

Lecture 5C

Burn Injuries

Thermal Injury

- Burns are thermal injuries caused by dry heat, moist heat, electricity, chemicals, friction, radiant energy, or electromagnetic energy.
- Major burn injuries **disrupt homeostasis** more than any other type of injury!
- Children younger than 4 years old and adults older than 65 years old have the highest risk of thermal injury.
- Scald injuries account for at least 60% of burn injuries in children younger than 15 years of age. Up to 20% of scald injuries are due to **abuse or neglect**.
- Drug abuse, alcohol abuse and psychological disorders also increase the risk of thermal injury.

Thermal Injury

Scald Injury Due to Child Abuse

(Note the absence of drip, spill, or splatter marks!)



Depth Classification of Burn Injuries

Burns are classified according to the depth of injury:

- First Degree Burns
- Second Degree Partial Thickness Burns
- Second Degree Deep Partial Thickness Burns
- Third Degree Burns
- Fourth Degree Burns

First Degree Burns

- Involve the outermost layers of the epidermis. The dermis is not injured. A mild sunburn is an example.
- Erythema, mild pain
- Healing occurs in 3-5 days without scarring or loss of pigmentation.
- Erythema is possible because superficial dermal blood vessels have not been injured.

Depth Classification of Burn Injuries

Second Degree Superficial Partial Thickness Burns

- Involve the epidermis and **upper part** of the dermis
- Appear red to pale ivory depending on the state of dermal blood vessels
- **Moist, thin-walled blisters** form superficially within minutes of injury.
- Pain is a major feature as **tactile and pain receptors are intact.**
- Healing occurs in 21-28 days in the absence of infection.
- The amount of scarring is genetically determined. African-Americans and Caucasians with red hair experience more scarring than Native Americans and Asian-Americans.
- Hair follicles remain intact and hair reappears in 7-10 days.

Depth Classification of Burn Injuries

Second-Degree Deep Partial Thickness Burns

- May involve the entire dermis and leave only remnants of the epidermis in the hair follicles.
- **Mottled** appearance with large areas of waxy white tissue surrounded by light pink or red tissue. White areas are due to destruction of dermal blood vessels.
- **Blisters are superficial and resemble flat dry tissue paper.**
- **Tactile and pain receptors are absent in the areas of deepest destruction, but intact in areas of lesser depth.**
- Healing occurs in about 4 weeks in the absence of infection with significant scarring, hair follicle loss, and depigmentation.
- **Debridement and grafting** are used to diminish scarring and achieve early wound closure.

Depth Classification of Burn Injuries

Third Degree Burns

- Involve the **epidermis, dermis, and underlying subcutaneous tissue**.
- Immediately following injury the area appears white, cherry red, or black.
- **Deep blisters may be present under a dry layer of dehydrated skin**.
- Superficial blood vessels coagulated by the heat of injury may be visible through the skin.
- The dermis loses its elasticity resulting in a wound with a dry, hard, leathery texture.
- The massive edema and loss of elasticity may produce a tourniquet-like effect if the wound occurs circumferentially around a limb or the torso. This may require **escharotomy (removal of burned tissue) or fasciotomy (removal of muscle fascia)** to restore circulation.
- **Full-thickness burn areas are painless to touch**, but an area of lesser injury in which pain and tactile receptors are intact usually surrounds the third degree burn.
- Third degree burns **require grafting** because so little residual tissue is left for regeneration. Small injuries may heal by **second intention** (a result of ingrowth of dermal elements from the margins of the wound toward the center).

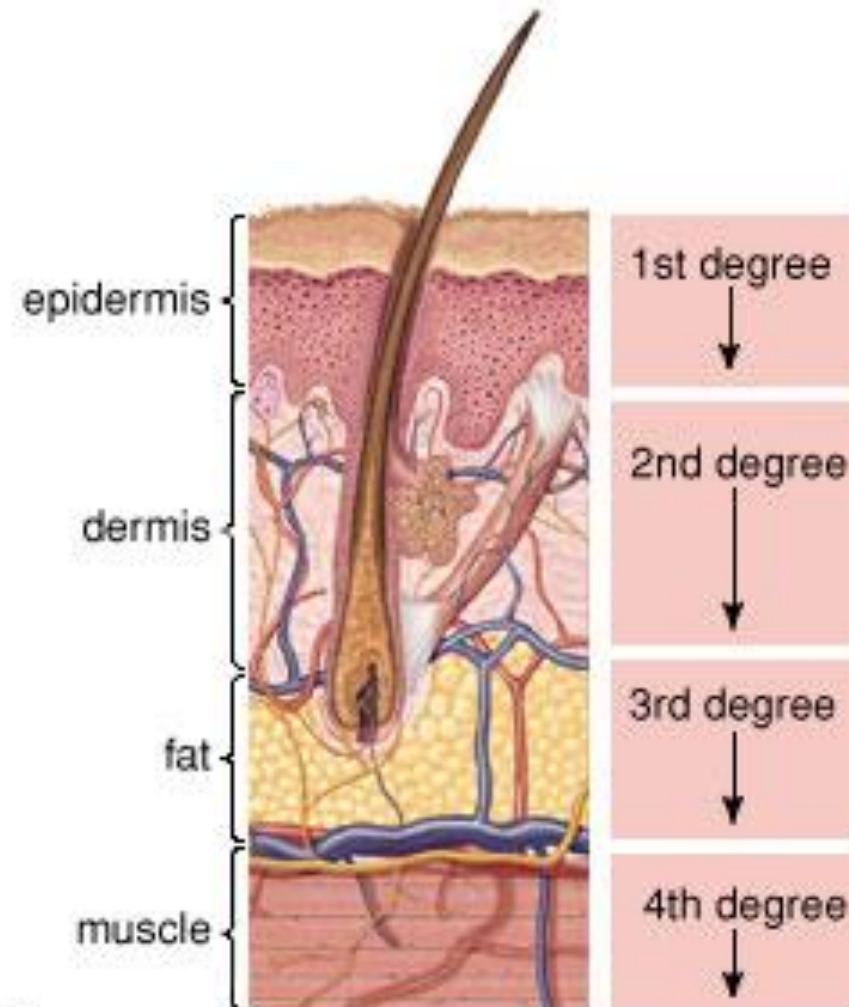
Depth Classification of Burn Injuries

Fourth Degree Burns

- Full-thickness injuries that extend beyond the dermis **and involve muscle, bone, or both.**
- The burn appears dry, charred, and mottled. There is no sensation and limited or no movement in affected limbs or digits.
- Common in high voltage **electrical** injury or **prolonged** exposure to intense heat (unconscious fire victims).
- **Grafting is required and possibly amputation** of involved limbs or digits.

NOTE: Keep in mind that serious burn injuries are often not uniform, but contain areas multiple degrees of injury. Burns that are primarily full thickness will often have edges of partial thickness damage where pain sensors are intact.

Depth Classification of Burn Injuries



Depth Classification of Burn Injuries

First Degree



Second Degree
Superficial Partial Thickness



Second Degree
Deep Partial Thickness



Depth Classification of Burn Injuries

Third Degree

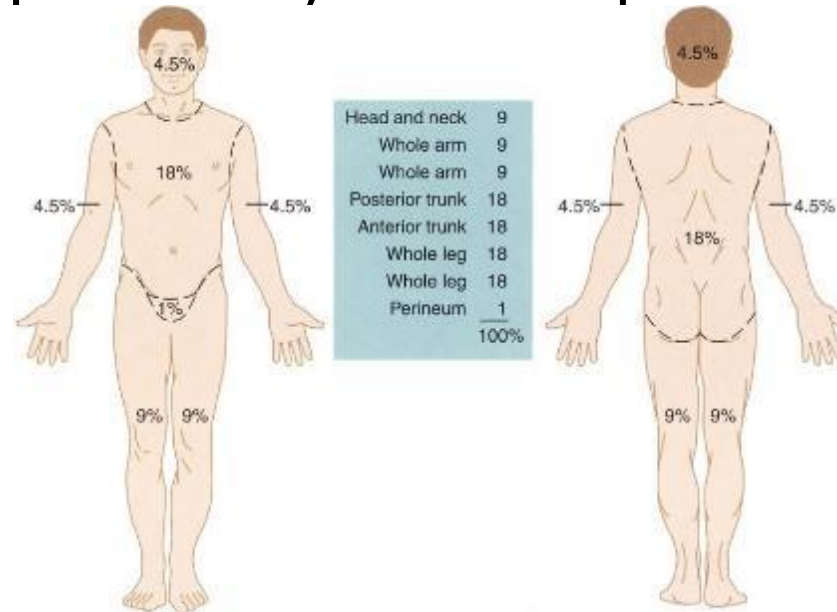


Fourth Degree



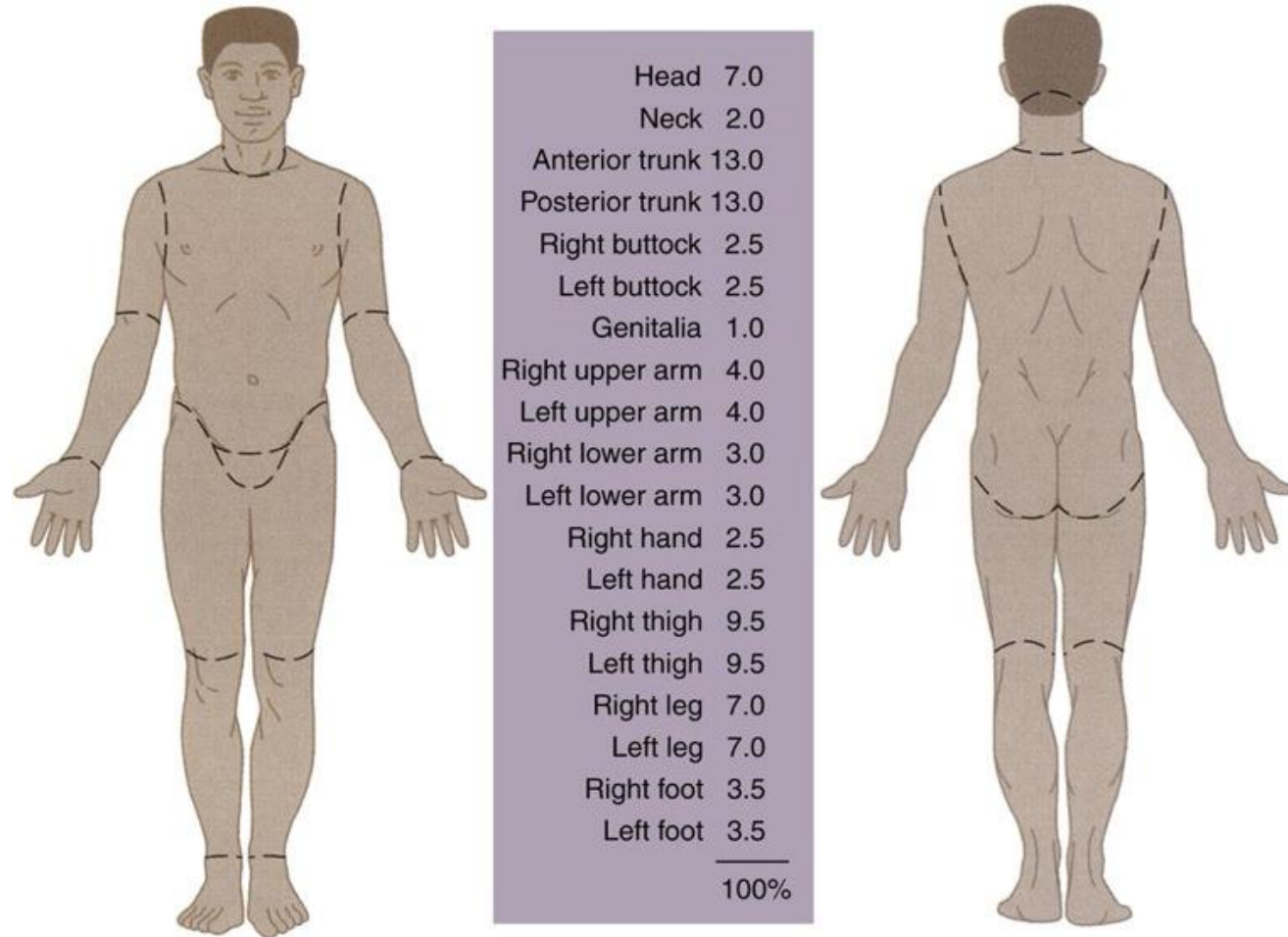
The Rule of Nines: %TBSA

- Burns are described both in terms of their depth and the percent of the total body surface area (**%TBSA**) affected.
- The **Rule of Nines** is used to determine %TBSA affected. The diagram below lists the body areas and their respective %TBSAs. Note that the %TBSAs in the list (except for the perineum) are multiples of 9.



