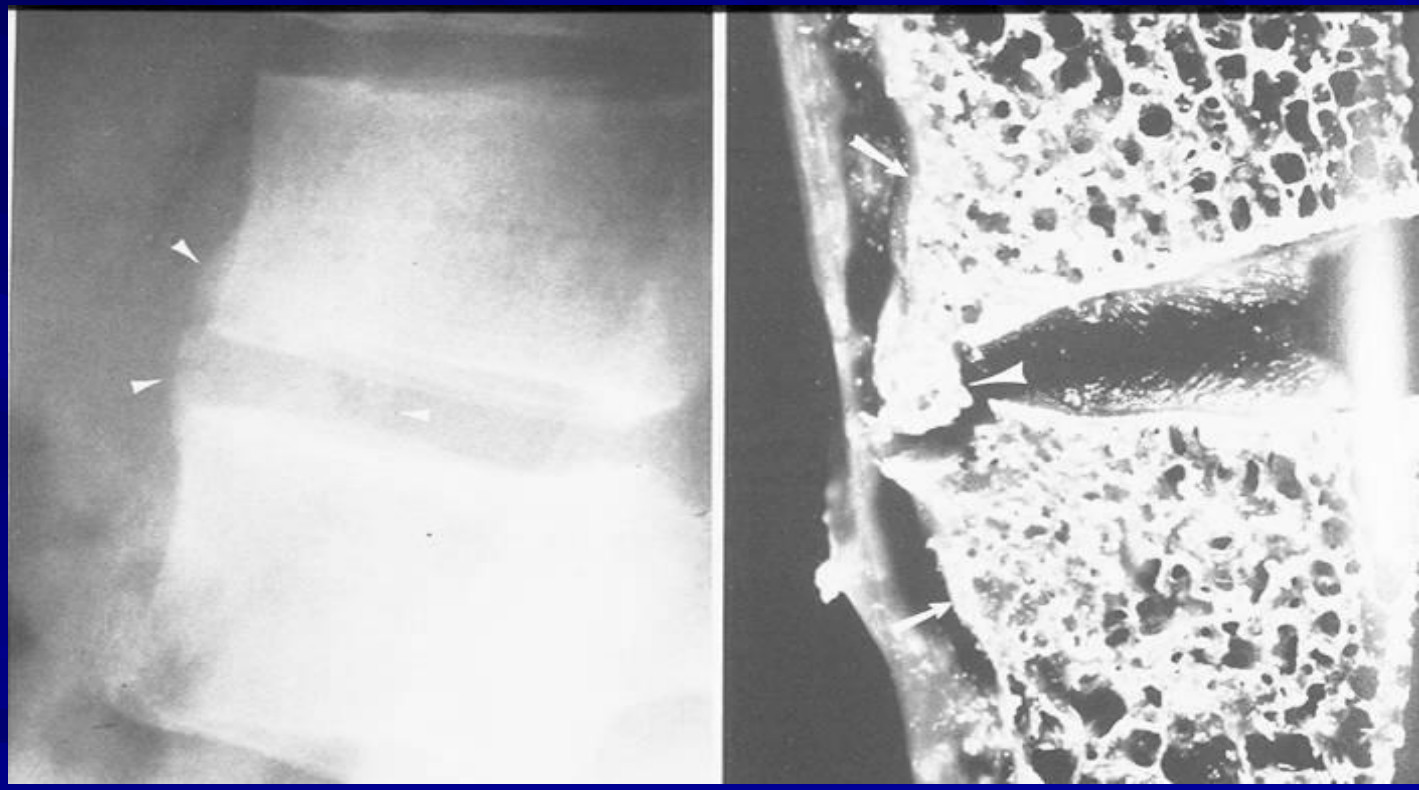
Normal SI Joint



Fused SI Joint

Ankylosing Spondylitis

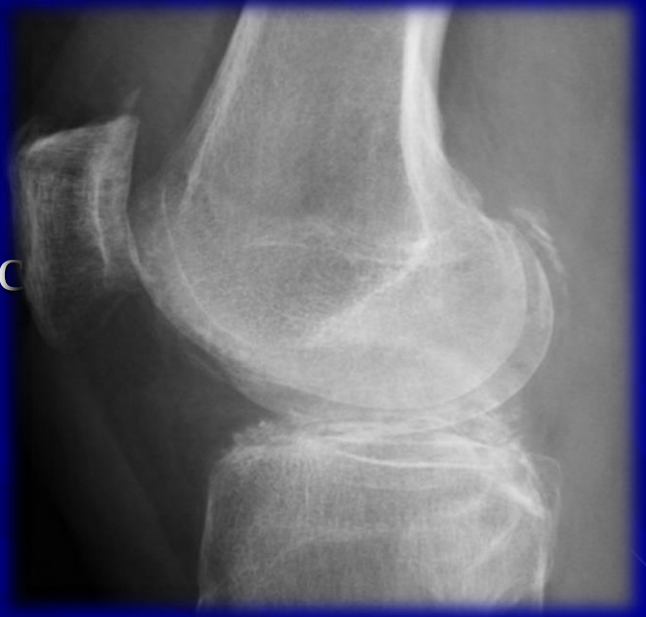


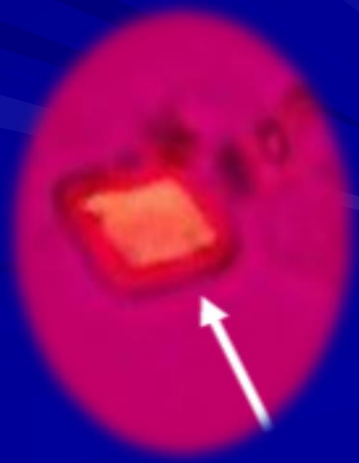


Chondrocalcinosis

Chondrocalcinosis

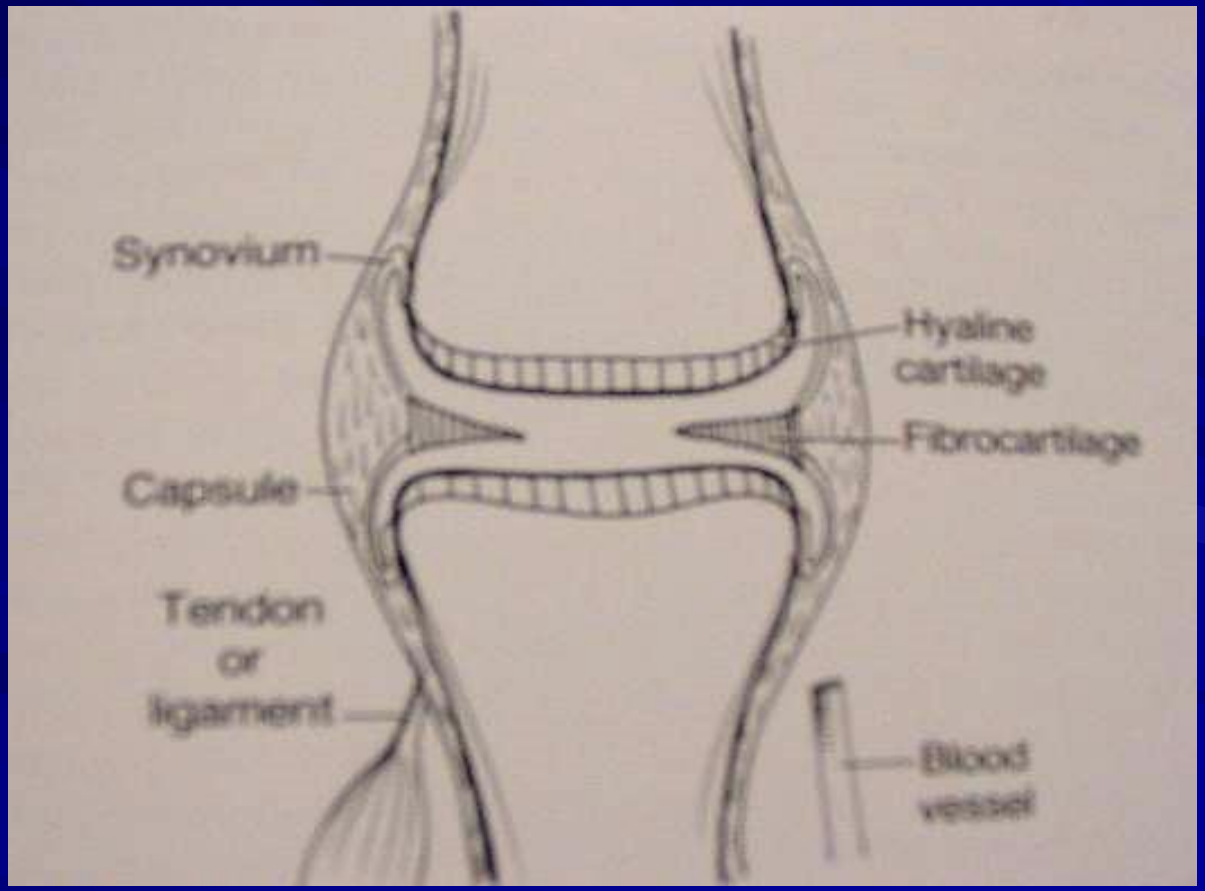
- **Gout-like symptoms with CPPD crystals**
- Clinical features
  - Age: >30 yoa, peak at 6th decade
  - 50% of people older than 85 years have radiographic evidence of **chondrocalcinosis**
  - Gender: Males = females
  - Can simulate acute gout attack- erythema, edema, fever, increased calor at the joint, & pain
- Lab findings
  - Elevated ESR
  - Polarizing microscopy – **weak positive birefringence of the rod/rhomboid shaped crystals**



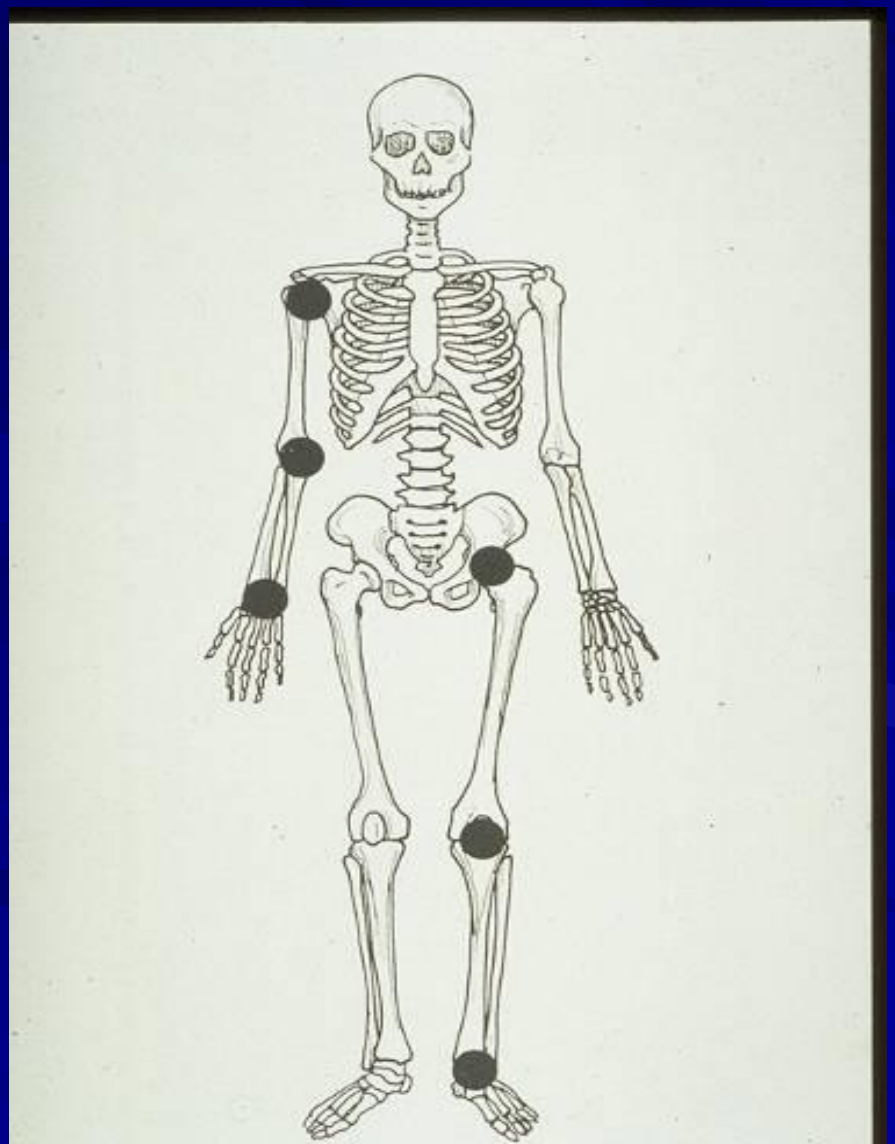


Chondrocalcinosis

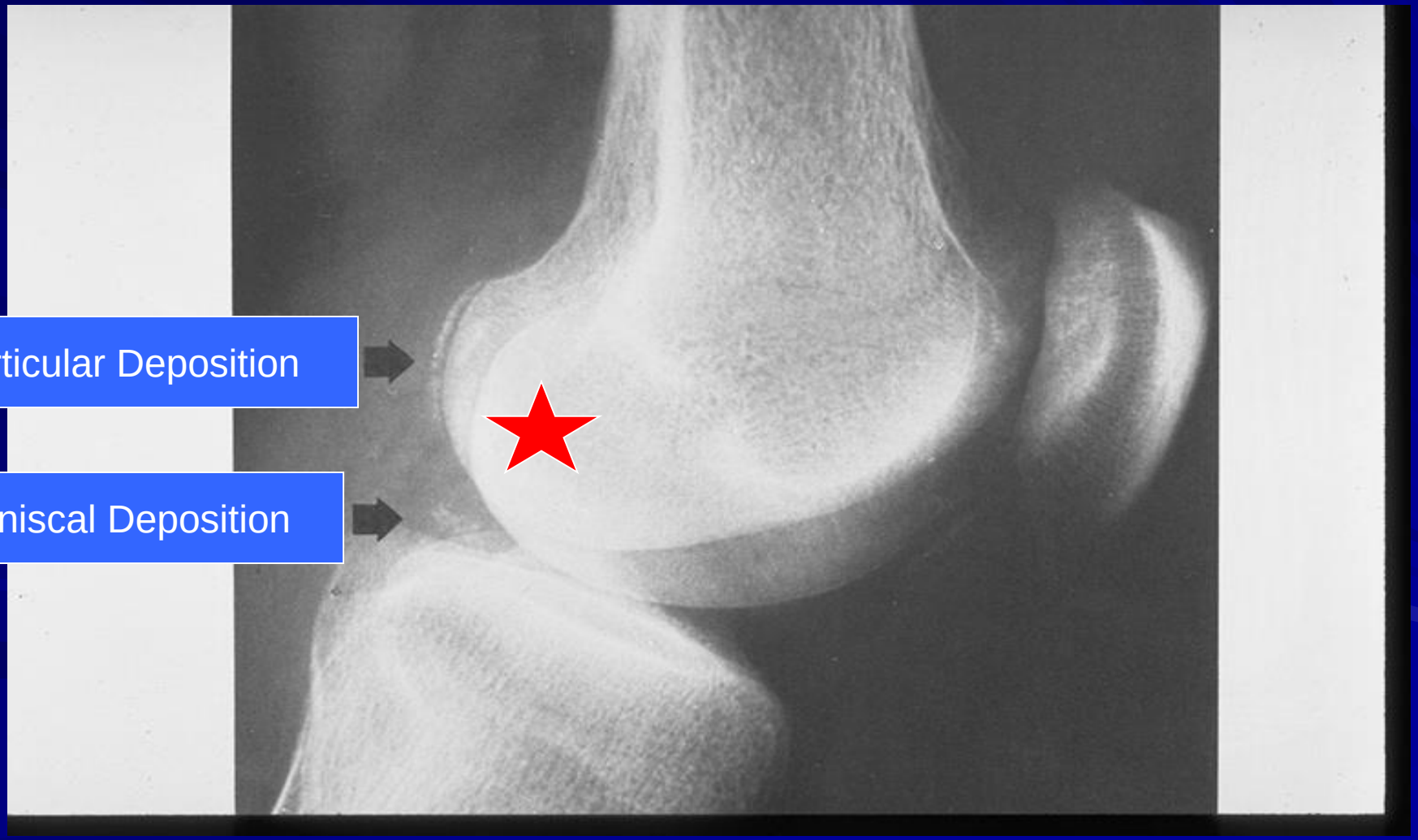
Potential sites for calcification in CPPD



Chondrocalcinosis

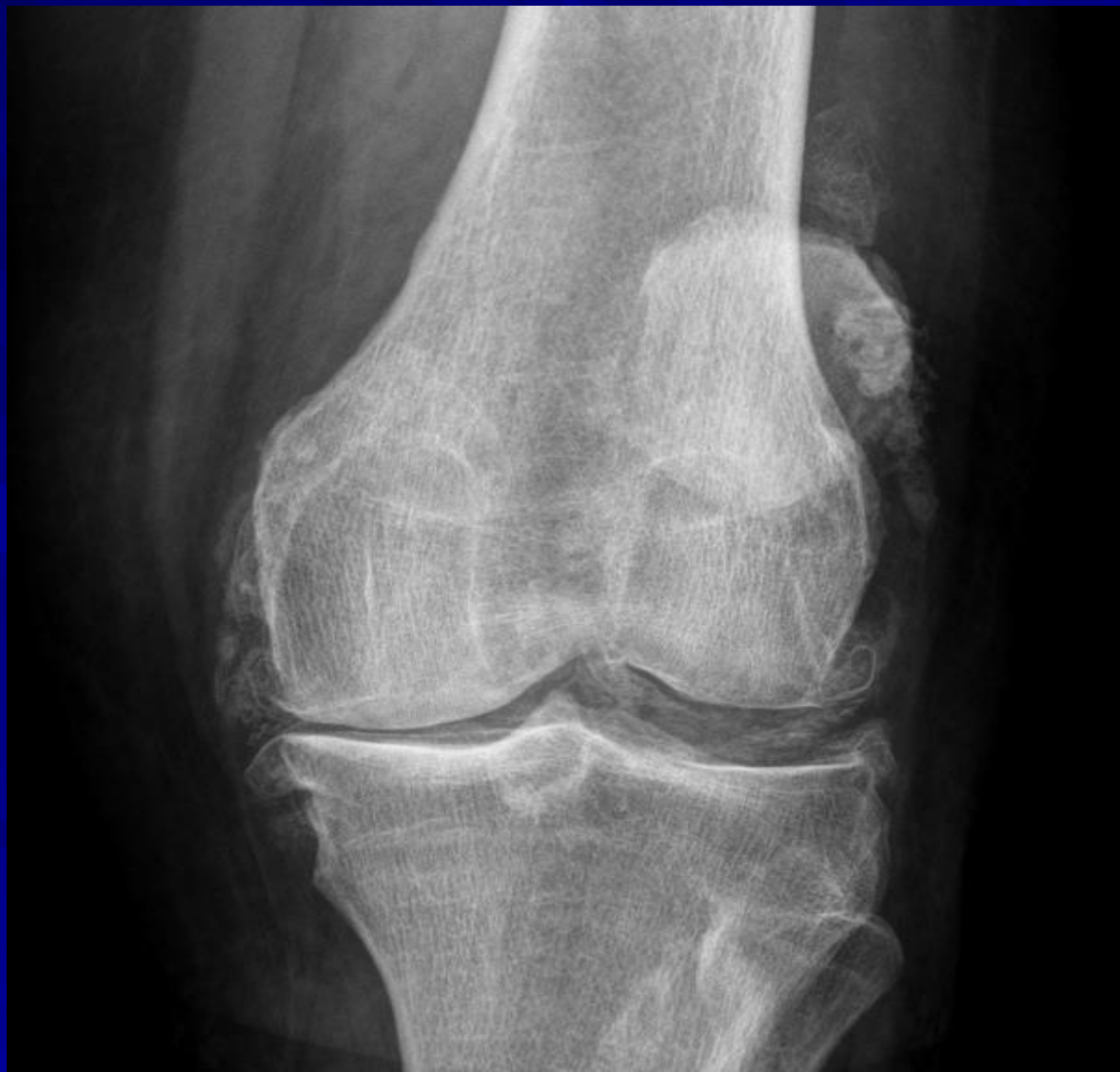


Chondrocalcinosis

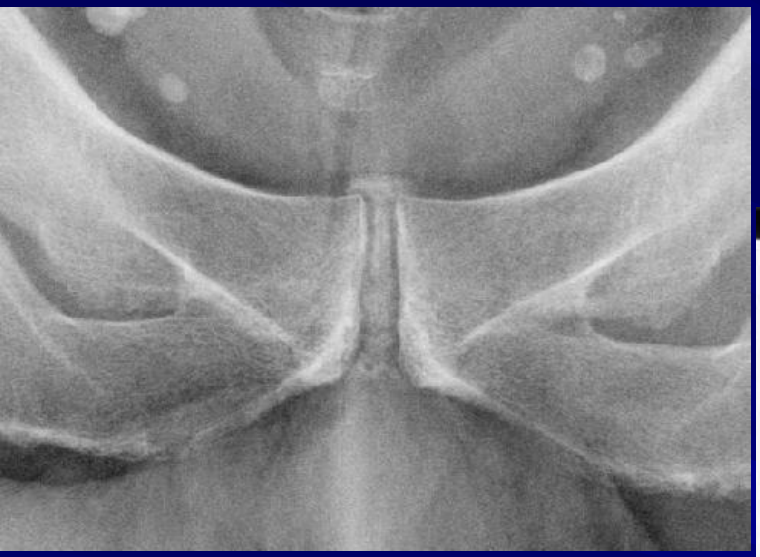


Chondrocalcinosis

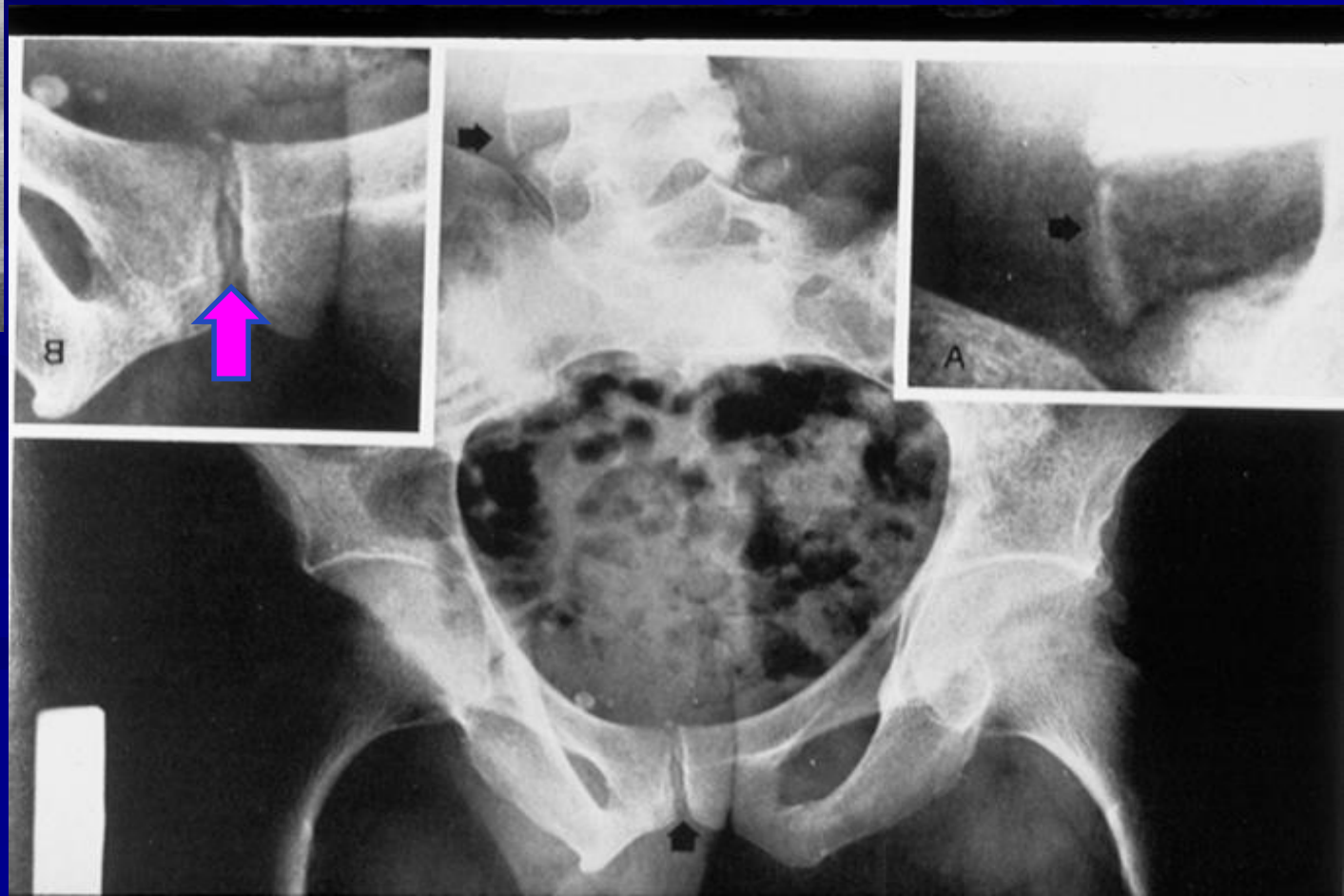
- Distribution
  - **Knee**
  - Wrist
  - Hand
  - Ankle
  - Symphysis pubis
  - Elbow



