Infection=Only thing w/ no gender bias or age bias
Acts much quicker than anything else (including tumors)

Look at Marchiori (Dr. Kuhn wrote that chapter, and he writes the test not Yochum)

Subdivisions of infection (Osteomyelitis)
  Separative-most frequent (Staph Aureus accounts for 90% of all bone and joint infections)
  Humerus infections are typically Streptococcus-Proximity to entry points
  Pseudomonas-affects S-joints (Spine, SC, Symphysis Pubis, SI)
  Transplant, HIV, immune suppressive meds
  The rest of the infections (opportunistic
  Non-separative-most famous=TB
  Fungal
    Regional distributions in the US
  Syphilitic
    Young victims

How do we get these infections?
  Hematogenous spread
  Initial infection via breech of vessel wall 1st
    URT (respiratory)
    UTI (genital urinary)
    Skin infection
  May also include direct infection-puncture of skin into bone carrying pathogen
  Contiguous spread
    Skin infection to blood to spine to lung
  Metaphysis=most metabolically active part of bone
**Osteomyelitis**

**Osteomyelitis** = bacterial infection of bone

Entry sites/infection (Leading to hematogenous spread)-how infection spreads in body
- URT (respiratory)
- UTI (genital urinary)
- Dermatitis may present problem for diabetics

Direct infection
- Does occur but not as often
- Acts as event driven

**Contiguous spread** = direct outspreading

Post surgical complication - inadvertent introduction of organism

**Osteomyelitis** = big problem w/ orthopedic surgery

Higher concentration b/t 2 - 12

No definitive predisposition

Prone to show up in metaphysis

**Sequestrum** = remaining dead bone in middle of infection

**Involucrum** = bodies attempt to fully contain infection site (a prominent periosteal response)

**Draining sinus** = release area for pressurized system (bacterial pressure my extrude from bone and form a sinus or ulcer to drain the infected bone)

May form cloaca on skin

**Cloaca** = end point for a draining sinus formed along the skin which oozes material from osteomyelitis infection

Margolin’s ulcer = degenerative form of cloaca (area of may turn into squamous cell carcinoma)

Rapid increase in pressure within infection site decreases blood flow to the area leading to necrosis of the bone

**Film: lateral Skull View**

- Showing increased retropharyngeal space
  - Diff Dx for soft tissue swelling/missing bone
    - Trauma (blood)
    - Tumors (cells)
    - Infection (pus)

**Lateral cervical**

- Destruction of C3 body, C4 upper body
- Crosses multiple joints

Dx: Osteomyelitis

**Film: Sagittal MRI**

- Previous multilevel Decompressive Laminectomy (L5-L1)
- Wound isn’t taken care of well
- Orthopedist doesn’t answer calls
- Weeping wound seen at imaging center

Dx: Osteomyelitis

IV antibiotics used for Tx

**50% of bone has to be missing for plain film to see it**

**Film: lateral knee**

- Sail sign in periosteum
- Lifted off of bone

Secondary film is bone scan showing increased uptake

**Film: AP thoracic**

- Widened periosteal stripe along vertebra

Dx:
- Blood
- Cells
- Pus
Film: tomogram
   Sorry I can’t tell what we are looking at
   Diff Dx:
   Diskitis
   Septicemia

Film: AP pelvis
   Pubic symphysis is destroyed!
   Pain generator is SI sourced

Film: AP shoulder
   Moth eaten bone (pin holes)
   Could be either tumor or infection
   Infection=over a couple of weeks
   Tumor=months/years

Kids vs adults
   Kids get sicker quicker
   Adults have more WBCs
   Adults have larger volume of blood
   Adults=insidious onset
   Kids=sudden onset

Signs
   Rubor
   Tumor
   Dolor
   Calor

ESR elevation early in kids, late in adults
   Adults have greater ability due to greater exposure to pathogens previously
Localized lucency=very bad thing
Widened vertebral stripe over multiple levels
  Schmorl's node will typically just be 1 level at a time
  Since on multiple levels=infection
Film: AP shoulder
  Lots of mixed density soft tissue swelling evident
  Soft tissue w/air bubbles
  Lytic destruction and widened spacing b/t AC joint
  Absence of distal clavicle
Film: AP Foot
  2nd toe missing
  Diabetics lose toes quite often
  Ulceration on foot lead to osteomyelitis
Film: Bone Scan
  Osteoblastic activity seen in right foot and left shoulder
  Crossing joint spaces=osteomyelitis
  Residual T99 seen in bladder & kidney
Film: photo of legs w/ red spots from vascular insufficiency
Film: AP knee
  Open physis-Child
  Film shows no deformity
  Severe symptomatology which doesn’t always show on film
  Not enough bone change to see on plain film
    Only after 50% change from normal do we see radiographic findings
Bone Scan (same patient)
  Soft tissue phase shows increased uptake on 1 leg vs another
  Bone phase shows increased uptake seen on 1 tibia
  The speed of the infection will cause the patient distress before it shows up on plain film
Film: comparison of R and L lower extremity
  One side has normal contour of the gastrocnemius and soleus
  The opposite side has swelling where the gastrocnemius and soleus should be tapered
Film: AP foot
  DM patient has loss of neurologic Fx in lower extremity (looks
  Neurotrophic)
  Adjacent soft tissue swelling is not seen in Neurotrophic so it must include osteomyelitis
Brodie's Abscess-walled off supportive (pus forming) infection
  Classic pain presentation: most often at night (may wake pt up)
  Relieved by aspirin
  May go through the growth plate
  Intra process
  Tx: curettage (& packing if large enough)
Nidus=recognized central lucency

Film: AP knee
  Not much seen
Bone Scan
  Increased uptake in 1 tibia
MRI
  Brodie's Abscess overlying tibial tuberosity in AP view
    Bull's Eye sign (aka Target Sign)=radiographic finding associated w/ Brodie's Abscess and the appearance of the walled off bone
  Sagittal view shows abscess in posterior portion
Diff Dx (night pain relieved by aspirin)
  Brodie's Abscess
    Larger nidus
    Osteoid Osteoma
      Outside cortex but under periosteum (which adds bone)
      Nidus typically <1cm
Non-suppurative infection
No organism is able to be grown out of this
Commonly forms in psoas muscle
Act as slow moving compared to Staph Aureus
Acts faster than tumors
Destroys both end plates and can destroy vertebra often
creating a Gibbus formation

Gibbus Formation = section of Hyperkyphosis/angular deformity due to vertebral fracture
Pott’s Paraplegia = myelopathy due to compression of the cord due to compression from Gibbus Formation

Film: AP Chest
Central white area overlying heart but smaller
TB Granuloma

Film: AP Lumbar
Long vertebra = enlarged vertebral margin unilaterally w/ contralateral reduction due to TB infection (Pott’s disease)
Structural scoliosis produced by long vertebra

Film: AP and lateral left hand
Large scale destruction of proximal phalanx of 5th digit
Cortical margin destroyed in multiple locations
Large scale soft tissue swelling
History tells us slow onset (non-suppurative)

dx: TB Dactylitis

Film: AP pelvis
Left femur head is pushed up to S2 level in the ileum
TB is present in left femur head but was not as weak

Carpal count is approximate to age for 1st 8 years

Syphilitic

Film: AP lower extremity
Bilateral solid periosteal reaction = hallmark sign of syphilis
Since this is a child it is likely a congenital infection

Congenital syphilis
Transfers occurs in utero
Stats: 25-50 out of 100 die before birth and the rest have continued health problems
Bilateral symmetrical
Decreased integrity of tibia

Wimberger Sign of Syphilis = medial tibial erosion
Wimberger Sign of Scurvy = bright, white ring around the epiphysis
Saber Shin Deformity = softening of the bone w/ anterior bowing and brightened periosteal reaction

Film: AP lower extremity
Syndesmosis b/t Tibia and Fibula is thinned due to syphilis

Fungal Infection
4-we will discuss later
Madura foot = Madura mycosis
Finishing up infections

Fungal infections (4)

Case study
- 52 year old female w/ neck pain and stiffness
- History of headaches and dysphasia
- Ortho exam: decreased ROM, para-cervical mass effect
- Neurologic: dysphagia, dysphasia
  
  **Dysphasia = disordered thinking**

Radiographic Findings
- Huge retropharyngeal space (blood, pus, cells)
- Destruction of C5/C6 bodies halfway done

**Diff Dx: local bone loss**
- Tumor
- Infection-obliterates disc spaces

9 out of 10 osteomyelitis infections = **staph aureus**
- TB #2 (happens slowly)
- Fungal infection turned out to be **Blastomycosis**

Coccidioidomycosis
- **Initial infection in lung**
- If unsure of what infection then culture
- Mostly found in SW United States
- **Film: AP ankle**
  - Focal bone loss

Histoplasmosis
- May be lung, liver, spleen lesion
  - **Never seen as bone lesion**

Madura Foot (maduramycosis)
- **Tends to target foot 1st**
### Table 16-1 Marchiori

#### Table patterns of Osteopenia

<table>
<thead>
<tr>
<th>Definition</th>
<th>Classic examples</th>
</tr>
</thead>
</table>
| **Generalized** | Osteopenia affecting the majority of the skeleton | Senile osteoporosis  
| | | Post-menopausal  
| | | Hyperparathyroidism  
| | | Cushing's disease  
| | | Wide spread malignant disease (metastasis, multiple myeloma) |
| **Regionalized** | Osteopenia affecting one limb or section of the body | Disuse arthropathy (immobilization)  
| | | Reflex sympathetic dystrophy  
| | | Transient regional osteoporosis  
| | | Regional migratory osteoporosis |
| **Localized** | Focal osteopenia in one or multiple discrete portions of bone | Lytic metastasis  
| | | Osteomyelitis  
| | | Inflammatory arthritides |

#### Generalized

- **Senile osteoporosis**: not enough osteoclasts due to advanced age  
  - Fluoride therapy: possible therapy to increase bone density
    - Doesn’t mean good bone but lots of it  
    - Absorbs X-rays well but has increased risk of fracture
  - **Hyperparathyroidism**  
    - Fairly clinically silent  
    - Will have greater incidence than expected
  - **Cushing's disease**  
    - Interference of bone creation
  - **Wide spread malignant disease**  
    - Lytic patterns may seem like generalized osteopenia