Lund Browder Chart: %TBSA

- The **Lund Browder Chart** presents body areas in sections. Some clinicians prefer to use this chart to estimate % TBSA .



Burn Severity Chart (American Burn Society)

- Burn Severity-depends on several factors in addition to depth and %TBSA. **KNOW the characteristics of MAJOR burns.**

Minor	<15% TBSA; < 40 yr old <10% TBSA; > 40 yr old <10% TBSA; < 10 yr old <2% TBSA full thickness burn without cosmetic or functional impairment
Moderate	12%-25% TBSA; < 40 yr old 10%-20% TBSA; > 40 yr old 10%-20% TBSA; < 10 yr old <10% TBSA full thickness burn without cosmetic or functional impairment in the following areas: face, eyes, ears, hands, feet, or perineum
MAJOR	>25% TBSA: 10-40 yr old >20% TBSA: >40 yr old >20% TBSA: <10 yr old >10% TBSA: full-thickness burn; any age Any burn with cosmetic or functional impairment in the following areas: face, eyes, ears, hands, feet, or perineum High-voltage electrical burn All burns with concomitant inhalation injury or major trauma

Response to Thermal Injury

Disruption of Skin Functions

- Burns are capable of disrupting any or all of the functions served by the skin:
 - **Protection** against infection, water and electrolyte imbalance, caustic chemicals, UV light
 - **Sensory Processing** by skin receptors for temperature, pressure, pain
 - **Production** of Vitamin D
 - **Thermoregulation** by integration of skin sensory receptors, the CNS and dermal blood vessels
 - **Immunologic** function by tissue Langerhans cells, macrophages and immune elements in dermal blood and lymphatic vessels
 - **Aesthetic** function provides the individual identity of the person.

Response to Thermal Injury

Local Response to Burn Injury: “Sick cell syndrome”

- It is initiated by inactivity of sodium potassium pumps. It allows sodium to accumulate inside cells. This leads to the entry of water and **hydropic swelling** with possible cell rupture.
- An associated **decrease in resting membrane potential** leads to a decreased amplitude in action potentials and prolonged repolarization and depolarization times.
- **Potassium leaks out** of cells due to sodium potassium pump failure.
- Calcium ion channel transport is disrupted leading to **increased intracellular calcium ion** and further membrane damage due to activation of destructive intracellular enzymes.
- **Cell membrane damage** causes cells to spill intracellular potassium, magnesium, phosphate and **lactate dehydrogenase (LDH)** into the extracellular space. Although that enzyme has a high concentration in many cells, it normally has a low concentration in the serum. A rise in **serum LDH** indicates necrosis.

Response to Thermal Injury

Systemic Response to Serious Burn Injury

- Serious burn injuries are responsible for these dangerous systemic effects:
 - Capillary Leak Syndrome
 - Burn Shock
 - Hypermetabolism
 - Metabolic Acidosis
 - Negative Nitrogen Balance
 - Organ Dysfunction

Systemic Response to Thermal Injury

Capillary Leak Syndrome and Burn Shock

- Serious burn injuries elicit a **massive inflammatory response**. Vasodilation and increased capillary permeability occur not only at the burn wound site, but systemically as well. This phenomenon is termed “**capillary leak syndrome**”. The loss of fluid volume from the circulatory system causes “**burn shock**”, a form of hypovolemic shock (very low blood pressure).
- The extra interstitial fluid is exposed to the atmosphere in the burn wound area. This leads to **extensive evaporation of body fluid**, further contributing to burn shock.
- The extra interstitial fluid internally results in **systemic edema**.
- Daily evaporative fluid loss is in the range of **20 times** normal during the acute phase of burn shock with gradual decline until wound closure occurs.
- Loss of circulatory volume leads to **reduced cardiac output**.
- Major organs are affected due to low blood pressure and flow.
- **Fluid resuscitation is a crucial component of burn management.**

Systemic Response to Thermal Injury

Fluid Resuscitation During Burn Shock

- The **Parkland formula** is used to determine the minimum fluid resuscitation volume for the **first 24 hours** of burn treatment.
- **Volume (mL) = 4 x body mass in kg x %TBSA of the burn**
- **Example: 70 kg patients with 50%TBSA burn**
 $4 \times 70 \times 50 = 14,000$ ml (14L) in the first 24 hours.
- **Lactated Ringer solution (LRS)** is employed initially.
 - Half of the fluid volume (7000 ml) is administered during the first 8 hours.
 - A quarter (3500 ml) is administered during the second 8 hours.
 - A quarter (3500 ml) is administered during the third 8 hours or in quantities to maintain adequate urine output.
- **Indicators for adequacy** of fluid resuscitation: normalization of mental status, blood pressure, pulse, capillary refill, arterial pH, base deficit, and maintenance of adequate urine output. **Burn shock normally resolves in 24-72 hours.**
- Once the capillary leak syndrome is resolved, a colloid solution, such as **albumin or plasma**, is administered to replace the **protein** lost during the acute phase.

Systemic Response to Thermal Injury

The Hypermetabolic State

- The extensive evaporation of body fluid that occurs in the burn wound area leads to **extensive loss of body HEAT (Recall that evaporation causes the water molecules with the highest kinetic energy to leave the body surface as they transition from the liquid to the vapor state.)** to the atmosphere. The thermoregulatory response to the loss of body heat energy is to increase the **basal metabolic rate (BMR)** via the **fight or flight response** in order to maintain the homeostatic set point for body temperature. Recall that enzymes have an optimal temperature.
- The hypermetabolic state is triggered by massive release of **epinephrine and norepinephrine** from the adrenal glands in to the blood.
- The hypermetabolic state is characterized by a higher level of **catabolic reactions compared to anabolic reactions**. Catabolic reactions are **exothermic**; they release **heat** to the surroundings.
- **Aerobic cellular respiration** is the **catabolism of glucose**. It consumes huge amounts of **oxygen**, about 150% of normal. Most of that oxygen that would normally be expended on other body functions. Thus the entire body is affected.
- The **heart rate** also climbs (120-140 bpm). Elevated heart rate (increased cardiac muscle contraction) consumes ATP, and thus demands even more oxygen.

Systemic Response to Thermal Injury

Negative Nitrogen Balance

- **Muscle wasting** occurs during the hypermetabolic state. During severe stress patients become **insulin-resistant**. Their bodies must then rely on fats and proteins (instead of carbohydrates like glucose) for the source fuel molecules.
- **Triglyceride fats** are converted to glycerol and fatty acids. Fatty acids are converted to acetyl CoA for Krebs's cycle.
- **Muscle proteins**, are hydrolysed, and their amino acids are converted to acetyl CoA or other Krebs's Cycle molecules.
- The amino acids must be **deaminated** (Amine groups are removed.) before they are converted to fuel molecules.
- The amine groups (NH₂) are then used to synthesize nitrogen compounds (urea, uric acid) that are regularly **excreted in the urine**.
- Thus considerable amounts of **nitrogen** are excreted and lost from the body.

Systemic Response to Thermal Injury

- Although protein synthesis (anabolism) is increased during hypermetabolism, it fails to keep pace with proteolysis (catabolism) and amino acid conversion to fuel molecules.
- The excretion of nitrogen compounds in the urine leads to a **negative nitrogen balance** in the body.
- Hypermetabolism lasts until the %TBSA is reduced to 20%.

Management of Hypermetabolism

- Propranolol, a **beta blocker**, blocks beta adrenergic hormone receptors for epinephrine and norepinephrine. It reduces resting heart rate and energy expenditure and improves net muscle protein synthesis.
- Insulin and growth hormone stimulate **protein synthesis**
- Insulin-like growth factor (ILGF)-(similar in structure to insulin). It stimulates **protein synthesis**.
- Management of negative nitrogen balance is necessary for months after a serious burn injury. See below.

Systemic Response to Thermal Injury

Acidosis

- Serious burn injury is associated with a decline in blood pH for multiple reasons.
 - The low blood pressure and low cardiac output during burn shock means that many cells will not receive enough oxygen for cellular respiration. They will have to use **anaerobic respiration** to survive. Thus they will produce **lactic acid** and it will enter the blood causing **metabolic acidosis**.
 - The kidneys normally filter excess acid into the urine for excretion. But the kidneys are often damaged by **the decreased the blood supply during burn shock**. They may also suffer during hypermetabolism when the blood contains excessive amounts of fat and protein fuel molecules.
 - If the lungs have been damaged by smoke inhalation, CO₂ may accumulate in the blood leading to **respiratory acidosis**. When CO₂ is elevated it combines with water to form **carbonic acid**.

Systemic Response to Thermal Injury

Organ Dysfunction: Major burns upset homeostasis more than any other injury!

- **Cardiac dysfunction**

- A major drop in cardiac output occurs during burn shock that does not parallel the gradual reduction in blood volume.
- Metabolic and immunologic factors are involved.
- Renal failure may occur.

- **Respiratory dysfunction**

- **Obstruction secondary to edema** begins within the first few hours after burn injury, but tends to manifest later as resuscitation is undertaken.
- Prophylactic endotracheal **intubation** is undertaken.
- Smoke or fume inhalation often leads to **chemical denaturation** of pulmonary tissue.
- The resulting edema **thickens the respiratory membrane** and leads to acute hypoxia. Adult respiratory distress syndrome (**ARDS**) results.
- **Carbon monoxide** poisoning often accompanies smoke inhalation. The treatment is oxygen administration.

Systemic Response to Thermal Injury

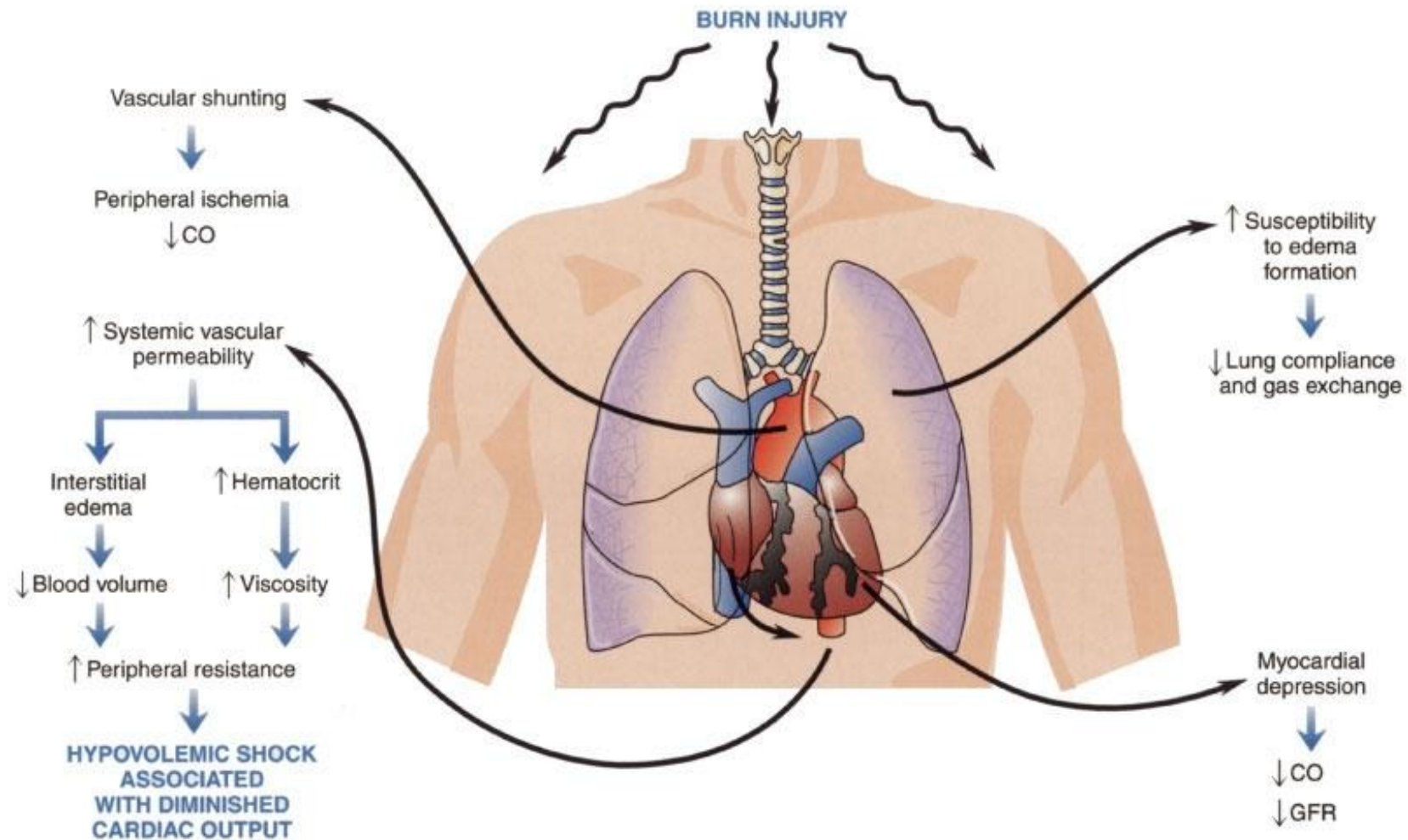
Renal dysfunction

- Reduced cardiac output leads to **reduced** glomerular filtration rate (GFR). The kidneys are not able to filter and clean the blood properly.
- The kidney tissue suffers ischemic damage. Renal failure may occur.

