• DISCLAIMER: As previously mentioned with study guides of the same type, I copied these images from Dr. Meuten’s slides. Please don’t distribute to others. If the content is incorrect, I apologize and please let me know so I can make corrections.

• Choose your ‘path’ wisely!!! (are we nerds or what!)
Identify labeled structures.
• A: metaphysis
• B: physis
• C: epiphysis
Describe lesion
• Osteochondrosis
Identify lesion. Etiology?
• Osteochondrosis
• Etiology
  – Local ischemia
  – Overnutrition,
  – Rapid growth
  – Trauma and weight bearing
  – Zn tox in horse, pig
  – Cu deficiency in horse, deer
Identify lesion. Pathogenesis?
• Osteochondrosis

• Pathogenesis
  – Ischemia => necrotic cartilage => FAILURE OF ENDOCHONDRAL OSSIFICATION => plug of dead cartilage
  – Trauma to wt-bearing region => necrotic plug of cart fractures => flap => leakage of synovial fluid into cart/subchondral bone => inflamm => synovial hyperplasia => joint mice
Identify lesion. Describe structure(s) denoted by arrows.
• Osteochondroses
• “joint mice”
  – Fragments of cartilage separated from healed lesion
  – Grown w/l joint space
  – Derive nutrients from synovial fluid
Identify lesion. Signalment?
• Osteochondrosis
• Signalment: young, rapidly-growing, male
• Osteochondrosis
• During healing, exostoses/soft tissue prolif encroach/compress spinal cord => cervical vertebral stenotic myelopathy (wobbler syndrome)
Identify most common site in dog, pig, horse, cow, turkey
• Dog: caudal aspect of humeral head
• Pig: medial condyle of stifle
• Horse: lateral trochlear ridge of femur
• Cow: lateral trochlear ridge of femur
• Turkey: tibia (tibial dyschondroplasia = new name, same great taste!!)
Summary........
• OC (once separation occurs = OCD)
• Common disease in veterinary medicine
• Young rapid growing large males
• Ischemia of AE cartilage due to ……
• Early lesions are asymptomatic
• Weight bearing & trauma contributes to progression and fx
• By time lesions produce clinical signs the lesions are chronic
• Predisposes to DJD
• Treatable
Identify/describe condition
• Bovine chondrodysplasia (bulldog type)
• Extremely short limbs
• Domed forehead w/ reduced muzzle/protruding mandible
• Tongue is normal size (marked protrusion)
Identify condition and characteristics.
• Bovine chondrodysplasia (snorter type)
• Short head
• Bulging forehead/protruding mandible
• Shortened vertebral column and bloat are typical
Identify condition. Describe characteristics
• Ovine chondrodysplasia (spider lamb synd)
• Most born alive (some aborted/stillborn)
• Limbs disproportionately long/deformed
• May include scoliosis, kyphosis, ‘roman’ nose
• Stippled appearance on radiographs (multiple ossification centers)
Dog. Identify/Describe condition.
• Chondrodysplasia
• Irregularly thickened physis
• Physes have extra cartilage d/t failure of endochondral ossification
• Usually in radius
• Could be confused w/ rickets grossly/radiographically
Dog. Identify/describe condition.
• Pseudoachondroplastic dwarfism
• Epiphyses are a cap of cartilage
• Cart ‘mushrooms’ over metaphysis b/c ossification centers fail to form/are delayed
• Radiolucent cart on rads
Describe condition.
• Pituitary dwarfism (symmetrical)
• d/t decreased STH
Identify/describe condition.
• Osteopetrosis
• Defect in bone resorption
• Unresorbed cones of primary spongiosa extend from physes to center of diaphysis
  – Absence of medullary cavity
Identify lesion/condition. Pathogenesis. Prognosis?
• Osteopetrosis
  – Solid vertebrae ("butterfly" vertebrae)
• Defect in osteoCLASTS in mammals
  – Excess endosteal bone
• Defect in osteoBLASTS in birds
  – Excess endosteal AND periosteal bone
• LETHAL w/o bone marrow transplant
• Osteopetrosis (avian)
• Defect in osteoBLASTS
  – Excess bone production
    • Periosteal and endosteal
Identify/describe condition.
