Michael Miloro · G. E. Ghali ·
Peter E. Larsen · Peter Waite *Editors*

Peterson's Principles of Oral and Maxillofacial Surgery

Fourth Edition

Springer

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To Beth and Macy, and my family, for their love and support, and my parents for always being there for me. To my students and residents of oral and maxillofacial surgery for teaching me and making me realize every day that I made the right career choice. To Pete Peterson, who I think about quite often even though it has been 20 years since his death; thank you for being such an excellent mentor – I would not be here if you had not come into my life in 1994.

Michael Miloro

This book is dedicated to my wife, Hope, and our wonderful children, Gregor, Gracie, Gabrielle, and Garrisyn. Thanks for always reminding me of the important things in life. To my students, residents, fellows, and staff, who always keep it relevant and enjoyable for me.

G. E. Ghali

To my wife Patty, and my sons, Michael, Matthew, and Mark, for reminding me that my most important role is that of father and husband. To my father, who inspired me to enter medicine, and to my mother, who convinced me that I could accomplish whatever I put my mind to. To my residents, many of whom have become close friends. I am proud of the fine surgeons they have become. Lastly, to the many faculties with whom I have had the great privilege to work with throughout my career.

Peter E. Larsen

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Peter Waite

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Preface

Nearly 30 years ago in 1992, Dr. Larry J. "Pete" Peterson published the first edition of *Principles of Oral and Maxillofacial Surgery* when he was Professor and Chairman of Oral and Maxillofacial Surgery at The Ohio State University in Columbus, Ohio. In his preface for that book, he recognized that "(t)he specialty of Oral and Maxillofacial Surgery has advanced dramatically over the last 15 years [1977–1992]." Over the next 10 years (1992–2002), Pete certainly experienced some of the advancements in our specialty until his premature death in 2002. Yet, I wonder how he would see the subsequent expansion of the scope of the specialty, as well as the significant and explosive technological advances that have occurred over the past 20 years and have shaped the manner in which we practice medicine, dentistry, and specialty care today. I am certain that he would have had restrained enthusiasm, and would have reserved judgment until the published studies had validated the efficacy of these advances in improving patient care while maintaining a favorable cost-to-benefit ratio; Pete was never one to "jump on the bandwagon," and evidence-based practice was mandatory. Dr. Peterson also recognized "…that much of the surgery that the individual practitioner is called on to do today was learned *after* formal residency training." Most surgeons would agree, and this basic premise served as his impetus to have a complete and comprehensive reference textbook for the practicing surgeon. Dr. Peterson's intention was that the first edition "…emphasizes new and innovative methods and techniques," and it certainly accomplished that goal within the constraints of the technological limitations of the time.

With similar themes in mind from 1992, in 2021 we have created the fourth edition of *Peterson's Principles of Oral and Maxillofacial Surgery* to reflect the significant changes in clinical practice, and contain contemporary, state-of-the-art knowledge and clinical techniques for the oral and maxillofacial surgeon, both in training and in practice. We have sought to assemble an excellent authorship composed of both experienced surgeons and young surgeons to collaborate on the specific chapters in order to include both historical and experienced points of view combined with a fresh perspective on each chapter topic. The clear purpose of this fourth edition is to provide an authoritative textbook that is concise, easy to read, contemporarily referenced, and that contains the requisite information that the competent oral and maxillofacial surgeon should possess. Throughout the prior three editions, this textbook has been used as a *required* resource for residency training programs in the USA and abroad, and it has served well those residents in training and candidates for board certification well as a resource for didactic conference preparation, clinical and operating room preparation, and examination preparation.

Unfortunately, many who will benefit from this fourth edition, and future editions, would have never met Dr. Peterson, but those who knew Pete Peterson as the *teacher* would know how important it would have been for him to see his *teachings* continue through this textbook.

Peterson's Principles of Oral and Maxillofacial Surgery is, without a doubt, *the* authoritative textbook for the specialty of oral and maxillofacial Surgery.

