Musculoskeletal Objectives

Week 1

5.3.04 Introduction to the Anatomy of the Limbs
- GA Dissection 19A: Scapular and Pectoral Regions
- GA Dissection 19B: Gluteal Region and Posterior Thigh

5.4.04 Connective Tissues II - Bone Development and Growth
- Lab: BONE DEVELOPMENT

5.5.04 Innervation Patterns of the Upper and Lower Limbs
- GA Dissection 20: Axilla

5.6.04 Human Limb Development
- Physiology of Skeletal Muscle
- Gross Structure & Function of Skeletal Muscles
- Skeletal Muscle Plasticity & Adaptations to Exercise aka Exercise Physiology, Muscle Conditioning

5.7.04 Functional Anatomy of the Hand and Forearm
- GA Dissection 21A:
- GA Dissection 21B:

Week 2

5.10.04 Review of Week One Gross Anatomy
- GA Dissection 22A: Posterior Forearm & Dorsal Hand
- GA Dissection 22B: Leg and Foot

5.11.04 Orthopaedic Soft Tissue Injury
- USC Athletic Medicine: Working to Provide the Best Possible Medical Care
- Biomechanics of Fractures

5.12.04 Gait & Posture
- GA Dissection 23: Palmar Hand

5.13.04 PPP Case - Shoulder Injury & Pain
- PPP Case - Carpal Tunnel Syndrome

5.14.04 The Knee Joint
- GA Dissection 24A: Joints of Upper Limb
- GA Dissection 24B: Joints of Lower Limb

Week 3

5.17.04 Overview of Hand Surgery / Pathophysiology of the Hand
- Pharmacology of Muscle Relaxants
- Local Anesthetics
- Arthritis Overview / Rheumatoid Arthritis (RA) / Seronegative Arthritis
- Drugs and RA Seronegative Arthritis (Gopa review sheet)

5.19.04 Review of Autoimmunity
- Systemic Rheumatic Disease and Lupus
- PPP Cases: Lupus and Scleroderma

5.20.04 Pathogenesis of Degenerative Arthritis / Osteoarthritis (OA)
- Artificial Joint Replacements
- Crystal-Induced Arthritis and Gout
- Pharmacology of Gout

5.21.04 Pathophysiology of Joints
- Radiographic Imaging Neck & Back Pain
- Mechanics of the Cervical and Lumbar Spine
- PPP Cases: Low Back Pain

Week 4

5.24.04 Bone and Joint Infections
- Non-Neoplastic Diseases of Bones and Joints
- Lab - Pathology Non-Neoplastic Diseases Of Bones and Joints

5.25.04 Neurological Disorders of the Musculoskeletal System
- Pathology Tumors of Mesenchymal Origin
- Case Discussions Tumors of Mesenchymal Origin
- Pediatric Orthopaedics
Points from Study Guide:
p.6 abduction/adduction in frontal plane
p.7 involves extension, adduction, and lateral rotation of the 1st metacarpal bone, plus extension of the MP and IP joints of the thumb. The same movements occur with opposition involving the little finger
- Ankle. The foot is rotated about 90° to a position perpendicular to the leg as an adaptation to bipedality and about 180° during development; embryonic dorsal surface of the foot faces upward in the adult.
  - dorsiflexion = extension @ ankle
  - plantar flexion = flexion @ ankle
  - inversion = sole/plantar surface face in/medially
  - eversion = sole/plantar surface face out/laterally

1. Upper Limb.
   a. Deltoid as a multifunction muscle.
   b. Muscles used in raising your arm above the head and lowering it down again.
   c. Biceps muscle (anatomical action versus function).
   d. Pronation and supination (or, why screws are designed to turn clockwise).
   e. Making a fist and the power grip.
   f. Precision movements of the fingers.

2. Lower Limb.
   a. Extension at the hip against resistance (running, climbing stairs, sitting down in and getting up from a chair)
   b. Muscles involved in kicking a ball.
   c. Sartorius as a multi-joint, multifunctional muscle.
   d. The functional role of muscles in walking (gait).

   - pronation rotates the radial tuberosity of radius so it points posteriorly
   - “wraps” the biceps tendon around radius; biceps cause supination by “unwrapping” (rotating) the radius
5.3.04

Introduction to the Anatomy of the Limbs

Dr. Snow

1) Present an overview of the dissection approach and schedule.

2) Define the scope of the material to be learned and how best to study it.
   - NOTE: do not need to know blood supply to muscle (M); yes ID and relation of blood vessels (BV)
   - Do need to know nerves, location, relations, 1ary actions, attachments

3) Review joint classification and structure.
   Study joints in 3 ways:
   - (a) classification
     - (i) fibrous joints
       - articulate surfaces firmly connected by dense connective tissue (CT) → little movement
       - e.g. interosseous membranes between long bones of forearm and of leg
     - (ii) cartilaginous joints, aka symphyses
       - articulate surfaces joined by cartilage
       - e.g. intervertebral disks
     - (iii) synovial joints
       - greatest degree of movement
       - features:
         - (1) joint cavity / space
         - (2) hyaline cartilage on bony surfaces
         - (3) joint capsule
         - (4) synovial lining membrane (lots of VAN in here)
         - (5) synovial fluid
   - (b) basic morphology
   - (c) possible movements around its axis(es)

4) Review nerve components within nerves to the limbs.
   - cutaneous: sensory (nerve cell bodies (NCBs) in DRG), sym/post (NCBs: pre in lateral gray T1-L2, post in sympathetic chain ganglia)
   - motor: sensory (pain/proprioception; NCBs in DRG), motor (NCBs ventral horn: upper limb - C5-T1, lower limb – L2-S3; sym/post (NCBs: pre in lateral gray T1-L2, post in sympathetic chain ganglia)

5) Define axes of motion around which muscles act at pectoral and pelvic girdles.
   - 3 axes at glenohumeral / hip joint:
     - (a) flexion / extension around transverse axis
     - (b) abduction / adduction around anteroposterior axis
     - (c) medial / lateral rotation around vertical axis
   - Rotation of the Scapula
     - (1) abduction of humerus at glenohumeral joint
     - (2) lateral rotation at glenohumeral joint
     - (3) upward rotation of scapula (involve upper/lower trapezius, serratus anterior)

6) Introduce concept of collateral circulation around joints.
   - Anastomoses, esp. around joints w/lots of mov’t, e.g. scapula, hand
   - e.g. transverse cervical artery / dorsal scapular artery to get around axillary or subclavian artery blockage
Musculoskeletal Objectives

5.3.04
GA Dissection 19A: Scapular and Pectoral Regions

Osteology
- **Pectoral girdle** (clavicle and scapula)
- **Scapula**
  - Scapular spine expands laterally to become...
  - Acromion process
  - Infraspinous fossa
  - Supraspinous fossa
  - Scapular notch on superior border of scapula, supra scapular VAN (vessels, arteries, nerves) runs thru here
- **Inferior angle**
- **Medial (vertebral) border**
- **Glenoid cavity** location of glenohumeral joint
- **Coracoid process** projects anteriorly
- **Humerus**
  - Head of the humerus at proximal end
  - Glenohumeral joint - smooth articular surface
  - Greater and lesser tubercles - roughened portion of the head
  - Intertubercular groove - between G/L tubercles
  - Surgical neck
- **Clavicle**
  - Sternoclavicular joint - medial end articulates with sternum
  - Acromioclavicular joint - lateral end articulates with acromion
- **Surface anatomy**
  - Dorsal venous arch - superficial vein on dorsum of hand
  - Cephalic vein - extend superiorly from ? on thumb-side of forearm
  - Basilic vein - extends superiorly from arch on little finger-side of forearm
- **Dermatomes**
  - C6 = skin of thumb
  - C7 = skin of middle finger
  - C8 = little finger

Dissection
- **Basilic vein** - courses within superficial fascia
- **Cephalic vein** - courses within superficial fascia
- **Medial cubital vein** - connection between basilic and cephalic veins

Dorsal Scapular Region
- **Superficial Muscles of the Back**
  - **Trapezius muscle** - fan-shaped; arises from back of skull & cervical and thoracic vertebrae; inserts into spine of scapula, acromion process, and lateral third of clavicle; can contract as a whole, or in parts (upper, middle, lower);
  - **Accessory nerve** - on reflected deep surface of trapezius M as well as...
  - **Branches of the transverse cervical artery and vein**
  - **Latissimus dorsi muscle** - broad extent; inserts onto humerus; superficial to & confused w/ ...
  - **Serratus anterior muscle** - fibers run to latissimus dorsi M
  - **Rhomboideus muscle** - attach to medial border of scapula; retracts scapula
  - **Levator scapulae muscle**
  - **Dorsal scapular nerve** - innervates rhomboid and levator scapulae Ms
  - **Deltoid M** - superior attach to scapular spine, acromion, clavicle; broad origin at clavicle, tapers onto lateral shaft of humerus at midpoint; ? shape, : many actions at shoulder - anterior - flexion / medial rotation of humerus, middle - abduct humerus, posterior - extension and lateral rotation
  - **Axillary N (nerve) / Posterior circumflex humeral artery** - enters deep surface of ? ; axillary N adjacent to surgical neck; [C/D:401B] N/A reach deltoid by passing thru space bordered by teres minor M, teres major M, and ...
  - **Lateral and long heads of triceps brachii muscle**
  - **Supraspinatus M** - occupies supraspinous fossa of scapula; passes deep to acromion to insert onto superior head of humerus; : action at glenohumeral joint - abduction of arm
Bursitis – inflammation of a bursa (CT sac w/small amount of synovial fluid from synovial membrane lines inner surface of bursa. Occur at points of friction to save tendons and soft tissue.

**Infraspinatus M** – occupies infraspinous fossa of scapula; attaches to head of humerus inferior to supraspinatus tendon; action – laterally rotates arm

**Teres minor M** – inferior to / fused with infraspinatus M; similar attach to humerus and action; different innervation (teres minor – axillary; infraspinatus – suprascapular)

**Teres major M** – passes medial to shaft of humerus; attaches onto anterior aspect of humeral shaft below head

**Rotator cuff** – shoulder (glenohumeral joint) depends on four deep tendons to reinforce joint capsule as cross joint to attach to head of humerus (supraspinatus, infraspinatus, teres minor, subscapularis); ¾ rotate humerus and tendons a most completely envelope joint capsule. “rotator cuff” tendons. Overuse in athletes; supraspinatus tendon esp. vulnerable to wear and tear when chronically pinched between humerus and acromion while abducting upper limb

**Suprascapular nerve & Suprascapular artery** - enter infraspinous fossa by passing around lateral end of scapular spine. A off thyrocervical trunk

**Circumflex scapular artery** [CD:402B] – in ? space of teres minor, teres major, long head of triceps Ms medial to ? a round axillary N / posterior circumflex A; CSA arises from subscapular A off axillary A; contributes to collateral circulation – anastomose w/ branches of suprascapular A and dorsal scapular A (runs along medial scapula)

**Scapular Anastomosis** – alternate pathway if subclavian or axillary A’s occlude; dorsal scapular, suprascapular, circumflex scapular A’s interconnected on dorsum of scapula, no valves; e.g. block subclavian → blood reach axillary A thru thyrocervical trunk --> suprascapular --> “backwards” thru circumflex scapular / subscapular As to axillary

**Pectoral Region**

**Pectoralis major M** – FROM clavicular, sternal, costal attach to converge near insertion in proximal humerus

**Pectoralis minor M** – inferior attach to ribs, converge superiorly and insert on coracoid process

**Medial pectoral nerve** – pierces thru minor to reach major, innervates both

**Lateral pectoral nerve** – reachespec. major by passing onmedial border of pec minor

**Serratus anterior M** – anterior digitations deep to pec minor, arises from several ribs

**Cephalic V** – in groove btn deltoid & pec major, empties into axillary V = subclavian V as passes under 1st rib anterior to attachment of anterior scalene M, joins w/ internal jugular to form brachiocephalic
5.3.04
GA Dissection 19B: Gluteal Region and Posterior Thigh

**Osteology**
- Pelvic girdle (ilium, ischium, pubis fused into hip and sacrum)
- Hip Bone: [CD:457B]
  - Iliac crest
  - Ala of ilium
  - Greater & lesser sciatic notches
  - Ischial spine (btwn g/l sciatic notches)
  - Ischial tuberosity
- Sacrum: [CD:147B]
  - Sacrotuberous / sacrospinous ligaments
  - Convert G/L sciatic notches into greater / lesser sciatic foramina
- Femur:
  - Greater / Lesser trochanters
  - Head of femur
  - Neck of femur
  - Shaft
  - Medial / Lateral condyles

**Dissection**
- Dermatomes - [CD:511A]
  - L4 = skin of big toe; L5 = skin middle toe; S1 = little toe
  - Great saphenous V - w/in superficial fascia on anterior/meatial thigh/leg
  - Iliopsoas M - convergence of Ilacus M. / psoas M. on posterior abd wall; IP M passes into anterior thigh beneath inguinal ligament, attaches inferiorly at femoral trochanter
  - Femoral N - lateral psoas M sup to inguinal lig
  - Obturator N - medial and deep to psoas M, into obturator canal.
  - Obturator A - accompanies N

**Gluteal Region**
- Gluteus maximus M - from dorsum of sacrum & posterior iliac crest to proximal femur and iliotibial tract oblique; fascia from muscle to iliac crest
- Sacrotuberous ligament - strong ligament attached to & near inferior margin of posterior gluteal max
- Superior / inferior gluteal vessels - enter deep surface of gluteal max
- 4 Ms inserr onto greater trochanter of femur (in the middle of the pears is a hole with four great seeds):
  - glutus medius M
  - piriformis M
  - obturator internus M
  - quadratus femoris M

- Lateral Rotators of the Hip: piriformis, obturator internus, obturator externus, quadratus femoris inserr onto, or adjacent to (obt ext), greater trochanter of femur; cross hip joint posteriorly. rotate hip laterally; medial / lateral rotation round vertical axis reference point ant femoral head; lateral rotators have 2 essential fx's: (1) provide stability to hip joint - pull head of femur tightly into acetabulum., (2) keep foot pointing straight ahead during swing of gait, else foot points inward (pigeon-toed)

- Piriformis M relationships: superior gluteal VAN sup to M, inferior gluteal VAN inf to M, the large sciatic N enters from pelvic cavity inf to muscle and curves inf/lat’ly to enter the post thigh

- Gluteus minimus M - superior gluteal vessels pass btwn glut med and glut min; from ilium to greater trochanter; action of g-min and g-med is abduction of femur at hip joint; N’d: superior gluteal N

- Glut med & glut min as Stabilizers of the Pelvis: Gmed/Gmin abduct hip AND stabilize the pelvis during walking. In stance of gait, Ms resist gravity, prevent hip from collapsing toward opposite side when foot elevated; paralyzed super glut N → distinct waddling gait - sagging pelvis to opp side when opp limb in swing of gait, compensate by lurching trunk to side of paralysis = Trendelenburg gait

- Hamstrings - from ischial tuberosity to bones of leg (the long ten covers the short mem), cross hip joint (except short head); action: flexion of leg (cross knee), extension of thigh or trunk (cross hip joint)
  - Semitendinosus M - pass medial to knee
  - Semimembranosus M - pass medial to knee
  - Short & Long heads of biceps femoris M - pass lateral to knee
  - Sciatic N - from entry into gluteal region thru greater sciatic foramen to division into: tibial N (semimem, semilen, long head, adductor magnus) & common (peroneal) fibular N (short head) just above knee
  - Hamstring blood supply from perforating As off. femoral artery
Musculoskeletal Conditions have an Enormous Impact on the Population of the United States

- They Rank Highest Amongst Disease Groups when Indicators of Quality of Life, Such as Impairment, Disability, or Limitation of Activity are Considered
- Ranks 1st in Frequency of Visits to Physicians
- 2nd in Frequency of Hospitalizations
- 4th in Frequency of Surgical Procedures within Hospitals
5.4.04
Connective Tissues II - Bone Development and Growth
Aaron Logan

1. Be able to list and define the characteristic structures and functions of the cells and extracellular matrix in cartilage and bone.

   Bone consists of a calcified extracellular matrix (ECM) comprised of organic and inorganic components. The organic component of bone consists of type I collagen, fibronectin, GAGs, proteoglycans, and several specialized proteins (e.g., osteonectin, osteocalcin, osteopontin). The organic matrix, called osteoid, becomes ossified by the deposition of hydroxyapatite \([\text{Ca}10(\text{OH})2(\text{PO}_4)6]\) crystals to form calcified bone.

2. Be able to distinguish the difference between intramembranous and endochondral bone formation and describe the steps involved in each.

   **Intramembranous:** bones created in richly vascularized mesenchymal tissue, mesenchymal cells differentiate into osteoblasts, they cluster to form 1ary ossification center and ground substance and collagenous fibers come along to constitute the bone matrix. Bony trabecular joint as spongy bone. Appositional growth. Eventually compact bone on spongy bone.

   **Endochondral:** originates in a cartilage model, replaced by bone. Vascularization of perichondrium at diaphysis of hyaline cartilage causes chondrogenic cells to differentiate into osteoprogenitor cells \(\rightarrow\) osteoblasts, lay down osteoid into bone collar. Trabecular bone and marrow cavity

3. Be able to explain how bones grow in length and width.

   Length: proliferation and hypertrophy of chondrocytes w/in the epiphyseal plate (Growth plate between diaphysis/epiphysis), then calcification of cartilage matrix

   Width: bone deposition on outer surface (osteoblast) of bone collar & resorption on inner (osteoclast)

4. Be able to describe the processes involved in bone remodeling.

   Bone renewed by coordinating bone-resorbing osteoclasts (from hematopoietic stem cells) and bone-forming osteoblasts. Responsive to physical forces (provided by weight-bearing and muscular contractions), hormones, blood Ca++ levels, paracrine effector proteins in BM

5. Be able to describe the sequence of events in repair of bone fractures.

   (1) fracture hematoma: BVs broken, lack of circulation \(\rightarrow\) cell death and inflammation \(\rightarrow\) PMNs, MFs, osteoclasts (absorb bone fragments)

   (2) cartilaginous callus formation: granulation tissue formed from infiltrating capillaries, fibroblasts, osteoprogenitor cells from periosteum, BM LCT; fibrous CT between ends of bone, hyaline cartilage callus around ends of bone

   (3) bony callus formation: osteoprogenitor cells from (2) produce bony trabeculae by intramembranous ossification (see Q2); ? blood supply \(\rightarrow\) endochondral ossification forming a bony callus 1st bone

   (4) bony remodeling: 1st bone remodeled to 2nd bone. Original (compact + spongy) bone restored

6. Be able to describe the basic structure of synovial joints.

   1) periosteum
   2) fibrous layer of capsule
   3) synovial membrane
   4) articular cavity
   5) articular cartilage
   6) spongy bone
   7) compact bone
   8) marrow cavity
#1 Define what is meant by intramembranous versus endochondral bone development.

**Intramembranous:** bones created in richly vascularized mesenchymal tissue, mesenchymal cells differentiate into osteoblasts, they cluster to form **1ary ossification center** and ground substance and collagenous fibers come along to constitute the bone matrix. Bony trabeculae joint as spongy bone. Appositional growth. Eventually compact bone on spongy bone.

**Endochondral:** originates in a cartilage model, replaced by bone. Vascularization of perichondrium at diaphysis of hyaline cartilage causes chondrogenic cells to differentiate into osteoprogenitor cells → osteoblasts, lay down osteoid into bone collar. Trabecular bone and marrow cavity

#2 Explain how and why all bone growth occurs by appositional deposition of lamellae.

Osteoclasts work on inside; osteoblasts work on outside

#3 Prepare a simple diagram to explain how osteoblasts and osteoclasts function in bone remodeling.

#4 Be able to identify the bolded terms in this lab lesson.