#### Regionalized

- **Disuse atrophy**: immobilization decreases piezoelectric effect and decreased bone production  
  - May be decreased by working contralateral joint (up to 25%)  
  - Use muscles therapy and progressive loading to keep muscles from atrophying
  - **Reflex sympathetic dystrophy (RSD)**  
    - Relatively frequent (distant 2nd place from disuse atrophy)  
    - Related to injury of sympathetic nerve can be a painful cause of regional osteoporosis
  - **Clinical indications**  
    - Loss of hair  
    - Sensitivity of skin (touch/cold)  
    - Decreased skin tone  
    - Prior trauma  
    - Chronic deep bone ache
  - **Transient regional osteoporosis**  
    - Idiopathic, likes the hip  
    - Shows up and goes away w/out warning  
    - Preserve shape by decreasing mechanical load
  - **Regional migratory osteoporosis**  
    - Moves around  
    - Can’t predict where it will show up next

#### Localized

- Go through benign vs aggressive list to rule out inflammatory arthritides  
  - **Lytic metastasis**  
  - **Osteomyelitis**  
  - **Inflammatory arthritides**  
    - 1st stage of ankylosing spondylitis (Romanus lesion)
Pseudowidening of SI joint

Patterns of osteopenia

Table 14.4 in Yochum and Rowe

A=Normal vertebral body
   Screen door like pattern of trabeculae

B=Osteoporosis
   Thinner cortical margin
   Accentuation of vertical trabeculae
   Osteoblasts are following forces applied to them
   Vertebral body shape is retained
   Approaches fracture threshold (reorganization and decreased strength)

C=Wedge shaped fracture
   May be related to trauma
   Intrinsic load applied causing wedged shaped vertebra
   **Wedge shaped vertebra definitive factors**
   Shape=taller in back, shorter in front
   Recognize that posterior vertebral body height has maintained at least 80% of former height (loss of <20% of height)
   Typically in T11-L1
   Called a benign fracture
   Rule out breast cancer and lung disease which may metastize to spine

D=Vertebra plana
   Loss of height on anterior and interior side
   Roots in bone pathology
   Worry about lytic metastasis

E=Biconcave (fish vertebra)
   Deepening of sup/inf end plates
   No trabeculae seen

F=Angular endplate deformity (Check sign)
   Statistically associated w/ aggressive bone disease
   Must be investigated for underlying bone pathology

Qualitative vs quantitative assessment

- Tops of femur are seen on L/s spine and F/s series
- When skeleton demonstrates general osteopenia, not all bone demonstrates the same percentage of decreased bone density
- Other factors that determine bone density are not the same

**SEXA scanner** (Single Energy X-ray Absorbtometry)
- Measures density

**DEXA scanner** (Dual Energy X-ray Absorbtometry)
- Measures bone density and quality
  - Can only assess the forearm (doesn’t always show what is happening in the rest of the body)
  - Some models can do T-L junction, lumbar spine, femur head
  - 30% mortality w/ hip fractures

**Ward’s triangle** = area b/t vertical trabecular bundles from head of femur inferior femoral neck (1), lateral diagonal trabecular bundles from inferior/medial femoral neck to greater trochanter (2), and medial diagonal trabecular bundles from medial femoral head to inferior lateral portion of greater trochanter (3)

1=principle compression group
   - Receive weight from pelvis translating to medial cortex
2=secondary compressive group
   - Inter-trochanter region compressed by muscle contraction
3=principle tensile group
   - Has suspension function
   - Fibers intermingle w/ principle compression group and translates compression to lateral cortical bone

If Ward’s triangle is small then bone is small
If Ward’s triangle is large due to retreat of trabecular bundles
If Ward’s triangle is not complete then we are at or near fracture

**Quantitative CT** may be done using CT machine to sample bone density by comparing Hounsfield unit assignments to known values

0 score is average for age group

35 is considered the key age for osteoporosis

Females

- Lose .1-.3% bone each year 35-peri-menopause
- After peri-menopause>1-3% per year linearly

**Estimating your chances of developing osteoporosis**

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Category</th>
<th>Points</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Your age</td>
<td>Under 35</td>
<td>0</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>Over 35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Your sex</td>
<td>Male</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>25</td>
<td></td>
</tr>
</tbody>
</table>

Dx Imaging Page 8
Men only lose .1-.3% bone per year
Race
Has been debunked (only 1 small group study)
Body type
Farm girlfriends-hard working/non-sedentary-no risk
Small boned +25
Sender +25
Family history
Genetic role

Many additional risks factors are for women only

Diary problems=counteract w/ supplementation, exercise

We should try to slow/stop loss
You can almost not restore lost bone
Supplement w/ calcium alone=no change
Supplement w/ D alone=non statistically sig reduction in loss
Supplement w/ Ca and D alone=stat sig reduction of bone loss
Exercise=decrease in rate of loss
Exercise + D + Ca=greatest non-drug reduction in loss
Conservative care home run!
Activity that constitutes mechanical loading
Used for people at or below fracture threshold

Good inclusion criteria/bad exclusion criteria

<table>
<thead>
<tr>
<th>Race</th>
<th>Black</th>
<th>Caucasian</th>
<th>Oriental</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>25</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Body type</th>
<th>Large boned</th>
<th>Small boned</th>
<th>Average/Weighted</th>
<th>Slender</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>25</td>
<td>0</td>
<td>25</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Family history</th>
<th>No family history of osteoporosis</th>
<th>Mother, grandmothers, or sisters with osteoporosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>25</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Additional Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>If you have never been pregnant</td>
</tr>
<tr>
<td>If you have experienced menopause</td>
</tr>
<tr>
<td>If you have had your ovaries surgically removed</td>
</tr>
<tr>
<td>If you breast fed your child/children</td>
</tr>
<tr>
<td>If you are allergic to milk/dairy products</td>
</tr>
<tr>
<td>If you are inactive</td>
</tr>
<tr>
<td>If your daily routine is stressful</td>
</tr>
<tr>
<td>If you smoke</td>
</tr>
<tr>
<td>If you consume large amounts of caffeine</td>
</tr>
<tr>
<td>If you drink alcoholic beverages</td>
</tr>
</tbody>
</table>

**Total _____**

<table>
<thead>
<tr>
<th>If score is</th>
<th>Your category is</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 85</td>
<td>Lowest risk</td>
</tr>
<tr>
<td>85-175</td>
<td>Medium risk</td>
</tr>
<tr>
<td>175-250</td>
<td>High risk</td>
</tr>
<tr>
<td>Above 250</td>
<td>Highest risk</td>
</tr>
</tbody>
</table>
Film: lateral lumbar
Codfish vertebra seen w/ increased vertical trabeculae (actually horizontal fibers are gone so they seem increased)
Limbus vertebrae
Looks like hemangioma (typically seen only on 1 level)

Film: circle shot lateral
Wedged vertebra
Good posterior height seen
Benign pattern of collapse
T11-L1+65+ female=good
No Breast/lung cancer=non cancerous

Film: lateral lumbar
Vertical trabeculae
Codfish vertebra seen at L4
Cupids bow likely at L5

Film: Lateral lumbar
No trabeculae seen inside vertebra
Inner body density is approximately equal to soft tissue density
Put finger over cortical line to see

Film: lateral thoracic
Hyperkyphosis
Compression fracture (wedge)
Very little difference b/t body density and ST density

Film: lateral thoracic
Overlying aorta shows some density over anterior part of bodies
Almost no bone density seen (about the same density as lungs)

Film: right hand AP
Looks like generalized osteopenia but shouldn’t occur in the hand due to plenty of use
Regionalized osteopenia—all the carpals, metacarpals, distal wrist
Reflex sympathetic dystrophy
Raynaud’s phenomenon

Film: AP wrist film
Very little bone density seen
Previous fracture of distal radius
Metabolic, endocrine, and nutrition category

Dr. Kuhn wrote the metabolic, Nutrition, endocrine chapters in Marchiori!!!

RSD
Fracture or surgery may prove to be trigger fracture

Film: AP Hand
Same film as last time
Looking at how thin the cortical margins are compared to Reflex Sympathetic Dystrophy

Film: AP Wrist
Obvious fracture leading to RSD

Foot films in Marchiori

Film: AP lower extremity
Bowed femurs
Ragged metaphyseal margin
**Dx: Rickets**
Hypovitaminosis (low Vitamin D)
ZPC doesn’t form in Rickets
Rickets rarefies the ZPC
Underlying bone may not support teeth so tooth loss may occur

Film: AP chest
Spatulated anterior rib
Costochondral appears like string of rosary beads ()

Film: lat chest
Rosary bead Costochondral appearance
Arteriosclerosis seen on multiple fingers
Dx: Hyperparathyroidism

Hyperparathyroidism
Arteriosclerosis
Hypercalcuria results from Hypercalcemia
Generalized hypocalcaemia
3 types
Primary
  Tumor
Secondary
  Starts off as renal disease
  Early stages of renal failure (diuresis)
  Kidney is wasting Ca so the parathyroid gland enters excessive production to keep up with the rapid loss of Ca
Tertiary
  Oliguria = reduced urinary output (seen in late kidney disease)
  Seen in late kidney disease and may be associated w/ dialysis
Rugger Jersey = hyper parathyroidism
Sandwich vertebra = Osteopetrosis
Film: Lateral Spine

Scurvy
  Scurvy sclerosis the ZPC
  Weinberger’s sign of Scurvy
  Change at ZPC (Zone of Provisional Calcification)
  Bleeding gums is large sign
  Scurbutic rosary bead appearance

*Weinberger’s sign of Syphilis* Remember
Sandwich vertebra=Osteopetrosis
Film: Lateral Spine

Scurvy

Scurvy sclerosis the ZPC

**Weinberger’s sign of Scurvy**

- Change at ZPC (Zone of Provisional Calcification)
- Bleeding gums is large sign
- Scurbutic rosary bead appearance

*Weinberger's sign of Syphilis* Remember
Film: lateral thoracic spin
- Rugger Jersey spine/Sandwich vertebra
- Could be Osteopetrosis or Hyperparathyroidism
  - Differentiate w/ lab findings
  - Anemia=Osteopetrosis
  - Increased serum Ca=hyperparathyroidism

Film: AP hand view
- Acroosteolysis (along radial side of metacarpals and phalanges noted
  - Is also seen in Scleroderma but cannot be b/c no soft tissue changes
  - Also seen in Pyknodysostosis
- Hand films are the most reliable films to see hyperparathyroidism