Immune Dysfunction

- **Burns upset homeostasis more than any other injury!**
- The **first line of defense** is compromised.
- Burns profoundly suppress the immune system and thereby increase the **risk of infection**.
- Many inflammatory and stress-related chemicals are found in elevated concentrations in burn patient plasma: histamine, serotonin, complement proteins, prostaglandins, kinins, endotoxins, catecholamines, and corticosteroids.

Systemic Response to Thermal Injury



Elements of Burn Injury Survival

- The **second phase** of burn injury care occurs between the end of burn shock (24 to 72 hours post injury) and the closure of the wound to less than 20% TBSA.
- The requirement for fluid resuscitation ends when burn shock ends.
- **Three elements** are essential for survival during this time.
 1. Meticulous wound management and prevention of infection
 2. Timely surgical excision and grafting of full-thickness wounds
 3. Adequate nutritional support to restore a positive nitrogen balance

Elements Of Burn Injury Survival

Wound Management and Prevention of Infection

- The most common source of burn wound infection is the resident flora: patient's own hair follicles, sweat glands, pulmonary tract, and GI tract.
- Poor hand-washing by staff members can lead to cross-contamination from other patients.
- **Initial wound care**-wound debridement with soap and water. Removal of blisters is controversial and varies from institution to institution.
- **Daily wound care**-bathing patient at least once daily with mild soap and water to remove accumulated bacteria and previously applied ointments, and to debride necrotic tissue.
- Cleansing of wounds is a **very stressful and painful** experience for the patient. Unfortunately, most analgesics work best on visceral (deep) pain rather than the superficial pain of burn injuries. Benzodiazepines are used to decrease anxiety.

Elements Of Burn Injury Survival

Wound Management and Prevention of Infection

- After the wound has been cleaned, **topical antibacterial agents** are applied and covered with a light dressing. Systemic antibiotics are **not effective** in controlling burn wound flora because the burned skin (eschar) has **no blood vessels**.
- Appropriate antibiotic selection is based on cultures of the patient's wound tissue.
- **Systemic antibiotics** are used when the patient exhibits signs of systemic infection and prophylactically at times of surgical procedures.
- Partial-thickness burns will heal in 14-21 days, but full-thickness burns require **autografting** to achieve wound closure.

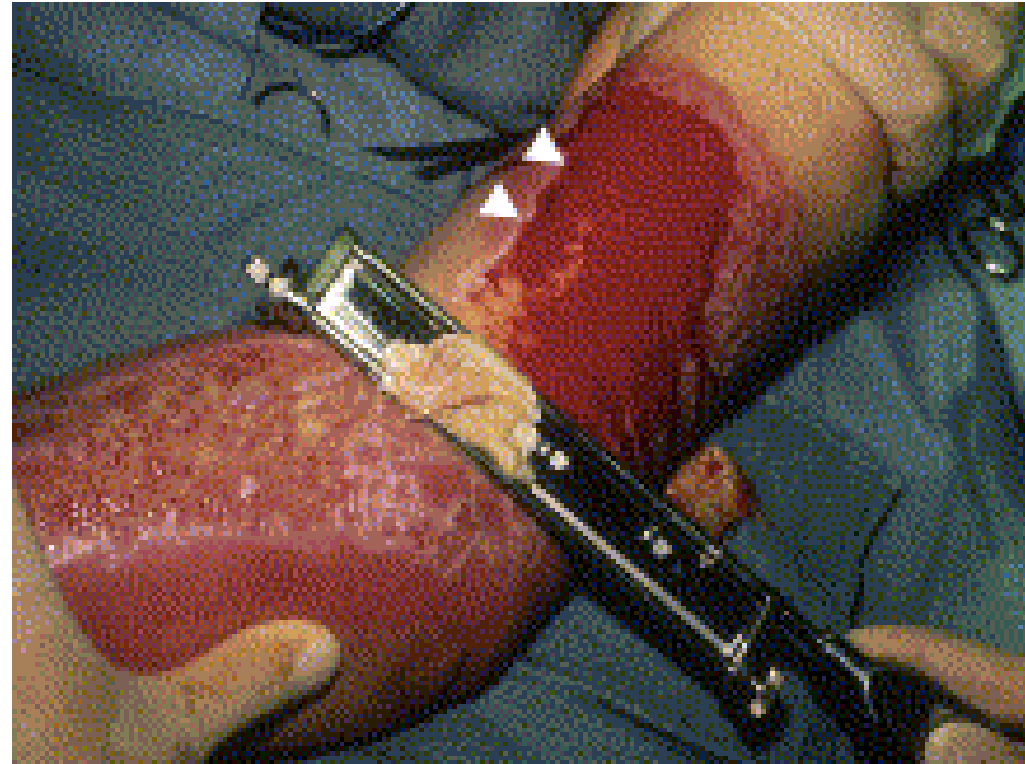
Elements Of Burn Injury Survival

Burn Surgery

- **Dead burned skin (eschar) must be excised** BEFORE a graft of the patient's own skin or a skin substitute is applied. There are two modes of surgical excision:
 - **Tangential excision**-thin layers are removed with a dermatome until viable tissue is revealed. The procedure is usually done 2 to 7 days after injury.
 - **Full-thickness excision**-is done with surgical knives and removes the eschar to the level of the muscle fascia. This type of excision often leaves an uneven contour that is difficult to graft with poor resultant cosmetic effects.
- Both tangential and full-thickness excision involve **massive blood loss** that often requires **transfusion**.
- After bleeding has been controlled application of an autograft, a skin substitute or dressing follows.
- The excised area is often covered with wet dressings soaked in antibiotic solution for 24 hours before autografting.

Elements Of Burn Injury Survival

Burn Surgery
(Tangential Excision)



Elements Of Burn Injury Survival

Burn Surgery

- Several **skin substitutes** are available for patients with extensive burns that do not permit initial autografting. Skin substitutes are in place only temporarily. Autografting is the goal.
 - Homograft-cadaver skin
 - Xenograft-pig skin (the mammalian skin most like human skin)
 - Amnion-amniotic sac
 - Synthetic skin (Integra)
 - Dermal layer of bovine collagen
 - Epidermal layer of silicone to prevent desiccation.
 - The silicone matrix allows ingrowth of capillaries and slowly degrades as **neodermis** develops.
 - The silicone layer is removed and graft is applied over the neodermis.

Elements Of Burn Injury Survival

Burn Surgery

- The “**donor site**” is the unburned area of the patient from which a skin graft is harvested as a paper-thin sheet. Donor sites heal in 5 to 7 days and can be reharvested at that time.
- To expand the surface area that a sheet of autograft will cover, donor skin is cut in a manner that resembles a net or mesh by using a **skin mesher**.
- The skin mesh covers up to **four times** the original area of the skin removed from the donor site.

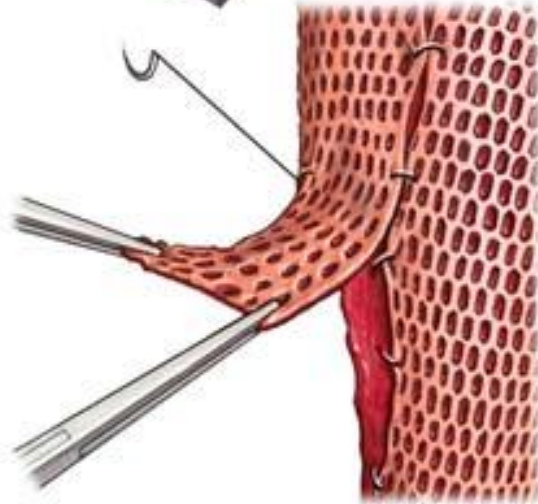
Elements Of Burn Injury Survival

Preparation of a Skin Graft

Graft taken from patient's healthy skin



Skin is meshed to cover a large wound



ADAM.



Elements Of Burn Injury Survival

After Grafting

- The area must be protected from infection, pressure, shearing, and any trauma that could produce bleeding under the graft.
- Bulky dressings are used the first few days followed by the use of wraps and elastic bandages.
- Patients are **returned to the OR once a week** until grafting procedures are complete.
- The **greatest risk of infection occurs on day 3** when bacteria begin to recolonize the area.
- Grafts are usually stable by day 4.
- After day 4, occupational and physical therapy begin.

Elements Of Burn Injury Survival

Nutritional Support: Positive Nitrogen Balance

- In the absence of adequate nutritional support after burn injury there is massive loss of body weight due to the **conversion of body tissue into fuel for hypermetabolism.**
- The protein loss creates a **negative nitrogen balance** in the body. Amine groups are removed from amino acids, converted to nitrogenous waste molecules such as urea and excreted in the urine.
 - Loss of 10% of total protein mass of the body impairs immune function.
 - Loss of 20% of total protein mass of the body impairs wound healing.
 - Loss of 30% of total protein mass of the body leads to severe infections.
 - Loss of 40% of total protein mass of the body is fatal.
- In patients with **greater than 40% TBSA** burn, providing early protein and calorie support is necessary for optimal outcome.

Elements Of Burn Injury Survival

Nutritional Support: Positive Nitrogen Balance

- Enteral feeding (or parenteral) should begin **within 18 hours** of admission.
- Feedings should be continuous and should not be stopped when the patient goes to the ER.
- Monitoring **daily nitrogen balance** is essential throughout the course of burn treatment to ensure a positive nitrogen balance.
- The **Curreri formula** (for adults) and the **Polk formula** (for children) are used to determine **24-hour caloric requirement** for a burn patient. Both formulas are based on pre-burn body mass (or total body surface area) and %TBSA of the burn.
- A **positive nitrogen balance** requires intake of at least **2 grams of protein per kg of body weight per day** and a **non-protein kilocalorie to protein kilocalorie ratio of no more than 100:1**.
- The **serum prealbumin concentration** is a useful indicator of nutritional progress. Prealbumin is a plasma protein synthesized by the liver. It functions as a binding protein for thyroxine (thyroid hormone).
- Nutritional status and dietary habits of burn patients should be continually evaluated for **many months** after discharge from the burn unit.

Rehabilitation Phase

Wound Healing at the Cellular Level

- **Fibroblasts** in the dermis contribute to wound repair by producing collagen and elastin fibers. **Macrophages** clear debris and bacteria.
- New vascular networks form and **surviving keratinocytes** move inward from the edges of the injury toward the center causing **wound contraction**.
- **New keratinocytes** are formed in the stratum basale of the surviving epidermal areas and push up to the stratum corneum over a 14-day period.
- Normal skin color is restored in a delayed manner by **melanocytes** and they melanin they produce.
- **Hair** grows back at a rate of about 1 cm per month, in viable hair follicles.
- New **nail** formation is irregular and of abnormal thickness.

Rehabilitation Phase

Wound Healing (Superficial Observations)

- The skin graft appears **flat and pink initially** even in dark-skinned people.
- In approximately **4 to 6 weeks** the area becomes **raised and hyperemic**.
- If adequate range of motion exercises are not instituted early, the new tissue will shorten, and **contracture** will result.
- Mature healing for a full thickness burn is reached in **6-12 months** when suppleness has returned and the pink or red color has faded to a slightly lighter hue than the surrounding unburned tissue.
- It takes longer for dark skin to regain color because many of the melanocytes have been destroyed. The skin may never regain the original color.
- The mesh pattern in meshed autografts fades with time, but in large expansions (4:1 or greater) the pattern may persist.

