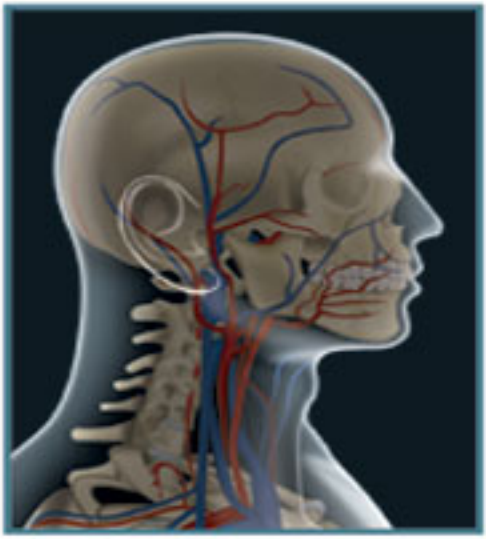
Sataloff's Comprehensive Textbook of

Otolaryngology
Head & Neck Surgery

Facial Plastic and Reconstructive Surgery

Series Editor
Robert T Sataloff



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Vol. 3

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Dedicated to

Matthew, James and Anthony Sclafani, who have not only tolerated but encouraged me in this endeavor, deserve special thanks. Without Peggy Hanrahan, who has given me more love and support than anyone could ever ask, my journey through medicine, otolaryngology, facial plastic surgery and life would not have been possible. It is to these wonderful people that I dedicate this volume.

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Foreword

Sataloff's Comprehensive Textbook of Otolaryngology: Head and Neck Surgery is a component of the most extensive compilation of information in otolaryngology—head and neck surgery to date. The six volumes of the comprehensive textbook are part of a 12-volume, encyclopedic compendium that also includes a six-volume set of detailed, extensively illustrated atlases of otolaryngologic surgical techniques. The vision for the *Comprehensive Textbook* was realized with the invaluable, expert collaboration of eight world-class volume editors. Chapter authors include many of the most prominent otolaryngologists in the world, and coverage of each subspecialty is extensive, detailed and scholarly.

Anil K Lalwani, MD edited the volume on otology/neurotology/skull base surgery. Like all six of the volumes in the *Comprehensive Textbook*, the otology/neurotology/skull base surgery volume is designed not only as part of the multivolume book, but also to stand alone or in combination with the atlas of otological surgery. Dr Lalwani's volume covers anatomy and physiology of hearing and balance, temporal bone radiology, medical and surgical treatment of common and rare disorders of the ear and related structures, occupational hearing loss, aural rehabilitation, cochlear and brainstem implantation, disorders of the facial nerve, and other topics. Each chapter is not only replete with the latest scientific information, but also accessible and practical for clinicians.

The rhinology/allergy and immunology volume by Marvin P Fried and Abtin Tabaee is the most elegant and inclusive book on the topic to date. Drs Fried and Tabaee start with a history of rhinology beginning in ancient times. The chapters on evolution of the nose and sinuses, embryology, sinonasal anatomy and physiology, and rhinological assessment are exceptional. The volume includes discussions of virtually all sinonasal disorders and allergy, including not only traditional medical and surgical therapy but also complementary and integrative medicine. The information is state-of-the-art.

Anthony P Sclafani's volume on facial plastic and reconstructive surgery is unique in its thoroughness and practicality. The volume covers skin anatomy and physiology, principles of wound healing, physiology of grafts and flaps, lasers in facial plastic surgery, aesthetic analysis of the face and other basic topics. There are extensive discussions on essentially all problems and procedures in facial plastic and reconstructive surgery contributed by many of the most respected experts in the field. The volume includes not only cosmetic and reconstructive surgery, but also information on diagnosis and treatment of facial trauma.

The volume on laryngology edited by Dr Michael S Benninger incorporates the most current information on virtually every aspect of laryngology. The authors constitute a who's who of world experts in voice and swallowing. After extensive and practical discussions of science and genetics, the volume reviews diagnosis and treatment (traditional and complementary) of laryngological disorders. Chapters on laser physics and use, voice therapy, laryngeal dystonia, cough, vocal aging and many other topics provide invaluable "pearls" for clinicians. The volume also includes extensive discussion of surgery for airway disorders, office-based laryngeal surgery, laryngeal transplantation and other topics.

For the volume on head and neck surgery, Drs Patrick J Gullane and David P Goldstein have recruited an extraordinary group of contributors who have compiled the latest information on molecular biology of head and neck cancer, principles of radiation, immunobiology, medical oncology, common and rare head and neck malignancies, endocrine neoplasms, lymphoma, deep neck space infections and other maladies. The surgical discussions are thorough and richly illustrated, and they include definitive discussions of free flap surgery, facial transplantation and other subjects.

Dr Christopher J Hartnick's vision for the volume on pediatric otolaryngology was expansive, elegantly scholarly and invaluable clinically. The volume begins with information on embryology, anatomy, genetics, syndromes and other complex topics. Dr Hartnick's contributors include basic discussions of otolaryngologic examination in a pediatric patient, imaging, hearing screening and aural rehabilitation, and diagnosis and treatment of diseases of the ear, nose, larynx, oral cavity, neck and airway. Congenital, syndromic and acquired disorders are covered in detail, as are special, particularly vexing problems such as chronic cough in pediatric patients, breathing and obstructive sleep apnea in children, pediatric voice disorders, and many other subjects. This volume will be invaluable to any otolaryngologist who treats children.

All of us who have been involved with the creation of the six-volume *Sataloff's Comprehensive Textbook of Otolaryngology: Head and Neck Surgery* and its companion six-volume set of surgical atlases hope and believe that our colleagues will find this new offering to be not only the most extensive and convenient compilation of information in our field, but also the most clinically practical and up-to-date resource in otolaryngology. We are indebted to Mr Jitendar P Vij (Group Chairman) and Mr Ankit Vij (Group President) of M/s Jaypee Brothers Medical Publishers (P) Ltd., New Delhi, India, for their commitment to this project, and for their promise to keep this work available not only online but also in print. We are indebted also to the many otolaryngologists who have contributed to this work not only by editing volumes and writing chapters, but also by asking questions that inspired many of us to seek the answers found on these pages. We also thank especially the great academic otolaryngologists who trained us and inspired us to spend our nights, weekends and vacations writing chapters and books. We hope that our colleagues and their patients find this book useful.

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Preface

Facial Plastic and Reconstructive Surgery is, depending upon one's point of view, either a distinct subspecialty or an integral part of otolaryngology. Techniques of soft tissue and bone repair, central to all aspects of otolaryngology, are of paramount concern in facial plastic surgery. As much as any other part of otolaryngology, facial plastic and reconstructive surgery relates intimately with all other subspecialties of the field. Reconstruction after removal of cancer or facial trauma, correction of congenital defects of the ears, nose, lips and palate, and many rhinoplasty procedures improve the function of facial structures. The sound understanding of head and neck anatomy and physiology is at the core of a successful facial plastic surgeon. Knowledge, techniques and approaches in facial plastic surgery are essential to anyone practicing otolaryngology—head and neck surgery.

This volume of *Sataloff's Comprehensive Textbook of Otolaryngology: Head and Neck Surgery* covers the breadth of facial plastic surgery. Basic principles, anatomy, physiology, and reconstructive and cosmetic topics in facial plastic surgery are well-illustrated and covered in detail. This volume, like the rest of this work, serves the needs of the most junior resident as well as the most seasoned surgeon. It is hoped that the reader will fully appreciate not only the techniques of facial plastic and reconstructive surgery, but also its significance within otolaryngology.

Anthony P Sclafani MD FACS

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The editor would like to thank Joseph Rusko, Marco Ulloa, Carol Rogers Field, Bridget Meyer, Tom Gibbons and the rest of the Jaypee Brothers team. Without their perseverance and hard work, this volume would not have been possible. Special thanks are offered to the authors, who have shared their expertise and experience in order to improve the care of the Facial Plastic and Reconstructive Surgery patient.

I would also like to thank Mr Jitendar P Vij (Group Chairman), Mr Ankit Vij (Group President), Ms Chetna Malhotra Vohra (Associate Director), Mr Umar Rashid (Development Editor) and Production team of Jaypee Brothers Medical Publishers (P) Ltd., New Delhi, India.

Contents

CHAPTER

1

Skin Anatomy and Physiology

Joseph J Rousso, Michael Bassiri-Tehrani

INTRODUCTION

The skin is the largest organ in the human body and often, the most overlooked. The integumentary system serves as the ultimate canvas for the plastic surgeon, and its surface can only be appropriately camouflaged with an intricate knowledge of its form and function. The skin serves an important protective function as the first line barrier to potential harm such as infection or trauma, and its contiguous structure envelopes and largely defines a considerable majority of the human body.

The skin is perhaps the most important biologic tool that the plastic surgeon has to work with; its healing capacity, malleability, mobility, functional adnexa, and complex cellular matrix make flaps as well as grafts available to the surgeon. Although the dermatologist is, indeed, the skin specialist, it is the plastic surgeon's responsibility to have a comprehensive understanding of this organ for both aesthetic and reconstructive purposes. Whether it be aging face procedures or cancer reconstruction, the end result is directly related to the postoperative status and appearance of the visible skin.

The face, more so than anywhere else on the human body, parades the skin and all its features in a manner that is most noticeable to the observer. For example, severe acne is easily identified in the unmasked face, whereas equal levels of acne on the torso or back may not immediately grasp the observer's attention. Detailed knowledge of all layers of the facial skin and associated adnexal structures, as well as an in-depth understanding of its vascular supply is vital and will be discussed in this chapter. In addition, the physiologic role of temperature regulation and how it contributes to integumentary attributes will be described with a brief description of sensation and metabolic function of the skin.

Ideally, skin flaps and grafts would uniformly follow rules and surgical principles to give an aesthetic postoperative result; this is not always the case and it is the knowledge of the skin anatomy and physiology that helps the surgeon adapt accordingly to different skin types. For example, some patients have scalp skin that is more immobile than others. Based on the knowledge of vascular factors and influences, a relatively immobile flap can be improved with the use of adjunctive surgical techniques such as tissue expansion. Tissue expansion is just one example of a technique that requires an intricate knowledge of the layers of the skin and the microphysiologic changes that lead to improved survival.

On a more basic level, the facial surgeon spends a great deal of time elevating and manipulating soft tissue for reasons that include cancer reconstruction, redraping of lax skin, and coverage of soft tissue defects. Each of these may require elevation in a specific plane or layer; the surgeon may want to have a thinner and more immediate subdermal plane of elevation when redraping cutis laxa, whereas a large soft tissue defect may require a deeper and thicker plane of elevation for increased blood supply as well as tissue bulk. Placing these sorts of specific descriptions into practice is easier to perform when they are well conceptualized. A distinctive part of the surgical complexity of the skin is that every layer is not grossly distinctive to the naked eye, and clinical judgment plays a large role in deciphering the road map that is the integument. This chapter emphasizes the anatomy of the

facial skin with a comprehensive outline that is broken down into the skin layers, the physiologic role of the skin, and finally a conclusion that ties this large organ system together.

THE ANATOMIC LAYERS OF THE SKIN

In order of superficial to deep, the skin is composed of the epidermis, dermis, and subcutaneous tissue. The layers of the skin are traditionally described, in dermatologic texts, from deep to superficial. This is intuitive in the sense that the development of the cell structures begin at the basal layers and progress superficially. Nonetheless, this chapter will take an alternative approach and describe the anatomic elements from superficial to deep as this is the method in which the surgical practitioner encounters these components. While trying to grasp the concept of skin anatomy, it is best to consider the entire system as a map and each layer as a road that gives you access to varying structures. Figure 1.1 gives an overall picture of the gross structure of the skin from the depth perspective.