Film: B/L hands
- Wider than normal bone width
- Sclerosed cortical margin
  - Acromegaly=elevation of growth hormone but growth plates are closed
  - Intramembranous ossification is where we are seeing changes
  - Length/width ratio
  - Hand will appear wide/squatty
  - May have decreased joint spaces due to ligamentous limitation leading to early onset DJD

Film: lateral foot
- Bone spurring seen on anterior portion of the calcaneus
  - Dx: Acromegaly
  - Findings: increased heel pad thickness
    - Hypertrophic change on calcaneus at Achilles insertion and plantar aponeurosis insertion

Film: lateral skull
- Findings: double floor sign
  - Dx: pituitary tumor causing increased growth hormone production leading to Acromegaly
    - Sella turcica measurement has great specificity but poor Pituitary tumor may cause distortion of sella turcica into a J shape

Film: lateral skull
- Dx: Acromegaly
  - Lantern jaw (Aka prognathism)=Elongation and thickening of jawbone
    - Loss of teeth are seen

Film: lateral chest
- That is a terrible view of anything
  - Long term use of corticosteroids
  - Hyperkyphosis doesn't begin to describe this
    - This person’s head is below their mid thoracic spine
    - They are freaking bent at 90 degrees

Heavy metal intoxication
- Lead poisoning
  - Netter slide of toxicity
    - Basophilic stippling=dots w/in RBCs caused by lead poisoning
  - Increased ZPC width (Lead intoxication)
Increased whiteness and thickness (maybe double layer)
ZPCs

Film: Skull View
Thickening along margins of sutures due to lead incorporation

Histiocytosis X
Eosinophilic granuloma
If we don’t make the situation worse then they will recover completely
Should not have recurrent attacks
Not a brittle bone disease but decreased strength is present
Film: lateral thoracic
Spontaneous collapse of vertebral body
Silver dollar sign=looks like silver dollar on its side
Maintenance of disc space above and below
Film: AP pelvis
Widened portion of femoral diaphysis w/ lucency seen
Disappearing bone=bone will feel and function pretty normal
Film: lower extremity
???Aunt mini lesion=disappearing bone that reappears??
Film: lateral cervical
Loss of space b/t C3/4
Loss of cortical margin (local lucency)
C4’s body is messed up
Do we have disc space?
Could be infection
Needle aspiration will show granulation tissue and eosinophils

Hemochromatosis
Film: PA left hand
Hook on distal end of 3rd metacarpal (may show up on other metacarpals/metatarsals as well)
Strongly associated w/ hemochromatosis
Finding: non-osteophytic growths

Tumors
Category of tumors
Metastasis=starts somewhere else and moves to bone, Most common aggressive tumor to skeleton
Osteosarcoma
Ewing’s sarcoma
Multiple myeloma=most common primary tumor to skeleton
Benign=longest list (know it, ID it, forget it)
Hemangioma=Most common primary benign tumor to spine
Tumor-like
Padgett’s great imitator
Will be tested on classic form
Fibrous dysplasia - great imitator
Will be tested on classic form

Metastasis

Table 11.4 in Yochum (radiologic presentation of metastatic carcinoma)
Bone doesn’t have a wide response range
Lytic = goes away
Blastic = builds up
Combo of lytic and blastic

Blastic metastasis = Prostate, carcinoid, lymphoma (only 3 that don’t reliably do lysis)

Table 11.5 radiologic features of metastatic carcinoma to bone

General
Axial skeleton predilection
Multiple sites
Osteolytic mets 75%
Osteoblastic 15%
Others 10%

Table 11.6
2:1 ratio of metastasis to bone: tumor in bone
Never have joint involvement (that will be infection)

Statistically high areas of metastatic distribution
Skull
Shoulder
Ribs
Spine
Pelvis
Proximal femur

Nothing beyond knee or elbow

Batson’s Plexus
More like portal vein system than systemic veins
NO VALVES!!!
Venous plexus that traverses from the proximal femur, pelvis, along the spine, ribs, shoulders, and skull
Pretty much the same site of high areas of metastatic distribution

Most common primary sites that metastasize to skeleton
Lung
Breast
Prostate
Kidney

Most common primary aggressive = multiple myeloma has negative bone scan

Create a most common list (good for part 2 national board, also fun Dx and positioning)

Film: AP lower cervical view
Big soft tissue in front of vertebral body
Diff Dx: blood, pus, cells
Behind soft tissue mass we are missing a vertebral body = lysis
Top 2 sites males = lung and kidney
Top 2 sites females = breast and lung
Film: lateral C spine
- Local lucency
- Doesn’t cross the joint space (not infection)
- Enlargement of pre-vertebral space
  - Blood - moves up and down the facial planes
  - Puss - not an infection
  - Cells

Differential of lytic stuff
- Female
  - Lung
  - Breast
- Male
  - Lung
  - Kidney

Film: lateral lumbar
- **Check sign=angular end plate deformity**
  - Think pathology 1st until proven otherwise

Film: lateral lumbar
- Decreased disc space L3-L4 and check sign seen
- No subchondral sclerosis

Film: AP Lumbar
- Winking owl sign=missing pedicle

Film: lateral c/s
- Local lucency
- Lytic mets is 1st thought

Film: lateral L/s
- Lumbar body is not concave in anterior portion
- There is a S-shape to the front of the vertebra
- Associated bone scan
  - + result seen
- 3 weeks later another lateral lumbar shows huge check sign and further distortion (compression deformity)

Film: AP lumbar
- Missing L3 pedicle
- Lytic mets is most common cause of ‘winking owl sign’

Film: lateral thoracic
- Wedge shape fracture - must maintain 80% of pre collapse height
  - [If beyond 20% collapse=Vertebra Planar]

Film: PA shoulder
- Sometimes see pseudotumor/pseudocyst on plain film but that disappears
  - w/ external rotation
- True lytic mass will not disappear w/ rotation
- Biopsy of tumor to determine origin

Film: lateral Lumbar
- Normally L3, L4 look similar but in this case L4 looks like L5 (which is
Osteoblastic metastasis

Ivory vertebra differential

- Osteoblastic metastasis (50+ years old)
  - Women = breast
  - Males = prostate
- Padgett's disease (3rd stage) (50+ years old)
  - Thickens cortex and coarsens trabecular pattern
- Lymphoma (20-40 years old)

Film: AP pelvis

- Apparent OCI but not really
- Mass above right femur, changes in left teardrop distance
- Osteoblastic mets (breast)

Film: AP Lumbar

- White mass overlying a pedicle
- So local and bright it must be osteoblastic disease
- Could be bone island or bone metastasis
- Age will be your differentiator

Primary tumors

**Lytic skull pattern decision tree**

**Rain drop skull-multiple myeloma**
- Uniform hole sizes
- Metastasis is more common
  - Will have blood spreading of tumor emboli
  - Will not be symmetrical
- Padgett's = osteoporosis circumscripta

**Multiple myeloma**
- Will not show 'hot' on bone scan
- Inhibition of osteoblastic activity
- Want MR
- Want immunoglobulin electrophoresis

Film: AP shoulder

Dx: Multiple Myeloma
- Most aggressive patterns of bone destruction
  - Moth eaten = larger holes, uneven size
  - Permeative pattern of destruction = smaller holes w/ even sizes
- Lack of periosteal reaction
- Generalized osteopenia
Dx: Multiple Myeloma
Most aggressive patterns of bone destruction
  Moth eaten: larger holes, uneven size
  Permeative pattern of destruction: smaller holes w/ even sizes
Lack of periosteal reaction
Generalized osteopenia
Continued *Multiple Myeloma*

A primary cancer which produces uniform sized lytic lesions

*Causes suppression of osteoblastic activity (why the bone scan will be negative)*

Bence Jones protein is associated w/ Multiple Myeloma (only elevated in 40% of cases)

Use as inclusive criteria

**Immunoglobulin electrophoresis**—great test for Multiple Myeloma

May be GADEM

Biopsy is the gold standard for diagnosing Multiple Myeloma

Red marrow spaces may be taken over by tumor resulting in Normocytic, normochromic anemia (made correctly but just not enough)

**Film: lateral lumbar**

Compression deformity of L4, L3 (vertebra plana at L4)

Generalized osteopenia

Cold on bone scan

*Dx*: must be Multiple Myeloma

**Film: lateral C-spine**

C4 has bubbly expansile lesion w/ protrusion into tracheal space

Local lucency seen in body

Infection or *tumor*

**Hx**: dealing with pain for weeks to months (rules out infection)

**Diff Dx**

- Male—lung
- Female—breast/lung
- Primary bone tumor

Perform bone scan—comes back negative

MRI and biopsy performed

Histology is same as MM

MR shows only in one place

*Dx*: **Solitary Plasmacytoma**

- 30% will stay same
- 70% will progress to multiple myeloma

**Film: AP knee**

Young adult male (closure of growth plates almost finished)

Don’t think mets due to age/history

**Diff Dx:**

- Ewing’s sarcoma
  - Predominates diaphyseal area of bones
  - Hair on end periosteal reaction
  - Osteosarcoma
  - Strong association w/ metaphysis of long bones

- **Osteosarcoma**
  - Double matrix tumor (makes it great at absorbing bone)
  - #1 location=distal femur
  - #2 location=proximal tibia
  - VERY aggressive tumor

*Dx*: Osteosarcoma

**Film: AP knee**

Proximal portion of fibula is GONE!