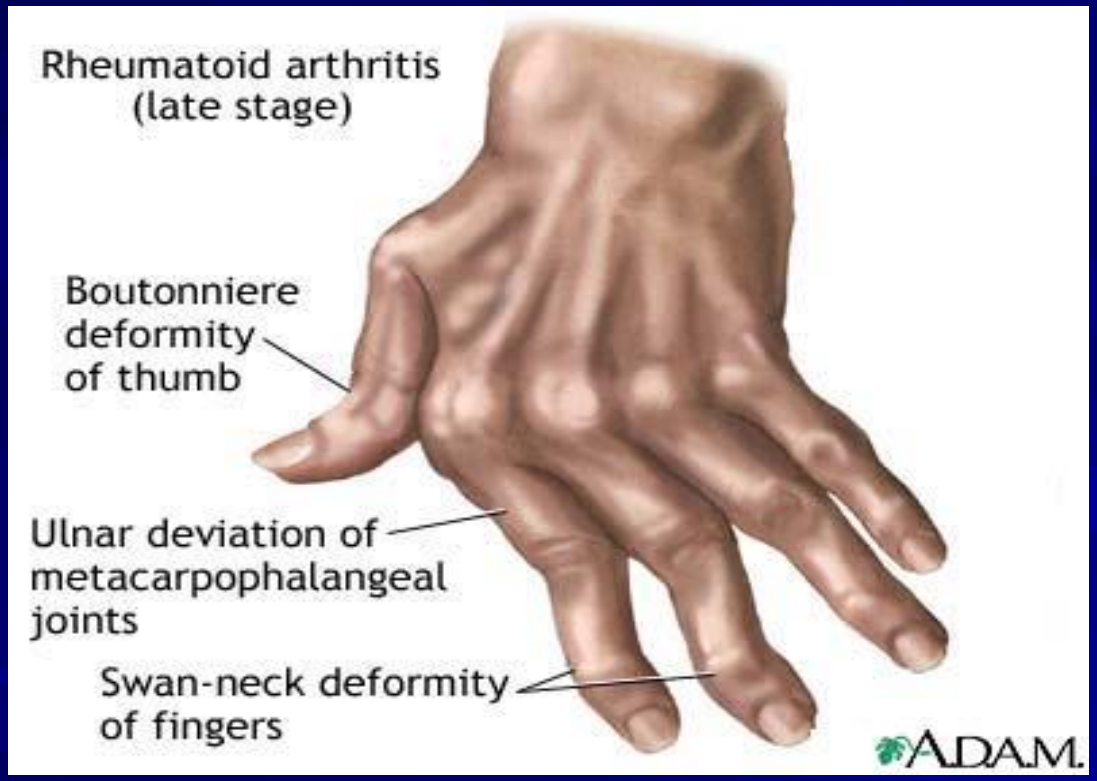
Chondrocalcinosis



Pubic Symphysis
CPPD Deposition

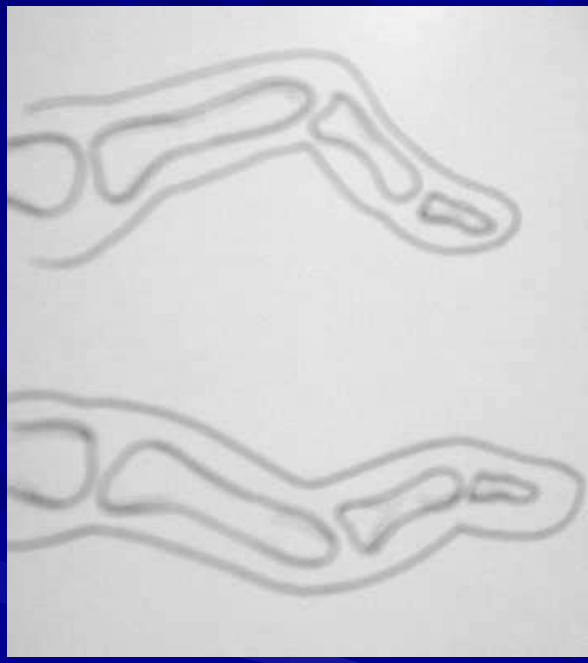


Rheumatoid Arthritis



Boutonniere Deformity

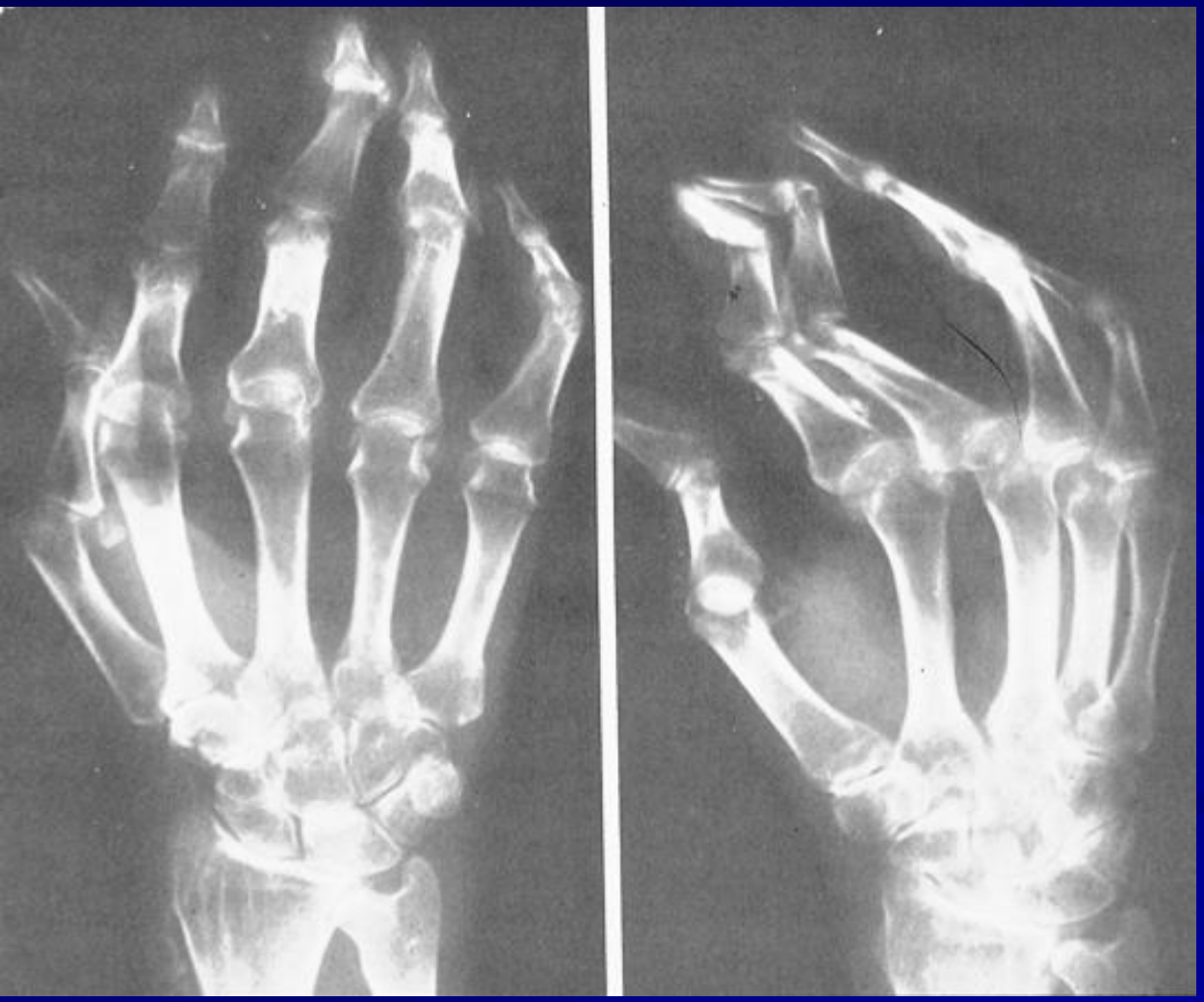
Swan-neck Deformity

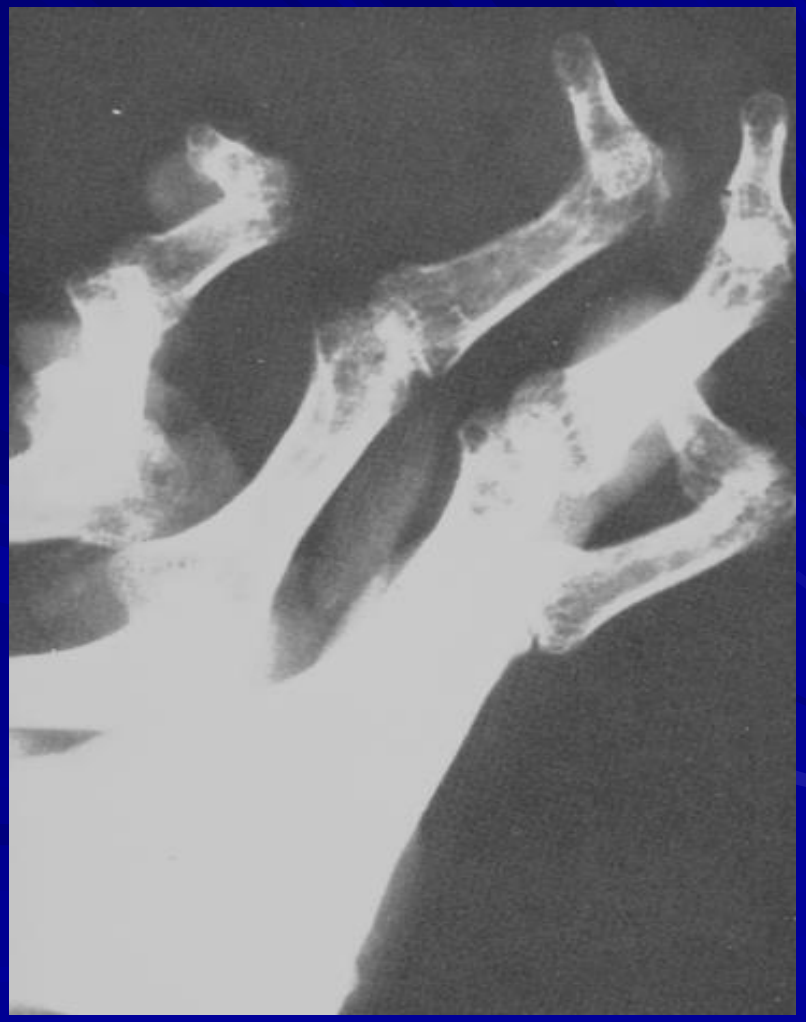


Rheumatoid Arthritis

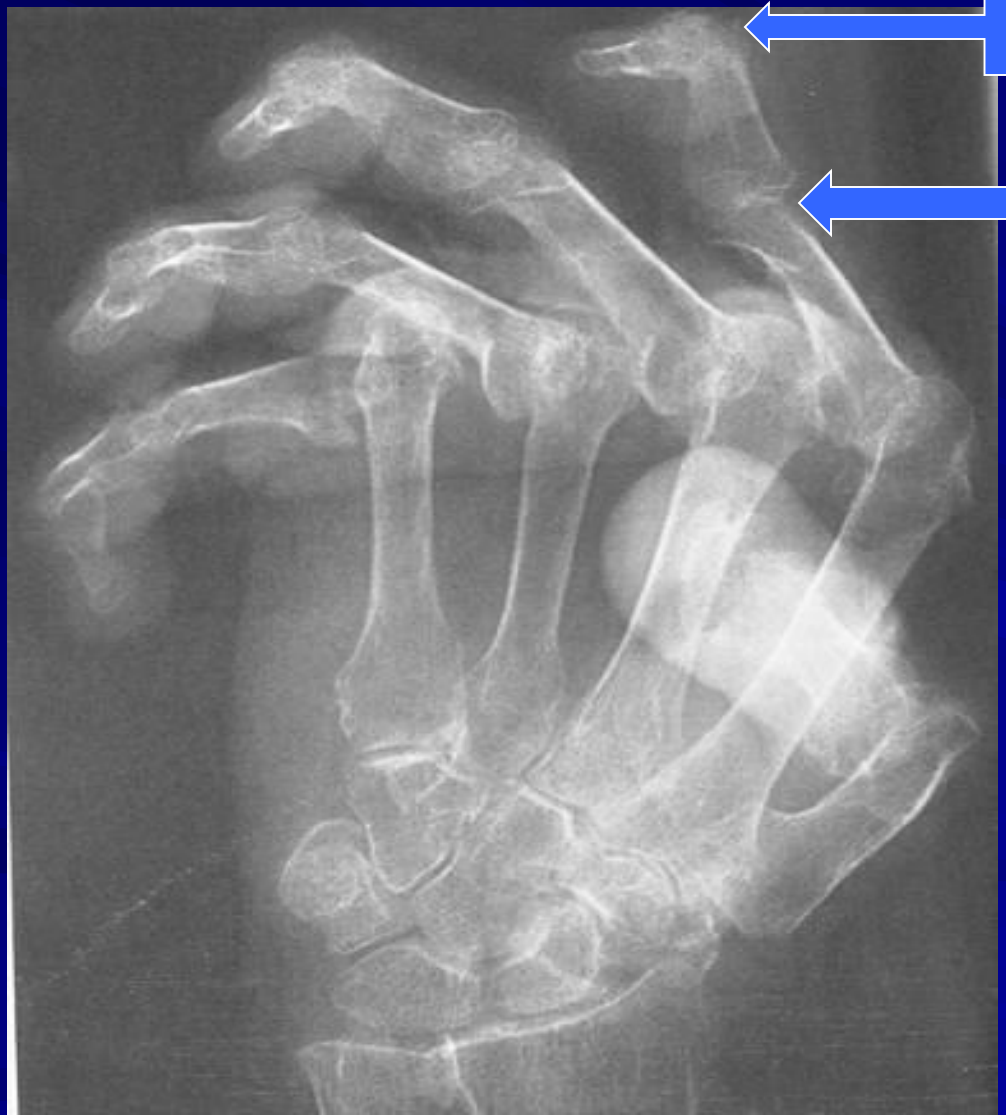
- Late Radiographic Changes:
  - SWAN-NECK DEFORMITY
    - Flexion of the DIPJ and hyperextension of the PIPJ
    - Seen in SLE and scleroderma

Rheumatoid Arthritis





Rheumatoid Arthritis



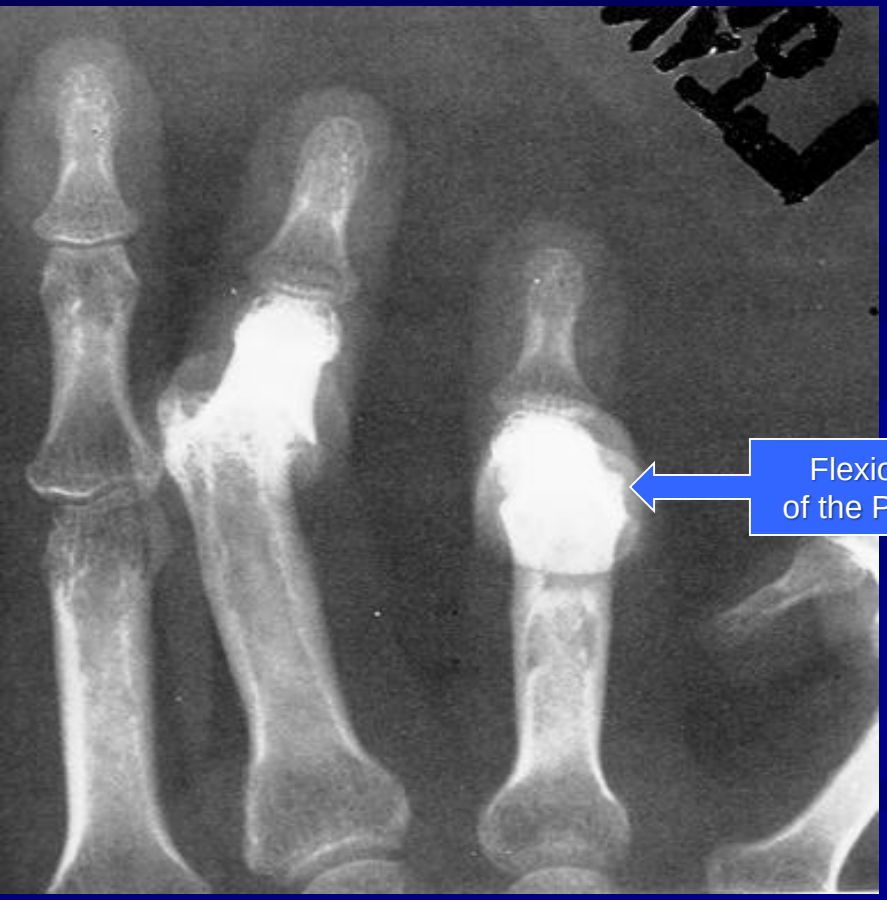
Flexion of the DIPJ

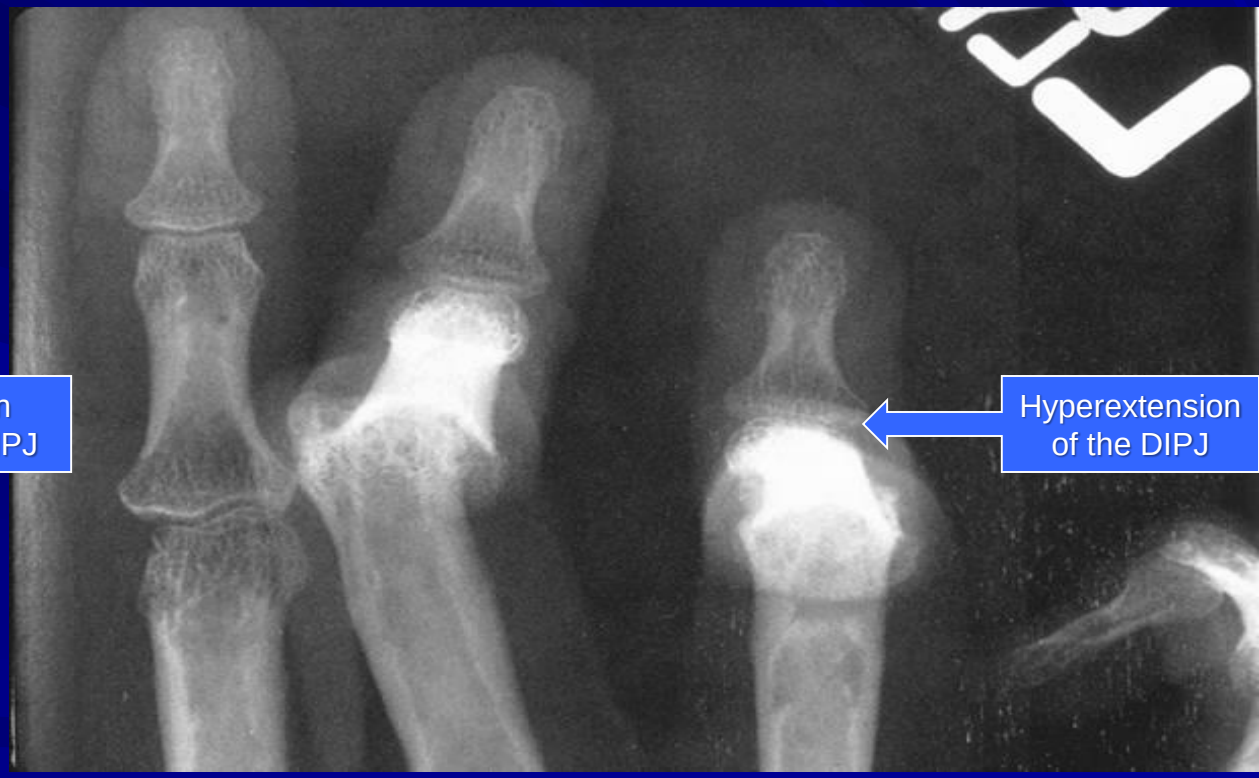
Hyperextension of the PIPJ

Rheumatoid Arthritis

- Late Radiographic Changes
  - BOUTINNEIRE DEFORMITY
    - Flexion of the PIPJ and hyperextension of the DIPJ
    - Named because of the appearance of the fingers placing a carnation in a button hole
    - Common in RA but also seen in SLE and Jaccoud's (post-rheumatic fever) arthritis

Rheumatoid Arthritis

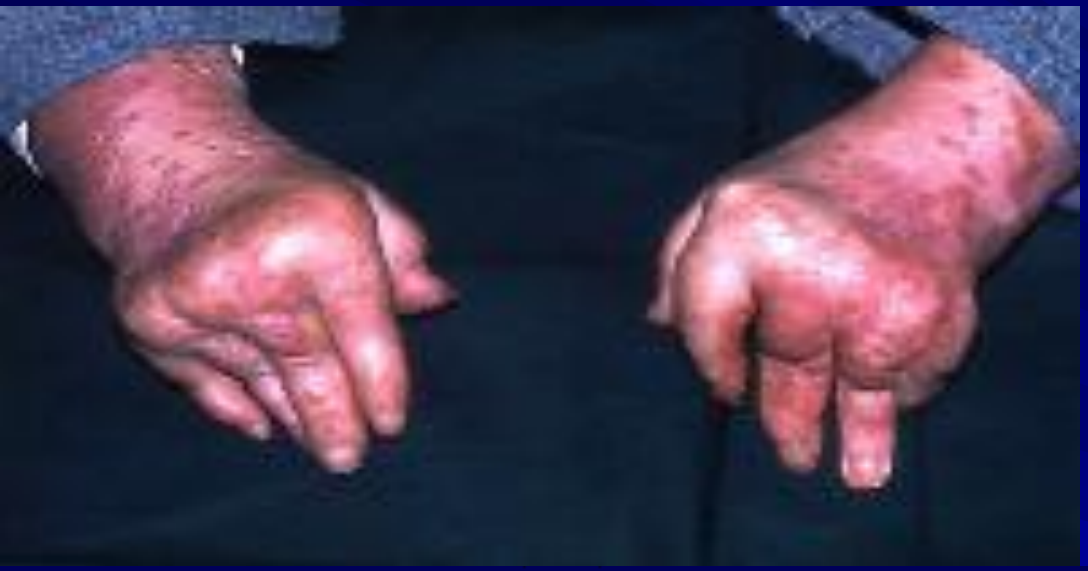


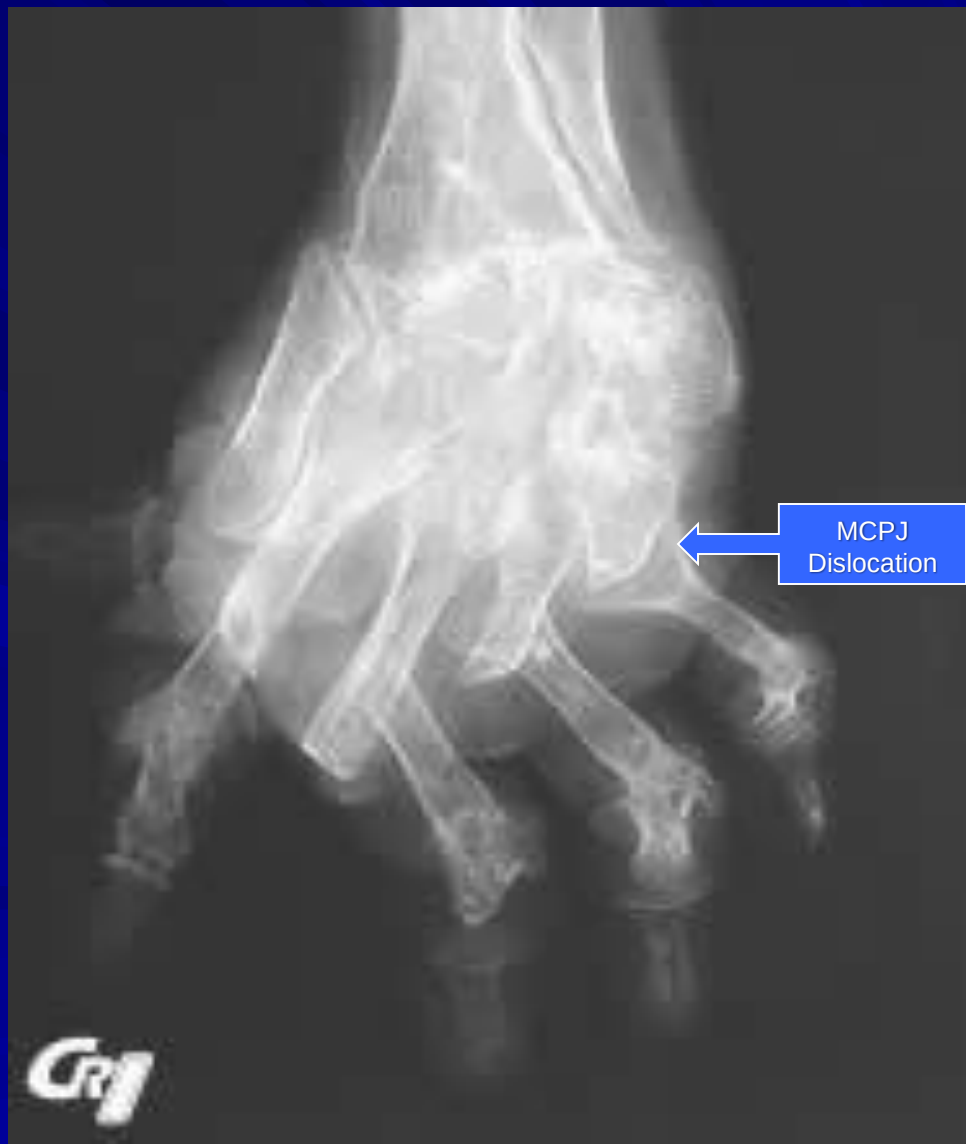


Rheumatoid Arthritis

- Late Radiographic Changes:
  - MAIN EN LORGNETTE DEFORMITY
    - AKA "Opera Glass Hand"
    - Seen in advanced RA, advanced PA, & erosive osteoarthritis
    - Develops as a result of:
      - Shortening of several proximal phalanges secondary to compressive erosions & destruction of bony ends
      - MCPJ dislocations causing a "telescoping" & retraction of the fingers

