# Wound

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- Define and classify wound.
- Write in details about wound healing and factors affecting wound healing.
- Describe the biological process of wound healing.

Wound can be defined as an injury, usually involving division of tissue or rupture of the integument or mucus membrane, due to external violence or some mechanical agency rather than disease. General wounds are classified as being:

- Superficial (loss of epidermis only)
- Partial thickness (involve the epidermis and dermis)
- Full thickness (involve the dermis, subcutaneous fat and sometimes bone)

According to level of contamination a wound can be classified as

- Clean wound, a wound made under sterile conditions where there are no organisms present in the wound and the wound is likely to heal without complications.
- Contaminated wound, where the wound is as a result of accidental injury where there are pathogenic organisms and foreign bodies in the wound.

- Infected wound, where the wound has pathogenic organisms present and multiplying showing clinical signs of infection, where it looks yellow, oozing pus, having pain and redness.
- Colonized wound, where the wound is a chronic one and there are a number of organisms present and very difficult to heal as in a bedsore.

Open wounds can be classified according to the object that caused the wound.

The types of open wound are:

- Incisions or incised wounds, caused by a clean, sharp-edged object such as a knife, razor or glass splinter.
- Lacerations, irregular tear-like wounds caused by some blunt trauma. Lacerations and incisions may appear linear (regular) or stellate (irregular). The term *laceration* is commonly misused in reference to incisions.
- Abrasions (grazes), superficial wounds in which the topmost layer of the skin (the epidermis) is scraped off. Abrasions are often caused by a sliding fall onto a rough surface.

- Avulsions, injuries in which a body structure is forcibly detached from its normal point of insertion. A type of amputation where the extremity is pulled off rather than cut off.
- Puncture wounds, caused by an object puncturing the skin, such as a splinter, nail or needle.
- Penetration wounds, caused by an object such as a knife entering and coming out from the skin.
- Gunshot wounds, caused by a bullet or similar projectile driving into or through the body. There may be two wounds, one at the site of entry and one at the site of exit, generally referred to as a "through-and-through."

Closed wounds have fewer categories, but are just as dangerous as open wounds.

The types of closed wounds are:

- Hematomas, also called a blood tumor, caused by damage to a blood vessel that in turn causes blood to collect under the skin.
  - Hematomas that originate from internal blood vessel pathology are petechiae, purpura, and ecchymosis. The different classifications are based on size.
  - Hematomas that originate from an external source of trauma are contusions, also commonly called bruises.
- Crush injury, caused by a great or extreme amount of force applied over a long period of time.

Surgical wounds:

 Class I: Clean 

 Uninfected operative wound where no inflammation is encountered and respiratory, GI, genital, and urinary tracts aren't entered.
 Wounds are primarily closed, and a drain (if needed) is connected to a closed system.

  Class II: Clean Contaminated have no signs of infection at the time of surgery but do involve repairing or removing an organ.

Example of this type of wound include surgery on lungs, appendix and vaginal procedure

- Class III: Contaminated 

   Open, fresh, accidental wound from surgery with a major break in sterile technique or gross spillage from GI tract; incision in which acute, nonpurulent inflammation is encountered (including necrotic tissue without evidence of purulent drainage, such as dry gangrene)
- Class IV: Dirty/infected Old traumatic wounds with retained devitalized tissue; procedures with existing clinical infection (purulence already present in wound) or perforated viscera.

### Local and Systemic Impediments to Wound Healing

Systemic	Local
Malnutrition	Wound infection
Diabetes mellitus	Wound necrosis
Drugs (steroids, cytotoxins)	Foreign bodies
Obesity	Wound hypoperfusion and hypoxia
Shock	Repeat trauma
Immunodeficiency	Irradiated tissue
Renal failure	Neoplasm

Intrinsic or local factors:

 Ischemia and hypoxia are common contributing causes of nonhealing wounds. Atherosclerosis or local damage to vessels in the form of trauma or vasculitis causes ischemia and subsequent hypoxia in the wound. Hypoxia leads to impaired collagen synthesis, prevents fibroblast migration, and increases the susceptibility of the wound to infection.

 Infection in the wound delays healing. The host's resistance can be impaired by diabetes, malnutrition, malignancy, steroids, or other immunosuppressive therapies. If there are allowed to persist, wound infections lead to increased tissue destruction and alter the effect of cytokines on wound healing.