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Medicine, Surgery, and Anesthesia

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Wound Healing

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Learning Aims

1. Wound healing restores tissue integrity and function through a coordinated series of cellular events.
2. Healing continuum consists of overlapping inflammatory, proliferative, and remodeling phases.
3. Quality of healing depends on wound and tissue type.
4. Multiple local and systemic factors can cause wound healing complications.
5. Healing may be modulated spatiotemporally with growth factors, gene therapy, and biologic scaffolds.

1.1 Introduction

The healing wound is an overt expression of an intricate and tightly choreographed sequence of cellular and biochemical responses directed toward restoring tissue integrity and functional capacity following injury. Although healing in the orofacial region culminates uneventfully in most instances, a variety of intrinsic and extrinsic factors can hamper or facilitate the process. Understanding wound healing at multiple levels – biochemical, physiologic, cellular, and molecular – provides the surgeon with a framework for basing clinical decisions aimed at optimizing the healing response. Equally important, it allows the surgeon to critically evaluate and selectively use the growing collection of biologic approaches that seek to assist healing by favorably modulating the wound microenvironment.

1.2 The Healing Process

The restoration of tissue integrity, whether initiated by trauma or surgery, is a phylogenetically primitive but essential defense response. Injured organisms survive only if they can repair themselves quickly and effectively. The healing response depends primarily on the type of tissue involved and the nature of the tissue disruption. When restitution occurs through tissue that is structurally and functionally indistinguishable from native tissue, *regeneration* has taken place. However, if tissue integrity is reestablished primarily through the formation of scar tissue, then *repair* has occurred. Repair by scarring is the body's version of a spot weld and the replacement tissue is coarse and has lower cellular content than native tissue. Except for bone and liver, tissue disruption invariably results in repair rather than regeneration.

At the cellular level, the rate and quality of tissue healing depend on whether the constitutive cells are labile, stable, or permanent. Labile cells, including the keratinocytes of the epidermis and epithelial cells of the oral mucosa, divide throughout their life span. Stable cells such as fibroblasts exhibit a low rate of duplication but can undergo rapid proliferation in response to injury. For example, bone injury causes pluripotent mesenchymal cells to speedily differentiate into osteoblasts and osteoclasts. On the other hand, permanent cells such as specialized nerve cells do not divide in postnatal life. The surgeon's expectation of "normal healing" should be correspondingly realistic and based on the inherent capabilities of the injured tissue. Whereas a fibrous scar is normal for skin wounds, it is suboptimal in the context of bone healing.

At a more macro level, the quality of the healing response is influenced by the nature of the tissue disruption and the circumstances surrounding wound closure. Healing by first intention occurs when a clean laceration or surgical incision is closed primarily with sutures or other means and healing proceeds rapidly with no dehiscence and minimal scar formation. If conditions are less favorable, wound healing is more complicated and occurs through a protracted filling of the tissue defect with granulation and connective tissue. This process is called healing by second intention and is commonly associated with avulsion injury, local infection, or inadequate closure of the wound. For more complex wounds, the surgeon may attempt healing by third intention through a staged procedure that combines secondary healing with delayed primary closure. The avulsion or contaminated wound is debrided and allowed to granulate and heal by second intention for 5–7 days. Once adequate granulation tissue has formed and the risk of infection appears minimal, the wound is sutured close to heal by first intention.

1.3 Wound Healing Response

Injury of any kind sets into motion a complex series of synchronized and temporally overlapping processes directed toward restoring the integrity of the involved tissue. Reparative processes are most commonly modeled in skin [1]; however, similar patterns of biochemical and cellular events occur in virtually every other tissue [2]. To facilitate description, the healing continuum of coagulation, inflammation, reepithelialization, granulation tissue, and matrix and tissue remodeling is typically broken down into three distinct overlapping phases: inflammatory, proliferative, and remodeling [3, 4].