**Summary of the stages in sequence of the Development of Long Bone**

1. Cartilaginous model. Cartilage (generally hyaline) is formed from mesenchyme, at first by interstitial growth, later by appositional growth. The model is surrounded by a condensation of mesenchyme called perichondrium (Greek: peri, around; chondros, cartilage), except at the joint surfaces.

2. The osteogenic cells in the perichondrium of the shaft or diaphysis begin to lay down bone in the form of a collar.

   The perichondrium becomes known as periosteum since it now invests and forms bone and not cartilage. Technically, the formation of a subperiosteal, bony collar in the collagenous tissue around the diaphysis is a type of intramembranous bone formation. This is referred to as periosteal bone formation.

3. A cavity develops in the center of the diaphyseal cartilage through degeneration of cartilage cells (chondrocytes). This cavity is invaded by blood vessels which grow through breaks in the periosteal collar and bring in osteoblasts. (It is these osteoblasts which later become active in the endochondral bone formation, mentioned under #4). The cavity becomes the marrow cavity lined with an endosteum.

   As cartilage degenerates, bone begins to be deposited on the surfaces of fragments of calcified cartilage; this central region is now termed the primary center of ossification.

   Beyond the proximal and distal ends of the growing cavity are the epiphyses (epiphysis, Greek: epi, upon; physis, growth, i.e., growth upon the shaft).

4. The cartilage column between the epiphysis and diaphysis is known as the epiphyseal growth plate and will establish the linear growth pattern of the bone. Chondrocytes are arranged in columns, with multiplication occurring at the epiphyseal aspect and degenerating cells disappearing at the diaphyseal aspect. On the remaining cartilage matrix, osteoblasts form bone. The basophilic remains of cartilage may be seen within the newly-formed bony spicules. This cartilage matrix is eventually totally absorbed. Growth in length of the bone results from continual growth of cartilage cells away from the epiphyseal plate and replacement by bone as they degenerate (endochondral bone formation). Each epiphyseal plate of any long bone has a characteristic rate and pattern of growth. The metaphysis (meta = between) is the transitional area between the cartilaginous growth plate and the diaphysis. It is also referred to as the zone of provisional ossification.

5. As bone (and cartilage) grow, secondary centers of ossification arise in the epiphyses. The process of endochondral ossification is essentially the same in these centers as described under #4.

6. During adolescence, the cartilaginous growth centers gradually become inactive. When this happens, the epiphysis fuses with the diaphysis and, with this union, linear growth ceases.
5.5.04
Innervation Patterns of the Upper and Lower Limbs
Dr. Slavin

1. Review the anatomical features of a typical spinal nerve
   motor component supplying skeletal muscle, a sensory component
   transmitting sensation from skin, muscle, tendon and joints and a
   postganglionic sympathetic component innervating blood vessels,
   sweat glands and erector pili muscles.

2. Define a nerve plexus
   communicating network or an intermingling of ventral nerve rami and their branches supplying certain
   regions of the body, causing a widening of the influence of the individual segments of the spinal cord.

3. Make a diagram of the brachial plexus including its major or chief branches supplying the upper limb
   RTDCB [really tiny dicks can break]
   rami: C5 – T1
   Trunks: superior (upper), middle, inferior (lower)
   Divisions: 3 anterior, 3 posterior
   Cords: Lateral, Medial, posterior
   Branches: MMURA [mean men ultimately run away] muscular cutaneous, median, ulnar, radial, axillary

4. Indicate muscular compartmentalization and innervation of upper limb
   ARM: anterior/extensor N-musculotaneous; posterior/extensor N-radial
   FOREARM: anterior N-median & ulnar; posterior N-radial

5. Name the chief branches of the lumbar and sacral plexuses innervating the lower limb, and indicate
   muscular compartmentalization and innervation of lower limb
   thigh:
<table>
<thead>
<tr>
<th></th>
<th>Anterior / extensors</th>
<th>Posterior / flexors</th>
<th>Medial / adductor</th>
<th>Lateral / abductor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thigh</td>
<td>Femoral</td>
<td>Tibial &amp; common fibular</td>
<td>Obturator</td>
<td></td>
</tr>
<tr>
<td>Leg</td>
<td>Deep fibular</td>
<td>Tibial</td>
<td>(evert) Superficial fibular</td>
<td></td>
</tr>
<tr>
<td>Foot</td>
<td>(dorsum) deep fibular</td>
<td>(plantar flexor) tibial</td>
<td></td>
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</tbody>
</table>

   anterior/extensors (femoral); posterior/flexors (sciatic=tibial); medial/adductor (obturator)
   lumbar (L1/2-L4) – femoral (quadriceps M, extensor/anterior) / obturator (adductor) Ns
   sacral (L4-S3) – sciatic N = tibial + common peroneal (aka fibular) Ns
   leg:
   anterior/extensors/dorsiflexors (deep fibular); lateral/evertors (superficial fibular); posterior/flexor/plantar flexor (tibial)

6. Discuss the cutaneous nerves and dermatome distribution of upper and lower limbs
   cutaneous N? dermatome because several cutaneous Ns are components of each spinal nerve

7. Differentiate between trauma to the upper and lower regions of the brachial plexus
   Klumpke’s palsy: trauma to C8-T1 lower trunk; (claw) hand paralyzed; see U/L Trunk Injuries
   Erb-Duchenne syndrome: trauma on C5, C6 upper trunk; shoulder paralyzed; see U/L Trunk Injuries
Osteology & Surface Anatomy

- Triangular area: **clavicle, first rib**, upper border of **scapula** - roots of brachial plexus and subclavian A/V pass thru here to enter axilla; apex of axilla
- **Coracoid process**
- **Humerus: intertubercular groove** = lateral border of axilla, **lesser tubercle** = subscapularis tendon inserts onto humerus
- Ampi = base of axilla, **anterior axillary fold** formed by pec major M
- **Posterior axillary fold** = teres major + latissimus dorsi Ms
- **Deltoid M**

Dissection

Structures w/in the posterior cervical triangle

- **Accessory N (CN XI)** = descends across posterior triangle, disappear under ant free edge trapezius
- **Subclavian V** = ant to anterior scalene M; lateral to 1st rib becomes the **axillary V**
- Two branches of thyrocervical trunk. Cross ant to ant scalene M from med to lat - **Transverse cervical artery** - across neck to trap M
- **Suprascapular artery** - | s clavicle, near subclavious M
- **Subclavian A** = post to ant scalene M cross 1st rib = **axillary A**
- **Dorsal scapular A** = off subclavian A post/lat to ant scalene M b/t parts of brachial plexus
- **Brachial plexus** = emerge b/t ant/mid scalene Ms; Ns to upper limb

Compression of Roots of the Brachial Plexus: ant/mid scalene M/1st rib narrow interval, can compress brachial plexus and subclavian A from spasm of ant scalene M. S/S: ? arterial pulse, paresthesia of limb (C8-T1 dermatomes). Also, can have a cervical rib - extension of C7 transverse process, can compress plexus/subC A as pass 1st rib

Short head of biceps & coracobrachialis Ms - attach to coracoid process, deep to pec major/minor

- **Lateral thoracic A** = off axillary A; courses w/in fat of axilla to reach **serratus anterior M**
- **Latissimus dorsi M** - attach to proximal humerus; N/blood: thoracodorsal N/A (thoracoDORSAL to latissimusDORS)
- Axillary sheath envelope **axillary A/V**
- Five branches of axillary artery:
  - **Thoracocervical trunk** = deep to medial pec minor, off superiorly
  - **Lateral thoracic A** = deep to lateral pec minor, off inferiorly
  - **Subscapular A** = largest branch of axillary = trunk of thoracodorsal A + circumflex scapular A
  - **Anterior circumflex humeral A** = off axillary A opposite subscapular A
  - **Posterior circumflex humeral A** = off axillary A deep to/larger than ant CHA; runs with axillary N
  - Major parts of brachial plexus: (really tiny dicks can break)
  - Five ventral rami of spinal Ns (C5,6,7,8,10) = roots of brachial plexus = post to ant scalene M
  - Trunks (3) = lat to ant scalene M @ 1st rib; superior, middle, inferior
  - **Upper (superior) trunk** = C5/C6 fuse lateral to an scalene M
  - **Middle trunk** = C7
  - **Lower (inferior) trunk** = C8/ T1
- **Divisions (3)** = deep to clavicle; 3 anterior, 3 posterior
  - **Cords (3)** = deep to pec minor, surround axillary A; medial, lateral, posterior
  - **Terminal branches (5)** = lat to pec minor; [mean men ultimately run away]
- **Musculocutaneous N** = pierces belly of coracobrachialis M, proximal origin from lateral cord
  - **Medial N** = from lateral/medial cords
  - **Ulnar N** = from medial cord
  - **Radial N** = deep to axillary A; from post cord; large, deep; passes obliquely to dorsum of arm
  - **Axillary N** = from post cord; courses sup to radial N; pass deep to surgical neck humerus w/posterior circumflex humeral A

Test for Possible Damage to Axillary Nerve: axillary N has cutaneous branch supplies skin over deltoid M. may be damaged following fracture of surgical neck of humerus, determine by testing sensation over skin of shoulder. Don’t test fx of deltoid, too painful

Upper/Lower Trunk Injuries: injure upper trunk (Erb-Duchenne)(C5,C6) by sudden excessive stretching of angle b/t neck and shoulder → weakness/paralysis of Ms of shoulder, limb hang limp in adducted shoulder, medially rotated arm, extended elbow; injure lower trunk (Klumpke’s palsy)(C8,T1) in sudden forcible upward stretch of upper limb, e.g. fall and catch on branch → paralysis of Ms of hand → “clawhand”

- **Suprascapular N** = from upper trunk (C5,C6), course lat to course w/suprascapular A
- **Long thoracic N** = courses vertically on **serratus anterior M**; from ventral rami C5,6,7; SAM forms medial wall of axilla

Winged Scapula: paralysis of serratus anterior M; SAM arises from several ribs, inserts a long medial border of scapula near inferior angle; action: protract (draw forward) as in throwing a punch; action w/trapezius: rotate scapula superiorly as in
reach high above head; damage long thoracic N, lose both fx's. TEST: look from behind which pt places both hands on wall and pushes; if paralyzed, medial border of scapula will project posteriorly.

- Thoracodorsal N - from posterior cord, to latissimus dorsi M
- Subscapularis M’s tendon attaches to lesser tubercle of humerus
- Subscapularis M + teres major + latissimus dorsi = posterior wall of axilla

Axilla [Dissection]

Anterior View
5.6.04
Human Limb Development
Dr. Snow

1. **Describe the formation of the limbs from the somite stage to the adult.**
   a. **trilaminar disc** (ectoderm, mesoderm, endoderm)
   b. **mesodermal subdivisions**
      i. **somites**
         a. **spinal cord segments**
            1. **sclerotome**
            2. **dermatome**
            3. **myotome**
            a. epimere → deep back Ms
            b. hypomere → skeletal Ms of ventral body wall
            c. near origin of limb bud
               i. posterior (extensor) mass
               ii. anterior (flexor) mass
      ii. **intermediate mesoderm**
      iii. **lateral plate mesoderm**

2. **Define the embryonic origin of the extensor and flexor compartments of the adult.**
   Myoblasts from myotomes near origin of limb buds migrate into limb bud; organize into 2 muscle blocks relative to long axis of embryonic limbs
   a. blocks near origin of limb bud
      i. posterior (extensor) mass
      ii. anterior (flexor) mass

3. **Define the rationale for the anterior and posterior divisions of the brachial plexus.**
   N’d by ventral primary rami of spinal Ns at each limb’s base (upper: C5-T1, lower: L2-S3). As each ramus penetrates base of bud, divides into anterior and posterior division along with the blocks, continues into adulthood as ant/post divisions of brachial plexus

4. **Describe the key steps in muscle formation (myogenesis).**
   Ant / post M blocks
   Small mononucleated cells (myoblasts) divide mitotically, then fuse to form syncitial cell (myotube) w/ nuclei in central position. Form M proteins assembled into myofibrils, which ? length/girth of myotubes. Central nuclei to periphery. Satellite cells (some myoblasts) are reserve cells in adulthood

5. **Describe the morphological expressions of the more common limb malformations.**
   (a) reduction defects
      ? full development of limbs; e.g. meromelia, amelia
   (b) duplication defects
      e.g. polydactyly
   (c) dysplastic defects
      Includes fusion and gigantism; e.g. syndactyly

The Thalidomide Story...
5.6.04
Physiology of Skeletal Muscle
Dr. Farley

1. Describe the cellular structure of a skeletal muscle fiber
   thick / thin filaments → sarcomere → myofibrile → muscle fiber → muscle
   spindle shaped M composed of bundles of M fibers → a single M cell 100µ x Xcms separated by
   sarcolemma contain bundles of smaller units of myofibrils → are contractile els of M, cylindrical stx in
   cytoplasm of M fiber, 1-2µ x Xcms containing repeating structure sarcomeres → ordered arrangement
   of thick & thin filaments w/ proteins for M contraction

2. Know the difference between Type I and Type II skeletal muscle fibers
   Type I: red fibers; mitochondria for aerobic contraction, slow twitch response, resistant to fatigue,
   efficient for generating sustained forces
   Type II: white fibers; depleted in mito, energy from glycolysis, fast twitch response, fatigue easily, for
   quick, intermittent movements

3. Name the major protein components of the thick and the thin filaments
   Thick: myosin
   Thin: actin, tropomyosin, troponin

4. Explain the sliding filament model of muscle contraction, including the relationship between crossbridge
   formation, force generation, and ATP hydrolysis
   thin / thick overlap; contraction → filaments slide past each other increasing overlap by breaking and
   reformation of crossbridges between thick myosin heads and thin actin molecules; swing from 90° to
   45° pulling thin past thick so pulling thin filaments closer → mechanical work
   energy for work from hydrolysis of ATP by myosin ATPase, which it does 100's x better when actin binds
   myosin
   Crossbridges are protein-protein contacts between the fibers, and the total force exerted by a muscle
   is proportional to the number of crossbridges between the filaments. Crossbridges break and reform
   during muscle shortening.

5. Describe the role of calcium in the regulation of skeletal muscle contraction
   Ca2+ binding to troponin C causes a conformational change of other troponins (I, T) which induces
   mov't of tropomyosin molecule from one position on actin to another and activates myosin ATPase
   and :: contraction of M

6. Distinguish between isotonic and isometric contractions
   isotonic: contraction resulting in a change in M length
   isometric: contractions that cause no change in M length

7. Describe the force-velocity relationship for an isotonic contraction
   when M shortens while lifting a load, rate/extent/duration of M shortening decreases as load increases, lag time between AP and shortening increases

8. Explain the length-tension curve for an isometric contraction
   curve: relation between amount of active force generated (to resistive force)
   and length of M = "active tension"; SkM narrow range; cardiac below optimal

9. Describe the mechanism whereby force of contraction can be increased by
   increasing the frequency of motor nerve firing
   AP short (5ms), twitch (10s-100s ms), refractory period short, can generate 2nd AP
   before M relaxed; :: ?stim freq → twitches fuse to yield max (tetanus) tension
   • Hydrolysis of ATP is required for muscle relaxation (need ATP to get Ca++ back in SR) and contraction
   • Malignant hyperthermia
   • Dystrophin defects: Duchenne's muscular dystrophy, Becker's muscular dystrophy
   • Maximum power output obtained when M moves against a load 1/3 maximum load can be lifted
A. Define and apply the terms, principles, and concepts shown in bold italic type.

IV. Muscular System – muscular system, skeletal Ms,

B. Contrast the basic features of skeletal muscle compared to cardiac and smooth muscle.

Skeletal: striated, long, move bone + attach, all-or-none, voluntary, motor NCB in CNS, axon via spinal Ns PNS
Cardiac: striated, short, branch, electrically coupled, constrict heart, rapid/continuous, involuntary,
smooth: nonstriated, fusiform, short, constrict lumen, slow, peristalsis, involuntary, pre/CNS post/ganglia PNS

C. Describe the hierarchical structure of muscle from molecules to whole muscle, including its connective tissue layers.

Epimysium → perimysium → M fascicle → endomysium → M fiber → myofibril → myofilaments actin/myosin

D. List the components of a motor nerve and specify the location of cell bodies for each nerve component.

Motor ventral horn SC; sensory pain/proprio-DRG spinal N; sym: (preT1-L2 lat grey), post (trunk, gray, plexus)

E. Define the term “motor unit” and give several examples of how motor unit size relates to muscle function.

ID: a single motor N and all the M fibers it innervates; size 1/α fineness of control (5= fine ctrl e.g. vocals)

F. Discuss how you would determine if a muscle is specialized for movement as opposed to power in terms of fiber length, cross-sectional area, and fiber architecture.

Force < cross-sectional area & # pennate fibers < to axis; mov’t < length & | | | fibers

G. Define shortening (concentric), isometric, and lengthening (eccentric) contractions of muscle and give several examples of each.

Concentric: M shorter in contraction; isometric: M same length in contraction; eccentric: M longer
Biceps short when flex with weight; same length if can’t lift; eccentric when lower weight back down

H. Discuss the role of tendons as part of the musculoskeletal system.

Fibrous cords of CT attach striated M to other stx; dense || collagen fiber bundles; provide length to a
M at low metabolic cost; in | | | ? mov’t; in \// Ms? mov’t

I. Define the terms prime mover (agonist), antagonist, fixator (stabilizer), and synergist relative to muscle function.

Prime mover aka Agonist: M regularly active in initiate & maintain a particular mov’t; e.g. brachialis flex
Antagonist: M oppose/resist action of a prime mover; e.g. triceps antagonize brachialis
Fixator aka stabilizer: M stabilize joint position & integrity; e.g. 4 rotator cuff Ms
Synergist: M eliminate unwanted mov’ts; e.g. triceps/shoulder Ms when biceps supinating

J. Discuss the importance of gravity and inertia in movement and muscle function.

Can be prime mover or antagonist; Ms control as antagonists eccentrically

K. Define the difference between anatomical action and muscle function when discussing what a muscle does.

Anatomical action: what M does concentrically act alone; M fx: what it does coordinated w/other Ms

L. List the anatomical actions of the deltoid muscle relative to each axis for movements that occur at the shoulder joint.

{actions – axis}; {actions of each set of fibers}
flexion/extension – transverse; ab-/adduction – anteroposterior; med/lat rotation – vertical
Anterior fibers – flex, med’l rotate, adduct; Lateral – abduct\\/\\/; posterior – extend, lat rotate, adduct

M. List the anatomical actions of the biceps brachii muscle and explain its role as an antagonist to gravity during extension at the elbow.

Weak flex arm @ shoulder; powerful flex forearm @ elbow; supinate pronated forearm
Elbow extension: gravity prime mover; biceps smoothly control rate/amplitude w/ eccentric conx

N. Define pronation and supination, explain how the radius rotates relative to the ulna, and identify the muscles responsible for these movements.

Pronate = palm down; supinate = palm up
Radial head rotates in place proximal; distal, styloid process @ radius end moves 180° about ulnar head
Pronate: pronator teres, pronator quadratus. Supinate: biceps brachii, supinator

O. Discuss the roles of the biceps brachii, supinators, triceps, and shoulder muscles in supination (S’n).

Supinators: prime mover – slow unresisted S’n, fast S’n w/ elbow F’d, fast S’n w/ elbow E’d, vs resistance
Biceps: prime mover – fast S’n w/ elbow flexed, S’n vs. resistance
Triceps: synergist – fast S’n w/ elbow F’d, S’n vs. resistance
Shoulder: stabilizers – fast S’n w/ elbow F’d, S’n vs. resistance
1. Describe muscle fiber type characteristics and adaptations to exercise and aging.