• Porcine congenital hyperostosis
• (bottom limb=affected)
• Thickened d/t increased periosteal bone along diaphysis.
  – Surrounding tissues swollen/edematous
• LETHAL
Differential diagnoses.
• Rickets
• Osteogenesis imperfecta (dx)
• Joint laxity
• Vit C deficiency
Identify condition. Etiology/pathogenesis
Other lesions?
• Osteogenesis imperfecta (heritable)
• Defect in TI collagen
  – Blue eye b/c sclera mostly type I
  – Dentin mostly type I
• Broken tooth=18d old (usually death before)
• Other lesions: rib fractures in utero, callus on ribs, marked joint laxity,
Identify condition. Dx
• Domed forehead, undershot mandible, blue sclera, pink teeth, joint laxity, bone fragility, fragile skin
• Bone(right) show diaphyseal thickening and absence of medullary cavity
Ddx. Dx
• Osteogenesis imperfecta (Dx)
• osteopenosis
Dx. Non-skeletal lesions
• Porphyria
  – Brown discoloration of teeth; red fluorescence under UV light; bones normal in strength; pathologic lesions in skin

• photosensitization
Describe lesion. Pathogenesis.
• Yellow discoloration
• Only in recently formed during tetracycling admin
  – Tetracycling deposited at sites of current mineralization
Neonate. Dx. Etiology. Primary/secondary lesions
• Arthrogryposis (crooked calf dz)
• Familial (charlois, herefords), toxic plants (lupinus)
• Defect is spinal cord; “stuck” in position in utero (unable to “correct” joints)
• Primary=spinal cord/muscle
• Secondary=skeletal
Dx. etiology
• Cyclopia
• Ewe ingests veratrum californicum day 14 of gestation.
• Lamb may have pituitary hypoplasia therefore prolonged gestation
Ddx. Dx. pathogenesis
• Angular limb deformity(dx), rickets, OC, osteoporosis, FOD, osteogenesis imperfecta, Vit C deficiency
• Asymmetric lesion in rapidly growing physis
• May be secondary to abnormally shaped carpal/tarsal bones or joint instability
• Older patient=likely damage to physis
Dx. Possible causes
• Angular limb deformity
• Hypothyroidism (goiter in foals), poor conformation, OC, osteomyelitis, trauma, joint lax,
• Many will self-correct
Describe lesions. Ddx
• Rickets
• Failure of mineralization
• Tongues of cartilage protruding into metaphysis
• Flaring from extra woven/soft bone formation
• Other conditions w/ tongues of cartilage
  – OC, non-pit dwarfism
Describe lesions. Antemortem Ddx; etiology/pathogenesis.
• Ddx: FOD(dx), sinusitis, tooth abscess, field rickets
• PTH (nutritional/renal),
Describe lesions. Dx. Non-skeletal lesions
• Decreased bone, growth arrest lines
• Serous atrophy of fat
• osteoperosis
Identify lesions
Clockwise: rickets, FOD, osteoporosis
Describe lesions. Dx. Etiologies.
• Osteoperosis. Lesions described perviously
• Starvation, parasitism, pneumonia, age, disuse
State lesions. Dx. Describe pathogenesis.
• Radiolucency of bone with multiple holes (from old plate) and significant cutaneous inflammation & necrosis
• Disuse osteoporosis + osteitis
• Disuse -> osteoporosis + inflamm -> cytokine induced osteolysis
State lesions. Ddx?
• Multiple fractures in multiple bones
• Which metabolic dz?
Describe lesion. Ddx.
• Growth arrest lines in metaphysis parallel to physis
  – Indicate physeal growth ceased then recommenced on several occasions
• Osteoporosis (starvation, parasitism), lead tox, K9 distemper, BVD
Describe histo characteristics. Dx.
