Proper Care of Early Wounds to Optimize Healing and Prevent Complications

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KEYWORDS

• Wounds • Wound complications • Wound healing
• Contaminated wounds • Avulsed wounds

The proverb states, "time heals all wounds" and, fortunately, through the resilience of the human body, this often holds true. As medicine advances, so has understanding of the mechanisms of wound healing. Elucidation of the healing process has permitted better opportunities to promote healing while minimizing scar formation. We possess a gross understanding of the key cells and factors that manipulate healing; however, the ability to translate this knowledge into clinical use is still lacking and limits the potential to completely control and enhance the process.

Surgery inherently implies the creation of a wound and, consequently, all wounds create scar. Therefore, an important and unspoken goal in surgery is to accomplish the procedure while minimizing scar formation. Proper surgical techniques for wound closure have long been described, and through continued affirmation, these concepts have evolved into tenets rather than mere recommendations.

Wound healing is important in a wide range of scenarios, whether a patient is healing from a chronic open ulcer or healing from a planned scar revision. The ability to optimize the early care of wounds can greatly aid in the expediency of a wound healing, as well as in the final aesthetic appearance. Although there is no absolute way to care for a wound, this article describes key points that can aid in promoting improved healing while preventing future complications, such as the development of a chronic wound, a hypertrophic scar, or a visibly undesirable scar.

WOUND HEALING PHASES

Understanding the basic stages of wound healing is imperative to best regulate the process. The physiology of wound healing involves multiple phases over time and can be organized into the following phases (**Fig. 1**):

- Inflammatory
- Proliferative
- Remodeling/maturation.

Inflammatory Phase

During the initial injury, hemostasis is of primary importance, and immediate vasoconstriction occurs. Vasoconstriction is mediated by thromboxane A_2 and lasts for 5 to 10 minutes.[1] Endothelial cell injury and exposure of collagen, fibronectin, and laminin lead to activation of the coagulation and complement cascades, which initiate the formation of a clot consisting of fibrin and aggregated platelets.[2] Activated platelets release prostaglandins and vasoactive materials, such as serotonin, histamine, proteases, and thromboxane, which go on to activate their target cells (**Box 1**).

After initial vasoconstriction, active vasodilation occurs, likely secondary to histamine release from mast cells and circulating serotonin. Subsequently,

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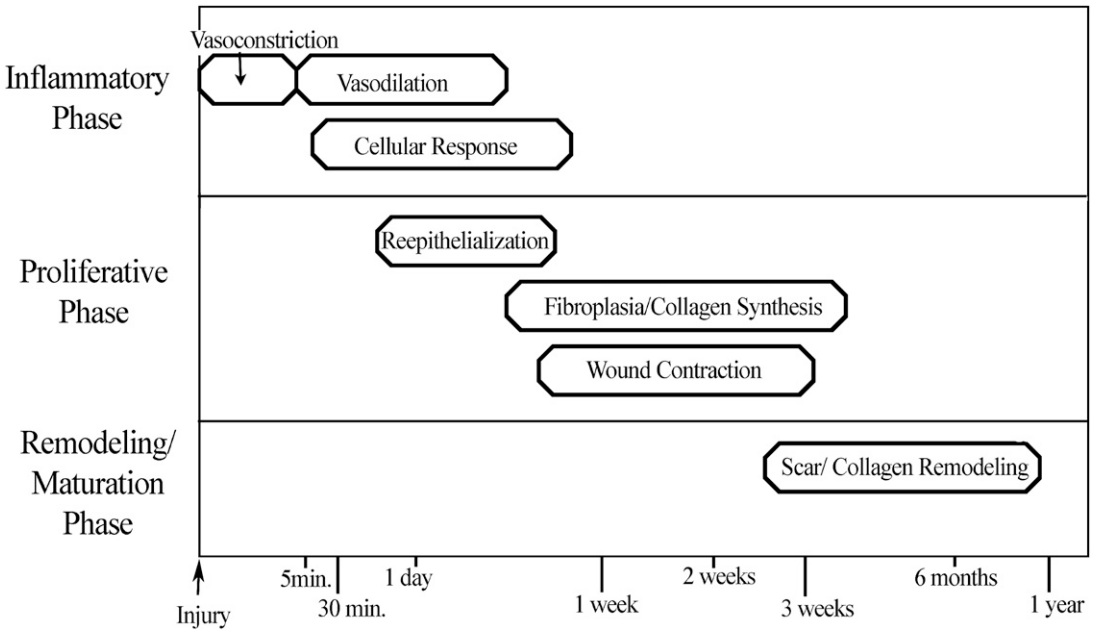

Fig. 1. Phases of wound healing. (*Adapted from* Fisher E, Frodel JL. Wound healing. In: Papel ID, editor. Facial plastic and reconstructive surgery. 3rd edition. New York: Thieme; 2009; with permission.)

kallikrein is activated, leading to kinin activation and endothelial cell separation, which then allows increased vascular permeability that continues for the first 48 to 72 hours.[3]

The cellular response in the inflammatory phase lags somewhat behind the vascular changes, and it begins as fibronectin promotes the migration of neutrophils, monocytes, fibroblasts, and endothelial cells into the region of injury.[2] Fibronectin forms cross-links with clot, which epithelial cells and fibroblasts use as a temporary matrix to proliferate in the wound.[4] Polymorphonuclear leukocytes (granulocytes) and monocytes are among the first cells to appear after an injury. Stimulated by chemotactic factors, granulocytes appear within 6 hours of an insult, and act to clean the wound by phagocytic removal of bacteria and debris. In a noncontaminated wound, the presence of granulocytes is generally short-lived; however, in a contaminated wound, granulocytes can persist and prolong the inflammatory phase. Lengthened periods of inflammation may account for worsened scarring.[2]

Macrophages are essential for wound healing by providing a critical regulatory function in the inflammatory phase and by transitioning a wound into a stage of repair. Attracted by platelet-derived growth factor (PDGF), macrophages are the predominant cell type in a wound by 48 to 96 hours.[1] Macrophages release chemotactic and growth factors, such as transforming growth factor β (TGF-β), basic fibroblast growth factor (FGF), epidermal growth factor, transforming growth factor-α (TGF-α), and PDGF, that result in endothelial and fibroblast proliferation[2] (**Table 1**). If macrophage function is diminished, granulation tissue formation, fibroplasia, collagen production, and, subsequently, overall wound healing, are decreased.[5] The immune response is also closely linked to wound repair, because lymphocytes produce important factors, such as TGF-β, interferons, interleukins, and tumor necrosis factor, that interact with macrophages.[6]

As wound inflammation subsides, collagen deposition can begin, resulting in increased wound tensile strength. Wound strength early in the inflammatory phase is minimal and based on fibrin clot and early epithelialization.[7] At the end of the inflammatory phase, approximately 5 to 7 days after injury, a wound has only approximately 10% of its final tensile strength.[2]

Proliferative Phase

The proliferative phase is the next major step in wound healing, and it is characterized by:

- Re-epithelialization
- Neovascularization
- Collagen deposition
- Wound contraction.

Re-epithelialization begins within 24 hours of injury as epithelial cells from wound margins or deeper adnexal structures, such as hair follicles or sebaceous glands, migrate into the wound to re-establish a protective barrier over underlying