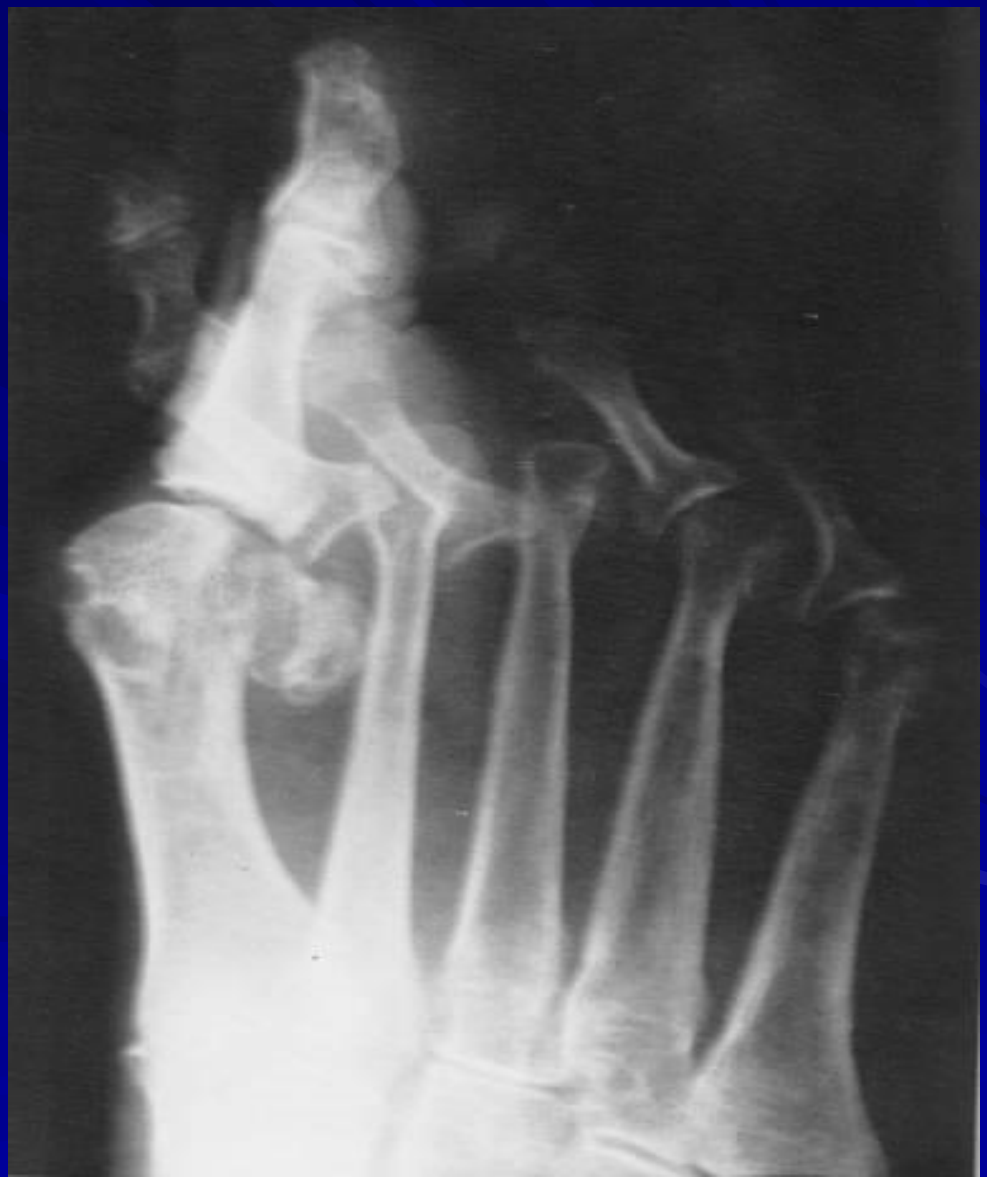
Rheumatoid Arthritis



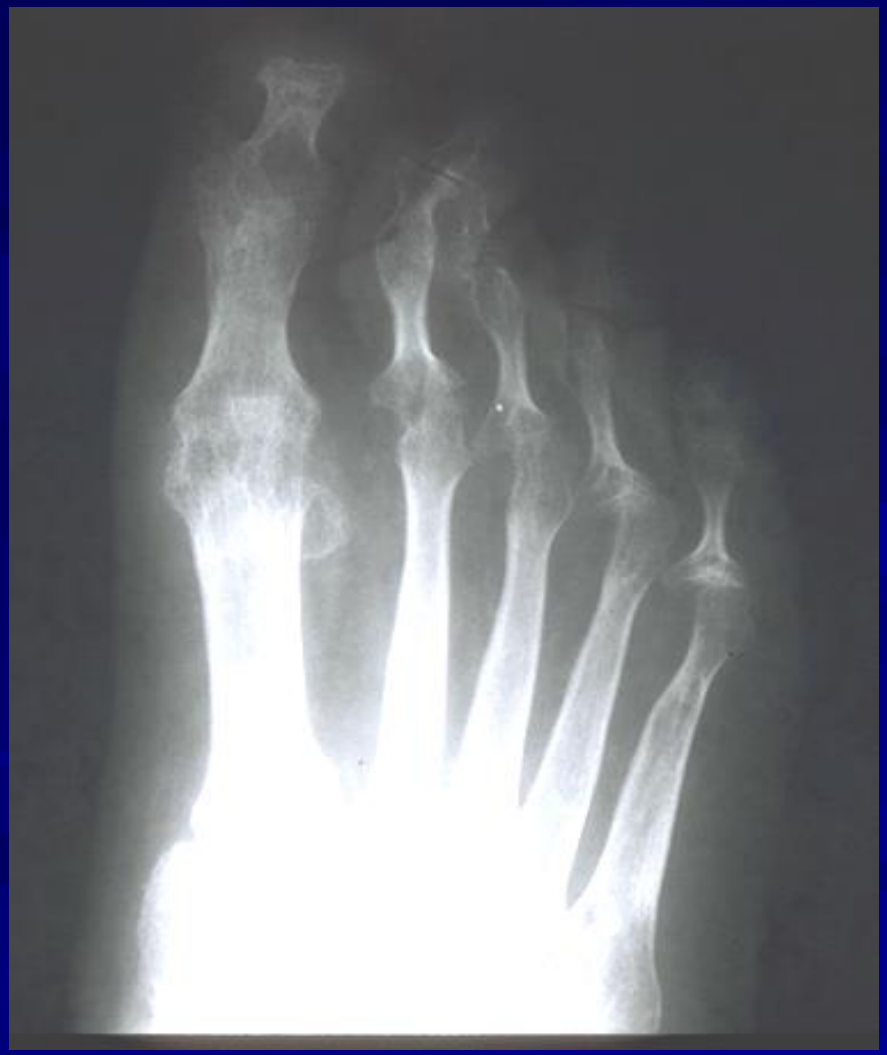


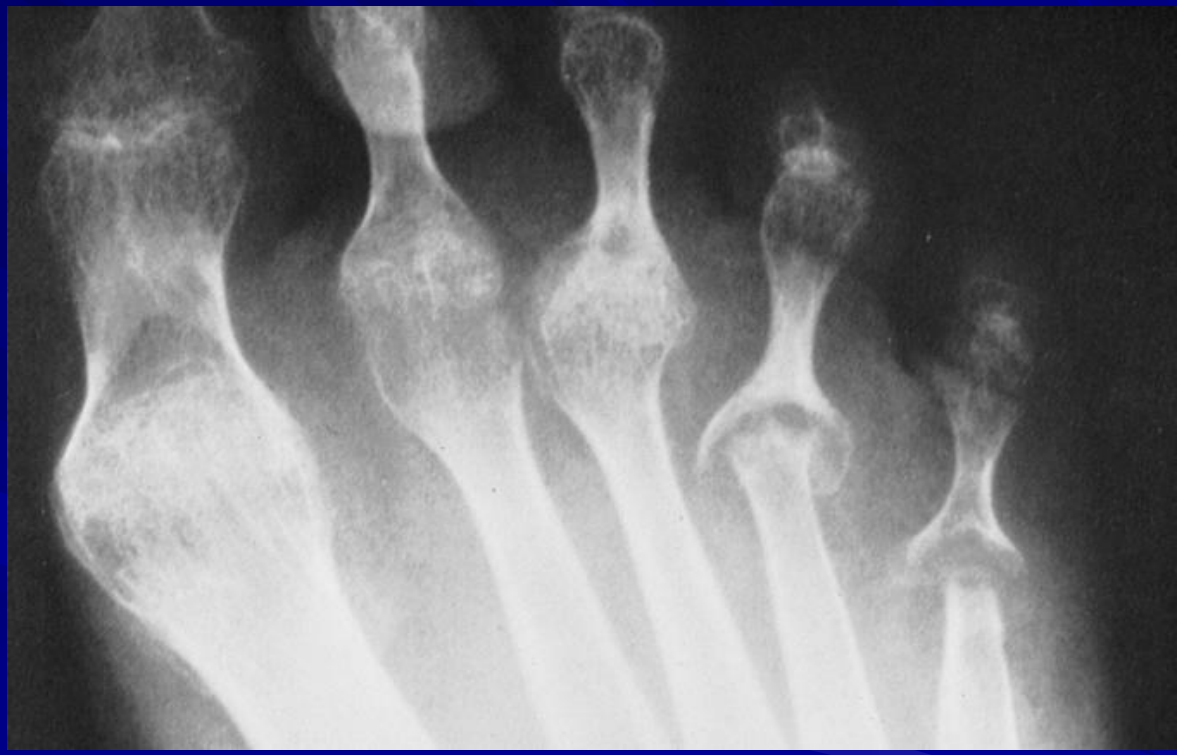
Rheumatoid Arthritis

Dorsal Dislocation of MPJs



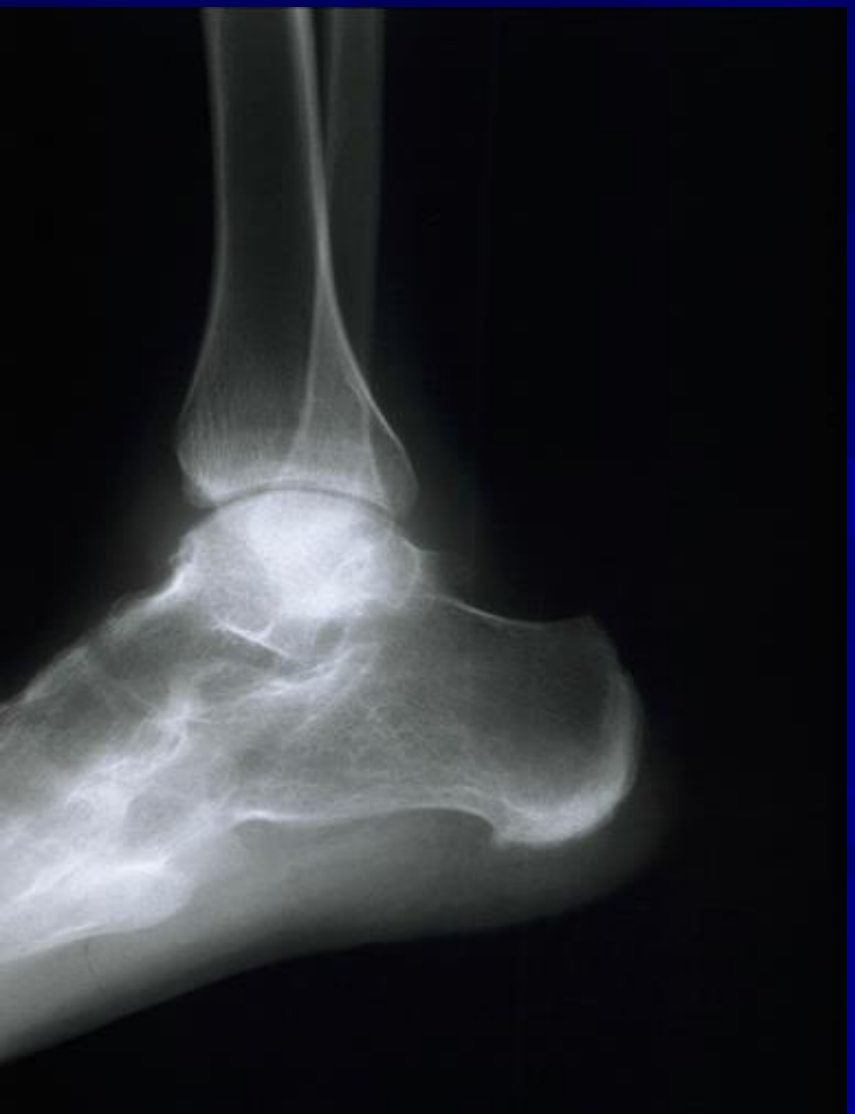
Rheumatoid Arthritis

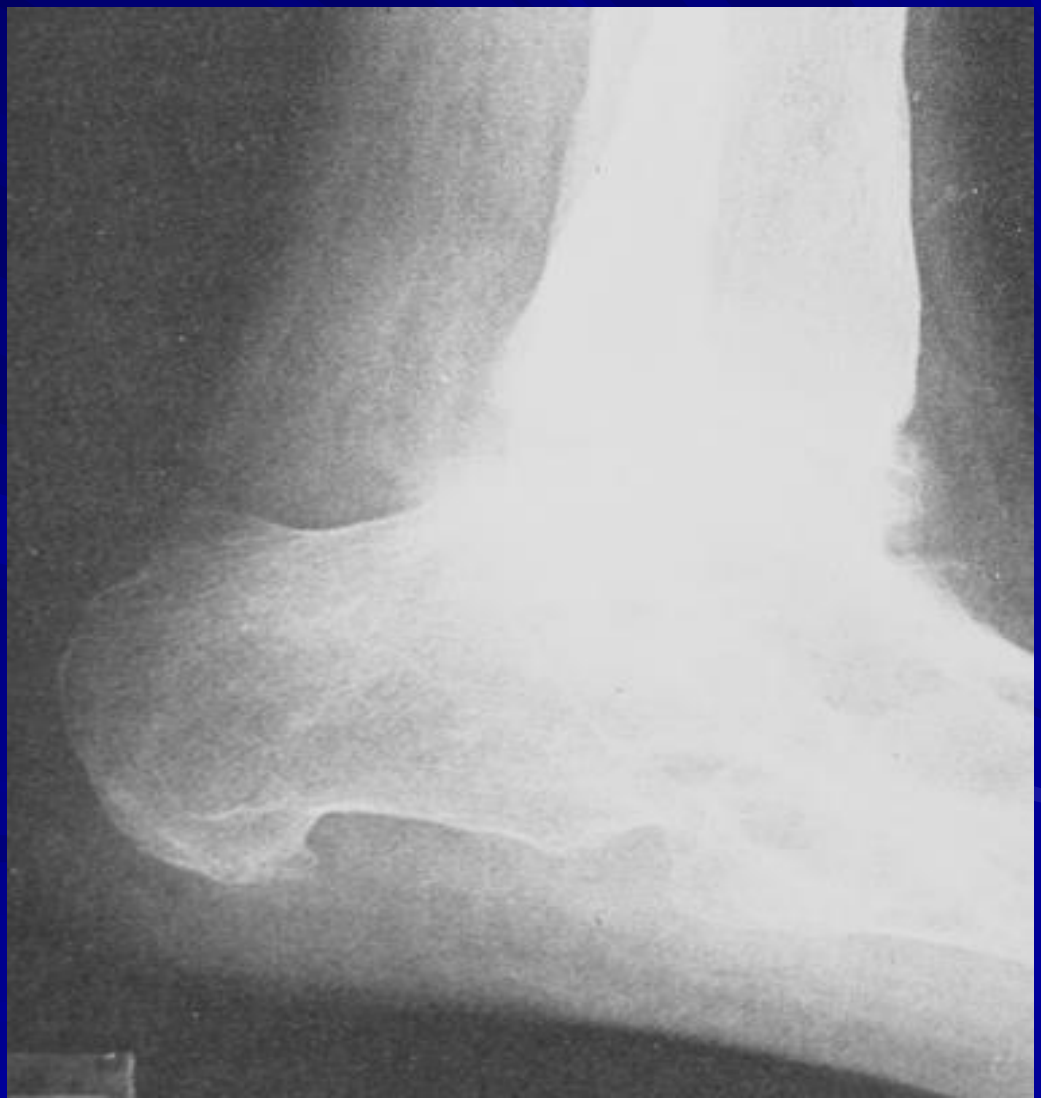




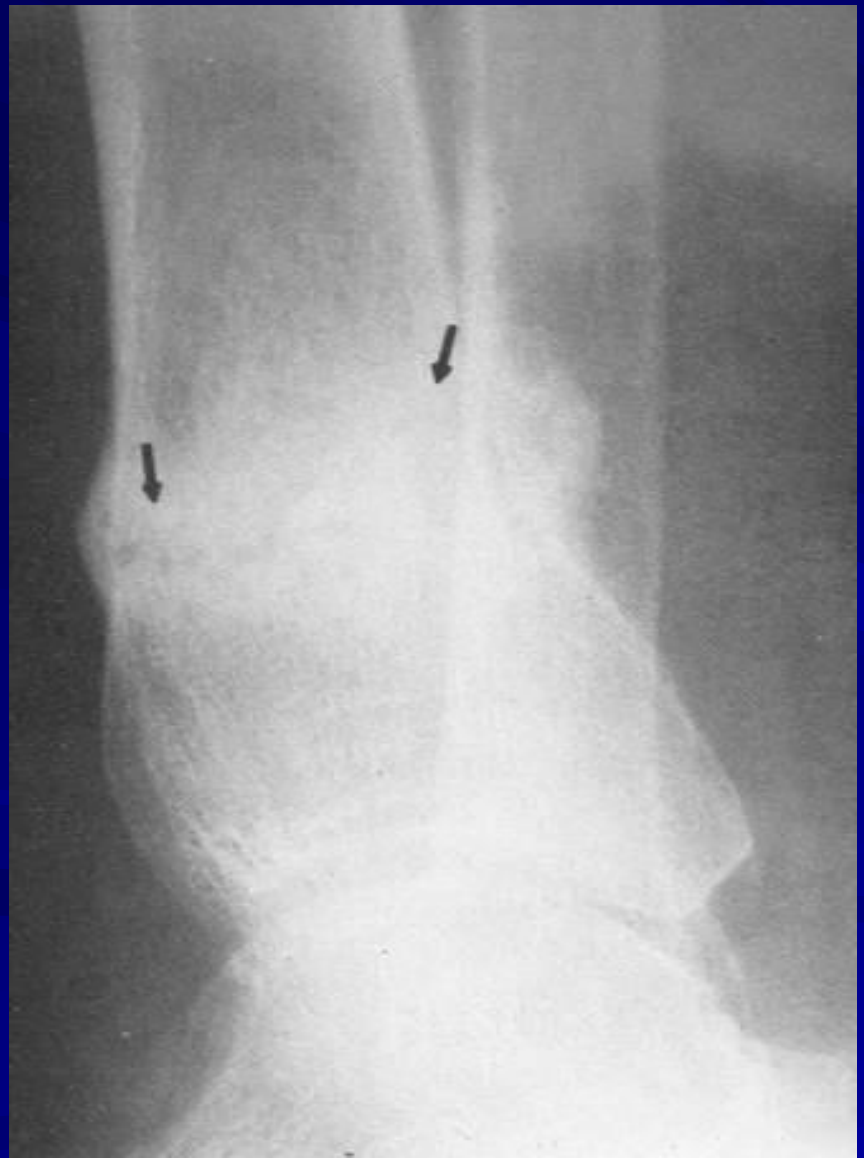
Bony Ankylosis of MPJs

Rheumatoid Arthritis

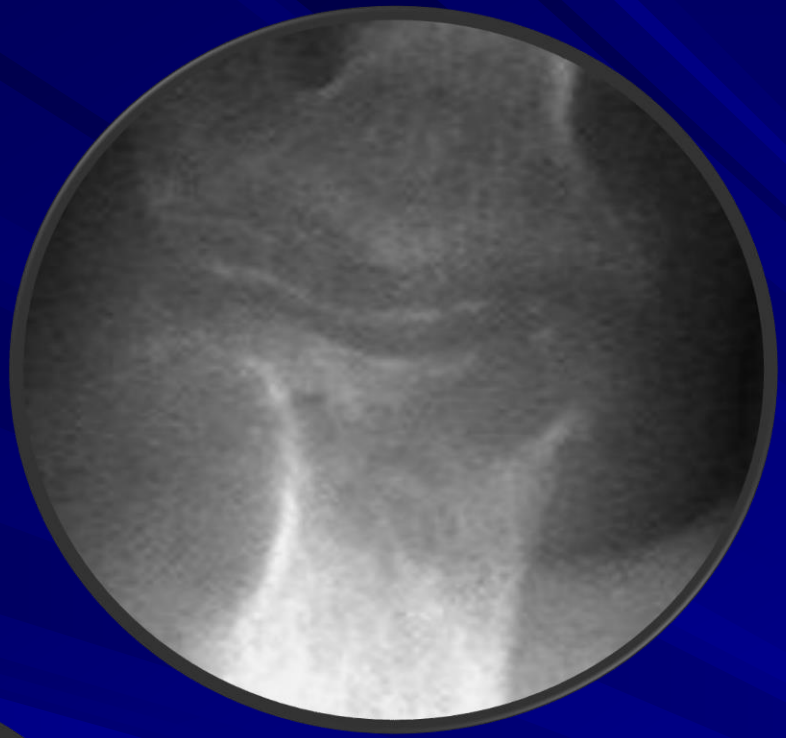




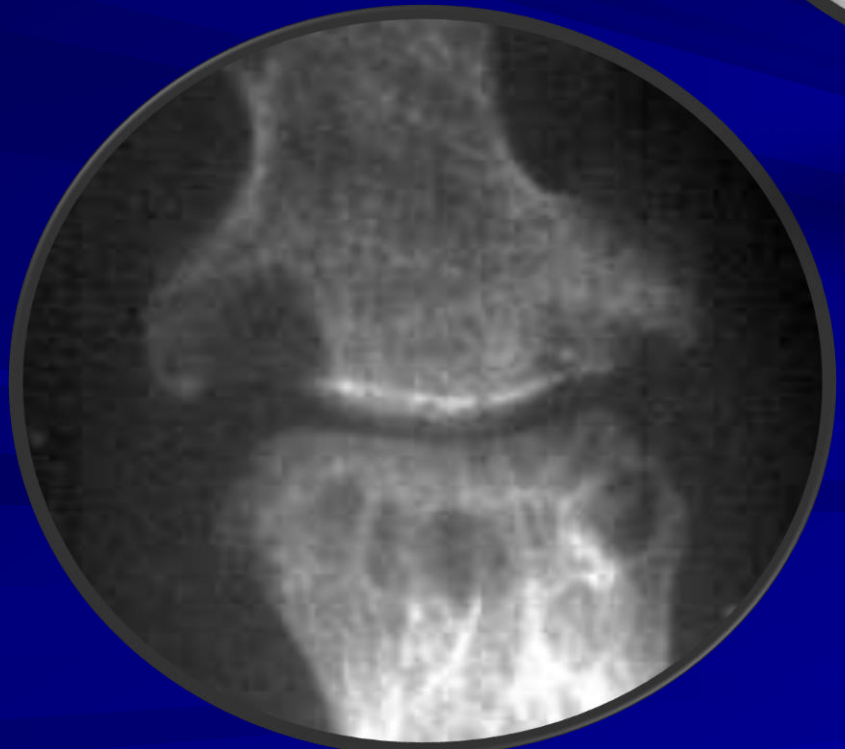
Rheumatoid Arthritis

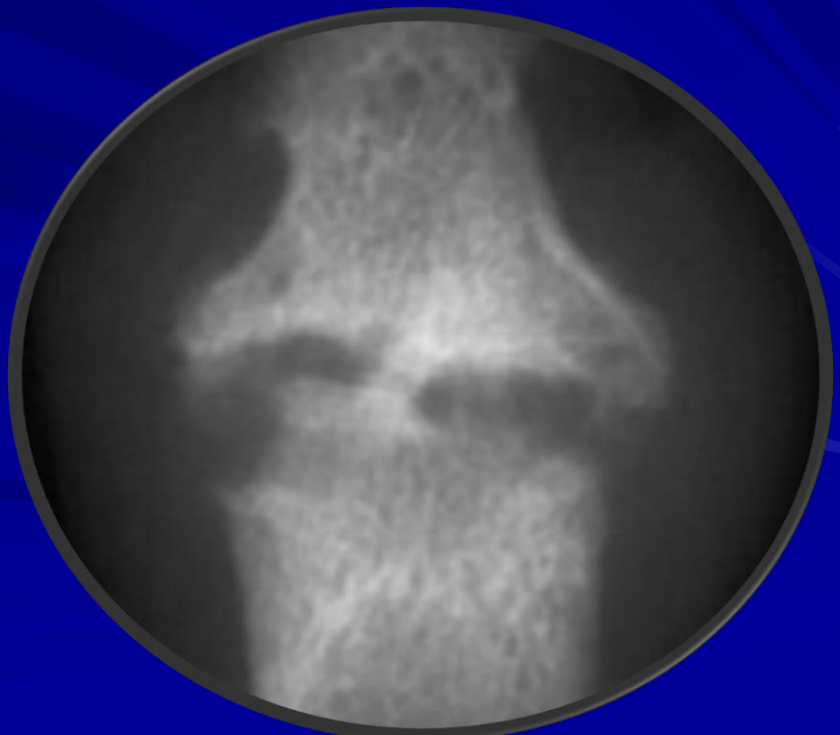


Pathological Fractures Secondary To The Osteopenia Not Uncommon

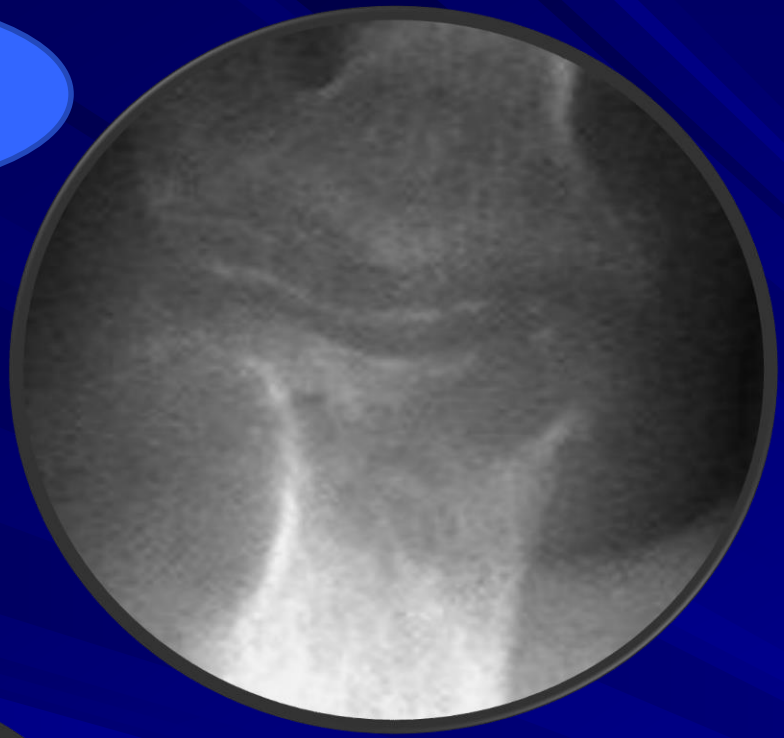


State the diagnosis for each of the following X-rays.



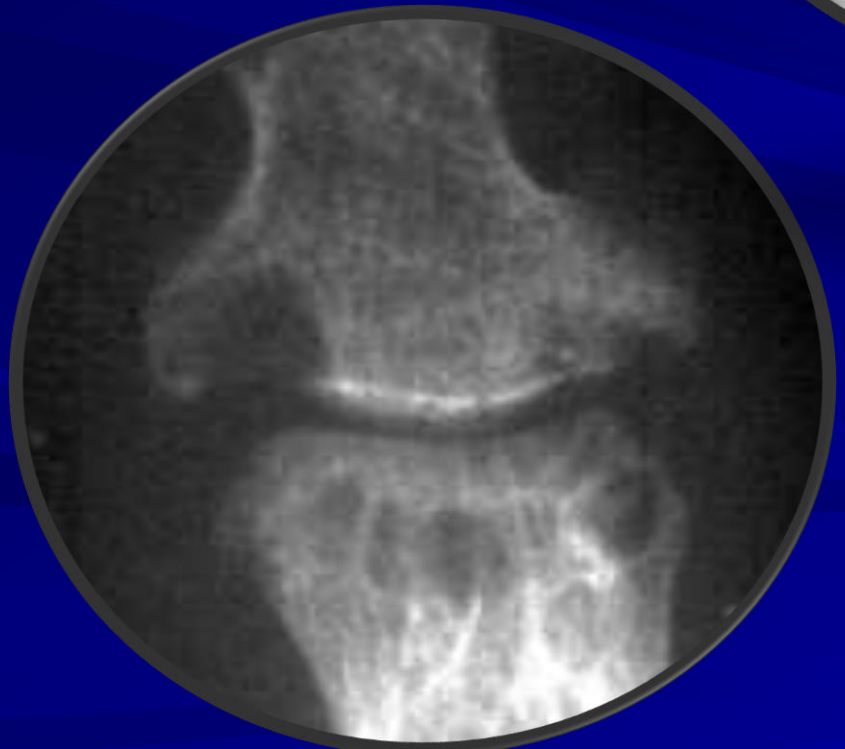


GOUT

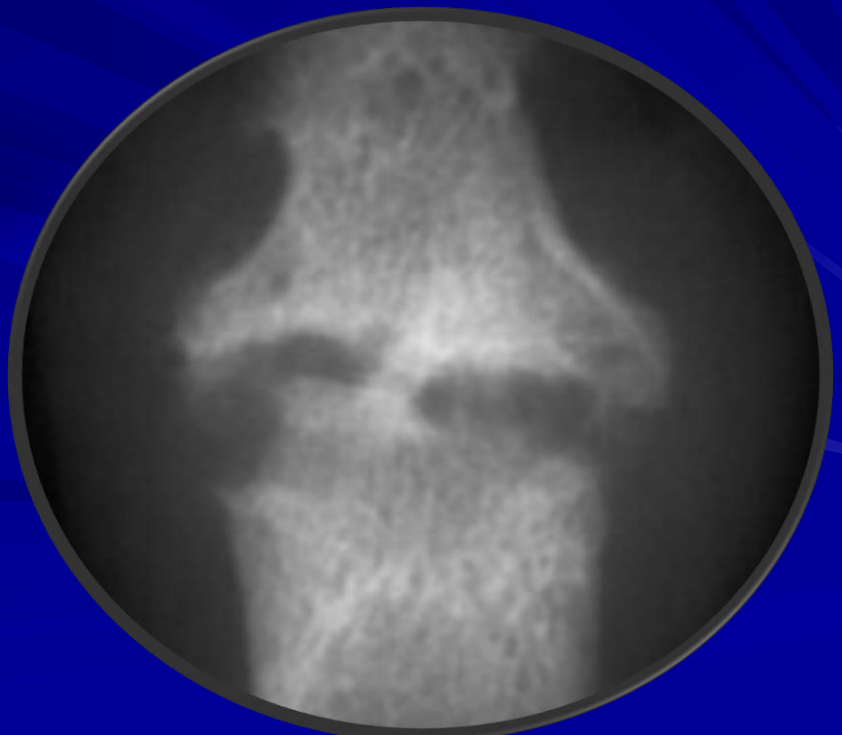


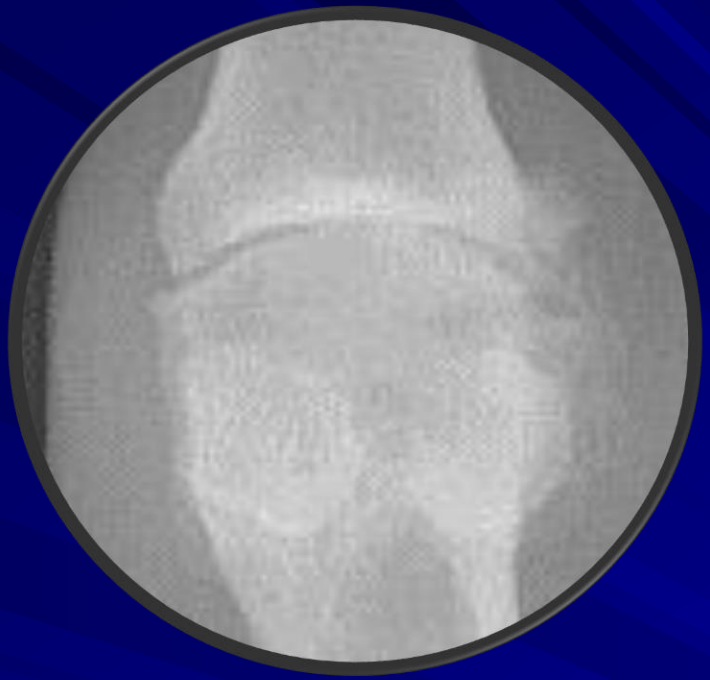
State the diagnosis for each of the following X-rays.

RA

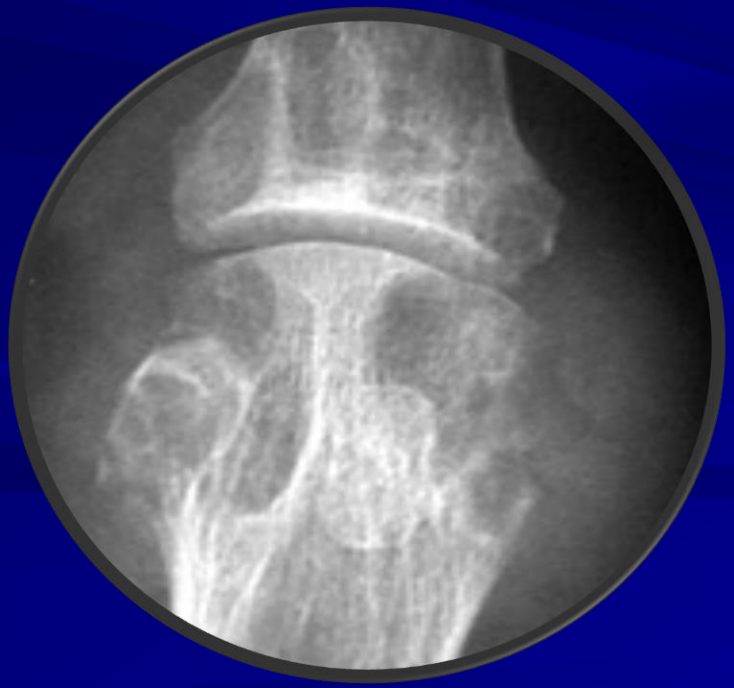


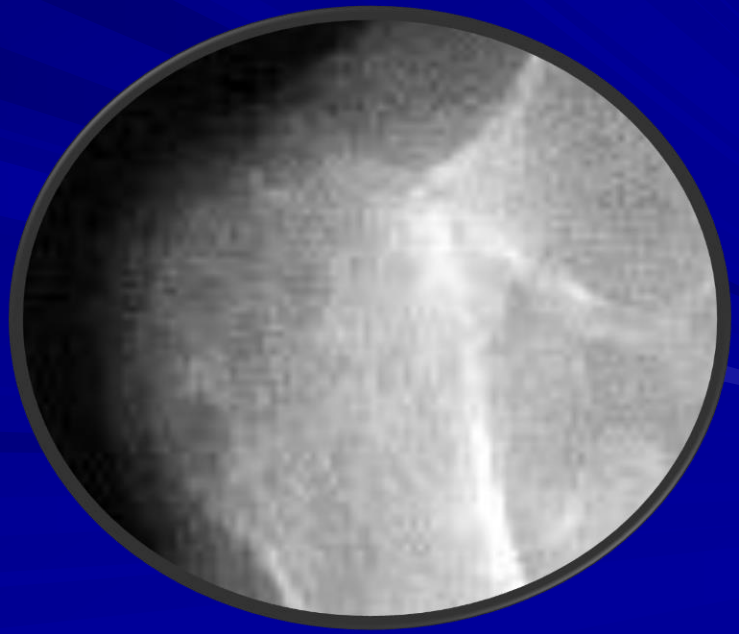
PA



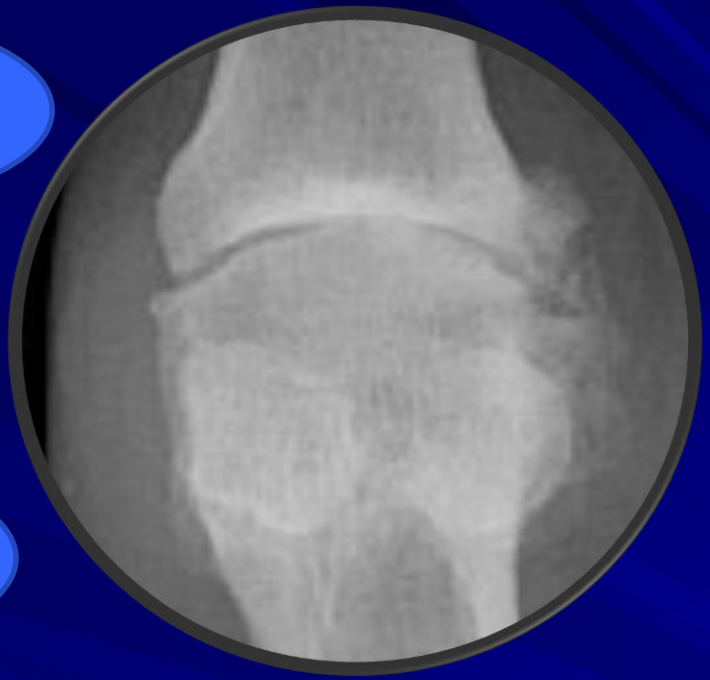


State the diagnosis for each of the following X-rays.



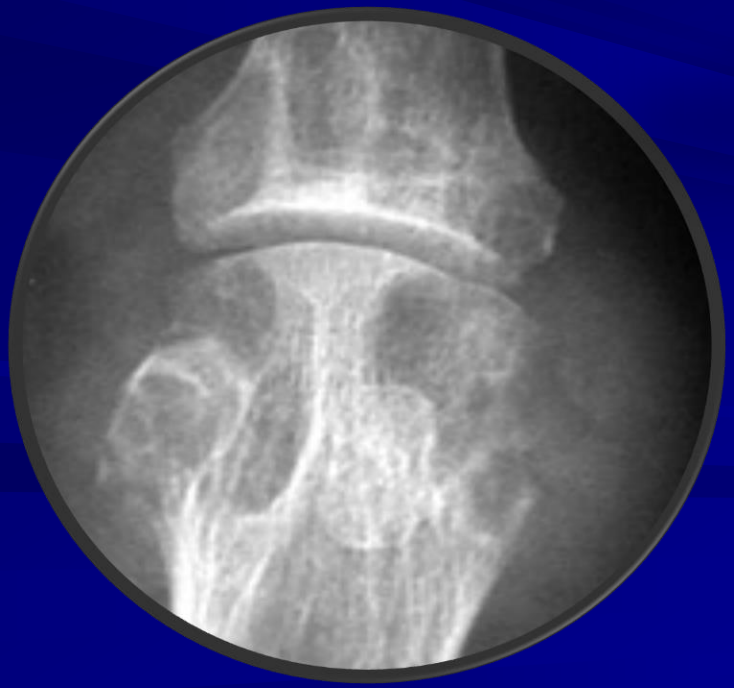


DJD

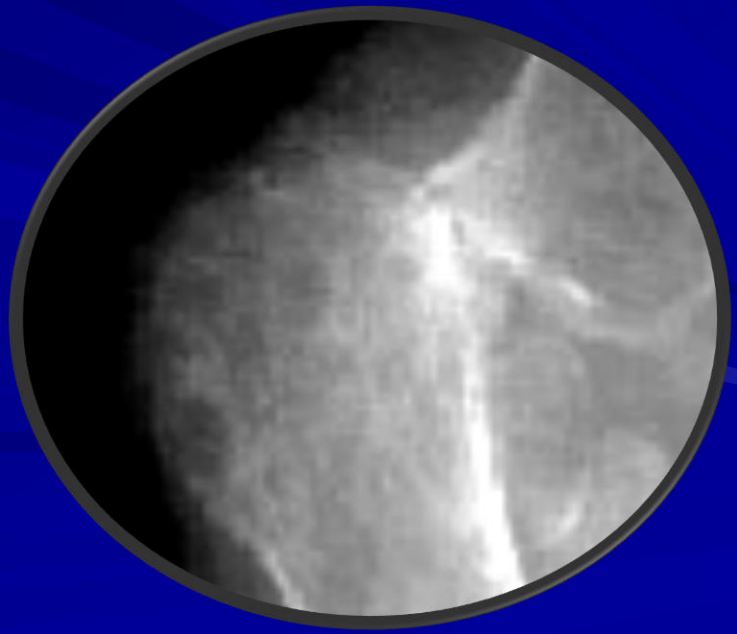


State the diagnosis for each of the following X-rays.

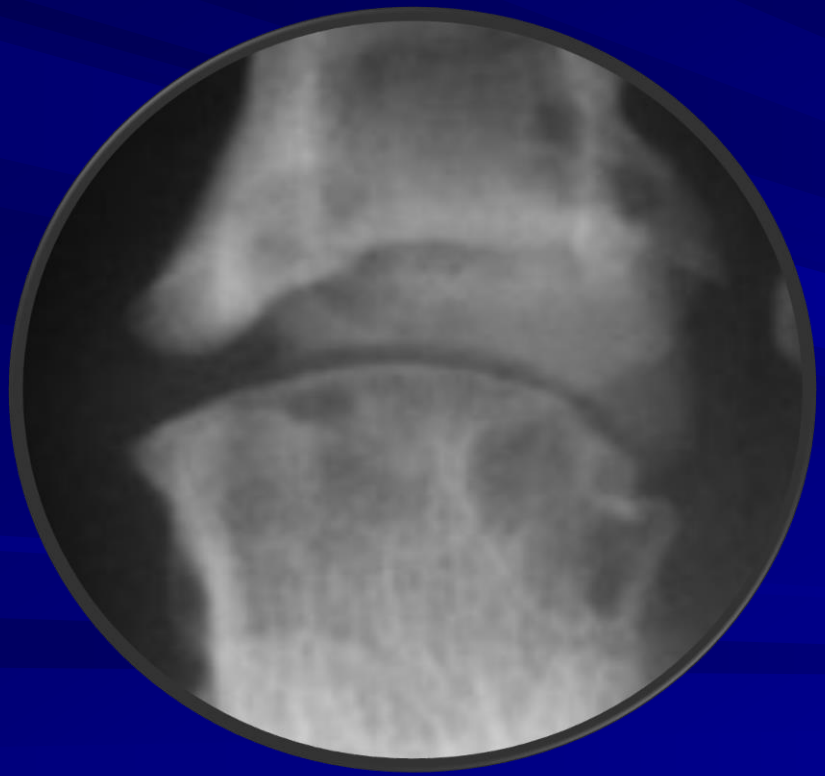
GOUT

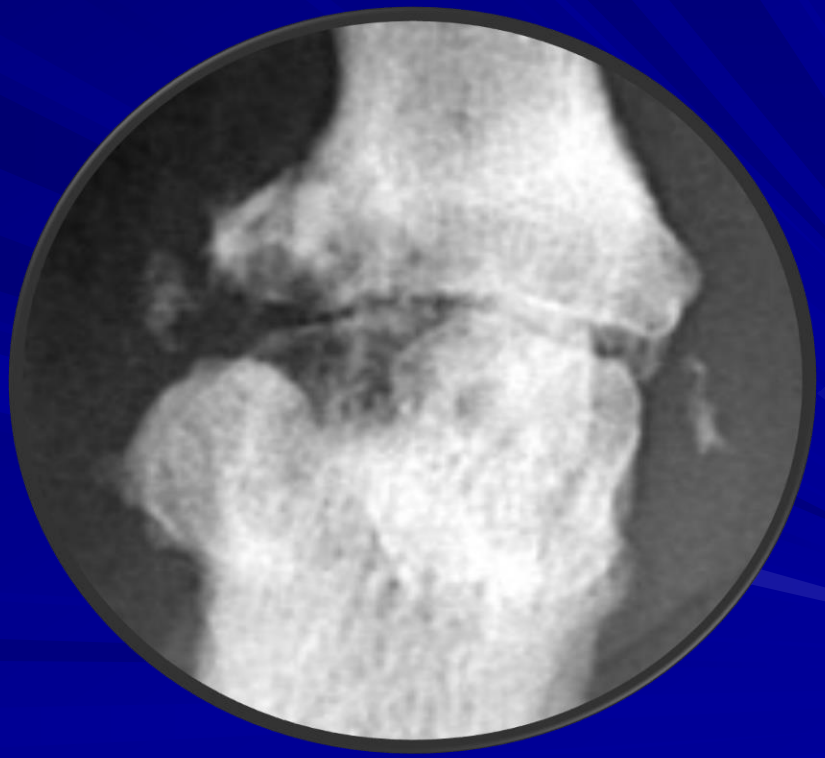


RA



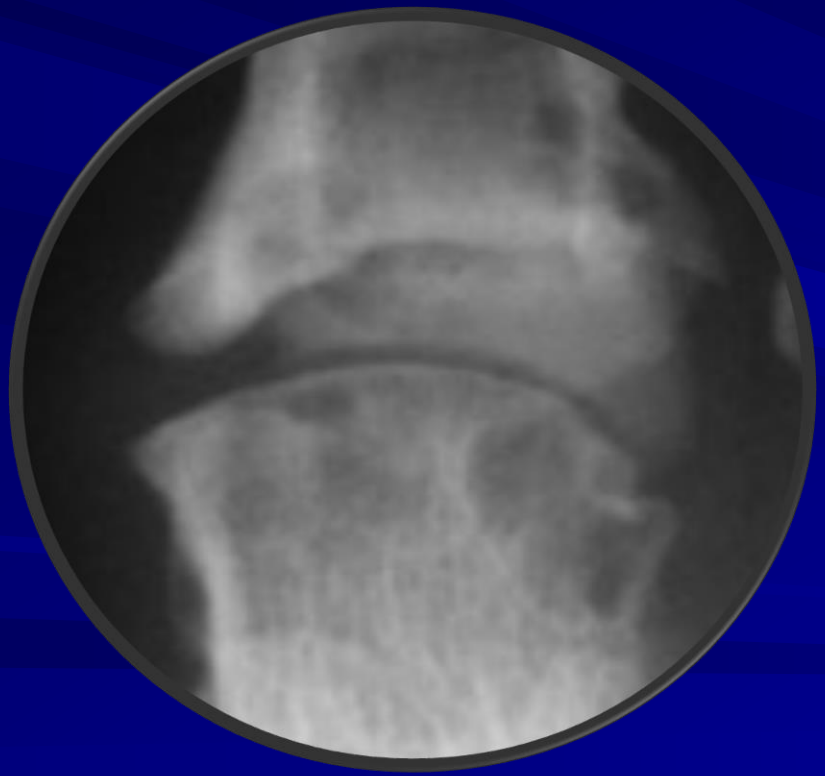
What do you think is going on with these two different radiographs?



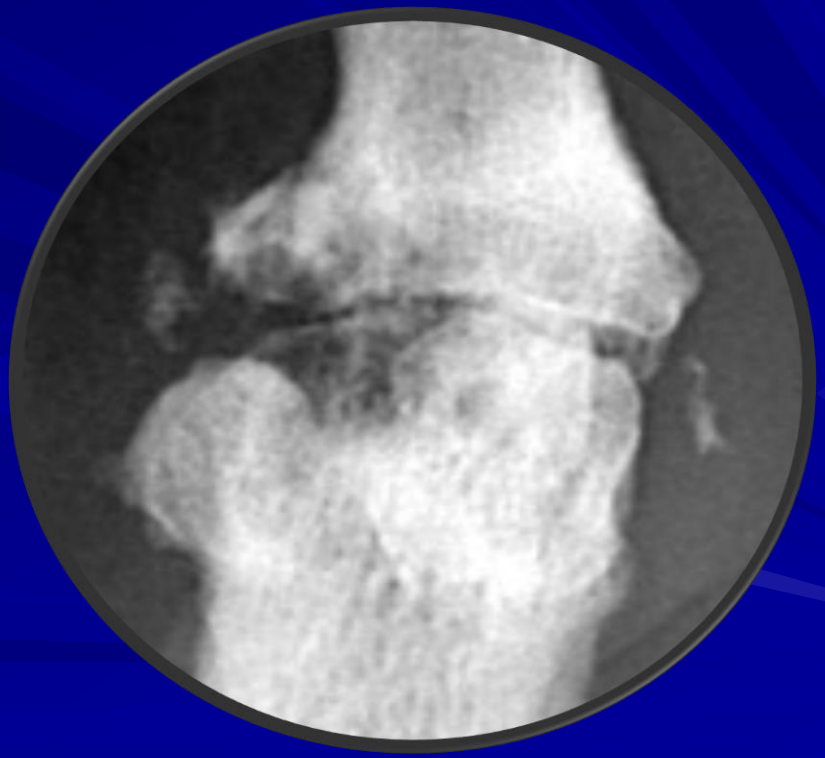


What do you think is going on with these two different radiographs?