• Growth ceased, cap of bone formed on metaphyseal side; trabecular bone in metaphysis is sparse
Describe lesion. Dx. Pathologic characteristics
• Thin cortices, depletion of trabecular bone in metaphysis, serous atrophy of medullary fat
• Osteoporosis
• Bone retains normal shape; trabecular bone depleted first (>surface area for resorpt); lesions most prominent in bones consisting of trabecular bone; thin cortices in advanced stages
Dx. Describe gross and histo characteristics.
• Left=normal; right=rickets
• Gross: defective mineralization of physeal cartilage at sites of endo ossif; irregular thickening of rapidly growing physes; in adults, defective mineralization confined to osteoid formed during remodeling
• Histo: persistence of hypertrophic chondrocytse at sites of endo ossif; trab bone in metaphysis is disrupted; islands of hypertrophic chondrocytes in metaphysis
Describe lesion. Dx. Etiologies.
• Enlarged CCJ; tongues of cartilage extending into metaphysis from physis; growth arrest line
• Rickets
• Vit D deficiency; phos deficiency
Describe lesions. Dx.
• Irregularly thickening of physis; focal replacement of trabecular bone w/red, gelatinous, fibrous CT; 2 white foci of cartilage protruding below physis
Histo prep from previous gross specimen. Describe characteristics.
• Marked focal thickening of physeal cartilage b/c mineralization did not occur; endochondral bone formation also did not occur

• Virtually pathognomonic for rickets
Describe lesion. Dx
• Thick plugs of cartilage along physis
• rickets
Ddx.
• sinusitis, FOD, neoplasia, others
• Scurvy d/t Vit C deficiency
• VitC is coenzyme in collagen synth-> decreased osteoid, collagen, dentin
• Abnormal weak collagen in blood vessels, scars, dentin, osteoid
• Lesions: hemarthrosis, periodontal hemorrhage, subperiosteal new bone rxn, osteoperosis
Describe lesions. Dx. Etiologies.
- White mineralized muscle (VitD tox)
- Oversupplementation, plants (Cestrum), rodenticide
- Excessive soft tissue mineralization; hypercalcemia/hyperphosphatemia
- Die of renal/cardiac mineralization before bone lesions develop (osteopetrosis)
• Band of increased density on metaphyseal side of physis (metaphyseal sclerosis)
• Inc density visible on rads (lead line)
• Can also occur from CDV
• Growth arrest line WITHOUT serous atrophy of fat
Describe lesion. Dx
• Dense bands in metaphysis on rads (lead line)
• Metaphyseal sclerosis (lead tox)
Describe lesion. Ddx (lots!!!)
• Hyperostoses (excessive bone formation)
• VitA tox, flurosis, HOD, HO, craniomandibular osteopathy(if mandible 😊), eos panosteitis, infectious
Describe lesion. Dx.
• Central incisors normal; others show accelerated wear and brown discoloration of enamel
• Virtually pathognomonic for fluorine tox
• Brown discoloration of molars (not shown) is normal and must not be mistaken for dental fluorosis
• HOD; young, fast growing, large breed dog
• Swelling/pain in metaphyseal regions of long bones (esp rad/ulna)
• Etiol: VitC def, VitD tox, CDV, bact infection,
• Lesions: PNB, microfractures subjacent to physis, osteomyelitis subjacent to physis; multiple bones involved
Dog. Describe lesion. Dx. pathogenesis
• Increased density in metaphyses, early PNB formation
• Canine metaphyseal osteopathy (HOD)
• Metaphyseal trabeculae become necrotic -> bone resorption -> fibrous tissue (suppurative osteomyelitis) -> PNB in metaphysis
Describe lesions. Dx. Px.
• Canine metaphyseal osteopathy (HOD)
• Alternating radiopaque and radiolucent zones in metaphysis parallel to physis
• May progress to develop PNB around metaphysis
• Usually bilaterally symmetrical
• Px= mild, moderate, severe…..MOST affected animals recover and excess bone is eventually resorbed
dx.