Type I – fast-twitch < exercise = endurance athletes
Type II – IIa – fast shorten, E transfer aerobic & anaerobic = fast-oxidative-glycolytic FOG < sprint athletes
    IIb – GREATEST anaerobic potential, most rapid shortening velocity = “true” fast-glycolytic FG
Aging: decrease # & area type II; M fiber atrophy & loss of motor units; 1st concentric then eccentric

2. Explain the neuromuscular and hypertrophic adaptations that are responsible for enhancing skeletal muscle force production.

NM adaptations:
1) increased motor unit recruitment
2) increased firing frequency of motor units
3) increased motor unit synchronization
4) increased activation of the synergist muscles, coordinate firing
5) increased inhibition of antagonist muscles

Hypertrophic Factors:
1) hypertrophy
2) increase II : I
3) increase myofilament packing

3. Describe the process of exercise induced muscle damage and its importance to muscular adaptation.

delayed-onset muscle soreness (DOMS) appears later and can last for 3 or 4 days.
DOMS may be produced by one or a combination of the following factors: 1) Microscopic tears in muscle tissue or damage to its contractile components with accompanying release of creatine kinase (CK), myoglobin (Mb), and troponin I, 2) Osmotic pressure changes that cause fluid retention in the surrounding tissues, 3) Muscle spasms, 4) Overstretching and tearing of the muscle’s connective tissue, 5) Acute inflammation, and 6) Alteration in the cell’s mechanism for calcium regulation.

Once the muscle fiber is injured (e.g. the sarcolemma is damaged) large amounts of calcium may enter the cell, which is cytotoxic.

The muscle damage associated with exercise acts as a stimulus for initiation of the repair process and adaptation. The greater the magnitude of muscle fiber damage the greater the compensatory adaptation. It has been demonstrated that eccentric compared to concentric resistance training results in greater muscle damage and hence greater muscle hypertrophy. Therefore, the muscle fibers undergo repair and remodeling to build a muscle that can meet the functional demands placed on it.

4. Explain the role of the satellite cell in skeletal muscle repair and adaptation.

Activation of muscle via specific types and intensities of long-term use stimulates otherwise dormant myogenic stem cells (satellite cells) under a muscle fiber’s basement membrane to proliferate and differentiate to form new fibers. Fusion of satellite cell nuclei and their incorporation into existing muscle fibers enables the fiber to synthesize more proteins to form additional myofibrils. This most likely contributes directly to muscular hypertrophy with chronic overload and may stimulate transformation of existing fibers from one type to another.

5. Explain the factors that contribute to improving muscle mass and strength.

Six primary factors contribute to the development and maintenance of muscle mass and strength:

1) Genetics,
2) Nervous system activation,
3) Physical activity,
4) Nutritional activity,
5) Endocrine influences, and
6) Environmental factors.

Of these factors, resistance training & anabolic steroids are the two most common & effective interventions.
Functional Anatomy of the Hand and Forearm
Dr. McNeill

1. Know the basic anatomical movements of the wrist, fingers, and thumb.
Fingers: Abduction away from / adduction towards - sagittal plane thru 3rd digit;
Thumb: abduction & adduction ⊥ to palm plane

<table>
<thead>
<tr>
<th>Joint</th>
<th>Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>IP, 1-5</td>
<td>flexion &amp; extension</td>
</tr>
<tr>
<td>MP/MCP, 1-5</td>
<td>flexion &amp; extension, abduction &amp; adduction, limited rotation</td>
</tr>
<tr>
<td>Carpometacarpal, 1-5</td>
<td>flexion &amp; extension, abduction &amp; adduction, rotation</td>
</tr>
<tr>
<td>Midcarpal (IC) &amp; radiocarpal/wrist</td>
<td>flexion &amp; extension, abduction &amp; adduction</td>
</tr>
</tbody>
</table>

2. Know the difference between the innervation and anatomical functions of the muscles located in the medial and lateral compartments of the forearm.

Flexor wad attaches to **medial** epicondyle of humerus/ Anterior Forearm
- **Median nerve:**
  - Pronator teres (pronates forearm)
  - Pronator quadratus (pronates forearm)
  - Palmaris longus [may be absent] (flexes hand)
  - Flexor carpi radialis (flexes & abducts hand)
  - Flexor digitorum superficialis (flexes fingers at MP & proximal IP joints)
  - Flexor digitorum profundus [part for lateral 2 fingers] (flexes fingers at MP & both IP joints)
  - Flexor pollicis longus (flexes thumb at MP & IP joints)

Ulnar nerve:
- Flexor carpi ulnaris (flexes & adducts hand)
- Flexor digitorum profundus [part for medial 2 fingers] (flexes fingers at MP & both IP joints)

Extensor wad attaches to **lateral** epicondyle of humerus/ Posterior Forearm
- **Radial nerve:**
  - Brachioradialis (flexes forearm)
  - Extensor carpi radialis longus (extends & abducts hand)
  - Extensor carpi radialis brevis (extends & abducts hand)
  - Extensor carpi ulnaris (extends & adducts hand)
  - Supinator (supinates forearm)
  - Extensor digitorum (extends fingers)
  - Extensor indicis (extends index finger)
  - Extensor digiti minimi (extends little finger)
  - Extensor pollicis longus (extends thumb)
  - Extensor pollicis brevis (extends thumb)
  - Abductor pollicis longus (abducts thumb)

3. Know the innervation and anatomical functions of the intrinsic muscles of the hand.

**Median nerve, recurrent branch:**
- Opponens pollicis (rotates & abducts 1st metacarpal during opposition)
- Abductor pollicis brevis (abducts thumb)
- Flexor pollicis brevis (flexes thumb)
- Lumbricals [lateral 2 muscles] (flex at MP & extend at IP joints)

**Ulnar nerve:**
- Opponens digiti minimi (rotates & abducts 5th metacarpal during opposition)
- Adductor pollicis (adducts thumb)
- Flexor digiti minimi (flex little finger)
- Dorsal interossei [4 muscles arranged around middle finger as central axis of hand] (abduct 2nd, 3rd, & 4th fingers; flex at MP & extend at IP joints)
- Palmar interossei [3 muscles arranged around middle finger as central axis of hand] (abduct 2nd, 4th, & 5th fingers; flex at MP & extend at IP joints)
- Lumbricals [medial 2 muscles] (flex at MP & extend at IP joints)

4. Describe the blood circulation in the hand.
Deep palmar arch = radial A ⬌ anastomose with → superficial palmar arch = ulnar A, test blockage with Allen’s test

5. Know the cutaneous innervation of the arm, hand, and fingers.
Mca = medial cutaneous nerve of the arm; MF = medial cutaneous of forearm

6. Describe the difference between the resting position of the normal hand and the hand of a patient with a median nerve, ulnar nerve, or radial nerve injury.
- Median N Lesion = Ape hand = paralyze thenar - thumb adducted, straight, extended
- Ulnar = claw hand = paralyze interossei, medial 2 lumbricals, adductor pollicis; MP hyperE, IP F, thumb abducted
- Deep Radial = wrist drop = lose E wrist/fingers; can E IPs (lumbricals), cutaneous OK
Review of Week One Gross Anatomy
5.10.04
GA Dissection 22A: Posterior Forearm & Dorsal Hand

Osteology & Surface Anatomy
- **lateral epicondyle** of humerus – posterior forearm Ms (extensors) attach here
- **head of ulna** – bump on distal little-finger side of forearm, just proximal to wrist
- **styloid process of ulna** – distal to head of ulna
- **styloid process of radius** – distal most of radius; Clinical: slightly DISTAL to styloid of ulna (~½ cm) (Colle’s fracture)
- **metacarpals**: knuckles of fist are distal ends
- **phalanges** (proximal, middle, distal); thumb has no middle
- Thumb extension / abduction at 90° angles to digits 2-5 (extension || plane of hand; abduction away from hand)
- **8 Carpals** of wrist; proximal/distal rows; lat to med, prox to dis:
  - Scaphoid
  - Lunate
  - Triquetrum
  - Pisiform
  - Trapezium
  - Trapezoid
  - Capitate
  - Hamate

  Some Lovers Ty Positions That They Can’t Handle
  Scaphoid Lunate Triquetrum Pisiform Trapezium Trapezoid Capitate Hamate
- **anatomical snuff box** = tendons that raise skin at jxn thumb/wrist = (post) extensor pollicis longus + (ant) abductor pollicis longus, extensor pollicis brevis (abductor inserts more proximally)

Dissection
- **extensor retinaculum**
- superficial group of extensor Ms of forearm (lat/thumb to med/pinky):
  - brachioradialis (flexor)
  - extensor carpi radialis longus
  - extensor carpi radialis brevis
  - extensor digitorum
  - extensor digiti minimi
  - extensor carpi ulnaris
  - brachioradialis M – doesn’t follow developmental rule (posterior = radial N = extensor), it’s a flexor of elbow, still radial N
  - deep layer of extensor Ms of forearm
    - supinator
    - abductor pollicis longus
    - extensor pollicis brevis
    - extensor pollicis longus
    - extensor indicis
  - VAN btn deep / superficial layers
  - deep radial N exits distal supinator
  - skin on dorsum of hand: loose vs palmar surface. Dorsal venous arch site for IVs
  - index finger: 2 independent tendons (extensor indicis, extensor digitorum)
  - little finger: 2 tendons (independent extensor digiti minimi, slip from extensor digitorum)
  - Can you do this? Middle finger against palm, hand flat against surface, try to raise ring finger. Good luck
  - Abduction of wrist: extensor carpi radialis longus and brevis and flexor carpi radialis
  - Adduction of wrist: extensor carpi ulnaris and flexor carpi ulnaris
  - dorsal interossei Ms – between adjacent metacarpals on dorsum of hand
    - Dorsal Interossei Muscles: ? 4; each has 2 bellies from shafts of metacarpal bones; insert into proximal phalanges of Fs2,3,4; N’d = ulnar
  - radial artery – cross floor of snuff box btn extensor pollicis longus and brevis; distally dive deep btn heads of origin of 1st dorsal interosseous M
5.10.04
GA Dissection 22B: Leg and Foot

**Osteology and Surface Anatomy**
- **medial malleolus** - distal end of tibia
- **lateral malleolus** - distal end of fibula; proximal fibula has no action at knee
- **greater saphenous V.** - ant to med malleolus
- **lesser saphenous V.** - post to lat malleolus
- **posterior tibial A** - btn medial malleolus & calcaneal tuberosity
- **ankle joint.** - articulation between tibia, fibula, talus
- **calcaneal tendon (Achilles tendon)** attaches to calcaneal tuberosity on calcaneus; continuous proximally with calf Ms
- **lesser saphenous V.** - post to lat malleolus
- **posterior tibial A** - btn medial malleolus & calcaneal tuberosity
- **ankle joint.** - articulation between tibia, fibula, talus
- **calcaneal tendon (Achilles tendon)** attaches to calcaneal tuberosity on calcaneus; continuous proximally with calf Ms
- **medial malleolus** - distal end of tibia
- **greater saphenous V.** - ant to med malleolus
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- **posterior tibial A** - btn medial malleolus & calcaneal tuberosity
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- **calcaneal tendon (Achilles tendon)** attaches to calcaneal tuberosity on calcaneus; continuous proximally with calf Ms

**Dissection**

**The Leg - Posterior Compartment**
- **gastrocnemius M** - & its 2 heads of origin, medial and lateral
- **plantaris M**; deep to lateral head of gastrocnemius, its tendon (freshman’s N) long, skinny, pass between gastrocnemius and soleus to insert in common with calcaneal and soleal tendons
- **Achilles Tendon Reflex**: examiner dorsiflexes ankle to stretch calcaneal tendon; tap calcaneal tendon → short, quick plantarflexion. **TESTS**: S1 (guide says S2)
- **Popliteal A / Tibial N** - cross popliteus M & disappear deep to soleus; descend down angling toward med malleolus
- **Deep Ms of posterior compartment:**
  - **Flexor digitorum longus M** (medial most)
  - **Tibialis posterior M** (in middle)
  - **Flexor hallucis longus M** (lateral most)
- **Popliteal A branches into posterior & anterior tibial As** inferior to popliteus M proximal attachment; anterior tibial A dive deep to reach anterior compartment
- **Structures in interval btn med malleolus & calcaneal tuberosity under flexor retinaculum. Start immediately posterior to med malleus [Tom Dick And Harry]:**
  - **Tibialis posterior tendon**
  - **Flexor Digitorum longus tendon**
  - **Posterior tibial Artery**
  - **Tibial Nerve**
  - **Flexor hallucis longus tendon**
A. Understand the Structure & Function of Collagen in Soft Tissues

resilience and elasticity
- The three chains coil together in a right handed triple helix to form a molecule of collagen
- 2/3 of the molecule consists of the following amino acids: glycine, proline, hydroxyproline. Every third amino acid is glycine, essential for the formation of the triple helix
- Crosslinks between the chains and the molecules of collagen which give collagen its strength
- As collagen matures, the crosslinks become more stable.
- Arrangement of collagen fibers in tendons is parallel, equipping them to withstand high unidirectional tensile loads.
- The arrangement of collagen fibers in ligaments are not completely parallel. They sustain loads mainly in one direction, but can bear smaller tensile loads in other directions.
- Collagen stronger at higher speeds.

B. Be Familiar With Three Common Soft Musculoskeletal Tissue Injuries:

1. Meniscus
   - Behave under compression, tension, & shear; fx: load-bearing, shock absorption, joint stability, joint lubrication.
   - Poor healing ability – vascularity is peripheral
   - Don’t repair, yes meniscectomy → OA in long term.
2. Ligament
   - Anterior Cruciate Ligament Injury: noncontact deceleration situation that produces a valgus twisting injury - when the athlete lands on the leg & quickly pivots in the opposite direction; tibia comes forward.
   - Associated Injuries: Meniscal tear - 62% - lateral > medial, Capsular tears - 21%, Chondral fracture - 10%.
   - Nonoperative (brace, educate, rehab) vs. operate (younger, isometric graft placement).
3. Tendon
   - Tight collagen bundles, tensile strength for repetitive motion.
   - Pathology: age related.
   - Older patient (>40) think of possible rotator cuff tear with dislocation of shoulder until proven otherwise.

C. Appreciate the Healing/Surgical Treatment of These Tissues as it Pertains to the Athlete / High Demand Pt.

Arthroscopy:
- Minimally invasive, definite dx, excise pathology, suture/anchor repair in joints, early recovery, less cost.
Rehab:
- I - recovery; II - motion; III - strengthen; US/ electrical stimulation; no pain with full ROM.

D. Know the Treatment Plans for These Common Injuries

Meniscus
- Resect torn portion
- Repairs rare, on younger pts.
- Long term arthritis develops without intact meniscus.
Ligament
- Older patient (>40) think of possible rotator cuff tear with dislocation.
- Anterior Cruciate Ligament Injury
  - Nonoperative – brace, rehabilitation, education.
  - Operative – sudden deceleration injury, younger pt, controlled rehab – return to sport 6-12 months, graft choice (bone tendon-bone - 90% graft tx, hamstring tendons, allograft).
  - Isometric graft placement.
  - Associated injuries: meniscus 50%, articular cartilage 20-25% true damage.
Tendon
- Tight collagen bundles, tensile strength; pathology age related.
- PT to increase motion & strength and decrease pain.
- If PT fails, need surgical correction - open vs. arthroscopic, subacromial decompression, too; less likely to perform surgery on an older pt.
- Rotator cuff tendon most commonly torn (vs. e.g. achilles, patellar, biceps, ...).
A. Understand what an athletic trainer is and the domains of athletic training
   1. Injury Prevention
   2. Recognition, evaluation and assessment
   3. Immediate Care
   4. Treatment, rehabilitation and reconditioning
   5. Organization/administration
   6. Professional development and responsibility

B. Appreciate the dynamic of the USC Department Athletic Medicine and its team approach
   1. Team Physicians
   2. Certified Athletic Trainers
   3. Specialists
   4. Student Assistants

C. Understand the challenge of some pertinent injuries
   1. MRSA skin and soft tissue infections
   2. Syndesmosis Ankle Sprains
      - high ankle sprain - more debilitating, ant/inf tib/fib ligament
      - vs. inversion - etiology different
   3. Concussion Management
5.11.04
Biomechanics of Fractures
Dr. Jackson Lee

• Understand factors that contribute to fractures
  o metabolic factor - osteomalacia: renal osteodystrophy, disturb Ca++ metabolism, ? Ca++in matrix, nl amount of matrix; decrease in bone quality
  o metabolic factor - osteoporosis: Ca:Matrix nl, decrease in bone quantity
  o strength – cross-sectional diameter: small w/ thick walls stronger
  o Fractures occur on tension side of force application
  o Bone is weaker in tension than in compression
  o Rate of force application: rapid – fracture, slow – ligament failure
  o Femoral neck fracture in young pt \textit{à} SURGICAL EMERGENCY

• Understand factors that contribute to fracture healing
  o Metabolic
    o Nutrition (need more calories)
    o Smoking
    o NSAIDs
  o Local biology
    o Blood supply
  o Mechanical
    o Primary bone healing
      ▪ Stabilize fracture rigidly – eliminate all motion at fracture
      ▪ Bone heals by remodeling; skips earlier stages
    o Failure - nonunion / malunion

• Understand principles of fracture treatment
  o Goals:
    o Achieve union in shortest time
    o Restore function to pre-injury state / ASAP
    o Maintain proper alignment for function / axial alignment; restore anatomy
    o Minimize complications
    o Cast, internal fixation

• Understand Complications of fractures
  o Adult respiratory distress syndrome (ARDS) – multiply injured pts
  o Fat emboli syndrome – release of marrow contents into systemic circulation
  o Pulmonary emboli – blood clots from injury and immobilization
  o Compartment syndrome – accumulation of fluid in a compartment of fixed volume
    5 P’s: \textit{Pain}, Pressure, Paralysis, Pulselessness, Pallor
  o Nerve injury – e.g. common peroneal, axillary, radial
  o Vascular injuries – e.g. superficial femoral if fx distal femur; popliteal A w/ knee dislocation
  o Injury to surrounding soft tissues

Summary
  ✞ Bone is living tissue
  ✞ Fractures occur when forces are applied to bone that exceeds it’s strength
  ✞ Bone fails in tension
  ✞ Deforming forces results in loss of anatomy
  ✞ Fracture treatment is guided by the restoration of function
1. Gain familiarity with the mechanism of injury, clinical findings, and basic treatment of ankle sprains

- Most common injury to ankle region
- **MOI:** inversion while ankle plantar flexed
- Most common injury presenting to emergency rooms
- **PE:** edema, tenderness, ecchymosis over lateral ankle Ls, poss medial tenderness (deltoid ligament), poss tenderness above ankle (syndesmotic sprain, high ankle sprain)
- Radiographs: R/O fracture; 2nd set if persistent pain >6 weeks
- **Tx:** RICE = Rest - ace wrap, gel brace, lace up brace, cast, crutches; Ice; Compression; Elevation

2. Become familiar with pathoanatomy, physical examination, and treatment principles of bunions

- Painful bunions / hallux valgus
- Bunion sx most commonly performed foot sx
- **Pathoanatomy:** Hallux valgus = deviation of great toe away from midline, bunion is prominent medial eminence, not exostosis (benign proliferation of bone); 8F:1M affected - high-heeled, narrow-toed shoes
- **PE:** Prominent medial eminence, tenderness to palpation; generally no pain with hallux MP joint ROM; splayed forefoot; radiographs: splay 1st 2 metatarsals and hallux valgus; no degeneration
- **Tx:** Conservative: wide shoes, low heels, stretch leather over bunion $5/shoe
  Surgical: ONLY for pain, NOT cosmesis; not for prophylaxis - won’t progress; sx - osteotomy → prolonged disability

3. Understand pathogenesis and basic treatment principles of diabetic foot ulcers

- Most inpatient days for diabetic complications
- **Pathogenesis:** Ulcers tertiary to excess pressure, neuropathy (painless), & vasculopathy
- Neuropathy: sock of numbness; monofilament test - 10g of pressure is protective sensation
- Radiographs: bony destruction with osteomyelitis or Charcot arthropathy
- **Tx:** Conservative: debridement, antibiotics, total contact casting; post-healing - custom orthotics w/ excavation beneath site of ulcer for LIFE; surgical revascularization pm
- **TCO2** (transcutaneous O2 levels): <20 likely won’t heal; 20-30 iffy; 30+ probably heal
- Callus care: relieve pressure, shoe insert; NO corn pad

4. Understand pathoanatomy, clinical findings, and treatment of plantar fasciitis

- Most common source of disabling foot pain
- **Pathoanatomy:** 90% of time inflammation / microscopic tearing of plantar fascia origin from inferior aspect of calcaneus;
  - 50% pts w/ heel pain have spur; 25% gen pop has spur w/o heel pain
- **PE:**
  - Point tender over medial calcaneal tubercle (plantar fascia origin); 80% std dev above ideal weight;
  - Tight calf; 80%+ can’t dorsiflex w/knee extended → overstretch plantar fascia when walk
- **Tx:**
  - Address overuse (e.g. lose weight)
    - Stretch calf
    - NSAIDs
  - Heel orthotic, e.g. heel cup, pad
  - Warm soak
  - PT
  - Night splint (2nd office visit)
  - Steroid injections (3rd+ office visit) - Sheila's mom
  - Short leg walking cast 4-6 wks
  - Sx to partially release medial plantar fascia ONLY after 6 months of failed active conservative treatment

Summary:
1. Most chronic foot pain amenable to address underlying pathology, e.g. tight calf, overuse fasciitis
2. Vast majority of foot pain from acquired deformities amenable to nonoperative treatment
A. Understand the basic functional/mechanical characteristics and principles of walking.