1.3.1 Inflammatory Phase

The inflammatory phase presages the body's reparative response and usually lasts for 3–5 days. Vasoconstriction of the injured vasculature is the spontaneous tissue reac-

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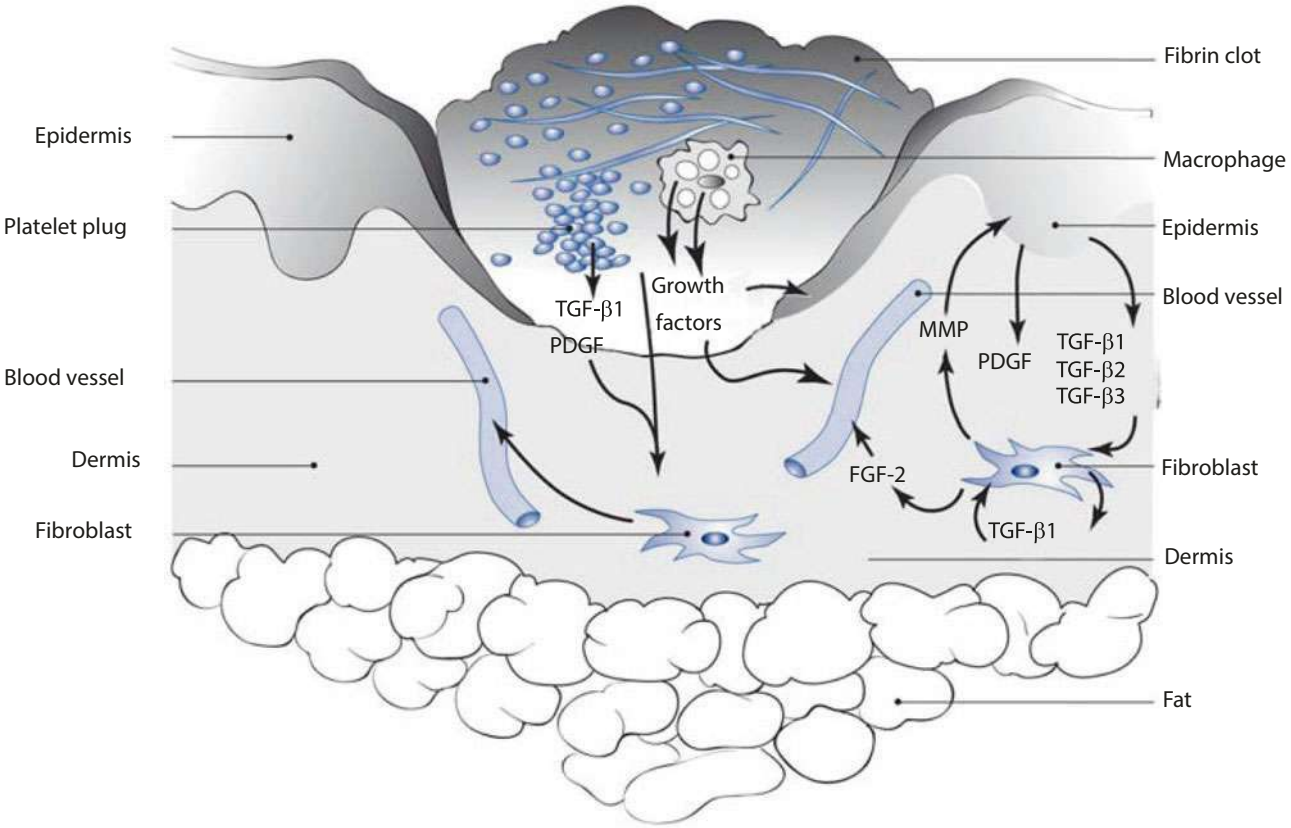