This is the most aggressive form of Osteosarcoma
Film: AP shoulder
Destruction/lytic but doesn't cross joint space
Osteosarcoma
May present w/ brush like periosteal reaction

Codman's cuff (Codman's triangle) = laminated periosteal response that gets punctured in middle w/ ragged ends on border
Saucerization = tea cup and saucer appearance provides 3 diff
Ewing's sarcoma
Osteomyelitis
Stress fracture

Film: AP femur/knee
Only 1 Quasi-malignant tumor = Giant Cell Tumor
20% aggressive
 Mostly silent history
Youngish age range
Loves going to end of the bone (subchondral)
ID by biopsy and find giant cells in tumor

Film: AP knee
Local lucency seen above and behind the patella
Small zone of transition = more benign
Lateral view
Shows more defined outline

Padgett's will convert from a mixed phase presentation to a blastic presentation

Film: AP knee
Osteochondroma
Looks like Supracondylar process but isn't on humerus and points away from joint
"Coat-hanger exostosis"
Cartilage will be at tip
Similar sessile lesion
Fracture = most common complication
May also present neurovascular compression
HME = Hereditary Multiple Exostosis
Looks like lots of osteochondroma
Film: lateral lumbar
**Hemangioma**=most common benign tumor of spine
May think osteoporosis but isn’t generalized
Barrel shaped vertebra (may exacerbate central canal stenosis)
Very bloody lesions w/ altered haemostatic responses (bleed out)
**Corduroy vertebra**=pattern made by increased trabecular pattern
Also predilicts to mandible
On CT the tumor is pushing the horizontal trabeculae around
See it, name it, forget it

Film: AP tibia
Diff Dx Night pain relieved by aspirin (both have nidus)
Brodie’s abscess
**Osteoid Osteoma**
Extra cortical

Shows up hot on bone scan

Film: AP hand
**Enchondroma**=most common benign appearing, tumor of the hand (foot) w/ a clear matrix
Some cortical stretch (but no destruction)
May become chondrosarcoma
Most common complication=fracture

Film: AP lower leg
**Benign fibroCortical Defect**=intercortical lucency lesion
Will usually fill in and can’t find
Clinically silent
Tx: drill holes=body will fill in

Dx Imaging Page 22
Non Fibrocortical defect
Like BCD but will not fill in over time
Clinically silent
Tx: drill holes=body will fill in

Aneurismal Bone Cyst=most likely benign tumor to cross growth plate
Like aunt mini lesion
Loves metaphysis

Intraosseous Lipoma
Most common tumor of calcaneus
Most common tumor w/ central calcified nidus
Can fracture

Padgett’s disease
Starts lytic then moves mixed then blastic
Osteoporosis circumscripita=pattern of lucency in skull
Cotton tipped skull=Sclerotic change of skull
Skull changes may lead to decrease CN function due to closing of foramen

Osteoporosis circumscripta=pattern of lucency in skull
Cotton tipped skull=Sclerotic change of skull
Skull changes may lead to decrease CN function due to closing of foramen

Bone softening disorder
May pressure erode the bone
Also present in neurofibromatosis
May pressure erode the bone

Fibrous dysplasia=never has calcium within center
Extremely irritating to periosteum and endosteum
Rind of sclerosis=irritation of periosteum and endosteum causes a rind-like sclerotic margin
Café au lae spots may be present
Also present in neurofibromatosis
Neurofibromatosis
Can push tracheal air shadow (shown on)

Film: AP cervical
Neurofibromatosis
Can push tracheal air shadow (shown on)

Film: AP chest
Café au lae spots
Coast of California appearance = smooth and consistent (fibrous dysplasia)
Coast of Maine = rough and inconsistent margin (neurofibromatosis)

Film: oblique cervical

AP Cervical-neurofibromatosis

AP Chest-neurofibromatosis

Oblique cervical-neurofibromatosis
A. Infection categories
   a. Suppurative Osteomyelitis
   b. Non Suppurative Osteomyelitis
   c. Syphilitic
   d. Mycotic

B. Most common organism associated with osteomyelitis?
   a. Staph. Aureus

C. Organism, which demonstrates a predilection for the SI, SC and Spinal Joints
   a. Pseudomonas – gets the “S Joints.” Mostly associated with drug addicts

D. Organism seen to affect humerus in newborns
   a. Streptococcus B

E. Most likely age range for osteomyelitis
   a. Typically between 2 and 12 with 3:1 male to female

F. Is osteomyelitis typically monoostotic or polysystolic
   a. Monoostotic

G. Affected population in cases of osteomyelitis
   a. Immunosuppressed
   b. Alcoholics
   c. Newborns
   d. Drug addicts – predisposed to Pseudomonas infections of the S joints

H. Is osteomyelitis a primary or secondary process

I. Where is it most likely to start?
   a. Commonly large tubular bones in extremities [femur]. Tibia humerus and radius are also favorite spots.

J. How does it spread to bone?
   a. Hematogenous
      i. Deposition into blood stream of organisms that may reach distal skeletal sites. MOST COMMON
   b. From contiguous source of infection
      i. Extend into bone from adjacent contaminated site
      ii. Cutaneous, sinus and dental infections are common sites of origin for adjacent osteomyelits
   c. Direct implantation
      i. Result of direct penetrating injuries or puncture wounds, such as those by nail, splinter or glass common on feet. Open fractures could be a source of direct implantation
   d. Postoperative infection
      i. Contamination of surgical sites

K. Define the following
   a. Sequestrum: (pg. 1374 – 1375)
      ○ When periosteal and subperiosteal are involved in an inflammatory process there is a loss of blood supply to the cortical bone rendering it necrotic. Cortical and medullary infarcts result in the formation of a sequestrum, or dead bone.
   b. Involcrum: (pg. 1374 – 1375)
During osteomyelitis as the pus from the infection lifts the periosteum; it causes a modest degree of new bone proliferation and pain. The periosteal new bone is the body’s attempt to wall off the infective process. This bony collar is often referred to as the involucrum.

c. Cloaca: (pg. 1375)
   - The occurrence of a defect that may develop in the involucrum is referred to as a cloaca. The functions of these defects is to allow the continued discharge (decompression) of inflammatory products from the bone and has been referred to empyema necessitates. These cloacae are most frequently associated with chronic osteomyelitis, which is stubborn in its response to conventional antibiotic therapy.

d. Marjolin’s ulcer: (1375)
   - One rare but significant complication of the draining sinus (cloaca) is the development of a squamous cell carcinoma within the channel of the cloaca. This ulcerative channel and malignant transformation is found only with chronic osteomyelitis and may be referred to as Marjolin’s ulcer.

e. Saucerization:

f. Brodie’s abscess: (1386 – 1391)
   - It is defined as a localized, aborted form of suppurative osteomyelitis. The abscess is depicted as an oval, elliptical, or seriginous radiolucency with no visible matrix surrounded by a halo or doughnut rim of heavy reactive sclerosis.

L. List the clinical features for the child with osteomyelitis (pg 1385)
   - Early diagnosis is important and clinical signs and symptoms precede plain film findings by 7 – 10 days in the appendicular skeleton and 21 days in the spine. Young patients present with acute systemic symptoms. The most frequently affected age range is 2 – 12 years; 3:1 males. Affects tubular bones, most commonly the femur.

M. List the clinical features for the adult with osteomyelitis: (pg. 1385)
   - It is basically the same as in the adult except adult patients present with symptoms that vary and tend to be more chronic.

N. List the radiographic features for osteomyelitis (pg. 1385)
   - Bone scans are the earliest means of diagnosis
   - Radiographic latent period for plain films: extremities 10 days, spine 21 days
   - Soft tissue alterations: elevated fat plans, obliterated fat planes, increased density, and paraspinal edema.

O. How to differentiate between infection and neoplasm
   - a. Infection respects no boundaries – therefore it crosses jt margins
   - b. Manifests visible bony changes in a short amount of time

P. Define the following
   - a. Gibbus – angular deformity, found in TB of spine
   - b. Long vertebra – altered vertebra that has become longer than it is wide due to long standing gibbus formation just above it. Only found in patients in which the vertebral growth centers were not closed at the time of infection. Found in TB of spine
   - c. Pott’s paraplegia – a rare complication of Pott’s disease. Pressure paraplegia results from collapse of vertebral bodies, extensive granulation tissue and detached sequestra from the vertebral bodies. May have sudden onset
   - d. Cold abscess – coalition of several nodes due to their walls deteriorating (p1412)
     - i. Speckled white pattern in bone and soft tissue
     - ii. No longer an active process

Q. For TB complete the following
a. Compared to the rate of progression of suppurative infections how does TB compare (table 12-3 p 1412)
   i. TB – slower progression (months)
   ii. Suppurative – quick (2 weeks)

b. Where do TB infections begin
   i. Respiratory Tract (upper lungs)

c. How do they spread
   i. Initially via inhalation or ingestion
   ii. Hematogenous spread of a primary focus in the respiratory tract leads to infection of musculoskeletal system

d. Where are resistant strains more common today?
   i. An overuse of the same antibiotics

e. Why is TB on the rise again in USA?
   i. Modern travel
   ii. Immigration of groups from areas where TB is still endemic (Prevalent in or restricted to a particular region, community, or group of people).

R. For Fungal Infections:
a. m/c fungal infection worldwide? Maduramycosis (Madura Foot): comes from direct contact to fecal matter with bare feet that have an exposed wound, very common in Africa
b. m/c fungal infection in the SW US? Coccidioidomycosis: favorite cites are bony prominences-acromian, med and lat maleoli, and patella; just needs a blood supply to the bone to cause damage
c. m/c fungal infection in the northern US? Blastomycosis: spores went airborne from a landslide and the 52 y/o woman inhaled them, she had increased prevertebral soft tissue space on her cervical x-ray. Use a methylene blue for staining of this fungus
d. m/c fungal infection in the Miss. and ohio river valley? Histoplasmosis; soft tissue fungus that does not migrate to bone

S. For Syphilis:
a. Two types: Congenital and Acquired
b. Pathomechanics: Congenital- is passed from mother to child through the placenta after the 4 month of gestation. Acquired- is seen in adults as a consequence of poor behavior; sexually transmitted
c. Key radiographic features of the m/c type-congenital
   -phase 1: Metaphysitis
     - present at birth or shortly after
     - lodging of spirochetes beneath the fetal growth plates produces a metaphysitis in which the normal vascular fountain underneath the cartilage is replaced by syphilitic granulation tissue
     - bone formation and remodeling in the zone of primary ossification is decreased or absent
     - creates radiolucent metaphyseal bands, these bands often lead to metaphyseal irregularity with fragmentation and infractures = sawtoothed appearance; often bilateral and symmetrical, m/c the upper extremities
     - knees, shoulders, and wrists are the m/c affected areas
     - often symmetrical erosive defects occur on the medial surfaces of the proximal ends of the tibiae, representing Wimberger’s sign of congenital syphilis
     - doesn’t affect cartilage
     - lesions of phase 1 heal quickly with Tx

   -phase 2: Periostitis
     - periosteum may be infiltrated by syphilitic granulation tissue, creating a solid or laminated rxn
     - the periosteal response is often diffuse and symmetrical, affecting nearly all of the
major long bones
- complete remission with Tx

-phase 3: Osteitis
- only get if have not received Tx
- extension of infectious focus
- reactive sclerosis surrounds the osteolytic lesions, with associated periostitis of the long tubular bones
- extensive periostitis and cortical overgrowth may create an undulating, dense contour to the long tubular bones
- frequently, both tibiae will be involved, creating the classic saber shin; anterior bowing of the tibia is characteristic, with osteolytic defects scattered throughout the bone

-Additional Features
- will find multiple swollen, non painful joints, called Clutton’s jts
- deformity of the teeth, creating peg-shaped, hypoplastic, and notched teeth, Hutchinson’s teeth

a. definitions of Wimberger’s sign, Saw tooth, and Saber shin are in above descriptions
1. Osteoporosis
   A. Know the 3 patterns of osteopenia and the most common causes of each
      
      1. **Generalized**: An “all over” loss of bone density, especially in the axial components of the spine, pelvis, and proximal long bones. The most common cause is postmenopausal status and aging.