B. Define the terms and sequence of events used to describe the gait cycle during walking.

C. Analyze the movements that take place at the hip, knee, and ankle joints during the different parts of the gait cycle during walking.

D. Discuss the roles of the major muscles of the lower limb during walking.

E. Describe the roles of muscles versus ligaments in maintaining posture of the upper and lower limbs.
Osteology & Surface Anatomy

- **Metacarpals**
- **Phalanges** (proximal, middle, distal; Note: no middle on thumb)
- **Thumb extension / abduction at 90° angles to digits 2-5 (extension | | plane of hand; abduction away from hand) [sign language ‘mnemonic’]
- **3rd digit doesn’t move much when fingers ab/ad-ducting; it’s the point of reference to define finger ab/ad-duction; 3rd digit mov’t always abduction, whether medial or lateral**
- **Mov’ts of the thumb: extension/flexion; ab/ad-duction; opposition**
- **Thenar eminence** = fleshy mass at base of thumb; **hypothenar eminence** = fleshy mass along length of 5th metacarpal
- **Webbing btn thumb/1st finger = adductor pollicis M on palmar side; first dorsal interosseous on dorsal side**
- **Surface landmark: recurrent branch of the median N (million dollar N) where pierces Ms of thenar eminence; resting hand → flex middle finger till touches palm = loc’n of N**

Dissection

- Three Ms of **Thenar Eminence**
  - **Abductor pollicis brevis** - most superficial
  - **Flexor pollicis brevis** - closest to center of palm
  - **Opponens pollicis** - deep to abductor pollicis brevis; inserts along shaft of 1st MC (metacarpal), ? cross metacarpophalangeal joint. :: action only on metacarpophalangeal joint of thumb
- **Adductor pollicis M** - seed in webbing; N’d by ulnar branch
- Three Ms of **Hypothenar Eminence** [not UFO, but AFO]
  - **Flexor digiti minimi**
  - **Abductor digiti minimi**
  - **Opponens digiti minimi**
- **Palmar aponeurosis; continuous w/ tendon of palmaris longus M**
- **Flexor retinaculum** (aka transverse carpal ligament) - broad, thick ligament btn carpals bones btn (hypo)thenar eminenci
- **ulnar N passes on little finger side, superficial to flexor retinaculum & deep to extensor retinaculum, forms the superfical palmar arch; accompanies ulnar A; divides into superficial & deep ulnar branches distal to pisiform bone (medial)**
- **common digital As** - course distal from arch to webbings of fingers
- **proper digital As** - distal to proximal IP (interphalangeal) joint → terminal branches of a common digital A; course along sides of each digit; accompanied by proper digital Ns
- **Carpal Tunnel: median nerve terminates as multiple digital Ns on radial side (digital Ns from ulnar on medial/ulnar side); recurrent branch of Median N** - distal to flexor retinaculum to thenar eminence Ms

- **Carpal Tunnel Syndrome: unyielding boundaries, 9 tendons (4 flexor digitorum superficialis, 4 flexor digitorum profundus, 1 flexor pollicis longus), their synovial sheaths, one large nerve; frequent mov’t of fingers/wrist → overuse → inflammation of tendons or synovial sheath → swelling in tunnel → Median N damaged by pressure from swelling → s/s: weak thenar Ms, tingling of 1st-3rd digits, thenar atrophy**
- **Synovial sheaths** - surround tendons of long finger flexors; thin, opaque, LCT

- 4 **flexor digitorum superficialis tendons** - distal forearm → carpal tunnel → distal fingers; near MP, tendons disappear into .fibrous digital sheaths.
- Tendon of flexor digitorum superficialis splits to allow **flexor digitorum profundus tendon** to continue distally; 4 tendons pass thru carpal tunnel w/median N and flexor digitorum superficialis tendons
- **Flexor pollicis longus tendon** - flexes distal phalanx of thumb
- 4 **lumbicals** - ARISE FROM tendons of flexor digitorum profundus
- Deep palmar space: deep palmar arch lies on interossei Ms
- **Palmar (3, adductors) / dorsal (4, abductors) interossei Ms**
- Interossei Ms: dorsal – abductors of MP joints 2,3,4 Fs, DAB; palmar - adductors 2,4,5, PAD
- Tendons of **lumbical Ms (radial side) & interossei Ms (dorsal - radial, abduct; palmar - ulnar side, adduct) insert into sides of Extensor aponeurosis; interossei & lumbicals flex at MP, extend at IP
5.13.04
Non-narcotic analgesics
Dr. Gopalakrishna

513 1 Non-narcotic Analgesics.pdf
513 2 NSAIDS.pdf
513 Gopa review.pdf
1. **Describe normal shoulder anatomy and function.**
   **Major Muscles of Shoulder:**
   - Serratus anterior - protract scapula
   - Trapezius, levator scapulae, rhomboids - elevate & rotate scapula
   - Latissimus dorsi, pectoralis major - adduct and internally rotate humerus
   - Deltoid - forward flex, abduct, extend arm
   - **Subscapularis, supraspinatus, infraspinatus, teres minor - rotator cuff**

2. **Appreciate the pathophysiology of the most common shoulder problems.**
   - **SUPRASPINATUS TENDON** - most common site of pathology
     - Rub tendon on under surface of arch (coracoid - coracoacromial L - ant acromion) → "impingement" syndrome; common in pt > 40y/o
     - Impingement from repetitive overhead activity & pinch cuff beneath arch; sports, work, idiopathic
     - Pain insidious or sudden after incident of overuse
     - Pain - lateral aspect of upper arm; ~ go distally; worse @ night & when reach overhead

3. **Describe the epidemiology of rotator cuff pathology.**
   - < 30 → shoulder instability w/ impingement & inflammation
   - > 40 → chronic rotator cuff / supraspinatus tendonitis
   - > 60 → partial / full-thick ness tears of supraspinatus tendon & OA common
   - Sudden with injury → mechanical event, e.g. tear; insidious → degenerative ds
   - ? pain over time → progressive pathology; ? weak → painful inhibition; profound loss of strength → progressive disrupt muscle-tendon unit, possibly rotator cuff
   - ? stiff (loss of ROM) → inflammatory, possible adhesive capsulitis (frozen shoulder)
   - **Site of pain is important**
     - Front of shoulder → subacromial pain; a veces radiation to deltoid insertion (also deltoid radiation in glenohumeral and cervical (C5) pathology)
     - Posterior joint line → glenohumeral
     - Involve hand → neurological or neurovascular

4. **Assess the risk factors commonly associated with rotator cuff disease.**
   - **Age:** 40 or older
   - **Heavy lifting**
   - **Activities that involve repetitive overhead arm motion, e.g. baseball**
   - **Weakened shoulder muscles from inactivity**

5. **Understand some principles of medical and surgical treatment of rotator cuff disease.**
   - **Corticosteroids**
     - Inject into area of inflammation
     - **S/E:** infection, atrophy / weaken CT with repeated use
   - **PT**
     - Exercise to strengthen rotator cuff to hold humeral head down and away from coracoacromial arch, reducing risk of further impingement
   - **Surgery**
     - If pathology too extensive to tx arthroscopically, open repair of rotator cuff

**Cases:**
- Fall off bike → clavicle fracture
- 10-foot fall backward onto arm / shoulder → fracture of humerus
- Fall while skiing, severe pain, can’t move shoulder → shoulder dislocation, usu. Anterior
- Trip & fall on steps onto front of shoulder → AC (acromion/clavicle) separation; no tx, self-healing
5.13.04
PPP Case - Carpal Tunnel Syndrome
Dr. Cantwell

1. Obtain a focused history on a patient complaining of numbness of the fingers.
   More focused ?s:
   - When does the numbness/tingling occur? (time of day, body position, relation to provocative activity)
   - Does it occur when you are asleep or driving?
   - What do you do to relieve the numbness?
   - Do you have problems dropping things or picking up coins?
   - Do you have diabetes, thyroid disease, joint or vessel inflammation?
   - Have you ever injured your neck, shoulder, elbow, wrist?
   - Do you wear a tight wrist-watch or use tight clothing or bands about the wrist?

2. Describe the causes of CTS.
   Local compression of median N w/in carpal canal 2ary to narrowing or crowding the N in carpal tunnel; N is most superficial stx in canal, above finger flexor tendons & their tenosynovium

3. Describe the risk factors leading to CTS.
   - Space occupying lesions – recent or old wrist fracture, infection, local edema, ganglion or lipoma, aberrant muscle belly, foreign body, flexor tenosynovitis (e.g. seen with RA)
   - Systemic cond’ns – pregnancy (fluid retention), obesity, diabetes, thyroid dysfx, arthritis, amyloidosis
   - Overuse syndromes – postural habits of wrist joint which produce forced flexion or extension positions, constricting bands, bandages, watches around wrist; trauma
   - > 30 y/o; females 3-5x males

4. Describe the symptoms and signs of CTS.
   - Numbness & tingling of thumb, index and middle fingers starts overnight, waking the pt who then dangles or shakes hand over side of bed for relief
   - May provocate: neck extension, sleep on stomach w/ shoulder elevated & abducted, tote heavy shoulder bag, Driving car / wrist extension
   - Decrease sensation (e.g. to light touch and pin prick) over median nerve distribution; later motor
   - Slight thenar M atrophy
   - (+) Tinel’s sign, (+) Phalen’s sign

5. Perform the pathognomonic tests to diagnose CTS (Phalen’s sign, Tinel’s sign).
   - Tinel’s: tap directly over volar center of wrist; (+) if tingling or electric shock type feeling radiating to 1st-3rd fingers → N irritation
   - Phalen’s: hold pt’s wrist in full flexion for 30 sec. (+) if pt gets “numb/tingly” 1st, 2nd, or 3rd digit; test increases pressure in carpal tunnel

6. Be familiar with the differential diagnoses in CTS.
   - Carpal tunnel syndrome
   - Cervical radiculitis/ radiculopathy
   - Peripheral neuropathy
   - Brachial plexopathy (Injury, e.g. stretch brachial plexus
   - Thoracic outlet syndrome
   - Occlusive vascular disease
   - Nervous system / vascular system
   - Systemic disease

7. Describe the electromyographic and nerve conduction findings found in CTS.
   EMG + N conduction studies show increase in distal median sensory latency time and distal medial motor conduction time at level of wrist and early denervation of thenar muscles

8. Outline the non-operational and surgical treatment of CTS.
   Conservative: wrist splint (prevent provocative pos’n of wrist), NSAID (reduce inflammation/swelling)
   Next step: steriod injection
   Surgery: if conservative fail, incise over volar wrist and release transverse carpal ligaments (canal roof)
The Knee Joint
Dr. Slavin

A. List the bones entering into the formation of the knee joint, and compare the shape and size of femoral condyles as they relate to motion at the joint
Femur, tibia, patella
Femoral condyles are curved, but tibias are flat, so incongruency and unstable
Large bearing surface area of condyle

B. List the functions of the semilunar cartilages (menisci), collateral, and cruciate ligaments
Menisci: See 7 below; collateral: see 6 below; cruciate: see 4,5 below

C. Describe the fibrous capsule and synovial membrane of the knee
Fibrous capsule
- Anteriorly it's the quadriceps tendon, patella, and patellar ligament
- Iliotibial tract and patellar retinacula blend w/ capsule anteromedially & anterolaterally
- Posteriorly, capsule thin, but strengthened by ligaments
- Covers cruciate ligaments

Synovial membrane
- Responsible for secretion and absorption of synovial fluid
- Membrane lines fibrous capsule, not over cartilage or menisci

D. Describe the anatomy and function of the patellofemoral joint
- Articular surface of patella and patellar surface of femoral condyles; patella glides on patellar surface during flexion/extension of leg at knee
- Patella provides additional leverage to quadriceps during extension of knee by holding tendon forward and acting as a pulley
- Articular cartilage on patellar posterior surface injured by rubbing on quads \(\rightarrow\) chondromalacia
- Patellar fractures \(\rightarrow\) removal \(\rightarrow\) knee at dec mechanical advantage \(\rightarrow\) weakness/ disability

E. Describe the anatomical bases for injury to the knee - A case study - “The Unhappy Triad?”
See 8 below
Unhappy triad: torn anterior cruciate ligament, tibial collateral ligament, AND medial meniscus

1. Primarily hinge type synovial joint between rounded femoral condyles & flattened tibial condyles
2. Joint includes articulation between patella & patellar surface of femur between its condyles
3. Maximum stability of joint is in full extension when ligaments are most taut & articular surfaces are most congruent brought about partially by medial femoral rotation that helps lock knee at end of extension
4. Anterior cruciate ligament prevents posterior displacement of femur relative to tibia (or vice versa)
5. Posterior cruciate ligament prevents anterior displacement of femur relative to tibia (or vice versa)
6. Collateral ligaments are attached so as to prevent abduction/ adduction in all positions of knee but allow some degree of rotation when knee is flexed; prevent hyperextension
7. Medial & lateral menisci are slightly moveable, crescent-shaped, fibrocartilaginous wedges attached to tibia that increase congruity between rounded femoral condyles & flat tibial condyles
8. Knee joint injuries usually involve: (1) soft tissue damage to joint capsule; (2) torn or damaged menisci which then interfere with normal movement of femoral condyles on tibia, test - pain on lateral rotation indicates injury of lateral meniscus, pain medial rot'tn injury medial meniscus; (3) torn collateral ligaments that result in lateral instability of knee joint (test by trying to passively abduct/ adduct extended leg while thigh is held fixed); (4) torn cruciate ligaments that lead to anteroposterior instability of knee joint (test anterior cruciate for positive anterior draw sign, which is abnormal anterior movement of flexed leg when it is pulled anteriorly while thigh is held fixed; test posterior cruciate for positive anterior draw sign, which is abnormal posterior movement of flexed leg when it is pushed posteriorly while thigh is held fixed); (5) or some combination such as damage to medial meniscus when tearing medial collateral ligament because they are attached to one another
GA Dissection 24A: Upper Joints
GA Dissection 24B: Lower Joints
Objective of Hand Surgery: restore function
Don’t treat (especially with surgery) unless it’ll help function

I. Congenital Differences
   C. Embryology
      1. Upper limb develops 4th – 8th week
      2. Viscera & other internal organs develop at this time, any child with a congenital difference of the upper limb (& lower?) should be evaluated for other congenital differences
   D. Function
      1. Timing important. Assess: (1) patient status (2) limb viability (3) progression of deformity (4) “patterning” – by 3 years old, you’re used to what God gave you

II. Trauma
   A. Fractures – deform in relation to articular congruity (how bones line up), angulation, & rotation
      1. Extraarticular - NO rotational deformity
   B. Flexor Tendons
      1. Zones of Verdan
      2. “no man’s land” = Zone 2
         a. Lacerations difficult → scar formation

III. Infections
   B. Flexor tenosynovitis
      a. Tendons scarred if infection
      b. Classic signs of tendon sheath infection aka “Four Signs of Kanavel”:
         i. Diffuse swelling of finger
         ii. Tenderness along flexor sheath
         iii. Finger held in slight flexed position
         iv. Excruciating pain with passive extension of finger
   2. Necrotizing Fasciitis
      a. A medical emergency
      b. 38% mortality (18% at LAC + USC)
      c. Infects subQ tissue / fascia, Muscles spared; can watch progression

IV. Vascular (radial / ulnar Ars)
   B. Collateral good in most
   C. Repair w/ microvascular techniques
   D. Prior to procedures, confirm collateral circulation (Allens Test)
      1. Occlude radial & ulnar arteries → release one side → all fingers should regain circulation / pink up / test via pulse-ox
      2. Repeat releasing other Ar
      3. (+) result = some digits don’t regain circulation

V. Reconstructive
   B. Plan initial care & possible future procedures
   C. Allow for any subsequent procedures to be optimized

Carpal Tunnel Syndrome:
   Tinel’s sign – tap anterior wrist to elicit S/S; (+) if tap and ow
   Phalen’s sign – wrists flexed & pushing on each other for 30 seconds; (+) if elicit S/S
   Compression test – examiner’s thumb over carpal tunnel; (+) if elicit S/S

What do you do?
(1) Complete H&P
(2) Debride
(3) Save what you can
(4) Stabilize structure
(5) Think ahead for future reconstruction needs
5.17.04
Inflammatory and Metabolic Diseases of Muscles
Dr. Ehresmann

1. Know the clinical features that distinguish inflammatory myopathies from non-inflammatory myopathies

Idiopathic inflammatory myopathies clinical criteria: (polymyositis = 4 or 5; dermatomyositis = 3 or 4 + rash)

1. proximal muscle weakness
2. elevated serum levels of muscle enzymes (CPK, aldolase, AST, ALT, LDH)
3. myopathic changes seen by electromyography (EMG)
4. inflammation – muscle bx
5. skin rash = dermatomyositis
6. circulating myositis-specific auto-antibodies (MSAs) present in ~50% of patients

 Polymyositis: insidious weak 3-6 mo; shoulder, pelvis, neck, dysphagia \( \leftrightarrow \) esophageal, dysphonia – nasal voice; not facial / ocular Ms; pulmonary crackles / cardiac – EKG abnl, arrhythmia, cardiomyopathy, CHF;
(2) above; EMG triad: (1) \(^{\Delta} \) insertional activity, (2) high-freq discharge, (3) low amp, short duration; biopsy: necrosis & regeneration – infiltrate focal / endomysial; T-CD8+; replace w/ fibrous CT & fat; in some only type II fiber atrophy

 Dermatomyositis: polymyositis + cutaneous; rashes before proximal M weak; Gottron’s papules

 Juvenile dermatomyositis: polymyositis + cutaneous; rashes before proximal M weak; Gottron’s papules; eyelid discoloration, periorbital edema, shawl sign, V-sign; histopath – perivascular infiltrate infl’y cells – B-cell & CD4+; perifascicular atrophy; some amyotrophic demato- w/ fatigue \( \rightarrow \) Ca

 Non-inflammatory = metabolic \( \rightarrow \) abnormalities in M energy metabolism \( \rightarrow \) skeletal M dysfx