Detritic Synovitis from Silastic Hemi-Implant



Septic Arthritis



Of the arthritides that we have discussed thus far, which one results in the most “bone production mineralization”?

Osteoarthritis (DJD)

Which of the arthritides that we have discussed results in the least “bone production/mineralization” ?

Rheumatoid Arthritis

Match the term with its corresponding arthritide(s):

1) Heberden's Node
2) Ivory Phalanx
3) Gull Wing Sign
4) Romanus Lesion
5) Swan-neck Deformity
6) Fluffy Heel Spur
7) Martel's Sign

A) PA
B) RA
C) OA
D) Erosive OA
E) Gout
F) AS
G) Reiter's Dz

1C,D

2A

3D

4F

5B

6G

7E

Neuropathic Arthritide

Neuropathic Joint Disease

Neuropathic Joint Disease

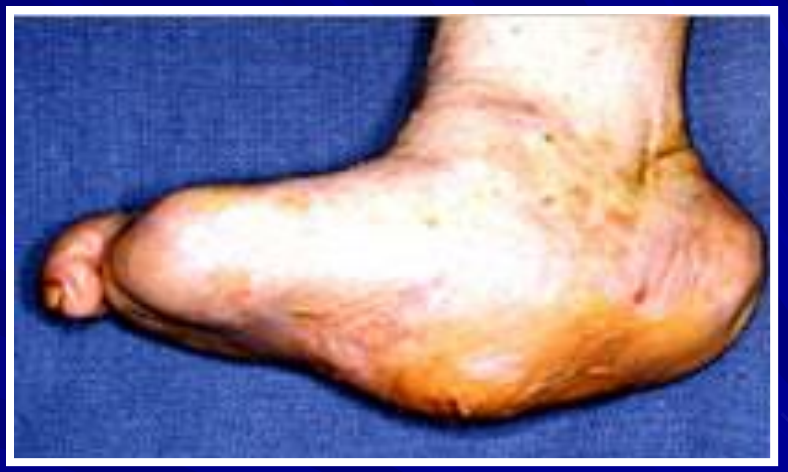
- Destructive articular disease process associated with conditions that cause sensory & autonomic neuropathy.
- Pathophysiology:
  - Neurotraumatic Theory (Volkman & Virchow 1886): Bony destruction is attributed to loss of pain sensation & proprioception combined with repetitive, mechanical minor trauma to the foot.
  - Neurovascular Theory: Suggests that joint destruction is secondary to an autonomically stimulated vascular reflex that causes hyperemia & periarticular osteopenia.
  - Pro-inflammatory Cytokines Theory: Activation of cytokines like tumor necrosis factor alpha & interleukin-1B result in increased expression of RANKL which causes osteoclast maturation and ultimately local osteolysis.

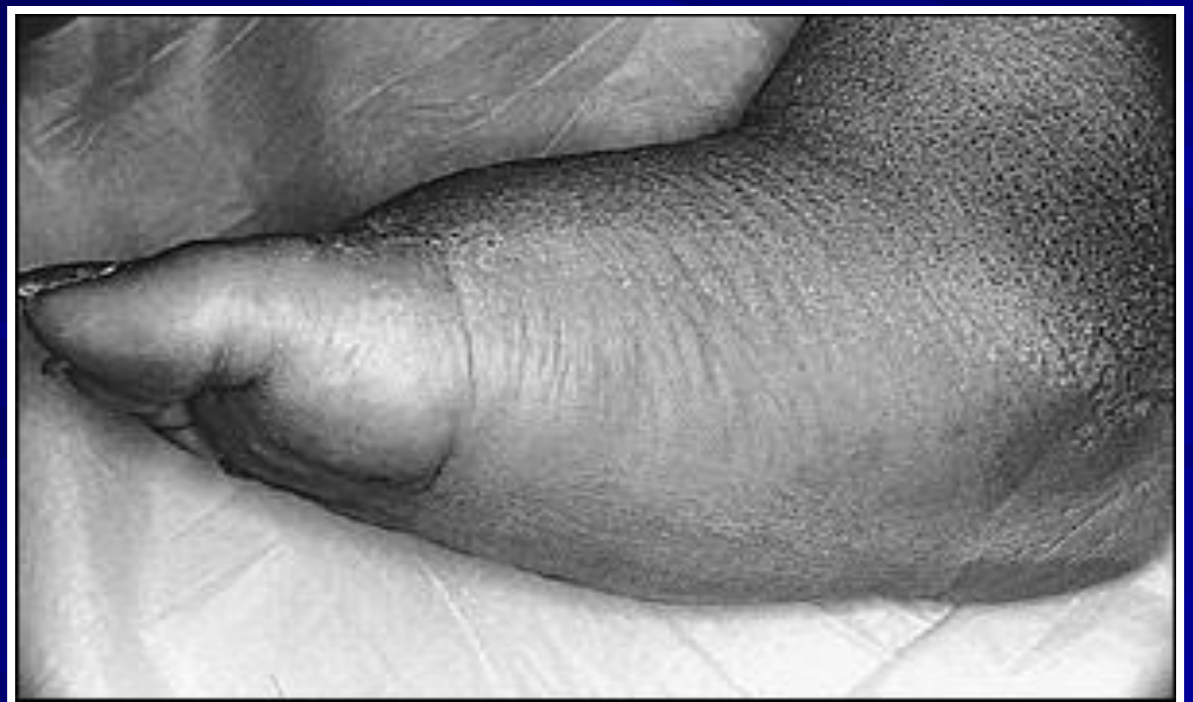
Neuropathic Joint Disease

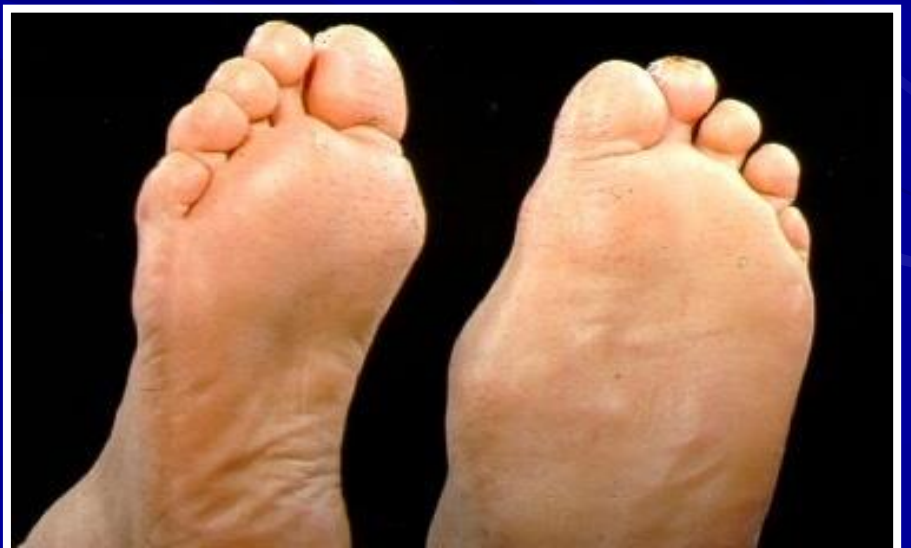
- Pathophysiology (Biochemical):
  - Physiologic balance between pro- & anti- inflammatory cytokines is lost
  - Increase in proinflammatory cytokines like TNF, IL-1B, IL-6
  - Prolonged inflammatory response triggers the RANKL pathway
  - RANKL pathways increase osteoclast production/activity which induces bone lysis & destruction

Neuropathic Joint Disease

- Lab findings:
  - Negative except for etiologic factor







Neuropathic Joint Disease

- Clinical features:
  - Vary widely depending of the stage of the disease
    - Mild swelling/no deformity to significant swelling/moderate deformity
  - Signs of inflammation such as:
    - Profound unilateral swelling
    - Increase in local skin temperature (3-7 degrees greater that the non-affected foot's skin)
    - Erythema
    - Joint effusion
  - Joint instability & crepitation (Loose bag of bones)
  - Loss of protective sensation, DTRs, ataxia
  - Pain (severity significantly less that what would be expected based on the clinical/radiographic findings)

Neuropathic Joint Disease

Causes of Neurotrophic Arthropathy

CONGENITAL
- Congenital indifference to pain
- Dysautonomia
- Spina bifida vera (meningocele, etc.)

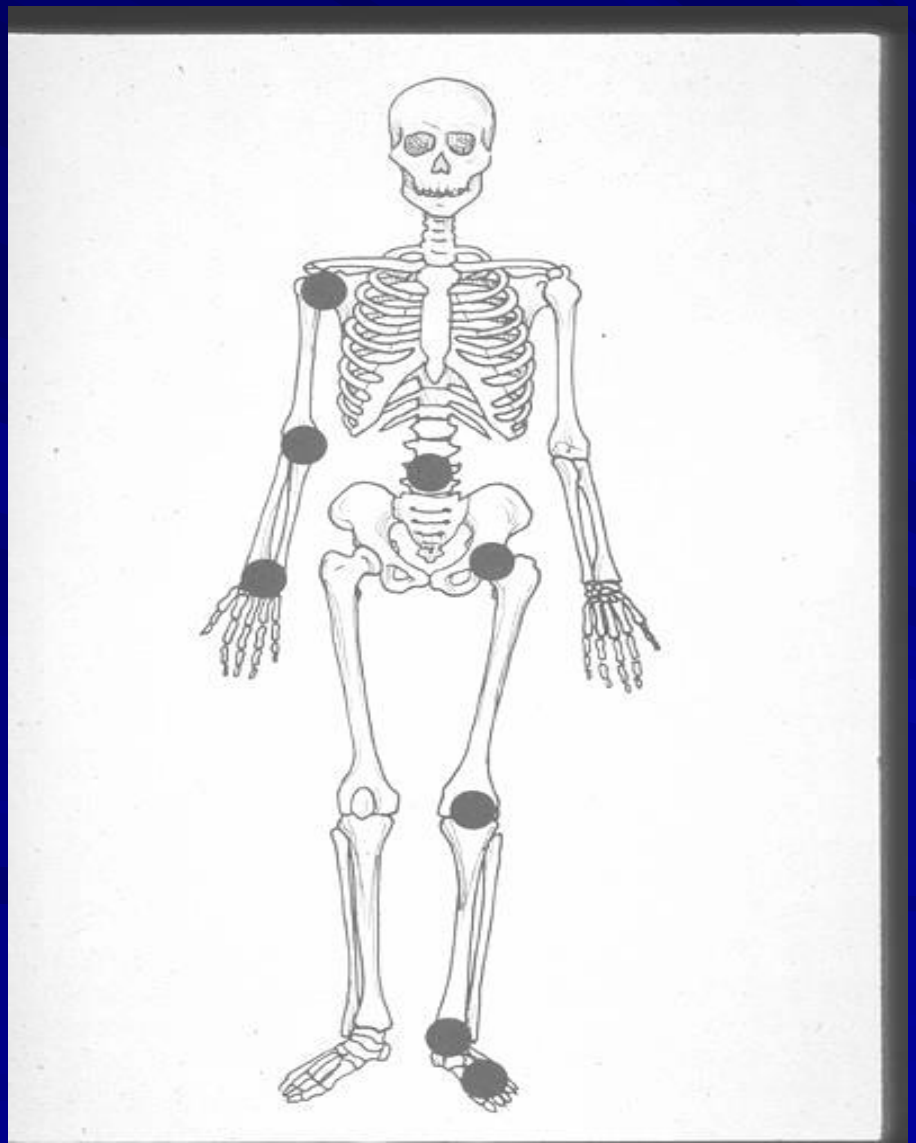
ACQUIRED
- Alcoholism
- Amyloidosis
- Charcot-Marie-Tooth disease
- Diabetes mellitus
- Leprosy, yaws
- Multiple sclerosis
- Neurosyphilis
- Syringomyelia
- Trauma
- Tumor

IATROGENIC
- Indomethacin
- Phenylbutazone
- Steroids

Charcot Arthropathy:

- Diabetes is the most common cause
- 1 out of 700 patients with diabetes will develop Charcot joint

Neuropathic Joint Disease

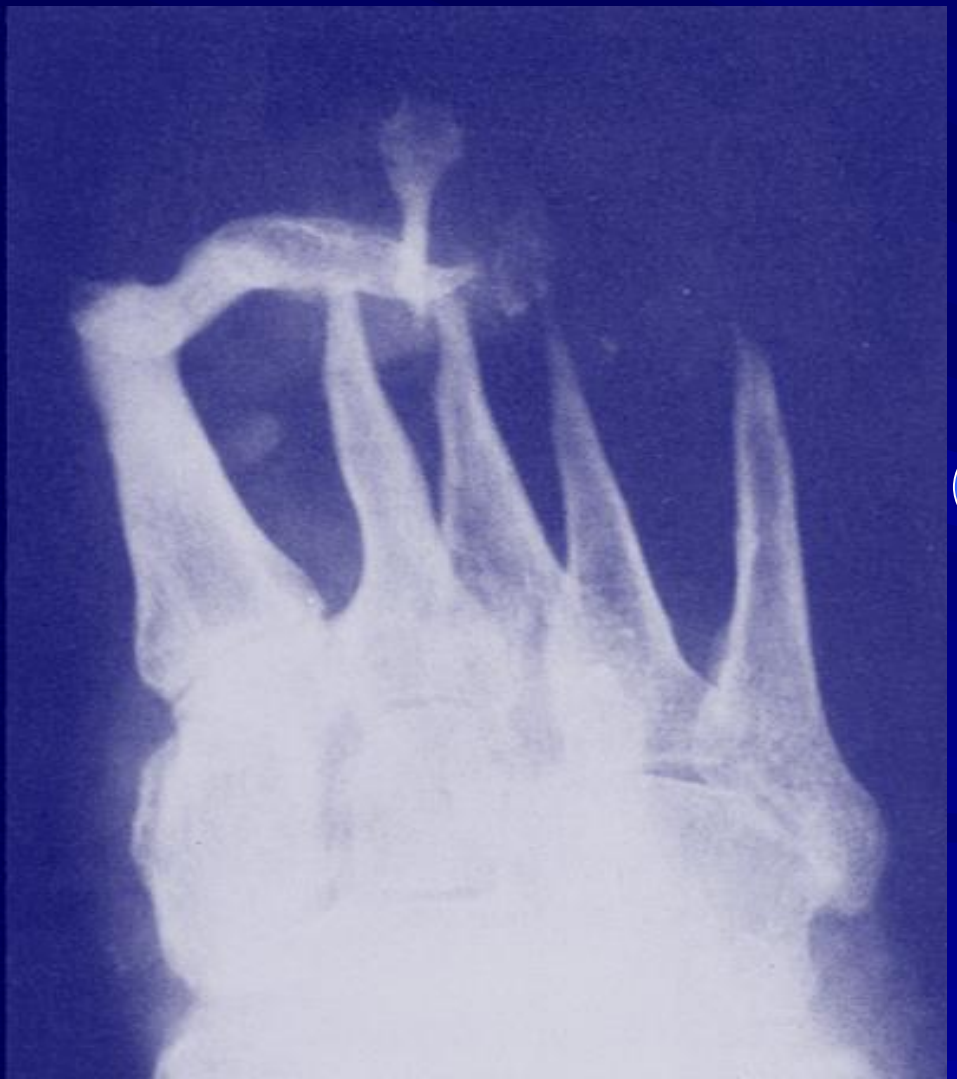


Neuropathic Joint Disease

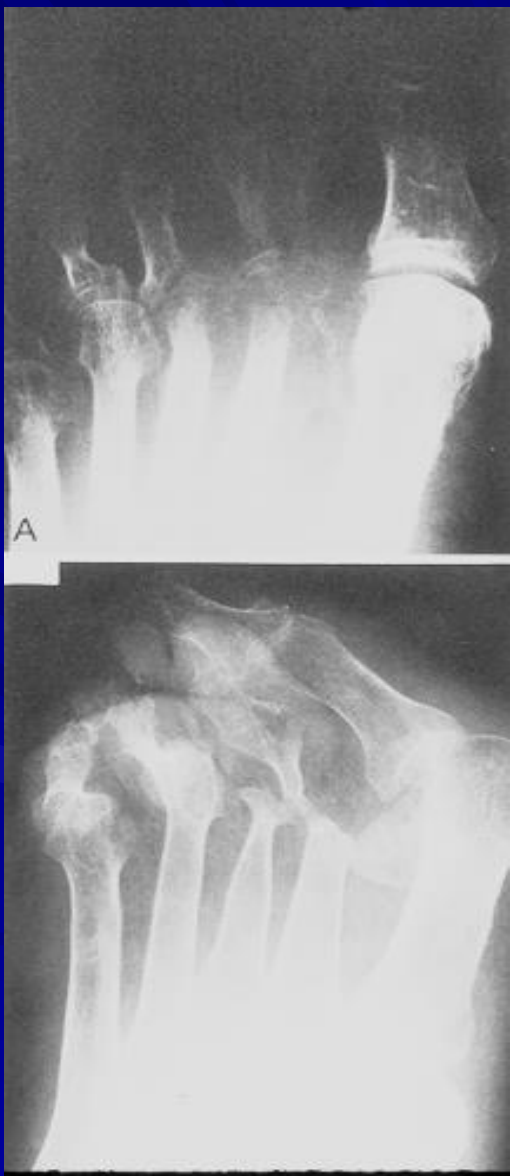
- Distribution:
  - Hypotrophic form (Atrophic) – non-weightbearing joints (forefoot)
  - Hypertrophic form – weightbearing joints (Ankle, rearfoot, midfoot)

- Radiographic features:
  - Atrophic form
    - Usually localized to the forefoot (metatarsal heads/shafts)
    - Bone resorption
      - Osteolysis is more pronounced in atrophic charcot than with psoriatic arthritis
    - "Licked candy stick"
    - Diabetic osteolysis

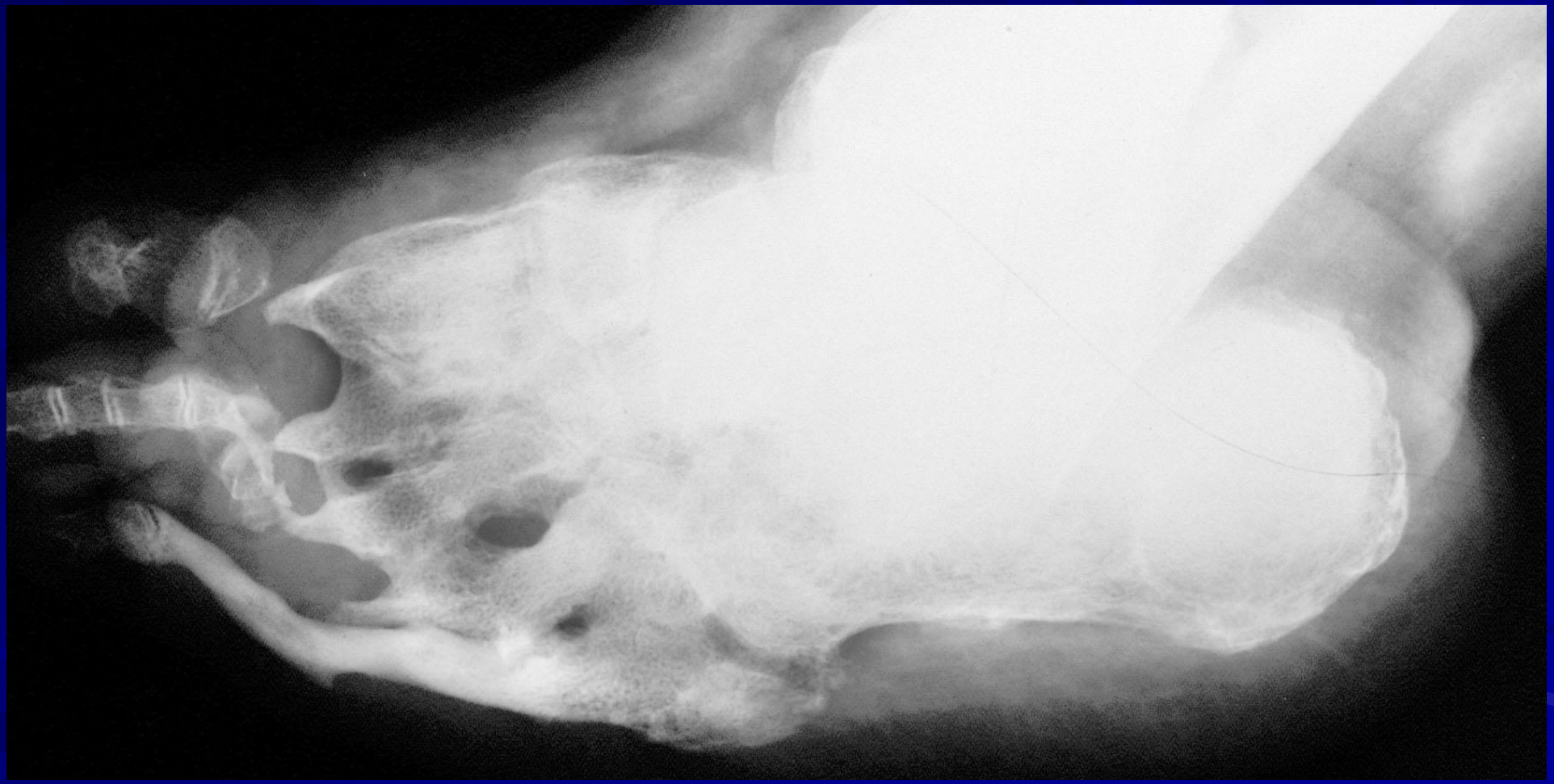
Neuropathic Joint Disease



"Licked Candy Stick" Appearance (Atrophic Form)



Neuropathic Joint Disease

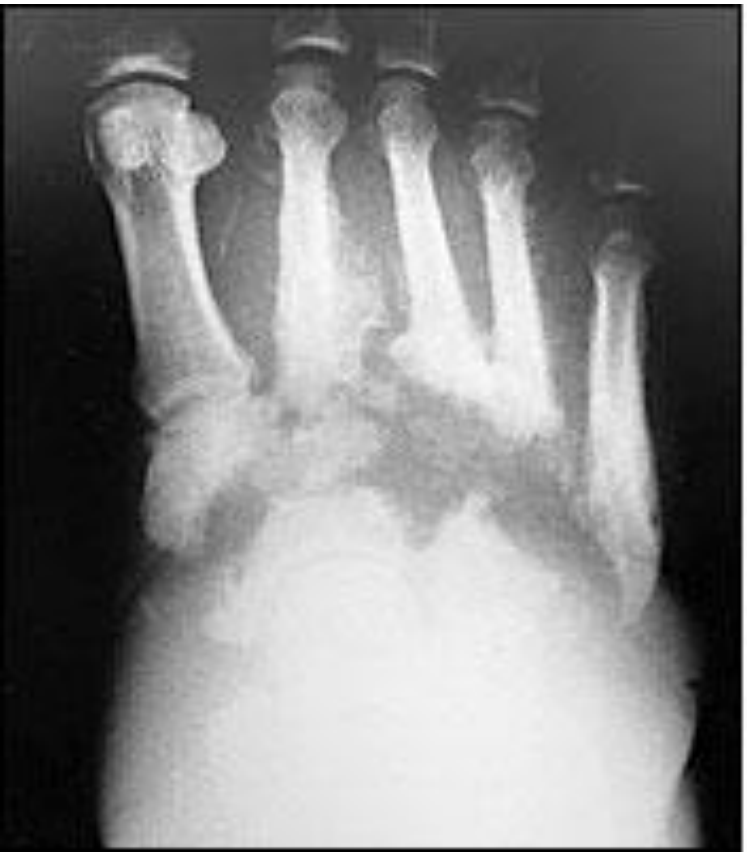


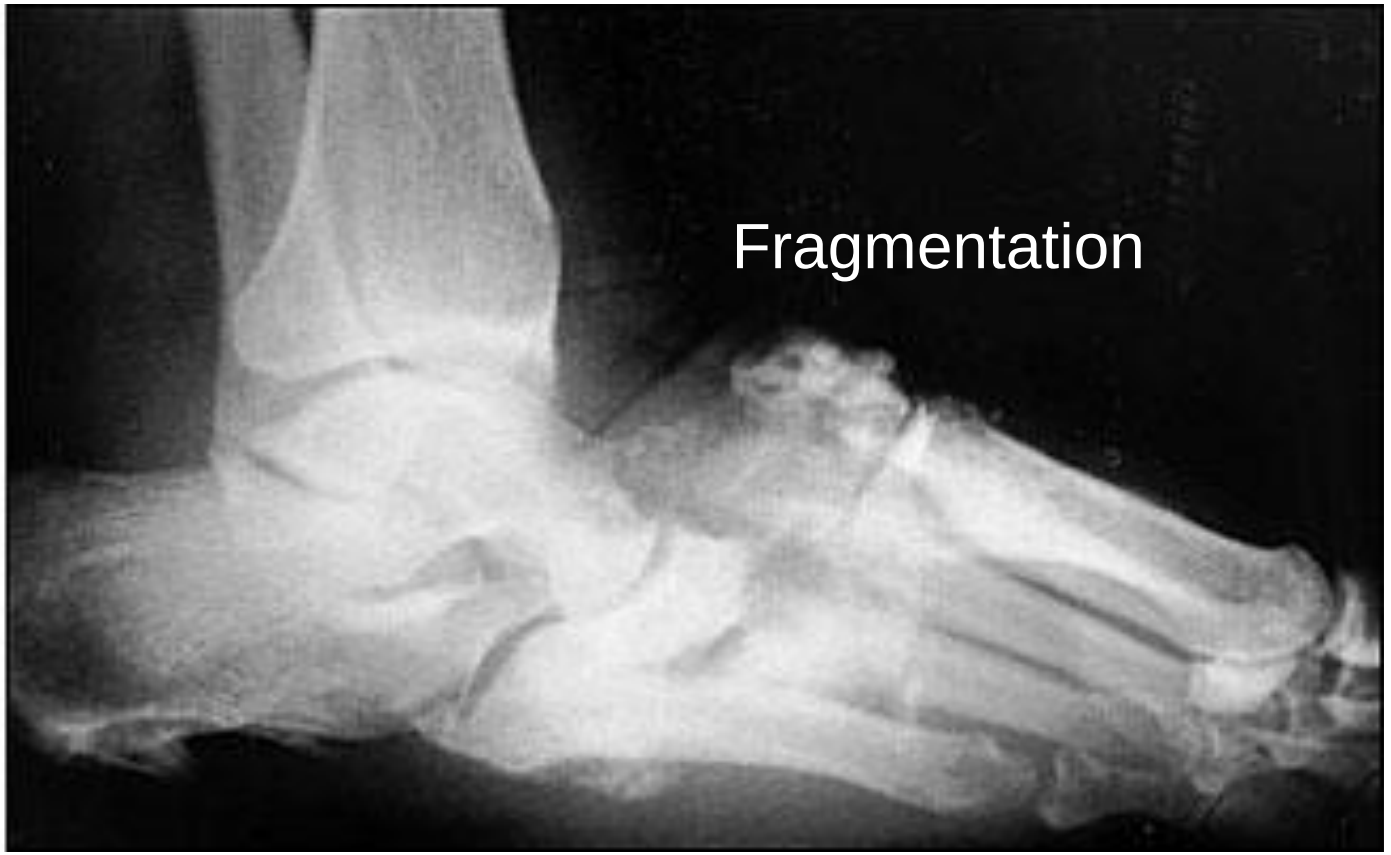
Osteolysis of the distal metatarsals and phalanges with tapering results in a pencil-like appearance in the late stage of diabetic neuropathy.

Neuropathic Joint Disease

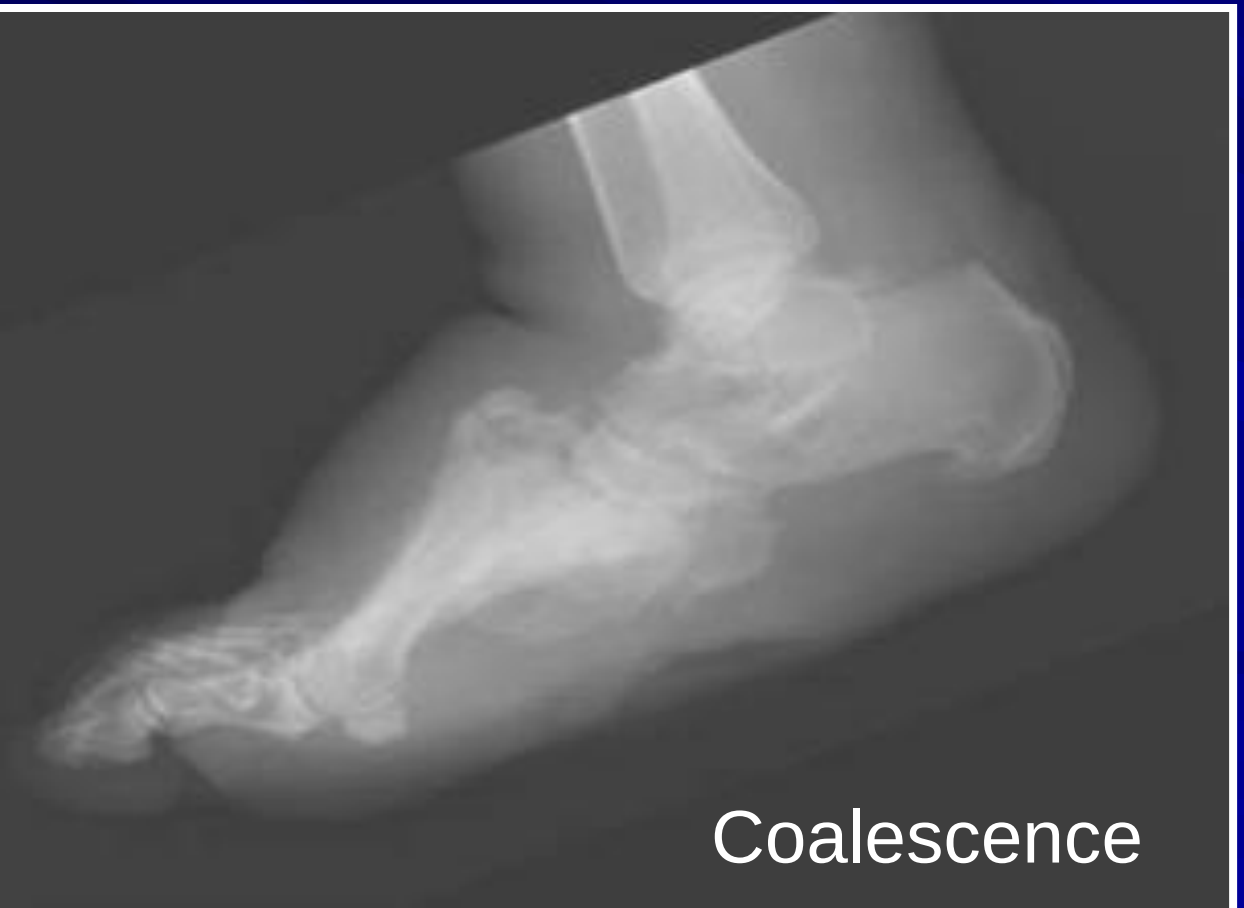
- **Eichenholtz Classification** for **Hypertrophic** Charcot:
  - 3 stages
    - Destruction/Fragmentation (Acute Charcot)- characterized by inflammation & joint effusion. Capsular/ligamentous structures become lax and subsequently periarticular fracture/erosions & joint dislocation occurs.
    - Coalescence (Subacute Charcot)- resorption of fine bone debris & coalescence of larger boney fragments. Bone becomes sclerotic and joint stability starts to increase.
    - Reconstruction (Chronic Charcot)- associated with re-stabilization of the foot with continued ankylosis, remodeling/rounding of bone, formation of pseudarthroses, & reduction in sclerosis.