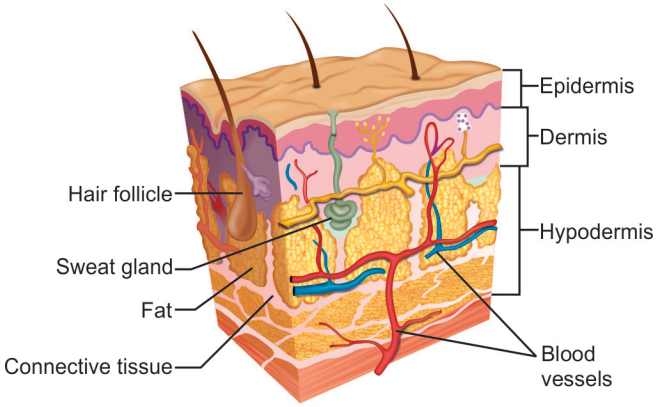
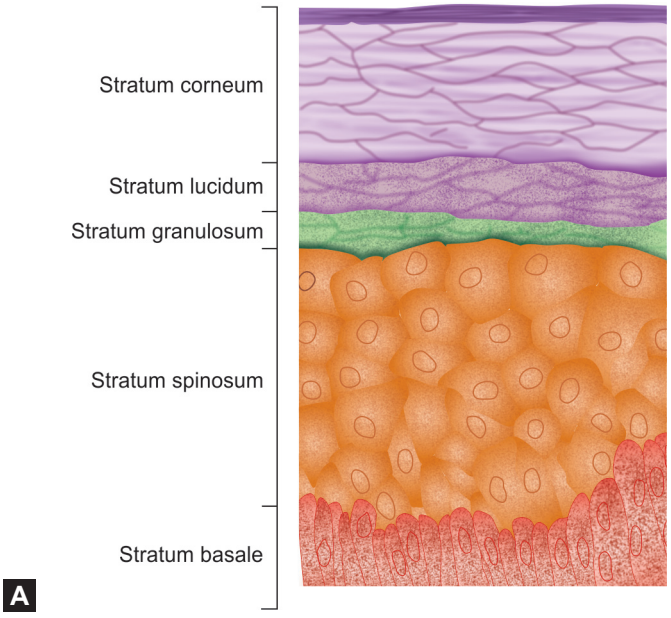
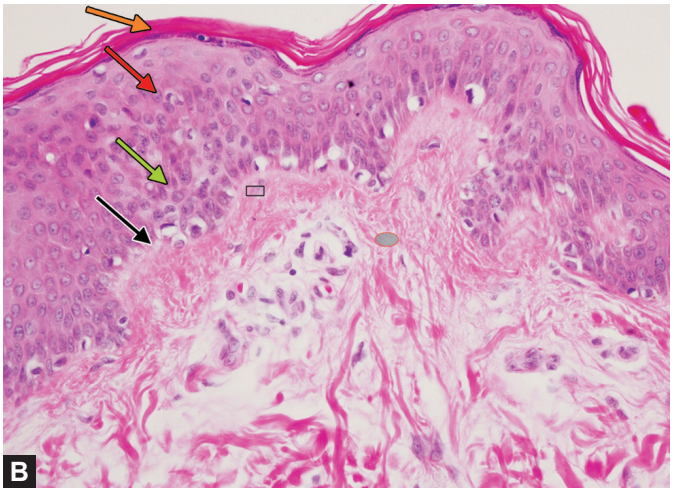


Fig. 1.1: The gross structure of the skin from the depth perspective. The structural layers of the epidermis, dermis, and subdermal tissues are depicted.

The Epidermis

The epidermis, the outermost surface, is composed of cell types that include dendritic cells, Langerhans cells, melanocytes, and Merkel cells. None of these aforementioned cell types is as populous as the keratinocytes, which comprise at least 80–85% of epidermal cells.[1,2] Keratinocytes result in the formation of keratin, which establishes a stratified squamosal network that can be described as the epidermal coat. The epidermis does not contain its own layer of blood vessels, but it is thin enough that it can be supported entirely by underlying dermal networks. Within the epidermis are layers, termed stratum, and named as individual components; these are described in this chapter, and Figure 1.2A shows a representation of where each is located, whereas Figure 1.2B shows a histologic slide depicting these layers as well as the underlying dermis. The epidermis contains the melanocytes, which are the cells of origin of malignant melanoma.[3,4] Melanin, which comes from melanocytes, has a protective function against ultraviolet light and is situated mainly in the stratum





Figs. 1.2A and B: (A) Representation of the location of each of the individual subcomponent layers of the epidermis and how they correlate with each other. (B) Histologic slide depicting the layers that make up the subcomponents of the epidermis. The solid circle indicates the reticular dermis; the open rectangle marks the papillary dermis. The black arrow points to the stratum basale; green arrow, to the stratum spinosum; red arrow, to the stratum granulosum; orange arrow, to the stratum corneum.

basale of the epidermis.[5] The keratinocyte moves from dermal–epidermal attachments up toward the surface, creating distinct epidermal layers through its progression.[6] The thickness of the epidermis varies depending on location with the eyelids and postauricular area being the thinnest (~0.05 mm thick) and the palms and soles being the thickest (~1.5 mm thick).[4]

The stratum corneum is the outermost layer of the epidermis and is largely lipophilic; although it does contain some water, it is <20% of its total composition. Accordingly, its thickness varies on the basis of its state of hydration from 10 to 20 μm.[7] It is the stratum corneum, which plays the most important role as the barrier that helps prevent entry of harmful pathogens.[8,9] The slightly acidic pH of the stratum corneum contributes to its pathogenic averting properties. The stratum corneum serves as a conduit for conducting skin sensation, and this is a direct result of its mechanical properties such as elasticity and yield stress.[10] There is a network of cells, known as corneocytes, which are completely surrounded in a lipid layer and comprise the majority of the stratum corneum. These corneocytes are actually the terminally differentiated form of the keratinocyte cell. It is these corneocytes that are directly responsible for the mechanical barrier that is created, and its lipophilic properties permit fluid retention.[11] One important aspect to bear in mind is that the stratum corneum consists of anucleated or dead cells, making it the final phase of keratinocyte differentiation.

The stratum lucidum will be mentioned briefly for purposes of completion. However, it should be noted that the facial, head, and neck skin does not contain this layer. The stratum lucidum is a layer found exclusively in thickened areas of skin such as the palms of the hands and soles of feet and is composed of dead skin cells.

The stratum granulosum is often referred to as simply the granular layer. This is a thin layer in which reside keratohyalin granules, which promote cross-linking of keratin.[12,13] This zone in addition to the overlying corneum helps maintain water and avoid volume loss from the body via its lipophilic nature.

The stratum spinosum is also known as the spinous layer and described as "prickly". These descriptions are a result of the histologic appearance of their cellular desmosomes or intercellular bridges.[14] The majority of this epidermal layer has tonofilaments that can be seen in large numbers, gathered in coarse bundles when studied by electron microscopy. This differs from the stratum granulosum where there are significantly less tonofilaments.[15] In addition, on electron microscopy the more

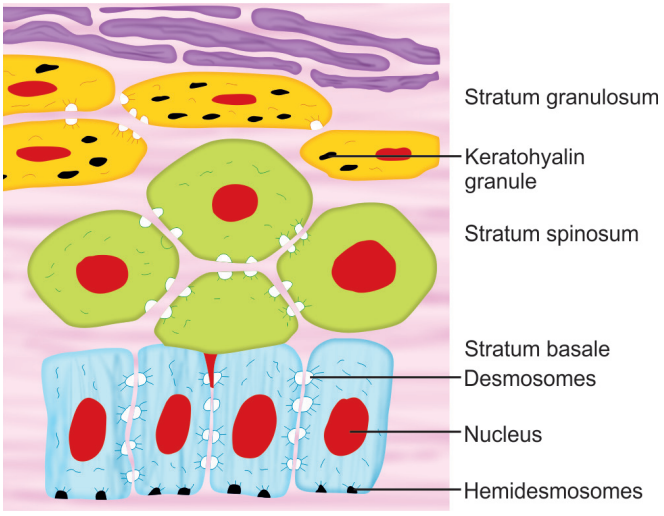


Fig. 1.3: The microstructure and environment of the stratum basale.

complete structure of the desmosome can be appreciated when compared with light microscopy. There is a distinct size difference in the intercellular bridges based on the depth level of the stratum spinosum. Specifically, the desmosomes that connect the basal cells to the spinous cells, or the deeper layers, are smaller than those in the remainder of the spinosum layer.[15] The stratum spinosum together with the stratum basale is termed the Malpighian layer.

The stratum basale is the deepest layer of the epidermis and therefore separates it from the underlying dermis. This tier consists of columnar or cuboidal cells, which are in direct contact with the basement membrane. The Malpighian layer contains melanin pigment, mostly in the stratum basale. The stratum basale represents an important anatomical landmark for the facial plastic surgeon; it is the deepest part of the skin that is treated with a superficial skin treatment such as microdermabrasion.[16] The microstructure of the stratum basale is illustrated in Figure 1.3.

The Epidermal Adnexa

The adnexa refers to the appendages of the integumentary system. The epidermal adnexa consists of important appendages including pilosebaceous units, sweat glands, and sebaceous glands not associated with hair follicles (such as those encountered in the eyelids).

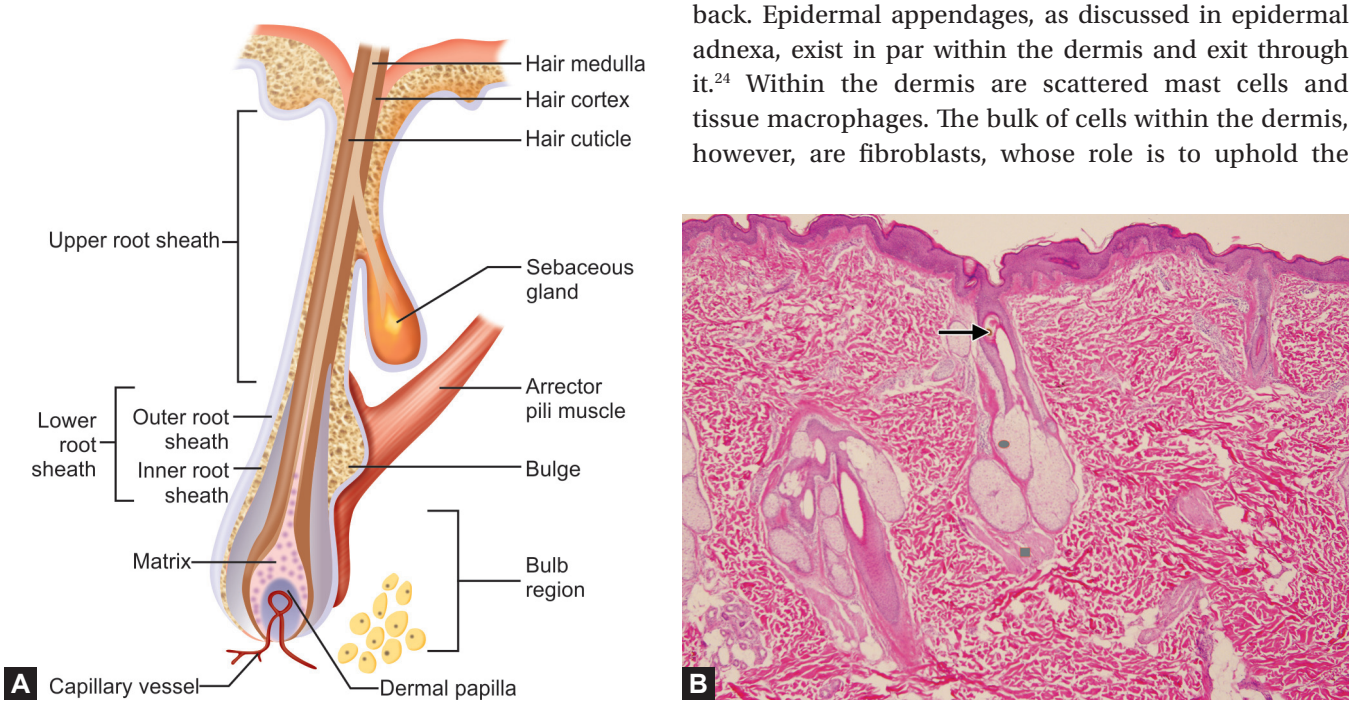
The physiology of the pilosebaceous unit will be described in conjunction with its anatomy as it is a key dynamic structure of the integumentary system. Figure 1.4A is a depiction of a pilosebaceous unit with its components.

