Wound Management
Surgical Anatomy

Skin is composed of

Epidermis: outermost layer, avascular, receiving nourishment from fluid penetrating the deeper layers and from dermal capillaries

Dermis: lies deep to the epidermis, composed of collagenous, reticular, and elastic fibers surrounded by a mucopolysaccharide ground substance. Fibroblasts, macrophages, plasma cells, and mast cells are found throughout this layer,

The dermis contains blood and lymph vessels, nerves, hair follicles, glands, ducts, and smooth muscle fibers

Associated adnexa: (hypodermis) or subcutis, lies below the dermis

Terminal arteries and veins branch from direct cutaneous vessels and

Form

Subdermal (deep) plexus: supplies hair bulbs and follicles, tubular glands, the deeper portion of the gland ducts, and arrectores pilorum muscles, major importance to skin viability. In areas where there is a panniculus muscle (cutaneous trunci, platysma, sphincter colli superficialis, sphincter colli profundus, preputialis,

supramammarius muscles)

Cutaneous (middle) plexus: It supplies sebaceous glands and reinforces capillary networks around hair follicles, tubular gland ducts, and arrectores pili muscles. , and

Subpapillary (superficial) plexus: Lies on the outer layer of dermis, and capillary loops from this plexus project into and supply the epidermis.

Wound: Disruption of normal tissue integrity.

Wound Healing

Wound healing is a biologic process that restores tissue continuity after injury. It is a combination of

- ❖ Physical,
- ❖ Chemical, and
- ❖ Cellular events that restore wounded tissue or replace it with collagen.

Stages of Wound Healing

1. Inflammatory phase.

This phase is characterized by:

- ❖ Increased vascular permeability,
- ❖ Chemotaxis of circulatory cells,
- ❖ Release of cytokines and growth factors, and
- ❖ Cell activation (macrophages, neutrophils, lymphocytes, and fibroblasts).
- ❖ Hemorrhage cleans and fills wounds immediately after injury.

- Blood vessels constrict for 5 to 10 minutes to limit hemorrhage,
- But then dilate and leak fibrinogen and clotting elements into wounds. Vasoconstriction is mediated by catecholamines, serotonin, bradykinin, and histamine.
- Platelet aggregation and blood coagulation form a clot that ensures hemostasis and provides a scaffold for cell migration
- Platelets also release potent chemoattractants and growth factors (epidermal, platelet-derived, transforming growth factors: a and b) that are necessary in later stages of wound healing
- Fibrin and plasma transudates fill wounds and plug lymphatics, localizing inflammation and "gluing" wound edges together.

- Inflammatory phase cells such as platelets, mast cells, and macrophages secrete growth factors or cytokines, which initiate and maintain the proliferative phase of healing.

- Inflammatory mediators (i.e., histamine, serotonin, proteolytic enzymes, kinins, prostaglandins, complement, lysosomal enzymes, thromboxane, and growth factors) cause inflammation that begins immediately after injury and lasts approximately 5 days.

2. **Debridement phase**:

- Chemoattractants encourage neutrophils and monocytes to appear in wounds (approximately 6 hours and 12 hours after injury, respectively)
- Degenerating neutrophils release enzymes and toxic oxygen products that facilitate the breakdown of bacteria, extracellular debris, and necrotic material, and they stimulate monocytes
- Monocytes are major secretory cells synthesizing growth factors that participate in tissue formation and remodeling
- Monocytes become macrophages in wounds at 24 to 48 hours.

- Macrophages secrete collagenases, removing necrotic tissue, bacteria, and foreign material.
- Macrophages also secrete chemotactic and growth factors. Growth factors (i.e., platelet-derived growth factor, transforming growth factor-α, transforming growth factor-b, fibroblast growth factor, and interleukin-1) can initiate, maintain, and coordinate formation of granulation tissue. Chemotactic factors (i.e., complement, collagen fragments, bacterial endotoxins, and inflammatory cell products) direct macrophages to injured tissue
- Macrophages also recruit mesenchymal cells, stimulate angiogenesis, and modulate matrix production in wounds. Platelets release growth factors important for fibroblastic activity.

❖ Lymphocytes appear later in the debridement phase

3. Repair phase:

- Usually begins 3 to 5 days after injury
- Macrophages stimulate deoxyribonucleic acid (DNA) and fibroblast proliferation. Cytokines, in concert with extracellular matrix molecules, stimulate fibroblasts in the surrounding tissue to proliferate.
- A tissue oxygen content of approximately 20 mm Hg and slight acidity also stimulate fibroblast proliferation and collagen synthesis.
- Fibroblasts originate from undifferentiated mesenchymal cells in surrounding connective tissue and migrate to wounds along fibrin strands in the fibrin clot.
- The amount of collagen reaches a maximum within 2 to 3 weeks after injury.
- Capillaries invade wounds behind migrating fibroblasts by the process of angiogenesis which relying on interaction of extracellular matrix with cytokines that stimulate migration and proliferation of endothelial cells.