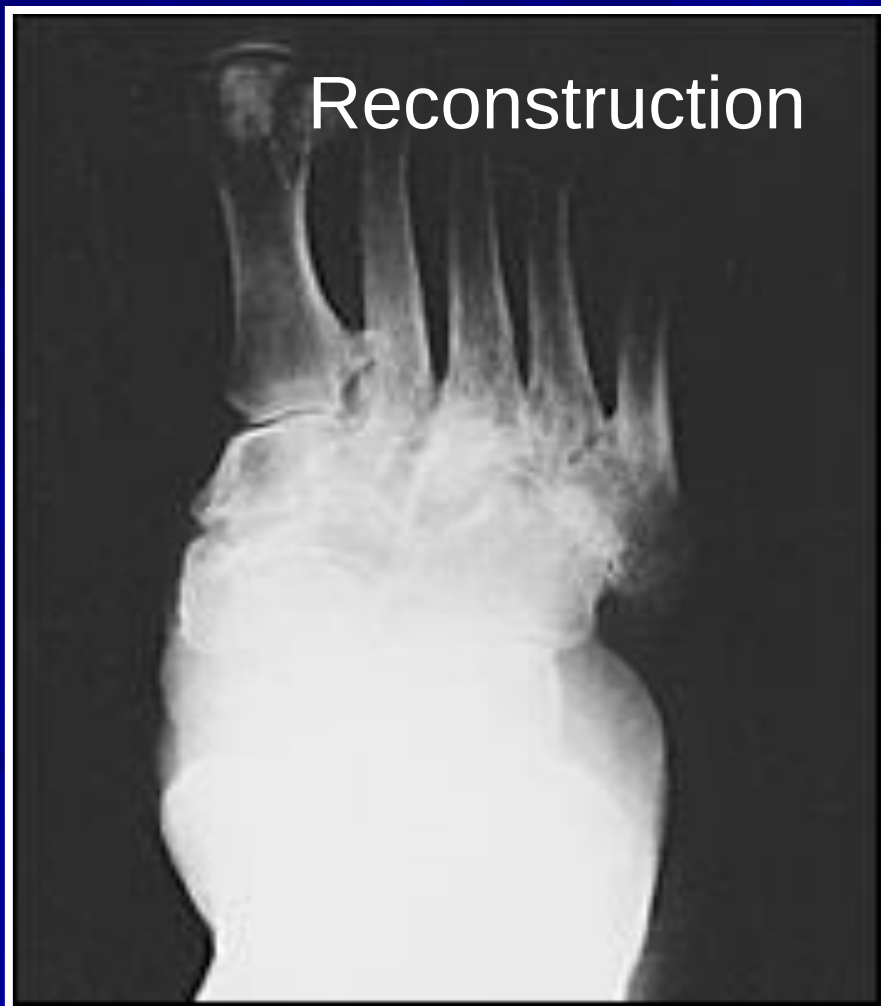
Neuropathic Joint Disease





Neuropathic Joint Disease

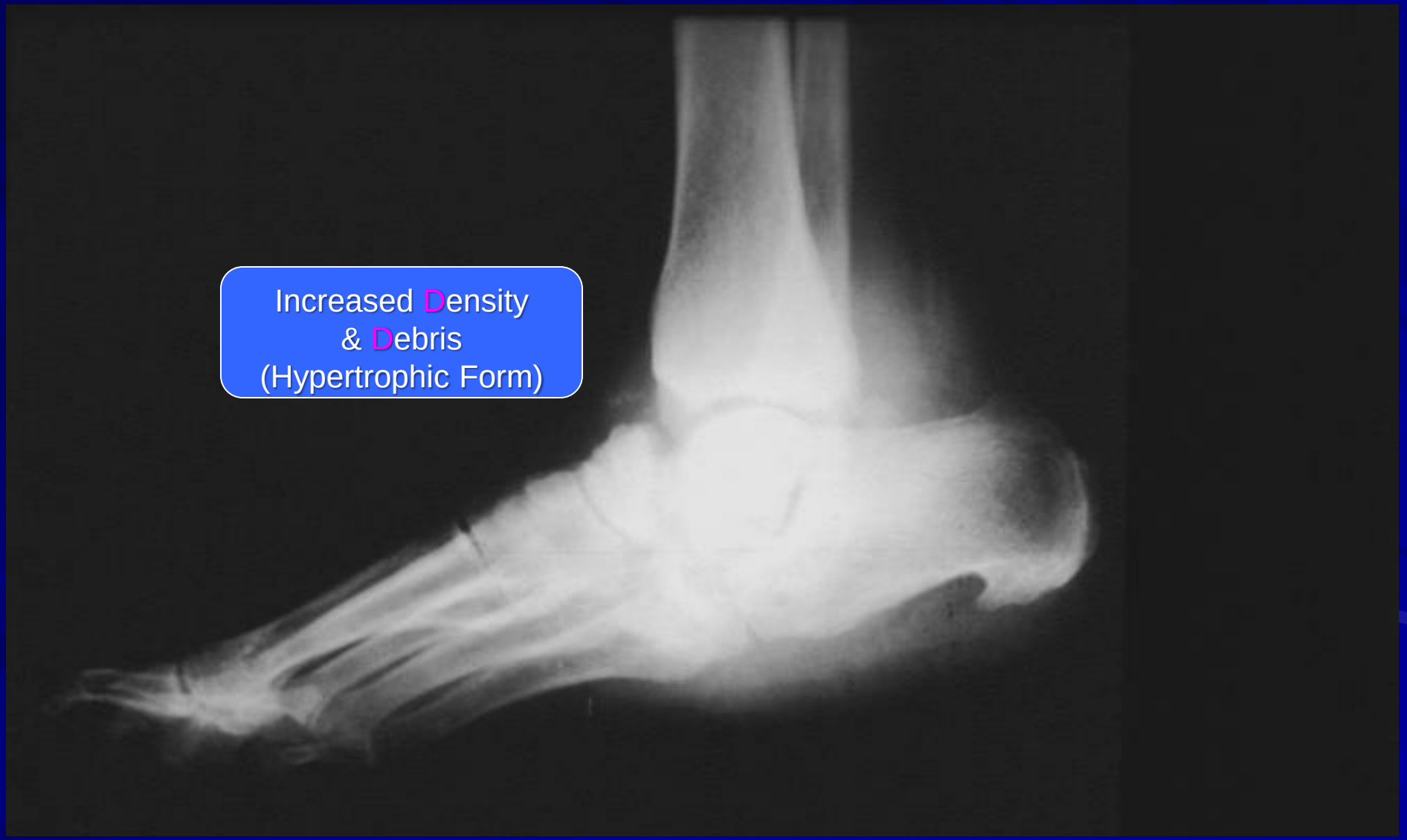




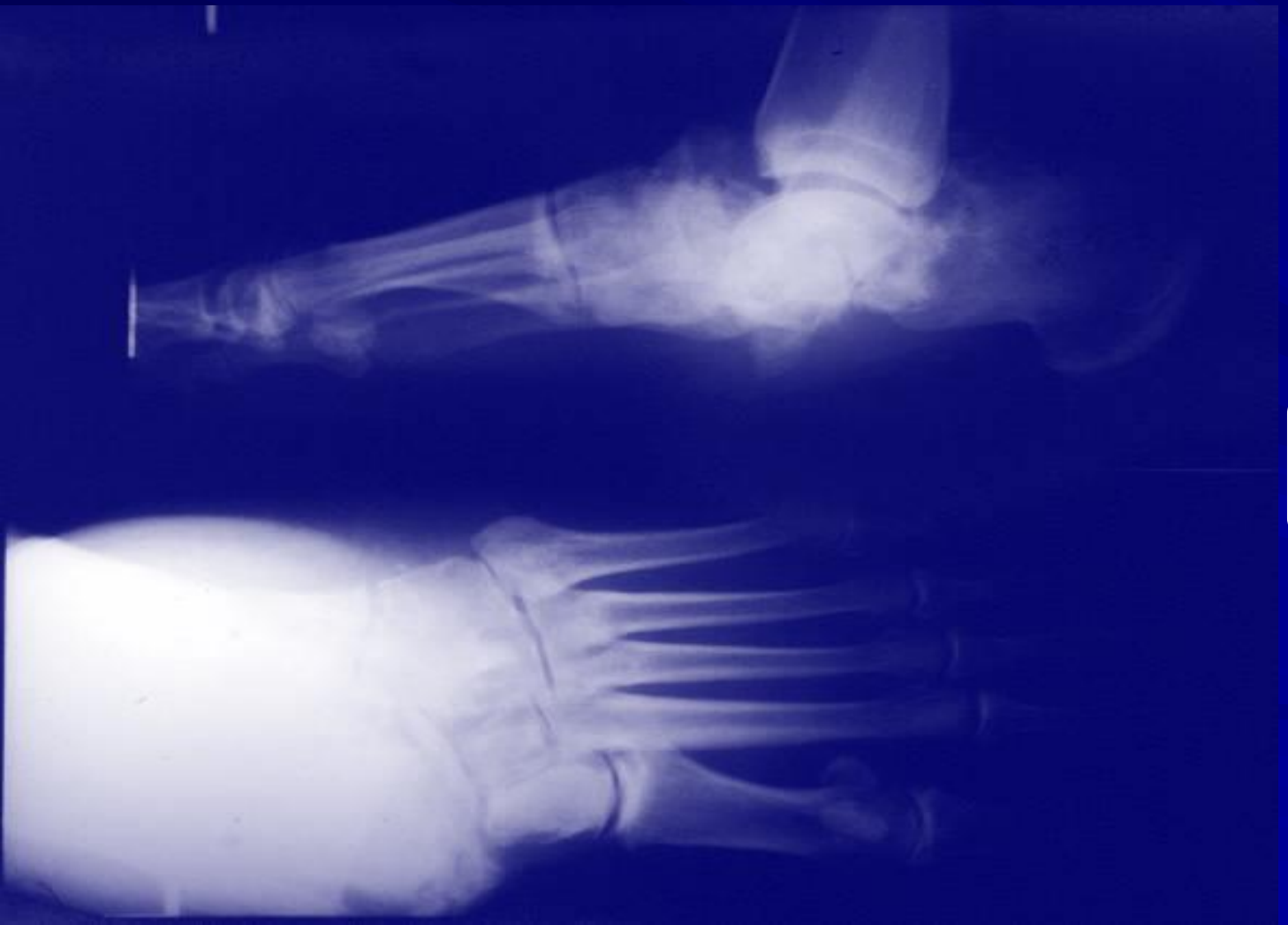
Neuropathic Joint Disease

- Radiographic features:
  - Hypertrophic form – 6 Ds (3 “De-” & 3 “Dis-”)
    - **D**estruction
    - **D**ensity increases
    - **D**ebris production
    - **D**islocation
    - **D**isorganization
    - **D**istension of joint

Neuropathic Joint Disease

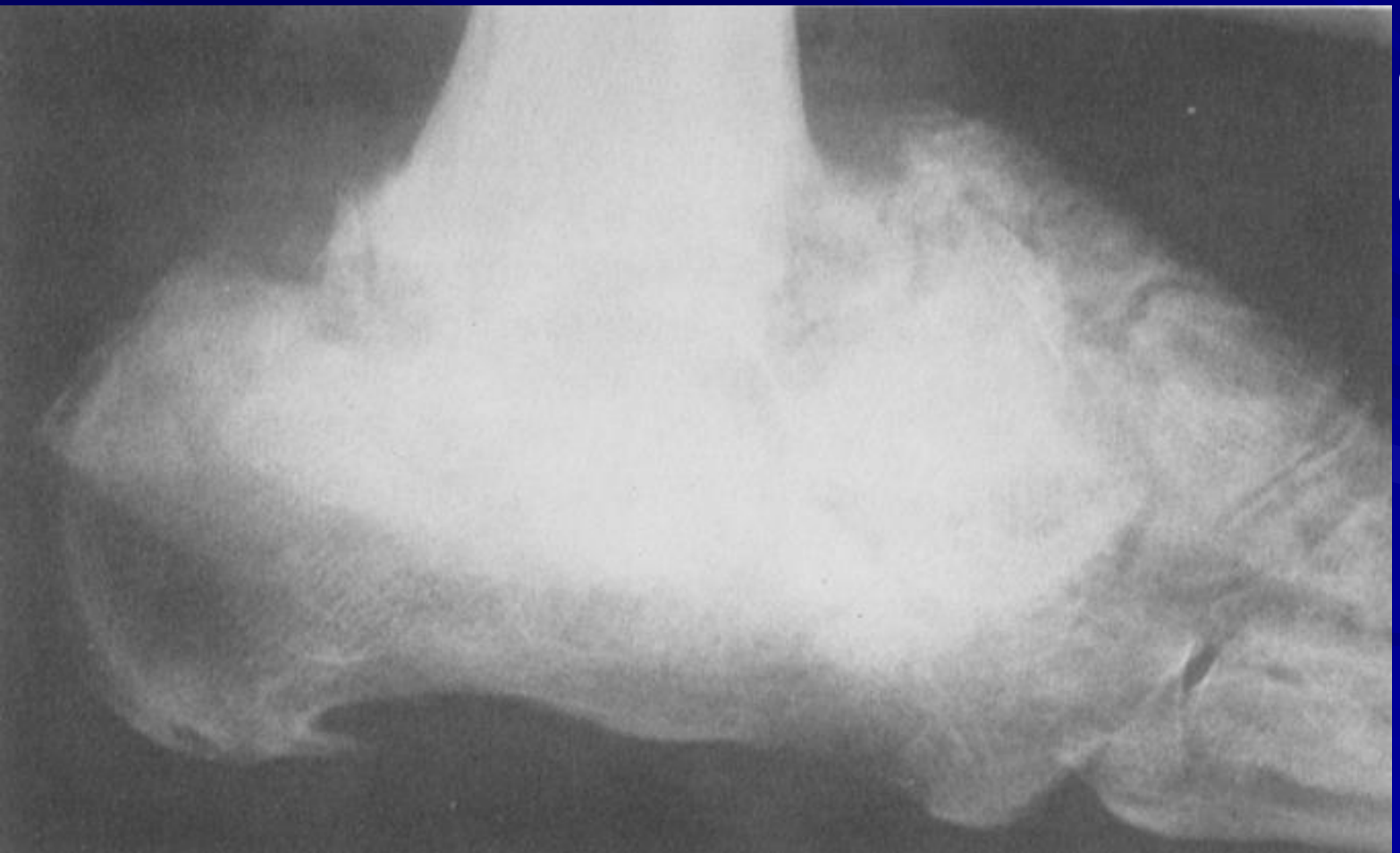


Neuropathic Joint Disease



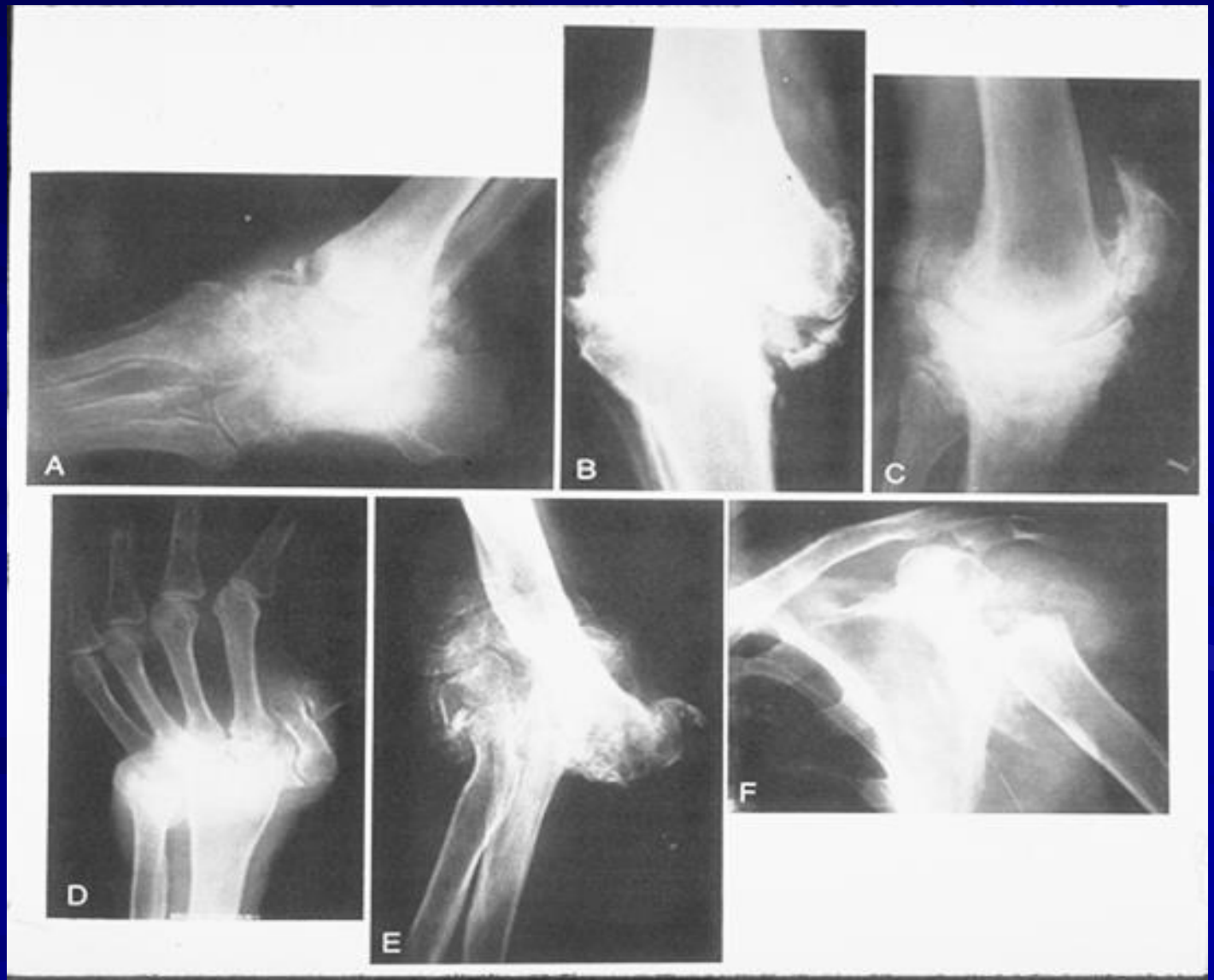
Fragmentation & Destruction
With Talar Dislocation
(Hypertrophic Form)

Neuropathic Joint Disease



Disorganization of the ankle & subtalar joints (Hypertrophic Form)

Neuropathic Joint Disease



Miscellaneous Arthritides

Diffuse Idiopathic Skeletal Hyperostosis

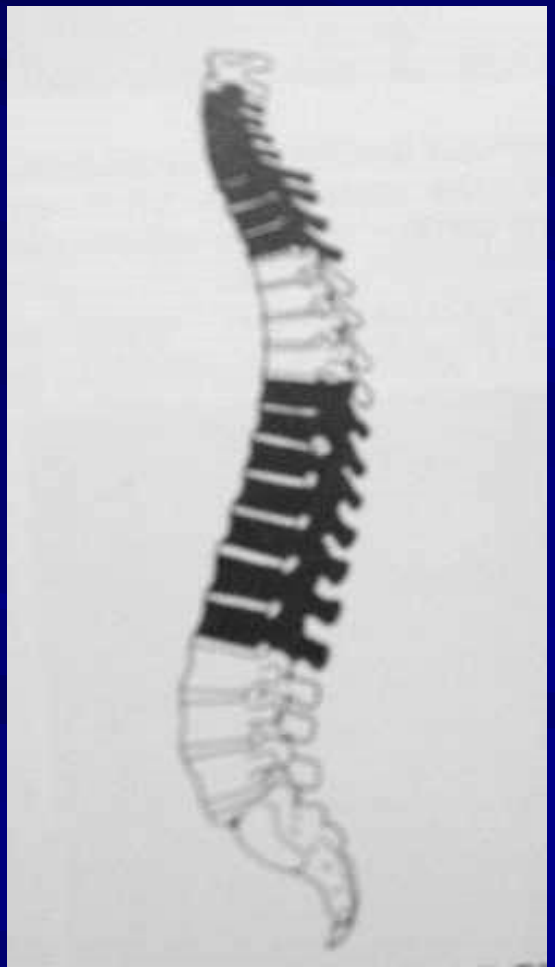
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

- Overview:
  - Ligamentous/Tendinous ossification and calcification at their respective attachment sites to bone (entheses)
  - Also known as a diffuse variant of Forestier's Disease (OALL: ossification of the anterior longitudinal ligament) or senile ankylosing spondylitis
  - Spinal & extra-spinal articulations
  - 12% middle aged and elderly

Diffuse Idiopathic Skeletal Hyperostosis (DISH)

- Clinical features:
  - Age: >50 yoa (AS 15-35 y/o)
  - Gender: males predominant (like AS)
  - 20% have concurrent diabetes; dyslipidemia & hyperuricemia also commonly occur simultaneously
  - Obesity (high BMI or large waist circumference) is a common feature of many patients with DISH
  - May exhibit postural abnormalities & limitation in spinal mobility similar to AS
  - Dysphagia is common because calcification of the anterior longitudinal ligament & extraspinal ligaments, may impinge on esophageal space

Diffuse Idiopathic Skeletal Hyperostosis (DISH)



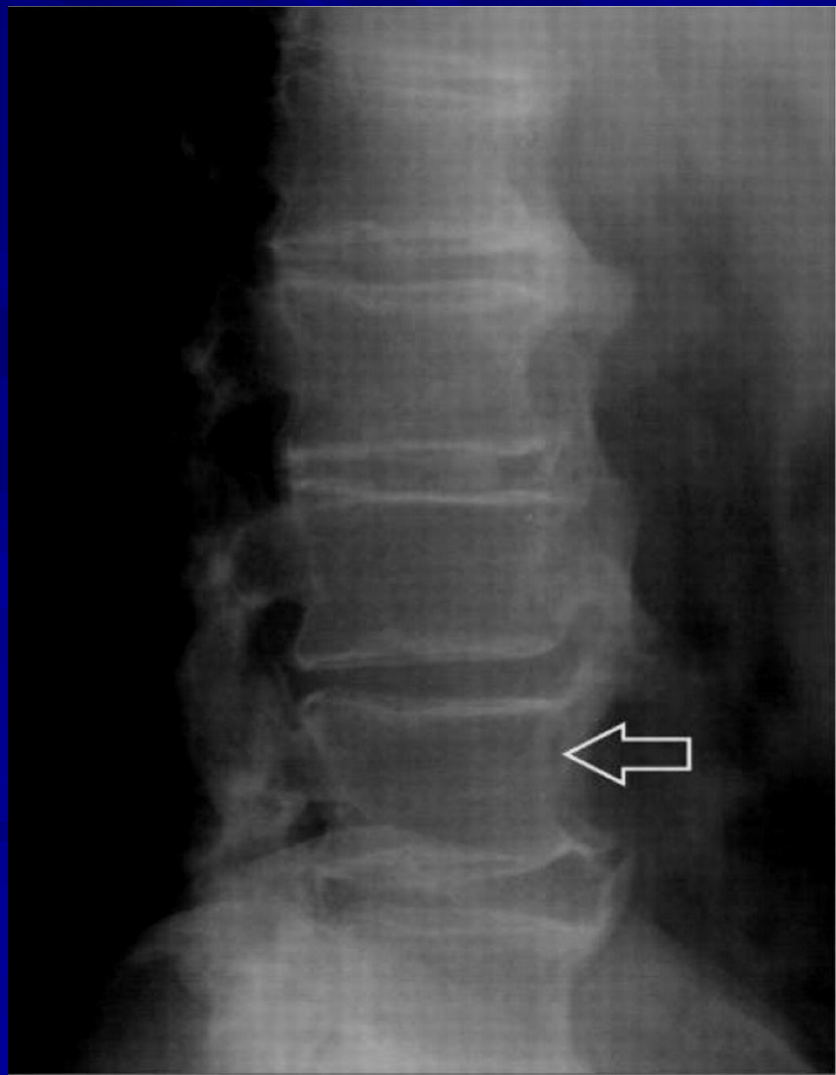
- Typically affects the lower thoracic spine, but the cervical & lumbar spine might also be involved
- Left side of the spine is usually spared
  - Due to pulsating aorta
  - Finding is supported by the fact that individuals with DISH & situs inversus (organs on reverse side of body) have sparing of the right side of the spine b/c the aorta is located on the right side

Diffuse Idiopathic Skeletal Hyperostosis (DISH)

- Lab findings:
  - HLA-B8 antigen 40% (AS positive for HLA-B27)
- Distribution:
  - Cervical, thoracic and lumbar spine
- Radiographic features:
  - Spinal
    - Flowing hyperostoses over 4 contiguous vertebral bodies
      - Anterior longitudinal ligament (PLL in 50% - cervical)
    - Preservation of normal disk height with absence of vertebral body squaring/shiny corner sign (differentiates DISH from AS)
    - Absence of bony ankylosis of facet joints (differentiates DISH from AS)
    - Absence of sacroiliac erosion, sclerosis, or bony fusion, although narrowing and sclerosis of facet joints are acceptable (differentiates DISH from AS)

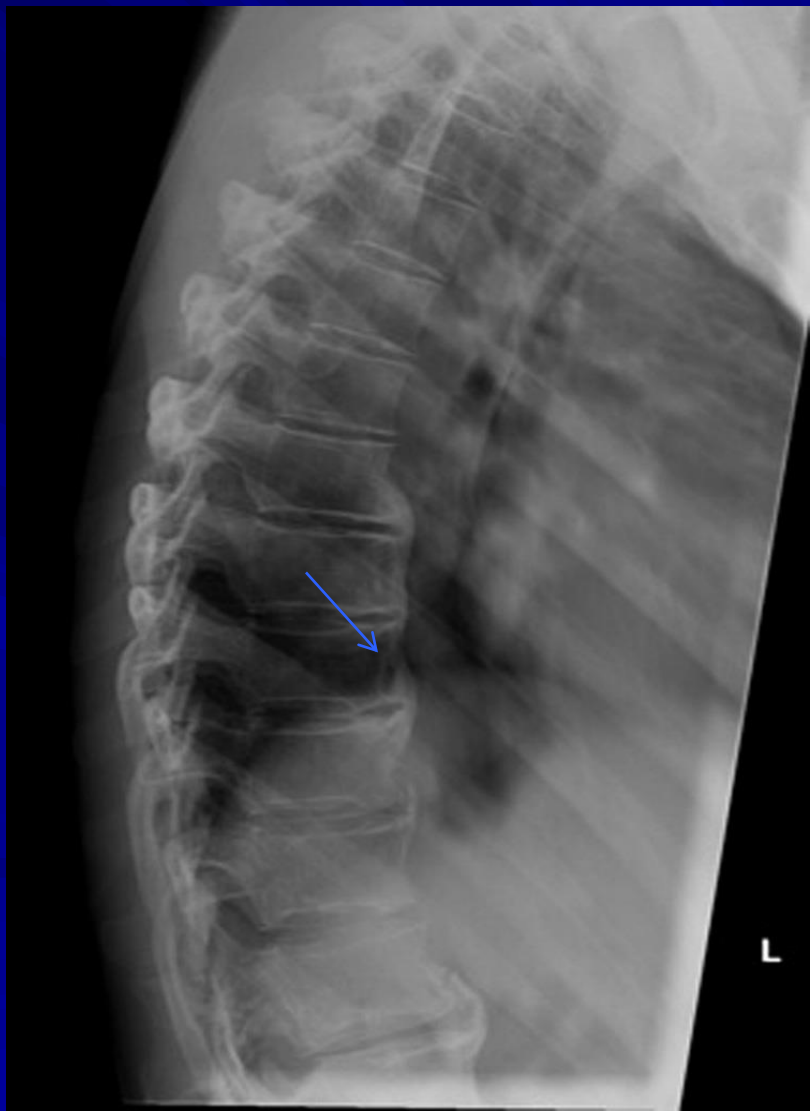
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

- DISH is characterized by ‘flowing’ ossification of the anterior longitudinal ligament.
- Ossification is separated from the anterior aspect of the vertebral body by a thin radiolucent line.
- Anterior ossifications often meet **without** fusion, thereby allowing continued motion of the spine.



Diffuse Idiopathic Skeletal Hyperostosis (DISH)

- Lateral radiograph of the thoracic spine shows flowing ossification of the anterior longitudinal ligament of several thoracic vertebral bodies. The disc spaces and vertebral body heights are normal.

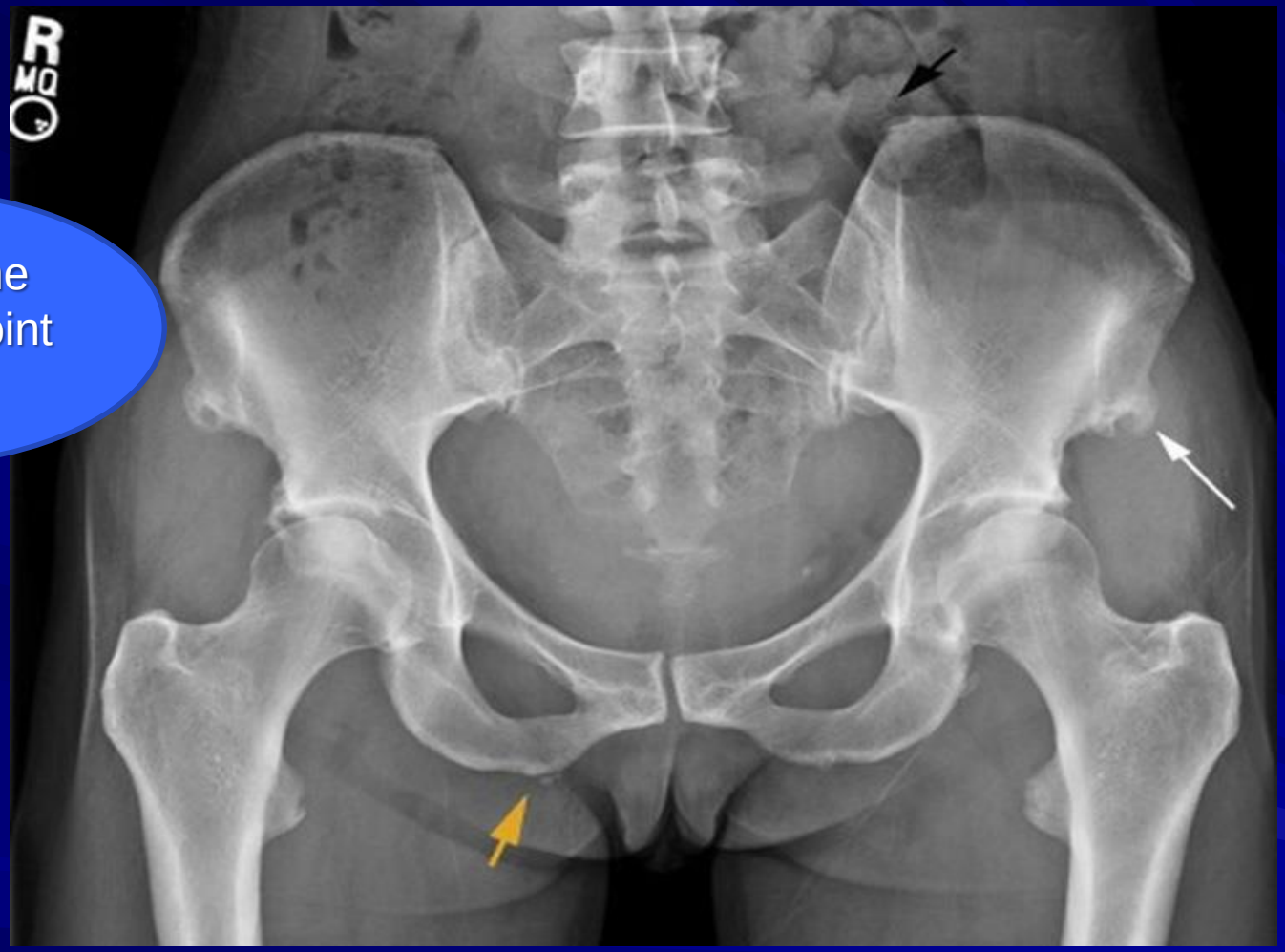


Diffuse Idiopathic Skeletal Hyperostosis (DISH)

- Radiographic features (continued):
  - Extra-spinal
    - Can occur at any tendinous or ligamentous insertion
    - Roughening at bony attachment (whiskering)
    - Ossification of ligament or tendon
    - Normal joint space preserved
    - Hypertrophy/enlarged sesamoids
    - Hypertrophy/broadening of the distal phalangeal tufts (arrowheading)

Diffuse Idiopathic Skeletal Hyperostosis

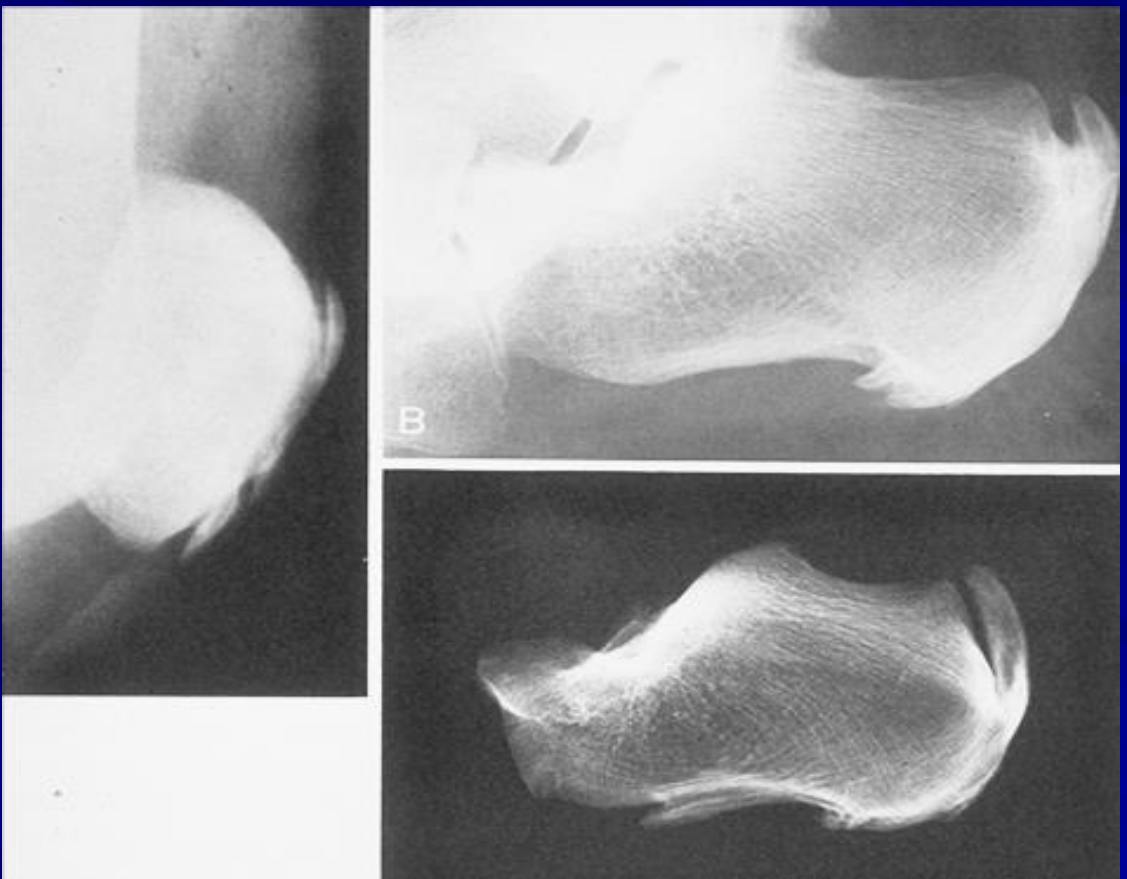
Note that the Sacro-iliac joint is spared.

