Enhancement of Facial Scars With Dermabrasion

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KEYWORDS

• Dermabrasion • Wound healing • Facial scars
• Facial trauma

Dermabrasion is a well-established method of skin resurfacing, used for both facial rejuvenation and scar revision. The earliest known use of dermabrasion dates back to Egypt, 1500 BC, when sandpaper was used to revise scars.[1] Many forms of skin resurfacing are available, including chemical peels, laser resurfacing, and mechanical resurfacing, known as *dermabrasion*. Dermabrasion results in the removal of the epidermis along with partial removal of the dermis. Dermabrasion devices consist of a powered hand piece and either a wire brush or diamond fraise. Although not viewed as "high tech" or glamorous, dermabrasion continues to be a popular adjunct to scar revision, with many benefits over other resurfacing options. As with any procedure, technical proficiency, experience, and understanding of its applications and limitations are paramount.

PHYSIOLOGY OF WOUND HEALING

Understanding the physiology of wound healing is important when considering any skin resurfacing procedure. Wound healing occurs in progressive phases: the inflammatory phase, the proliferative phase, and the maturation phase; with significant overlap between the inflammatory and proliferative phases (**Fig. 1**).

Inflammatory Phase

During the inflammatory phase, injury to endothelial cells results in exposure of subendothelial collagen, which acts as a binding surface for aggregation of platelets and results in their activation. The extrinsic and intrinsic coagulation cascades then occur, ultimately resulting in the activation of thrombin, which converts fibrinogen to fibrin. Fibrin then acts as the substrate for further platelet aggregation, migration of inflammatory cells, and plasma proteins. The inflammatory phase initially involves a period of vasoconstriction, mediated by epinephrine, norepinephrine, prostaglandins, serotonin, and thromboxane. This stage is followed by vasodilation, which is activated by histamines, prostaglandins, kinins, and leukotrienes. Macrophages function in phagocytosis and also release chemotactic and growth factors, including transforming growth factor (TGF)-β, basic fibroblast growth factor, epidermal growth factor, TGF-α, and plate let-derived growth factor, which is critical in endothelial cell and fibroblast proliferation.[2–4]

Proliferative Phase

The proliferative phase begins within 24 hours of injury and has significant overlap with the inflammatory phase. Epithelial regeneration, fibroplasia, collagen formation, wound contraction, and neovascularization all occur during the proliferative phase. Epithelial regeneration begins within 24 hours of injury and is at its peak between 48 and 72 hours.[5] Reepithelialization occurs as a result of the migration of epithelial cells from the wound margins and from within adnexal

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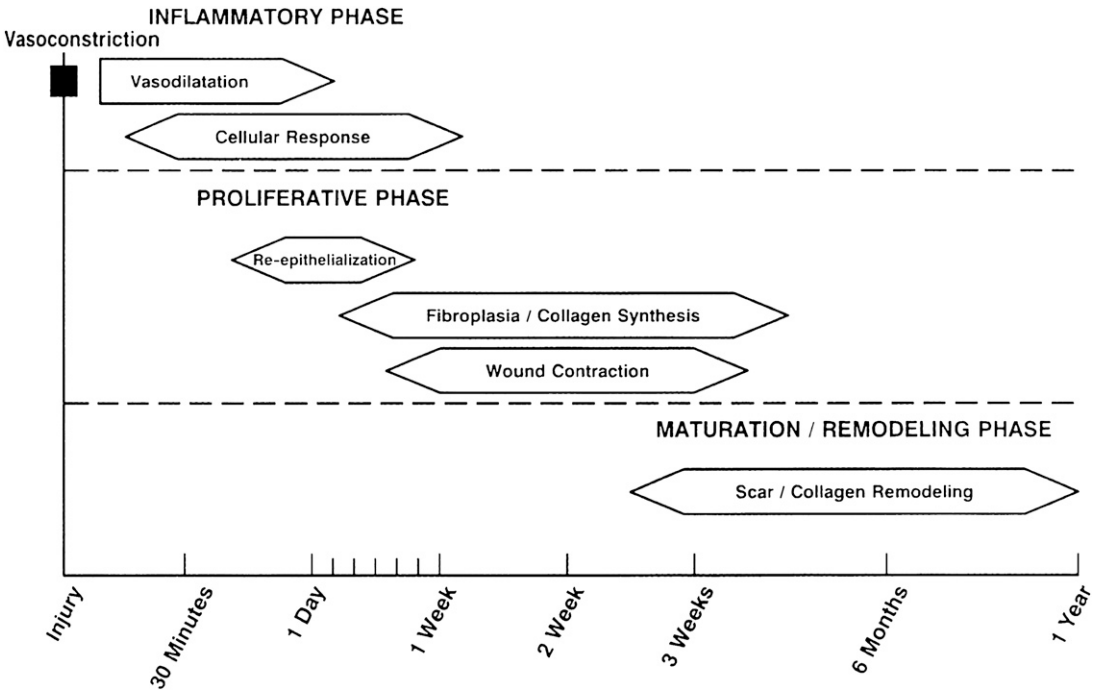

Fig. 1. Phases of wound healing. (*From* Fisher E, Frodel J. Wound healing. In: Papel I, editor. Facial plastic and reconstructive surgery. New York: Thieme; 2009. p. 17; with permission.)

structures of the skin, which include sweat glands, hair follicles, and sebaceous glands.[2,6] Basal stem cells within the adnexae undergo differentiation and subsequent migration, which is why it is paramount to dermabrade only down as far as the superficial reticular dermis, otherwise epithelialization will be impaired and scarring may result (**Fig. 2**).[5] Apposition of advancing epithelial cells results in inhibition of further migration, and in stratification and differentiation.

Fibroplasia, which is the growth of fibroblasts within the wound, occurs 48 to 72 hours after injury and is associated with a significant increase in collagen synthesis, which is most prominent 4 days after injury.[5,7] In addition to collagen, fibroblasts also secrete elastin, fibronectin, glycosaminoglycans, and collagenase. Granulation tissue and neovascularization are also noted during the proliferative phase. Granulation tissue forms during the reepithelialization process, usually beginning at day 3 or 4, and continues until reepithelialization is complete. Wound contraction occurs as fibroblasts differentiate into myofibroblasts, reaching its maximum around 10 to 15 days after injury.[5]

Maturation Phase

The maturation or remodeling phase begins approximately 3 weeks after injury. During this phase, type III collagen is replaced by type I collagen, with reorientation of collagen fibers parallel to the scar, and regression of neovascularization. Scar tensile strength is ultimately 70% to 80% of that encountered in nonwounded skin.[8,9] The maturation phase can take up to 18 months after injury.

Yarborough[10] proposed that dermabrasion created a reorientation of collagen fibers parallel to the lines of wound tension, which may account for some of the scar contour smoothing effects noted after the procedure. Harmon and colleagues[11] performed ultrastructural evaluation of scars resulting from excision and primary closure of cutaneous malignancies in patients who underwent primary closure and those who underwent primary closure with dermabrasion. Serial punch biopsies over the course of 6 weeks showed organized unidirectional collagen fiber orientation parallel to the epidermal surface in the dermabrasion specimens, whereas the control specimens were found to have more-sparse and less-well-organized collagen fiber orientation.

PREOPERATIVE CONSIDERATIONS

Surgeons should obtain a complete past medical history, including a current list of all medications, especially anticoagulants. Patients taking anticoagulants should discontinue these 2 weeks before dermabrasion if medically feasible. Acne scarring is