These units represent an extremely important clinical structure for the plastic surgeon as acne is a manifestation of inflammation of the pilosebaceous units. The pilosebaceous unit is formed by a hair follicle that contains both hair and sebaceous glands. Its three basic components are the hair follicle, the sebaceous gland, and the arrector pili muscle that causes the hair to stand up in response to sympathetic stressors. Figure 1.4B is a histologic slide showing these components. The sebaceous glands secrete lipids and are found in high densities in the face and scalp. It is after puberty that these glands begin to function as a result of the hormonal influence of androgen.[17,18] In males, the vellus hair on the face transforms into terminal hairs and the opposite occurs on the scalp.[19] The sebaceous glands produce sebum, which produces an individual-specific odor and is thought to be involved in sexual and social attractions. Furthermore, sebum delivers vitamin E and other compounds to the stratum corneum. The sebum has antimicrobial properties and is known to be fungistatic.[19] The pilosebaceous unit can present as one of three anatomic forms, the terminal hair follicle, the vellus hair follicle, and the sebaceous follicle. The terminal follicle, such as that found in the distribution of the male beard, consists of thick stiff and long hair. Because its diameter is wide, it occupies most of the canal, thereby preventing debris from depositing and acne from forming. The vellus follicle is smaller than the terminal one and has larger sebaceous glands, and the hairs from these follicles are much smaller and are barely perceptible to the observer. The vellus hairs are commonly referred to as "peach fuzz." Finally, the sebaceous follicle is the most likely site of acne, because it has a very deep and wide canal, which is easily filled with debris or other irritant. It has a very tiny hair, which is imperceptible in relation to the large canal and sebaceous glands.[20,21]

Possibly, the most important function of the pilosebaceous unit, in the eyes of the plastic surgeon, is its role as a reservoir for keratinocytes and stem cells, which proliferate and aid in epithelialization and complete wound healing.[22,23] To summarize, the very important pilosebaceous unit plays a role in thermal regulation, hair production, sebum production, cell signaling, and wound healing. Additional roles include ultraviolet protection and sensory perception.

The Dermis

The dermis is the next layer of skin, immediately below the epidermis. It serves to provide support and nutrients to the overlying epidermis. Its thickness varies throughout the body, from 0.3 mm on the eyelid to 3.0 mm on the back. Epidermal appendages, as discussed in epidermal adnexa, exist in par within the dermis and exit through it.[24] Within the dermis are scattered mast cells and tissue macrophages. The bulk of cells within the dermis, however, are fibroblasts, whose role is to uphold the



Figs. 1.4A and B: (A) Depiction of a pilosebaceous unit with its components. These units represent an extremely important clinical structure for the plastic surgeon as acne is a manifestation of inflammation of the pilosebaceous units. (B) A histologic image of the pilosebaceous unit. The black arrow indicates a hair follicle; solid rectangle marks a sebaceous gland; solid square marks the arrector pili muscle.

structural components. The key structural component of the dermis is collagen. Abundant collagen is responsible for the tensile strength of dermis as well as most of dermal fat free dry weight.[24]

Several varieties of collagen types are found in the dermis with a distribution of approximately 80% type I collagen, 15% type III, 5% type IV, and type V.[24] The 4:1 ratio of type I to type III collagen is also present in scars after wound healing. Elastic fibers are present in dermis and arranged in all directions contributing to skin recoil. Aging and ultraviolent light damages these fibers and causes wrinkles.[25]

The elastic nature of skin can be exploited for reconstructive purposes. Tissue expanders placed subcutaneously gradually inflate and stretch collagen and elastin in the dermal layer ultimately increasing the surface area of skin. Ground substances in the dermis are displaced with a resultant dermal structure that is thinner while the epidermis thickens.

Dermal architecture is subdivided into two layers: papillary dermis (pars papillaris) and the deeper reticular dermis (pars reticularis). There is no sharp demarcation between the two layers; however, each has its own specialized composition and function.[25] Figure 1.5 is representative of the two subcomponents of the dermal structure.

Papillary Dermis

Papillary dermis is the thinner more superficial layer that forms the junction of the epidermis and dermis. Its form consists of wavy, undulating finger-like projections of dermis into the epidermis reminiscent of a mountainous landscape. These projections, called rete pegs, maximize surface area between the layers and expedite oxygen and nutrient transport.[24] These rete pegs diminish with age and can cause epidermal gliding and shearing.

Structurally, connective tissue of the papillary dermis is arranged in a more chaotic fashion than that of the reticular dermis. The loose configuration of this tissue is occupied by more ground substance composed of a variety of anionic polysaccharides or glycosaminoglycans. This matrix is governed by fibroblasts and mast cells and has implications in water binding and collagen interaction.[24] Its fluid gel-like composition facilitates nutrient and hormone transport through the dermis. Ground substance also provides fullness to the skin and protects against compressive forces directed toward surface of the body.[25]

Papillary dermis also contains unmyelinated nerve endings that provide pain, itch, and temperature sensations.

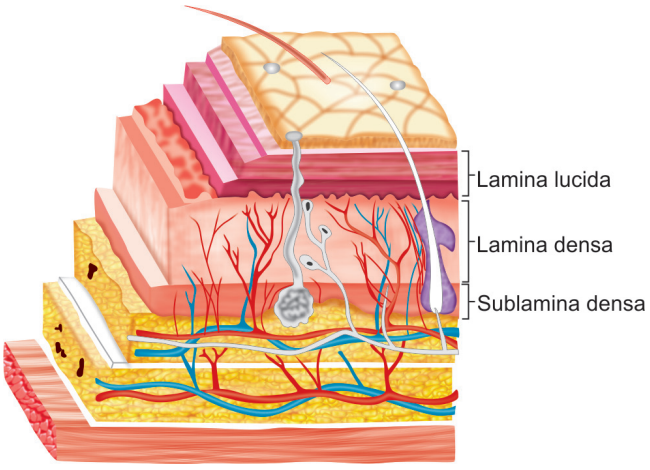


Fig. 1.5: The two subcomponents of the dermal structure.

Reticular Dermis

Situated just deep to the papillary layer of dermis, the reticular dermis extends to the subcutaneous tissue. It is thicker and contains more collagen and elastic tissue arranged in parallel to the skin, in a more organized fashion than the papillary dermis. In 1861, Langer described the collagen orientation as a "lattice-like network with much extended rhomboidal meshes".[24] The coarse bundles of collagen give this layer strength aiding in surgical skin closure.

Subcutaneous Tissue

Located beneath the dermis, the subcutaneous tissue or hypodermis consists mainly of adipocytes and loosely joins the skin to deeper structures. Medications administered in this layer have rapid uptake because it is replete with vasculature.[26]

Skin Vasculature

Cutaneous arteries arise, either directly or indirectly, from underlying source arteries particularly from underlying muscles.[3] These source arteries penetrate the muscle and fascia and exit toward the surface in an orientation that is perpendicular to the skin. Its distal and superficial branches comprise the dermal and subdermal plexus, which are essentially webs of interconnected vessels. It is these dermal and subdermal plexus that provide the basis of random patterned skin flaps used in reconstructive surgery. A few examples of such flaps include advancement, transposition, bilobed, and rotation flaps.

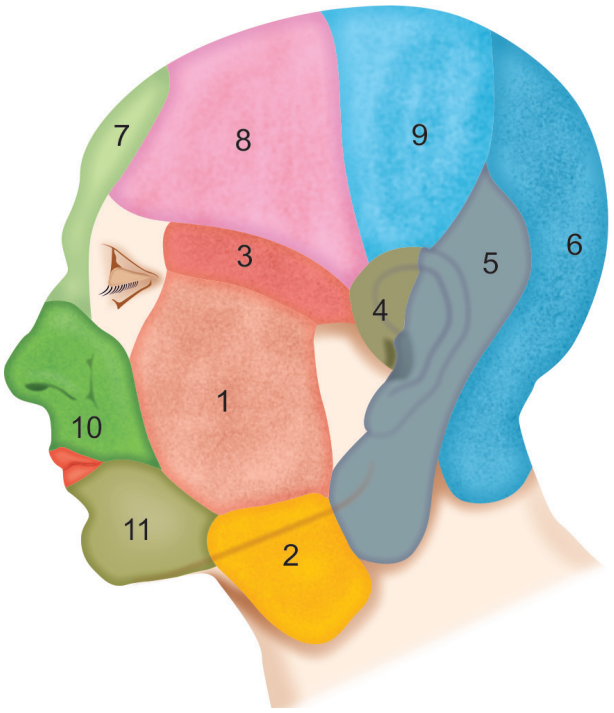


Fig. 1.6: A basic distribution of the vascular territories that supply the skin of the face and scalp. These supplying arteries are in relatively constant locations and knowledge of their territories is useful in designing local flaps.

The survival of these flaps is directly related to the vascular supply, which can be increased by adjusting the base to length ratio of the flap. As a general and loose rule, in a random patterned flap, the flap should be no longer than three times its width, and additional increase in vasculature can be obtained by delaying the flap inset.[27]

Facial vasculature territories have been well delineated by studies performed by Whetzel and Mathes. They described a detailed analysis of 11 vascular territories of the face and head and divide them up into three patterns of vascularization. These include:

1. Small and densely populated arteries that supply the anterior face (facial and infraorbital artery perforators)
2. Large and more sparsely populated arteries that supply the lateral face (transverse facial, submental, and zygomatico-orbital)
3. Small and densely populated arteries that supply the scalp (superficial temporal and posterior auricular).[28]

Figure 1.6 shows a basic distribution of the vascular territories that supply the skin of the face and scalp. These supplying arteries are in relatively constant locations, and knowledge of their territories is useful in designing local flaps.

PHYSIOLOGIC ROLE OF THE SKIN

The integumentary system serves its role as a major protector from the outside world, but its additional dynamic responsibilities are diverse and include sensation, thermoregulation, and metabolism.[26] Each of these is described separately, although their interrelated factors may blur the lines of separation in vivo, particularly with regards to thermoregulation and metabolism.