Fig. 1.1 Immediately following wounding, platelets facilitate the formation of a blood clot that secures hemostasis and provides a temporary matrix for cell migration. Cytokines released by activated macrophages and fibroblasts initiate the formation of granulation tissue by degrading extracellular matrix and promoting development of new blood vessels. Cellular interactions are potentiated by reciprocal signaling between the epidermis and dermal fibroblasts through growth factors, MMPs, and members of the TGF-β family. FGF fibroblast growth factor, MMP matrix metalloproteinase, PDGF platelet-derived growth factor, TGF-β transforming growth factor beta. (Adapted with permission from Bissell MJ and Radisky D[70])

tion to stop bleeding. Tissue trauma and local bleeding activate factor XII (Hageman factor), which initiates the various effectors of the healing cascade including the complement, plasminogen, kinin, and clotting systems. Circulating platelets (thrombocytes) rapidly aggregate at the injury site and adhere to each other and the exposed vascular subendothelial collagen to form a primary platelet plug organized within a fibrin matrix. The clot secures hemostasis and provides a provisional matrix through which cells can migrate during the repair process. Additionally, the clot serves as a reservoir of cytokines and growth factors that are released as activated platelets degranulate (Fig. 1.1). The bolus of secreted proteins, including interleukins, transforming growth factor β (TGF-β), platelet-derived growth factor (PDGF), and vascular endothelial growth factor (VEGF), maintains the wound milieu and regulates subsequent healing.

Once hemostasis is secured, the reactive vasoconstriction is replaced by a more persistent period of vasodilation that is mediated by histamine, prostaglandins, kinins, and leukotrienes. Increasing vascular permeability allows blood plasma, leucocytes, and other cellular mediators of healing to pass through the vessel walls (diapedesis) and populate the extravascular space. Parallel clinical manifestations include swelling, redness, heat, and pain. Cytokines released into the wound provide the chemotactic cues that sequentially recruit the neutrophils and monocytes to the site of injury. Neutrophils normally begin arriving at the wound site within minutes of injury and rapidly establish themselves as the predominant cells. Migrating through the scaffolding provided by the fibrin-enriched clot, the short-lived neutrophils flood the site with proteases and cytokines that help cleanse the wound of contaminating bacteria, devitalized tissue, and degraded matrix components. Neutrophil activity is accentuated by opsonic antibodies leaking into the wound from the altered vasculature. Unless a wound is grossly infected, neutrophil infiltration ceases after a few days. However, the proinflammatory cytokines released by perishing neutrophils, including tumor necrosis factor α (TNF-α) and interleu-

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kins (IL-1a, IL-1b), continue to stimulate the inflammatory response for extended periods [5].

The initial levels of neutrophils begin to taper over the next 24–72 h with an increasing deployment of blood-borne monocytes to the site of injury. Activated monocytes, now termed macrophages, continue with the process of wound microdebridement initiated by the neutrophils. The macrophages secrete collagenases and elastases to break down injured tissue and phagocytose bacteria and cell debris. Beyond their scavenging role, the macrophages also serve as the primary source of healing mediators. Once activated, macrophages release a battery of growth factors and cytokines (TGF-α, TGF-β1, PDGF, insulin-like growth factor [IGF]-I and -II, TNF-α, and IL-1) at the wound site, further amplifying and perpetuating the action of the chemical and cellular mediators released previously by degranulating platelets and neutrophils. Macrophages influence all phases of early wound healing by regulating local tissue remodeling through proteolytic enzymes (e.g., matrix metalloproteases and collagenases), inducing the formation of new extracellular matrix, and modulating angiogenesis and fibroplasia through local production of cytokines such as thrombospondin-1 and IL-1b [6]. The centrality of macrophage function to early wound healing is underscored by the consistent finding that macrophage-depleted animal wounds demonstrate diminished fibroplasia and defective repair. Although the numbers and activity of the macrophages taper off by the fifth post-injury day, they continue to modulate the wound healing process until repair is complete.

