

Normal Wound Healing

The Wound Healing Process is defined by the Wound Healing Society (WHS) as “a complex and dynamic process that results in restoration of anatomic continuity and function”. Wound healing, though often taken for granted, is a very dynamic and delicate process. The wound healing process is a cascade of events, beginning with injury to tissue. Appropriate wound management is dependent on an understanding of the normal repair process, the factors affecting this process and the interventions that can impact either positively or negatively on the outcome. Healing progresses in a series of overlapping phases.

There are four phases of normal wound healing:

1. **Vascular Response** (Hemostasis)

- ❑ Vasoconstriction – within seconds, regardless of the source of injury, blood vessels constrict to stop bleeding and reduce exposure to bacteria
- ❑ Platelets cluster together at the site of injury to form a ‘clot’
- ❑ A clot is formed by the conversion of thrombin to fibrinogen to *fibrin*
- ❑ Wound healing begins within minutes after tissue damage
(Black & Matassarini-Jacobs, 1997; Silver, 1994)

2. **Inflammatory Response** (Inflammation)

- ❑ This is the body’s early defense system against microbial invasion
- ❑ Neutrophils are the first and most numerous white blood cells to arrive at the injured site. Their role, along with macrophages, is to ingest injurious agents, thereby protecting against bacterial invasion
- ❑ Monocytes and macrophages are next on the scene (usually about 4 days) Monocytes can phagocytose foreign material. The macrophages are critical cells in wound healing because they secrete angiogenesis factor (AGF). AGF stimulates the formation of new blood vessels. Wound healing is significantly impaired without macrophages
- ❑ Leukocytes and macrophages serve as phagocytes that recognize foreign protein or damaged tissue, bind to it, engulf it and destroy it
- ❑ Cell membranes are disrupted by the release of chemicals, resulting in edema
- ❑ There are other mediators of inflammation – the inflammatory response, mast cells, the kinin system, free radicals and the complement system
- ❑ Disorders that lead to reduced numbers of phagocytic cells slow the inflammatory process and make the person more prone to infection
(Black, Matassarini-Jacobs, 1997)

3. **The Proliferative Phase** (Granulation, Epithelialization) (The Active Growth Phase)

This phase contains overlapping of collagen deposits, angiogenesis, granulation, tissue development and wound contraction.

- ❑ Collagen is secreted reconstructing connective tissue. Vitamin C, zinc, oxygen and iron are required for this process
- ❑ Granulation occurs. Collagen, capillaries and cells begin to fill the wound space with new connective tissue. Granulation tissue is red and bumpy, with a meaty appearance
- ❑ The wound contracts as newly formed granulation tissue pulls wound margins inward; this is caused by the action of myofibroblasts
- ❑ Epithelialization occurs as epithelial cells migrate from surrounding skin. This tissue is very fragile
- ❑ Skin re-growth occurs
- ❑ The cells eventually begin to differentiate into various layers of the *epidermis*
- ❑ Epithelialization can be hastened if a wound is kept moist
- ❑ The initial scar is bright red, thick and blanches with pressure (Black & Matassarini-Jacobs, 1997; Silver, 1994)

4. **Maturation Phase** (Reconstruction phase)

- ❑ Remodeling of the scar continues for approximately 1 year
- ❑ Scar tissue regains about two thirds of its original strength
- ❑ Scar tissue is never as strong as the original tissue it replaces (Black & Matassarini-Jacobs, 1997; Silver, 1994)¹

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