Sensation

Sensory receptors are located throughout the skin and serve a host of functions. Nerves carrying sensation signals from the skin can be either myelinated or unmyelinated (naked nerve fibers). Those that are unmyelinated nerve fibers are terminally exposed in epidermis and respond to itch, pain, and temperature. These sensations can be particularly noticeable when felt on the face. Myelinated fibers correspond to end organs categorized as mechanoreceptors, thermoreceptors, and nociceptors. Mechanoreceptors respond to stretch, vibration, pressure, and touch. Thermoreceptors detect temperature changes. Nociceptors are specific for pain. Specific examples of mechanoreceptors include Merkel cells in epidermis for touch, Meissner corpuscles in dermal papillae for textural touch, Pacinian corpuscle in hypodermis for vibration, and finally peritrichial nerve endings associated with hair follicles for hair movement.[29] The distribution of receptors varies depending on the location of the body. With age and certain diseases, that can cause neuropathies such as diabetes, the function of the receptors becomes less sensitive and can result in traumatic or thermal injury.

Thermoregulation

Temperature control is a major function of skin. External temperature, as detected by skin afferents, enters a feedback loop with the anterior hypothalamus. The hypothalamus correlates this with the body's internal core temperature and serves as a thermostat, altering blood flow to the skin in concert with sweating or shivering to return temperature to its thermoneutral set point. Anatomically, arteriovenous shunts governed by the autonomic system link the subpapillary plexus with the deep plexus of vessels near the junction of the dermis and subcutaneous tissue. Flow through arteriovenous shunts is altered by sympathetic vasoconstrictor nerves acting on α-1 and α-2 arterioles. These arterioles are tonically active at neutral temperatures,

allowing the body to adjust to minor temperature fluctuations with blood flow manipulation. When thermoneutral, blood flow through the skin is approximately 250 mL/min.[30] In cold temperature, flow through these anastomoses decreases and blood flow is diverted to the deeper plexus of vessels. During overheating, blood flow to the superficial skin can increase massively, up to 6–8 L/min, partially because of relaxation of arterioles but mainly because of cutaneous vasodilation.[30] As a result, blood shunts to the superficial subpapillary plexus from deeper areas of circulation and its heat can dissipate by convection with the coordinated cooling effects of evaporation of sweat released by eccrine sweat glands.[26] Interestingly, the temperature threshold for sweating and blood flow alterations is not the same, with sweating usually occurring at a higher threshold. Cutaneous vasodilation, also controlled by the cholinergic autonomic system, is not understood as well as the vasoconstrictor system. It in part works with the production of nitric oxide and is responsible for most of the increase in cutaneous blood flow during hyperthermia. It has a limited role in cold temperature, where shunt arterioles are stimulated and blood flow is diverted to deeper vessels reducing heat loss at the surface. Aside from reflex neurologic control of temperature, local temperature changes to a region of skin will affect blood flow. Heating skin locally increases blood flow and is thought to be mediated by local neural control via C-fibers. Local cooling can almost stop blood flow to a region of skin by activating arterioles, this time without input from the central nervous system.[30] Cutaneous blood supply to some areas of the body, such as the face, can also be affected by emotional state.[29]

Knowing the mechanisms of blood flow regulation to the skin makes it easy to understand why certain surgical practices are followed. Simple maneuvers such as avoiding ice packs or caffeine can be employed to maximize blood flow to a new reconstruction. The topical application of nitropaste to a reconstruction is another example. Savvy surgeons can implement the fundamentals of thermoregulation to their advantage.

Other Functions of Skin

Aside from providing a waterproof barrier to the outside world, skin has important immunologic and metabolic functions. Antigen presenting cells, such as Langerhans cells, situated in the epidermis constantly sample the environment and activate T lymphocytes when exposed to an antigen.[29] Metabolically, skin is essential for the transformation of provitamin-D to previtamin-D during ultraviolet light exposure. In addition, skin is part of sexual signaling. As, the most exposed organ in the body, it is a visible proxy measure for health and youth, attracting the opposite sex.[26]

CONCLUSION

This chapter illustrates the composition, both structurally and functionally, of the integumentary system in a comprehensible way. The skin has several layers that should be thought of as individual components with unique characteristics and functions. Like peeling away the layers of an onion, each individual depth level or stratum should be visualized in the mind's eye as its own unique structural entity when performing surgery.

The surgeon should understand that the structural epidermis, in its entirety, is composed of cells in a series of phases of the life cycle of the keratinocyte, with the most visible exterior made up of a nuclear keratin. The stratum corneum is the most important of the barrier-providing layers, and the stratum basale separates the epidermis from the underlying dermis.

The epidermal adnexa, which largely originates in the dermis and makes its way into the epidermis, is one of the keys to skin regeneration and wound healing. Its role in providing skin-derived stem cells, with further study, may prove to be of significant clinical value for therapeutic use in other parts of the body.[31] It is specifically the follicular stem cell niche or the "bulge" of the appendage that is composed of multipotent cells, making the epidermis and its appendages a unique entity.[32]

The dermis, structurally maintained by collagen and fibroblasts, is the foundation and substantive nutrient providing groundwork for the epidermis. Its two subcomponents are the papillary and reticular dermis. The papillary dermis is intimately related to the epidermis with its projections and tight adherence, whereas the reticular dermis is a stronger framework-type structure. This is clinically important for the surgeon in suturing techniques. A satisfactory plastic closure often requires a deep dermal element to reduce wound tension as well as eliminate dead space. It is the more structurally sound reticular dermis that has the strength for good apposition with deep dermal sutures. Although it is not easy to tell the exact demarcation point of reticular dermis grossly, the strength on closure lets the surgeon know that they

are indeed in the stronger, deeper layer. Another very important situation in which the surgeon needs to be aware of their dermal depth is in skin resurfacing. Whether by dermabrasion, laser techniques, or chemical peels, the deep reticular dermis should be avoided to prevent scar formation. This is done by clinical judgment and observation, such as identification of uniform white fibrils and pinpoint bleeding.

The vascular supply of the skin is based on plexuses and arcades of small vessels originating from larger axial vessel. These arcades anastomose liberally and form the basis of the so-called "random patterned" flaps. The skin of the face, scalp, and neck has a disproportionately stronger blood supply when compared with other parts of the body, allowing for improved wound healing and decreased rates of infection. The specific territories of vascular supply to the face have been well established and are extremely useful in the planning and execution of operative procedures. When rhytidectomy procedures are performed with this vascular preservation principle in mind, the postoperative wound-healing course can improve and complications such as distal edge necrosis can be reduced.

Skin physiology can be difficult to separate from skin anatomy as there is a dynamic component to the structural evolution that is constantly occurring, particularly in the epidermis. However, the distinctive sensation, thermoregulation, immunity, wound-healing factors, and metabolic activity attest to both the complexity and the importance of this large organ system.

The ability of the surgeon to work intelligently with the integumentary system provides endless possibilities for his/her procedural armamentarium. It is a lack of understanding of the capabilities and anatomic intricacies of the skin that leads to poor results and unexpected outcomes.

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CHAPTER

2

Wound Healing

Virginia P Teti, Sam P Most

OVERVIEW

The skin serves as a protective barrier to the environment; hence, the primary goal in the treatment of wounds is rapid closure with functional preservation. Wound healing is a complex yet coordinated series of events that begins with tissue injury, followed by hemostasis, inflammation, regeneration and remodeling. Recent advances in cellular and molecular biology have expanded our understanding of the dynamic molecular processes involved in wound healing. This chapter serves to review skin anatomy, the phases of wound healing, recent developments and special considerations in wound healing.

ANATOMY

A rudimentary understanding of basic skin anatomy is imperative to fully appreciate the complex yet coordinated biological events occurring during cutaneous wound healing. Essentially, the skin is composed of two layers, the dermis and epidermis.

The epidermis overlies the dermis and provides a protective, waterproof covering characterized by its color, quality, texture and scar tissue. The epidermis is a dynamic covering composed of keratinocytes arranged in four layers. From deep to superficial, the layers include the stratum basale, stratum spinosum, stratum granulosum and stratum corneum. The stratum basale is a unique layer that continually replicates, allowing keratinocytes to migrate and eventually form the stratum corneum until they are shed from the surface of the epidermis.[1] The average turnover time of the epidermis is 30 days. Destruction of the superficial layers via abrasions or superficial burns may form without scarring if the basal layer is not disrupted. The epidermis is thin at birth, becomes thicker at puberty and in early adulthood, and thins again in the fifth to sixth decade of life.[2]

Other cells involved in the epidermis include the Langerhans cells, melanocytes, and Merkel cells. Derived from bone marrow, Langerhans cells are mediators of immunologic response and serve as potent antigen presenting cells. Melanocytes are of neural crest origin and reside in the basal layer. Melanin serves to protect keratinocytes from ultraviolet radiation. Merkel cells originate in the neural crest and are found in the epidermis and dermis, with a function not completely understood.

The epidermis is connect to the underlying dermis via the epidermal junction, a layer composed of basement membrane type IV collagen, serving to provide attachment and mechanical support for the epidermis while providing an additional mechanical barrier to the environment (Fig. 2.1).[2]

Beneath the epidermis lies the dermis, fashioned into two layers: an overlying papillary portion and deeper reticular layer. The dermis is composed collagen, elastin and ground substance, which all provide structural support. In addition, the dermis contains the vascular supply to the skin and epithelial lined skin appendages such as sebaceous or sweat glands. The fibroblast, the main cell of the dermis, produces collagen, elastin and ground substance. Dermal collagen provides strength and extensibility of the skin, whereas elastic fibers provide recoil for the skin. Ground substance is composed of glycosaminoglycan, hyaluronic acid, chondroitin-4-sulfate, dermatan sulfate, and fibronectin. These substances play an important role in skin

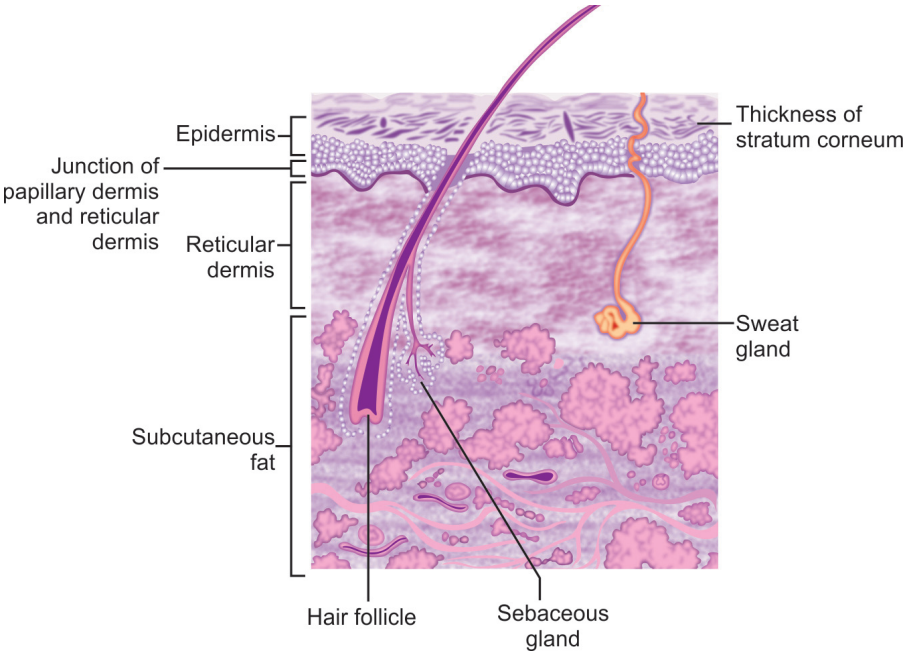

Fig. 2.1: Normal skin and appendages.
Source: Modified with permission from Koranda FC. Dermabrasion. In: Thomas JR, Hold GR (Eds). Facial Scars: Incision, Revision and Camouflage. St Louis, MO: CV Mosby; 1989.

hydration and preservation of skin elasticity.[3] Disruption of the dermis results in loss of wound tensile strength and requires longer periods to heal.