      2. **Regionalized**: Loss of bone density in one region or segment of the body. Most common cause is immobilization such as after a fracture. Other causes are Sudex atrophy and transient regional osteoporosis.

      3. **Localized**: Focal losses of bone density affecting a relatively small area of bone are usually the result of local disease such as inflammatory arthritis, neoplasm, or infection.

2. **How can we assess bone density?**
   A. Quantitative CT
   B. Dual Photon Absorptiometry (I guess this is DEXA)
   C. Single Photon Absorptiometry
   D. Radiogrametry

3. **For Postmenopausal Osteoporosis**

   Clinical Features:
   A. Presentation is usually in the 5th or 6th decades in females by a ratio of 4:1
   B. Usually causes pain only when complicated by fracture and deformity, especially in the spine
   C. No lab findings useful

   Key radiographic features:
   A. increased bony radiolucency
   B. cortical thinning
   C. altered trabecular patterns
   D. fracture deformity

Table 14-3
- Decreased bone density
- Trabecular changes
  - Accentuation of primary trabeculae (pseudo-hemangiomatous appearance)
- Washed out appearance
- Cortical thinning
- Changes to vertebral shape
  - Vertebra plana (pancake vertebra or silver dollar vertebra)
  - Wedged vertebra
  - Biconcave deformities (fish vertebra)
  - Localized endplate deformities
  (chart continued)
  - Schmorl’s nodes
Review Figures 14.4, 14.10
see text pages 1501 and 1504

A. Briefly define senile osteoporosis (1497)

Reduction in bone quantity w/ the actual quality of the bone remaining normal, in this form of osteoporosis it is associated w/ old people.

B. For Reflex Dystrophy: (1508-1509)

a. Define the entity
   
   Aka: posttraumatic osteoporosis, Sudeck’s atrophy, acute bone atrophy, casalgia
   
   Def: complex regional pain syndrome to emphasize the multisys. Disturbances of the somatic, psychological, and behavioral aspects of the pt's life

b. Key clinical features

   *Characterized by an acute onset of painful regional osteoporosis, usually following trivial trauma
   *Progressive onset of pain, stiffness, swelling, and atrophy@ and distal to the site of injury over a 3-6 month period
   *Characteristic changes can be classified into 3 distinct stages: 1) acute/hyperemic 2) dystrophic/ischemic 3) atrophic
   *Recovery slow over many months, may never completely heal w/ residual atrophy, contracture, and joint stiffness

c. Key radiographic features

   *Rapidity of appearance and progression of osteoporosis
   -early: bone appears mottled
   -later: entire bone density is diminished
   *Bone scans will always be negative

d. Discuss likely etiologies

   *Reflex overactivity of the sympathetic nervous sys. That mediates trophic changes in bone and soft tissues in response to external stimulus
   *Hyperemia of bone augments osteoclastic resorption, which rapidly demineralizes the involved skeletal structures.

C. Define disuse and immobilization osteoporosis (1509)

*Regional osteoporosis resulting from traumatic injuries that are immobilized, motor paralysis, and inflammatory lesions of bones and joints
   i. immobilization inhibits osteoblastic activity while osteoclastic-mediated bone resorption is accelerated

*Four patterns of osteoporosis: uniform, spotty, bands, cortical/scalloping

7. Osteomalacia

   a. Defined = bone softening, abnormal (K-Mart) bone; the skeletal endpoint of many, many different diseases
      i. Osteopenia = decreased bone density (just a quantitative description)
      ii. Osteoporosis = normal bone, but less of it (qualitative and quantitative description)

   b. M/C examples (table 14.4)
      i. Deficiency – Vit D, calcium, phosphorus, dietary chelators
      ii. Absorption – gastric abnormalities, biliary diseases, enteric malabsorption
iii. **Renal tubular** – proximal tubular lesions, proximal and distal tubular lesions, distal tubular lesions (primary and secondary)

iv. **Renal osteodystrophy**

v. **Unusual forms and associations** – fibrous dysplasia, neurofibromatosis, neoplasm, anticonvulsant drugs (Dilantin), hypophosphatemia

c. **Radiographic features**
   i. Decreases bone density – 2º to diminished bone mineral content
   ii. Coarsened trabecular pattern – from overall loss of bony trabeculae
   iii. Loss of cortical definition – thinner and altered in structure
   iv. Pseudo-fractures – bilateral and symmetrical linear radioluencies; Paget’s fibrous dysplasia, rickets, hyperphosphatemia
   v. Deformities – (in weight bearing bones) protrusio acetabuli, bowing of femur and tibia

d. **Common complications** – fracture from trivial trauma, progressive bone deformities

8. **Rickets** = systemic osteomalacia in an infant or young child
   a. **Cause** – deficiency in Vit D, phosphorous, calcium
      i. **In the past** – primary
      ii. **Today** – renal rickets or secondary hyperparathyroidism
   b. **Clinical features** – short in stature, bowed long bones
   c. **Radiographic features** – “rickets rarifies the ZPC,” rachitic rosy (bulbous spade-like ribs)
   d. **Treatment** – Vit D, sunlight exposure

9. **Scurvy** = Barlow’s Disease, hypovitaminosis C
   a. **Cause** – long-term deficiency of Vit C
   b. **Clinical features** – petechiae, bleeding gums, melena, hematuria
   c. **Radiographic features** – “scurvy scleroses the ZPC,” scorbutic rosy (bowed, spade-like ribs), generalized osteopenia, Wimberger’s sign (ring epiphysis)
   d. **Treatment** – Vit C therapy

D. **For osteomalacia: (1511-1514)**

   a. **Define the term**
      metabolic disorder that alters the quality of bone
      -lack of calcium salts being deposited in osteoid tissue
      -“soft bones”

   b. **List the most common examples (table 14.4)**
      **Deficiency:**
      *Vit. D
      *Calcium
      *Phosphorous
      *Dietary chelators
      **Absorption:**
      *Gastric abnormalities
      *Biliary Ds.
      *Enteric malabsorption
      **Renal tubular:**
      *Proximal tubular lesions
      *Proximal and distal tubular lesions
      *Distal tubular lesions (tubular acidosis), primary and secondary
      **Renal Osteodystrophy**
      **Unusual forms and associations:**
      *Fibrous dysplasia
      *Neurofibromatosis
*Anticonvulsant Rx (Dilantin)
*Hypophosphatasia

c. Radiographic features
d. Common complications

E. Define rickets
   a. Causes
   b. Clinical features
   c. Radiographic features
   d. Treatment

F. Define Scurvy (Barlow’s disease, Hypervitaminosis C) – Pg 1516-1517
   a. Cause
      Vitamin C deficiency results in impaired collagen synthesis. Must be at least 4
      months of avitaminosis before symptoms and skeletal changes become
      apparent. The typical pathological manifestations of vitamin C deficiency are
      noted in dentine, osteoid, and capillary vessel wall tissues. Pathological
      changes are a function of the rate of growth of the affected tissues; hence, the
      bone changes are often observed only in infants during periods of rapid bone
      growth.
      Initial symptoms are nonspecific and include the following: Loss of appetite,
      Peevishness, Poor weight gain, Diarrhea, Tachypnea, Fever
      Specific symptoms include the following: Irritability, Pain and tenderness of the
      legs, Pseudoparalysis, Swelling over the long bones, Hemorrhage.
   b. Clinical features
      Hypovitaminosis C which affects mainly infants fed solely on pasteurized milk.
      Latent period of months
      Usually age 8-14
      Spontaneous hemorrhages, swelling, irritability, pain, lying motionless (frog-Legged), and costal rosary.
      Serum ascorbic acid < 0.6mg/100ml
   c. Radiographic features
      Osteopenia
      Dense Zone of provisional calcification
      Ring epiphysis (Winberger’s sign)
      Corner (angle) sign
      Pelken’s spur
      Scorbutive zone (Trummerfeld’s zone)
      Subperiosteal hemorrhage
   d. Treatment
      Vitamin C therapy – all changes are reversible although Frankel’s line (dense
      zone of provisional calcification) may remain. Adequate diet
      http://www.wrongdiagnosis.com/treat/diet_changes.htm - sources include citrus
      fruits (lemons, limes, oranges), Berries, Capsicum, Parsley, Pawpaw, Leafy
      green vegetables

