Late Revision or Correction of Facial Trauma–Related Soft-Tissue Deformities

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KEYWORDS

• Facial trauma • Maxillofacial surgery • Soft-tissue injury • Wound healing • Treatment

KEY POINTS

- Facial soft-tissue deformities can arise that require specific evaluation and management for correction.
- The contemporary maxillofacial surgeon has continued to expand on the historical management of facial fractures established by pioneers in the specialty.
- Advances in understanding the biological aspects of wound healing and surgical options to rectify acute and delayed facial soft-tissue deformities, combined with current technology, facilitate improved outcomes.

INTRODUCTION

Facial trauma is the foundation underlying contemporary oral and maxillofacial surgery. We have a long history of managing these patients and providing excellent care for their complex injuries. As specialists we owe a great deal to those who pioneered various treatments for facial fracture repair and paved the way for oral and maxillofacial surgeons to further expand their scope to include all aspects of facial trauma. The surgical approaches used in accessing the facial skeleton for fracture repair are often the same as or similar to those used for cosmetic enhancement of the face, an aspect that has contributed to the expansion of cosmetic procedures performed by maxillofacial surgeons. Rarely does facial trauma result in injuries that do not in some way affect the facial soft-tissue envelope either directly or as sequelae of the surgical repair. Knowledge of both skeletal and facial soft-tissue anatomy is paramount to successful clinical outcomes. Facial soft-tissue deformities can arise that require specific evaluation and management for correction. This article focuses on revision and correction of these soft-tissue–related injuries secondary to facial trauma.

PHASES OF WOUND HEALING

A basic knowledge of the principles of wound healing is critical for surgeons to understand when managing soft-tissue injuries resulting from trauma. An awareness of the intricacies of wound healing enables the surgeon to endeavor to minimize complications that might occur during this process, and helps ensure the best possible postoperative outcome. Wound healing is an extremely

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complex process of overlapping phases whereby numerous cell types are responsible for executing a multitude of cellular functions, and can be organized into the 4 following phases:

- Immediate response
- Inflammatory phase
- Proliferative phase
- Remodeling/maturation

Prolongation of any one of these phases can result in delayed or compromised healing of a wound and can eventually lead to suboptimal outcomes from a clinical standpoint.

Immediate Response

The immediate response to an injury in which there is a violation of tissue integrity is a rapid alteration in cell-signaling pathways that modify cellular gene expression, metabolism, and cell survival.[1] Pathways leading to hemostasis are triggered and lead to local vasoconstriction of damaged blood vessels, platelet activation, platelet aggregation, and formation of a provisional fibrin matrix that also serves as a medium through which cells are recruited to participate in wound repair.[2] Activated platelets are also responsible for the secretion of multiple cytokines, growth factors, and chemotactic agents needed for progression of the healing process, such as vascular endothelial growth factor (VEGF), platelet-derived growth factor (PDGF), fibroblast growth factor, transforming growth factor β (TGF-β), CXCL4, and RANTES.[1]

Inflammatory Phase

The inflammatory phase is typified by the passive ingress of circulating leukocytes (predominantly neutrophils) from damaged blood vessels coupled with activation of immune cells present within the damaged tissues (mast cells, T cells, and Langerhans cells).[1] This ingress of phagocytic cells represents the transition of the wound into a state of active repair, and is facilitated by an increase in vascular permeability secondary to locally released factors such as nitric oxide (NO), mast cell–derived histamine, and tissue plasminogen activator.[1] Neutrophils are recruited to the site of injury to cleanse the wound through phagocytosis of invading microorganisms and removal of cellular debris. Monocytes are slowly recruited to the site of injury, and will peak in numbers approximately 24 hours after the initial injury.[1] Monocytes further the efforts set forth by the neutrophils, and will clear additional cellular debris along with nonviable neutrophils in preparation for the next phase of wound healing. In noncontaminated wounds the presence of neutrophils and monocytes is relatively short lived; however, in contaminated wounds the presence of phagocytic cells can persist, leading to a prolongation of the inflammatory phase and compromised outcomes in terms of wound healing and scar formation.

Proliferative Phase

The second phase of wound healing is the proliferative phase, during which the processes of reepithelialization, angiogenesis, collagen deposition, and wound contraction predominate. Reepithelialization will occur through the migration and proliferation of epithelial stem cells present at the wound margins and in deeper adnexal structures such as the hair-follicle bulge.[2] The provisional fibrin matrix that was deposited during the inflammatory phase will be replaced with granulation tissue within about 72 hours following initial injury, and will persist for approximately 14 days.[2] Fibroblast proliferation in response to TGF-β, PDGF, and fibroblast growth factor leads to synthesis of extracellular matrix components including glycosaminoglycans, proteoglycans, and collagen (predominantly type III).[2] Angiogenesis leads to extension of capillaries from the wound edge into the zone of injury.[3] This process is critical for providing the oxygen and nutrients necessary for continuation of healing. Collagen deposition will increase wound strength, while fibroblasts in close proximity to the wound edges will begin producing weakly contractile actin bundles to facilitate contraction of the wound.[1] The proliferative phase culminates with restoration of tissue integrity via newly formed epithelial barrier and reapproximation of deeper layers through the process of wound contracture and synthesis of new extracellular matrix: essentially, formation of a scar.

Remodeling and Maturation

The final phase of wound healing is the longest of the 3 phases, and can take anywhere from several months to several years depending on the patient, the integrity of their immune system, and overall systemic health. This phase is essential for modification of tissue integrity and the normalization of tissue appearance. The hallmark of this phase is the reorganization of the collagenous matrix created during the proliferative phase, and involves the sequential replacement of type III collagen fibrils with stronger and more robust type I collagen. Larger collagen bundles with a higher proportion of intermolecular cross-linking are created,[2] and the microvascular network within the scar is also revised to become a more functional network of blood vessels.[1] The extracellular matrix that was formed in the dermis will