PHASES OF WOUND HEALING

The mechanisms of wound healing are described in four basic phases: hemostasis, inflammation, proliferation and remodeling. However, these four phases are oversimplified descriptions of a complex and dynamic interplay of molecular processes occurring in synchrony rather than independent, autonomous events.

The hemostatic and inflammatory phases involve platelet aggregation on a fibrin clot leading to hemostasis followed by migration of inflammatory cells to remove debris allowing a favorable environment for repair. The proliferative phase involves the formation of granulation tissue, collagen deposition, angiogenesis, epithelial migration and wound coverage. Lastly, the remodeling phase involves collagen reabsorption and replacement. These phases will be further discussed with specific attention to cellular and biochemical interactions (Fig. 2.2).

Hemostatic Phase

Tissue injury causes hemorrhage, allowing platelet interaction with thrombogenic, subendothelial connective tissue, and thus, platelet activation. Platelet activation results in the immediate release of vasoactive substances such as serotonin and catecholamines, thus further initiating vasoconstriction, platelet aggregation, and formation of a primary hemostatic plug. The remaining vasoactive mediators, bradykinin, histamine and serotonin, induce vasodilation and small vessel vascular permeability with transudation of fluid.

Platelets are the primary mediator in the hemostatic phase. Initially, platelets induce vasoconstriction, followed by a complex cytokine-induced cascade of events based on the release of preformed mediators from α-granules of the platelets. Some preformed mediators released from activated platelets include platelet-derived growth factor (PGDF), fibroblast growth factor (FGF), epidermal growth factor (EGF), and coagulation factors of both the intrinsic and extrinsic clotting pathways (Table 2.1). The resultant fibrin clot provides a scaffold promoting migration of fibroblasts, leukocytes and keratinocytes.[4,5]

Inflammatory Phase

Within the first 6–8 hours after tissue disruption, the inflammatory phase of healing commences with an infiltration of white blood cells, migrating through vessel walls, in a process known as diapedesis. Leukocytes and

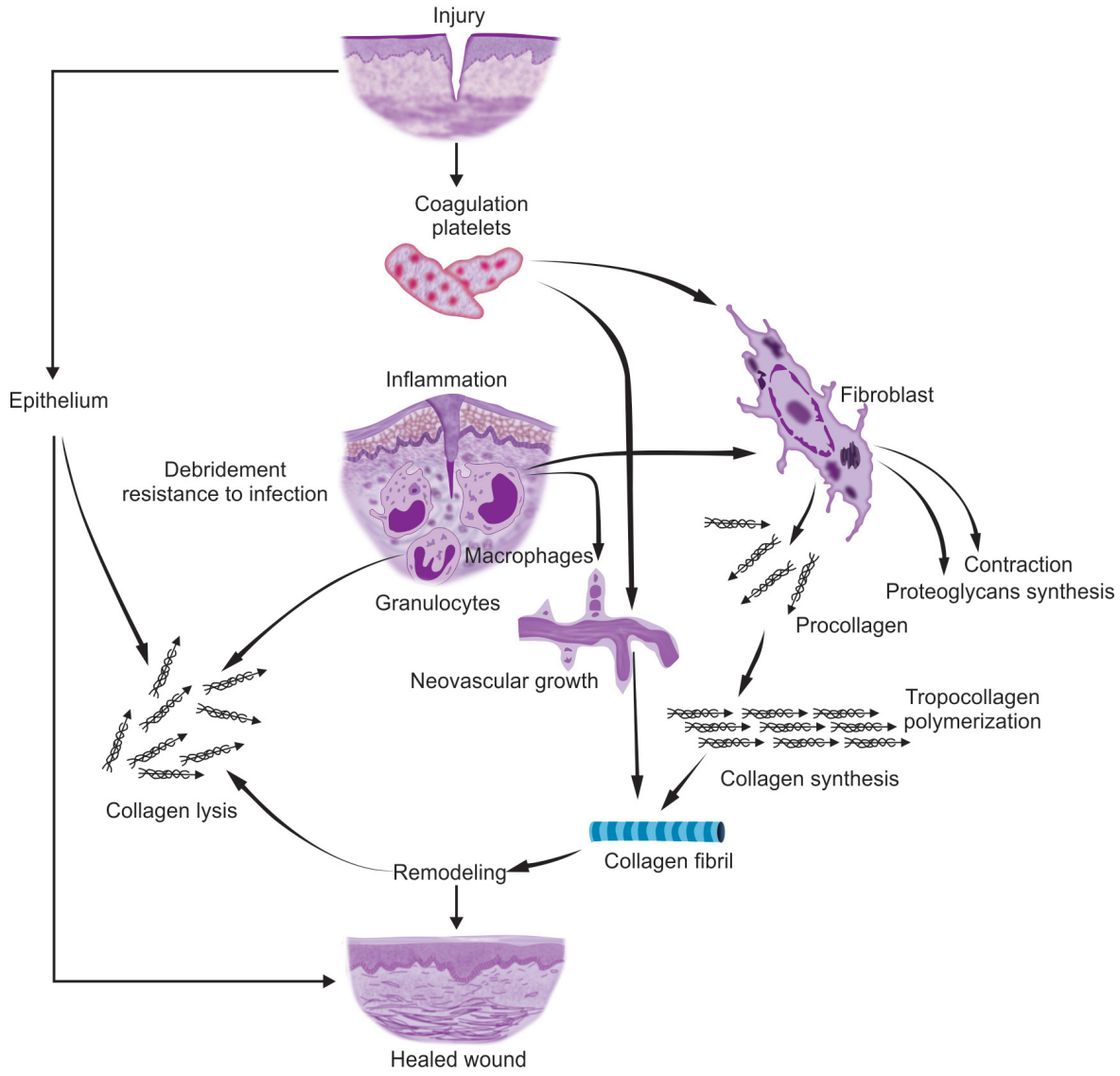

Fig. 2.2: Overview of the complex and coordinated interactions occurring during wound healing.
Source: Modified with permission from Feinberg SE, Larson PE. Healing of traumatic injuries. In: Fonseca RJ, Walker RV (Eds). Oral and Maxillofacial Trauma. Philadelphia, PA: WB Saunders; 1991.

monocytes are recruited via platelet-derived cytokines. Initially, polymorphonuclear leukocytes engulf cellular debris and foreign material for the first 24 hours.[5] Next, the monocyte predominates and matures into the macrophage. Macrophages bind to proteins of the extracellular matrix, thereby stimulating phagocytosis of microorganisms.[4-6] This process of cellular debridement continues for several days in clean wounds and may persist for weeks in contaminated wounds. Most importantly, monocytes and macrophages produce growth factors to recruit fibroblasts and endothelial cells, creating the scaffold for the proliferative phase of wound healing. Some growth factors include colony-stimulating factor-1, tumor necrosis factor alpha and PDGF, interleukin-1 (IL-1), transforming growth factor beta (TGF-β), and insulin-like growth factor (IGF) (Table 2.2). Monocytes and macrophages play an essential role in the transition between the inflammatory and regenerative phases of wound healing. In some conditions, these cells may inhibit fibroblast collagen synthesis.[6] Some studies have demonstrated that macrophage-depleted animals have defective wound healing.[7]

Table 2.1: Substances released from the α-granules of platelets during wound healing

Platelet-derived growth factor
Basic fibroblast growth factor
Vascular endothelial growth factor
Transforming growth factor β1
Transforming growth factor α
Epidermal growth factor
Thrombospondin
Platelet thromboplastin
Coagulation factors
Serotonin
Histamine
Platelet-activating factor
Hydrolytic enzymes
Endostatin (antiangiogenic)

Source: Reproduced with permission from Hom DB, Linzie BM, Huang TC. The healing effects of autologous platelet gel on acute human skin wounds. Arch Facial Plast Surg. 2007;9(3):174-83.

Proliferative Phase

The next significant component of wound healing includes the proliferative phase, which involves epithelial regeneration, fibroplasia, collagen formation, wound contraction and neovascularization.

Epithelial regeneration serves to provide a barrier to infection and foreign materials, protecting the underlying tissues from desiccation and bacteria. Commencing within 24 hours, basal epithelial cells differentiate and actively divide with pseudopod extensions of their cytoplasm, leading to migration from surrounding wound margins or adnexal structures. Production of collagenase by epidermal cells and activation of plasmin by plasminogen activator facilitates the degradation of collagen and extracellular matrix proteins, thereby allowing the epidermal cells to migrate between the fibrin eschar and collagenous dermis (Fig. 2.3).[4] A moist wound environment will improve epithelialization, thereby preventing desiccation and necrosis of the underlying dermis, which may delay migration of epithelial cells.[6] The epithelial cells of opposing

Table 2.2: Cytokines that affect wound healing

Cytokine	*Major source*	*Target cells and major effects*
Epidermal growth factor family		Epidermal and mesenchymal regeneration
Epidermal growth factor	Platelets	Pleiotropic-cell motility and proliferation
Transforming growth factor α	Macrophages, epidermal cells	Pleiotropic-cell motility and proliferation
Heparin-binding epidermal growth factor	Macrophages	Pleiotropic-cell motility and proliferation
Fibroblast growth factor family		Wound vascularization
Basic fibroblast growth factor	Macrophages, endothelial cells	Angiogenesis and fibroblast proliferation
Acidic fibroblast growth factor	Macrophages, endothelial cells	Angiogenesis and fibroblast proliferation
Keratinocyte growth factor	Fibroblasts	Epidermal-cell motility and proliferation
Transforming growth factor β family		Fibrosis and increased tensile strength
Transforming growth factor β1 and β2	Platelets, macrophages	Epidermal-cell motility, chemotaxis of macrophages and fibroblasts, extracellular-matrix synthesis and remodeling
Transforming growth factor β3	Macrophages	Antiscarring effects
Other		
Platelet-derived growth factor	Platelets, macrophages, epidermal cells	Fibroblast proliferation and chemoattraction, macrophage chemoattraction and activation
Vascular endothelial growth factor	Epidermal cells, macrophages	Angiogenesis and increased vascular permeability
Tumor necrosis factor α	Neutrophils	Pleiotropic expression of growth factors
Interleukin-1	Neutrophils	Pleiotropic expression of growth factors
Insulin-like growth factor 1	Fibroblasts, epidermal cells	Reepithelialization and granulation tissue formation
Colony-stimulating factor 1	Multiple cells	Macrophage activation and granulation-tissue formation

Source: Modified with permission from Singer and Clark.[4]