G. Hyperparathyroidism [HPT] – Pg 1517-1523
   This condition of excessive calcium in the blood, called hypercalcemia, is what usually
   signals the doctor that something may be wrong with the parathyroid glands. In 85
   percent of people with this disorder, a benign tumor (adenoma) has formed on one of the
   parathyroid glands, causing it to become overactive. In most other cases, the excess
   hormone comes from two or more enlarged parathyroid glands, a condition called
   hyperplasia. Very rarely, hyperparathyroidism is caused by cancer of a parathyroid gland.
a. Define 3 forms of HPT
1. Primary hyperparathyroidism: elevated parathormone stimulates osteoblastic resorption, liberating calcium and phosphorus into the bloodstream. Phosphorus is more readily excreted and, owing to the constant calcium-phosphorus product, calcium is retained disturbing the homeostasis. The net result is hypercalcemia and hypophosphatemia. This is the most common cause of hypercalcemia and may be owing to parathyroid adenoma, carcinoma, hyperplasia or ectopic tumors producing parathormone type substances. Characteristically there are elevated levels of parathormone, hypercalcemia, and hypophosphatemia.
2. Secondary hyperparathyroidism: a combination of calcium loss and abnormal renal vitamin D formation creates continuous hypocalcemia and increase the release of parathormone and bone resorption. Complication of chronic renal disease, allowing for persistent loss of calcium and phosphorus thus stimulating the parathormone release.
3. Tertiary hyperparathyroidism: the parathormone gland acting independently of serum calcium levels (from dialysis/renal failure)

b. Clinical features
Affects women 3:1
Clinical profile of women 30-50 y/o with weakness, lethargy, polydipsia, and polyuria
Weak and hypotonic muscles
Kidneys Calculus formation (maybe the reason why patient presents for examination)
Bone tenderness
Increase alkaline phosphatase level in the presence of bone disease
Elevated parathormone concentration
c. Radiographic features
Subperiosteal resorption
Subarticular resorption and associated vascular calcification
Widened joint space and irregular joint margins
Brown tumors and chondrocalcinosis
Soft tissue calcification
Classic salt and pepper appearance of the skull
Rugger Jersey Spine
Target Sites:
Hand: subperiosteal resorption, radial margins of proximal and middle phalanges of the 2nd and 3rd digits, w/ acro-osteolysis
Skull: salt and pepper, resorption of lamina dura
Spine: osteopenia, trabecular accentuation, endplate concavities, rugger jersey spine, widened sacroiliac joints, DRSA manifesting as loss of endplate, vertebral body destruction, and decreases disc height.
DRSA – Dialysis related spondyloarthropathy
d. Treatment
Surgery to remove the enlarged gland (or glands) is the only treatment for the disorder and cures it in 95 percent of cases. Patients who are symptom-free, whose blood calcium is only slightly elevated, and whose kidneys and bones are normal, may wish to talk to their doctor about long-term monitoring. Biphosphonates are currently used to inactivate osteoblastic activity.
Estrogen therapy may be beneficial in postmenopausal women to reduce the severity of osteoporosis.

11.) ACROMEGALY
   a.) Cause: Increased growth hormone in skeletally mature person
   b.) Clinical Features –
       • thick joint – hip, knee, spine
       • facial: malocclusion, prominent forehead, thickened tongue, broad, large forehead
       • thickened skin on hands
   c.) Radiographic Features
       • Heel sign – heel pad > 20 mm
       • Skull – sella turcica enlargement, sinus overgrowth, malocclusion
       • Hand and foot – widened shafts, bony protuberances, enlarged distal tufts (spade-like), widened jt. Spaces
       • Spine – platyspondyl, hyperostoses, widened disc and facet spaces, posterior body scalloping, widened ADI

I. Heavy metal intoxication
   a. Clinical features
      Abdominal pain
      Encephalopathy
      Disturbances of the nervous system
   b. Radiologic features
      Linear, transverse densities at the metaphyses (lead lines)/ radiodense metaphyseal bands
      Remodeling abnormalities

J. Histiocytosis X-Focus on eosinophilic granuloma aka: Langerhans cell histiocytosis LCH
   a. Cause
      It is of unknown origin but the hallmark of the disease is an abnormal proliferation of reticulo-endothelial cells, predominantly the histiocyte from which the disease derives it’s name.
      (intense proliferation of reticulohistiocytic cells)
      3 Types
   b. Clinical features
      Eosinophilic Granuloma: 60-80% of Histiocytosis X, age 5 - 10 yrs, presentation: bone pain, local swelling, irritability,
      Bones: 50 - 75% solitary / monostotic, skull/mandible (50%): "punched-out" lucencies, "hole within a hole", "button sequestrum", "floating teeth", spine/pelvis (25%): vertebra plana http://chorus.rad.mcw.edu/doc/00848.html (most common pediatric cause), long bones (15%): medullary lucency +/- thin sclerotic rim
      Lungs: involved in <10%, signals worse prognosis, apical reticulonodular infiltrates, honeycomb lung.
   c. Radiographic features
      Letterer-Siwe disease: Skeletal lesions are infrequent; lytic lesions in the calvaria. Uncommon long bone lesions stimulate Ewing’s sarcoma
Hand-Schuller-Christian Disease: Polyostotic destructive foci in and immature skeleton with lesions occurring anywhere particularly in the skull, pelvis and long bone. Lesion as a wide spectrum of appearances from benign geographic from to a permeative, cortex destroying malignant process. Lesions may consist of multiple lytic defects involving the entire bone from the diaphysis to the metaphysis. Coalescence creates larger defects with bevel cortex, producing the whole-within-hole appearance.

Eosinophilic Granuloma:
Study Guide for Tumors

Tumor material is divided between Metastasis, Primary Tumors, Secondary Tumors, and Tumor-like conditions.

Must review Tables: 7.5, 11.1, 11.2, 11.4, 11.5, 11.6, 11.9, 11.10, 11.11
Must review figures: 11.2, 11.3
Look in Normal variance packet it is also very good.

1. Metastasis:
   a. Define
      i. Metastatic bone tumors are the most common malignant tumors of the skeleton.
      ii. 70% are malignant tumors are metastatic in origin
      iii. 30% are primary in nature
      iv. Most malignant tumors of bone are metastases from a primary extraskeletal focus, the majority are epithelial in origin

| Table 11.1 Overview of Common Malignant Bone Lesions |
|------------------------|------------------------|
| **Primary (30%)**      | **Secondary 70%**      |
| Multiple Myeloma (m/c) | Lytic 75%              |
| Osteosarcoma (second m/c) | Blastic 15%          |
| Chondrosarcoma (third m/c) | Mixed 10%            |
| Ewing’s Sarcoma (fourth m/c) |                   |

| Table 11.2 Most Common Causes for Osseous Metastases |
|------------------------|------------------------|
| **Population**         | **Lytic**             |
| **Blastic**            |                        |
| Female                 | Breast (80%)          |
|                        | Breast (10%)          |
| Male                   | Lung (75%)            |
|                        | Prostate (80%)        |
| Young (<20 years)      | Neuroblastoma (80%)   |
|                        | Hodgkin’s (50%)       |

<table>
<thead>
<tr>
<th>Table 11-5</th>
<th>Radiologic Features of Metastatic Carcinoma to Bone</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td></td>
</tr>
<tr>
<td>Axial skeleton predilection</td>
<td></td>
</tr>
<tr>
<td>Multiple sites</td>
<td></td>
</tr>
<tr>
<td>Osteolytic metastases (75%)</td>
<td></td>
</tr>
<tr>
<td>Cortical and trabecular destruction</td>
<td></td>
</tr>
<tr>
<td>Lack of periosteal response</td>
<td></td>
</tr>
<tr>
<td>Moth-eaten, percutative destruction</td>
<td></td>
</tr>
<tr>
<td>Small or absent soft tissue mass</td>
<td></td>
</tr>
</tbody>
</table>
b. Most common primary sites of cancer associated with metastasis to bone are: **Breast, Lung, Prostate, Kidney, Thyroid, and Bowel.**

c. **Key Clinical Features**
   i. **Age:**
      1. Most patients presenting with skeletal metastases are in their second half of life, M/C past the 4th decade
      2. Children <5 yoa caused by **Neuroblastoma**
      3. 10-20 yoa caused by **Ewing’s sarcoma and Osteosarcoma**
      4. 20-35 yoa caused by **Hodgkin’s lymphoma**
   
   ii. **Appearance at presentation:**
      1. Most patients present with a history of **recent weight loss, appear cachectic, and experience anemia and fever** in advanced stages of the disease
      2. Secondary skeletal deposits create the first symptoms of the carcinomatous process. Common w/ carcinoma of the thyroid, liver, and kidney
      3. Sign and symptoms are pain and pathologic fracture. Pain is insidious onset w/ bouts of remission and exacerbation.

   iii. **Key laboratory Findings**
      1. Elevated **erythrocyte sedimentation rate (ESR)** is often present but not pathognomonic of metastatic disease
      2. Elevation of **serum calcium** may occur in diffuse osteolytic metastatic carcinoma. Serum calcium in most cases, even if lytic, are normal.
      3. Alkaline phosphatase is frequently elevated in blastic metastatic lesions but overall is a insensitive indicator of bone metastasis.
      4. Prostate Specific Antigen (PSA) is elevated >10ng/mL in cancer patients which the prostate gland tumor has broken through

d. **Lytic vs. Blastic Metastasis**

<table>
<thead>
<tr>
<th>Lytic Metastasis</th>
<th>Blastic Metastasis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>M/C Primary</strong></td>
<td><strong>M/C Primary</strong></td>
</tr>
<tr>
<td>(M) 1. Lung</td>
<td>(M) 1. Prostate</td>
</tr>
<tr>
<td>(F) 1. Lung</td>
<td>(F) 1. Breast</td>
</tr>
<tr>
<td>2. Breast</td>
<td></td>
</tr>
</tbody>
</table>
e. Key radiographic features for each:

Table 11.4 Radiologic Presentation of Metastatic Carcinoma

<table>
<thead>
<tr>
<th>Primary Organ Involvement</th>
<th>Lytic (%)</th>
<th>Mixed (%)</th>
<th>Blastic (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast</td>
<td>80</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Lung</td>
<td>75</td>
<td>20</td>
<td>5</td>
</tr>
<tr>
<td>Real Wilm’s Tumor</td>
<td>80</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Urinary bladder</td>
<td>90</td>
<td>10</td>
<td>---</td>
</tr>
<tr>
<td>Thyroid</td>
<td>90</td>
<td>10</td>
<td>---</td>
</tr>
<tr>
<td>Prostate</td>
<td>10</td>
<td>10</td>
<td>80</td>
</tr>
<tr>
<td>Salivary glands</td>
<td>100</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Neuroblastoma</td>
<td>80</td>
<td>15</td>
<td>---</td>
</tr>
<tr>
<td>Esophagus</td>
<td>85</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Stomach</td>
<td>90</td>
<td>10</td>
<td>---</td>
</tr>
<tr>
<td>Colon or rectum</td>
<td>75</td>
<td>5</td>
<td>20</td>
</tr>
<tr>
<td>Pancreas</td>
<td>80</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Liver</td>
<td>70</td>
<td>30</td>
<td>---</td>
</tr>
<tr>
<td>Gallbladder</td>
<td>90</td>
<td>10</td>
<td>---</td>
</tr>
<tr>
<td>Uterine cervix</td>
<td>90</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>Uterine corpus</td>
<td>90</td>
<td>10</td>
<td>---</td>
</tr>
<tr>
<td>Ovary</td>
<td>90</td>
<td>7</td>
<td>---</td>
</tr>
<tr>
<td>Testis</td>
<td>75</td>
<td>5</td>
<td>20</td>
</tr>
<tr>
<td>Skin carcinoma</td>
<td>95</td>
<td>5</td>
<td>---</td>
</tr>
<tr>
<td>Malignant melanoma</td>
<td>90</td>
<td>10</td>
<td>---</td>
</tr>
<tr>
<td>Carcinoid</td>
<td>5</td>
<td>15</td>
<td>80</td>
</tr>
<tr>
<td>Hodgkin’s Lymphoma</td>
<td>40</td>
<td>10</td>
<td>50</td>
</tr>
</tbody>
</table>

f. Provide the differential dx. of pedicle destruction (table 11.9)