Rehabilitation Phase

Contractures

(due to inadequate range of motion exercise
after burn unit release)



Rehabilitation Phase

Scarring

- Scar tissue tends to raise above the natural contour of the skin (hypertrophic scarring)
- Areas of the **face** tend to scar in an **even plane** deleting the natural contours around the nose, chin, and mouth. Psychological counselling is imperative in the case of severe facial burns.
- Scarring on the cheeks can contract and pull the lower eyelid down far enough to prevent closure, a condition called **ectropion** that must be corrected by plastic surgery.
- Pressure can help keep a scar flat if the pressure is slightly higher than the capillary pressure and is applied continuously.
- This knowledge led to the development of **burn garments** which are custom-made for each patient.
- The garment is worn continuously for 12-18 months except for bath times. **Patient compliance** is an issue.

Rehabilitation Phase

Healing After Burn Surgery on the Face



Rehabilitation Phase

Ectropion



Rehabilitation Phase

Anti-scar Support Garment



From Black JM, Matassarin-Jacobs E, editors: Medical-surgical nursing: clinical management for continuity of care, ed 6, Philadelphia, 2001, Saunders, p 1356. Courtesy Medical Z Corp., San Antonio, Tx.

Rehabilitation Phase

Excessive Itching

- Persists for many months.
- Related to the **absence of sebaceous glands and the hyperactivity of sweat glands.**
- Topical lotions and oral antihistamines give partial relief but **tolerance develops** and patients require a series of medications over time.

Extremely Sensitivity to Trauma

- New skin **blisters** form after very slight pressure or friction.
- New skin may be hyper- or hyposensitive to **cold, heat, or touch.**
- The area should be **protected from direct sunlight for 1 year** to prevent hyperpigmentation.

Burns Due To Electrical Injury

Incidence

- Account for **fewer than 2%** of burn unit admissions, but incidence is increasing in the US.
- More common in **rural** areas than in urban areas

Classification

- Low voltage electrical injuries: **household current (120v or 220v)**
- High voltage electrical injuries: **high-tension sources (7200v to 19,000v)**
- **Lightning carries 1 million or more volts.** It kills between 150 and 300 people per year in the US.

Pathophysiology

- Electrical energy is converted to heat energy as it enters, traverses, and exits the body.
- High voltage electrical **injury is deeper than full-thickness burn injuries.** Burns are categorized as **fourth degree.**
- Voltage, the type of current, and the length of contact influence the extent of damage.

Burns Due To Electrical Injury

Pathway of Electricity in the Body

- Because skin has a high resistance to electric current, external evidence of a true electrical injury is limited to an **entrance wound and one or a few exit wounds.**
- Electrical current follows the path of least resistance: **in humans, this path is through muscle, blood vessels, nerves, tendons, and bone.**
- The current rarely causes visceral damage, but **cardiac issues** are more common for electrical injuries than other major burns.
- Severe injury to the extremities is common.
- The **amputation rate after severe electrical injury to a limb is more than 90%**

Burns Due To Electrical Injury

Chewing on an electrical cord (low voltage) caused this burn.



Burns Due To Electrical Injury

High Voltage Electrical Injury

Entry Wound



Exit Wound



Burns Due To Electrical Injury

Local Effects

- Direct cellular denaturation and heat coagulation of arteries and veins
- These events are followed 48 to 72 hours later by tissue **necrosis and gangrene** resulting from lack of blood flow.
- Amputation is often required early to prevent fatal sepsis and/or rhabdomyolysis.
- **Rhabdomyolysis** is the necrotic death of muscle fibers and the resultant release of muscle cell contents (lots of protein) into the blood.

Systemic Effects of Electrical Injury (Acute Period)

- **Shock**
- **Respiratory difficulty**
- **Heart dysrhythmias or cardiac arrest**
- **Metabolic acidosis**
- **Myoglobinuria and kidney damage**

Management of Electrical Injury

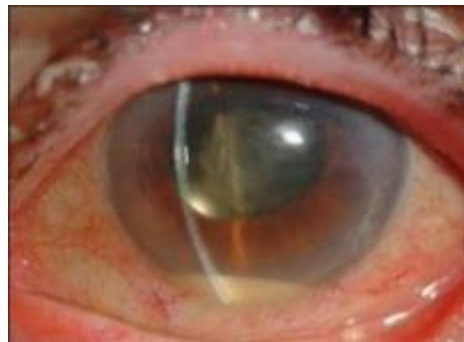
- Airway management is the primary focus of concern immediately after admission, and **endotracheal intubation** is often required due to capillary leak and associated airway edema.
- A **condition similar to burn shock** occurs within a few minutes after injury.
- **No formula exists** to predict fluid requirements for patients with electrical injury shock because often the only visible damage is the entrance and exit wound.
 - An adult patient is given a **1 L bolus** (children a smaller amount) of **Ringer lactate solution IV**, within 15 minutes of intravenous line placement; thereafter, fluid is infused to **maintain a urine volume of 100 mL/hr** (1 to 2 mL/kg per hour for children).
 - Adults often require 1 to 2L of fluid per hour.
- **Twenty-four ECG monitoring** is required for patients with abnormal initial ECG, a history of coronary disease, loss of consciousness at the scene, and/or voltage >240 v.

Management of Electrical Injury

- Electrical injury patients often have **initial serum pH values of 6.8 to 7.2** on admission (ph 7.4 is normal; pH <7.0 is not compatible with life.).
 - The source of the acidity is the **release of cellular contents, including potassium ions, and the lactic acidosis that accompanies hypotensive shock.**
 - Treatment is IV administration of **sodium bicarbonate** to return pH to normal (7.4).
 - The problem usually **resolves 24 to 48 hours** after injury.
- **Myoglobinuria** is due to the release of myoglobin from damaged muscle tissue into the general circulation.
 - Myoglobin is a large muscle protein that **precipitates in the renal tubules** and leads to cast formation. Obstruction of tubules leads to necrosis of the kidney tubules. The accumulation can be prevented by **maintaining a high urine output** until the urine clears of myoglobin.
 - **Mannitol, an osmotic diuretic**, is given along with fluids to increase urine output. **Sodium bicarbonate** may be given to alkalinize the urine. This increases the solubility of myoglobin.

Management of Electrical Injury

- **CNS alterations** include loss of short-term memory for many weeks, ataxia, and sensory deficits due to interference with ion currents at the moment of the injury and also due to hypotension.
- **Corneal cataracts** are a unique complication the cause of which is unknown.
 - These appear anywhere from **1 to 12 months after injury** and may occur in one or both eyes.
 - Progression is rapid with the cornea becoming completely opaque within one month.
 - Treatment is **corneal transplantation**.



Lecture 5D
Soft Tissue Injuries
Bone & Joint Trauma
Infections Of The Bone

Soft Tissue Injuries

- **Inert (Noncontractile) Soft Tissues**
 - Ligaments, Bursae, Fasciae
 - Joint Capsules
 - Cartilages
 - Neural Structures: Dura Mater and Nerve Roots
- **Contractile Soft Tissues**
 - Tendons
 - Bony Muscle Insertions (Entheses)
 - Muscles

Inert Soft Tissue Injuries

Ligament injuries

- Dense regular connective tissue (essentially avascular, so damage is slow to heal) that connects bone to bone.
- Injury (**sprain**) occurs when tension exceeds physiological range of motion.
- Classified by extent of tear
 - **Mild** – few fibers damaged, strength not lost
 - **Moderate** – many fibers damaged, strength is lost
 - **Severe** – complete rupture; function lost
- Common sites: **ACL** (anterior cruciate ligament) in the knee, **talofibular** ligament in the ankle region
- Treatment-pain relief, surgery to repair tears

Inert Soft Tissue Injuries

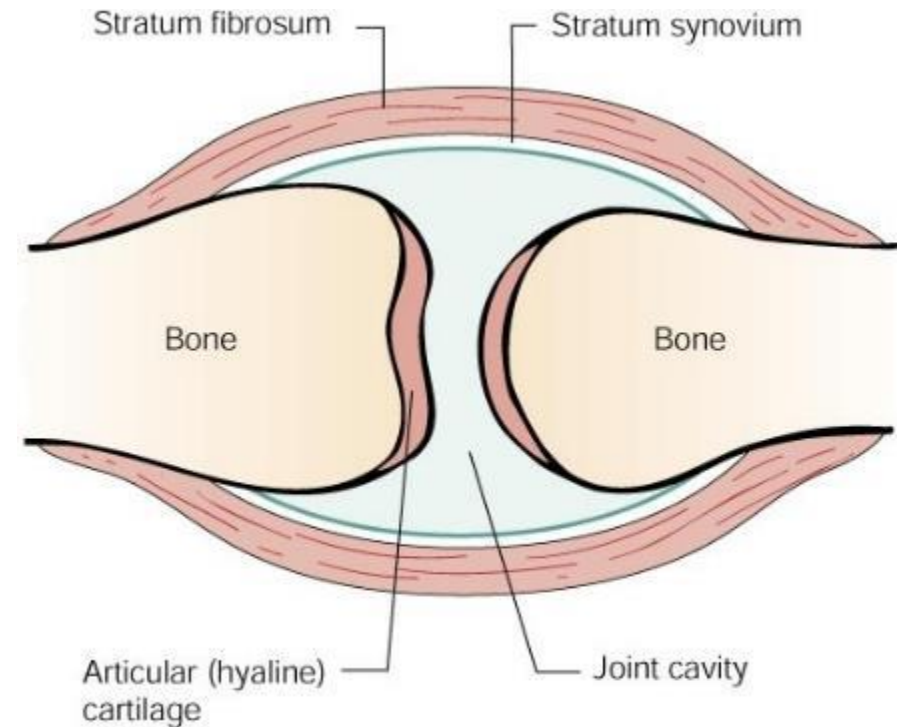
The Joint Capsule

Stratum synovium-inner layer

- highly vascularized
- poorly innervated
- synthesizes the **hyaluronic acid** component of synovial fluid and matrix collagen fibers
- essential for joint **nutrition**

Stratum fibrosum-outer layer

- poorly vascularized
- highly innervated by joint receptors for proprioception, motion, vibration and pain



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Inert Soft Tissue Injuries

- **Joint capsule injuries**
 - **Immobilization** of the joint occurs due to
 - Inflammation with edema
 - Fibrotic thickening
 - Immobilization has **two effects**:
 - **Reduced flow** of synovial fluid reduces joint nutrition. Joint movement is especially important for nourishment of cartilage due to its avascularity.
 - **Fatty tissue builds up** in the joint space causing adhesions. Contracture of the capsule and the muscles spanning the joint occurs.
 - **Frozen shoulder (adhesive capsulitis)** is an example of the effects of immobilization.
 - Treatment-intra-articular corticosteroid injections, gentle stretching, anti-inflammatory oral meds

Inert Soft Tissue Injuries

- **Internal joint derangement** – injury to inert soft tissue structures inside a joint
 - Menisci (cartilage) in knee, labrum (cartilage cuff for a rounded bone marking) in shoulder or hip joint or TMJ (temporomandibular joint)
 - Restriction of motion, pain
- **Injuries to fasciae and bursae**
 - Injury may cause pain and restriction of motion due to edema and scarring
 - **Fasciitis**-inflammation of a fasciae (fibrous connective tissue); plantar fasciitis affecting the foot, for example
 - **Bursitis** – inflammation of a bursal sac (fibrous connective tissue) at a stress point in joint. Synovial fluid is located inside the bursal sac. Bursitis commonly effects the shoulder, hip and elbow (joints that experience repetitive motion).
- **Joint abnormalities causing damage to nerves, nerve roots, dura mater**
 - Pain, altered sensation, weakness, reduction of reflexive activity
 - Pinched nerves, herniated discs

Inert Soft Tissue Injuries

- Inflammation or pressure on a neural structure causes pain that radiates throughout the structures served by the nerve. Severe compression can lead to loss of function (sensory and motor).
- The most common site of such injuries is due to trauma to the vertebrae of the **lumbosacral** region. Most commonly an intervertebral disc is subjected to unequal compression causing it to herniate. Common sites of **disc herniation** are in the L3 to S1 region. Nerve roots affected are associated with spinal nerves L4, L5 and S1. These nerves contribute to the formation of the **sciatic nerve**.
- The term **sciatica** is used to describe the pain in structures innervated by the sciatic nerve: posterior structures from the waist down and the foot.

