Chapter 6

Skeletal System

• The structural framework that gives the body shape & provides protection for internal organs & soft tissue.

• Skeleton derived from Greek: meaning dried

• Dynamic, living tissues that are capable of growth, adaptation to stress, & undergoing repair after injury
Chapter 6 Outline

I. Fxn of the Skeletal System
II. Cartilage
III. Bone Histology
IV. Bone Anatomy
V. Bone Development
VI. Bone Growth
VII. Bone remodeling
VIII. Bone repair
IX. Calcium Homeostasis
X. FX of aging on Skeletal System
I. Fxns of the Skeletal System

4 components make-up SS

1. Bones
2. Cartilage
3. Tendons
4. Ligaments
5 fxns of SS

1. Support
   - Bone: rigid, strong well suited for bearing weight & major supporting tissue for body
   - Cartilage: firm yet flexible support
   - Ligaments: strong bands of fibrous CT that hold bones together

2. Protection
   - Forms a hard shell protecting organs within

3. Movement
   - Muscles attach to tendons wh/attach to bone and use the joints of those bones as pivot points. Hyaline cartilage also covers bones ends and allows for smoother movement. Ligaments help prevent XSV movement

4. Storage
   - Mineral Storage (Ca PO4) can be deposited and extracted when needed
   - Yellow Bone Marrow: stored in bone cavities and can be extracted for energy use

5. Blood cell production
   - Red Bone Marrow: gives rise to blood cells & platelets
II. Cartilage
II. Cartilage

3 types of cartilage:
- Elastic, Fibrocartilage, & hyaline

Hyaline (HC) is most closely associated w/bone:
- Most bones develop from it
- Bone growth in length involves HC
- Bone repair involves prod’n of HC followed by bone replacement

Figure 6.1 Pg 174
II. Cartilage

- **Chondroblasts**: produce cartilage matrix
- **Chondrocyte**: cell left occupying a *lacuna* after chondroblast is surrounded by matrix
- Matrix of HC:
  - Contains collagen (strength) proteoglycans (resiliency by trapping water)
- Perichondrium double layered CT sheath covering most cartilage lined with blood vessels & nerves (means diffusion is req’d for $X\Delta$)
- Articular cartilage: covering the ends of bones at the joint contains no blood vessels or nerves

Figure 6.1
Pg 174
III. Bone histology

A. Bone Matrix
B. Bone Cells
C. Woven & lamellar bone
D. Cancellous & compact bone
III. Bone Histology

Bone Matrix

- Responsible for the major final characteristics of bone:
- Described as reinforced concrete, w/collagen (rebar) and minerals (concrete)
- 35% Organic material
  - Collagen
    - Flexible strength
  - Proteoglycans
- 65% Inorganic Material
  - Compression strength
  - Hydroxyapatite primarily
    - Calcium phosphate crystal

Figure 6.2 pg 175
III. Bone Histology: Bone Cells

**Origin of Bone Cells**

- Mesenchymal Stem cells
- Osteochondral Progenitor cells
- Red Bone Marrow Stem Cells
- Osteoblasts
- Chondroblasts
- Osteocytes
- Osteoclasts

Figure 6.3 pg 176
III. Bone Histology: Bone Cells

• Osteoblast:
  – Prod. collagen & proteoglycans that will become the bony matrix connecting to each other via cell processes. Also prod. Matrix vesicles containing Ca & PO4 act as seeds for matrix growth
  – Ossification → formation of bone by osteoblasts; appositional growth (surface outward)

• Osteocytes:
  – Osteoblasts surrounded by bony matrix transition into osteocytes that occupy lacunae with processes connected thru canaliculi (dense matrix prevents diffusion)
III. Bone Histology: Bone Cells

• Osteoclasts:
  – Responsible for resorption (breakdown) of bone
  – Ruffled border contacts matrix and seals the osteoclast in place so it can digest matrix via acid. This product is taken into the osteoclast and processed b4 being released into the body
III: Bone histology: Woven & lamellar bone

- Woven bone is made up of collagen fibers randomly oriented in many directions. This is what is initially formed during fetal development & repair in a fracture it will be replaced with Lamellar bone.