 1°: biochem defects, affect maintain ATP levels; 2°: endocrine disorders, e.g. thyroid, adrenal, electrolyte

 Polymyositis: insidious weak 3-6 mo; shoulder, pelvis, neck, dysphagia \( \leftrightarrow \) esophageal, dysphonia – nasal voice; not facial / ocular Ms; pulmonary crackles / cardiac – EKG abnl, arrhythmia, cardiomyopathy, CHF;
(2) above; EMG triad: (1) \(^{\Delta} \) insertional activity, (2) high-freq discharge, (3) low amp, short duration; biopsy: necrosis & regeneration – infiltrate focal / endomysial; T-CD8+; replace w/ fibrous CT & fat; in some only type II fiber atrophy

2. Understand the pathogenesis of inflammatory myositis

(1) immune mediated
(2) associated with other autoimmune diseases
(3) MSAs; in <50% of pts, but presence indicates clinical manifestation and prognosis

 i) anti-Jo-1 = most common MSA; vs. anti-histidyl-tRNA synthetase; more in polymyositis, anti-synthetase syndrome; anti-Jo-1 extra M\( \Delta \) features: interstitial lung disease, arthritis, Raynaud’s, mechanic’s hands; don’t sustain remissions; HLA-DR52
ii) anti-Mi-2 \( \rightarrow \) helicase; dermatomyositis; good tx response
iii) anti-SRP; rapid onset polymyositis; tx-resistant; \( \rightarrow \) cardiomyopathy, distal M weakness

(4) Genetic; HLA-DR3 \( \rightarrow \) risk inflammatory Ms; anti-Jo-1 \( \rightarrow \) HLA-DR52
(5) pathologic – cell mediated antigen specific cytotoxicity; CD8+; B-cells not in polymyositis or inclusion body, yes in demato-; inclusion body – rimmed vacuoles, amyloid deposits & prion proteins

3. Recognize other causes of muscle weakness that may mimic myositis

Medications: alcohol, clofibrate, corticosteroids, D-penicillamine, Zidovudine (AZT), statin drugs

 Test: Chem, EMG, forearm ischemic-glycogen storage; MRI; muscle bx histoch, histochem, EM, assay enzs; CXR; Ca screen

 Before start tx: M strength, clinical status, CXR, lung fx, swallow study, Ca; enzs: CK, aldolase, AST, ALT, LDH
Tx: PT – bed rest; severe inflmn + passive ROM exercises; corticosteroids – prednisone (90% response); other 10% - + methotrexate / azathioprine, other agents; hydroxychloroquine for cutaneous lesions of demato-
5.17.04
Pharmacology of Muscle Relaxants
Dr. Miller

1. Diagram the reflex arc and indicate the major sites of action of spasmolytic drugs

<table>
<thead>
<tr>
<th>Generic name</th>
<th>Trade name</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Diazepam</td>
<td>Valium</td>
<td>GABA-A agonist</td>
</tr>
<tr>
<td>*Baclofen</td>
<td>Lioresal</td>
<td>GABA-B agonist</td>
</tr>
<tr>
<td>*Tizantidine</td>
<td>Zanaflex</td>
<td>Alpha-2 block?</td>
</tr>
<tr>
<td>**Dantrolene</td>
<td>Dantrium</td>
<td>Ryanodine R block</td>
</tr>
<tr>
<td>Gabapentin</td>
<td>Neurontin</td>
<td>Ca channel block</td>
</tr>
<tr>
<td>**Riluzole</td>
<td>Rilutek</td>
<td>Glutamate release block</td>
</tr>
<tr>
<td>#Botulinum toxin</td>
<td>Botox</td>
<td>ACh vesicular release block</td>
</tr>
<tr>
<td>Cyclobenzaprine</td>
<td>Flexeril</td>
<td>5HT2 block?</td>
</tr>
</tbody>
</table>

* FDA approved for spasticity
** FDA approved for ALS / glutamate neurotoxicity
# peripheral action

2. Contrast the mechanisms of action of centrally and peripherally acting spasmyotics

- **Centrally acting**
  - baclofen: open K+/close Ca2+ → inhibit NT release, inhibit sub P fibers on dorsal hom
  - neurontin: block Ca2+
  - zanaflex: / pre-/post-synaptic inhibition, analgesic
  - rilutek: glutamate release blocker
  - cyclobenzaprine: block descending serotonergic projection on dorsal hom w/ 5HT2 Rs; muscarinic block → sedation/ hallucination

  **Peripherally acting**
  - dantrolene: block Ca++ release from sarcoplasmic reticulum, bind ryanodine R-gates Ca++ release → block actomyosin form’ n → / M contraction; Tx Malignant Hyperthermia
  - botox: block ACh vesicle release

3. Specify the clinical use of cyclobenzaprine and related centrally acting muscle relaxants

- relieve local muscle spasm from tissue trauma or muscle strain
- not useful for spinal injury or cerebral palsy
- block descending serotonergic projection on dorsal hom Ns
Local Anesthetics (LA)  Dr. Thangathurai

Notes:
- PEN: procaine (pseudoChE) ester novocain; LAX: lidocaine (liver) amide xylocaine
- High frequency pain transmission carried by small-diameter, lightly myelinated Aδ and non-myelinated dorsal root type C fibers
- Time course of regional anesthesia: (1) block sym Ns / vasodilation, (2) loss pain / temp, (3) loss touch / deep pressure, (4) loss motor
- Basic Chemistry: non-ionized - penetrate lipid membranes; ionized - active form
- Metabolism: esters-hydrolytic met’z by plasma pseudo-ChE to allergenic PABA; amides-hepatic P450
- Unwanted effects & Toxicity: *dosage, *administration, *use of vasoconstrictors

1. Explain the mechanism of action of local anesthetics and list pharmacological, chemical and physiological factors including local pH and pKA that determine the onset or intensity of blockade of nerve impulses.
   - reversibly block voltage-gated Na+ channels → ↓ ion influx → prevent nl depolzn → block AP cond’n
   - bind inside Na+ channel pore near intracellular end; better block activated/inactivated than at rest;
   - LAs only reach binding site inside cell, : cross lipophilic plasma mem via non-ionized, Lipid-soluble forms
   - effects: ↑excitation threshold; ↓ depolarization; ↓ AP amp; ↓ impulse propagation; ↓ excitability
   - Factors of intensity: penetration via uncharged base, lipid soluble, non-ionized; active form - charged cation, water soluble, ionized
   - ↔ Depends on pKa; LAs less effective at lower pH (e.g. infection, inflammed, ischemia) cuz ionized form can’t penetrate
   - relative lipid solubility - dependent on aromatic end of chemical structure
   - protein binding % - higher → longer action

2. List factors that influence the sensitivity of different nerves to local anesthetics and state which neurons are most susceptible to blockade and the expected order of sensory loss.
   Sensitivity Factors  Time course / expected order of sensory loss
   - Size: Fiber diameter & myelination 1. block sympathetic nerves & vasodilation
   - Activity: Firing frequency & duration of AP 2. loss of pain & temperature sensation
   - Anatomical fiber pos’n in nerve bundle 3. loss of touch sensation & deep pressure
   - Small, unmyelinated, less/short acting loner susceptible 4. loss of motor function

3. Describe the different metabolism of amide- and ester-type local anesthetics. See notes above

4. Describe the advantages and disadvantages of co-administering a vasoconstrictor like epi with LAs.
   - Faster onset & Lengthen duration of location by reducing blood flow to/from area w/ ↓ systemic toxicity
   - Not for high potent / long duration locals (bupivacaine); not for local to distal extremities, ear
   - Yes for ischemic nerve lesions, systemic effects (RR↑, tachycardia, anhythmia)

5. List common toxic effects of local anesthetics, what precaution should be taken to avoid toxic reactions and what measures can be used to treat adverse cardiovascular and CNS effects.
   - S/E: Numb tongue, tinnitus, sleepy, sedat’n, lighthead, dizzy, disoriented, M twitch, tremor, unconscious, sz, coma, resp arrest, CVS depression, cramping, brain damage; allergic reactions (esp. esters)
   - Tx: Benzo’s, short-acting barbs and M relaxants to control seizures; O2 and hyperventilation therapy, EPI
   - Contraindicate: KDA inka, bradycardia, arrhythmias, coag disorders, neuro ds (e.g. MS), local inflmn/infx

6. Compare routes of administration and clinical use of local anesthetics.
   - Surface (Tropical): spray mucosa → superficial sensible N endings; poor permeate intact skin; diagnostics e.g. broncho-/laryngo-scopy, eye sx; skin - BENZC/CAINE, mucous mem -TETRA-/LIDO-CAINE
   - Infiltration: inject into tissue w/vasoconstrictor (EPI); small sx; PRO/LIDO/BUPIVA/mepiva – CAINE
   - Block peripheral Ns (regional): inj near N trunks/plexus; mixed N: large dose +vasocons; sx extremities; LIDO/prlo-/BUPIVA-CAINE; spinal & epi-/peridural special forms
A. INTRODUCTION TO RHEUMATOLOGY

1. Identify that there is a great variety of rheumatic diseases. >100 systemic diseases & localized conditions
2. Recognize that rheumatic diseases affect all age groups. Overall 14% <18:3%;18-44:7%;45-64:35%;64+:52%
3. Appreciate the financial impact of rheumatic diseases on society. 1995 arthritis cost $82.5 billion = 1% GNP

B. RHEUMATOID ARTHRITIS

1. Become familiar with the definition and clinical picture of Adult and Juvenile Rheumatoid Arthritis.
   **Adult:** chronic, inflammatory, systemic disease, major target is synovial joints
   - symmetric involve; small joints of hand and feet, wrists, knees, ankles, shoulders, hips, any synovial joints
   - inflammation causes swelling & pain; morning stiffness and fatigue; duration ∝ RA activity; → joint damage, deformities, dysfx; 3F:1M
   - 1%-of-pop; bimodal incidence - 20s/30s & 50s
   **clinical correlation:** inflammatory activity waxes & wanes; inflammation & synovial proliferation - joint swelling - thick ken & ↑ volume, ↑ temp/erythema, pain/stiff, ↓ ROM; joint contractures; tissue damage → joint deformities, tendon ruptures, ↓ cartilage thickness, bone erosion, 2ary OA, joint ankylosis
   **Juvenile:** onset <16 y/o; modes of onset (vs RA): polyarticular (40-50%), mon/oligo-articular → iridocyclitis; systemic or acute febrile onset; rash salmon pink, (+) Koebner phenomenon
   - involves joints, presents with high fever, rash, hepatosplenomegaly, peri/myocarditis, lymphadenopathy
   - growth abnormalities: aggressive ds or tx w/ systemic corticosteroids; premature epiphyseal closure

2. Identify the role of genetics in rheumatoid arthritis. HLA-DR4, HLA-DRB1; families higher risk; QR/KRAA risk; seq also on Epstein-Barr virus Gp110 / E. coli heat shock protein dnaJ → molecular mimicry poss. MOA

3. Gain an understanding of the pathogenetic mechanisms of rheumatoid arthritis.
   - Synovial inflammation & prolif n mediated by humoral and cellular immune responses → tissue damage
   - initiating event, e.g. entry agent in synovium thru microcirculation → initial inflammatory response
   - bind Ag, Ab, RF in synovial tissue, fluid → complement sequence → biologically active products
   - synovium: edema, lining cell prolif, vasodilation, infiltration by T-cells, B-lymph, plasma, mono/macrophage → cytokines IL-1β, TNF-α, IL-6, IL-8, TGFB, PDGF, G-M-CSF, (lymphocytes) INF-γ, IL-10, (granulomas) IFN-γ, IL-4; (B-lymphocytes) IgGs, RFs, complement
   - PMNs engulf immune complexes, release lysosomal enzymes: elastase, cathepsin G, proteinase 3, collagenase, gelatinase, osteoclast diff' factor, IL-1, TNF-α
   - tissue damage: enzymes above & synovial fluid contents: oxygen radicals, PGE2, lysosomal/synovial enzymes
   - angiogenesis, synovium infiltrated by lympho-mono-cytes - fuel response
   - pannus formation - granulation tissue at the edges of synovial lining
   - no immune complex clearance on cartilage → perpetuation of immune inflammatory response

4. Identify the mechanisms of tissue damage. See tissue damage above
   Collagen degraded by synovial & neutrophil collagenases; proteoglys ans via neutral protease and elastase; collagen & matrix via stromelysin; bone resorption via osteoclasts (ODF, IL-1, TNF-α, TGF, PGE2)

5. Recognize the multisystemic nature of the disease.
   - Extraarticular RA manifestations: osteopenia, osteoporosis; anemia of chronic disease; M atrophy → disease or rheumatoid myopathy; rheumatoid nodules - painless in pressure areas = more severe ds
   - Some RA pts: ↑ circulating immune complexes IC in serum w/ deposition in organs; vasculitis - IC in vessel wall → inflammation, vascular obstruction, infarction; Pleuropulmonary; carditis; neurologic - peripheral or spinal N compression; ocular inflammation

6. Identify therapeutic modalities for RA and comprehend their mechanisms of action.
   - Pt/family education
   - RX: (a) NSAIDS; (b) disease modifying anti-rheumatic drugs (DMARDs) aka Remittive drugs - hydroxychloroquine, methotrexate, combo; (c) biologic drugs - TNF-alpha receptor dimer, monoclonal Ab; (d) corticosteroids - injections, small oral doses; (e) immunoabsorbent column; (f) PT, rehab - learn joint protection, energy conservation, program of ROM & M strengthen exercises; (g) reconstructive ortho sx
C. SERONEGATIVE (HLA-B27 associated) ARTHRITIDES or SPONDYLOARTHROPATHIES

1. Be familiar with the definition & clinical picture of Ankylosing Spondylitis, Reiter's syndrome & Psoriatic arthritis.

Common Clinical Features of HLA-B27 arthritides:
1. high incidence of spinal joint (axial skeleton) involvement
2. asymmetrical lower extremity joint involvement
3. men > women
4. high frequency of HLA-B27, esp. with spinal arthritis
5. rheumatoid factor (RF) usually absent

Ankylosing Spondylitis (AS)
ID: Chronic inflammatory systemic disease, with major targets of the sacroiliac joints, cartilaginous and synovial joints of the spine; symmetrical joint involvement (except peripheral joints); >90% HLA-B27 (+); young men (9:1)
clinical manifestations:
1. low back pain & stiffness of >3 mo duration unrelied by rest
2. limited motion of lumbar spine, esp. flexion
3. reduced chest expansion (<5cm), w/reduction of vital capacity
4. bilateral sacroiliitis (rarely unilateral) on x-ray when 1st seen
5. eventually 40% develop peripheral arthritis, mainly of hips, shoulders, and knees
   - Inflammatory Δs start sacroiliac → progress cephalad to cervical spine; inflamed area heals w/ankylosis of joints and ossification of paraspinal ligaments; stiff posture & walk w/stiff gait; loss of lordosis, kyphosis, hyperextension of c-spine, flexion contractures, enthesitis; synovitis :: RA
   - LAB: ↑ ESR, bone scan, CT, MRI detail and spatial info RE spinal canal compromise
   - Extraarticular: 1) acute iritis, 2) upper lobe lung fibrosis, 3) CV, 4) cauda equina syndrome, 4) amyloidosis

Reiter's syndrome (aka reactive arthritis)
ID: chronic, recurring, inflammatory disease, consisting of urethritis or infectious diarrhea, conjunctivitis, arthritis, and mucocutaneous lesions. HLA-B27 (+) >90% pts;
- reactive arthritis 2ary to exposure to infectious agents in HLA-B27+ person; postvenereal (endemic) form → urethritis often from chlamydia trachomatis or mycoplasma, epidemic form → post diarrhea from shigella flexneri, salmonella, yersinia; M > F = 9:1
- arthritis: (i) involve lower extremity asymmetrical (ii) self-limited 2-6 mo (iii) recurs in 50%(iv) sausage toes(v) periarticular d/o uncommon (vi) sacroiliac & spinal arthritis occ as (vii) x-ray → periostitis in involved joint
- mucocutaneous lesions: (i) painless circinate balanitis on glans penis & corona – dry/circum;wet/uncirc (ii) painless ulcers palate / buccal mucosa (iii) keratodermia blennorrhagica on soles of feet 15%

Psoriatic Arthritis
ID: an inflammatory arthritis that accompanies psoriasis (chronic skin ds, unknown etiology)
- involve peripheral joints, sacroiliac 30%, spinal 12% psoriatic spondylitis HLA-B27 30-50% peripheral not
- Characteristic of psoriatic arthritis: (i) psoriasis 1st (ii) DIP involved (iii) frequent psoriatic nail involve (iv) sausage toes (v) skin lesions / arthritis wax/wane together (vi) remit freq > than RA (vii) few develop severe arthritis (mutilans) (viii) radiologic: pencil-in-cup, whittline, periostitis, nonmarginal spinal syndesmophytes

2. Identify the genetic susceptibility to ankylosing spondylitis. Acquire HLA-B27, rel risk AS 100-150x

3. Recognize the multisystemic nature of these diseases. See manifestations, esp. extraarticular, mucosa

4. Identify therapeutic modalities and their mechanisms of action.
AS: 1) pt/family education – ds, process, prognosis, adjust to chronic ds; 2) pharm – NSAIDS, sulfasalazine, methotrexate (no remittive tx), anti-TNF-α, intraarticular steroid injx; 3) PT – postural (kyphosis) & breathing exercises (keep lung capacity high), swim; 4) reconstructive surgery of hips/ spine PRN
RS: same as for AS. In chlamydia, 3-mo tx doxycycline for pt & sex partner, RS 37% → 10% topical corticosteroid, eye drops, occ. Systemic steroid – methotrexate, azathioprine, cyclosporin
PA: same as for RA: pt education, NSAIDS, remittive drugs (methotrexate, gold, sulfasalazine), PT. Anti-TNF-alpha; skin – corticosteroid, tar creams, UV light
SLE is T cell dependent and caused by a breakdown of immune regulation.
   Clonal deletion normal;
   Clonal anergy defective - SLE cells more resistant to become anergic;
   Regulatory cells defective - suppressor CD4+CD8+NK T-cells
   Clonal ignorance defective - cross-reacting Ags not prevented cuz regulatory cells don’t work

Complex genetic basis
   Susceptibility genes - 9 chromosomal foci in humans
   Resistance genes - resistant if have 1 major resistance gene, regardless of # of susceptibility genes;
   C1,4,2 complement components

Principles of tolerance induction and maintenance
1. Self recognition by the T cells is physiological rather than pathological.
2. Profile of inherited MHC and non-MHC genes are strong susceptibility and resistance determinants for autoimmune diseases
3. T cells can be activated to become responsive or non-responsive. Two signals are needed to activate T and B cells to become responsive. A single signal can induce non-responsiveness
4. T cell effector function is determined by antigen presentation, co-stimulatory signals, which follow cell to cell interactions (co-stimulatory signals), and the cytokines in the local environment.
5. Autoimmunity arises when harmful self-reactive T cells recognize a self-antigen and escape normal regulation.
6. Infectious agents have a major role in pathogenesis because of their adjuvant properties and, cross-reactivity (mimicry) with host peptides.