Inert Soft Tissue Injuries

3 joints of the knee



Talofibular ligaments



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Inert Soft Tissue Injuries

Frozen Shoulder

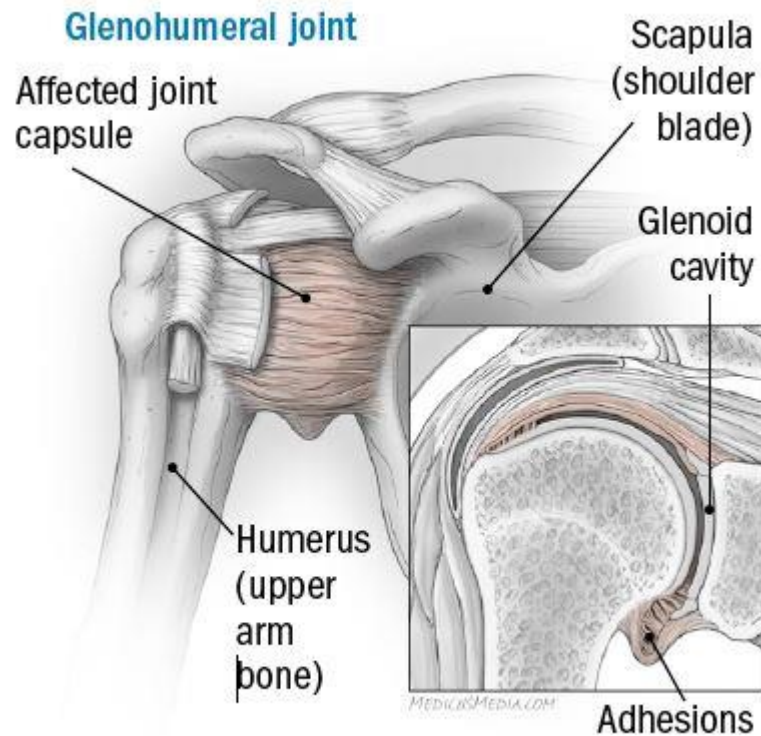
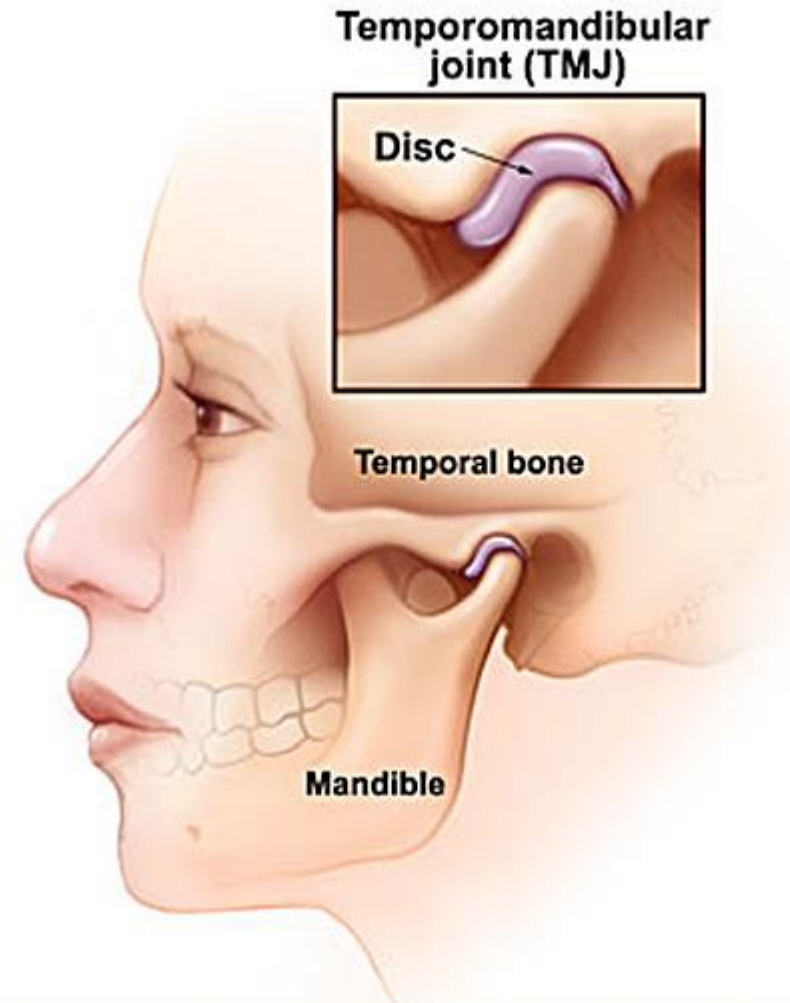
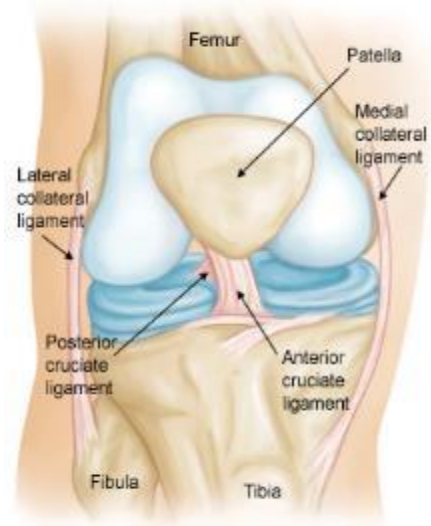


Illustration: Scott Leighton

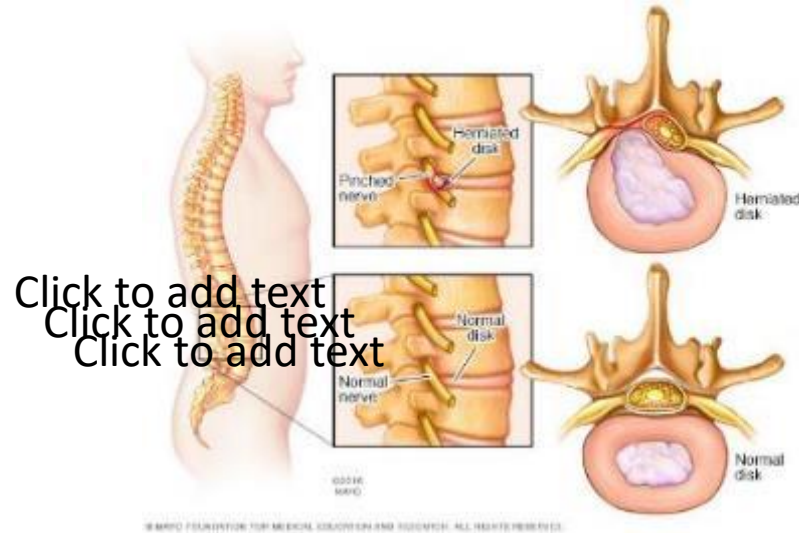


Inert Soft Tissue Injuries

ACL-Anterior
Cruciate Ligament

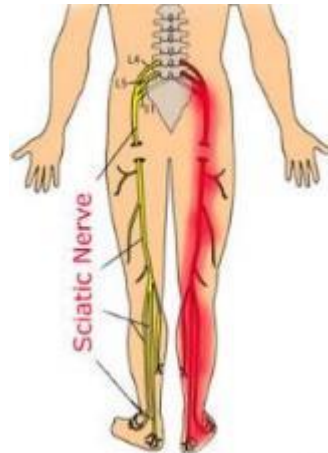


Herniated Disc and Nerve Impingement



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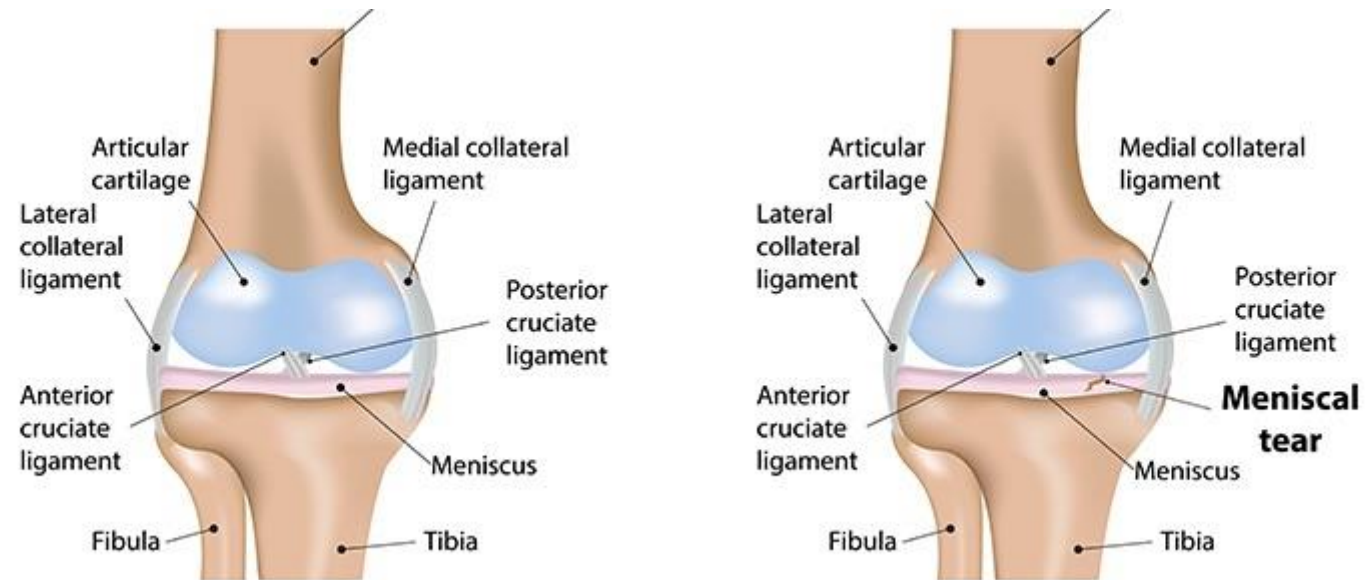
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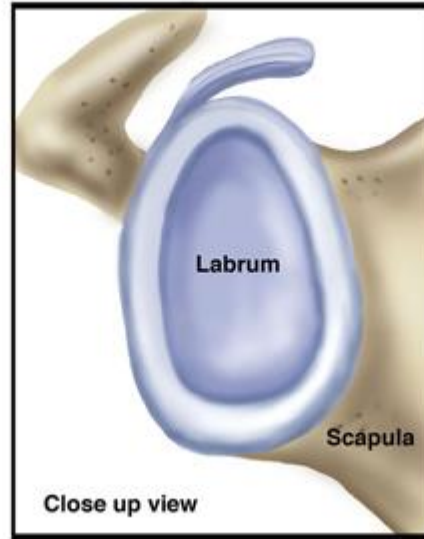
Inert Soft Tissue Injuries

