Fractures

- A fracture is a break in the rigid structure and continuity of a bone.
- The fracture may be:
  - Complete fracture
  - Incomplete fracture
  - Open fracture
  - Closed fracture

Types of Fractures

- Simple
- Comminuted
- Compression
- Impacted
- Pathologic
- Stress
- Depressed
- Others
Healing of a Fracture

- Five stages:
  - Hematoma
  - Granulation tissue
  - Procallus
  - Bony callus
  - Remodeling

Complications in Fractures

- Muscle spasms
- Infections
- Ischemia
- Compartment syndrome
- Fat emboli
- Nerve damage
- Failure to heal
- Osteoarthritis or stunted growth

Dislocations

A dislocation is the separation of two bones at a joint with loss of contact between the articulating bone surfaces.

- Usually only one of the bones is out of position
- If the bone is only partially displaced, with partial loss of contact between the surfaces it is termed subluxation.
Causes of Dislocation

- Trauma
- Sometimes associated with fracture
- Underlying muscular disease
- Rheumatoid arthritis
- Torn ligaments may predispose

Sprains and Strains

- Sprains involve a tear in a ligament
- Strains involve a tear in the tendon
- Avulsion involves complete separation of the ligament or tendon from their bony attachments
- Both are quite painful and are accompanied by tenderness, marked swelling, and often discoloration due to hematoma formation

Bone Disorders - Osteoporosis

- Common metabolic disorder
  - Decrease in bone mass and density
  - Loss of bone matrix and mineralization
- Bone mass usually peaks in young adults, then gradually declines (osteopenia)
- Calcium intake and exercise are important in children and young adults to build bone mass
Two forms of osteoporosis
- Primary including postmenopausal, senile and idiopathic osteoporosis
- Secondary following a specific primary disorder like Cushing’s Syndrome
- Both men and women are affected although the frequency and severity is more pronounced in women

Bone Matrix
- Bone is a hard connective tissue
- Matrix about 1/3 organic (mostly bone collagen)
- Matrix about 2/3 inorganic (mostly calcium phosphate and calcium carbonate; some fluoride, magnesium and sodium)
Bone and Mineral Homeostasis
- Osteoblasts deposit minerals from blood into bone matrix after meal
- Osteoclasts remove and release minerals from bone into blood during fasting
- Dietary deficiency of Calcium causes demineralization of osseus tissue

Aging of Cortical Bone

Aging of Trabecular Bone
Osteopenia

Changes in Bone Matrix
- Age changes are complex and not fully understood
- Due to variety of factors influencing matrix
  - Genes
  - Amount of exercise
  - Nutrition
  - Levels of hormones
  - Amount of skin exposure to sunlight
  - Levels of chemicals in the blood
  - Functioning of skin, intestines and kidneys

Proteins and Minerals
- With aging, the balance between the amount of protein and minerals shifts in favor of minerals
- Bones become rigid, brittle and more likely to break
Quantity of Bone Matrix

- Quantity of bone matrix decreases with age because matrix formation becomes slower than matrix resorption
- May begin as early as age 20 but by age 30 most people are losing matrix
- By age 35, everyone has begun to lose bone at a substantial rate

Structure

- At first, only trabecular bone is removed
- The trabeculae become thinner and weaker
  - Trabeculae can become thicker and stronger again if osteoblasts can be stimulated to replace missing matrix (e.g. increase in exercise)
  - Some trabeculae disappear completely and cannot be replaced; some disconnect from others and matrix becomes weaker
- The weakening at that spot is permanent

Structure

- Decline in cortical bone is not detected until about age 40 and is quite slow
- After age 45 the rate increases but remains about half the rate of trabecular loss
- Loss of cortical bone occurs only on the inside of the bone and so the layer of cortical bone becomes thinner and weaker
Effects of Menopause

- Rates of both trabecular and cortical bone loss increases in women after menopause
- Menopause usually occurs between 45-55 but the level of estrogen declines even before this
- Combined effects of aging and menopause result in a loss of 15-20 percent of the trabecular bone in the 10 years after menopause

Effects of Menopause

- This is 2-3 times the rate of loss in women before menopause or the rate of loss in men
- Very old women may have only half the trabecular bone they had at age 25
- Men have lost only 2/3 as much trabecular bone as women during the same period

Loss of Cortical Bone

- Cortical bone loss also accelerates because of menopause with a loss of 10-15 percent in the decade after menopause
- This is a 3-4 fold increase over the rate in the years before menopause
- Very elderly women have only about half the cortical bone they had at age 25
- Women begin with less bone matrix than men and suffer greater losses with aging
Variability is Matrix Loss

- Trabecular bone loss begins earlier and occurs faster than cortical bone loss
- Since some parts of the skeleton have a higher proportion of trabecular bone, some bones suffer the consequences more than others
  - Vertebrae are mostly trabecular bone
  - Higher incidence of vertebral fractures in elderly, especially women

Crush Fractures of Vertebrae

Fractures of the Femur

- The upper part of the femur which joins the pelvis contains a large percentage of trabecular bone
- The shaft of the femur is largely cortical bone
- The upper end of the bone loses matrix earlier and faster than the shaft
- This contributes to the high incidence of hip fractures as age increases
Hip fractures
Most hip fractures occur in one of two locations — the femoral neck or the intertrochanteric region.