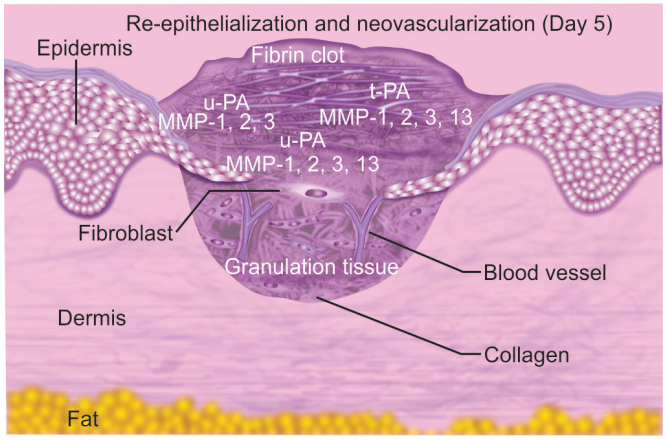

Fig. 2.3: A cutaneous wound after 5 days. Epidermal cells migrate across the wound as blood vessels proliferate into the fibrin clot. MMP, metalloproteinase; t-PA: tissue plasminogen activator; u-PA: urokinase-type plasminogen activator.
Source: Reproduced with permission from Singer and Clark.[4]

wound margins approach until contact inhibition leads to further differentiation and stratification of the basal cells. The new epithelial layer thickens and growth is maximal at 48–72 hours; however, it may continue up to 5 days in delayed wounds until a granulation bed is established. The stimulus for epithelial regeneration is unknown; however, the role of EGF and TGF-α has demonstrated enhanced fibroplasia and granulation tissue stimulation.[8]

Critical to wound healing, fibroblasts synthesize collagen, providing strength to the wound. Derived from undifferentiated mesenchymal cells, fibroblasts migrate into the wound between 48 and 72 hours. Collagen production is a complex series of events. In brief, procollagen undergoes enzymatic cleavage of nonhelical ends and spontaneously assembles into fibers. Next, hydroxylation of lysine and proline amino acids occurs allowing collagen to form cross-links. This important step of hydroxylation of amino acids relies on several cofactors to include vitamin A, C, iron and α-keloglutarate.[5,9] Finally, collagen is arranged into three α-peptide chains in a right-handed triple helix with glycine in every third position. Disulfide bridges between the carboxy terminal ends and intramolecular hydrogen bonding between the α-chains support the alignment of the triple helix structure. Most importantly, the nature and degree of collagen cross-linking establishes tissue-specific parameters and allows collagen to become insoluble in water.

Remodeling Phase

Maturation or remodeling phase involves a dynamic balance between collagen production and degradation. The remodeling phase may extend between 6 months and 24 months in duration. During this period, intense fibroplasia and angiogenesis subside. The scar may become softer and pale in color, all the while becoming stronger. Type III collagen is replaced by type I collagen and the collagen fibers eventually align in a more parallel fashion, thereby increasing the tensile strength and improving appearance. Tensile strength dramatically increases to reach a maximal level and plateaus to ultimately reach approximately 80% of the unwounded skin.[10] It is noteworthy to appreciate that scar tissue will never regain the inherent breaking strength of normal skin.[4,10]

Wound contraction is an ongoing process in wound healing. The mechanism of wound contraction is not entirely understood. During the 2nd week of wound healing, fibroblasts assume a myofibroblast phenotype with abundant actin-containing filaments, resembling contractile smooth muscle cells.[4] The stimulation for contraction is uncertain but likely involves TGF-β1 or β2 and PGDF and cross-linking between individual collagen bundles and attachment of fibroblasts to the collagen matrix.[4] Contraction is centripetal and maximal at 10–15 days.[9] Contraction of the myofibroblast occurs at a rate of 0.6 to 0.75 mm per day.[11] Water and glycosaminoglycans are displaced from the wound during the period of contraction, thereby providing further compression.[9,11] Wound contraction may be delayed or inhibited when antismooth muscle agents are topically applied.

TYPES OF WOUND REPAIR

Three basic types of wound repair exist: primary, delayed and secondary. Primary wound healing involves meticulous reapproximation of full-thickness wound edges via tape, suture, staples, or graft. Re-epithelialization and wound contracture play a minor role in the healing of a primary intention wound.

When a wound is contaminated or infected, the wound is allowed to remain open for several days to reduce the bacterial load. Once granulation tissue appears and the bacterial count is reduced, the wound is closed. This process of waiting to close the wound is considered delayed.

A spontaneous or secondary wound is one in which the wound is allowed to heal by contraction and re-epithelialization occurs over an extended period without surgical intervention to reapproximate the wound margins.

Several factors including the size, shape and depth of the defect may alter the time required for healing by secondary intention. The depth of injury will affect healing; the deeper wound will require more time to allow granulation tissue to fill the defect until epithelial migration may occur.[5,6] Exposed cartilage and cortical bone may demonstrate delays due to the impaired blood supply and thus impaired granulation tissue formation. Small fenestrations in the cartilage and burring or curettage of the bone may promote vascularity and hence improved epithelialization. The geometric shape of the wound may affect healing by secondary intention based on the fact that healing time is dependent on the diameter of the largest circle contained within the wound. Thus, fusiform or linear shapes contract more efficiently than circular designs.[12] Lastly, the time for secondary wound healing is proportional to the log of the area. A 1000-mm wound will heal twice as fast as a 100-mm wound, despite being 10 times larger than the smaller defect. Hence, larger wounds may heal faster than expected.[12]

Wounds that heal by secondary intention may demonstrate excellent cosmetic outcomes. Patient selection is paramount. The ideal location of a wound left to heal by secondary intention is an area within a concavity, surrounded by lax skin, thereby allowing wound contraction to prevent depression.[13] Finally, since melanocytes do not migrate during epithelialization, wounds healed by secondary intention wounds are relatively hypopigmented.[6,12,13] Considerations for secondary intention healing may include decreased perioperative morbidity, reduced postoperative infection or bleeding, and improved monitoring for recurrence.[13]

Local and Systemic Wound Healing Factors

There are numerous factors and conditions that may impair wound healing. We will subdivide these factors into local and systemic categories. Local wound factors include ischemia, infection, tissue trauma, desiccation, medications, antineoplastic agents and vitamin excess. Systemic factors impairing wound healing include hereditary conditions, vascular disorders, metabolic disorders, malignancy and immunologic deficiency states.

Local Wound Healing Factors

Appropriate wound closure techniques are essential for adequate wound repair. The tenets of Halsted must be observed: gentle handling of tissue, aseptic technique, sharp anatomic dissection, careful hemostasis, obliteration of dead space, avoidance of tension, and reliance on rest.[9] One must be mindful to avoid crushing skin edges, excessive electrocautery, strangulation of tissue, and inappropriate suture material as means to prevent tissue inflammation, necrosis and infection.

Wound humidity is an essential component of local wound healing, allowing epithelial cells to migrate if adequate surface humidity exists.[9] A moist wound environment improves epithelialization. Desiccated and crusted wounds heal slower than those kept moist.[6]

Systemic Wound Healing Factors

Certain specific vitamin deficiencies may impair wound healing. Vitamin C deficiency prevents the hydroxylation of proline and lysine, thereby significantly impairing collagen synthesis. Vitamin A deficiency reduces epithelialization and collagen synthesis, thereby reducing wound tensile strength and preventing wound closure, allowing increased susceptibility to infection. Supplementation with vitamin A may mitigate the deleterious effects of wound healing due to radiation and steroid therapy.[5] Zinc deficiency may affect fibroplasia and prevent cell proliferation.

BIOLOGIC CONSIDERATIONS OF WOUND HEALING

The normal healing process is an orderly and reproducible response regulated by inflammatory mediators via intricate and often overlapping mechanisms. These mediators include eicosanoids, cytokines, nitric oxide and growth factors.

Eicosanoids are biologically active arachidonic acid metabolites, including prostaglandins, prostacyclins, thromboxane, leukotrienes and lipoxins. The eicosanoid mediators primarily influence the inflammatory response to wound healing, including vasoconstriction, vasodilation, vascular permeability and chemotaxis, and adhesion of inflammatory cells.[14]

Cytokines are small molecular weight peptides and glycoproteins (5–30 kDa) that tightly regulate inflammation and the immune response. Cytokines include chemokines, lymphokines, monokines, ILs, colony-stimulating factors

and interferons. Produced by inflammatory cells, cytokines influence the activity of hematopoietic cells (monocytes, RBC, bone marrow), whereas growth factors mediate the activity of nonhematopoietic cells.

Nitric oxide influences the inflammatory response to healing, angiogenesis and matrix deposition, and tissue remodeling. Produced primarily from endothelial cells or neuronal cells, nitric oxide may express different isoforms depending on the cellular environment inducing the expression of nitric oxide.

Growth factors are larger molecular weight proteins between 4,000 kDa and 60,000 kDa, produced by nonhematopoietic cells such as macrophages, platelets, fibroblasts, and neutrophils. Growth factors bind to transmembrane glycoproteins that modulate tyrosine or serine kinase activity within the cell. Growth factors may regulate distant cells, neighboring cells or their own target cell. In response, the target cell will up- or downregulate the expression of growth factor receptors. Five families of growth factors exist to date: PDGF, EGF, FGR, the TGF and IGF. Growth factors exert their influence on angiogenic cells, mesenchymal cells and keratinocytes.[14,15]

SPECIAL CONSIDERATIONS IN WOUND HEALING

Fetal Wound Healing

In contrast to the adult wound healing, the mid-gestation fetus demonstrates rapid regenerative healing with indistinguishable scars. The mechanism of the scarless fetal wound healing is not fully elucidated. Early studies focused on the intrauterine wound environment containing the sterile, nutrient-rich amniotic fluid. Several animal studies demonstrate that fetal regeneration is independent of the intrauterine environment, suggesting intrinsic properties of the fetal skin capable of scarless healing.[16] These properties include alterations in TGFs, attenuation of the inflammatory response, and an increased extracellular matrix collagen type III collagen and hyaluronan.[16] Research has focused on the role of TGF (TGF-β) and three isoforms, β1, β2 and β3.[6] In the adult wound, there is a relative increase in TGF β1 and TGF β2, a scar promoting cytokine. In contrast, fetal wounds express decreased levels of TGF β1 and β2 and elevated levels of TGF β3.[16,17] Pathologic hypertrophic scars in adult wounds demonstrate increased levels of TGF β1 and functional inhibition of TGF β1 demonstrates reduced scarring.[17]

Animal studies in postnatal wounds demonstrated improved scarring after addition of recombinant TGF-β3, prompting the development of avotermin, human recombinant TGF-β3, for use as a potential antiscarring treatment. Avotermin was evaluated in a randomized, double-blinded, placebo-controlled, phase II clinical trial with intradermal injections of recombinant TGF-β3 prior and 24 hours after wounding, demonstrating improved scar appearance at 7 and 12 months postoperatively compared with placebo.[18] Unfortunately, phase III trials were terminated after demonstrating failure to meet the primary endpoint of decreased scar formation.[16]

The fetal wound demonstrates a paucity of inflammatory cells, including macrophages, neutrophils and platelets, resulting in diminished TGF-β and PDGF production.[19] The relative paucity in inflammatory cytokines led to the investigation of the anti-inflammatory cytokine IL-10. IL-10 has been shown to deactivate macrophages and neutrophils and decrease proinflammatory cytokines.[16] Early fetal skin, serum and amniotic fluid demonstrate high levels of IL-10 compared with neonatal skin demonstrating minimal levels of IL-10.[20] Animal studies demonstrating overexpression of IL-10 via viral vectors reported a dose-dependent recapitulation of fetal regeneration with imperceptible scars.[21] These laboratory findings led to the development of Prevascar, human recombinant IL-10, as a potential antiscarring therapeutic agent. In a randomized, double-blinded, placebo-controlled trial, Prevascar has been found to improve scar formation at 12 months.[22] Prevascar trials are ongoing.

Further research in the molecular mechanisms of scarless fetal wound healing may have far-reaching therapeutic benefits for a wide array of diseases beyond cutaneous wound healing.