Table 11.9 Differential Diagnosis of Pedicle Destruction

<table>
<thead>
<tr>
<th>Congenital</th>
<th>Agenesis (Contralateral Pedicular Sclerosis)</th>
<th>Hypoplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neoplasm</td>
<td>Benign</td>
<td>Aneurysmal bone cyst</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Osteoblastoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neurofibroma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Osteoid osteoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Malignant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lytic metastasis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Myeloma (rare</td>
</tr>
</tbody>
</table>
2. Features of Primary Malignant vs. secondary tumors. (Table 11.6)
Malignant Bone Neoplasms: Differentiating Radiologic Features between Primary and Secondary lesions

<table>
<thead>
<tr>
<th>Feature</th>
<th>Primary</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>30%</td>
<td>70%</td>
</tr>
<tr>
<td>Expansion of bone</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Joint involvement</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Length of lesion</td>
<td>&gt;6cm</td>
<td>2-4cm</td>
</tr>
<tr>
<td>Periosteal response</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Solitary lesion</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Multiple lesions</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Soft tissue mass</td>
<td>+++</td>
<td>+</td>
</tr>
</tbody>
</table>

3. List 3 ways in which tumors can spread from a primary site to bone.
   a. **Direct Extension**
      i. Direct invasion into a bone may occur from a soft tissue tumor lying adjacent to or near the bone.
      ii. An example is carcinoma of the uterus which is known to cause direct extension to the iliac bones
   b. **Lymphatic Dissemination**
      i. This is uncommon. It is thought that the absence of lymphatic channels in bone marrow is the reason for the relatively low incidence of lymphatic seeding of bone.
   c. **Hematogenous Dissemination**
      i. Spread through blood vessels, particularly the veins, is the most common pathway for tumor emboli. The three most commonly seeded in this manner are the lungs, liver, and the axial skeleton.
      ii. **Batson’s Venous Plexus** are valveless veins where blood can reflux when intrathoracic and intra-abdominal increases. Batson’s plexus also provides a series of venous passageways by which cancer cells can be directly seeded into bones. *(Figures 11.2 and 11.3)*

4. What is the most common primary malignancy of bone?
   a. Multiple Myeloma
   b. What does it look like on a bone scan?
      i. Bone scans are cold or normal except at sites of pathological fractures, which will be focally hot.
      1. This occurs because Multiple Myeloma causes the release of Osteoclast Activating Factor by the plasma cell. Bone scans depend on Osteoblastic activity. So when there is a fracture osteoblastic activity occurs.
   c. Where is it primarily located
      i. Located primarily in the spine.
      ii. Also located in the pelvis skull, ribs, and scapula.
d. Key clinical features.
   i. 75% of patients are between 50-70 yoa and 2:1 male preponderance
   ii. LB Pain is the cardinal initial symptom relieved with bedrest and aggravated with weight. With sciatica
   iii. Bacterial infections occur in 10% of cases most respiratory in nature.
   iv. Pathological fracture is common complication

e. Key radiologic findings.
   i. Bone Scans are cold
   ii. Gross osteoporosis may be the only early sign
   iii. Punched lesions are the radiologic hallmark of myeloma
   iv. Vertebra plana or wrinkled vertebra is characteristic.
   v. **Raindrop skull (lytic myeloma defects)** and pedicle sign of myeloma (preservation of pedicles) occur.
   vi. “**Moth Eaten,**” Permeative lesions
   vii. **Foggy cortical – Medullary border**

f. Key laboratory features.
   i. 40% show Bence Jones’ proteinuria.
   ii. Immunoglobulin Electrophoresis – to Elevation of IgG
   iii. May have infections do to decreased WBCs
   iv. Normocytic, normochromic anemia

g. Prognosis
   i. 90% die within 3 years.
   ii. Tx is usually radiotherapy, chemotherapy, and on occasion, local excision.

5. Define Solitary Plasmacytoma
   Localized form of plasma cell proliferation

   a. Key clinical features
      i. Most common bones affected: manible, ilium, vertebrae, ribs, proximal femur, and scapula.

   b. Key radiographic features
      i. The typical lesion has a geographic, soap bubbled, highly expansile radiographic appearance.

   c. Common Complications
      i. **70% of solitary plasmacytoma lesions develop into diffuse multiple myeloma**

6. Osteosarcoma (also look in normal variance packet)
   - Is a primary malignant tumor of bone; it is derived from undifferentiated connective tissue and forms neoplastic osteoid

   a. How common is Osteosarcoma? Age group?
      i. It is the second most common primary malignant bone tumor, representing 20% of all primary malignant bone tumors.
      ii. It is 2x more common than chondrosarcoma, 3x more frequent than Ewings sarcoma.
      iii. 75% of cases occur in the **10-25 year age range**, with 2:1 male predominance.
b. Key clinical features.
   i. Painful swelling of the involved limb is a common presenting symptom
   ii. The metaphyses of the distal femur, proximal tibia, and proximal humerus are the most common sites.
   iii. Only 3.5-7% occur in the spine

c. Key radiologic features
   i. The classic lesion presents as a permeative or ivory medullary lesion in the metaphysis of a long tubular bone with a poorly defined zone of transition.
   ii. A sunburst or sunray periosteal response is characteristic
      1. Sunburst = periosteal reaction that often takes place within an extracortical, dense soft tissue mass that displays transverse spicules or radiating striations.
   iii. Often, Codman’s triangle is found associated with the destructive lesions
      1. This is a reactive response to the lifting of the periosteum and is not pathognomonic for osteosarcoma b/c it may also be found in benign conditions such as traumatic periostitis, osteomyelitis, eosinophilic granuloma, and thyroid acropachy.
   iv. Cortical disruption with soft tissue mass formation, often growing to large dimensions, occurs. The peripheral edge of an eccentric lobulated mass whose margins are roughened and irregular may be referred to as the cumulus cloud appearance.

d. Prognosis
   i. A 20% 5-year survival rate has been traditional; studies using intensive chemotherapy report 80% survival rate
   ii. Amputation has offered the best tx when the lesion is surgically accessible.

7. Chondrosarcoma
   -Is a malignant tumor of chondrogenic origin that remains essentially cartilaginous throughout its evolution.

a. How common is Chondrosarcoma? What age group?
   i. Represents the third most common primary malignant bone tumor, following multiple myeloma and osteosarcoma.
   ii. Age group is 40-60 years 2:1 male predominance.

b. Key clinical features
   i. Pain usually presents late in the disease process, often after large soft tissue masses develop
   ii. Severe pain follows pathologic fracture
   iii. ? The most common sites are the pelvis, proximal femur and humerus, ribs, scapula, sternum, craniofacial bones, distal femur, and proximal tibia. The normal variance notes states that Chondrosarcoma is the m/c primary malignant tumor of the hand. And Used to an enchondroma.

c. Key radiological features
   i. Round or oval radiolucencies with ill-defined margins evident.
   ii. Lesions re metaphseal or diaphyseal
   iii. Endosteal scalloping occurs secondary to pressure erosion from the enlarging lobular mass
   iv. Popcorn matrix calcification in the lesion occurs in 2/3 of cases; 1/3 are purely radiolucent
v. Laminated or speculated periosteal response occurs
vi. Metastatic disease is usually to lung

d. Prognosis
i. Prognosis is good, with 90% survival after early surgery

8. Ewing’s sarcoma
- Is a primitive primary malignant tumor of bone it is composed of tumor cells derived from the connective tissue framework of bone marrow

a. How common? Age group?
i. 7% of all primary bone tumors. 4th m/c primary malignant bone tumor behind (multiple myeloma, osteosarcoma, and chondrosarcoma).
ii. 10-25 years of age

b. Key clinical features
i. Mimics: Infection; systemic signs of slight fever, secondary anemia leukocytosis, and increase ESR
ii. Affects long tubular bones of the Lower extremity.

9. Fibrosarcoma
- Primary malignant bone tumor that produces varying amounts of collagen and has no tendency to form tumor bone, osteoid, or cartilage, either in its primary site or in its metastases.

a. Age group?
i. 30-50 years of age

b. Key clinical features.
i. 2 types of lesions medullary and periosteal
ii. pain and swelling for long duration
iii. the metaphysis is the classic location
iv. Most occur about the knee, femur, tibia, and humerus

c. Key radiological features
i. Highly destructive medullary lesion, lytic and placed within long bone
ii. Produces the largest soft-tissue mass of all primary malignant tumors

10. Giant cell tumor (Quasimalignant)
- Is a neoplasm that originates from non-bone forming supportive connective tissue of the marrow. Contains multiple nucleated giant cells

a. Clinical Features
i. Males:
   1. Tumor tends to be malignant
ii. Females:
1. Tumor tends to be benign
   iii. 80% are benign, 60% are lytic

b. Key Radiologic features
   i. Most lesions begin in metaphysis and extend to subarticular location.
   ii. Thin, expanded cortex, which gives the Soap Bubble Pattern in 40%
   iii. May have delicate periosteal reaction.
   iv. M/c in distal femor and proximal tibia

c. Most worrisome complication
   i. 20% of the time could be malignant

11. Osteochondroma
   -Not to be confused with the congenital anomaly called Suprachondylar process.
   -Also known as Coat-hanger exostosis
   -Is a bony exostosis projecting from the external surface of a bone; usually has a cartilaginous cap.

   a. Key clinical features
      i. Occurs ~20 yoa
      ii. Points away from the joint, there is no ligament
      iii. Most are asymptomatic. M/c complaint is a painless mass around the joint

   b. Key radiological features
      i. 2 types of Osteochondroma:
         1. **Sessile**: On a broad, flat base and no stalk; common in the humerus and scapula.
         2. **Pedunculated**: On a long stalk, with a **cauliflower top**. Extending away from the joint.
      ii. Coat-hanger exostosis represents the pedunculated.
      iii. Has a continuous cortex until you reach the tip. Then it is cartilaginous

   c. Most common complication
      i. Aggressive biopsy
      ii. Trauma or fractures then can become malignant.