Torn (Cartilage) Meniscus of the Knee

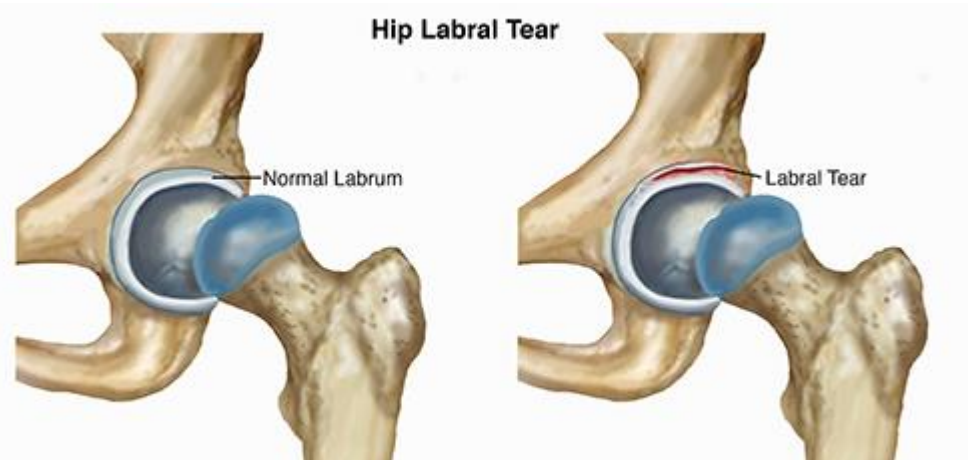


Inert Soft Tissue Injuries

Labral Tear



Hip Labral Tear



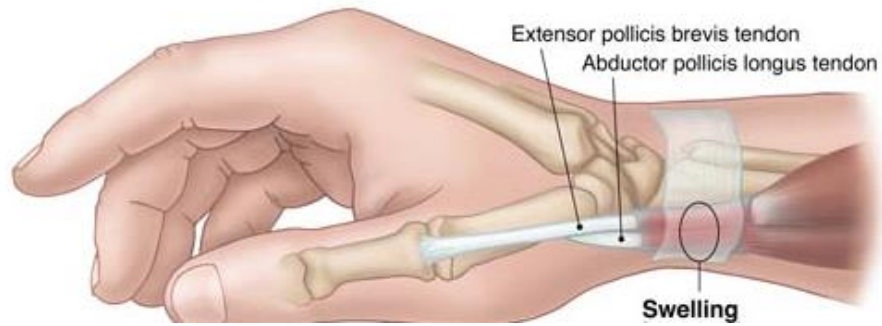
Contractile Soft Tissue Injuries

Injury to Tendons

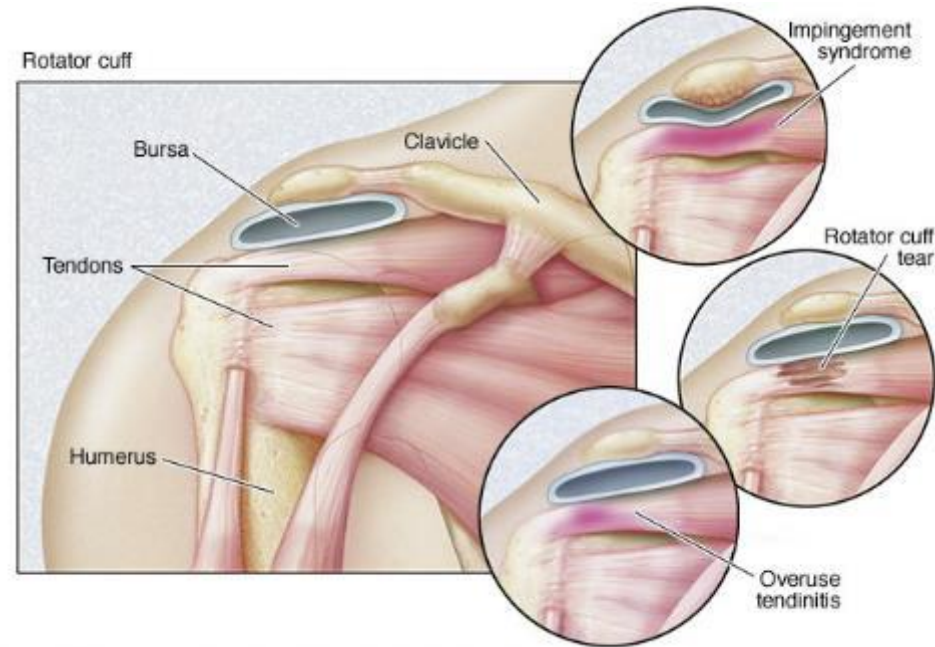
- **Tendonitis:** inflammation of a tendon due to injury, repetitive motion, or infection.
- Commonly injured tendons:
 - **Extensor pollicis brevis and abductor pollicis longus** tendons of the thumb (**de Quervain syndrome**); becoming more common due to texting and video gaming technology
 - **Rotator cuff tendons** of the shoulder
 - **Biceps brachii tendon**
 - **Quadriceps tendon, hamstring tendons, Achilles tendon, tibialis posterior tendon.**

Contractile Soft Tissue Injuries

deQuervain Syndrome

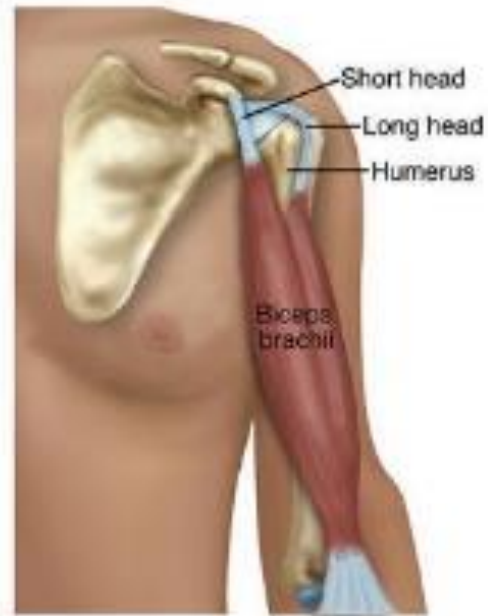


Rotator Cuff Tendon Injury



Contractile Soft Tissue Injuries

Biceps Tendinitis



Normal Anatomy



Tendinitis of the Long Head



Contractile Soft Tissue Injuries

Muscle and Tendon Strains

- Normally due to abnormal muscle contraction
- Classified by severity of injury
 - Grade I – minute tear of connective tissue and muscle fiber
 - Grade II – tear of large portion of contractile unit, segment still intact
 - Grade III – total rupture or loss of continuity of contractile unit

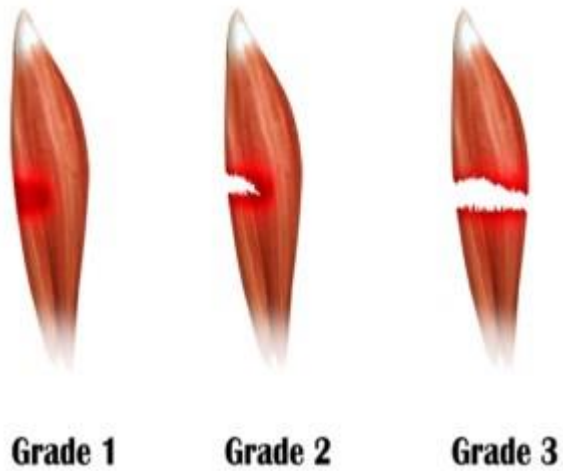
Blunt trauma

- Contusion or crush injury to soft tissue compromises contractile structure
- Bleeding into muscle belly can lead to inability to contract muscle.
- Hemorrhage may cause blood to coagulate and calcify, condition called **myositis ossificans**. (Myositis means inflammation of muscle and ossificans refers to calcium deposit.)

Contractile Soft Tissue Injuries

Myositis Ossificans

Muscle Strains



Contractile Soft Tissue Injuries

Compartment Syndrome

- Trauma to soft tissue (ischemic injury) caused by unyielding structure of **fasciae**.
- Three categories of cause:
 - Decreased compartment size (fibrosis of the fasciae)
 - Increased compartment content (edema or bleeding)
 - Externally applied pressure (bandage or cast)
- All three forms cause pressure that **reduces capillary blood flow**.
- Medical emergency; nerves can survive 2-4 hours of ischemia; muscles 6-8 hours
- The compartment **must be opened (fasciotomy)** to relieve pressure.
- Necrotic muscle tissue is later replaced by fat and fibrous CT.
- Compartments most often affected: leg, forearm, deltoid, biceps brachii, quadriceps, hand, gluteal

Contractile Soft Tissue Injuries

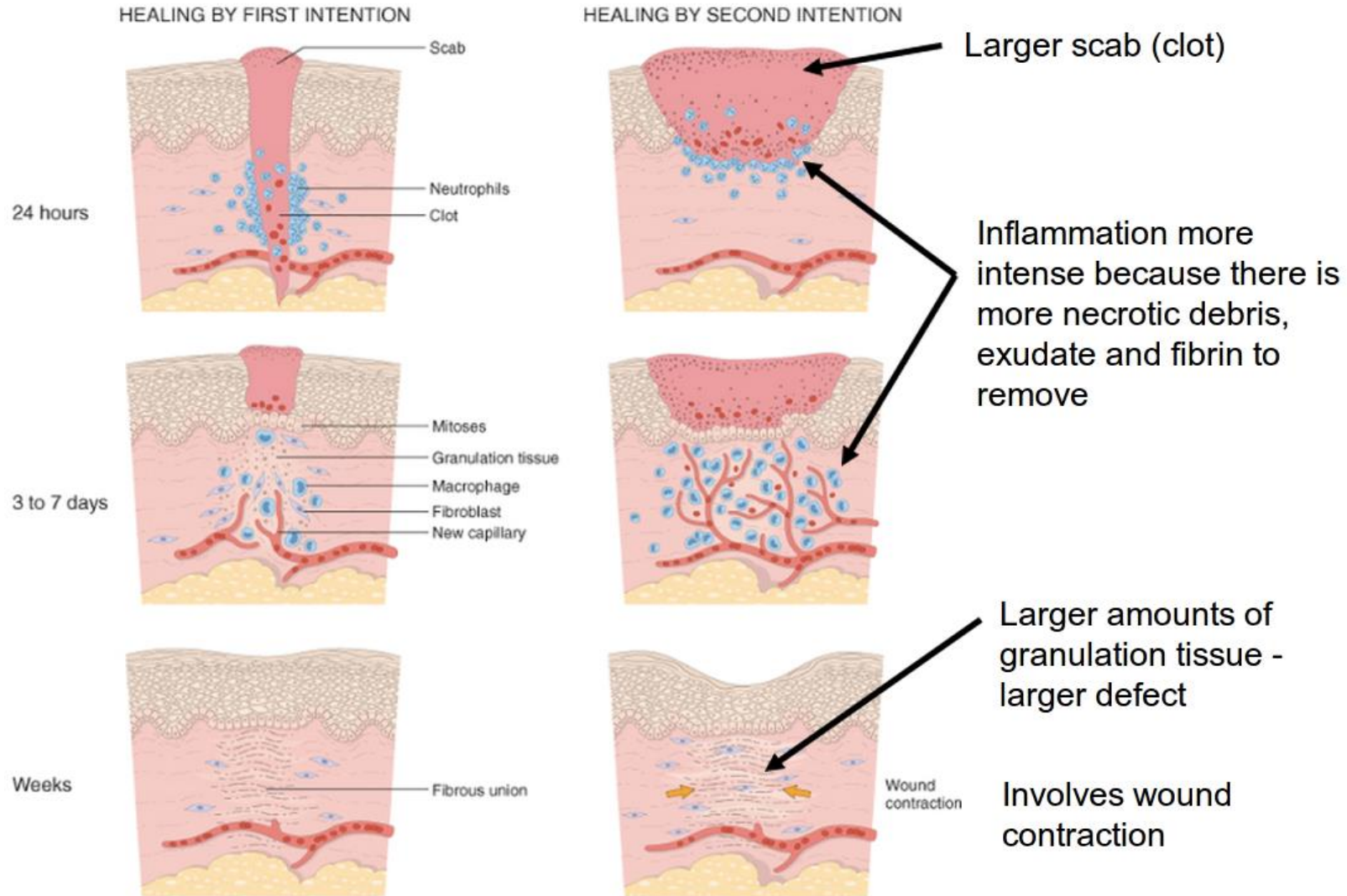
Fasciotomy: Compartment Syndrome with
Extensive Necrosis



Contractile Soft Tissue Injuries

- **Healing of Soft Tissue Injuries: 5 General Steps**
 - Blood clot formation
 - Inflammation
 - Angiogenesis
 - ****Granulation tissue formation**
 - ****Wound Contraction**
- Note that some soft tissues (**tendons, ligaments, cartilage**) are avascular or poorly vascularized so healing occurs more slowly. Biochemicals must diffuse in from surrounding vascularized tissue.
- Depending on the size of the wound, healing may be by **primary union/first intention** (small, clean wound) or **secondary union/second intention** (large, dirty wound).
- ****Granulation tissue formation and wound contraction are much more pronounced in secondary intention healing.**

Contractile Soft Tissue Injuries



Contractile Soft Tissue Injuries

Differences between Primary and Secondary union of wounds

Feature	Primary Union	Secondary Union
1. <i>Cleanliness of wound</i>	Clean	Unclean
2. <i>Infection</i>	Generally uninfected	May be infected
3. <i>Margins</i>	Surgical clean	Irregular
4. <i>Sutures</i>	Used	Not used
5. <i>Healing</i>	Scanty granulation tissue at the incised gap and along suture tracks	Exuberant granulation tissue to fill the gap
6. <i>Outcome</i>	Neat linear scar	Contracted irregular wound
7. <i>Complications</i>	Infrequent, epidermal inclusion cyst formation	Suppuration, may require debridement

Bone and Joint Trauma

Types of Bone Tissue

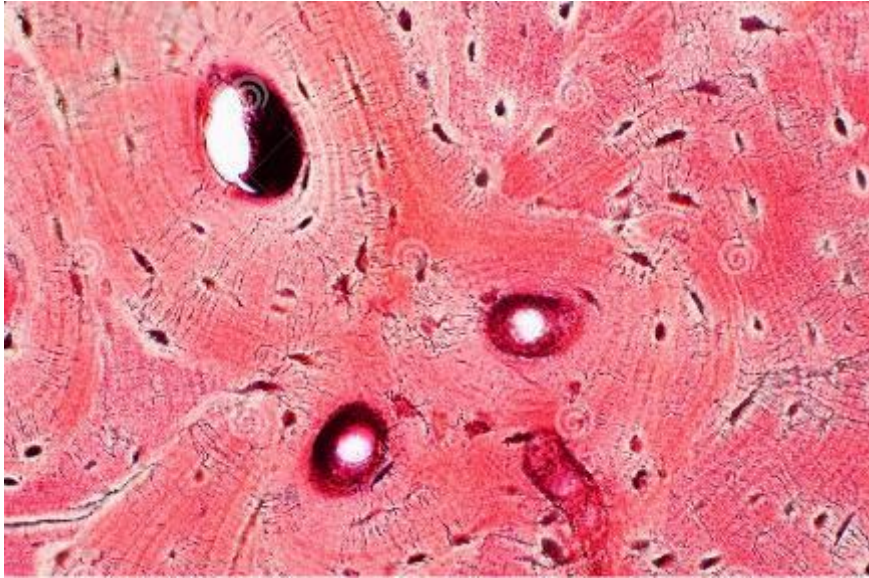
- **Cortical (Compact) Bone**

- Located on the surface of bones.
- Tolerates compression and shearing forces.
- Collagen fibers allow a great deal of tensile strength (resistance to pulling forces).
- Fractures occur when pulling forces exceed the tensile strength.