Nerve Healing

Management and treatment of peripheral nerve injuries poses significant clinical challenges and considerable patient morbidity. Despite technical advances in nerve repair, recovery is slow and rarely reaches a functional preinjury status when the nerve has been transected. To optimize nerve recovery, the timing of repair, surgical techniques, and adjunctive growth factors to support the regenerating nerve have been investigated.

The timing of nerve repair remains controversial and divides between early and late repair. The arguments for early repair, prior to Wallerian degeneration, include the

ability to stimulate distal nerve segments for improved reanastamosis. In addition, an earlier repair is considered technically easier with less scar formation. Support for the delayed repair hinges on the fact that the metabolic status of the nerve is at an optimal, regenerative peak approximately 3 weeks postinjury.[23] Tenets of surgical repair to achieve optimal physiologic results of peripheral nerves require tension-free repair and reduced scar formation. The standard method of repair of an injured nerve is by epineurial suture; however, perineurial and fascicular suture have been described. Technically, surgical reapproximation is ideally performed via direct coaptation of the nerve ends, or an interposition graft in the case of a nerve deficit.[24] Alternative forms of nerve repair include nerve tubules. These conduits serve as a guide to direct the sprouting axons toward the distal nerve stump. In addition, the tubes function as a barrier to infiltration by fibrous tissue and a boundary for neurotrophic factors secreted within the microenvironment of the regenerating nerve.[25] Recent animal studies utilizing biodegradable glass fabric entubulation technique demonstrate similar functional outcomes as standard suture neuroraphy, without the demands of microsurgery expertise.[24,26] Future developments in more precise pharmacologic options for enhanced nerve regeneration remain promising. For the present, meticulous microsurgical suture repair remains the gold standard to promote optimal neural healing.

FUTURE CONSIDERATIONS IN WOUND HEALING

A paradigm shift regarding the way we approach soft tissue wounds has occurred since the advent and approval of new biologic and clinical dressings by the Food and Drug Administration (FDA). These products include growth factor products, autologous platelet gel and bioengineered skin. Although several of the products were developed to assist with diabetic ulcers or post-radiated delayed healing wounds, applications to otolaryngologists and facial plastic surgeons will likely have a future impact on the way we treat normal and chronic wounds.

Topical Growth Factors

Topical growth factors were initially introduced in 1998 with the FDA approval of becaplermin [recombinant human PDGF–BB (rhPDGF); Regranex, 0.01% gel; Ortho-McNeil Pharmaceutical Inc, Raritan, NJ, USA] used to induce growth of soft tissue granulation tissue in diabetic ulcers.[27] Physiologic PDGF is stored in α-granules and released upon tissue injury during the blood-clotting cascade. Recombinant human PDGF increases fibroblast replication and induces fibroblasts to produce collagenase, thereby enhancing connective tissue remodeling.[28] Topical keratinocyte growth factor (KGF, palifermin, Kepivance, Biovitrum AB, Stockholm, Sweden) is currently approved by the FDA for oral mucositis. Off-label use of rh-PDGF includes treatment of chronic-irradiated wounds and even pharyngocutaneous fistulas.[29] This off-label use of rh-PDGF may be considered as an option to promote granulation tissue, along with optimal and routine wound care. In poorly healing wounds, one must consider the theoretical risk of local recurrence or malignant transformation and biopsy as warranted.[30] rh-PDGF may be considered in poorly healing wounds to induce further granulation tissue in refractory, poorly healing wounds.

Platelets

Platelets store bioactive molecules in their secretory organelles. Alpha granules contain many proteins involved in biologic processes including hemostasis, cell proliferation, extracellular matrix formation, angiogenesis, vascular remodeling, chemotaxis, and inflammation. Some of the proteins released from α-granules of activated platelets include TGF (TGF-β), PDGF, IGF (IGF-1), basic FGF (bFGF), vascular endothelial growth factor, and connective tissue growth factor.[31] To better simulate the native wound-healing environment, concentrated platelet preparations have been utilized clinically in hopes of modulating and accelerating the healing process.[31]

The concentrated levels of growth factors and other factors inherent to the platelet have propelled the medical industry to develop devices to collect autologous blood and concentrate platelets into plasma. Several systems to produce platelet rich plasma (PRP) are FDA approved; however, these methods are not standardized.

Due to the concentrated levels of growth factors and other substances, concentrated PRP is hypothesized to accelerate wound healing. Clinical results with the use of PRP have been equivocal. In an animal study of topical application of PRP to experimental wounds, Sclafani et al. noted a transient increase of fibroblasts and endothelial cells at day 7, which ultimately abated by day 14.[32] Other investigators report similar outcomes, demonstrating a temporary benefit to the use of topical PRP.[32-34]

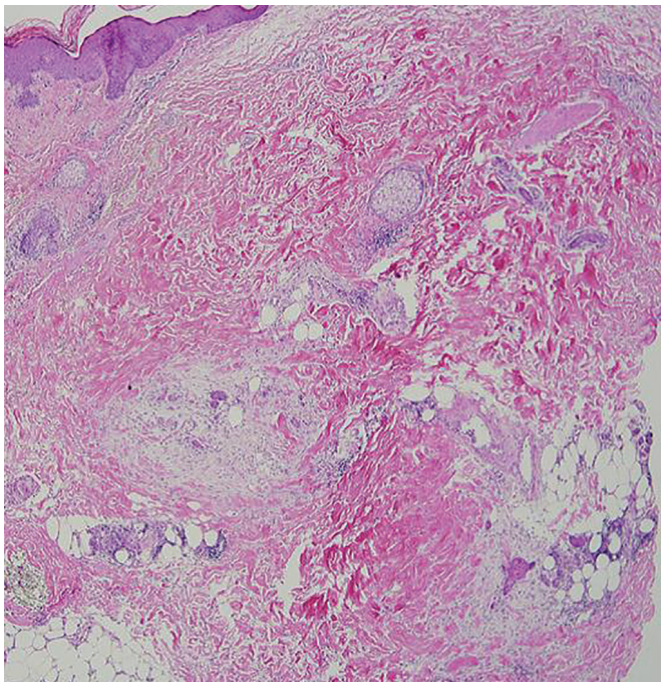

Fig. 2.4: Histological representation of skin 10 weeks after treatment with platelet-rich plasma matrix, demonstrating rests of mature adipocytes, thick new collagen bundles, and new blood vessels. *Source*: Reproduced with permission from Sclafani and Saman.[34]

To enhance the sustained delivery of growth factors inherent to the platelet, platelet-rich fibrin matrix (PRFM) systems were developed. In contrast to platelet-rich plasma, PRFM serves to provide a natural fibrin framework, thereby protecting the growth factors from proteolysis while providing mechanical support and stiffness.[35] Production of autologous PRFM involves centrifuged plasma/platelet suspension, which is transferred to a tube containing calcium chloride, which initiates the polymerization of fibrin. The PRFM is then injected through a 30-gauge needle. Histological changes associated with injection of PRFM in the dermis and subdermis demonstrate new collagen deposition, significant angiogenesis, and adipogenesis at 19 days with a sustained effect (Fig. 2.4).[33] Based on these angiogenic qualities, PRFM has been coinjected with autologous fat to improve the survivability and vascularity of the transferred fat.[33]

Biological Dressing

Prefabrication of tissue-engineered skin replacement products has demonstrated tremendous growth and innovation. Although bioengineered skin is rarely ideal and typically not preferred, improved variety and options for dermal and epidermal-engineered products are now commercially available (Table 2.3). Dermoepidermal products, referred as composite skin substitutes, are composed of allogenic skin incorporated onto a dermal lattice. These products may offer an aesthetic resemblance to normal skin.[30,36] Apligraf (organogenesis) is a living, permanent composite dermoepidermal graft cultured from neonatal foreskin, approved for coverage of venous and diabetic foot ulcers by the FDA. Off-label use includes thin split-thickness skin grafts in patients who cannot tolerate donor morbidity.[30,36,37] More tissue-engineered reconstructive products are required for cutaneous facial injuries.

Table 2.3: Bioengineered skin products

Product	*Composition*	*Approved clinical use*
Apligraf (Organogenesis, Canton, Massachusetts)	Living keratinocytes and fibroblasts derived from cultured neonatal foreskin	Partial- and full-thickness skin ulcers resulting from venous insufficiency, and full-thickness neuropathic diabetic foot ulcers
Transcyte (Advanced Biohealing, La Jolla, California)	Fibroblasts on nylon mesh	Full-thickness and deep partial-thickness burn wounds
Biobrane (Smith and Nephew, Hull, England)	Knitted nylon and silicone	Intended for the temporary covering of clean, excised, full-thickness burn wounds until autografting
Epicel (Genzyme, Cambridge, Massachusetts)	Autologous keratinocytes on a gauze mesh	Deep dermal or full-thickness burns comprising a total body surface area ≥30%
Dermagraft (Advanced Biohealing)	Fibroblasts on a poly lactic acid mesh	Full-thickness diabetic foot ulcers
Orcel (Forticel Bioscience, New York, New York)	Epidermal keratinocytes and dermal fibroblasts on a collagen sponge	Fresh, clean split-thickness donor site wounds in burn patients
Integra (Integra Life Sciences, Plainsboro, New Jersey)	Collagen sheet with glycosaminoglycans	Artificial skin to cover burns

Source: With permission from Hom DB. New developments in wound healing relevant to facial plastic surgery. Arch Facial Plast Surg. 2008;10(6):405.

Stem Cells

Stem cells are characterized by their ability to differentiate and replicate into specific cell lines based on environmental conditions and programming.[38] Pluripotent cells may differentiate into any cell in the body. Mesenchymal stem cells (MSC) differentiate into cells of the mesenchymal line as bone marrow- or adipose-derived stem cells. Many animal studies have been performed to investigate the effect on wound healing after topical or local injection of stem cells, primarily mesenchymal or adipose-derived stem cells. Steinberg et al. noted no difference in healing of ischemic wounds.[39] A report by Rigotti et al. described subjective improvement of wound healing in 20 patients with osteoradionecrosis treated with adipose-derived stem cells.[40]

Variability in the literature regarding therapeutic use of stem cells rests on the disparity between methods to harvest and isolate stem cells. No automated system for the isolation of stem cells has received FDA approval. Aesthetic and reconstructive surgery enters a new frontier as our understanding of regenerative medicine improves, coupled with innovative techniques to isolate stem cells for clinical application.

CONCLUSION

The goal in the management of wound healing is timely healing with an acceptable scar. Prerequisite knowledge of tissue injury and repair mechanisms allows the surgeon to optimize conditions for wound healing. The advent of technological advances in cellular biology, growth factor development and stem cell application provides exciting opportunities for future research and improved wound healing. Although there are not enough clinical data to support the routine use of growth factors, further refinement and development of substances may likely change our paradigm of wound healing in the future.

Disclaimer: Dr Teti is a military service member. This work was prepared as part of her official duties.

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CHAPTER

3

Soft Tissue Instruments and Sutures

Jonathan Sonne

SOFT TISSUE INSTRUMENTATION

Introduction

To perform complex surgical procedures, a surgeon needs high-quality precision instruments. Individual surgeons will select instruments best suited for the procedure, as the wrong instruments can result in unsatisfactory surgical outcome as well as damage to the instrument. Comfort of the surgeon is important with instruments appropriately sized for the surgeon's hand. Many soft tissue procedures can be performed with four basic instruments: scalpel, scissors, forceps, and a needle holder. More flexibility in performing complex procedures can be achieved by adding skin hooks, retractors, and hemostats to provide a sufficient supply and variety of instruments. Instruments for soft tissue procedures are often borrowed from related fields, and nomenclature can be confusing with instruments bearing the name of its primary function or person who designed it or popularized use.