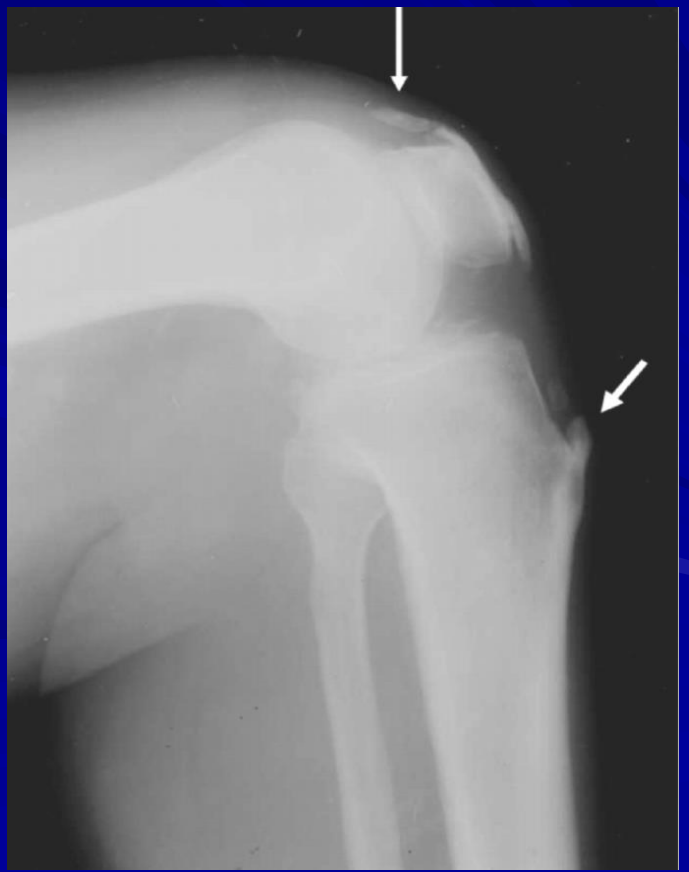


Enthesopathy at the iliolumbar ligament insertions (black arrow), anterior superior iliac spines (white arrow), and hamstring tendon (yellow arrow) attachments bilaterally.

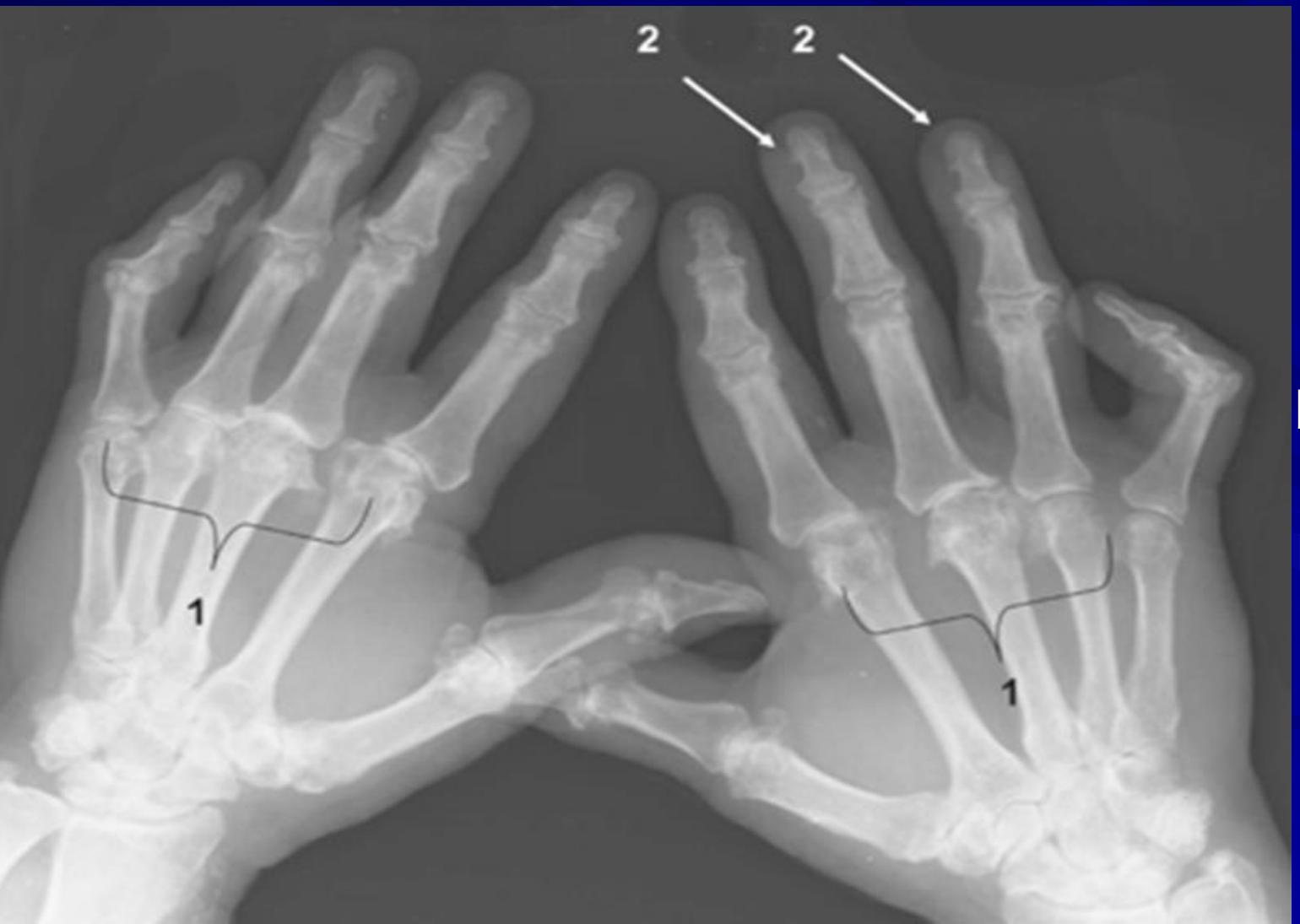
Diffuse Idiopathic Skeletal Hyperostosis (DISH)

Exuberant ossification at sites of tendon, ligamentous, or joint capsule insertion (entheses) is strongly suggestive of the diagnosis.





Diffuse Idiopathic Skeletal Hyperostosis (DISH)



Hypertrophic new bone formation at the metacarpals & distal phalangeal tufts.

Pulmonary Hypertrophic Osteoarthropathy

Pulmonary Hypertrophic Osteoarthropathy (PHO)

- Overview:
  - A clinical syndrome caused by intrathoracic neoplasm or infection (i.e. bronchogenic carcinoma) that produces the following triad of symptoms:
    - Digital clubbing
    - Symmetrical arthritis
    - Linear periostitis

(PHO)

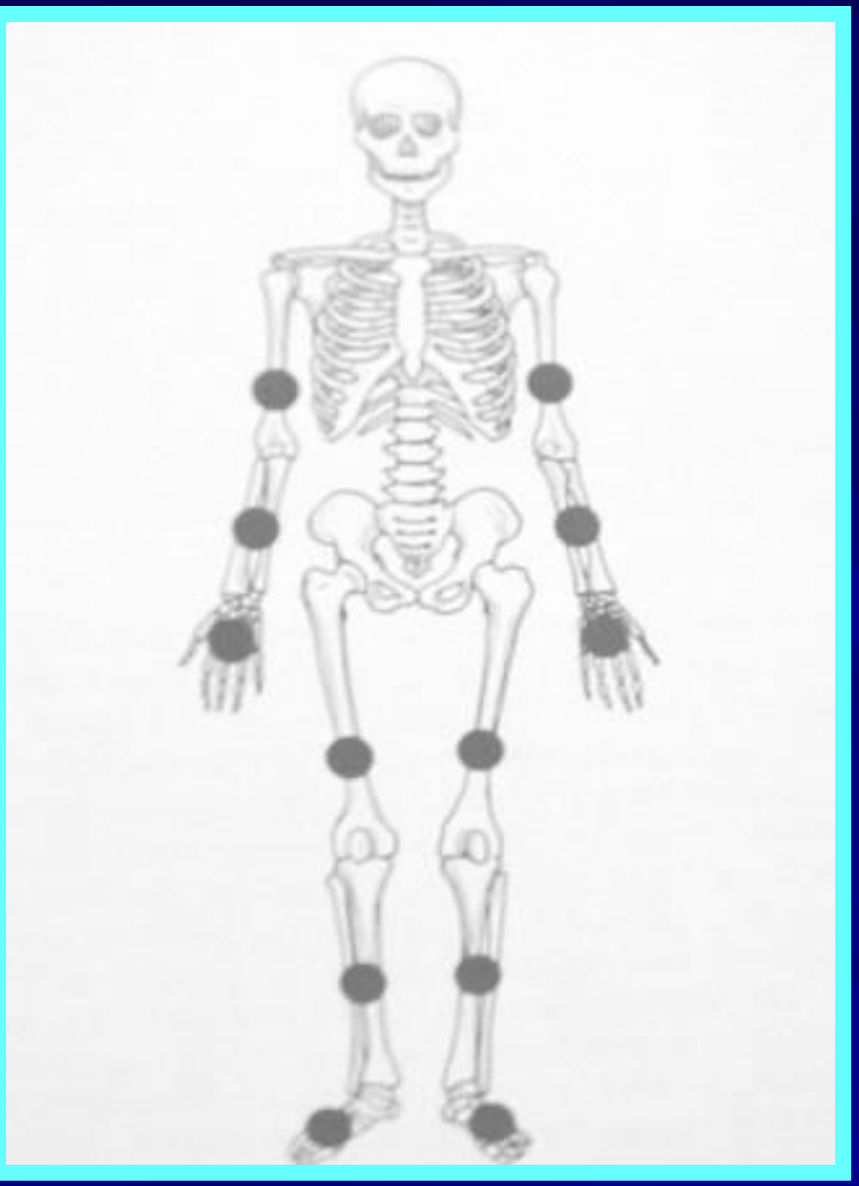
- AKA:
  - Hypertrophic Osteoarthropathy (HOA)
  - Primary Hypertrophic Osteoarthropathy (PHO)
  - Pachydermoperiostosis

  - “Pulmonary” dropped because the clinical findings may occur in several non-pulmonary diseases & even may occur without any underlying illness

PHO

- Clinical features:
  - Age: 40 – 60 yoa
  - Gender: males predominant
- Lab features:
  - Cause dependent
  - Elevated ESR
- Distribution:
  - Upper and lower extremities
    - Changes are most commonly observed in the tibia, radius, ulna, fibula, and femur

PHO



Most Common

Least Common

Location

Radius & Ulna
Tibia & Fibula
Proximal Phalanges
Femur
Metacarpals & metatarsals
Humerus & Distal Phalanges
Pelvis

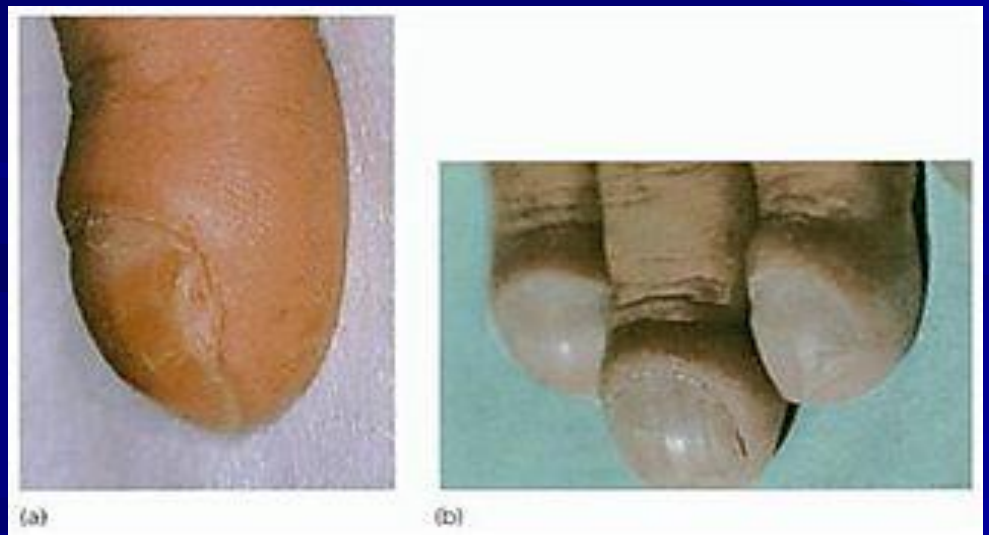
Bilateral/symmetric involvement

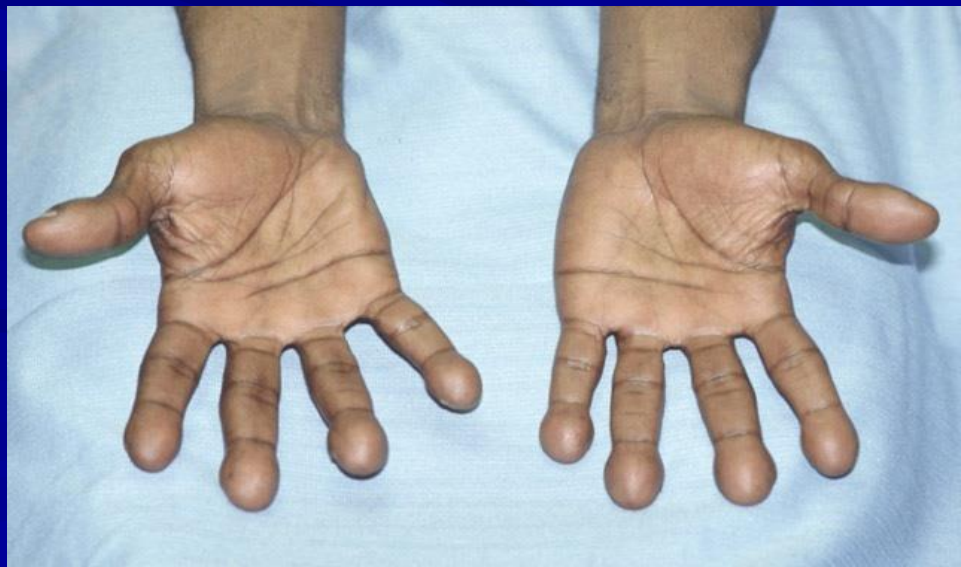
PHO

- Radiographic/Clinical features:
  - Joint effusion
    - No specific joint disease

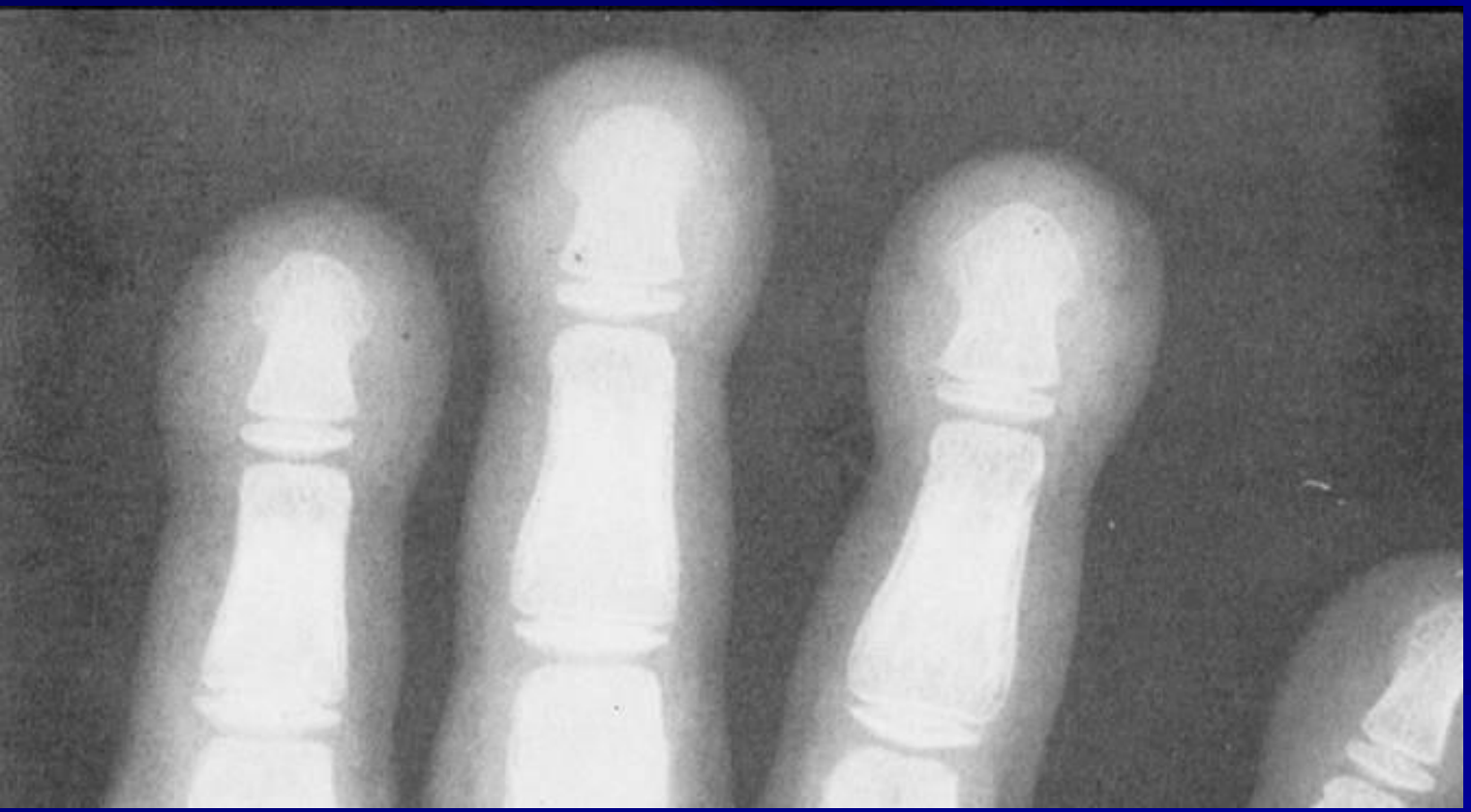
PHO

- Radiographic/Clinical features:
  - Digital clubbing
    - Bulbous enlargement of finger tips and rounding of nail plate
    - Clubbed portions consist of excessive collagen fiber deposition, accumulation of interstitial edema, & vascular hyperplasia between the nail matrix and the distal phalanx
    - AKA: Hippocratic finger; Watchglass nails, Drumstick fingers
    - Considered the oldest sign in clinical medicine-2500 yrs ago Hippocrates noted this finding on a patient suffering from empyema





Pulmonary Hypertrophic Osteoarthropathy (PHO)

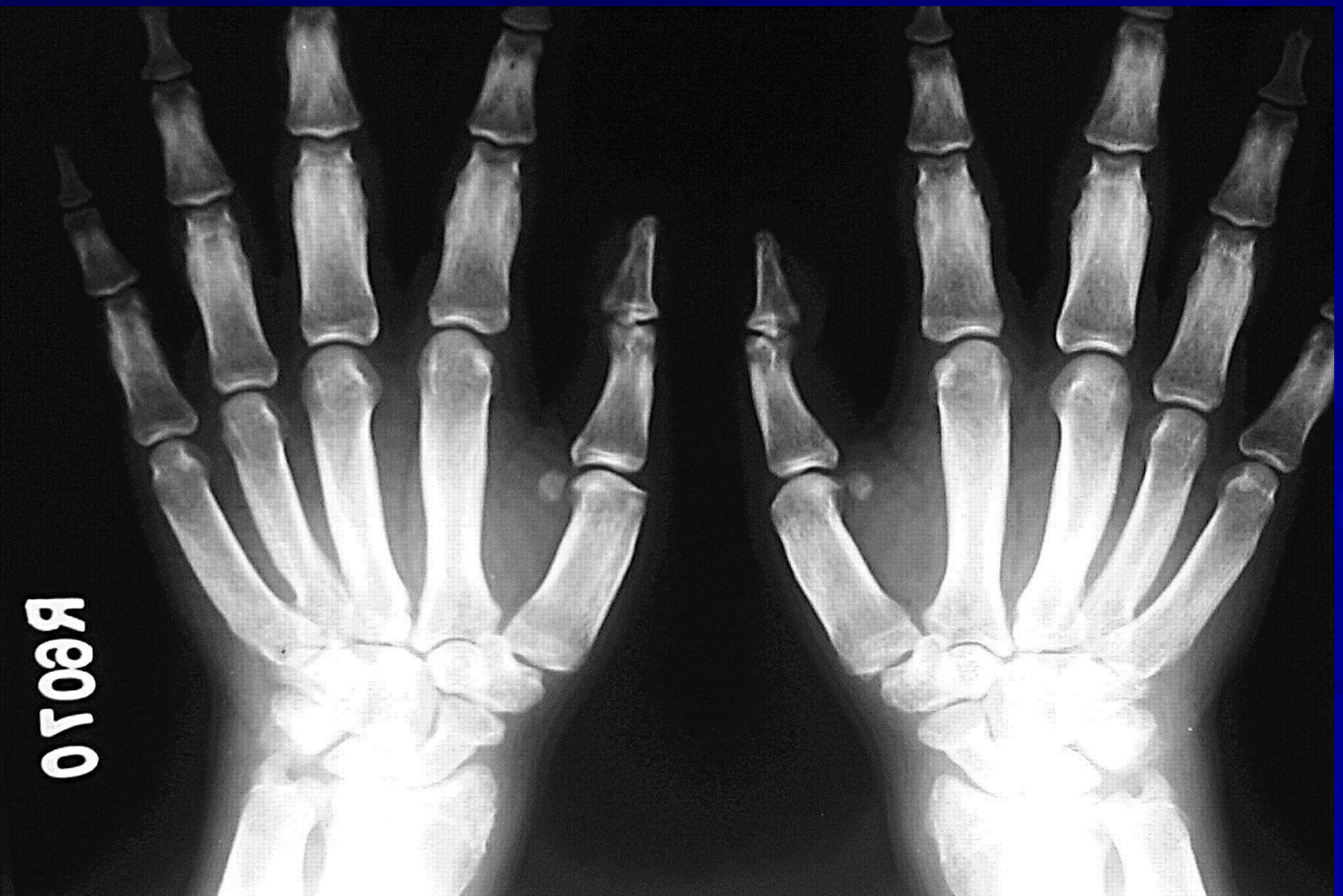


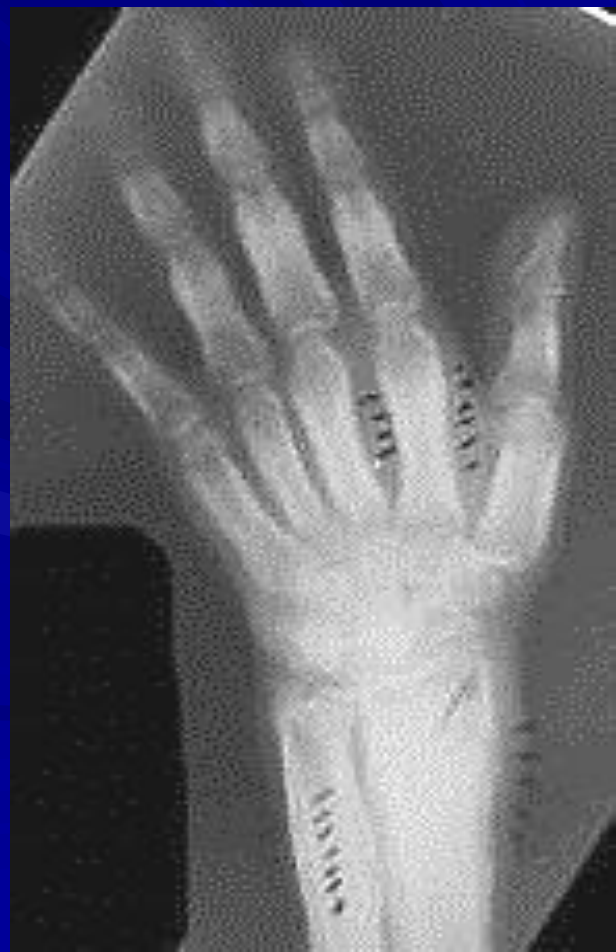
Digital Clubbing

PHO

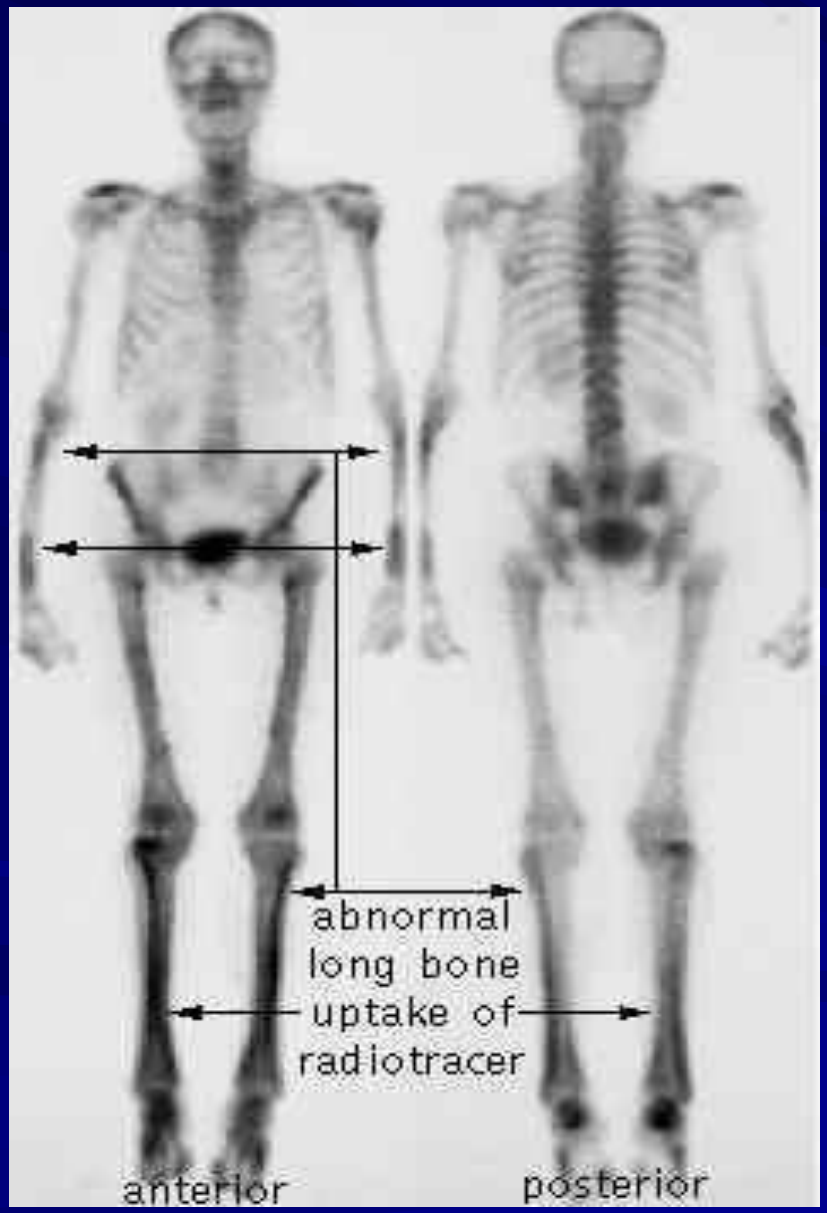
- Radiographic features:
  - Periostitis of long bones
    - **"Double stripe sign"** on bone scans
      - Symmetric diffusely increased uptake along cortical margins of diaphysis and metaphysis of tubular bones
    - Metaphyseal and diaphyseal regions typically involvement
      - Typically begins at the diaphysis of long bones & then extends into the metaphyseal region
      - Single periosteal proliferation: appears as a continuous thin line of sclerotic new bone separated from the cortex by a radiolucent space
      - Laminated periosteal proliferation: looks like onion skin in that it exhibits multiple layers/strips of new bone that eventually thicken and fuse with the cortex

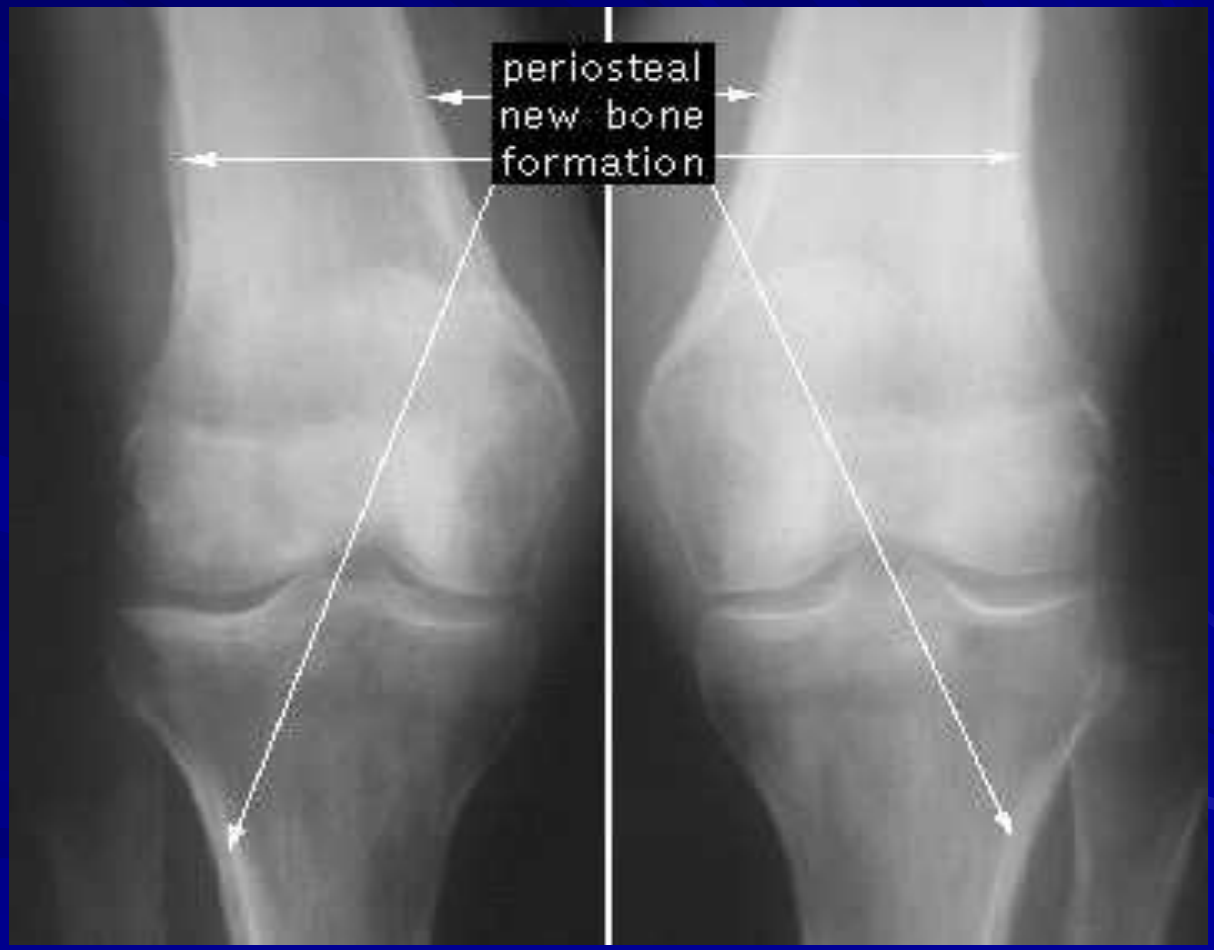
PHO





PHO

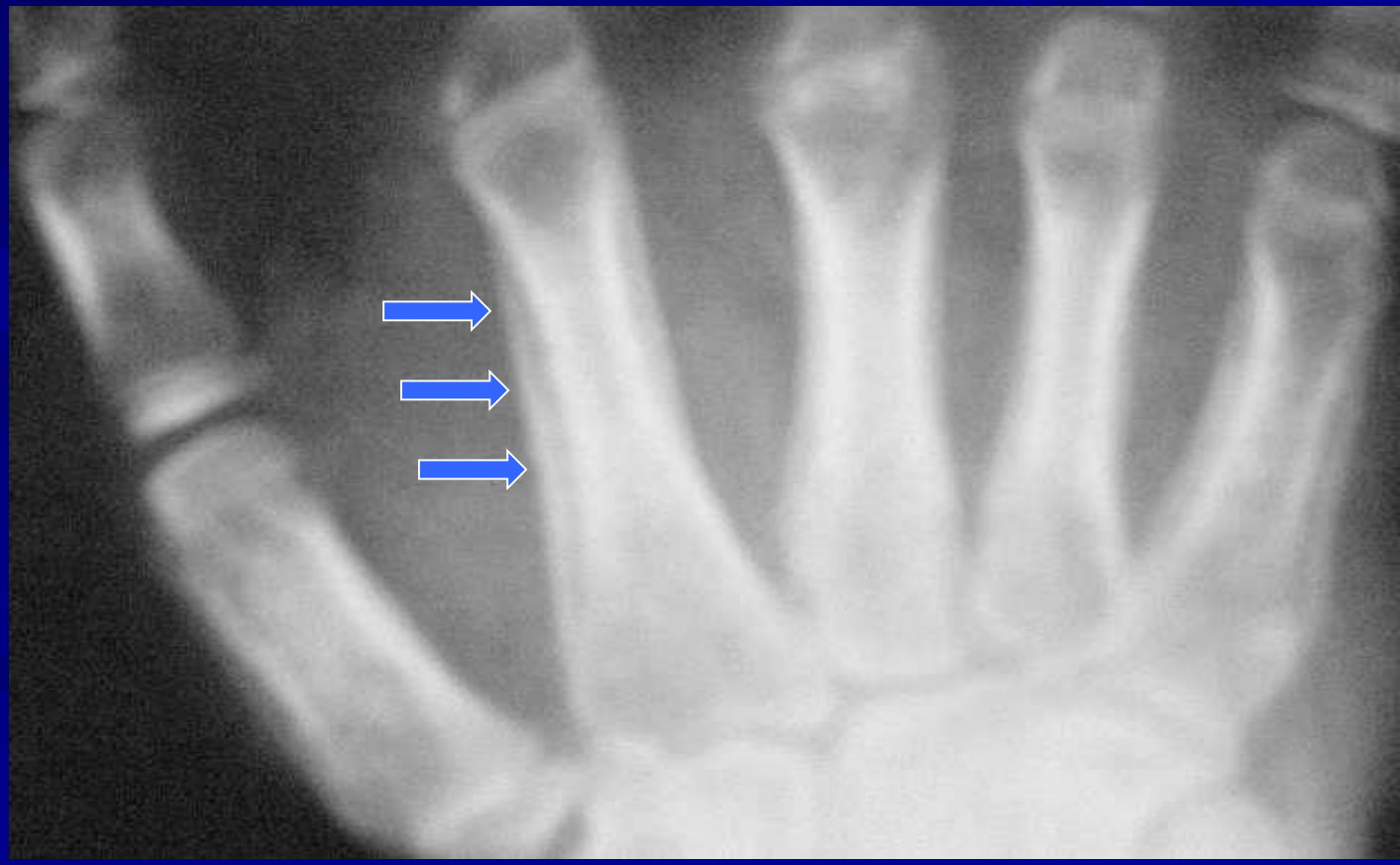

abnormal
long bone
uptake of
radiotracer
anterior
posterior


periosteal
new bone
formation

Pulmonary Hypertrophic Osteoarthropathy (PHO)

