Thomas Procedures in
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Facial Soft Tissue Reconstruction

英文原版

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GREGORY BRANHAM

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To my coauthors whose contributions have enriched this text in immeasurable ways

To Regan Thomas, my mentor and fellowship director

To my wife, Cindy, and my three wonderful children,
Allison, Matthew and Grace for their love and support

To my mother, Theodocia Hearon Branham, who taught me
the value of hard work, perseverance and a good education.

PREFACE

Facial Soft Tissue Reconstruction is a multi-step process that begins with a thoughtful and detailed preoperative plan followed by meticulous execution. Attentive postoperative care followed by scar enhancement/camouflage techniques will achieve the best result. A mindset of critical analysis of results, acquisition of new knowledge and refinement of techniques is imperative to hone the surgeon's skills and provide the best possible care to patients.

Facial Soft Tissue Reconstruction is a very rewarding discipline and offers many unique opportunities and challenges. Whether dealing with a Moh's surgical defect following skin cancer or repairing a soft tissue loss from trauma, the ability to "think on your feet" and problem solve is essential. Being prepared and acquiring experience are essential to success. An understanding of the healing process and how it impacts soft tissues through such forces as contracture and scar formation allow us to plan for these as we reconstruct a defect. By understanding the vascular anatomy of the skin and the physiology of the flaps that we raise we are able to avoid complications that plagued the early reconstructive surgeons.

While there are many possibilities for the reconstruction of a soft tissue defect, not all are equivalent. Education and experience are the best teachers with regard to which technique is best for a particular patient's defect. This text provides a useful beginning to the educational portion and experience over time will guide you forward. This text is not an encyclopedic or exhaustive review of every possible flap that has ever been used, nor is it an historical review of the origins of the local flaps and soft tissue reconstructive techniques.

Rather, in this text the authors have taken a pragmatic approach to the reconstruction of these defects and as such have attempted to outline commonly used techniques that yield consistent and reproducible results. We have emphasized the techniques and procedural aspects of this discipline providing step by step illustrations of the techniques.

I hope that you will find the text helpful and that it contributes to your success as a facial soft tissue reconstructive surgeon whether you are still in residency training, beginning your practice or are a seasoned veteran in the operating room.

Gregory H. Branham, MD, FACS

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Anatomy and Physiology of the Skin

Gregory H. Branham, MD, FACS

The skin is the largest organ of the body and provides an amazing protective barrier to disease and ultraviolet radiation. The skin is exposed to ionizing radiation and insults to the DNA. Most of these insults are able to be repaired at the cellular level, but after substantial chronic exposure, some are undetected and thus unrepaired and result in a cutaneous malignancy. The mainstay of treatment for these cutaneous malignancies remains surgical excision. Once the tumor has been completely removed, definitive reconstruction can be planned and executed.

It is essential that the surgeon understand the anatomy and biologic processes occurring in the skin before reconstructing a defect. The ultimate success of any flap or graft is dependent on its ability to maintain or quickly establish a reliable blood supply. Knowledge of the vascular anatomy of the skin allows us to plan a flap that will have a reliable vascular supply. In addition, understanding the biochemical processes that occur at the cellular level in the skin following injury provides us with an understanding of flap physiology. Such understanding allows us to ameliorate the effects of injury in the newly placed flap or graft as healing begins. This chapter focuses first on the relevant skin anatomy and concludes with a basic discussion of the physiology of the skin.

Histology of the Skin

The skin is comprised of several layers and cell types **(Figure 1-1).** The epidermis is the most superficial layer and is comprised primarily of keratinocytes but also contains melanocytes, Langerhans' cells, and Merckel cells. The most superficial layer of the epidermis is nonliving tissue called the stratum corneum **(see inset in Figure 1-1).** This layer is primarily keratinocytes that are flattened and devoid of nuclei and keratin granules and provides the first layer of protection. The next deeper layer of the epidermis is the stratum granulosum. This layer is several cells thick and these cells contain keratin and keratohyalin granules within the cytoplasm. This layer terminates in the prickle cell layer or stratum spinosum. This layer derives its name from the desmosomes that attach the cells to one another and appear as spines. Below the stratum spinosum lays the basal cell layer or stratum germinativum. It is this layer that is responsible for the regeneration of the layers above. The basal cell layer is one cell layer thick and rests on the basement membrane. Melanocytes are intermixed at varying ratios.