Host and environmental factors in pathogenesis
   Host Factors:
   • T cell defects
     hyperresponsive to self-Ags, resist tolerance, impaired Tregs
   • B cell abnormalities
     increased spontaneous activation, enhanced B cell receptor signaling; produce pathogenic IgG ANAs; immune complex-induced inflammation
   • Complement defects
     failure to clear immune complexes
   Environmental Factors:
   • Infectious agents
     adjuvants or co-stim signals to stimulate autoreactive T cells
   • Sunlight
     cellular injury altering self Ags
   • Drugs, e.g. procainamide, hydralazine
     cause lupus-like illness; activate ‘ignorant’ self-reactive T cells by inhibiting DNA methylation
5.19.04  
Systemic Rheumatic Disease & Lupus  
Dr. Quismorio

a) To present a clinical overview of systemic lupus erythematosus (SLE), a prototype of human "autoimmune disease"
SLE - a chronic inflammatory rheumatic disorder. Involves multiple organ systems; varied presentations; range of clinical manifestations & striking immunological abnormalities including antinuclear Abs (ANA) and other Ab types

- affects young women of child bearing age; 85% pts F; > AAs, Asians; 1/1969 pop; 1/700 F 15-64
- Clinical syndromes:
  - Classic: young F w/ butterfly skin rash, leucopenia
  - Articular: symmetrical polyarthritis of small J s of hands, feet; ‒myalgia, myositis, prox M weak; synovitis
  - Respiratory: pleurisy, fibrosis, pneumonia, pulmonary HTN, airway obstruction, diaphragmatic dysf
  - Febrile: fever of unknown origin (FUO)
  - Renal 50% nephrotic syndrome, HTN, edema, prot in urine, glomerulonephritis, granular deposits Ig
  - Neuropsych 1/3 pts; mental fx disturb * & sz*, neuropathy-central/peripheral, CVAs, mov’t disorders
  - Hematologic >50% anemia, AIHA, leucopenia, thrombocytopenia, circulating anticoagulants
  - Vascular: vasculitis, thrombosis, esp. if Ab to coag factors, fetal loss
  - Cardiac: pericarditis, myocarditis, valvulitis*, vasculitis of coronary As, accelerated atherosclerosis
  - Cutaneous - malar rash (butterfly), alopecia, demal vasculitis, Raynaud’s, purpura, subQ nodules
- most common in its category

b) To discuss the role of antinuclear antibodies and other types of autoantibodies in the pathogenesis of SLE
SLE Pathology: inflammatory changes → blood vessel abnormalities, immune complex deposits (fibrinoid necrosis, occlusion)

- non-organ specific AutoAbs: ANA, antiphospholipid, anticytoplasmic, rheumatoid factor, ...
  - " + " pathway: form immune cpxin situ or in circulation → deposit tissue/or organ → activate complement → chemotaxis / inflammatory changes → tissue damage
  - organ specific: Abs to: RBCs, lymphocytes, Platelets, coagulation factors, neurons, heart muscle
  - " + " pathway: anti-RBC, P, WBC → react w/ Ags on cell surface → activate complement → → cell lysis, premature removal of cell from circulation or impair cell fx
- genetic: HLA-DR2/3,; gender: F>M hormones?; environment: UV light, drugs
- deaths: early → opportunistic infection; late → cardiovascular/atherosclerosis complications

c) To present a clinical overview of systemic sclerosis (SSc) and discuss the possible role of lymphocytes, cytokines and other factors in the pathogenesis of the fibrosis in the skin and visceral organs

- ID: genz’d disorder of CT w/degenerative & inflammatory changes → fibrosis
- Affect skin, BVs, skeletal Ms, visceral organs - GI tract, lungs, heart & kidneys
- Skin & s: Edema (diffuse painless nonpitting) → induration (can’t pinch) → atrophy (folds gone; ↓ mov’t)
- Skin histopath: thin epidermis, sparse glands, THICKENING DERMIS, deposit collagen, perivascular lymph’cys
- Cutaneous: sclerosis of “sclerodema”, pigment, telangiectasia (spider), skin ulcer, edema, calcinosis
- MSK: symmetric polyarthritis & polyarthalgia presenting complaints; “squeaks” over tendons← fibrosis
- Myopathy: (1) non-inflm’y - fibrotic replace SkMs; (2)inflm’y myositis w/ proximal M weakness
- GI: atrophy/fibrosis-smooth M, submucosa, serosa, degen/infl BV; esophageal, small/large bowel
- Pulmonary: 1ary-interstitial fibrosis, hyperplasia, sclerosis → pulmonary HTN → CHF; 2ary-pleural, infx
- Cardiac: myocardial fibrosis, conduction defects, arrhythmias, pericarditis
- RENAL: pt w/rapid progress diffuse skin ↓s; sudden malignant HTN visual ↓s → renal failure; bx ACE-Is
- Diffuse SSc: thicken: face, neck, trunk, fingers, hands, arms, legs; Raynauds; lung, heart, GI, kidney; 50%
- Limited SSc: thicken symm: extremitities, face, neck; CREST: calcinosis, Raynauds, esophageal, sclerodactyl, telangiectasia
- Localized: fibrosis confined to skin; no vescera; morphea - localized, linear
- Immunologic: humoral: anti-topoisomerase=diffuse, -centromere=local; cell-mediated: Tcell collagen
- Why overprod’n? CKs? - TGF-b; from activated lymphocytes, mononuclear cells; stimulate fibroblasts
5.19.04
SLE and Systemic Sclerosis (SSc)
Dr. Quismorio

**SLE**

- Inflammatory arthritis of SLE milder than RA. No deformities; different pathology
- Skin lesion biopsy changes:
  - **Hydropic degeneration** (liquefaction) of basal zone of epidermis
  - Collections of mononuclear cells in the suprapidermis and around dermal BVs
  - **Parakeratosis** of the epidermis – imperfect keratinization with retention of cell nuclei in stratum corneum
- Knee
  - Bx synovium: synovitis, hyperplasia of synovial lining cells, lymphocytes, plasma cells, histiocytes in subsurface CT, villus formation non-specific
- Possible joint fluid aspirate Bx outcomes
  - Yellow, opaque fluid; WBC = 80,000/mm³ w/ 90% PMNs. Gram stain --> G(-) rods == infection
  - Yellow, translucent; WBC = 15,000/mm³ w/ 85% PMNs; needle shaped crystals seen on polarized LM, culture (-) == Gout
  - Straw color, transparent; WBC = 4300/mm³ w/ 65% mononuclear cells; culture (-) == mild arthritis
  - Straw color, clear; WBC = 200/mm³ w/ 90% mononuclear cells; culture (-) == nl
- The most characteristic serologic finding in SLE is the presence of **antinuclear antibodies (ANA)** in the blood; **anti-dsDNA** & **anti-Smith** characteristic and diagnostic of SLE
- ANA not species or organ specific; test with indirect immunofluorescent test
- **Drug-induced SLE** - procainamide (anti-arrhythmic) can cause lupus-like illness; inflammatory arthritis, pleurisy, pericarditis, heme abnl'ties, (+) ANA – anti-histone, not anti-dsDNA or anti-Sm; cannot induce anti-histone Abs except with procainamide
- **Glomerulonephritis** – frequent / serious feature of SLE; sig # morbidity; nl size, granular cortex, scattered punctate hemorrhages in renal cortex
  - Focal lupus glomerulonephritis (GN) – hypercellularity and obliteration of the lumen of caps; occurs in <50% of glomeruli
  - Diffuse lupus GN – architecture of glomerulus obliterated, hypercellularity, loss capillary lumina; intense eosinophilia = deposition of fibrin; >50% of glomeruli involved
  - Mesangial lupus GN – mild focal increase in mesangial cells
  - Membranous lupus GN – thickening of capillary basement membrane; no hypercellularity

**Systemic Sclerosis**

- Generalized disorder of CT characterized by fibrosis and degenerative changes in the skin “sclerodermia”, synovium, digital arteries, and in the parenchyma and blood vessels of certain internal organs notably the esophagus, intestines, heart, lungs, and kidneys
- Raynaud’s phenomenon: common & early complaint. Episodic attacks of paroxysmal vasospasm precipitated by extremes, e.g. cold, hypoxia (smoking); tingling, numbness, a vesicp pain with cyclic color changes of the digits --> pale (white), then cyanotic (blue/purple) → rewarm hyperemia (pink or red)
- Early stages → edematous changes in distal portions of limbs
- More advanced stages --> induration and tightness of the skin over fingers, hands, wrists; loss of skin folds and fingers appear shiny and slightly puffy = ‘scleodactyly’; flexion contractures 2ary to tight/indurated skin = characteristic finding in systemic sclerosis; areas of inc/dec-reased skin pigmentation; “mauskopf”
- Skin: atrophy of epidermis, loss of accessory skin structures; dermis markedly thickened
5.20.04

Osteoarthritis

Dr. Arkfeld

1. Know the definitions of osteoarthritis and its primary and secondary forms.

ID: disorder of synovial joints characterized by degeneration of hyaline articular cartilage, marginal osteophyte formation, and reactive subchondral bone changes
Primary: no identifiable predisposing cause
Secondary: identifiable predisposing factor (see Q3)

2. Know the pattern of joint involvement in osteoarthritis.

Primary:
- Hands: 1st CMC, DIP, PIP
- Cervical spine
- Lumbar spine
- Hips
- Knees
- 1st MTP (commonly affected by gout)

Secondary
- ANY joint
- Consider in DDx if OA in unusual locations (shoulder, elbow), or pt <50 y/o
- Associated conditions:
  - Trauma – acute / chronic
  - Structural abnormalities – local / diffuse
  - Neuropathic disorders – tabes dorsalis, DM, syringomyelia, meningomyelocele, peripheral N section
  - Metabolic disorders – acromegaly, alkaptonuria, gout, hemochromatosis
  - Chondrocalcinosis
  - Repeated hemorrhage (e.g. hemophilia)

3. Identify factors predisposing to osteoarthritis.

- Age
- Gender
- Race
- Genetic predisposition
- Obesity
- Mechanical stress
- Joint trauma
- Congenital & developmental bone and joint disease
- Prior inflammatory joint disease
- Endocrine & metabolic disease, including chondrocalcinosis

4. Be able to differentiate between the clinical presentations of osteoarthritis and inflammatory arthritis.

In inflammatory, not osteo-arthritis:
- joint effusion
- joint warmth
- positive anti nuclear antibody
- rheumatoid factor elevated
- erythrocyte sedimentation rate mildly or moderately increased
- synovial fluid white cells increased, marked c-reactive protein elevated

OA: labs normal

5. Identify the objectives of current treatment programs for osteoarthritis and the therapeutic modalities currently available to achieve these objectives.

Therapy
- rest joints
- avoid trauma, abnormal use
- weight reduction
- pharmacologic: analgesic, anti-inflammatory
- alternate exercise programs: yoga, pilates, water exercise programs
- surgical

Prevention
- recognize / treat congenital anomalies predispose to 2ary OA
- ortho manage fractures & soft tissue MSK injuries
- vigilance in suspect joint infection
- early dx, tx of metabolic and rheumatologic disorders predispose to 2ary OA
II. Surgical Treatment of Arthritis

B. Four categories of Surgical Treatment for Arthritis

1. Osteotomy (cutting)
2. Debridement (cleaning)
3. Arthrodesis (fusion)
4. Arthroplasty (repair)

C. Osteotomy

1. Joint malalignment distributes forces abnormally; OA joint destruction progresses
2. **Osteotomy corrects joint malalignment**
3. Reduces forces on involved surfaces by redistributing the abnormal forces more equally across the joint
4. Joint “sparing”
5. Possibly may prevent need for joint replacement (or buy more time until that’s necessary)
6. Does not preclude future replacement

H. Arthrodesis (fusion of joint)

1. **Eliminates motion at the joint**
2. No motion = no pain
3. Effect on function - depends on the joint
4. Common procedure in hand, wrist, spine, foot, and ankle
5. Requires mobility of adjacent joints
6. Increases stress on adjacent joints
7. Recovery depends on time required to get a solid fusion; may require long immobilization

L. Total Joint Arthroplasty (VIDEO)

1. **Best for pain relief, function, and motion (in major joints, i.e. hips & knees)**
2. Potential for wear, loosening, infection
3. Avoid impact activities, i.e. running, jumping, contact sports
4. OK to walk, swim, bike, golf, some skiing, doubles tennis
5. Cement fixation to bone or biologic fixation (bone to metal)

M. Contraindications to Total Joint Replacement

Absolute:
1. Active infection (in the joint to be replaced)
2. No arthritis in the joint to be replaced
   a. Referred pain, e.g. “hip” pain from lumbar spine; “knee” pain from arthritic hip

Potentially Reversible:
1. Uncontrolled medical problems
2. Inadequate soft-tissue structures
3. Unsatisfactory bone structures

P. Long term Results of THA

**MOST DON’T REQUIRE REVISION**

1. 2000 consecutive Charnley THA’s
   a) 80.9% free of revision or removal
   b) 3-16% revised - 16-20y (1st gen)
   c) 1.3-6% revised - 9-18y (2nd gen)
2. Cemented Stems
   a) Uncemented Cups
   b) 0% revised for loose (10y)
   c) 1.2% revised for loose (15y)
3. Uncemented Cups
   a) 90-98.6% at 10-23 years
   b) 91-98% at 5-16 years

R. Total Knee Arthroplasty (TKA)

3. **More difficult rehab than THA**
   a) Work on ROM

S. Long Term Results of TKA (survivorship with revision = endpoint)

**MOST DON’T REQUIRE REVISION**

1. Posterior stabilized
   a) 90-98.6% at 10-23 years
2. Cruciate Retaining
   a) 82-98.8% at 10-23 years
3. Mobile bearing
   a) 91-98% at 5-16 years

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**Case:**

- 67 year old
- R knee pain
- X 4 years
- NSAID’s
- Occ. codeine
- Walk < 1 blk
- Mild HTN, BPH

**Tx:** TKA
5.20.04
Crystal Induced Arthropathies - Gout
Dr. Ehresmann

1. Understand that naturally occurring crystals can cause acute and chronic arthritis.

2. Understand the relationship between hyperuricemia and acute and chronic gout.
   - Hyperuricemia - serum urate [ | > 2xSD; nl =5.1+/ -1.0 men; 4.0 +/- 1.0 women
   - Gout - disorder of altered metabolism of uric acid → tissue deposition of monosodium urate from supersaturated extracellular fluids
   - Acute gout - crystals initiate the acute attack: most initial attacks will be in the great toe J (1st MTP)
   - Chronic - tophi (urate deposits in CT), multiple J s J destruction

3. Understand the relationship btwn the purine biosynthetic pathway, overproduction of uric acid & gout.
   - Purine de novo synthesis - substrates, amidotransferase, IMP end product → other nucleotides synth’z;
   - overproduction associated with large excretion of urinary uric acid; i.e. lots of it floating around
   - Increased excretion due to overproduction via de novo pathway; tx: allopurinol - block xanthine oxidase
   - Underexcretion (90% of gout) normal purine metabolism, faulty renal excretion; tx: probenecid, sulfinpyrazone, colchicine prevent attacks

4. Distinguish between gout with increased and decreased excretion of uric acid and understand the basis for this and the appropriate treatment for each.
   - Increased excretion due to overproduction via de novo pathway; tx: allopurinol – block xanthine oxidase
   - Underexcretion (90% of gout) normal purine metabolism, faulty renal excretion; tx: probenecid, sulfinpyrazone, colchicine prevent attacks

5. Understand the difference between treatment of acute inflammation in gout and treatment of the hyperuricemic state.
   - tx hyperuricemic if symptomatic, few asymptomatic - tx to prevent attacks
   - acute attack tx to get inflammation down

6. Diagnose gout and identify characteristic crystals associated with arthritis.
   - Presenting s/S: intermittent attacks; initial s/S peak @ 12 hours; 1st MTP J freq 1st affected; systemic s/S - fever, chills; recurrent; tophaceous deposits over time (not presenting feature)
   - Clinical features: arthritis, tophi, kidney stones, nephropathy
   - Epidemiology: 8/1000; renal impairment; lipoprotein abnl’ties; inc body mass; excess ETOH; 10% overproduction uric acid; familial in 20% gout pts
   - Lab: (+) hyperuricemia; inc uric acid; visually confirm crystals in J fluid; synovial fluid J fluid - inflammatory - leukocytes: 15-20,000 WBC/mm³; occ WBC up to 80,000; peripheral WBC may be inc during attack; ESR inc in acute episodes
   - Radiography: x-ray nl early; soft tissue swelling; gouty destruction of bone; different joint densities (due to crystals?); erosion with overhanging edge of bone

7. Distinguish gout from calcium pyrophosphate deposition disease and know the clinical features of each.
   - PSEUDOGOUT (COMMON)
     - Clinical: KNEE PAIN, KNEE SWELLING, ARTHRITIS, JOINT WARMTH, JOINT ERYTHEMA, MONOARTICULAR, MONOARTHRITIS
     - Lab: SYNOVIAL FLUID WHITE CELLS INCREASED, CHONDROCALCINOSIS, SYNOVIAL FLUID WHITE CELLS INCREASED, MARKED
   - GOUT, PRIMARY
     - Clinical: CARTILAGE DEFORMITY, EAR CARTILAGE INFLAMMATION, SUBCUTANEOUS NO DULE, BURSAL TOPHUS, SUBCUTANEOUS TOPHUS, TO PHUS, ANKLE ARTHRITIS, INTEST PAIN, KIDNEY STONE, INTEST ARTHRITIS, GREAT TOE ARTHRITIS, GREAT TOE PAIN, JOINT ERYTHEMA, JOINT SWELLING, JOINT TENDERNESS, TO PAIN, MONOARTICULAR, MONOARTHRITIS, METATARSOPHALANG EAL ARTHRITIS
     - Lab: BONE TO PHUS, CARTILAGE TO PHUS, INTRAVENOUS URS PYELOGRAM NEPHROPATHY, SERUM URIC ACID INCREASED, MARKED, HYPERURICEMIA, SYNOVIAL FLUID WHITE CELLS INCREASED, MARKED, SYNOVIAL FLUID WHITE CELLS INCREASED

8. Distinguish between negative and positive birefringent crystal. (-) = uric acid = || ylw, ⊥ blue

9. Understand the usefulness of synovial fluid analysis. Can tell lots of stuff. E.g. hyperuricemic, inflammatory
Pharmacology of Gout
Dr. Gopalakrishna

Acute:
1º: NSAIDs
2º: colchicine
prophylactic: colchicine

Chronic:
Goal: decrease hyperuricemia
Uricosuric agents: probenecid & sulfinpyrazone - increase uric acid excretion
Allopurinol: decrease uric acid synthesis by blocking xanthine oxidase

Drug interactions:
Allopurinol - xanthine oxidase inhibition; cytochrome P450 inhibition
5.21.04
Joints: Pathophysiology – Introduction
Dr. Chandor

1. Discuss the pathophysiology and describe typical gross and microscopic changes of common disorders/diseases affecting the joints:
   - traumatic, degenerative, autoimmune, i.e., SLE, RA, infectious
2. Identify and describe the more common tumors of the joint.
   - benign: pigmented villonodular synovitis, synovial chondromatosis
   - malignant: synovial sarcoma

Notes
I. Joint types
   A. synarthroses – fibrous tissue, e.g. skull
   B. amphiarthroses – flexible fibrocartilage, e.g. pubic symphysis
   C. diarthroses – capsular structure lined by synovial membrane which produces synovial fluid

II. Joint components
   A. Capsule – dense fibrous tissue
   B. Articular Cartilage
      1. chondrocyte – in lacuna, secreting proteins; deepest layer undergoes calcification
      2. collagen – type II
      3. proteoglycans – hydrophilic; core protein + glycosaminoglycans; maintain water content and stability; pressure on AC, water released, water reabsorbed when weight lessened
      4. interfere with proteoglycan/water balance → disease; interfere with subchondral and synovial fluid flow → cartilage avascular, damage if don’t get nutrients
   C. Subchondral Bone Plate – thickened bone beneath cartilage
   D. Synovium – synovial lining cells (A = monocyte; B = synthesize lube) & fibrofatty vascular core
   E. Menisci – fibrocartilage, fit between bones (condyles) to provide joint support

III. Joint pathology
   A. General – injury / inflammation → increased vasculature, influx inflammatory cells; inc joint fluid