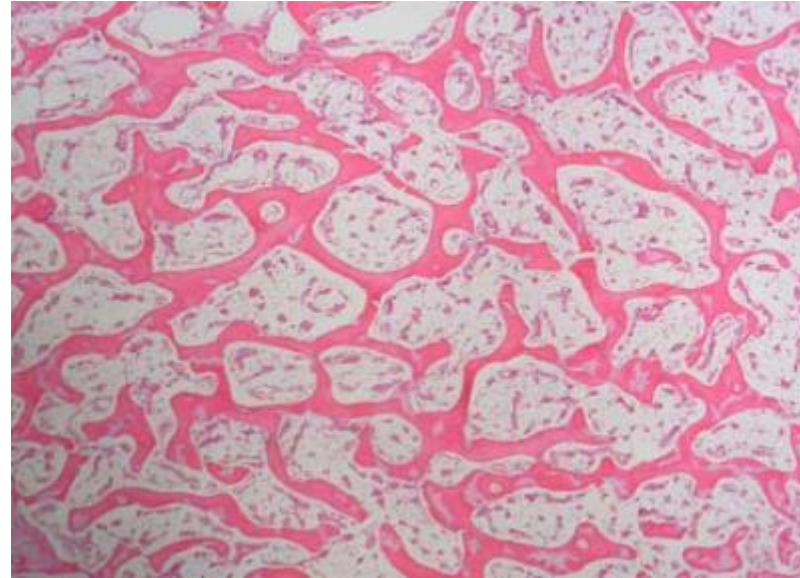
- **Cancellous (Spongy) Bone**

- Located deep to cortical bone.
- Protects red bone marrow especially in flat bones like the skull bones and the hip bones.
- 20-50x less resistant to compression forces than compact bone.
- 5-7x less resistant to pulling forces than compact bone.

Bone and Joint Trauma

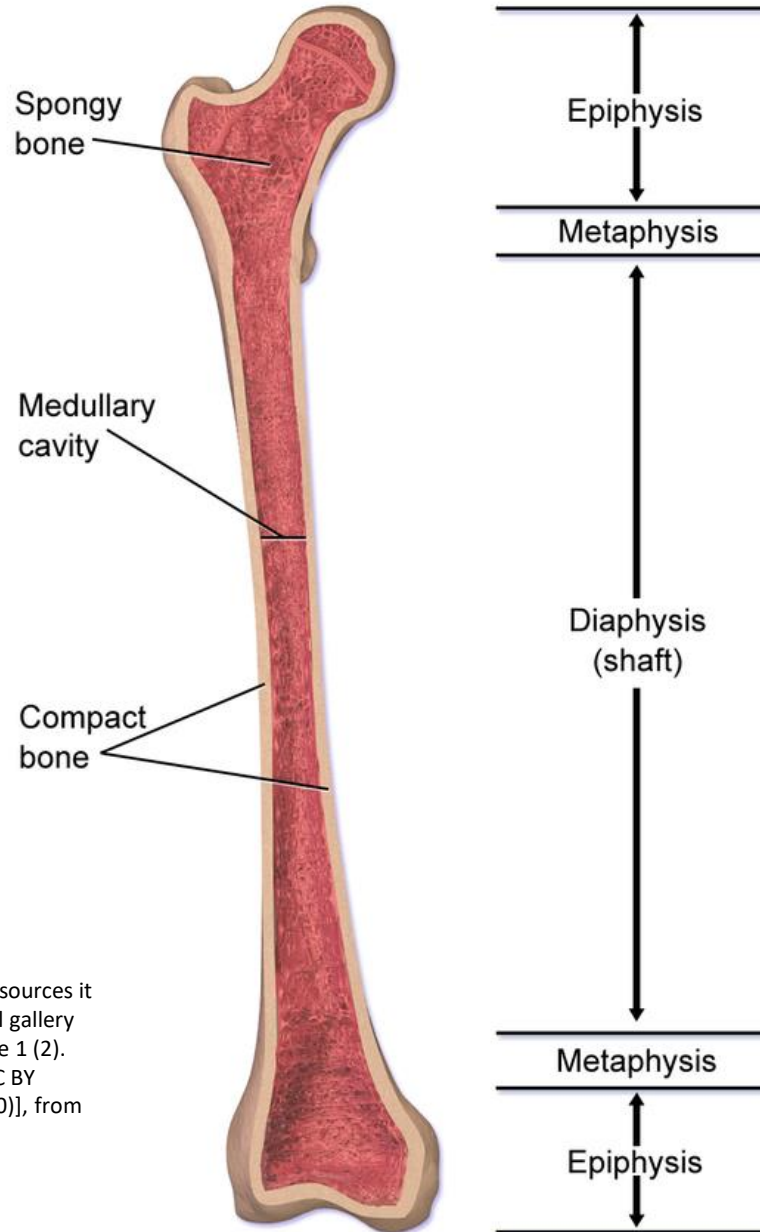


Compact Bone



Spongy Bone

Structure of a Long Bone

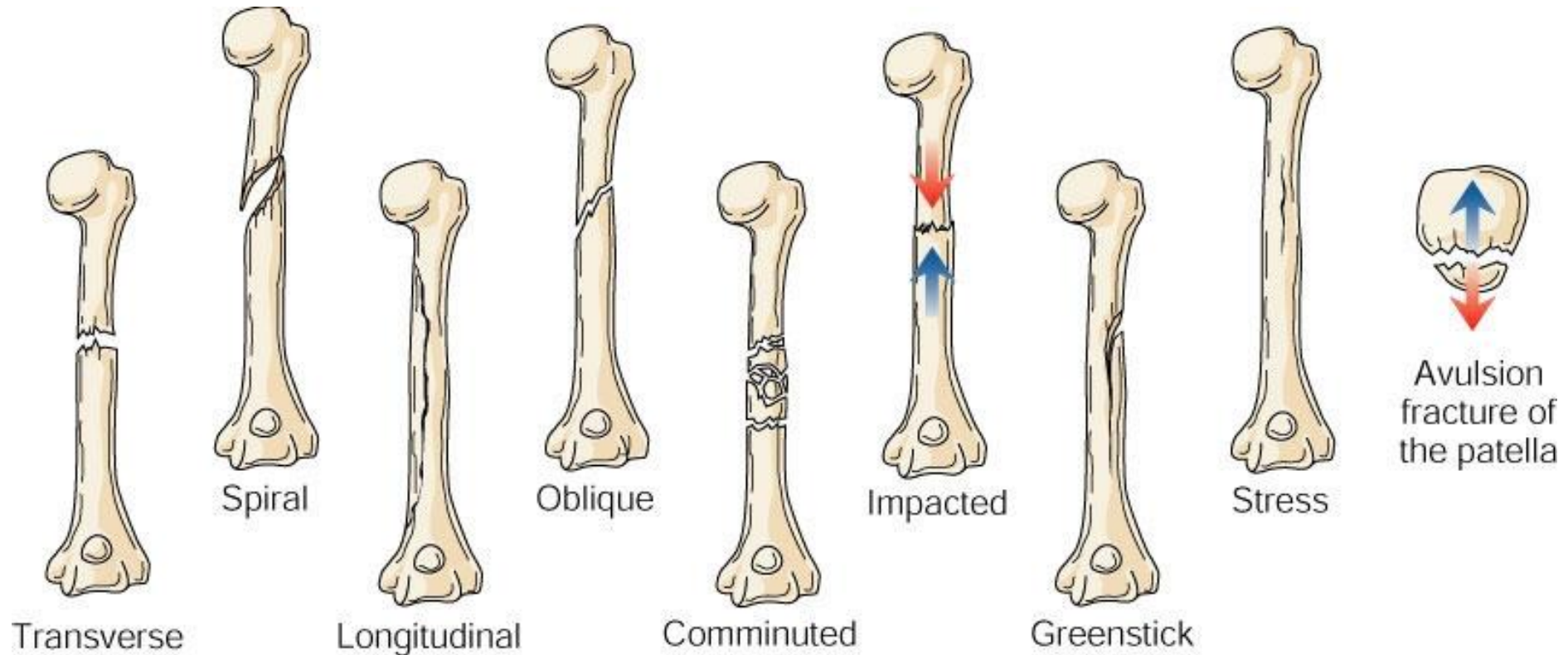


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By BruceBlas. When using this image in external sources it can be cited as:Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". WikiJournal of Medicine 1 (2). DOI:10.15347/wjm/2014.010. ISSN 2002-4436. CC BY 3.0 (<https://creativecommons.org/licenses/by/3.0/>), from Wikimedia Commons

Bone and Joint Trauma

Bone Fracture Types



Bone and Joint Trauma

Age-Specific Fracture Types

- **Epiphyseal plate (growth plate) fracture** may cause cessation of longitudinal bone growth in children.
- **Buckle (torus) fractures** occur in the long bones of children. The bone bends and folds in on itself on the **concave** (inside) surface of the bend. These fractures are often not visible on x-rays.
- **Greenstick fractures** also occur in children. The bone bends and cracks on the **convex** (outside) surface of the bend. They are visible on x-ray.
- **Compression fractures** often occur in the vertebral body if osteoporosis is present, as is often the case in older individuals.

Fracture Diagnosis

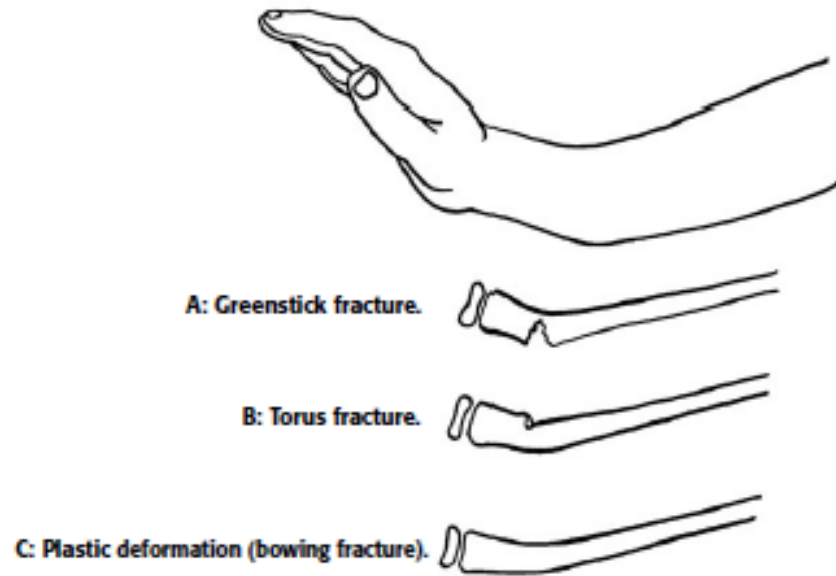
- Most commonly by x-ray
- CT or MRI may be required to reveal non-displaced fractures.

Fracture Treatment

- Ice, elevation and immobilization to minimize soft tissue damage
- Reduction (setting) and immobilization (casting) to provide optimal healing.