Coxa (hipbone)
Break
Femur (thighbone)

Hip fracture repair techniques
For a fracture in the femoral neck, internal fixation, bone grafting, or a total hip replacement may be used. A hip compression screw may be used for a fracture in the intertrochanteric region.
Consequences of Bone Loss

- Increase in spontaneous fractures and breaks from accidents with age
- Fractures heal more slowly in the aged
- This can lead to complications such as infection, bedsores, blood clots and pneumonia
- The prolonged immobility leads to an increase in the rate of matrix loss increasing the risk of another fracture

Osteopenia vs. Osteoporosis

- With age there is a general decline in bone mass (loss of bone mineral and collagen)
- If the loss of bone mass is serious enough to compromise bone structure resulting in easy fractures (crush fractures and broken bones) condition called osteoporosis.

Minimizing Loss of Matrix

- The cause of loss of bone matrix with aging is not known
- Much has been learned about factors that modify the rate of loss
- Best strategy is to develop as much bone matrix as possible during youth and then slow losses during adulthood
- It is much more difficult to reverse bone loss than to slow it
Reducing Risks of Osteoporosis

- Keep physically active
- Consume enough calcium
- Maintain adequate levels of vitamin D
- Women should maintain levels of estrogen (?)
- Avoid excessive consumption of alcohol
- Avoid smoking
- Avoid excessive caffeine in the diet
- Avoid consuming excess phosphates

Reducing Risks of Osteoporosis

- Choose antacids with calcium rather than aluminum
- Use corticosteroids only when needed
- Avoid consuming excessive insoluble fiber in the diet (usually only a problem with people with marginal calcium in diet)
- Do not consume excess protein

Rickets and Osteomalacia

- Due to deficit of vitamin D and phosphates needed for bone mineralization
- May be due to:
  - Dietary deficits
  - Malabsorption
  - Lack of sun exposure
Results in soft bone and rickets in children often with deformities including “bow legs”. Height is usually abnormal.
Results in soft bone and osteomalacia in adults resulting in compression fractures.
Renal rickets refers to osteomalacia associated with severe renal failure.

Paget’s Disease (Osteitis deformans)

- Progressive bone disease occurring in adults over 40 years.
- Cause not well understood but may include a slow virus and heredity.
- Includes excessive bone destruction with replacement by fibrous tissue and abnormal bone.
- Pathologic fractures are common.

Bone deformities of the long bones, vertebrae, pelvis and skull occur.
When the vertebrae are involved, compression fractures with kyphosis result.
Also causes cardiovascular disease and heart failure.
Egil, son of Skalla-Grim, an Islandic Viking in about 990

- Egil apparently had Paget’s disease
- He had many of the physical characteristics
- Stories of his prowess as a warrior taking direct blows to the skull from an ax

Bone Tumors

- Most primary bone tumors are malignant
- Bone is also a common site for secondary tumors
  - Particularly the spine and pelvis
  - Metastases usually have spread from breast, lung or prostate

Types of Bone Tumors

- Osteosarcoma (osteogenic sarcoma)
  - Primary malignant neoplasm
  - Usually develops in metaphysis of femur, tibia or fibula in children or young adults
  - More common in males than females
  - Steady, severe and persisting pain with rest and activity
Ewing’s Sarcoma

- Ewing’s sarcoma is another malignant neoplasm common in adolescents that occurs in the diaphysis of long bones
- Both grow quickly and can metastasize to the lungs
- Both cause constant bone pain

Chondrosarcoma

- Arises from cartilage cells and are more common in adults over 30
- Develop gradually in the pelvic bone or shoulder girdle at the points of muscle attachment
- Eventually metastasize to the lungs

Question 7
Disorders of Muscle, Tendons and Ligaments

- Muscular Dystrophy (MD) is a group of inherited disorders characterized by degeneration of skeletal muscle.
- The disorders differ in:
  - Type of inheritance
  - Area affected
  - Rate of progression

**Types of Muscular Dystrophy**

<table>
<thead>
<tr>
<th>Types of Muscular Dystrophy</th>
<th>Inheritance</th>
<th>Age of Onset</th>
<th>Distribution</th>
<th>Progress</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duchenne's</td>
<td>X-linked recessive (affects males)</td>
<td>2-3 years</td>
<td>Hip, legs, shoulder girdle</td>
<td>Rapid</td>
</tr>
<tr>
<td>Becker's Dystrophy (limb girdle)</td>
<td>Autosomal dominant</td>
<td>Before age 20</td>
<td>Shoulder, neck, face</td>
<td>Slow to moderate</td>
</tr>
<tr>
<td>Myotonic</td>
<td>Autosomal dominant</td>
<td>Birth to 10 years</td>
<td>Face, tongue</td>
<td>Slow</td>
</tr>
<tr>
<td>Jeune Syndrome</td>
<td>Autosomal recessive</td>
<td>Infancy</td>
<td>Shoulders, face, hands</td>
<td>Slow</td>
</tr>
</tbody>
</table>

![Duchenne's MD](image_url)

**Fig. 42-12** Duchenne’s Dystrophy

[Text continues...]