1.3.2 Proliferative Phase

The cytokines and growth factors secreted during the inflammatory phase stimulate the succeeding proliferative phase (Fig. 1.2) [7]. Starting as early as day 3 post-injury and lasting up to 3 weeks, the proliferative phase is distinguished by the formation of pink granular tissue (granulation tissue) containing inflammatory cells, fibroblasts, and budding vasculature enclosed in a loose matrix [8]. An essential first step is the establishment of a local microcirculation to supply the oxygen

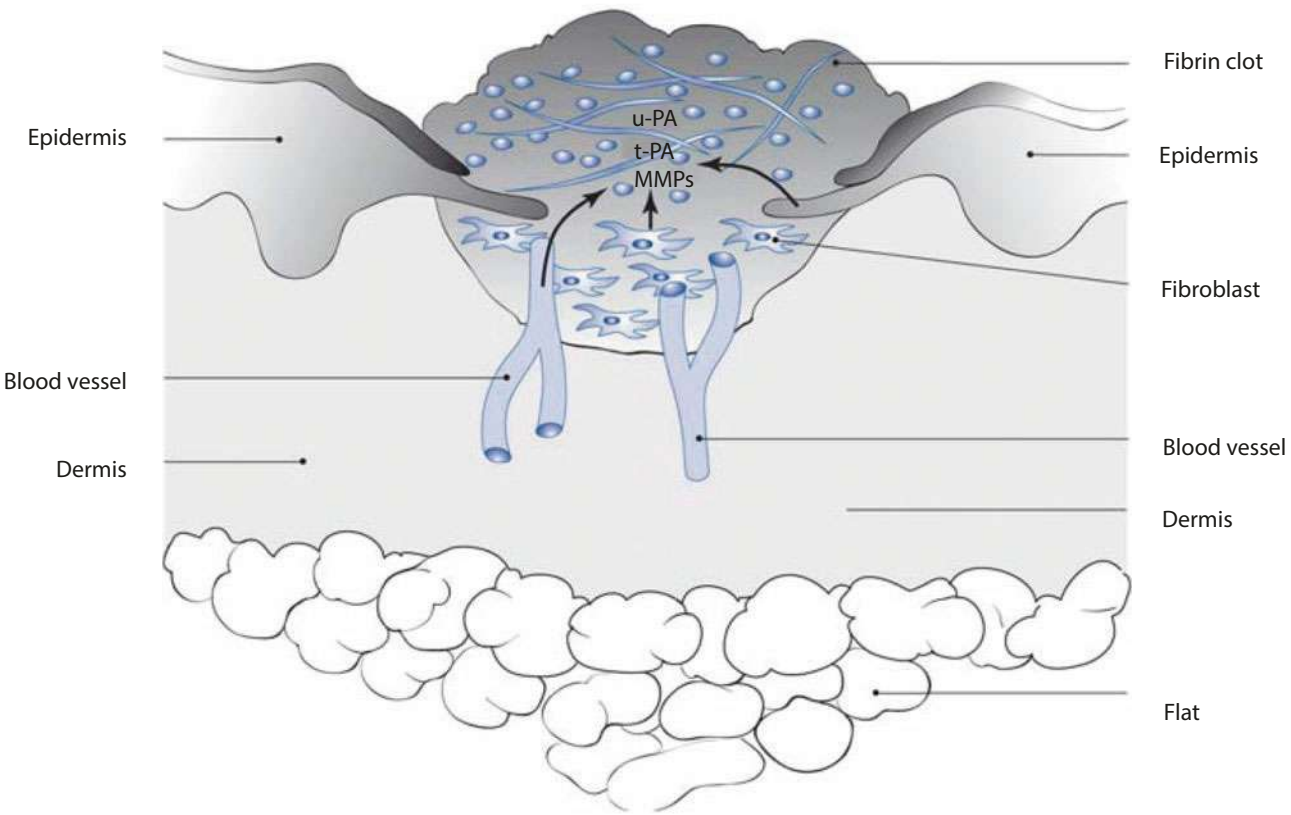

Fig. 1.2 The cytokine cascade mediates the succedent proliferative phase. This phase is distinguished by the establishment of local microcirculation and formation of extracellular matrix and immature collagen. Epidermal cells migrate laterally below the fibrin clot, and granulation tissue begins to organize below the epithelium. MMPs matrix metalloproteinases, t-PA tissue plasminogen activator, u-PA urinary plasminogen activator. (Adapted with permission from Bissell MJ and Radisky D[70])

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