- The combination of new capillaries, fibroblasts, and fibrous tissue forms bright red, fleshy granulation tissue 3 to 5 days after injury.

- Granulation tissue fills defects and protects wounds. It provides a barrier to infection, a surface for epithelial migration, and a source of special fibroblasts (i.e., myofibroblasts),
- Epithelial repair involves mobilization, migration, proliferation, and differentiation of epithelial cells.
- Migrating epithelial cells enlarge, flatten, and mobilize, losing their attachments to the basement membrane and other epithelial cells.
- Epithelial cells in the layers behind these altered cells migrate over them until they contact the wound surface.
- The migrating cells move under scabs and produce collagenase, which dissolves the base of the scab so it can be shed.
- ***Wound contraction*** reduces the size of wounds subsequent to fibroblasts, reorganizing collagen in granulation tissue and myofibroblast contraction at the wound edge
- Wound contraction involves a complex interaction of cells, extracellular matrix, and cytokines.
- Wound contraction stops when wound edges meet, when tension is excessive

4. Maturation phase

- Wound maturation begins once collagen has been adequately deposited in wounds (17 to 20 days after injury) and may continue for years.
- The cellularity of granulation tissue is reduced as cells die. There is also a reduction in collagen content of the extracellular matrix
- Nonfunctionally oriented collagen fibers are degraded by proteolytic enzymes (matrix metalloproteinases) secreted by macrophages, epithelial cells, endothelial cells, and fibroblasts within the extracellular matrix.

- ❖ As the number of capillaries in fibrous tissue declines, the scar becomes paler. Scars also become less cellular, flatten, and soften during maturation. Collagen synthesis and lysis occur at the same rate in maturing scars.

Management of Open or Superficial Wounds

❖A wound is classified

A: According to the etiology: skin involvement

Abrasion wound: superficial and involve destruction of varying depths of skin by friction from blunt trauma or shearing forces. Abrasions are sensitive to pressure or touch and bleed minimally

Laceration wound: A laceration is created by tearing, which damages skin and underlying tissue. Lacerations may be superficial or deep and have irregular edges.

Avulsion (degloving injuries) wound: Avulsion wounds are characterized by tearing of tissues from their attachments and creation of skin flaps. Avulsion injuries on limbs with extensive skin loss are called degloving injury.

Puncture wound: A penetrating or puncture wound is created by a missile or sharp object, such as a knife, pellet, or tooth that damages tissue. Wound depth and width vary depending on the velocity and mass of the object creating the wound. The extent of tissue damage is directly proportional to missile velocity. Pieces of hair, skin, and debris can be embedded in wounds.

Crush wound: can be a combination of other types of wounds with extensive damage and contusions to skin and deeper tissue

Burn wound: may be partial- or full-thickness skin injuries caused by heat or chemicals

B: According to the skin involvement

1. **Open wounds: When the whole thickness of skin is opened (Laceration and skinloss)**
2. **Closed wound: Skin is contact (not opened) include crush and contusion.**

C: According to the condition (Clinically) of the wound

1. **Clean wound:**
   Are generally those wounds that are not traumatic in nature, but are uninfected surgically created wound in which either the oropharyngeal cavities or the respiratory, gastrointestinal or genitourinary system are entered. Because clean wounds are created under aseptic or much cleaned condition. They are closed primary.
2. **Clean-contaminated wounds:**
   Also generally that is not traumatic in nature and are wounds are created in surgery that penetrate the respiratory, gastrointestinal, genitourinary tracts or oropharyngeal region system are entered. These wounds can be debrided, lavaged and closed primarily**.**
3. **Contaminated wounds** include open traumatic wounds. Other types of contaminated wounds include surgically created wounds in which there was break in aseptic technique, and surgical incision and wounds made in areas of acute Inflammation or in or near inflamed or contaminated skin

4. **Dirty and infected wounds** include old traumatic wounds as well as those involving clinical infection or perforated viscera. They also include surgical wounds or incision in which pus has contaminated the wound, and those in which infection was present in the operative field prior to the surgical procedure.

Wounds

Treatment

1. **Clean wounds**
   Primary closure (healing by first intention) is used on surgical wounds that are closed immediately.
2. **Contaminated wound**
   - ❖ The patient should be examined
   - ❖ The patient may need stabilization and treatment for shock, dehydration, fracture or other injuries prior treatment of any wound.
   - ❖ It may be necessary to apply a sterile water-soluble gel to the wound
   - ❖ Open wound is to clip the hair surrounding the wound
   - ❖ Sterile sponge is used to wipe the jelly from the wound
   - ❖ The wound should be lavaged using either sterile saline or a sterile dilute povidine-iodine solution or dilute chlrohexidine
   - ❖ Suture the wound, apply drains and/or bandage the wound.