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Fluid</th>
<th>Synovium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma, acute</td>
<td>RBCs</td>
<td>Hyperemic, edema fibrosis</td>
</tr>
<tr>
<td>old</td>
<td>mononuclear cells</td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>? viscosity; inflammatory cells PMNs, granuloma</td>
<td></td>
</tr>
<tr>
<td>Osteoarthritis (OA)</td>
<td>Slight ? in amt fluid</td>
<td>NI, non-specific changes</td>
</tr>
<tr>
<td>Rheumatoid arthritis (RA)</td>
<td>? viscosity, PMNs</td>
<td>Proliferative lymphocytes, plasma cells, pannus formation</td>
</tr>
<tr>
<td>Systemic lupus erythematosus (SLE)</td>
<td>PMNs</td>
<td>PMNs, edema, vasculitis</td>
</tr>
<tr>
<td>Gout (crystals)</td>
<td>PMNs, crystals</td>
<td>Acute inflammatory changes, deposition of crystals</td>
</tr>
<tr>
<td>Neoplasms</td>
<td>? cellularity; rarely contains malignant cells</td>
<td>Benign: pigmented histiocytes, nodules of cartilage malignant: synovial sarcoma, metastatic carcinoma</td>
</tr>
</tbody>
</table>

B. Arthritis
1. OA
   - articular cartilage 1ary affected tissue; injury → edema → release CKs; 1ary formation of nitrous oxide → damage cell → ¿block production & reduce joint disease?
   - synovium- non-specific reactive changes; type B synovial cells- extent friction & wear; maintain fluid prod’n & joint surface lub’n → damage deterred
   - subchondral bone - early marrow edema decrease blood flow to cartilage & itself → bone-on-bone =ebumation =painful!
2. RA
   - principal lesions synovitis, hyperplasia, pannus = edematous thickened villous projections of synovium; stroma: prolif lining cells, ? vascularity, lymphocytic plasma cell & m? infiltration
   - exposure microbial antigen initiate acute arthritis → persistent autoimmune → RF & immune complex deposition; inflm CKs induce articular cartilage damage + pannus → erosion of
underlying cartilage; when cartilage has been destroyed, the pannus bridges the apposing bones and forms a fibrous ankylosis → ossifies → bony ankylosis

C. Tumors
1. benign – pigmented villonodular synovitis (synovial membrane proliferation & infiltrate pigment histiocytes) & synovial chondromatosis (cartilagenous metaplasia in synovial membrane
2. malignant – synovial sarcomas – mesenchymal and epithelial tissues

D. Other conditions
1. Capsulitis / Prosthetic Synovitis - capsule rarely involved in pathology; open J surgery, infectious process → capsulitis; type A synovial cell, foreign body giant cells
2. Tendonitis - smaller J's of constant movement; chonic → sheaths undergo fibrosis & smooth mov't of Tendon interfered
3. Bursitis - closed sacs like joints; where T/M over bony prominences; enlarged / painful → increase in fluid, fibrin on inner surface, inflammatory cells in fluid & tissues
4. Ganglion - cystic lesion of T/sheath/J capsule → ganglion w/ viscid fluid
5.21.04
Lumbar & Cervical Spine Anatomy
Dr. Forrester

ID on Spine:
- sacroiliac joints
- vertebral body
- vertebral end plate
- disc space
- spinous process
- pedicles
- transverse process
- apophyseal joints
- superior and inferior articular facets
- intervertebral foramina

MRI T2 – water is bright
Bone scan, gallium scan – archaic, but detects metabolic activity (dark = active)
Image with radiographs or MRI if acute back pain doesn’t get better in 3-6 weeks
70% of acute discs resolve in 2 weeks with anti-inflammatories; 90% in 3 months

Acute back pain: when you should get an image immediately
- pain does not get better with rest
- night pain or pain that wakens pt up at night
- onset of lower extremity weakness
- fever, chills, night sweats
- wt loss, history of tumor (breast, lung, renal, prostate, etc.)
- back pain in children – very uncommon

CT much better for bone
Axial MRI – soft tissues
Identify important anatomical landmarks and structures of the cervical and lumbar spine, and understand their role in providing mobility and stability to the spine

Anatomy
- C2, T11-L5 = mobile presacral; 5 fused; 5 ossicles
- Vertebral body: ant = trabecular (90% of resistance to compressive forces; bone marrow increases compressive strength; wet = hydraulic cushion, inhomogeneous loss in osteoporosis – horizontal trabeculae that connect vertical trab. lost earlier = effect on compressive strength) + thin cortical shell; sup/inf surfaces are concave end plates; post = neural arch = pedicles + laminae + 7 processes
- Facet joints = synovial; orientation contributes to kinematics; C-spine plane of facet Js is ½ btn coronal & axial planes; L-spine plane btn coronal / sagittal planes; obligatory coupled motions of rotation w/ lateral bending

Spinal Ligaments
- functional spinal unit (FSU) = 2 adjacent vertebrae + intervertebral disc + 7 spinal ligaments
- resist tensile, not compressive forces; little resistance in physiologic range, strong resist forces exceeding
- anterior longitudinal Ls (ALL): wider @ vertebral bodies; 2x as strong as PLL; 2x cross-section area; extension
- interspinous Ls: protect nerve roots
- capsular Ls: cervical resist flexion; lumbar resist axial rotation
- ligamentum flavum: btn adjacent laminae; highest % of elastic fibers of any tissue in body; rest in tension
- supraspinous & interspinous Ls: resist flexion; supras stronger than flavum

Understand the physiology of the intervertebral disc and degenerative disc disease
Intervertebral discs
- Support compressive load with facet Js
- annulus fibrosis, nucleus pulposis, cartilaginous end plates; fibers in superficial rings attach to vertebral body Sharpey’s fibers, deeper rings attach to cartilaginous end plates = all resist torsional & tensile forces
- nucleus pulposis = collagen + proteoglycans + mucopolysaccharides = resist compressive spinal loads by increasing hydrostatic pressure
- disc degeneration (OA in synovial Js): age dependent, thicken annulus collagen fibers, nucleus? [1] mucopolysaccharides & proteoglycans ? retain water, osteophytes, disc height decreases → foraminial stenosis; DD + vertebral facet Js → spondylosis; of degeneration affects load-bearing; degenerated less ability to attenuate (reduce force) and evenly distribute applied loads to cartilaginous end plates instead forces go to periphery of end plates
- intervertebral disc herniation: protrusion of tissue from nuc pulp thru defect in annulus fibrosis - pain ← mechanical pressure & chemical irritant from extruded nuc pulp; 90% S/S subside in 3 months
- discs are viscoelastic - mechanical properties depend on rate of loading

Describe the kinematics of the cervical and lumbar spine
- Kinematics = motion of displacement, velocity, and acceleration
- See above

Understand the unique biomechanical characteristics of the normal and degenerated spine and how this may contribute to clinical signs and symptoms.
- Biomechanics - forces, internal or external on body
- Disc height decreases → foraminial stenosis → pressure on nerves
- clinical presentation of severe spinal stenosis: N’genic claudication (limbing), pain, numb, &/or weak in lower extremities ambulating: S/S better if lumbar spine in flexion

Functional Spinal Unit (FSU)
- load-deformation curve – nonlinear and biphasic; physiologic ROM = neutral zone NZ + elastic zone EZ
- two motion combos most dangerous to disc (i.e. cause hemiation): (1) axial rotation + lateral bending, (2) application of sudden compression to an FSU that is positioned in flexion & lateral bending
- T12-L1 FSU lowest flexibility → highest incidence of fractures
5.21.04
PPP Case: Low Back Pain
Dr. Foman

• Describe important features of the most common ailments of the back and spine, including:
  – sprains and strains
  – degenerative joint disease
  – degenerative disc disease
  – osteoporosis of the vertebral column
  – spinal stenosis

• Describe a general approach to the evaluation and management of our LBP PPP patient.

• Musculoskeletal Injury
  – Sprain (ligament injury)
  – Strain (tendon injury)
  - young = sprain; middle aged = degenerative disc disease; 60’s = DJD; 80’s = osteoporotic fracture

• Degenerative Joint Disease (OA)
  • J: Joint space narrowing
  • O: Osteophyte formation
  • B: Bone Cysts
  • E: Eburnation

why at spine and hip?
A) Osteoblasts less active
B) surface area of trabecular bone is higher compared to cortical bone
C) Vitamin D and Calcium are inefficiently absorbed
D) most common sites of acute injury

Cauda Equina Syndrome; bilateral leg pain, lower extremity weakness, saddle anesthesia and difficulty
holding urine → true medical emergency requiring immediate surgical decompression

Point, Palliation, Psychosocial History, Quality, Quality of Life, Radiation, Severity, Swelling, Timing, Trauma

RED FLAGS In history:
• AGE > 50 y.o. or < 20 y.o.
• History of cancer
• Constitutional symptoms
• IV drug abuse
• Immune suppression
• Recent infection
• Pain worse at night
• Trauma
• Progressive neurologic symptoms

PE:
(1) Focused physical exam based on history
(2) Observation: Posture, Expressions, Pain behavior, Stance, Gait
(3) Back examination: Inspection, Palpation, ROM, Mobility
(4) Neurologic evaluation: DTR’s, Sensation, Motor, Strength, SLR, Crossed SLR (straight leg raise)

Where’s the lesion?

<table>
<thead>
<tr>
<th>L4</th>
<th>SCREENING EXAM: SQUAT AND RISE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Strength: <strong>Weak extension of the leg at the knee</strong></td>
</tr>
<tr>
<td>2.</td>
<td>Sensory: Numbness at the knee</td>
</tr>
<tr>
<td>3.</td>
<td>Reflexes: Loss of knee jerk</td>
</tr>
<tr>
<td>4.</td>
<td>Pain: Along the front of the leg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>L5</th>
<th>SCREENING EXAM: HEEL WALKING</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Strength: <strong>Weak dorsiflexion of the foot</strong></td>
</tr>
<tr>
<td>2.</td>
<td>Sensory: Numbness at web of big toe and lateral calf</td>
</tr>
<tr>
<td>3.</td>
<td>Reflexes: No reflexes lost</td>
</tr>
<tr>
<td>4.</td>
<td>Pain: Along the side of the leg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>S1</th>
<th>SCREENING EXAM: WALK ON TOES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Strength: <strong>Weak plantar flexion of the foot</strong></td>
</tr>
<tr>
<td>2.</td>
<td>Sensory: Numbness along back of calf and lateral foot</td>
</tr>
<tr>
<td>3.</td>
<td>Reflexes: Loss of ankle jerk</td>
</tr>
<tr>
<td>4.</td>
<td>Pain: Along the back of the leg</td>
</tr>
</tbody>
</table>

Tx:
• Patient Education and Reassurance
• Comfort Measures
  • Pharmacotherapy
  • Physical therapy
• Activity Modification
  • Relative Rest
  • Exercise
• Complimentary Modalities
• Surgery

Conclusions
• 1/3 will feel better in 1 week, 70-90% better in 6 weeks
• 90% of patients are back to work by 3 months
• 40% Recurrence Rate within 6 months
• Reassess if no significant improvement after 4-6 weeks of treatment
• Few randomized controlled studies or objective unbiased articles
1. **Know the anatomic and physiologic classifications of osteomyelitis.**

**Osteomyelitis - Anatomical classification**
- Type I - Medullary - e.g., hematogenous osteomyelitis
- Type II - Superficial - e.g., ulcer with exposed bone
- Type III - Localized - e.g., cortex and medullary canal involvement
- Type IV - Diffuse - e.g., cortical involvement with extension into the medullary canal of bone. All ununited fractures and total joint infections with osteomyelitis are included

**Physiological classification**
- A Host - Normal
- B Host - Compromised
  - a. Local compromise - radiated area, vascular
  - b. Systemic - malignancies, diabetes, other systemic diseases
- C Host - No treatment indicated because of medical problems or treatment worse than disease

2. **Know the complications that occur from septic arthritis and osteomyelitis.**

**Complications of septic arthritis**
1. Cartilage and joint destruction; joint narrowing = cartilage destruction
   - a. By proteolytic enzymes released from bacteria, WBC, and synovial lining cells which release mucopolysaccharides from cartilage
2. Avascular necrosis of femoral head
3. Subluxations and dislocation
4. Degenerative arthritis
5. Pain and loss of function
6. Chronic synovitis
7. Recurrent infections

3. **Recognize and discuss the histological features of bacterial, tuberculosis, and coccidioidomycosis.**
   - **a. osteomyelitis**
   - **b. septic arthritis - synovial membrane**

**Synovial fluid analysis in septic arthritis**
1. Color - usually thick and cloudy
2. Viscosity - poor
3. Synovial fluid cell count and differential - usually over 50,000 with shift of 80% or more of PMN’s
4. Gram stain - not always positive - if it is, it start near specific antibiotics; *staphylococcus* - synthetic penicillin (methicillin)
5. Synovial fluid sugar - usually a difference of 40 mgm% or more difference between the blood and synovial sugar
6. Culture and sensitivity - make absolute diagnosis and allows for antibiotic selection

4. **Understand the pathophysiology of hematogenous osteomyelitis from onset of seeding of infection to the destruction of cortical bone.**

Bacterial seeding takes place through the nutrient or metaphyseal vessels and localizes in the venous sinusoids of the metaphysis. The reasons suggested for the predilection of the metaphyses include:
1. Slowing of the circulating blood in the sinusoids
2. The fact that epiphyseal arteries are end arteries allowing blood to accumulate in the sinusoid areas of the metaphysis
3. Reduced phagocytosis. Initially there is acute exudative inflammation with increased vascularity, edema and polymorphonuclear leukocytes. Within 2-3 days thrombosis and obliteration of the vessels through increased intramedullary pressure produces ischemia and bone necrosis. An intramedullary abscess forms and edema fluid and purulent exudate are forced through the Haversian and Volkmann’s canals, stripping the periosteum from the bone. This isolates the cortical bone from the blood supply and produces more dead bone. The pus, following the path of least resistance, may re-enter the medullary canal through the cortex at a distance, may perforate the periosteum and enter soft tissue, or extend into the neighboring joint.

5. Be familiar with topical antibiotic therapy including advantages and disadvantages.

Local Antibiotic Advantages
A. High local concentrations often are 10-20 times higher than what can be achieved by systemic administration
B. Low systemic blood levels
C. Low side effects
D. Decreased systemic treatment

Local Antibiotic Disadvantages
A. Foreign body
B. May cause resistant organisms
C. May require another operation

6. Know the radiographic features for osteomyelitis and septic arthritis.

Pediatric osteomyelitis x-rays:
1. Early - soft tissue swelling
2. 7-10 days periosteal elevation
3. Late - cortical and marrow destruction

7. Understand the concepts of bone graft management.

Bone Grafting of Tibia - Routinely done 6-7 weeks after muscle flapping
A. Anterior
B. Postero-lateral
C. Results: 88-100% healing reported by various investigators
D. Free vascularized fibula

Routinely done 6-7 weeks after muscle flapping
E. Following bone grafting fractures generally heal between 4-10 months average; average 6 months.

8. Understand antibiotic elution principles.

Antibiotic Elution Related to:
A. Dosage
B. Surface area
C. Fluid environment
D. Rate of fluid turnover
E. Antibiotic vehicle porosity

Not related to location
1. Define and use in context the following terms:

**articulat cartilage** - Ends of bones covered by

**involucrum** - new bone

**monostotic** - involve one bone

**osteoblast** - mononuclear mesenchymal cells which synthesize and secrete collagen propeptides. rich in alk phos

**osteoclast** - multinucleate monocyte-macrophage-derived cells involved in bone resorption.

**osteocyte** - osteoblasts that have become surrounded by bone matrix (osteoid).

**osteoid** - extracellular material of bone; becomes mineralized after being laid down.

**osteopenia**

**osteoarthritis** - bony outgrowth

**osteoporosis**

**pannus** - vascularised granulation tissue rich in fibroblasts, lymphocytes and macrophages, derived from synovial tissue, overgrows the bearing surface of the joint in rheumatoid arthritis and is associated with the breakdown of the articular surface.

**pathologic fracture** - fracture through a previously abnormal bone.

**polyostotic** - involve many bones

**rheumatoid nodule** - see RA

**sequestrum** - bone necrosis

**synovium** - joint space lined by synovial mem, composed of vascular fibro-fatty tissue covered by 1-2 cell layers.

**tophus** (pl. tophi).

2. Identify and explain the clinical and pathologic features of each of the following disease states:

**achondroplasia** - Autosomal dominant, relatively common. Defect in cartilage synthesis w/failure of proliferation at epiphyseal plates; short stature.

**acute pyogenic osteomyelitis** - children, fever, pain, limp; Staph, aureus, acute: sequestrum, IO abscess, rupture into periosteal space; reactive involucrum → elevated periosteum / xray lytic & sclerotic; IV abx, drain pus to avoid chronic

**ankylosing spondylitis** - HLA B-27, bamboo spine

**chronic osteomyelitis** - draining sinuses btwn bone & skin; 2ary amyloidosis & sq. cell Ca's at site draining sinus

**crystal-induced arthritis**

- **GOUT** - monosodium urate crystal deposits, hyperuricemia; primary - ? prod’n ? secretion or both; 2ary - rapid cell destruction, releases uric acid;
- acute gouty arthritis - deposit in synovial mem - big toe present; histo: hyperplasia, acute inflm synovium, needle-shape, birefringent in J fluid
- chronic tophaceous gout - deposit masses of crystals - tophi, in J scan be in soft tissue, histo: hyperplastic synovium w/baso/amphophilic masses, chronic inflm cells, foreign body rxn, destroy underlying cartilage
- PSEUDOGOUT - calcium pyrophosphate crystal deposition ds; rhomboid, short crystals; cause acute synovitis → destroy art cart ~ degen arthritis

**fibrous dysplasia** - replacement of a localized area of bone by an abnormal proliferation of benign fibrous tissue and bony trabeculae composed of haphazardly arranged woven bone; 70% monostotic - a rib, accidentally discovered on xray; 30% polyostotic - skull, long bones, craniofacial disfigurement; albright - unilateral polyostotic FD; early puberty; café-au-lait pigment; “ground-glass” lesions tan-grey/red, expand medullary bone leave shell intact cortex at periphery; 2ary cysts, hemorrhage; histo: mature fibrous tissue w/irr trabeculae thin, curved no blasts

infectious arthritis - see pyogenic arth, tuberculous arth, lyme disease
juvenile rheumatoid arthritis - fever, rash collagen vascular ds
osteoarthritis (degenerative arthritis) - most common form of arthritis; most age-related; 2ary DJ D in damaged or congenitally abnl J; weight bearing (spine, hips, knees), fingers involved; histo: prolif chondrocytes, fissure, cleft, loss art cart; synovium mild chronic inflm; subchondral bone thickens; osteophytes develop laterally; bone deformed and flattened

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osteomalacia adults; Structural abnormality of bone caused by defective mineralization of osteoid (nl or ?); vitamin D; bones are soft and weak with tendency to fracture Ca ?, P ?, Al Pase ?

osteopetrosis Marble Bone; Defect in osteoclastic reabsorption leading to dense brittle bone. Marrow space limited - anemia, nerve compression

osteoporosis Decrease in total mass of bone without structural abnormality. Affects all bones; Fx vertebrae, femoral neck, wrist (weight bear/stress); loss height / loss kyphosis; back pain;

Paget disease - white >40; mono/poly-ostotic; bone thicken defomyty e.g. big head (need larger hat), bow long bones; fx, esp. spine; xray-early-lucent,late-sclerotic; trabeculae: cement lines; ?? alk phos heart failure, work too hard cuz new bone needs lots blood; 2ary sarcoma/osteosarcoma

rheumatoid arthritis Chronic, relapsing inflammatory disease that affects multiple organ systems; joints, tendons, skin, blood vessels, eyes; rheumatoid factor (RF) - Ab vs. Fc of own IgG; histo: hyperplasia of synovium, lymphocytes, plasma cells, macrophages, fibrin deposit; inflamed cover art. cart pannus Æ destroys cartilage; 25% RA pt gets rheumatoid nodules - subQ w/central fibrinoid necrosis, palisaded epitheloid histiocytes, lymphocytes, plasma cells; extensor surfaces of joints

rickets - lack of vitamin D effect in children, resulting in lack of mineralization of osteoid and disruption of the epiphyseal cartilage and therefore growth; Dbones are soft and weak with tendency to fracture

Sjogren syndrome - collagen vascular disease, dry eyes, mouth
systemic lupus erythematosus - immune complex ds; skin rash
osteogenesis imperfecta Autosomal dominant; Defective synthesis of collagen; fractures heal improperly, blue sclerae,

Marfan syndrome autosomal dominancemutation in fibrillin gene FBNI, important for tissue with high elastic fiber content. arachnodactyly, mitral valve prolapse: dilation of the aorta

3. List the major microbiologic agent(s) responsible for each of the following diseases:
acute pyogenic osteomyelitis- Staphylococcus aureus; xray lytic & sclerotic; elevated periostea

infectious arthritis Pyogenic arthritis; Staph aureus most common cause, Neisseria sp. second
Tuberculous arthritis - mycobacteria

Lyme disease Borrelia burgdorferi,

Pott disease Tuberculosis of the spine; xray present with bone destruction; granulomass; (+) AFB
5.24.04

Lab: Non-Neoplastic Diseases of Bone, Joints, and Connective Tissue

Case 1: Slides 1-2  **Osteogenesis imperfecta**
1. What clinical features does this patient show? Short stature, short lower limbs, anterior bowing of tibia.
2. What features are present on the x-ray of the patient's legs? Slender, ill-formed, thinned bones with multiple fractures, one of which has been internally fixed with a metal rod.
3. What is the most likely diagnosis? **Osteogenesis imperfecta**.
5. What is the fundamental biochemical defect in these patients? Defective type I collagen synthesis.
6. How is this defect manifested in body sites other than bone? Other connective tissues rich in type I collagen can be affected, leading to lax ligaments, blue sclerae, ill-formed dentin.