Surgical Blades and Scalpel Handles

The scalpel is the most commonly used tool in soft tissue surgery. A scalpel consists of a snug-fitting disposable blade on a reusable or nickel-plated handle. There are two main types of scalpel handles, the standard Bard-Parker and Beaver. Bard-Parker style blades have a slot that slides over a corresponding raised portion on the scalpel handle, locking the blade in place. Although there are a variety of Bard-Parker handles, the number 3 handle is considered the workhorse. The number 3 handle may be obtained with ruler inscribed on the side, which is useful to gauge the size of lesions and plan flaps and grafts. Being flat, the number 3 handle is easy to hold, has little tendency to slip or roll, and will not roll around on surgical tray. Round-handled blade holders with knurled surfaces are also available and are popular among surgeons for their pencil-like feel. One such handle, the Siegel handle uses the standard system blades and is popular for delicate work, having a rounded handle and a knurled gripping surface that provides increased control.

The alternative to the Bard-Parker system is the Beaver style handle, which has a different attachment system and blades that are manufactured specifically for its use. The Beaver handle is typically composed of two pieces with the end of the handle being threaded to accept a second piece known as a collet. A slot in the collet accepts the blade and the two pieces, collet and handle, are screwed together to hold the blade securely. Beaver handles are small, narrow, and typically round with a knurled surface but also may be hexagonal in cross section. Beaver handles are especially useful for fine, delicate surgery, as the instrument is small and can be rolled with the fingers.

A variety of blades are available for both the standard Bard-Parker and the Beaver systems, with blade shapes and sizes referred to by numbers. With the Bard-Parker system, the most commonly used blades for soft tissue procedures are the number 15, 10, and 11. The number 15 blade has a small rounded belly and is ideally suited for delicate work on most areas of facial skin. The sharpest part of a number 15 blade is the belly, and thus, this is the area that should be used for cutting. The number 10 blade is large, broad

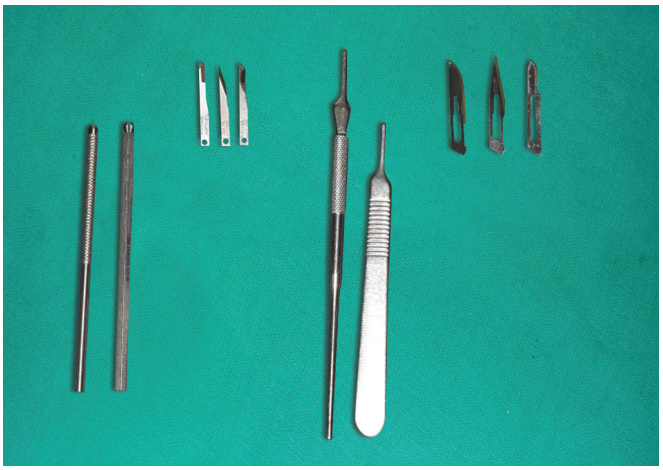

Fig. 3.1: From left to right; Rounded Beaver handle with knurled surface, hexagonal Beaver handle, 64 Beaver blade, 65 Beaver blade, 67 Beaver blade, Siegel handle, Standard number 3 handle with ruler, 10 blade, 11 blade, and 15 blade.

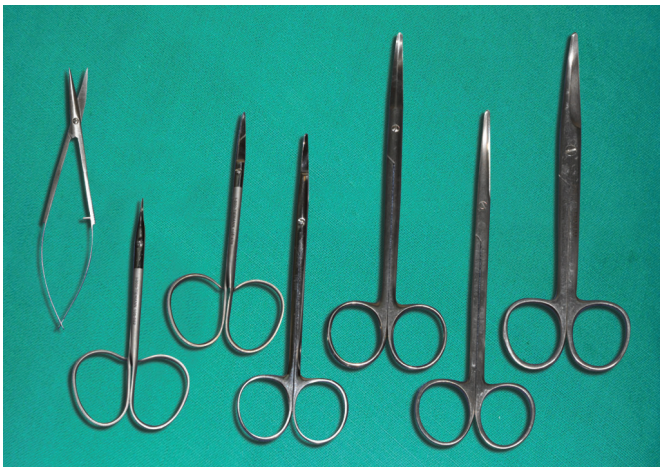

Fig. 3.2: Scissors from left to right; Wescott , Tenotomy, Iris, Ragnell, Kahn, Metzenbaum, Mayo.

and has a full, rounded convexly curved belly and a broad point with a long straight cutting edge. Like the number 15 blade, the sharpest part of the number 10 blade is also the belly. This blade is designed for larger excisional work and is too large for delicate procedures. This blade is especially useful for penetrating thicker skin, such as that found in the nuchal area or scalp. The number 11 blade is triangular with a long, narrow, sharp point. This blade is used for making stab incisions in drainage procedures, performing straight cuts on skin flaps, as well as cutting corners. The number 11 blade can be used as a microscalpel for making very tiny incisions.

Like the blades of the Bard-Parker system, Beaver blades come in a variety of sizes and shapes (Fig. 3.1). Generally, Beaver blades are smaller and sharper than their standard counterparts but tend to dull more rapidly. The number 67 Beaver blade is shaped like a smaller version of the standard number 15 blade with a gently curved belly and a sharp tip. The number 67 Beaver blade is popular for skin surgery and use on delicate tissues. The number 65 blade has a triangular configuration, similar to the standard number 11 blade. The number 64 Beaver blade has a cutting surface on a rounded tip that is used for incising in concavities such as the concha or the medial canthus.

Scissors

Scissors are used to cut and dissect tissue as well as to cut sutures and bandages. Scissors are preferable to a scalpel on flaccid tissue because they stabilize the tissue between the closing blades. Scissors are also preferable for blind dissection in areas where scalpels should be used with caution. Scissors vary in quality. German stainless steel tends to be the highest quality and most expensive, maintaining sharpness longer, resisting damage with routine use, and allowing for greatest precision. Instruments can be returned to the supplier for sharpening, maintaining tip approximation, and repair. Scissor nomenclature can be confusing as the same scissors have different names depending on the specialty using it, and the name often has little relation to the utility in soft tissue surgery.

Scissors vary in shape, size, and configuration, including long or short handles, curved or straight blades, plain or serrated blades, and blunt or sharp tips (Fig. 3.2). Each of these features has an impact on the overall utility of the instrument. Selection of scissors is based on personal preference but some basic principles apply. Long-handled instruments allow surgeons to reach under tissue for a longer distance, freeing up large flaps, or operating on areas of thicker subcutaneous tissue such as back of the neck. Short handles, on the other hand, are for fine, more superficial work. The farther the fulcrum from the hand (closer to the tip), the greater the stability and accuracy of the cut, but this benefit comes with a commensurate loss of mechanical advantage. The proper scissors should have adequate mechanical advantage to easily cut tissue at the tip. Cutting tissue at the fulcrum increases the force of the scissors but causes chewing of the tissue and unsatisfactory cutting. Curved blades allow better access to areas requiring fine dissection and provide better visualization

of the surgical field. Straight scissors are more accurate for straight cuts and have greater mechanical advantage than curved scissors. Blunt-tipped scissors cause less trauma to neural and vascular structures, making them safer when working underneath the skin, such as undermining, and give better visibility when working in deep spaces. Sharp-tipped scissors are more favorable for dissection. One serrated blade and one plain/smooth blade allows for less slippage of tissue between the blades, which can be useful when cutting thin skin without substantial subcutaneous tissue such as around the eye, or in trimming skin grafts.

Iris scissors are a generic term that defines a variety of small delicate tissue scissors. Originally intended for eyelids and ophthalmologic surgery, iris scissors have wide application in facial soft tissue surgery. Iris scissors are sharp tipped, lightweight and are manufactured in a variety of lengths, although most are approximately 4 in long. These scissors can have blades that are straight, which are useful in creating sharp corners or fashioning the tips of flaps, or curved, which improve visibility while cutting. Iris scissors can have a serrated blade to decrease tissue slippage. The Iris scissors are a workhorse instrument for fine dissection with their short handles and sharp tips. With a fulcrum situated farther back from tips, iris scissors have less mechanical advantage than Gradle or Stevens scissors. This can render them less effective for cutting thicker facial skin but allows them to have good precision with delicate tissues. Ribbon finger loops are available on Iris scissors and are felt to be more comfortable and controllable.

The (Stevens) tenotomy scissors are another delicate scissors with origins in ophthalmology. This instrument has a reverse curve on the blade that makes it accurate for small cuts. Like the Iris scissors, the tenotomy scissors have a short handle and a short, usually curved, blade. When compared with the Iris scissors, the tenotomy scissors have a more tapered tip, a more gradual curve, and are less sharp. The fulcrum is more distal, with a larger handle to blade ratio so less arc is traversed by the blade for a given finger motion. This makes the tenotomy scissors well suited for fine, delicate dissection, such as in the periorbital area. The tenotomy scissors have a slightly blunted tip; a similar instrument with sharp tips is called a Gradle scissors.

Wescott scissors are a spring-loaded, pinch-type scissors that are also used in ophthalmologic surgery. These scissors have no rings on the handles but instead are operated by spring action with the blades coming together as the arms of the instrument are squeezed. Wescott scissors have very delicate blades, which open when pressure is released. The tip is finely tapered. When held between thumb and second and third digit, they cut thin tissue with great accuracy. Because the spring opens the jaws, there is little jarring while the scissors are advanced. The spring handle allows for a light touch and good control of the tips, making it a good instrument for delicate procedures such as blepharoplasty.

Operating scissors are tissue scissors that are widely used in general surgery but less so for soft tissue surgery on the head and neck. Well-known operating scissors include the Mayo, Metzenbaum, Ragnell, and Kahn. Mayo and Metzenbaum scissors are larger, heavier instruments. The Mayo scissors have a handle-to-blade-length ratio close to 1:1 thus causing a large arc of the blade for a given finger movement, which makes is less useful for soft tissue work and better for coarser dissection. With its mechanical advantage, the Mayo scissors are popular among general surgeons and are used more as a utility instrument in soft tissue work. Metzenbaum scissors range in size starting from 5.5 in and have long handles with relatively short blades, making the handle-to-blade-length ratio of approximately 5:1. With blunt tips that are good for undermining, the curved Metzenbaum scissors are popular for face-lift surgery. With a sharp blade and heavier weight, Metzenbaum scissors are good for dissection of thicker tissue that might overwhelm a lighter instrument. Metzenbaum scissors come in both standard and small sizes, and the small version, which has a thinner and more delicate blade, is commonly referred to as a "baby" Metzenbaum. Another popular dissection scissors is the Ragnell, which range in size starting at 5 in. This versatile instrument has a blunt tip and a curved blade. The distal half of the Ragnell's blade is flattened, which provides a thin operating edge for subcutaneous dissection and ease of manipulation and control. Some scissors such as the Kahn not only have a cutting surface between the blades, but also sharpened edges on the outside of the blade for ease of dissection.

Fine, delicate scissors should not be used to cut suture material, which can dull the blades. This is especially true with fine scissors such as Iris and Wescott. Stitch scissors come in a variety of sizes and shapes. Personal preference comes to bear when selecting a scissor for this purpose. Some stitch scissors have notches at one tip to catch suture. Long handles on these scissors can give assistants better reach.