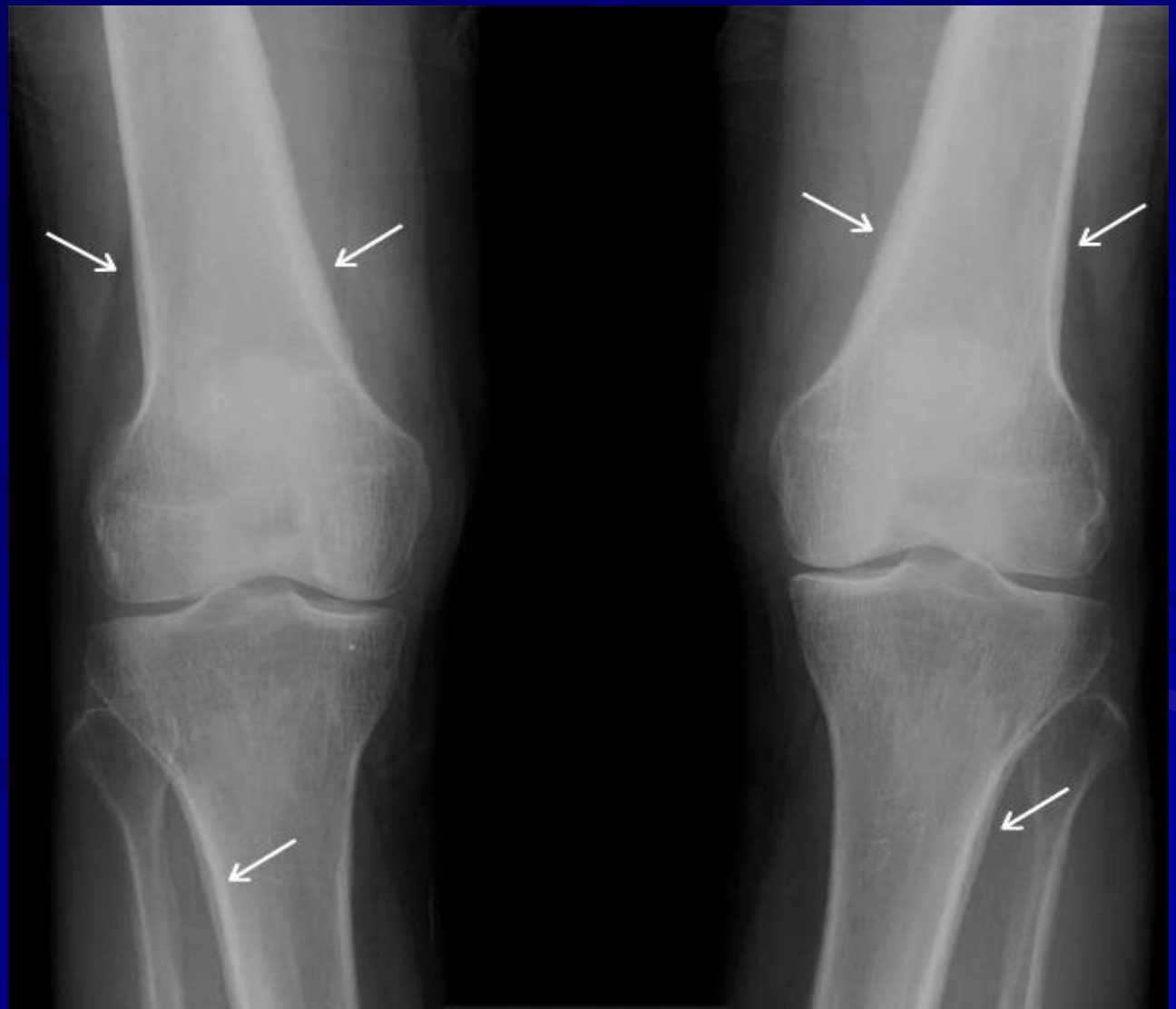
Linear Periostitis:

Subperiosteal edema elevates the periosteum & osteoid matrix is deposited beneath it. As this mineralizes, a new layer of bone is formed, and eventually the distal long bones may become sheathed with a cuff of new bone.



Pulmonary Hypertrophic Osteoarthropathy (PHO)

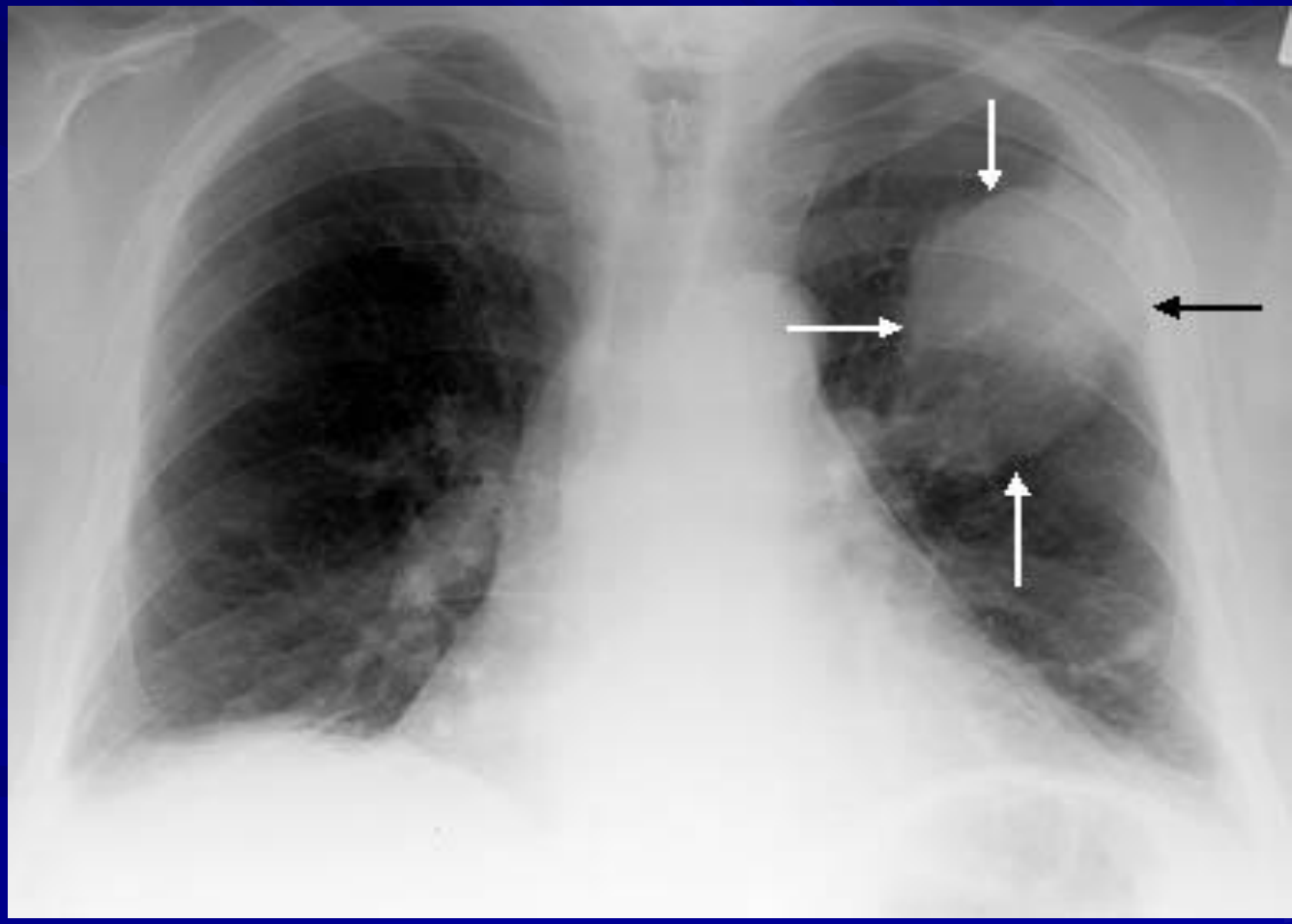
Linear Periostitis



Pulmonary Hypertrophic Osteoarthropathy (PHO)

Bronchogenic Carcinoma:

Begins as a small focus of atypical epithelial cells within the bronchial mucosa that grows and spreads to regional lymph nodes & organs like the liver, brain, and bone.



Pigmented Villonodular Synovitis

Pigmented Villonodular Synovitis (PVNS)

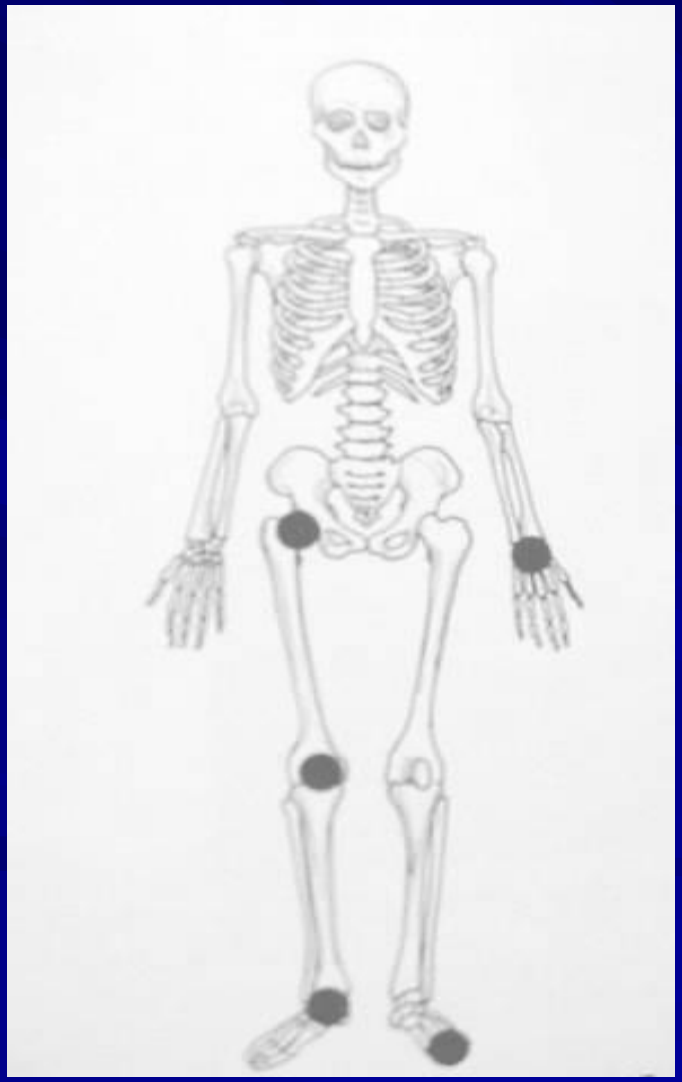
- Overview:
  - Localized , invasive nodular mass arising from synovial lining, tendon sheath, ligaments, or bursae
    - Locally destructive
    - Does not metastasize
- Clinical features:
  - Age: young adults
  - Gender: males predominant
  - Typically monoarticular arthritis in nature affecting the knee, hip, ankle or hand
  - Decreased ROM/locking/catching of the involved joint
  - Insidious progression with acute episodic attacks of pain & swelling
  - Synovial fluid is typically hemorrhagic & dark brown in color

Pigmented Villonodular Synovitis

- Radiographic features:
  - Soft-tissue mass with bony erosions/lysis
  - Pressure erosions well-demarcated with sclerotic borders (saucerization)
  - Calcification unusual
  - Typically monoarticular, but when polyarticular it will affect the midfoot due to its unique synovial compartmentalization

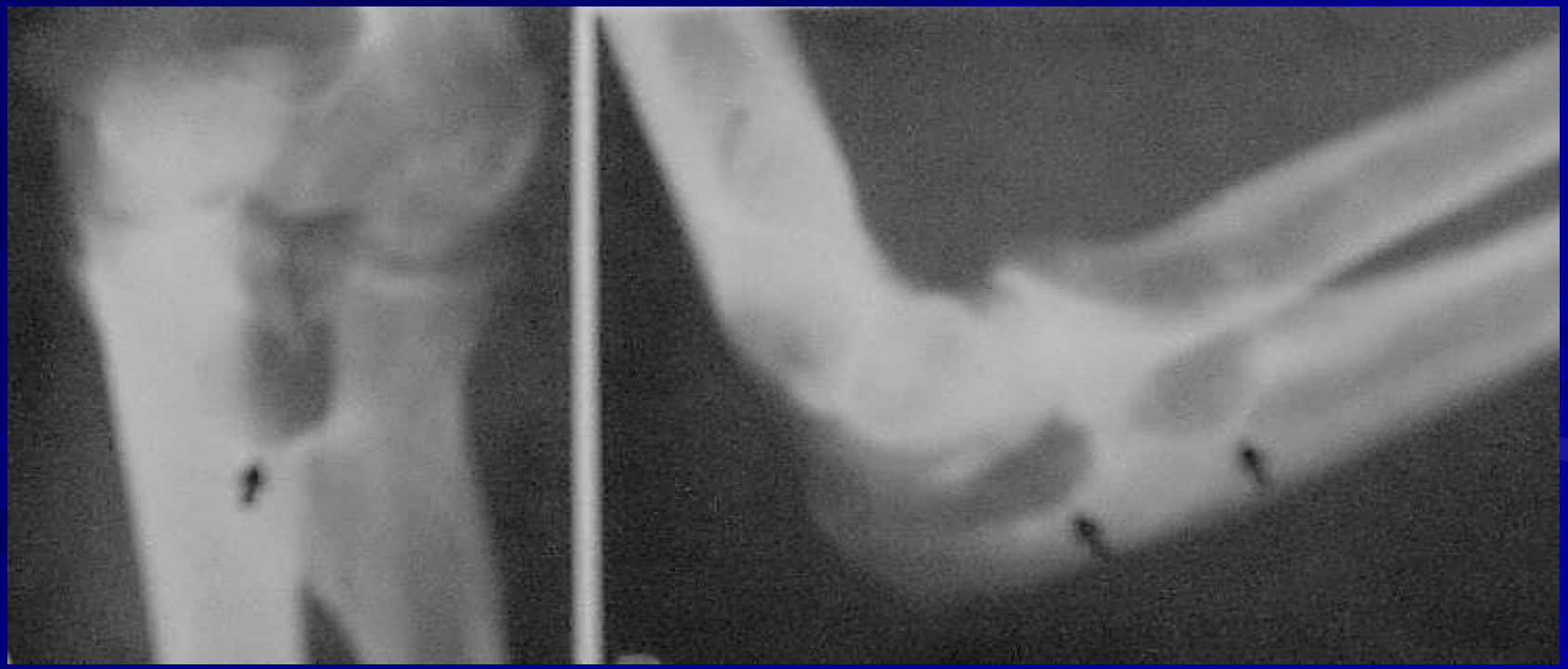
Pigmented Villonodular Synovitis

Monoarticular
&
Asymmetric Involvement

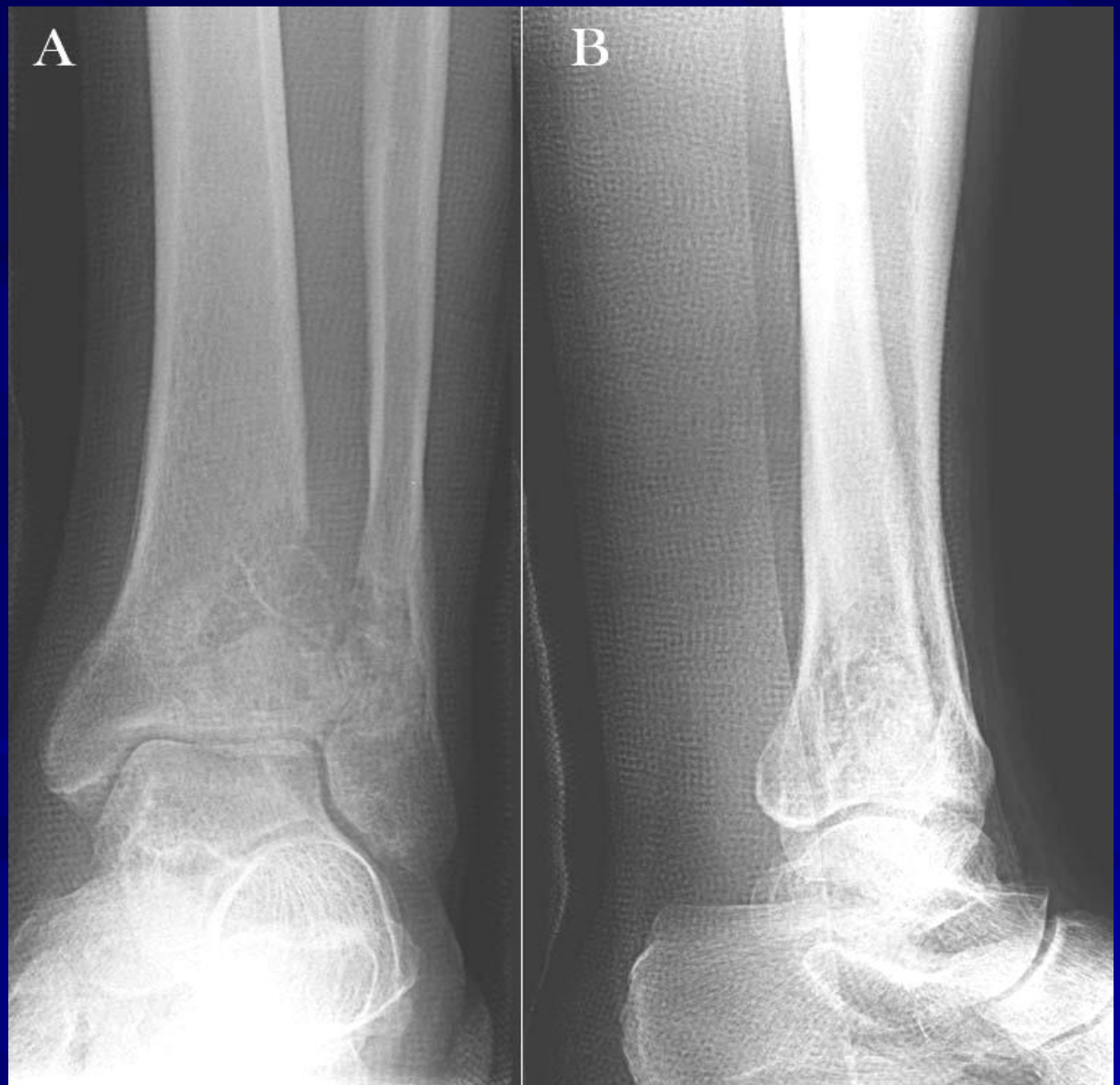


Pigmented Villonodular Synovitis

Extrinsic bone erosions in elbow

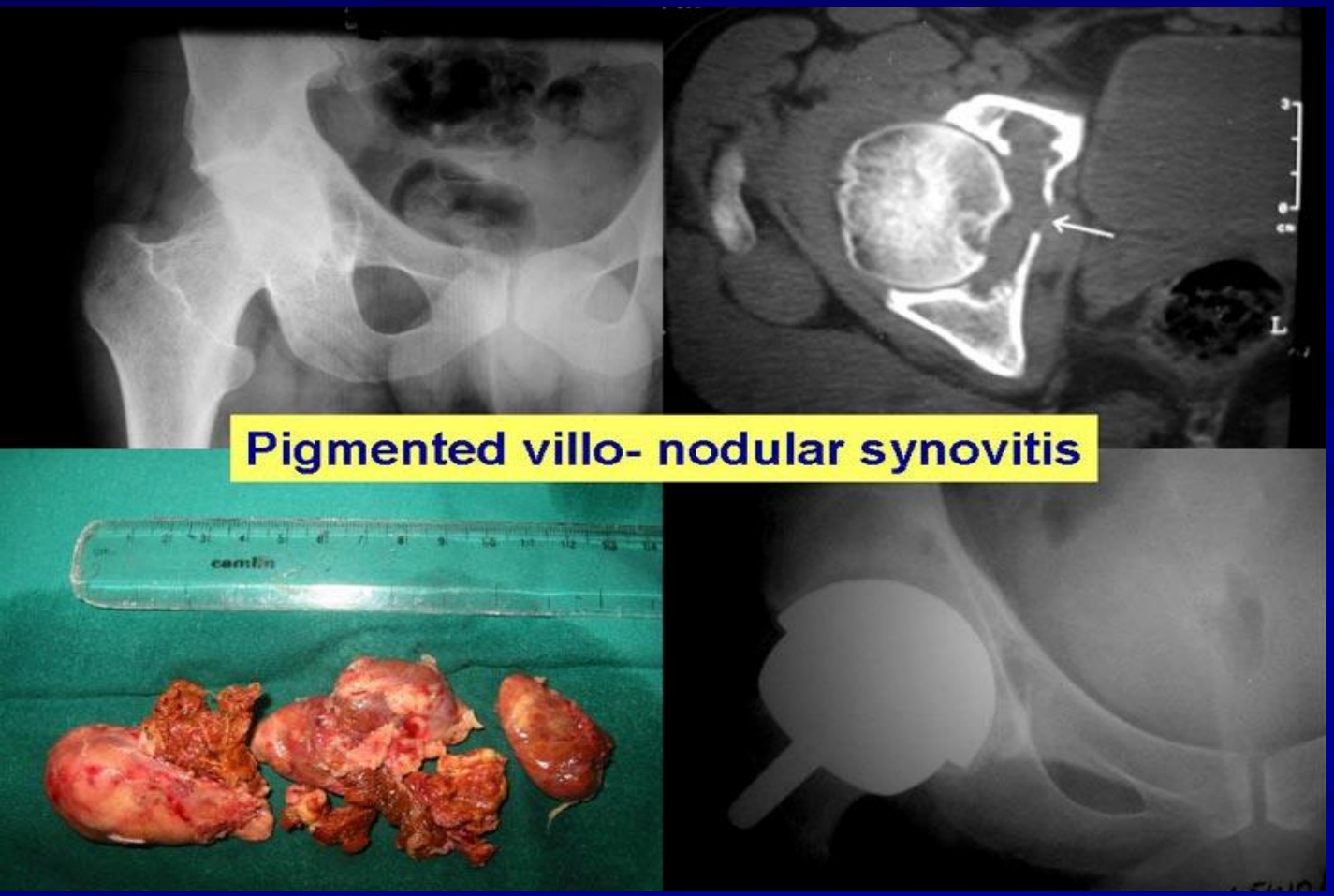


Pigmented Villonodular Synovitis



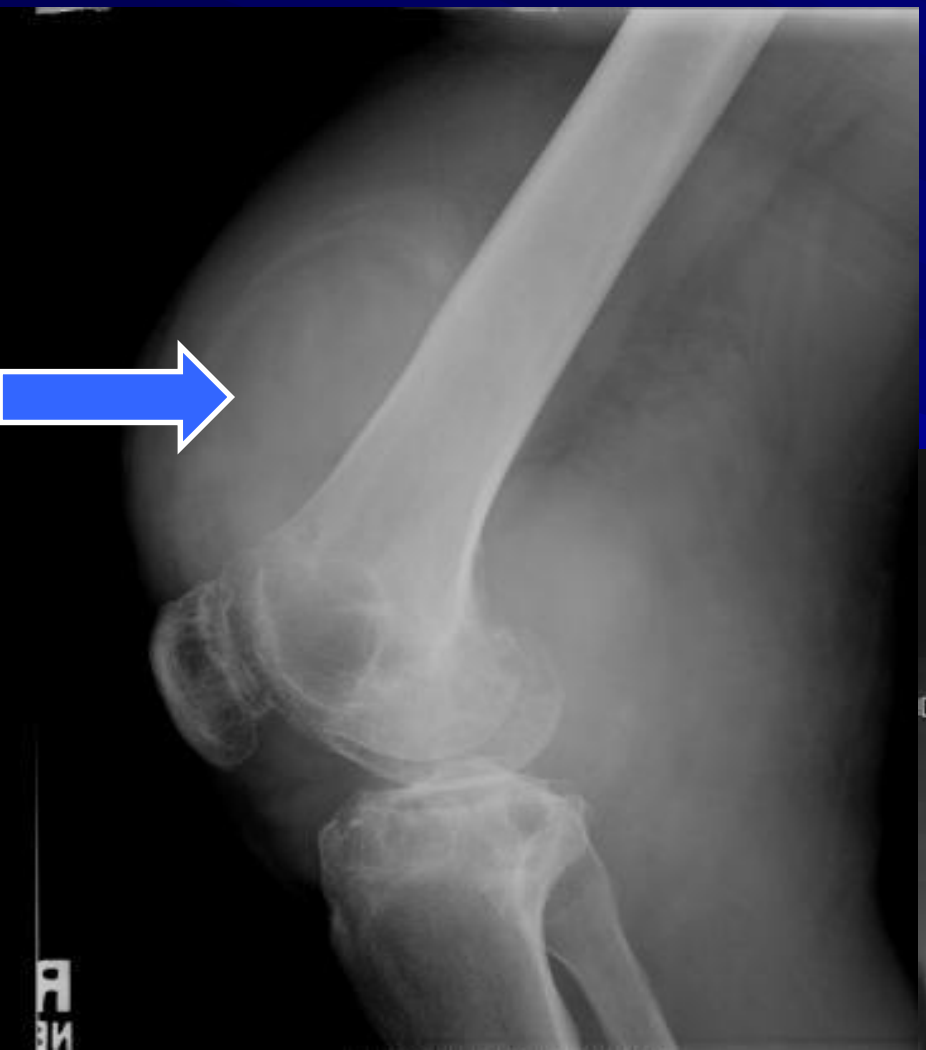
The hyperplastic synovium invades the subchondral bone & produces cysts and erosions.

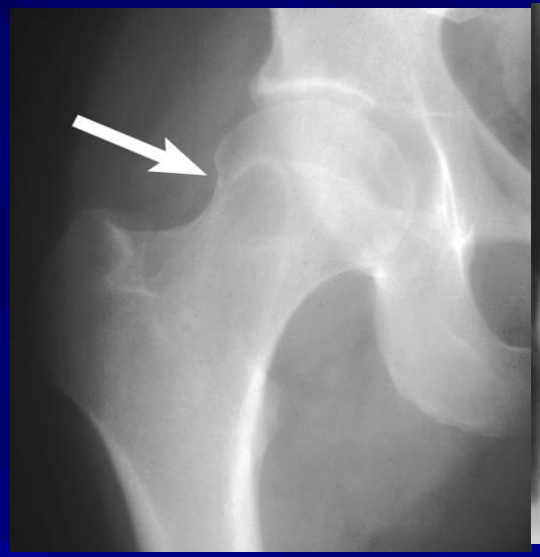
Pigmented Villonodular Synovitis

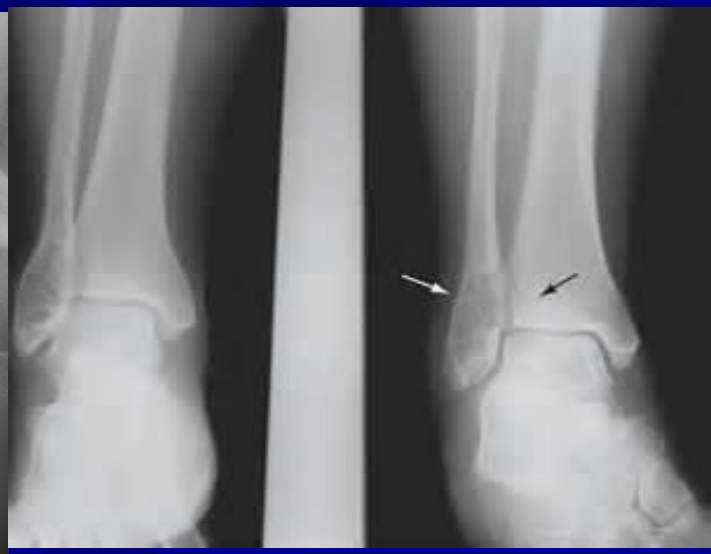


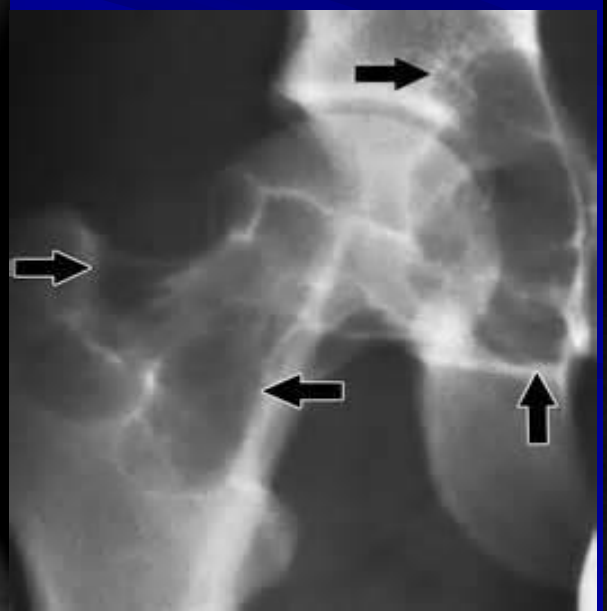
The nodular mass may have many synovial protrusions that affect joints, bursae, and tendon sheaths. It has a typical yellow-brown appearance due to excessive deposits of hemosiderin, fibrous stroma & lipids.