Treatment of infected wound

1. Put clean gauze on the wound, then prepare the area surrounding (clipping, shaving) clean, put antiseptic
2. Remove the gauze, irrigate the wound with warm sterile normal saline (profuse amount) if not available use tap water
3. Remove dead necrotic tissue in the wound (debridement), dead tissue appear as dry black senseless tissue, removal is done till blood oozes from the wound, also cut the wound edges (refresh) to create new fresh wound edge.
4. Irrigate the wound for the second time to remove all tissue debris in the wound using warm sterile normal saline (profuse amount).
5. Put local antibiotics solution
6. Suture the wound with non-absorbable suture interrupted stitches, leaving the last two stitches (at lower part of wound) not sutured for drainage.
7. Give systemic antibiotics for at least 3 days, antitetanic serum is also given in equine. .

Wound Closure

1. Primary wound closure: Wounds may be closed immediately (within 1 to 3 days after injury).
2. Delayed primary wound closure: when they are free of infection but before granulation tissue has appeared
3. Secondary closure: after the formation of granulation tissue
4. Secondary intention: allowed to contract and epithelialize

Factors that affect the decision to close wounds include the following:

1. Amount of time that has elapsed since injury. Wounds older than 6 to 8 hours are initially treated with bandages.
2. Degree of contamination. Obviously contaminated wounds should be thoroughly cleansed and initially treated with bandages.
3. Amount of tissue damage. Wounds with substantial tissue damage have reduced host defenses and are more likely to become infected; therefore they Initially should be treated with bandages.
4. Completeness of debridement. Wounds should remain open if the initial debridement was conservative and if further debridement is necessary.
5. Status of the wound's blood supply. A wound with questionable blood supply should be observed until the extent of nonviable tissue is determined.
6. The animal's health. Animals unable to tolerate prolonged anesthesia are best treated with bandages until their health improves.
7. Extent of tension or dead space. If excessive tension or dead space is present, wounds should be bandaged to prevent dehiscence, fluid accumulation, infection, and delayed wound healing.
8. Location of the wound. Large wounds in some areas (e.g., limbs) are not amenable to closure

Type of wound healing:

1. **Healing by first intention**:

Occurs with clean, incised wounds that are held together. Healing is initiated by movement of epithelial cells from the two edges of the wound toward the center that usually meet within 4 to 7 days

of the incision. It should *only* be performed if less than 6 to 8 hours (within the golden period) have elapsed since injury

2. **Healing by 2nd intention:**
   Wounds with considerable tissue loss, contamination, or infection, or those older than 6 to 8 hours should be treated as open wounds. The wounds will initially begin healing by contraction and epithelialization and may heal completely.

3. **Healing by 3rd intention**:

   When apart of sutured wound is opened and resutured again under aseptic condition, healing of this wound is called of healing by 3^{rd} intention

4. **Healing by mixed intention**:

   When a part of wound heals by 1^{st} intention (sutured part) and the other part heals by 2^{nd} intention.

5. **Healing under scab:**
   This type of healing occur in case of abrasion wound, the transudate dry and form a scab usually fixed to the underlying tissue and healing undergo under the scab. When healing is complete the scab will fall spontaneously. This type of healing is important in healing of wound in equine extremities.

Factor effect wound healing:

A. Local Factors That Influence Healing:

1. **Oxygenation**

   Oxygen is important for cell metabolism, especially energy production by means of ATP, and is critical for nearly all wound-healing processes. It prevents wounds from infection, induces angiogenesis, increases keratinocyte differentiation, migration, and re-epithelialization, enhances fibroblast proliferation and collagen synthesis, and promotes wound contraction. Temporary hypoxia after injury triggers wound healing; Hypoxia can induce cytokine and growth factor production from macrophages, keratinocytes, and fibroblasts. Cytokines that are produced in response to hypoxia include PDGF, TGF-β, VEGF, tumor necrosis factor-α (TNF-α), and endothelin-1, and are crucial promoters of cell proliferation, migration and chemotaxis, and angiogenesis in wound healing

2. **Infections:**

   Once skin is injured, micro-organisms that are normally sequestered at the skin surface obtain access to the underlying tissues.