12. Briefly define HME:
   -Hereditary Multiple Exostosis is an inherited autosomal dominant metaphyseal overgrowth that is characterized by multiple osteochondromas

   a. Key radiological features
      i. Multiple, painless lumps and bumps around joints
      ii. Bayonet deformity of the wrist
      iii. Broad metaphyses
      iv. Calcified cauliflower cartilaginous caps

13. ABC (Aneurysmal Bone Cyst)
   -Lesion consisting of a cystic cavity filled with blood.

   a. Key clinical features?
      i. 75% 5-20 yoa
      ii. Previous history of trauma. 80% occur in long bone (femur, tibia and spine)

   b. Key radiological features
14. Hemangioma  
-Hemangioma, a primary benign neoplasm, is a slowly growing lesion of bone composed of newly formed capillary, cavernous or venous vessels.  
   a. Key clinical features  
      i. M/c primary benign tumor of the spine  
      ii. No history of going malignant  
   b. Key radiologic features  
      i. **Corduroy cloth or striated vertebra** appearance which is accentuated vertical trabeculae.  
      ii. Barrel shaped vertebra  
      iii. In the skull, it appears as one large lesion. Most occur in the frontal bone, creating a round or oval radiolucency, radiating sunburst or poked-wheel appearance  

15. What is an Osteoma?  
-A cortical bone tumor with a smooth contour and a continuous cortex  
-Are found:  
  Ocning parts of the skull that SHB spaces (sinuses)  
  Attached to the skull as a raised lesion  
   a. What syndrome includes the Osteoma as part of its classic presentation?  
      i. **Gardener’s Syndrome**  

16. Define Bone Island (“Enostoma”)  
 a. Benign, extra bone within bone.  

17. Osteoid Osteoma  
 a. Shares a common presenting complaint with what process?  
    i. Brodie’s Abscess  
       1. The radiolucent nidus of Brodie’s is much larger (>1 or 2cm)  
       2. Hole will be in the marrow  
       3. The halo rim of sclerosis surrounding the nidus is much thicker and more irregular  
    b. Key clinical features.  
       i. Pain at night relieved by aspirin  
       ii. A “blister” on the bone between the cortex and the periosteum. Might show up as a hole or nidus, which is lucent.  
    c. Key radiologic features.  
       i. The nidus or hole is usually surrounded by sclerosis.  

18. Enchondroma  
-Can malignantly degenerate into **chondrosarcoma**  
 a. Key clinical features.  
    i. M/c primary benign tumor of the hand  
    ii. Can be Solitary or multiple (called Ollier’s Disease)  
       1. Solitary enchondroma, is a benign tumor arising in the cartilage in
the metaphysis as the physis goes away.

iii. Usually a painless incidental finding

iv. Most common complication is pathological fracture

b. Key radiological features.
   i. The bone looks less dense do to cartilage tissue within the bone
   ii. 40% will uptake Ca++, giving a speckled appearance- a round lesion with polka dots

c. What is Ollier’s disease? (Multiple enchondromatosis)
   i. Enchondroma’s in multiple sites

**Tumor-Like Conditions**

19. Paget’s Disease (aka *Osteitis Deformans*)
   - Is a bone disease of unknown origin characterized by osteolysis followed by extensive attempts at repair.
   a. Mimics: Paget’s disease mimics a Hemangioma. Hemangioma for the vertebral bodies produces a vertically striated pattern, which may closely mimic the vertical trabeculae of Paget’s disease.

b. Most likely site: Pelvis
   Least likely site is the Fibula

c. List the 4 phases and describe each
   i. **Stage one**: Osteolytic, Destructive, or Monophasic stage- Osteoclastic over activity creates gross loss of bone density described as **osteoporosis circumscripta**
   ii. **Stage two**: Combined, Mixed, or Biphasic stage – M/c encountered. Reflection of both destruction (Lytic) and production of bone (Blastic). Characterized by cortical thickening, increased radiopacity and accentuation of trabecular patterns with lucent areas mixed.
   iii. **Stage three**: Sclerotic or Ivory Stage – Uniform thickening of trabeculae with **ivory appearance**
   iv. **Stage Four**: Malignant Degeneration – Lethal stage

d. Clinical Features of Paget’s
   i. 2:1 males, m/c after 55
   ii. 90% are asymptomatic. Pain, when present, is low intensity and may be associated with bowing deformities or fractures
   iii. Increased hat size because enlargement of the calvaria

e. Radiological features
   i. Bone scan will be hot
   ii. Skull will demonstrate in earl lesions Osteoporosis circumscripta (described in c). More advanced or combined stage demonstrates cotton wool appearance, which is fuzzy, poorly defined edges of sclerotic areas.
   iii. Spine will demonstrate squared-off picture frame vertebra, which is thickened and enlarged vertebral endplates giving a squared look.
   iv. Homogenous increases radiopacity of vertebral body and creates an ivory vertebra
      1. The 3 m/c causes of an ivory vertebra are: osteoblastic metastatic carcinoma, Paget’s disease, and Hodgkins Lymphoma
Table 11-10 Solitary Ivory Vertebra

<table>
<thead>
<tr>
<th>Common Causes</th>
<th>Uncommon Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteoblastic metastasis</td>
<td>Sarcoïdosis</td>
</tr>
<tr>
<td>Hodgkin’s Lymphoma</td>
<td>Chordoma</td>
</tr>
<tr>
<td>Paget’s disease</td>
<td>Myeloma</td>
</tr>
<tr>
<td>Degenerative sclerosis</td>
<td>Osteosarcoma</td>
</tr>
<tr>
<td>Osteomyelitis (fungal or chronic)</td>
<td>Ewings sarcoma</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>Osteoid osteoma</td>
</tr>
<tr>
<td></td>
<td>Osteoblastoma</td>
</tr>
<tr>
<td></td>
<td>Bone island</td>
</tr>
</tbody>
</table>

Table 11-11 Differential Diagnosis of an Ivory Vertebra

<table>
<thead>
<tr>
<th>Factor</th>
<th>Blastic Metastases</th>
<th>Paget’s Disease</th>
<th>Hodgkin’s Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>&gt;45</td>
<td>&gt;50</td>
<td>20-40</td>
</tr>
<tr>
<td>Increased density</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Expansion</td>
<td>----</td>
<td>+++</td>
<td></td>
</tr>
<tr>
<td>Anterior Scalloping</td>
<td>----</td>
<td>----</td>
<td>+++</td>
</tr>
<tr>
<td>Acid phosphatase</td>
<td>+++</td>
<td>----</td>
<td>----</td>
</tr>
<tr>
<td>Alkaline phosphataes</td>
<td>++</td>
<td>+++</td>
<td>++</td>
</tr>
</tbody>
</table>

v. Pelvis includes protrusio acetabuli with Kohler’s teardrop and thickening of pelvic brim
vi. Long bones: the tibia is the m/c lytic site

f. Complications
i. Deformity of bone include: Shepherd’ crook deformity of the proximal femur (coxa vara), Saber shin (ant. Tibial bowing), Protrusio acetabuli.
ii. Cranial nerve neuropathies b/c foramen get smaller.
iii. Linked to increased wear and tear, early DJD
iv. Increased alkaline phospatase (up to 20x normal)

20. Fibrous Dysplasia pg. 1345
-is a disorder of unknown cause in which skeletal aberrations constitute the cardinal feature.

a. Key clinical features.
i. Café au Lait spots with a “coast of Maine” appearance, pigmentation changes
ii. Common in late childhood before puberty during skeletal growth
iii. Polystotic m/c for derormities

b. Key Radiological features
i. Clear zone in the center with a “Ground Gallss Appearance” – beveled / fine fuzzy opaque edges
ii. Stimulates blastic reaction of surrounding tissue = “Rind of Sclerosis”
iii. Can change the contour of bone (expansile), with widening of medullary,
endostal thinning and scalloping
iv. Sheperds crook deformity common

21. Coast of Maine Café au Lait spots is associated with **Polystatic fibrous dysphasia**

22. Coast of California Café au Lait spots are associated with **Neurofibromatosis**

23. Most common malignancy of bone? **METS**

24. Most common primary malignancy of bone? **Multiple Myeloma**

25. Most common benign tumor of the hand? **Enchondroma**

26. Most common Primary malignancy of the hand? **Chondrosarcoma**

27. Most common benign tumor to a growth plate? **ABC (Aneurysmal Bone Cyst)**

28. Most common benign tumor of the spine? **Hemangioma**

29. Define Solitary Bone Cyst? **Is not a true neoplasm of bone but rather a fluid-filled cyst that is lined with a thin layer of fibrous tissue**

30. Fibrocartilagenous bone defect?

31. **Non-Ossifying Fibroma**

### Table 7-5 Radiologic Criteria of Benign and Aggressive Lesions

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Benign</th>
<th>Primary aggressive</th>
<th>Secondary aggressive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (decades)</td>
<td>1-3</td>
<td>1-7</td>
<td>4-7</td>
</tr>
<tr>
<td>Size</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-6cm</td>
<td>+++</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>6+cm</td>
<td>+</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Monostotic</td>
<td>+++</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Polystotic</td>
<td>+</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Cortical destruction</td>
<td>----</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Periosteal reaction</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Solid</td>
<td>+++</td>
<td>+</td>
<td>----</td>
</tr>
<tr>
<td>Laminated</td>
<td>++</td>
<td>++</td>
<td>----</td>
</tr>
<tr>
<td>Spiculated</td>
<td>----</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Codman’s</td>
<td>++</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Destruction</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Geographical</td>
<td>+++</td>
<td>+</td>
<td>----</td>
</tr>
<tr>
<td>Moth-eaten</td>
<td>----</td>
<td>+++</td>
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</tr>
<tr>
<td>Permeative</td>
<td>----</td>
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<tr>
<td>Margins</td>
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</tr>
<tr>
<td>Sharp</td>
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<thead>
<tr>
<th></th>
<th>----</th>
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<tbody>
<tr>
<td>Imperceptible</td>
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</tr>
<tr>
<td>Soft tissue mass</td>
<td>----</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Joint space</td>
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<td>----</td>
<td>----</td>
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