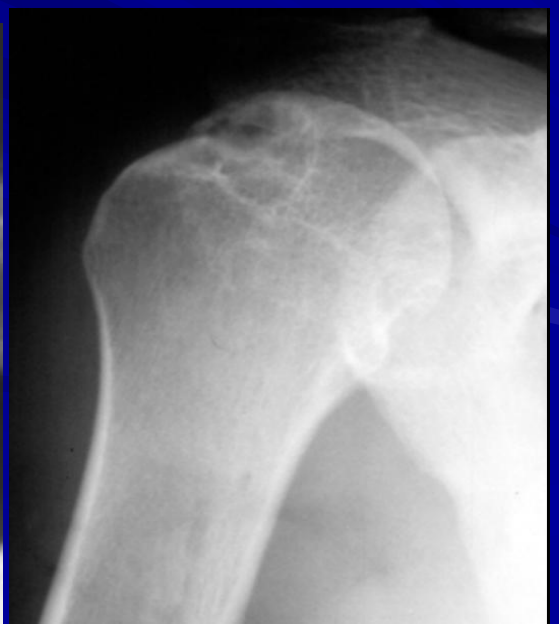
Pigmented Villonodular Synovitis











Arthritides associated with Collagen Vascular Diseases

Systemic Lupus Erythematosus

Systemic Lupus Erythematosus

- Overview:
  - Autoimmune, connective tissue disorder characterized by inflammation & vasculitis with multiple organ system involvement
- Clinical features:
  - Gender: Females (13:1 ratio)
  - Age: 20 – 40 yoa
- Lab findings:
  - Elevated ESR and ANA

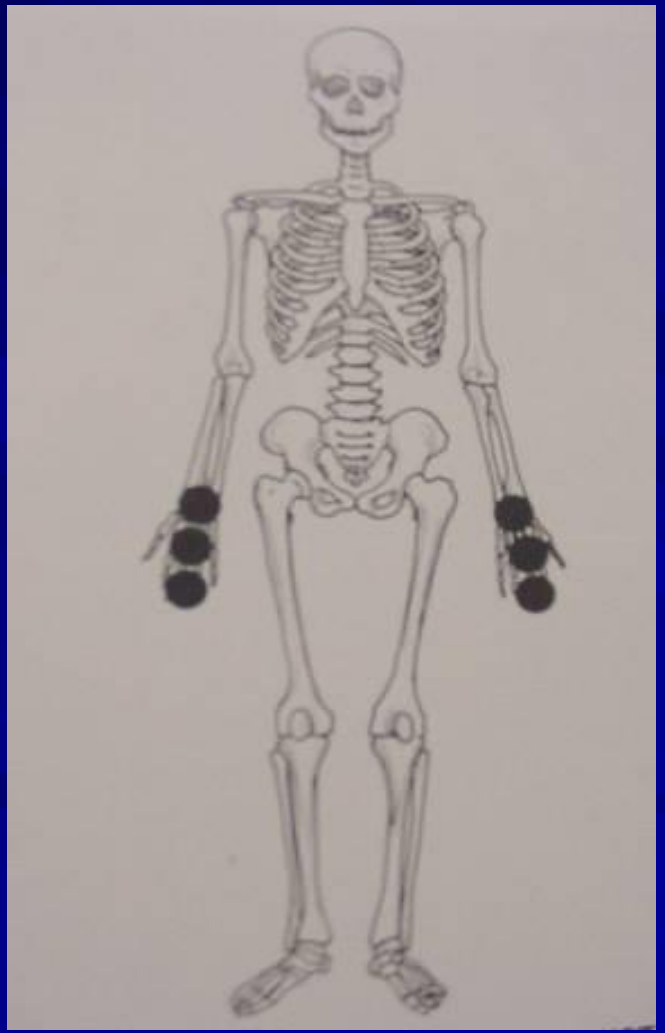
Systemic Lupus Erythematosus

- Radiographic features:
  - Most prominent x-ray features in hands
  - Juxta-articular osteopenia & soft tissue edema
  - Affects the small joints of the hands, wrists, knees, and shoulder
  - Polyarthritis (80%):
    - Symmetric
    - Non-erosive
    - Non-deforming

Systemic Lupus Erythematosus

- Radiographic features:
  - **Reversible** subluxations, dislocations, deformities are due to ligamentous instability & laxity of the supporting structures
  - Calcification & atrophy
    - Muscle atrophy & contractures cause the reversible deformities to become fixed/rigid
  - 10% will have irreversible deformities:
    - Ulnar drift at the metacarpophalangeal joints
    - Swan neck deformity (like RA)
    - Boutonniere deformity (like RA)

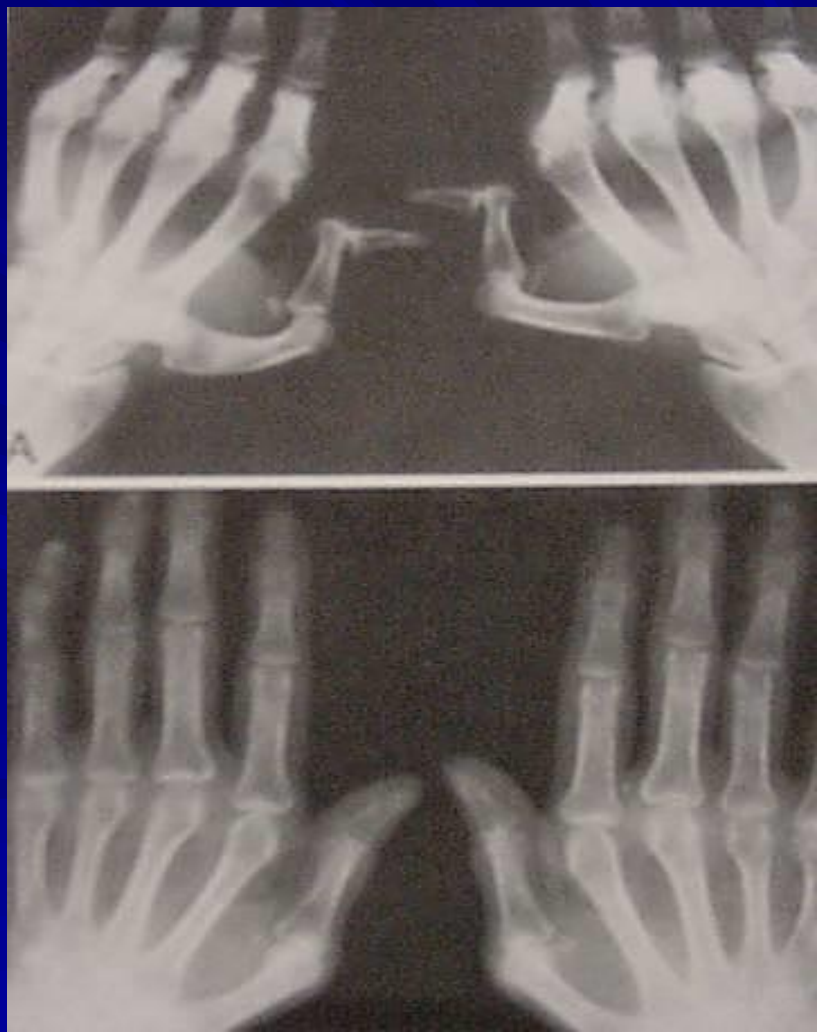
Systemic Lupus Erythematosus



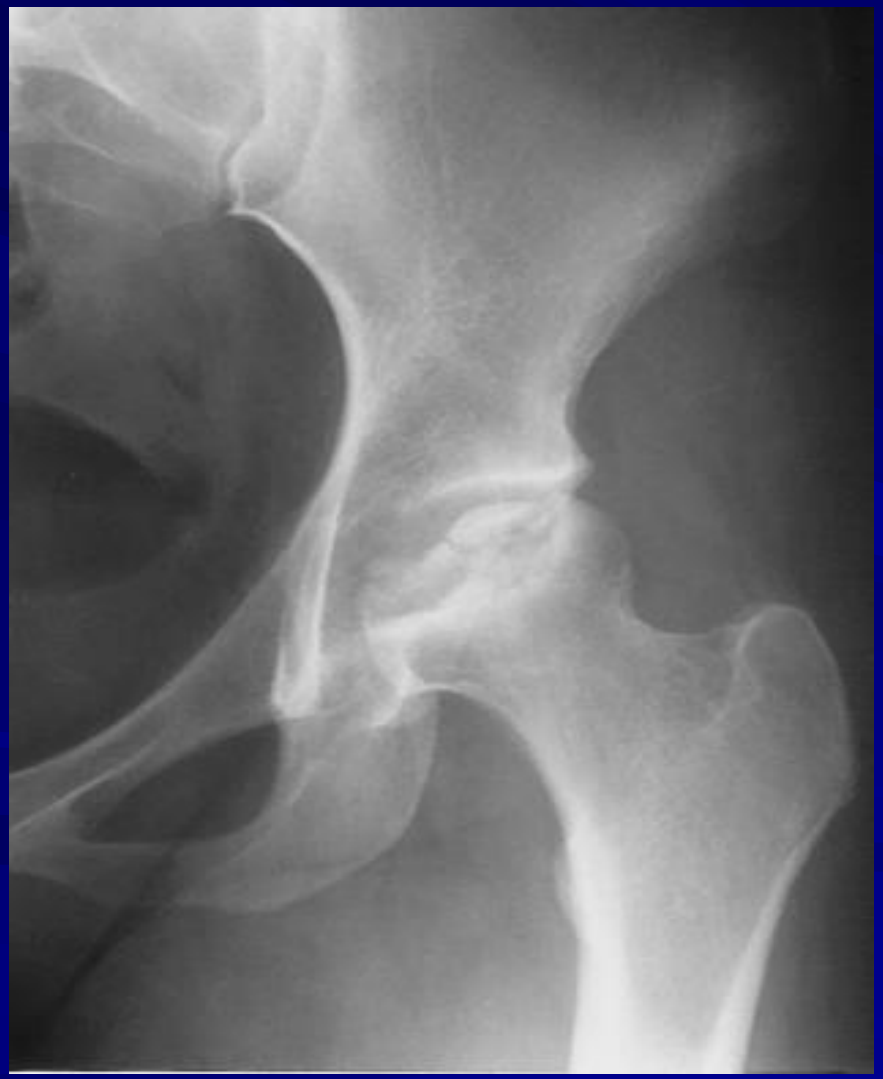
Systemic Lupus Erythematosus

Swan-neck and boutonniere deformities of the hand

Reversible when placed against x-ray cassette



Systemic Lupus Erythematosus



AVN attributable to SLE and/or steroid therapy. 5-50% of SLE patients develop osteonecrosis of weight-bearing joints. Note the subchondral fractures, density changes, & joint surface disruption of the femoral head.

Scleroderma

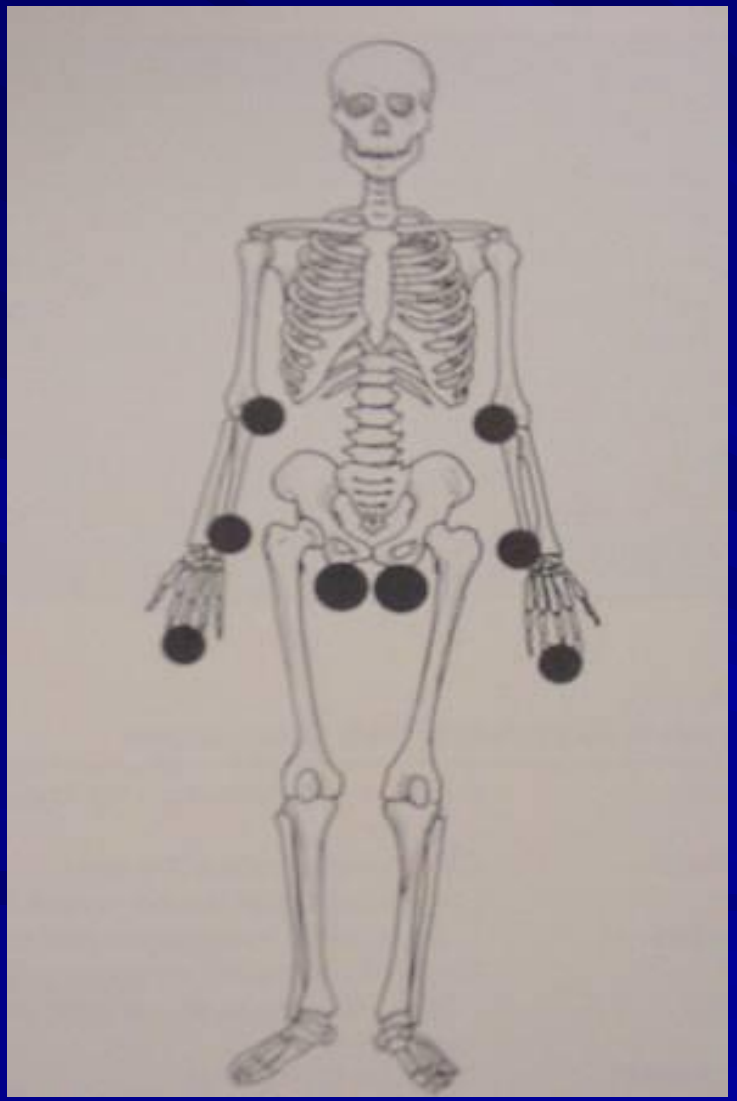
Scleroderma

- Overview:
  - Systemic inflammatory connective tissue disorder affecting skin, lungs, GI, heart, kidneys and musculoskeletal system
- Clinical features:
  - Females 30 – 60 yoa
  - CREST (Calcinosis Cutis; Raynaud's Phenomenon; Esophageal Dysmotility; Sclerodactyly; Telangiectasia)

Scleroderma

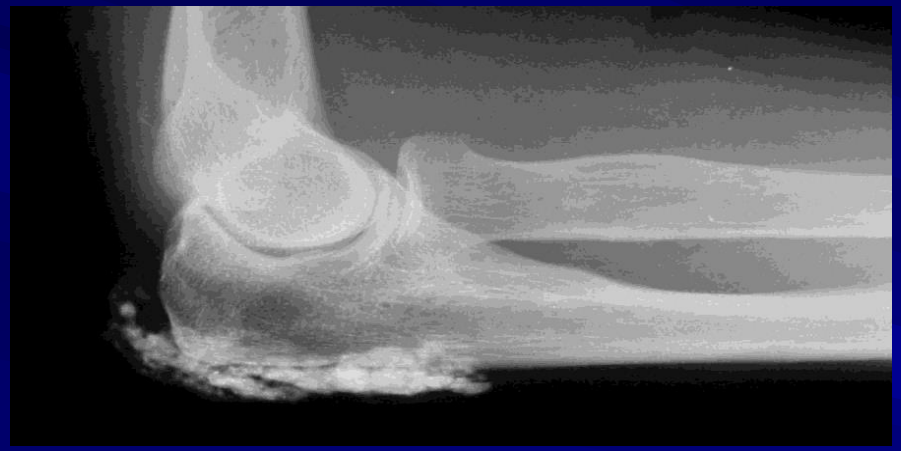
- Radiographic features:
  - **Hands** most commonly involved
  - Soft-tissue atrophy & calcification
  - Arthritis is initial symptom in 2/3^{rds} of patients, often preceding skin changes
    - Resembles RA but is less destructive/erosive
    - Symmetric joint space narrowing with marginal erosions (9-15%)
    - Acro-osteolysis
    - Juxta-articular osteopenia
    - Contractures are generally dermatogenic induced due to sclerotic changes of the overlying skin or surrounding connective tissue

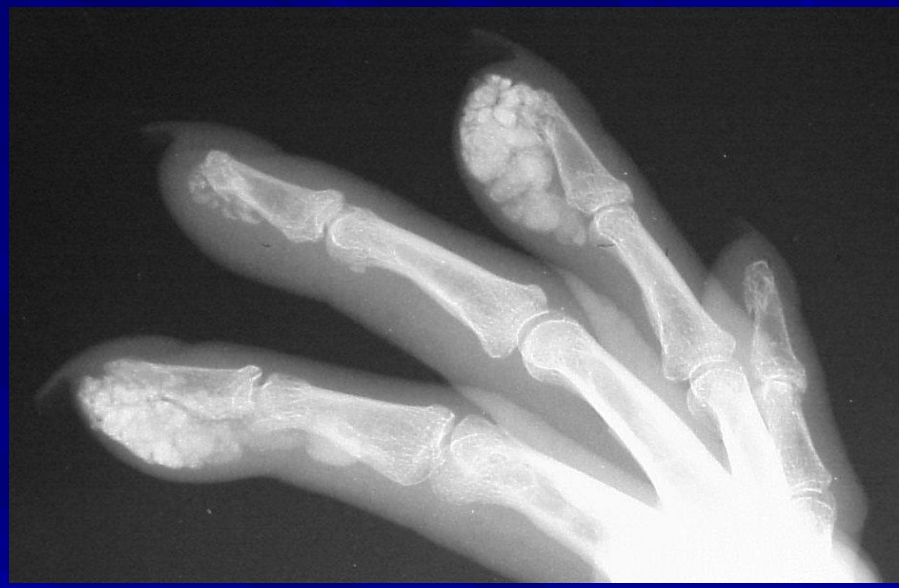
Scleroderma

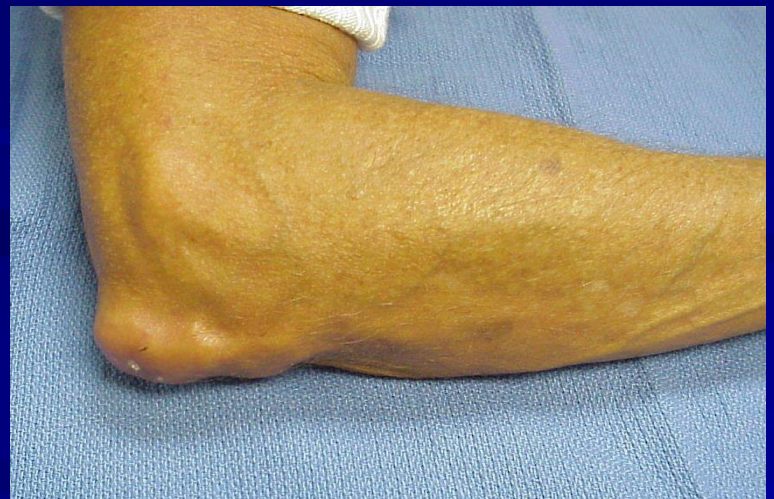


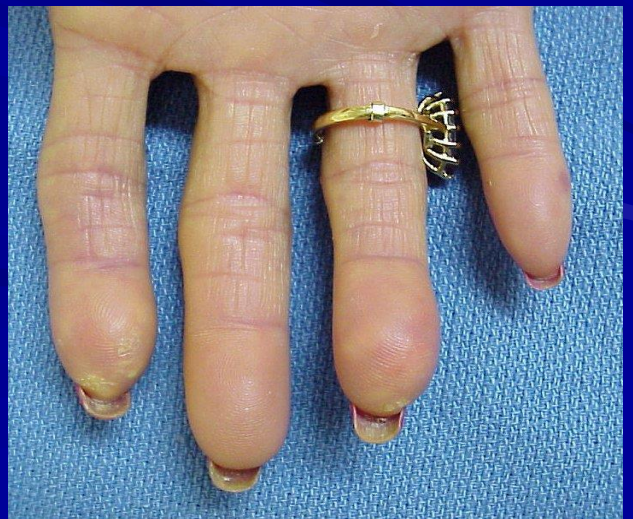
Scleroderma

Calcinosis cutis



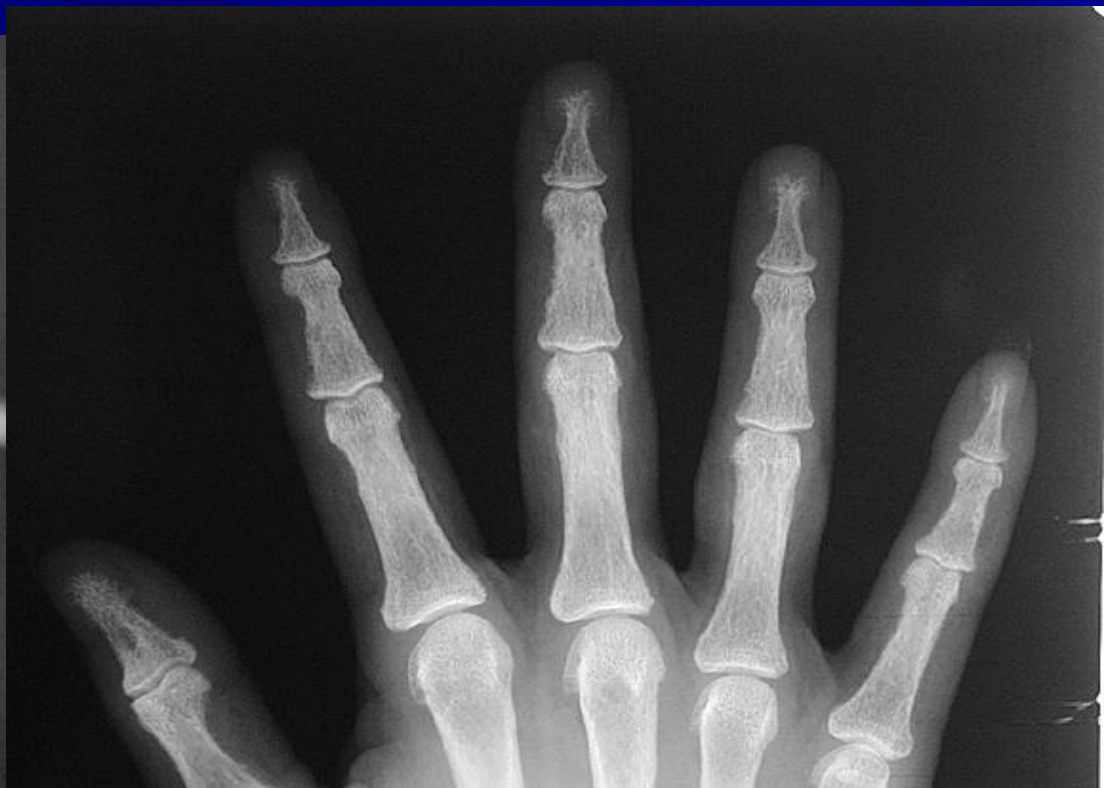




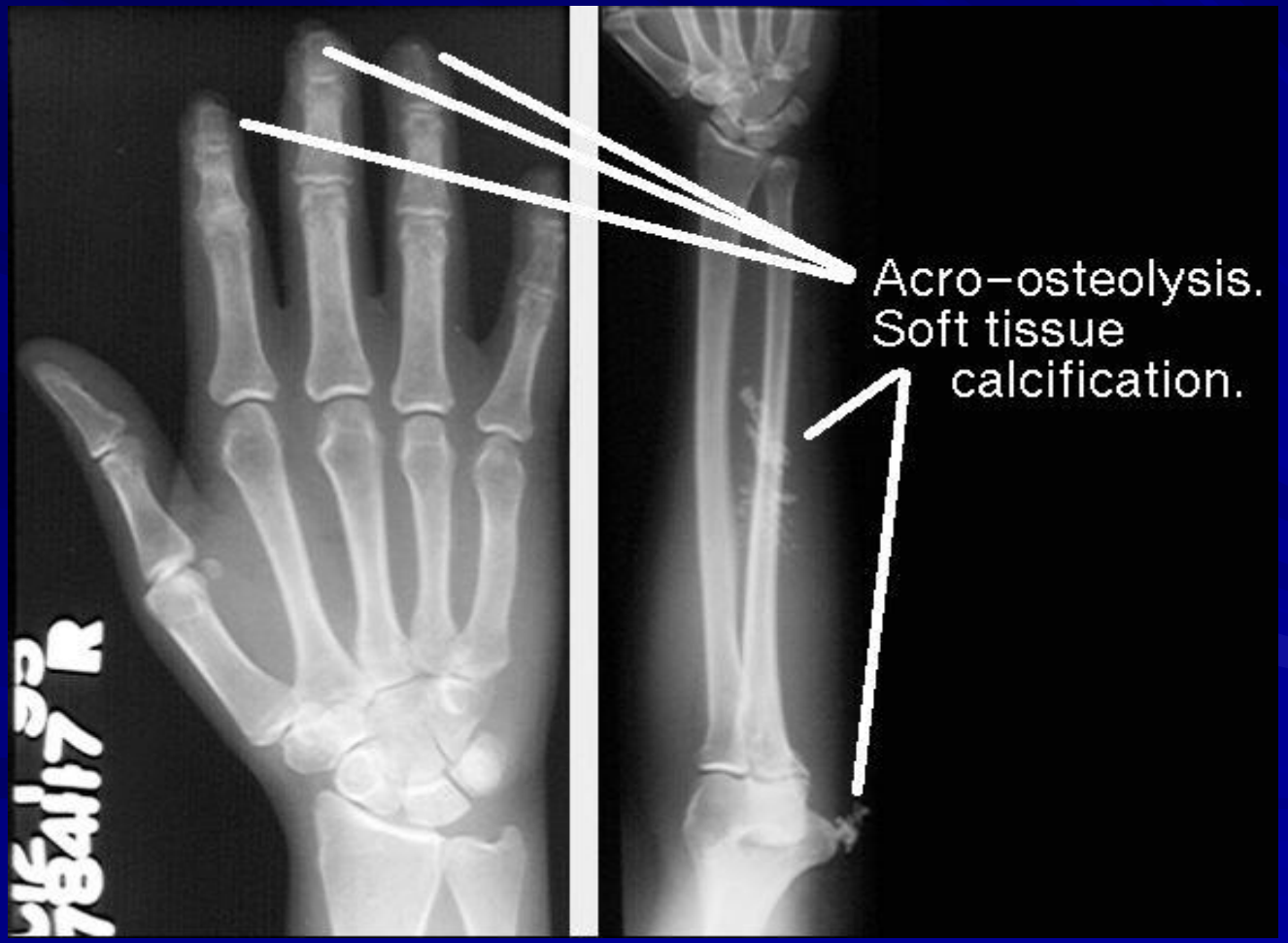


Scleroderma

Acro-osteolysis



Scleroderma

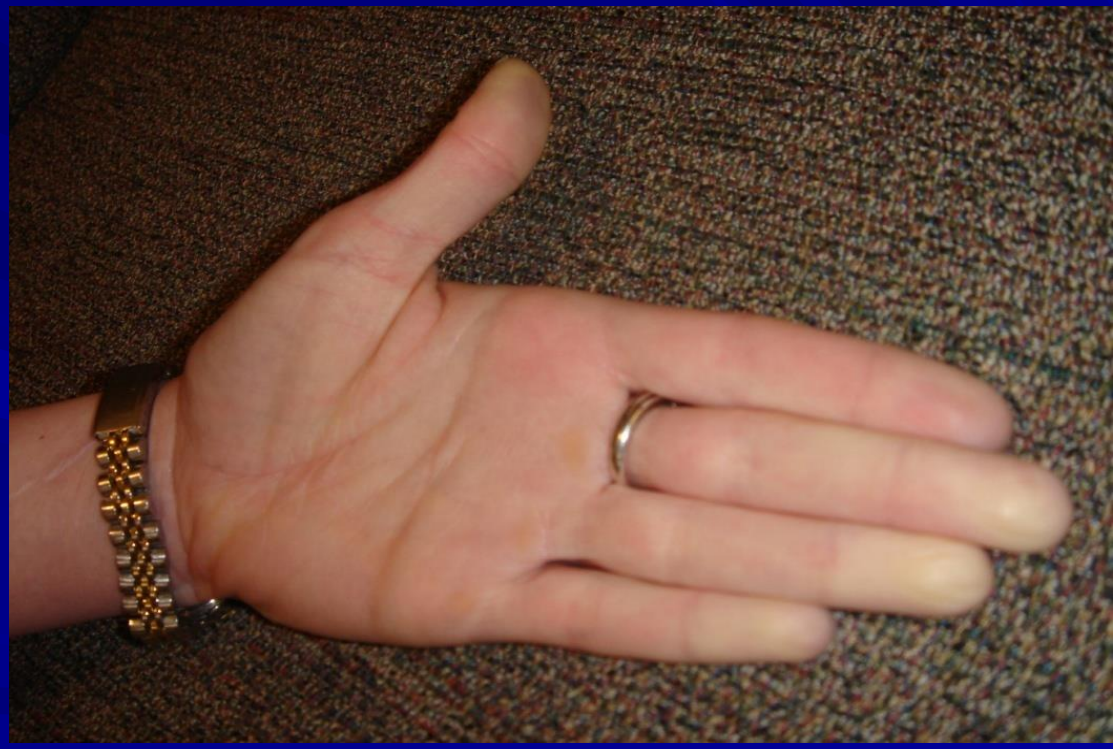


Scleroderma

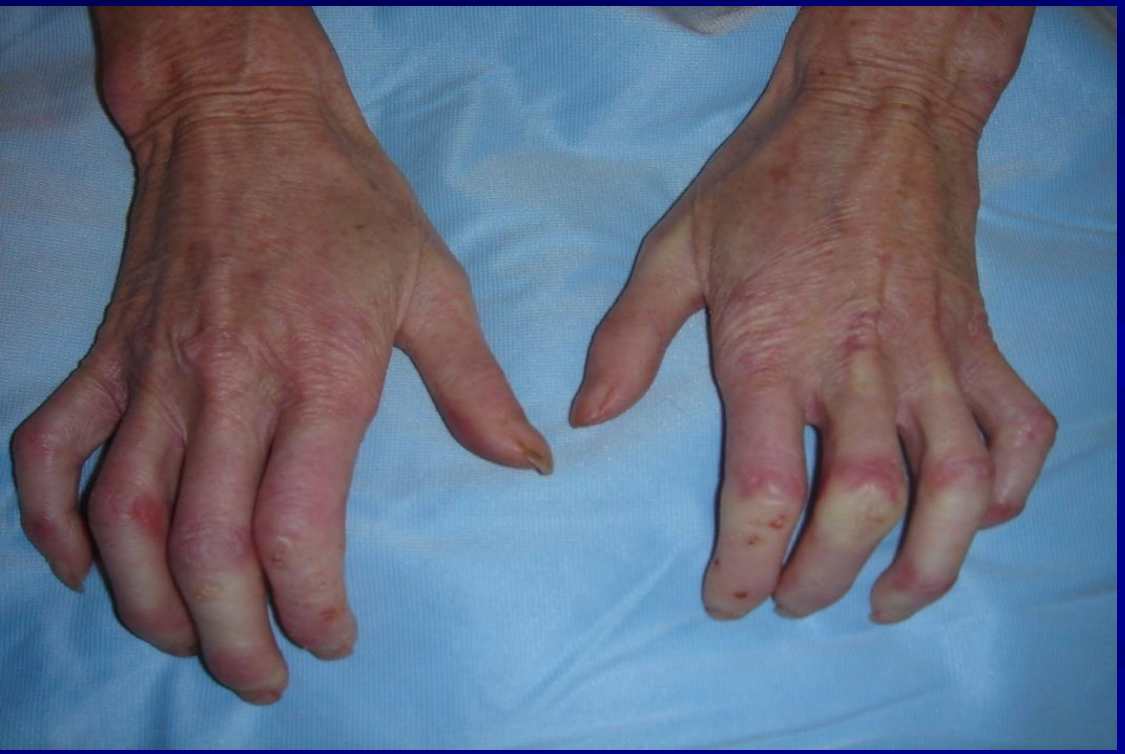
Raynaud's Phenomenon:

Triphasic color changes of pallor, cyanosis, and erythema (white/blue/red) represent phases of vasoconstriction, slow blood flow, and reperfusion.

Color changes extend proximally from the tips of digits to various levels, with a well-demarcated border



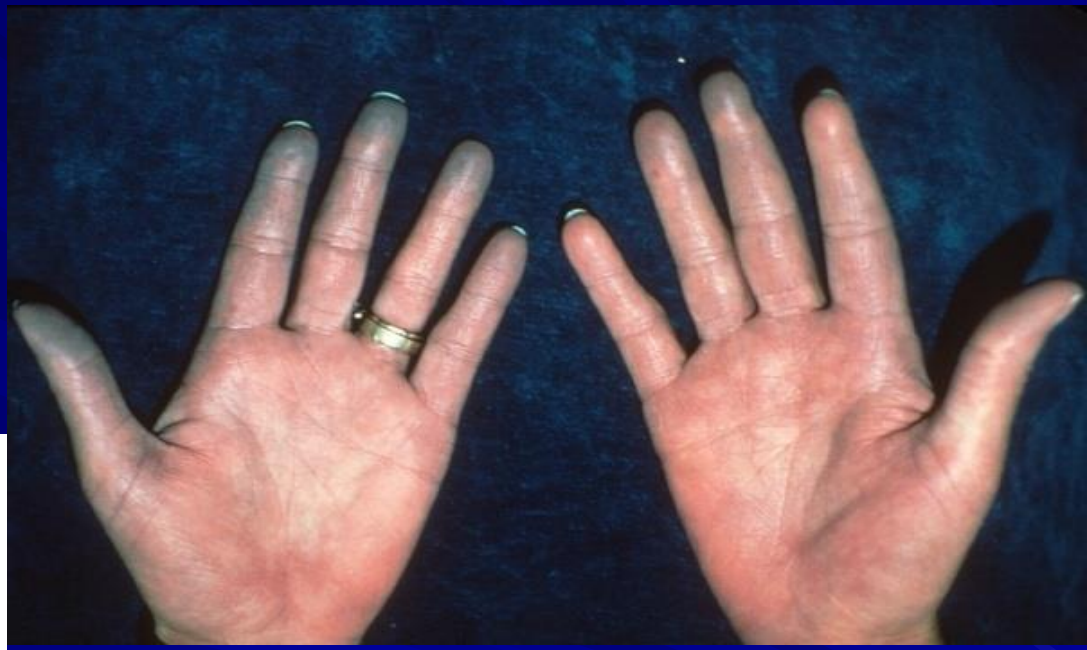
Scleroderma

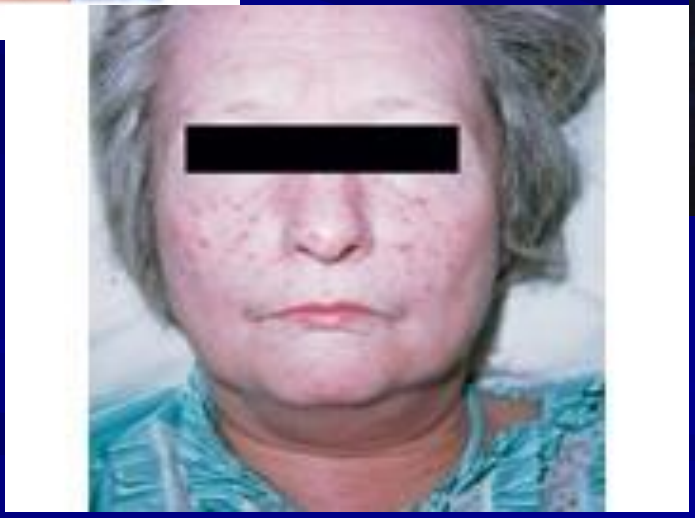


- Sclerodactyly:

  Initial stage of the disease involves swelling of the fingers. Later, as the connective tissue becomes fibrotic, while skin on the fingers and toes becomes hard & shiny. The fingers can become difficult to bend and can form contractures due to the severe tightening of the skin.

Scleroderma





Telangiectasias

In Summary:

- Remember your 6 broad arthritide categories.
- It is important to differentiate **degenerative** from **inflammatory** causes of joint space narrowing.
- **Degenerative arthritis** is characterized by subchondral cysts, osteophytosis, subchondral sclerosis, & asymmetric joint space narrowing.
- **Inflammatory arthritis** is characterized by bone erosions, osteopenia, soft-tissue swelling, & uniform joint space narrowing.
- Inflammation that involves multiple joints in a proximal distribution of the hands or feet **without** bone proliferation suggests Rheumatoid Arthritis.
- Inflammation that involves multiples joints in a distal distribution of the hands or feet **with** bone proliferation suggests a Seronegative Spondyloarthropathy.

The End