- Lamellar bone is arranged in thin layers called lamellae. Their collagen fibers are arranged parallel to one another.
III: Bone histology: Cancellous & compact bone

- **Cancellous Bone**
  - Has many spaces (spongy)
  - Lamellae combine to form trabeculae (lattice-like structure filled with bone marrow & blood vessels)
  - Trabeculae are oriented along lines of strength & provide structural strength

Fig 6.4, pg 178
III: Bone histology: Cancellous & compact bone

Compact Bone
- Dense with very few spaces.
- Organized in lamellae (circumferential, interstitial, concentric)
- Concentric surround a central canal & form osteons
- Interstitial are remnants of bone remodeling
  - Canals within bone provide means for $X\Delta$ of gases nutrients, & waste products.
    - Perforating canals, central canals, & canaliculi
IV. Bone Anatomy
IV. Bone Anatomy: Bone Shapes

- **Long bones**
  - Longer than wide
- **Short Bones**
  - About as long as wide
- **Flat Bones**
  - Relatively thin, flat shape with a slight curve
- **Irregular Bones**
  - Have odd shapes that do not fit into any category
IV. Bone Anatomy: Structure of long bone

- **Diaphysis**
  - Shaft of long bone

- **Epiphyses**
  - The ends of long bone

- **Epiphyseal Plate**
  - Site of bone growth in length

- **Medullary Cavity**
  - Hollow space in diaphysis

- **Red Bone Marrow**
  - Site of blood cell prod’n
  - Usually located in cancellous bone

- **Yellow Bone Marrow**
  - Consists of fat
  - Usually located within the diaphysis’ medullary cavity
IV. Bone Anatomy: Structure of long bone

• Periosteum
  – Covers the outer surface of the bone
  – Outer layer contains blood vessels & nerves
  – Inner layer contains osteoblasts, osteoclasts, & osteochondral progenitor cells
  – Perforating/Sharpy’s fibers hold the periosteum, ligaments & tendons in place

• Endosteum
  – Lines cavities inside the bone & contains osteoblasts, osteoclasts, & osteochondral progenitor cells
IV. Bone Anatomy: Structure of Short, flat, & irregular bones

- Flat bones have an interior framework of cancellous bone sandwiched between 2 layers of compact bone.
- Short & irregular have a composition similar to the long bone epiphysis. Most filled with marrow. Contain no diaphysis, but dependent on bone can have epiphyseal plate.
- Some flat or irregular bones in skull also have air filled spaces called sinuses that are lined by mucous membrane.
V. Bone Development
V. Bone Development

**Intramembranous Ossification**

In this process embryonic bone growth occurs at multiple places simultaneously, eventually growing together into a complete Bone. To make parts of the skull, mandible & diaphysis of the clavicles.

**Endocondral Ossification**

Bone forms within hyaline cartilage that develops from mesenchyme.
V. Bone Development: Intramembranous Ossification

1. Embryonic mesenchyme forms a collagen membrane containing osteochondral progenitor cells.

2. Embryonic mesenchyme forms the periosteum, containing osteoblasts.

3. Osteochondral progenitor cells become osteoblasts at the centers for ossification; internally the osteoblasts form cancellous bone; externally, the periosteal osteoblasts form compact bone which becomes the outer shell.

- Fontanels are the areas of membrane that are not ossified at birth.

Figure 6.11 page 185
V. Bone development: Endochondral Ossification

- **Most of the bones develop from a cartilage model**
- **End of the 4th wk of development:**
  - Formation of cartilage beings
- **8th wk of development**
  - Endochondral ossification begins & continues in different areas until age 18-21
V. Bone development: Figure 6.12 page 186-7

**Endochondral Ossification step by step**

1. A cartilage model is produced by chondroblasts. The cartilage model is surrounded by perichondrium, except where joints will form.