- The presence of foreign bodies and necrotic tissue can contribute to delayed wound healing.
- Chronic venous insufficiency leads to persistent venous hypertension and chronic edema in the lower extremities. These factors in turn lead to pericapillary fibrosis, tissue ischemia, and the liberation of superoxide radicals, which are thought to result in delayed wound healing in extremities with chronic venous insufficiency.
- Ionizing radiation to the wound leads to abnormal wound healing.
- Edema. Acute swelling, especially around joints, can lead to skin breakdown and full-thickness skin loss.

#### **Extrinsic or systemic factors:**

- Malnutrition alters normal healing through the indirect and the direct effects of vitamin and mineral deficiency. An example is the patient with clinical or subclinical scurvy (vitamin C deficiency); such an individual produces inadequately hydroxylated collagen, and the healed wound becomes significantly weakened as a result.
- Diabetes mellitus is believed to affect healing adversely at every level and in every phase of the healing process.

- Steroids and antineoplastic drugs can markedly diminish the speed and quality of the healing process. Vitamin A seems to cause a partial reversal of the detrimental effects of steroids on healing.
- Smoking contributes to delayed wound healing by causing cutaneous vasoconstriction, decreasing the oxygen-carrying capacity of hemoglobin, and contributing to atherosclerosis.
- Collagen vascular diseases are often accompanied by a vasculitic component, which needs to be controlled before healing can begin.

- Cleansing agents such as chlorhexidine gluconate (Hibiclens) or povidone-iodine (Betadine) or chemicals may impair wound healing by affecting cell migration.
- Repetitive trauma, intentional or otherwise, from shearing or pressure forces often leads to a failure in healing. Wound areas over pressure points often require stabilization of the overlying skin envelope with external taping or splinting.

- Wounds in patients with renal disease and liver disease often heal more slowly due to chronic protein deficiencies and reduced capacities for protein synthesis.
- Hematopoietic disorders. Sickle cell disease, with its high incidence of ankle wounds and leukoclastic and granulomatous processes, and mycosis fungoides are associated with poorly healing wounds. Maximal medical treatment for the underlying disorder is needed to effect meaningful healing.

## **Biological process of wound healing:**

 Wound healing is an intricate process where the skin or other body tissue repairs itself after injury. In normal skin, the epidermis (surface layer) and dermis (deeper layer) form a protective barrier against the external environment. When the barrier is broken, an orchestrated cascade of biochemical events is quickly set into motion to repair the damage.

 This process is divided into predictable phases: blood clotting (hemostasis), inflammation, the growth of new tissue (proliferation), and the remodeling of tissue (maturation). Sometimes blood clotting is considered to be part of the inflammation stage instead of its own stage.  Hemostasis (blood clotting): Within the first few minutes of injury, platelets in the blood begin to stick to the injured site. This activates the platelets, causing a few things to happen. They change into an amorphous shape, more suitable for clotting, and they release chemical signals to promote clotting. This results in the activation of fibrin, which forms a mesh and acts as "glue" to bind platelets to each other. This makes a clot that serves to plug the break in the blood vessel, slowing/preventing further bleeding.

 Inflammation: During this phase, damaged and dead cells are cleared out, along with bacteria and other pathogens or debris. This happens through the process of phagocytosis, where white blood cells "eat" debris by engulfing it. Platelet-derived growth factors are released into the wound that cause the migration and division of cells during the proliferative phase.

#### • Proliferation (growth of new tissue):

- In this phase, angiogenesis, collagen deposition, granulation tissue formation, epithelialization, and wound contraction occur. In angiogenesis, vascular endothelial cells form new blood vessels. In fibroplasia and granulation tissue formation, fibroblasts grow and form a new, provisional extracellular matrix (ECM) by excreting collagen and fibronectin. Concurrently, re-epithelialization of the epidermis occurs, in which epithelial cells proliferate and 'crawl' atop the wound bed, providing cover for the new tissue.
- In wound contraction, myofibroblasts decrease the size of the wound by gripping the wound edges and contracting using a mechanism that resembles that in smooth muscle cells. When the cells' roles are close to complete, unneeded cells undergo apoptosis.

 Maturation (remodeling): During maturation and remodeling, collagen is realigned along tension lines, and cells that are no longer needed are removed by programmed cell death, or apoptosis.