16
Primary Fibromyalgia Syndrome

- This is a group of disorders characterized by pain and stiffness affecting muscles, tendons and surrounding soft tissues (not joints)
- Specific trigger points where pain may be stimulated

No obvious signs of inflammation or degeneration in the tissues

No cause is known but appears to be related to altered central neurotransmission resulting in increased soft tissue sensitivity to substance P

Pain Theory and Pain Control

- Gate Control Theory says that control systems or “gates” are built into normal pain pathways in the nervous system that can modify the entry of pain into the spinal cord and brain
  - Gates can be open at synapses allowing pain to ascend the spinal cord to the brain
  - Gates can be closed, reducing or modifying the passage of pain impulses
Gate closure can occur in response to other sensory stimuli that diminish the pain sensations or modulate or inhibit impulses to the brain.

Example: application of ice to a painful site may reduce the pain because the patient is more aware of the cold than the pain.

Also, the brain can inhibit or modify incoming pain stimuli by producing efferent or outgoing transmissions through the reticular formation.

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**Brain Chemicals That Kill Pain**

- The key to blocking the transmission of pain impulses to the brain is the release of opiate-like chemicals (opioids) secreted by interneurons within the CNS.

- These chemicals block conduction of pain impulses into the CNS.

- They resemble morphine and are called endorphins; they include enkephalin, dynorphin, and beta-lipotropin.

---

Gate open and pain impulses carried to brain for perception.

Gate closed when opiate receptors are blocked by enkephalin preventing Substance P release; pain blocked from brain.
Inhibition of Pain by Opiodes

Fibromyalgia continued
- Incidence higher in women aged 20-50
- Often history of prior trauma or osteoarthritis
- Aggregating factors include:
  - Sleep deprivation
  - Stress
  - Fatigue

Joint Disorders
- Osteoarthritis
  - Degenerative or “wear and tear”
  - Non inflammatory joint disease
  - Articular cartilage especially in weight bearing joints (hips and knees) is damaged from excessive mechanical stress
  - Sometimes the breakdown is from unknown causes
- Surface of cartilage becomes rough and worn and interferes with smooth joint movement
- Tissue damage appears to cause enzyme release that accelerates breakdown of cartilage
- Eventually, the bone underlying the cartilage is exposed and damaged and cysts and bone spurs develop along the margin of the bone

- Pieces of the bone spur and cartilage break off inside the synovial cavity and cause further irritation
- No systemic effects are typically seen in osteoarthritis

- Primary form of osteoarthritis is considered to be idiopathic
- Secondary type follows injury or abuse
- Commonly seen after participation in sports and some physically demanding occupations
- Once cartilage is damaged, a vicious cycle ensues
- Pain is mild and incidious in the beginning
- Pain becomes more severe and degeneration progresses
Surgery may be needed to repair or replace joints such as the knee or hip.

Rheumatoid Arthritis

- RA is considered an autoimmune disease causing systemic inflammatory disease
- Affects both adults and children
- Remissions and exacerbations lead to progressive joint damage
- Often begins insidiously with symmetrical involvement of the small joints such as fingers followed by inflammation and destruction of additional joints (e.g. wrists, elbows, knees)

Severity varies from mild to severe depending on:
- Number of joints involved
- Degree of inflammation
- Rapidity of progression
In affected joints:
- An abnormal immune response causing inflammation of the synovial membrane
  - Vasodilation
  - Increased permeability
  - Formation of exudate
- This response results in the typical red, swollen and painful joint
- After first period of acute inflammation, the joint may appear to recover completely

During subsequent exacerbations:
- Synovitis
- Pannus formation
- Cartilage erosion
- Fibrosis
- Ankylosis

Mobility is impaired as the various joints become damaged and deformed
- Walking becomes difficult when knees and ankles are affected
- Other parts of the body may become affected:
  - Subcutaneous nodules may form on the extensor surfaces of the ulna
  - Nodules may also form on the pleura, heart valves or eyes
  - These systemic effects are thought to arise from circulating immune factors
Juvenile Rheumatoid Arthritis
- Occurs in several different types
- Similar in some ways to adult form but some differences:
  - Onset usually more acute
  - Systemic effects more marked but nodules are typically absent
  - The large joints are frequently affected

Other Types of Arthritis
- Infectious (Septic) Arthritis
  - Usually only one joint involved
  - Microbes like gonococcus, staphylococcus or others are the cause
  - Aggressive treatment necessary to prevent permanent joint damage
- Gout (Gouty Arthritis)
  - Mostly in men over age 40
  - Due to deposits of uric acid in joints

Ankylosing Spondylitis
- Chronic progressive inflammatory condition
- Affects sacroiliac joints, intervertebral spaces and costovertebral joints
- Usually develops in persons aged 20-30
- Remissions and exacerbations
- Deemed to be autoimmune disorder
- As calcification develops, the spine becomes more rigid and flexion, extension and rotation of the spine are impaired