2. The perichondrium of the diaphysis becomes the periosteum, and a bone collar is produced. Internally, the chondrocytes hypertrophy, and calcified cartilage is formed.
V. Bone development: Figure 6.12 page 186-7
Endochondral Ossification step by step

3. A primary ossification center forms as blood vessels and osteoblasts invade the calcified cartilage. The osteoblasts lay down bone matrix, forming cancellous bone.

4. The process of bone collar formation, cartilage calcification, and cancellous bone production continues. Calcified cartilage begins to form in the epiphyses. A medullary cavity begins to form in the center of the diaphysis.
V. Bone development: Figure 6.12 page 186-7
Endochondral Ossification step by step

5. Secondary ossification centers form in the epiphyses of long bones.

6. The original cartilage model is almost completely ossified. Unossified cartilage becomes the epiphyseal plate and the articular cartilage.
V. Bone development: Figure 6.12 page 186-7

Endochondral Ossification step by step

7. In a mature bone, the epiphyseal plate has become the epiphyseal line and all the cartilage in the epiphysis, except the articular cartilage, has become bone.
VI. Bone Growth

Bone can only grow via the process of appositional growth (putting new bone on the surface of older bone or cartilage)
VI. Bone Growth: Growth in bone length

- Long bones & bony projections increase in length because of growth at the epiphyseal plate. These separate the diaphysis from the epiphysis.
- Growth at the epiphyseal plate involves the formation of new cartilage by **interstitial** growth followed by **appositional** bone growth on the surface of the older cartilage.

Figure 6.13 pg 188
VI. Bone Growth: Growth in bone length

- Zones of the epiphyseal plate each have their own function.
- Growth results in an increase in diaphysis length. Bone growth ceases when epiphyseal plate becomes ossified and forms the epiphyseal line. (12-25 dependent on bone & individual)

Figure 6.13 pg 188

1. Zone of resting cartilage. Cartilage attaches to the epiphysis.

2. Zone of proliferation. New cartilage is produced on the epiphyseal side of the plate as the chondrocytes divide and form stacks of cells.

3. Zone of hypertrophy. Chondrocytes mature and enlarge.

4. Zone of calcification. Matrix is calcified, and chondrocytes die.

5. Ossified bone. The calcified cartilage on the diaphyseal side of the plate is replaced by bone.
VI. Bone Growth: Growth at articular cartilage

- The ends of long bones (epiphysis) increase in size because of growth at the articular cartilage.
- Similar to epiphyseal plate growth. The end closest to the bone is the part being ossified.
- One big difference is that the articular cartilage never ossifies.
VI. Bone Growth: Growth in bone width

1. Osteoblasts beneath the peristomeum lay down bone (dark brown) to form ridges separated by grooves. Blood vessels of the peristomeum lie in the grooves.

2. The groove is transformed into a tunnel when the bone built on adjacent ridges meets. The peristomeum of the groove becomes the endosteum of the tunnel.

3. Appositional growth by osteoblasts from the endosteum results in the formation of a new concentric lamella.

4. The production of additional concentric lamellae fills in the tunnel and completes the formation of the osteon.

- Appositional growth beneath the peristomeum increases the diameter of long bones & the size of other bones.
### VI. Bone Growth: Factors effecting Bone growth

- The potential size & shape of bone & an individual’s final adult height are determined genetically BUT factors such as nutrition & hormones can greatly modify the expression of those genetic factors.