• HOD/CMO
• Mild, moderate, severe (which is which? 🙁)
Describe lesion. Dx. Etiology.
• Hypertrophic osteopathy
• Thickened forelimb; extensive PNB formation
• Space-occupying mass in thoracic/abd cavity
“intermittent” swelling. Describe lesion. Dx.
• Thickening of forelimbs; excessive PNB formation on phalanges/humerus
• HO
• Mass in thor/abd cavity
Describe lesions. Ddx.
• Proliferative bony lesions along mandible, around tympanic bullae, on occipital bone
• Cranio-mandibular osteopathy (Dx), osteomyelitis, tooth abscess, trauma
Describe lesions. Dx. Signalment.
• Canine eos panosteitis (no itis=misnomer)
• Increased ENDOSTEAL bone production in diaphysis
• Increased radiographic density of diaphyseal endosteum
• Large/giant breed dog
• Usually self limiting
Describe lesion. Ddx. Etiologies.
• Discrete pale yellow necrotic foci
• osteonecrosis, osteomyelitis
• Infection, corticosteroids,
• Ischemia
  – Fracture, neoplasia, thromboembolism, peripheral vasoconstriction
Describe lesion.
• Osteonecrosis
• Usually resorbed by osteoclasts
  – Small fragments removed rapidly
  – Large fragments removed surgically
• Focal area of necrosis (sequestrum)
  surrounded by sclerotic bone (involucrum)
Describe histo characteristics. Dx
• Necrotic bone surrounded by layer of basophilic new bone
• Empty lacunae in necrotic bone
• Howship's lacunae indicating osteoclastic resorption
• osteonecrosis
Describe......again
• Osteonecrosis
• Layer of granulation tissue and reactive bone (involucrum) attempts to wall off sequestrum in the center
Describe lesion. Dx. Signalment. Pathogenesis
• Radiolucent/necrotic (aseptic) femoral head; spontaneous decapitation
• Legg-calve-perthes dz
• Sm breed dog, 4-8mo
• Femoral head vessels travel along femoral neck before entering bone; in susceptible breeds vessels remain superficial and venous drainage impaired; infarction of femoral head
Dx. Signalment. Complete pathogenesis.
• Osteomyelitis (hematogenous) = multiple jnts
• FPT -> neonatal septicemia -> hematogenous spread to patent venous-arterial loops in metaphyses (septic suppurative arthritis and osteomyelitis) -> bacteria exit into tissues
• Portal of entry: umbilicus = calf, foal; lamb = docking/castration; piglet = ears, tail chewing, teeth, castration; hi k d b ki t
Describe lesion
• Periostitis
  – Inflammation originating in the periosteum
Dx. pathogenesis
• Osteomyelitis
  – Direct extension/local invasion
  – One bone/joint
  – Worse px
  – More common than hematogenous
Dx. Etiology. Pathogenesis.
• Osteomyelitis (atrophic rhinitis)
• Bordetella/pasteurella
• Endotoxins-> inflammatory cells-> cytokines-> stimulate osteoclastic osteolysis AND endotoxin inhibits osteoblasts (bummer)
Describe lesion. Ddx
• Osteomyelitis
  – Atrophic rhinitis
  – Middle ear infection
  – Periodontal dz
  – Surgery, plates, orthopedic devices
Etiologic agents for cow, horse, dog, pig, turkey, calf/foal/piglet
• Osteomyelitis (generally ANAEROBES)
• Cow: Acinomyces bovis (lumpy jaw)
• Horse: Salmonella > Klebsiella > staph/strep
• Dog: staph, brucella, hepatozoon canis
• Pig: brucella
• Turkey: staph aureus + anaerobes = osteomyelitis-synovitis (green liver, abscess in distal ends of bones)
• Calf/lamb/piglet: Clostridium pyogenes, strep
• *****if culture neg for anaerobes/aerobes, check for mycotic: Coccidioides immitis, crypto histo blasto aspergillus
Dx. Predilection sites.
• Osteomyelitis
• Metaphyses of long bones and vertebral bodies d/t to unique blood supply