3. **Foreign body**
4. **Venous sufficiency**

B. Systemic Factors That Influence Healing

1. **Age:** increased age is a major risk factor for impaired wound healing. Many clinical and animal studies at the cellular and molecular level have examined age-related changes and delays in wound healing. Delayed T-cell infiltration into the wound area

with alterations in chemokine production and reduced macrophage phagocytic capacity

2. **Sex Hormones in Aged Individuals**

Sex hormones play a role in age-related wound-healing deficits. A partial explanation for this is that the female estrogens (estrone and 17β-estradiol), male androgens (testosterone and 5α-dihydrotestosterone, DHT), and their steroid precursor dehydroepiandrosterone (DHEA) appear to have significant effects on the wound-healing process

3. Stress

Many diseases-such as cardiovascular disease, cancer, compromised wound healing and diabetes—are associated with stress. Numerous studies have confirmed that stress-induced disruption of neuroendocrine immune equilibrium is consequential to health**.** Stress up-regulates glucocorticoids (GCs) and reduces the levels of the proinflammatory cytokines IL-1β, IL-6, and TNF-α at the wound site. Stress also reduces the expression of IL-1α and IL-8 at wound sites—both chemoattractants that are necessary for the initial inflammatory phase of wound healing

4. Diabetes:

Diabetic individuals exhibit a documented impairment in the healing of acute wounds. Moreover, this population is prone to develop chronic non-healing diabetic foot ulcers (DFUs), which are estimated to occur in 15% of all persons with diabetes. DFUs are a serious complication of diabetes, and precede 84% of all diabetes related lower leg amputations pathophysiological mechanisms. DFUs, like venous stasis disease and pressure-related chronic non-healing wounds, are always accompanied by hypoxia which may be derived from both insufficient perfusion and insufficient angiogenesis. Hyperglycemia can also add to the

oxidative stress when the production of reactive oxygen species (ROS) exceeds the anti-oxidant capacity.

The formation of advanced glycation end-products (AGEs) under hyperglycemia and the interaction with their receptors (RAGE) are associated with impaired wound healing in diabetic mice as well. High levels of metalloproteases (MMP) are a feature of diabetic foot ulcers, and the MMP levels in chronic wound fluids are almost 60 times higher than those in acute wounds. This increased protease activity supports tissue destruction and inhibits normal repair processes. Several dysregulated cellular functions are involved in wounds, such as defective T-cell immunity, defects in leukocyte chemotaxis, phagocytosis, and bactericidal capacity, and dysfunctions of fibroblasts and epidermal cells. These defects are responsible for inadequate bacterial clearance and delayed or impaired repair in individuals with diabetes.

5. **Medications**
   Many medications, such as those which interfere with clot formation or platelet function, or inflammatory responses and cell proliferation have the capacity to affect wound healing.

6. **Obesity:**
   Obese individuals frequently face wound complications; including skin wound infection, dehiscence, hematoma and seroma formation, pressure ulcers, and venous ulcers

7. **Nutrition**
   Malnutrition or specific nutrient deficiencies can have a profound impact on wound healing after trauma and surgery.

Complication of wound:

1. **Bleeding**.
2. **Syncope**: temporary unconsciousness due to anemia ,heart stops suddenly may be due to severe bleeding or a reflex action during rough manipulation .
3. **Shock**: a state in which the amount of oxygen delivered to the tissues is inadequate to maintain normal cellular respiration. The animal is unconscious.
4. **Traumatic neuralgia**.

   A-Primary neuralgia: pain at the wound area which persists for abnormal period may be local at the wound area or extend along the nerve in the injured region.

   B-Secondary neuralgia: pain appears at the time of scar formation, might be limited at the wound or extends to the surroundings.
5. **Damage to nerve**. May cause paralysis to the muscles supplied with that nerve or loss of sensation, may be temporary or permanent depending on degree of injury.
6. **Traumatic emphysema**.

   Occurs due to tissue infiltration with air or gas ,it occurs in penetrating wounds .there is limited soft swelling ,painless ,crepitating .un harmful unless infection supervene especially when the gas comes from the digestive tract.
7. **Traumatic fever**.

   Fever occurs due to toxins absorbed from the wounded area.
8. **Cellulitis**.

   Spread of infection in the connective tissue subcutaneously , the affected area tends to suppurate and necrotize ,the wound edges are swollen ,hot ,painful, superficial lymphatic's are prominent with fever and anorexia.
9. **Erysipelas**.

   Infectious disease affects skin and subcutaneous tissue caused by streptococcus infecting the wound. Occurs in equine, dogs, ovine, and workers in pig stables. There are two forms of the disease:

A-Cutaneous form: There is hot painful extensive swelling spreads quickly may be accompanied with lymphadenitis and lymphangitis

b-Phlegmonous and gangrenous form. In the phlegmonous there is extensive suppuration, fever, anorexia with bad prognosis. While in the gangrenous there are signs of moist gangrene

10. Septicemia & pyemia.

11. Venous thrombosis and embolism: Venous thrombosis occurs frequently because blood flow is slower it occurs due to phlebitis in the vein that passes through the wounded area

12. Gas gangrene.

Rarely occurs, happens in deep tortuous wound infected with anaerobs.