<table>
<thead>
<tr>
<th>Nutrition</th>
<th>Hormones</th>
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<tbody>
<tr>
<td>• Bone growth requires 2 things:</td>
<td>• Growth hormone, thyroid hormone, estrogen &amp; testosterone stimulate growth.</td>
</tr>
<tr>
<td>– New Cells &amp; New Matrix</td>
<td>– Deficiently results in decrease in size of an individual.</td>
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<tr>
<td>– Both of which need sufficient nutrition to divide or be created respectively.</td>
<td>• Estrogen &amp; testosterone also cause increased bone growth followed by closure of the epiphyseal plate.</td>
</tr>
<tr>
<td>• Bone grows at a slower rate when in severe nutritional deprivation. This may lead to someone not reaching their predetermined genetic height.</td>
<td>– Too much can result in someone not reaching their full height.</td>
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<tr>
<td>• Vitamin D: Required for Calcium absorption w/o it the boney matrix is weak “rickets”</td>
<td></td>
</tr>
<tr>
<td>• Vitamin C: required for collagen synthesis w/o collagen bones &amp; ligaments are weak or do not grow at all.</td>
<td></td>
</tr>
</tbody>
</table>
VII. Bone remodeling
VII. Bone remodeling

- Remodeling converts woven bone into lamellar bone & allows bone to Δ shape, adjust to stress, repair itself, & regulate body Ca^{2+} levels.

- Bone adjusts to stress by adding new bone & realigning bone using remodeling

Figure 6.17 pg 191

Cylinder with same height, weight, & composition as a solid rod can support much more weight than a rod w/o bending
VIII. Bone repair
Fractures

- Greenstick
- Spiral
- Comminuted
- Transverse
- Compound

Vertebral Compression

Tibial stress fracture

AP1 Chapter 6
Bone repair/Fracture Repair

Hematoma formation

1. Blood released from damaged blood vessels forms a hematoma. And inflammation & swelling (hematoma will be replaced by internal callus.

As clot dissolves macrophages clean-up cell debris, osteoclasts break down dead bone tissue, & fibroblasts produce collagen & other extracellular material.

Callus formation

2. The internal callus forms between the ends of the bones, and the external callus forms a collar around the break. External callus is bone-cartilage & stabilizes the ends of the broken bone.
Eventually fibers & cartilage of the internal callus are replaced by woven, cancellous bone, which further stabilizes it.

3. **Callus ossification**

4. **Bone remodeling**

External callus is reduced in size by osteoclast activity and although no evidence of the break remains the repaired zone usually remains slightly thicker.
IX. Calcium Homeostasis
IX. Calcium homeostasis

- Calcium must be maintained within narrow limits for functions such as muscle contraction & maintenance of membrane potentials.
- Because bone is a major storage site for calcium, it can be used to maintain homeostatic blood levels of calcium.
- When levels are low, osteoclast activity increases causing net movement of Ca into blood.
- When levels are too high osteoblast activity increases causing net movement of Ca into the matrix.
1. Osteoclasts break down bone and release Ca\(^{2+}\) into the blood, and osteoblasts remove Ca\(^{2+}\) from the blood to make bone. *(Blue arrows represent the movement of Ca\(^{2+}\).)* Parathyroid hormone (PTH) regulates blood Ca\(^{2+}\) levels by indirectly stimulating osteoclast activity, resulting in increased Ca\(^{2+}\) release into the blood. Calcitonin plays a minor role in Ca\(^{2+}\) maintenance by inhibiting osteoclast activity.

2. In the kidneys, PTH increases Ca\(^{2+}\) reabsorption from the urine.

3. In the kidneys, PTH also promotes the formation of active vitamin D *(green arrows)*, which increases Ca\(^{2+}\) absorption from the small intestine.

- PTH increases blood Ca levels by increasing bone breakdown, Ca absorption from the small intestine, & Ca reabsorption from urine.
- Calcitonin decreases blood Ca by decreasing bone breakdown.
X. FX of aging on the SS
X. FX of aging on the SS

- With aging, bone matrix is lost & the matrix becomes more brittle
- Cancellous bone loss results from thinning & a loss of trabeculae.
- Compact bone loss mainly occurs from the inner surface of the bones & involves less osteon formation
- Loss of bone increases the risk of fractures & causes deformity, loss of height, pain, stiffness, & loss of teeth