



Improving scars with minimally invasive and topical treatment

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Suboptimal facial scarring can be a stigma, leading many affected patients to seek treatment. An increasing array of minimally invasive techniques and topical treatments can improve many scars either as standalone interventions or as an adjunct to surgical scar revision. Familiarity with the science of normal wound healing, competence in minimally invasive techniques, and an evidence-based approach to using the wide array of available medical therapies will assist the head and neck surgeon in obtaining the best facial scars possible.

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Unsightly scarring following traumatic injuries, prior surgery, or abnormal wound healing is unacceptable to patients when it occurs on the face or neck. Unlike scars on other parts of the body, a facial scar can be a stigma, more often leading an affected patient to seek treatment. Surgical excision for camouflage and/or reorientation remains the gold standard for treatment of many scars. However, less invasive treatments can frequently offer improvement or serve as an adjunct following surgical treatment. Familiarity with the science of normal wound healing, competence in minimally invasive techniques, and an evidence-based approach to using the wide array of available medical therapies will assist the head and neck surgeon in obtaining the best scars possible. Some modalities are useful for mature scars and abnormal scar formation. Other treatments are more effective when used during earlier stages of wound healing; intervention must take place immediately after surgical scar revision or in the early postoperative period following primary reconstructive and even ablative procedures. Finally, there is a long list of widely known and

heavily marketed scar therapies that are backed by few documented benefits in the literature. Herein we discuss the merit of and technique for using dermabrasion, laser resurfacing, soft tissue fillers, pressure garments, massage, and radiation therapy for undesirable or abnormal scarring. Other treatments, including silicone sheeting, steroids, vitamin E, and quercetin, are addressed as well.

Normal wound healing

It is crucial that the head and neck surgeon be familiar with normal wound healing physiology to effectively intervene and improve scars. A comprehensive discussion of this broad topic is beyond the scope of the article. We instead present a focused review of fundamental primary wound healing with an emphasis on stages where aberration leads to undesirable scar formation and points where therapeutic intervention may be possible.

Wound healing begins immediately following any interruption of the epidermis whether traumatic or iatrogenic. The 4 basic stages and timeline of normal wound healing are summarized in [Figure 1](#). Coagulation is the first stage, typically lasting less than 30 minutes. Vasoconstriction, platelets, and the coagulation cascade form a hemostatic

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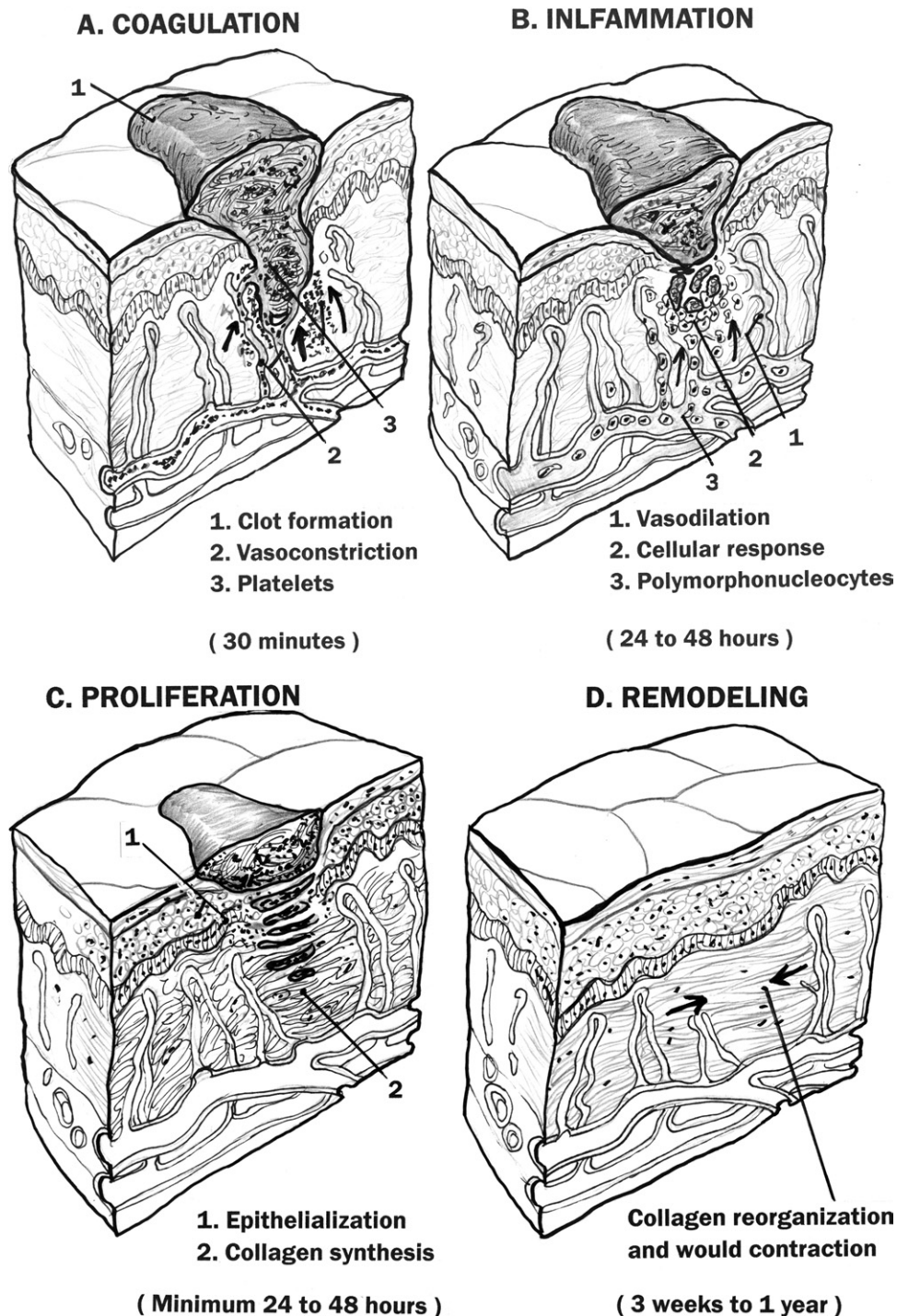


Figure 1 Coagulation is the first stage of wound healing (A). The endpoint of this stage is hemostasis and formation of a fibrin matrix. The second stage, inflammation, begins concurrently (B). Vasodilation, increased vascular permeability, and a cellular response mediated by polymorphonucleocytes and monocytes are hallmarks of the second stage. Early in the inflammatory stage, the third stage, or proliferation, begins simultaneously (C). Epithelial cells, collagen, and blood vessels begin propagating from the wound edges and continue until epithelialization is complete. As early as 3 weeks, the final, or remodeling, stage begins (D). Centripetal wound contraction occurs and wound tensile strength progressively increases.

plug and then fibrin matrix that will ultimately act as a scaffold during wound healing. Once hemostasis is achieved, the wound enters the second stage, or inflammation, with concomitant vasodilation, increased vascular permeability, and a cellular response. The initial cellular response consists of polymorphonucleocytes, ridding the

wound of most bacteria and other foreign debris. These polymorphonucleocytes are replaced after 24-72 hours by monocytes that continue the wound debridement and release essential cytokines. The inflammatory stage, accompanied by its classic signs of *calor*, *rubor*, *dolor*, and *tumor* (heat, erythema, tenderness, and swelling) lasts for up to a week or

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