Bone and Joint Trauma

Buckle (Torus) Fracture vs Greenstick Fracture



Epiphyseal Plate Fracture



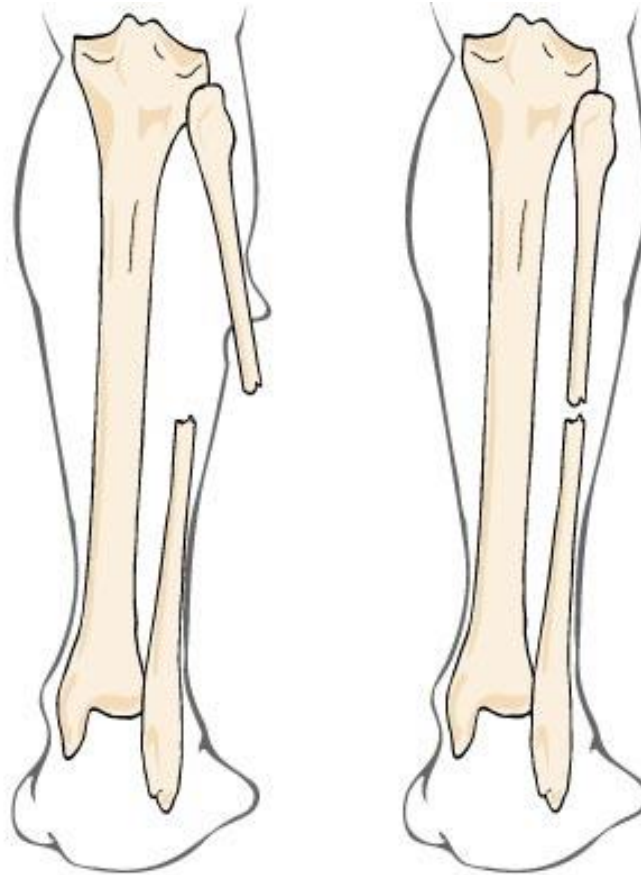
Bone and Joint Trauma

Extent of Bone Fracture

- **Displaced** – ends of fractured pieces are out of normal alignment.
- **Nondisplaced** – fracture fragments remain in alignment and position.
- **Depressed** – bone fragment is displaced below the normal level (skull).
- **Complete** – fracture disrupts bone continuity through whole bone thickness including the cortex.
- **Incomplete** – cortex buckles or cracks with no loss of continuity.
- **Open (compound)** bone fracture creates an external wound; increased risk of infection.
- **Closed (simple)** bone fracture does not create an external wound.

Bone and Joint Trauma

Open and Closed Fractures



Open (compound)

Closed (simple)

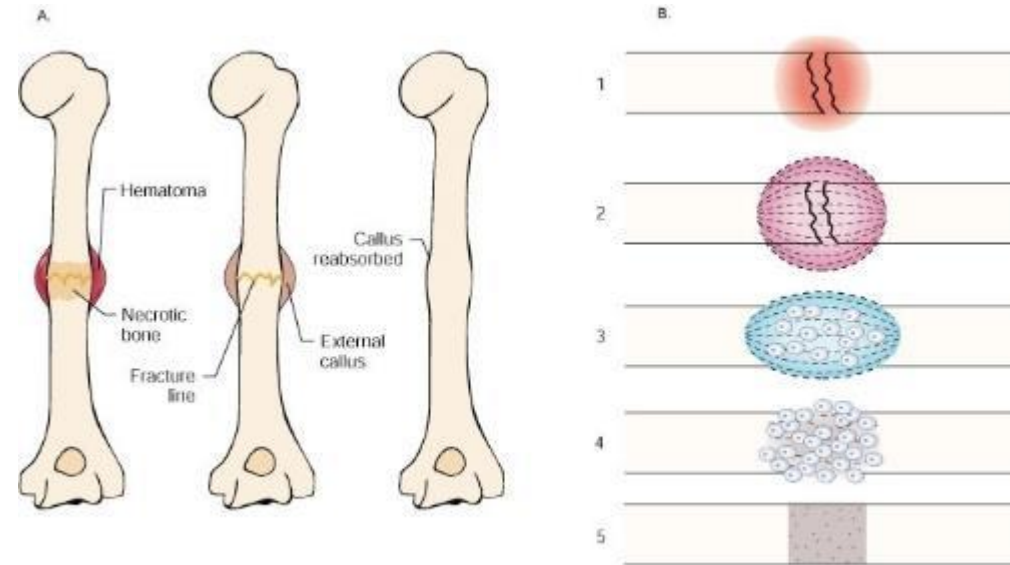
Healing of Bone Fracture

Cancellous Bone Healing

- The **rich blood supply** in cancellous bone prevents necrosis at the fracture site. (Some necrosis occurs in compact bone fracture.)
- **Osteoblasts** from the **endosteum** of the **trabeculae** form the new **woven bone** (a disorganized form of bone tissue) of **the internal callous**. It fills the open space in the fracture site.
- The woven bone is later **remodeled**--converted to mature cancellous bone.

Bone and Joint Trauma

5 Stages in Healing of Cortical Bone



From Lewis SM, Helikemper MM, Dirksen SR: Medical-surgical nursing, ed 6, St Louis, 2004, Mosby, p 1658.

1. Hematoma Formation
2. Fibrous Network Formation in the Hematoma
3. Invasion of Osteoblasts (or Chondroblasts where blood supply is low)
4. Bony Callus Formation: Woven Bone
5. Remodeling: Woven Bone to Cortical Bone

Healing of Bone Fracture

Bone Tissue Complications: Delayed Healing

- Subsequent injury or inflammation/infection
- Smoking, malnutrition, corticosteroids, poor circulation
- **Malunion** is due to unequal stress of muscle pull and gravity.
- **Nonunion** occurs when there is no fracture healing by 4-6 months after injury – factors listed above contribute.

Bone Tissue Complications: Osteonecrosis

- Due to restricted blood circulation and ischemia.

Bone Tissue Complications: Osteomyelitis

- Due to bone infection, most often associated with open fractures.

Bone Tissue Complications: Fat Emboli Syndrome

- Fat from the marrow cavity enters the circulation and causes emboli in the lungs (pulmonary embolus).

Healing of Bone Fracture

Bone Tissue Complications: Deep Vein Thrombosis (DVT)

- Thrombus, usually in an extremity, breaks loose and forms an embolus that may lodge in the lungs (pulmonary embolus).

Soft Tissue Complications of Bone Fracture

- Compartment syndrome
- Neurovascular injury

Joint Injury (often accompanies bone fracture)

- **Dislocation** – displacement of a bone so that articulating surfaces lose contact
- **Subluxation** – displacement of a bone so that articulating surfaces partially lose contact
- Most commonly affected joints: fingers, femoropatellar, shoulder

Infections Of The Bone

Osteomyelitis

- Severe pyogenic infection of red bone marrow-rich bones and local tissue, requires immediate treatment. If not caught early, the body attempts to **“wall off” the infection** making treatment difficult.
- Infectious route:
 - blood stream; most common
 - from adjacent soft tissue
 - direct introduction of pathogen into bone via trauma or surgery (periodontal surgery, prosthesis placement, etc.)
- The most common infective organism is *Staphylococcus aureus*. In young children *Haemophilus influenzae* was commonly causative, but is rarely causative now due to the vaccine.
- **In children**
 - Manifests as high fever, swelling and pain at site of bone involvement.
 - Child may refuse to walk.
 - **Brodie abscess** occurs when fibrotic tissue walls off the infected site.
- **In adults**
 - Manifests as more generalized symptoms of infection

Infections Of The Bone

Osteomyelitis

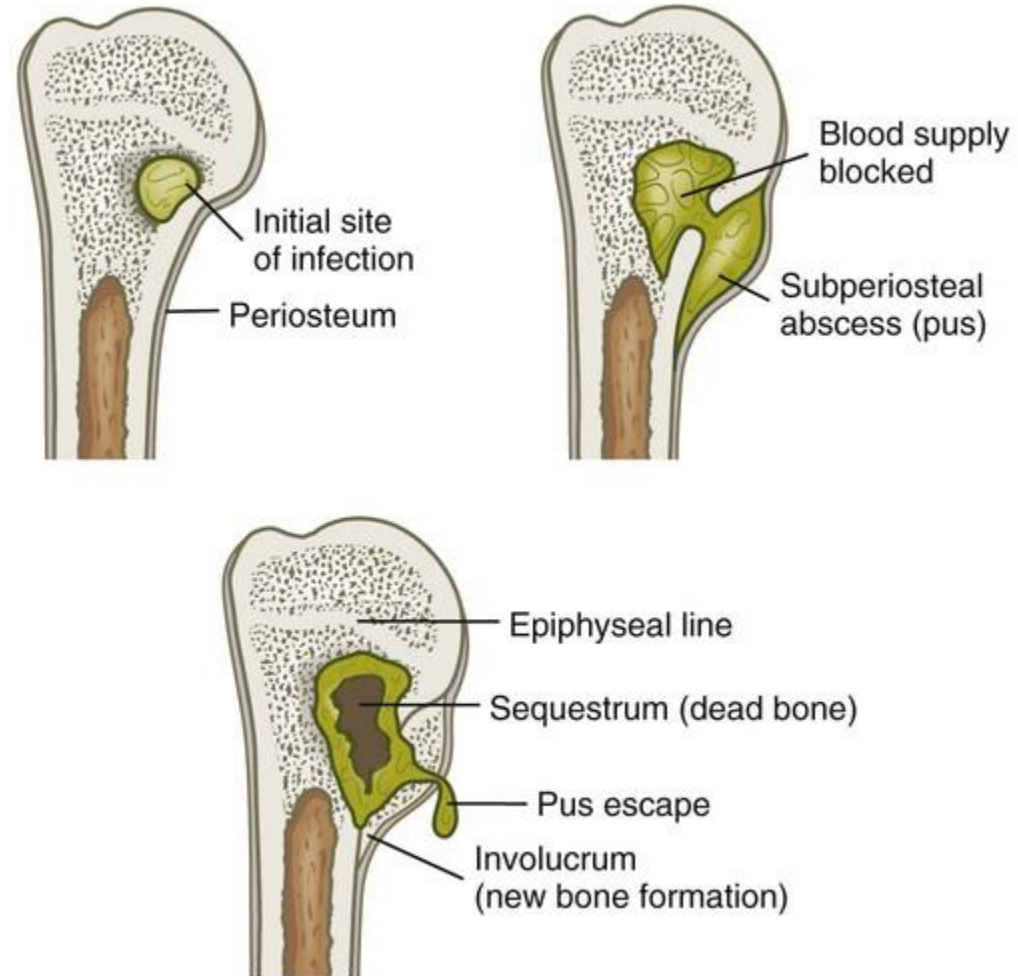
- May occur secondary to burns, sinus infection, tumor necrosis, periodontal infection, infection of a surgical site or infection of a pressure sore.

Osteomyelitis Pathogenesis

- Infection (pus) spreads from cancellous bone to medullary cortical bone. Before puberty, it may spread into the epiphyseal plate.
- **Vascular occlusion** due to edema leads to necrosis of bone tissue.
- Necrotic bone tissue separates from normal bone as a **sequestrum** initiating chronic disease. **Sinuses** from the sequestra to the body surface may form; pus escapes.
- Osteoblasts from the periosteum by forming an **involucrum**, new thick bone just deep to the periosteum near the sequestrum.
- **Treatment** 4-6 wk IV antibiotics; remove prosthesis if causative; debridement may be needed; possible amputation
- Antibiotics and phagocytes may be unable to reach the infected bone tissue due to vascular occlusion or involucrum formation.

Infections Of The Bone

Osteomyelitis Progression



Infections Of The Bone

Tuberculosis of Bones and Joints (Extrapulmonary)

- *Mycobacterium tuberculosis* spreads via lymph or blood from primary lung lesions in about 5% of pulmonary TB cases worldwide.
- It infects bone **where blood supply is richest**; spreads to other bone and joint spaces
- Most commonly (33% of cases) occurs in the vertebral column; **Pott's disease** is TB in thoracic or lumbar vertebrae.
- The highest risk occurs in at both ends of the age spectrum and in immunocompromised (HIV) or undernourished individuals.
- **Treatment:** antibiotics for 9-12 months; surgery may be required in case of severe deformity or neurological deficits.

QUIZ 5CD

- COMPLETE QUIZ 5CD.
- THEN PREPARE FOR EXAM 5.

EXAM 5

- COMPLETE EXAM 5.
- THEN GO ON TO MODULE 6AB PPT.