Case 2: Slides 3-7  **Acute pyogenic osteomyelitis**
1. What lesion is present on the x-ray from the patient's left femur? There is an ovoid, lytic lesion in the distal femur.
2. What cell type do you see in these photomicrographs from this patient's femur biopsy? Mainly PMNs, which are adjacent to bone.
3. What is the diagnosis? **Acute pyogenic osteomyelitis**.
4. Does the bone fragment in this field appear viable? What term is used to describe an area of bone such as this in osteomyelitis? No - no osteocytes are visible. necrotic bone in osteomyelitis is sequestrum.
5. This photomicrograph was taken from the periphery of the lesion in the femur. It shows cellular, new bone. What term is applied to this type of area in osteomyelitis? Involucrum.
6. What is the microorganism most commonly implicated in this disease? **Staph aureus**.
7. What complication is likely to ensue if this condition is not treated promptly? Chronic suppurrative osteomyelitis, squamous cell carcinoma.

Case 3: Slides 8-10  **Paget Disease**
1. What changes do you see in the x-ray from this patient's tibia? The tibia shows irregular sclerosis, thickening and deformation.
2. What histologic changes are present in the section from the biopsy of this patient's tibia? Cellular bone with prominent osteoblasts and osteoclasts. High power shows "mosaic" configuration with prominent cement lines.
3. What is the diagnosis? **Paget disease**.
4. Why is the patient's alkaline phosphatase markedly elevated? Because of marked osteoblast activity - osteoblasts contain alkaline phosphatase.
5. Name at least two complications of this condition. Deformities, fractures, pain, A-V shunts, secondary osteogenic sarcoma.

Case 4: Slides 11-15  **Pott Disease (TB osteomyelitis of spine)**
1. What does this x-ray show? A paraspinal mass.
2. What lesion is present in the vertebral column? A large destructive mass destroying vertebral bone and disc and extending into soft tissues.
3. What cell types are present in this photomicrograph from the paraspinal tissue mass? Plasma cells and lymphocytes.
4. What additional features are noted in this photomicrograph of an adjacent area? Necrosis, palisading epithelioid macrophages, Langhans-type giant cells.
5. What type of special stain is this? What structures are identified on it? An acid-fast bacilli stain - red-staining acid-fast bacilli.
6. What other laboratory test would be important to order on the tissue at the time it is biopsied? **TB culture**.

Musculoskeletal Objectives
Page 59 of 66
Lab: Non-Neoplastic Diseases of Bone, Joints, and Connective Tissue cont’d

Case 5: Slides 16-19  
**Rheumatoid Arthritis**
1. This photo shows a close-up of the radial head surface of this patient. What abnormality does it show?  
Hyperplastic synovium w/ a papillary configuration & is encroaching medially over the articular cartilage.
2. What abnormalities are noted in the synovial biopsy from this joint?  
Papillary hyperplasia, hypertrophy, and lymphoid follicle formation.
3. What inflammatory cells are present infiltrating the patient's synovium?  
Lymphocytes and plasma cells.
4. How does this differ from the type of cell that predominates in the patient's synovial fluid?  
Most in the patient's synovial fluid are PMNs. This is expected in rheumatoid arthritis.
5. What does this photomicrograph from one of the patient's subcutaneous nodules show histologically?  
Dermal nodules with central fibrinoid necrosis and peripherally palisading histiocytes.
6. What is this lesion called? A rheumatoid nodule.
7. What is the most likely diagnosis for this patient? Rheumatoid arthritis.
8. What percentage of patients develop the skin lesions illustrated in slide 5-4? 25%
9. Are other organs besides joints and skin involved in this disease?  
Yes, RA is a multisystem disease involving joints, skin, blood vessels, tendons, eyes, etc.
10. What is the etiology of this disease?  
RA is believed to be an autoimmune disorder.
11. What circulating autoantibody is often seen in patients with this disease?  
Rheumatoid factor, an Ab (usually IgM) against the Fc portion of the patient's own IgG.

Case 6: Slides 20-22  
**Osteoarthritis**
1. What gross changes do you see in the patient's femoral head resection specimen?  
Flattening and lateral osteophyte formation.
2. What histopathologic changes are noted in this joint?  
Clefts and fissures in cartilage, cartilage erosion, cysts in subchondral bone.
3. What is the diagnosis?  
Osteoarthritis.
4. How common is this condition? OA is the most common form of arthritis, affecting 85% of population >70

Case 7: Slides 23-25  
**Fibrous Dysplasia, Monostotic**
1. This x-ray shows a rib in the area where the pt described having pain. What abnormality does it show?  
A well-demarcated, "ground-glass" lytic lesion with sclerotic margins.
2. This gross photo shows a rib affected by the same condition from which the above patient suffers. What are the pertinent gross findings?  
A tan, intramedullary lesion that has cysts within it and spares the cortical bone, which is intact.
3. This photomicrograph shows the histologic appearance of the tissue curetted from the patient's rib lesion. What are the histologic findings?  
A proliferation of fibrous tissue with admixed, irregular woven bone trabeculae.
4. What is the diagnosis? Fibrous dysplasia.
5. What treatment options exist for a patient such as this?  
If asymptomatic, may just observe. If symptomatic, curettage or resection followed by bone grafts.
6. Name the two clinical forms of the disease this patient has. Which of the two does she have based on the information given?  
Monostotic & polyostotic fibrous dysplasia; only one bone involved, monostotic.
7. Name two complications that occur in patients with this disease.  
Pathologic fracture, deformity, rare secondary sarcoma.
5.25.04
Neurological Disorders of the Musculoskeletal System: Rehabilitation in Motor Unit Disorders
Dr. Hsu

1. Know some of the common neuromuscular diseases due to disorders of the motor unit
   ➔ Charcot-Marie-Tooth Disease (CMT)
     most common inherited neuropathy; main target = peroneal muscles; slowly progressive also get foot / lower leg; hand / forearm; spine; hips; does not affect life span
   ➔ Duchenne Pseudohypertrophic Muscular Dystrophy (DMD)
     most rapidly progressive & lethal form of dystrophy; dystrophin gene locus large – high mutation rate; 2ary complication: NM’ar spine deformity, pathological fractures respiratory failure; proteins involved in DMD don’t include laminin
   ➔ Poliomyelitis
     vaccine in 1953; static disease w/recovery of rx – asymmetrical M weakness post-polio – chronic mechanical strain on Ms; affects nerves

2. Apply the principles of rehabilitation in the management of patients with muscle weakness and gait disturbances
   PRINCIPLES OF REHABILITATION: PROVIDE HIGH QUALITY MEDICAL CARE & DISCIPLINE!
   1. Minimize the sequelae of the original insult – whether it was trauma or disease;
   2. Forestall all preventable complications; if they occur – deal with them aggressively.

3. Have knowledge of the multidisciplinary approach to treatment.
1. Define and use in context these words and roots:

- **actin**: cytoskeletal elements common to skeletal and smooth muscle.
- **angio-**: relating to blood vessels
- **chondro-**: relating to cartilage
- **desmin**: cytoskeletal elements common to skeletal and smooth muscle.
- **fibro-**: relating to fiber
- **histo-**: relating to tissue, esp. connective tissue (CT)
- **leio-**: smooth
- **lipo-**: relating to fat, lipid
- **mesenchyme**: undifferentiated tissue derived from embryologic mesoderm. This generally contains vimentin intermediate filaments.
- **myoglobin**: O2-carrying protein present only in skeletal muscle.
- **osteo-**: relating to bone
- **osteoid**: Uncalcified bone matrix, product of osteoblasts. Consists mainly of collagen, but has osteonectin.
- **pluripotent**: capable of differentiation into more than one type of tissue.
- **rhabdo-**: rod-shaped
- **S-100 protein**: acidic protein found in neural tissues, melanocytes, and chondrocytes + Langerhan cells
- **sarcoma**: malignant tumor of mesenchymal origin.
- **tumor grade**: I – low; II – intermediate; III – high
- **vimentin**: intermediate filament common to mesenchymal tissues.

2. Delineate the typical age, site, outcome, and gross and microscopic appearances and recognize clinically and histologically each of the following mesenchymal neoplasms:

- **angiosarcoma**: vascular malignant; lined by malig endothelial cells; liver – exposure: arsenic, dyes, PVC
- **chondrosarcoma**: well dif common; degenerate; mid-age+; pelvis; cart H, fibr, nec; mitoses, an aplasia, increased cellularity, binucleated, hyperchromatic nuclei in myxoid or cart matrix
- **desmoid tumor**: prolif of fibrous tissue; from M aponeuroses; rarely mets; locally invasive; low grade
- **Ewing sarcoma**: childhood, neuroectodermal, bone/soft tissue, small round blue cell tumor w/ glycogen; 60% survive with chemo/sx/radiation
- **Fibrosarcoma**: malignant prolif fibroblasts "homing bone" pattern
- **giant cell tumor**: knee, distal femur, prox fib; epiphysis; 20-55 y/o; red-brown, H, cyst, necrosis; histo: plump, spindled, fibroblast-like mononucleate cells, bkg = lots multinucleate giant cells
- **hemangioma**: nonmalignant; sequester blood / cardiac overload; capillary, cavernous
- **hemangioendothelioma**: prolif endothelial cell; classify basis: rel’n tumor cell to vascular space; low grade
- **Kaposi sarcoma**: vascular; myofibroblast/endo; old, immuno supp; purple skin; fibrotic, H, necrotic, spindle
- **Leiomyosarcoma**: malig of smooth M; anywhere; desmin, actin, NOTmyoglobin; predict: mitosis, size, pleo
- **Liposarcoma**: malig fibrous histiocyto ma MFH; deep thigh, retroperit’l; well-diff’d, local invasive, aggressive
- **lymphangioma** (cystic hygroma) vascular spaces – define by size/type - AVM, neck/axilla
- **lymphangiosarcoma**: vascular malignant; lined by malig endothelial cells; expose: tx radiation, lymph stasis
- **malignant fibrous histiocyto ma**: most common mesenchymal neoplasm in adults, fibroblast, phagocytic, spindle cells, plump histiocyte, tumor giant cells; grossly yellow
- **myositis ossificans**: tumor-like prolif of bone/cart/M; post-trauma; extraosseous bone form any mesench tissue; benign
- **nodular fascitis**: subQ forearm, leg, am, face; histo: “active-looking” fibroblasts, central myxoid, extension into surround tissue; rapid in size over few weeks; benign; doesn’t recur if partially resect
- **osteochondroma**: benign cart Ca; pedicle nl, rim prolif cart cells; near ends long bones; inherited (hereditary multiple exostoses)
- **osteosarcoma**: intramedullary, juxtacortical, extra skeletal; highly malignant, heme, 20s y/o, knee; later = Paget; produce osteoid = abnl nonmineralized bone matrix; soft -> friable, firm -> fibrous, foc of oss’n, calc’f’n; histo: anaplastic spindle cells w/ tumor osteoid or bone formation
- **rhabdomyosarcoma**: most common mesenchymal neoplasm in children; SkM; adult – pleomorphic; child – embryonal, alveolar, bunch of grapes; tan-red, soft, friable, hemorrhagic, necrotic, infiltrative; histo: cross striations, strap cells; desmin intermediate filaments & actin, a veces myoglobin
5.25.04  Lab - Tumors of Mesenchymal Origin

Case 1: Slides 1-3  **Myositis ossificans**
1. What cell/tissue types are found in this lesion?
   Muscle, connective tissue, vascular tissue, cartilage, and bone are all found in this lesion.
2. Neoplastic or reactive?  Benign or malignant?  This is a **reactive, benign** lesion.
3. What does the history of trauma indicate?  A history of trauma is very common.
4. What is the diagnosis?  **Myositis ossificans.**

Case 2: Slides 4-7  **Benign Osteochondroma**
1. In what age groups is this lesion most common?  Most common in patients 15-25 years of age.
2. What tissue types predominate in this lesion?
   This tumor consists of a cartilaginous cap overlying medullary bone. Periosteum usually covers the tumor, which overall shows a regular orientation, suggesting its benign nature.
3. In what bone is this lesion typically found?  Any bone that develops by endochondral ossification.
   The metaphyses of the femur and humerus are the most common sites.
4. What is the diagnosis?  **Osteochondroma.**

Case 3: Slides 8-9  **Malignant Fibrous Histiocytoma (MFH) or Liposarcoma**
1. What cell types are found in this lesion?  This lesion consists of **spindle cells** and **plumper cells which contain lipid.** Giant cells are found in some areas.
2. Do the histologic features indicate a good or poor prognosis?  Both benign and malignant forms of this neoplasm occur. Because of frequent mitoses and features of cellular anaplasia, this case would be considered malignant.
3. What is the diagnosis?  **Malignant fibrous histiocytoma.**

Case 4: Slides 10-15  **Rhabdomyosarcoma**
1. How would you characterize the cells of this lesion?  This lesion consists of **primitive mesenchymal cells** which occasionally demonstrate a more spindle shape, rarely **cross striations** can be delineated.
2. What is the patient's prognosis?  Prognosis is dependent on site and stage. This patient should do well, if there are no metastases and he is treated with chemotherapy, surgery, and radiation therapy.
3. What is the diagnosis?  **Rhabdomyosarcoma.**

Case 5: Slides 16-18  **Ewing Sarcoma** aka **primitive neural ectodermal**
1. Cell of origin of this neoplasm?  Controversial, considered to be derived from neuroectodermal cells.
2. Age group is this tumor most common?  Ewing sarcoma is most common in patients < 20 years.
3. Tumor looked like grossly?  Soft, grey to red, with areas of necrosis and hemorrhage.
4. What is the diagnosis?  Ewing sarcoma; a small round blue cell tumor; cells have glycogen.

Case 6: Slides 19-21  **Chondrosarcoma**
2. How would you evaluate the degree of differentiation in this tumor?
   Review all areas of a tumor before determining its "grade," may vary in different parts of the same neoplasm. grade is usually designated by the worst part found. Well-differentiated (low grade) chondrosarcomas may be very difficult to distinguish from benign chondromas. This shows mostly relatively well differentiated cells, but anaplastic, hyperchromatic, sometimes multinucleated chondrocytes and more primitive spindle cells with very scanty matrix may be found as well if multiple sites are sampled.
3. What are the three most common malignant neoplasms found in bone in adults?  Metastatic carcinoma, multiple myeloma and chondrosarcoma.
4. What is the diagnosis?  **Chondrosarcoma.**

Case 7: Slides 22-25  **Osteosarcoma**
1. relationship between trauma and lesion?  History of trauma is frequently given by patients with a skeletal or soft tissue neoplasm, esp. in kids, where injuries are common. Such a history is incidental, not causal.
2. Is this patient a typical age for this lesion?  Most common in second decade of life, so 15 is typical.
3. histo?  May contain fibroblastic, cartilaginous, osteoid, and giant cell components.
4. What is the diagnosis?  **Osteosarcoma.**
1. Identify some common pediatric orthopaedic problems.

**Common Hip Disorders**
- **Developmental dysplasia of the hip (DDH)**
  - Femoral head dislocation/subluxation; acetabular dysplasia (not formed well)
  - Test: Barlow, Ortolani
- **Transient or toxic synovitis**
  - ESR, CRP nl to ~?; R/O septic hip
- **Legg-Perthes “disease”**
  - Get round femoral head fitting well in round acetabulum; Test: Trendelenberg
- **Slipped femoral capital epiphysis (SCFE)**
  - Obese teens, obligate external rotation when hip is flexed up; medical emergency

**Spinal Problems**
- **Scoliosis**
  - Lateral curvature of spine; postural, congenital, neuromuscular, idiopathic; progress if growth left
- **Kyphosis**
  - Excessive rounding of back; postural,

**Foot Problems**
- **Metatarsus adductus or varus**
  - “searching” great toe
- **Clubfoot**
  - 3 primary components: heel varus (twisted in), heel equinus (plantar declination), metatarsus varus
- **Pes cavus**
  - Equinus deformity of forefoot on hindfoot (high arch)

**In-Toeing**
- **Internal tibial torsion**
- **Femoral anteversion**

**Bowed Legs**
- **Genu varum or bowed legs**
  - Blunt disease, physiologic, rickets

2. Classify fractures of the growth plate.

**Classification of Children’s Fractures**
1. **Plastic Deformation** – bending of the bone with microscopic failure resulting in deformity. No propagation of fracture. Ulna and fibula most common.
2. **Buckle fracture** – compression failure of bone that usually occurs at the metaphyseal-diaphyseal junction. Porous metaphyseal bone buckled out by denser diaphyseal bone. Also called torus fracture because of similarity to the raised band around the base of a Greek column.
4. **Complete fracture** – fracture propagates completely through the bone.
   a. Spiral – usually from rotational force
   b. Oblique – fracture occurs diagonally across the bone.
   c. Transverse – fracture line perpendicular to long axis of bone.
5. **Physeal fracture** – fracture involving the growth plate (physis)
   a. **Salter-Harris classification**
      i. I – fracture line through the physis only
      ii. II – fracture line through the physis and metaphysis
      iii. III – fracture line through the physis and epiphysis
      iv. IV – fracture line through the physis, metaphysis, and epiphysis
      v. V – axial compression of the physis → crush

**Growth plate or physeal fractures**
- Weak link in child's bone
  - If non-displaced, need to differentiate from sprain
    - Physeal fracture more common than sprain in young
    - Ankle sprain could be tibial physeal fx
  - Point of tenderness should allow diagnosis
    *If tender, 1 to 2 cm from the joint probably physeal fx even with nl xray
    *If tender right at joint probably sprain
  - Untreated or undertreated physeal fx may lead to growth problem

3. Understand initial management of pediatric orthopaedic lumps and bumps. **The 6 Week Rule**