The dermal-epidermal junction divides the epidermis from the dermis. This area, known as the basement membrane, is a highly specialized area that is responsible for the attachment of the epidermis to the underlying dermis. The dermis is comprised of the papillary and reticular dermis. The papillary dermis is a loose network of collagen, blood vessels, and fibroblasts. The fibroblasts are responsible for the production of collagen. The reticular dermis is a thicker layer comprised primarily of collagen with some elastin fibers and a few fibroblasts. Embedded in the dermis are glandular elements such as hair follicles or pilosebaceous units and eccrine and

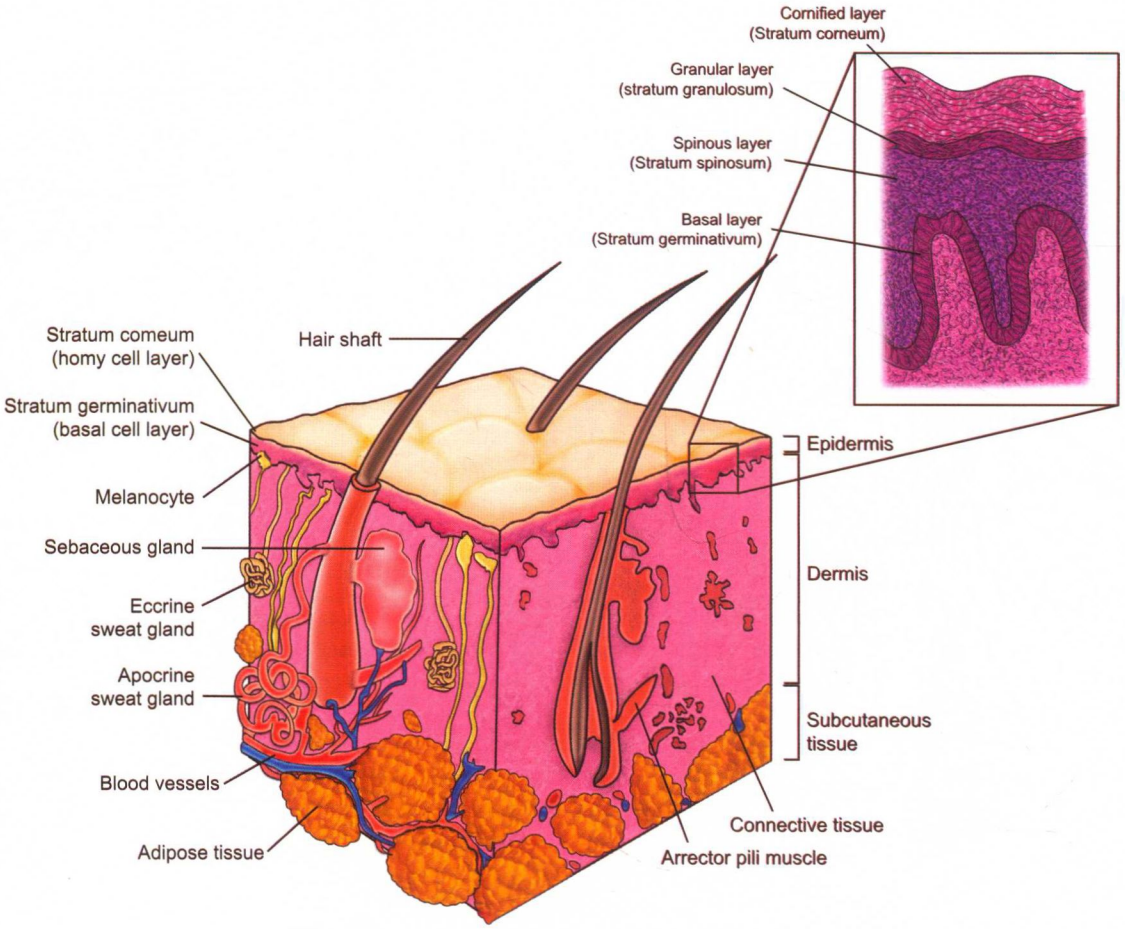

Figure 1-1. Anatomy of the skin. Inset shows layers of the epidermis.

apocrine sweat glands. These provide sources for basal cells that assist in reepithelialization following injury. Just below the dermis is the superficial fascial layer or subcutaneous layer that consists of fat and fascia.

Vascular Anatomy

Piercing the subcutaneous or superficial fascial layer are the musculocutaneous perforating vessels that arise from named arteries and terminate in the subdermal plexus (**see Figure 1-2**). The subdermal plexus is a network of arteries and veins that run along the junction of the subcutaneous fat layer and the dermis. The small vessels of the subdermal plexus pierce the dermis to terminate at the junction of the papillary and reticular dermis in an arcade known as the subpapillary plexus or the papillary dermal plexus (**see inset in Figure 1-2**). It is the subpapillary plexus that gives rise to the vertically oriented capillary loops that extend into the dermal papillae. In summary, there is a vascular arcade of the skin that is located at the subdermal layer (subdermal plexus) and another within the dermis at the junction of the papillary and reticular dermis (subpapillary plexus).

Neural Anatomy

The nerves in the skin follow the pattern of the vascular anatomy and, as such, form part of the neurovascular bundle or pedicle. Histologically, nerves in the skin are similar to those throughout the peripheral nervous system. Cutaneous nerves are sensory or autonomic, as there is no need for motor nerves at the skin level. Cranial nerves V, VII, IX, and the cervical nerves are the sensory afferent fibers of the head and neck. There is a number of specialized

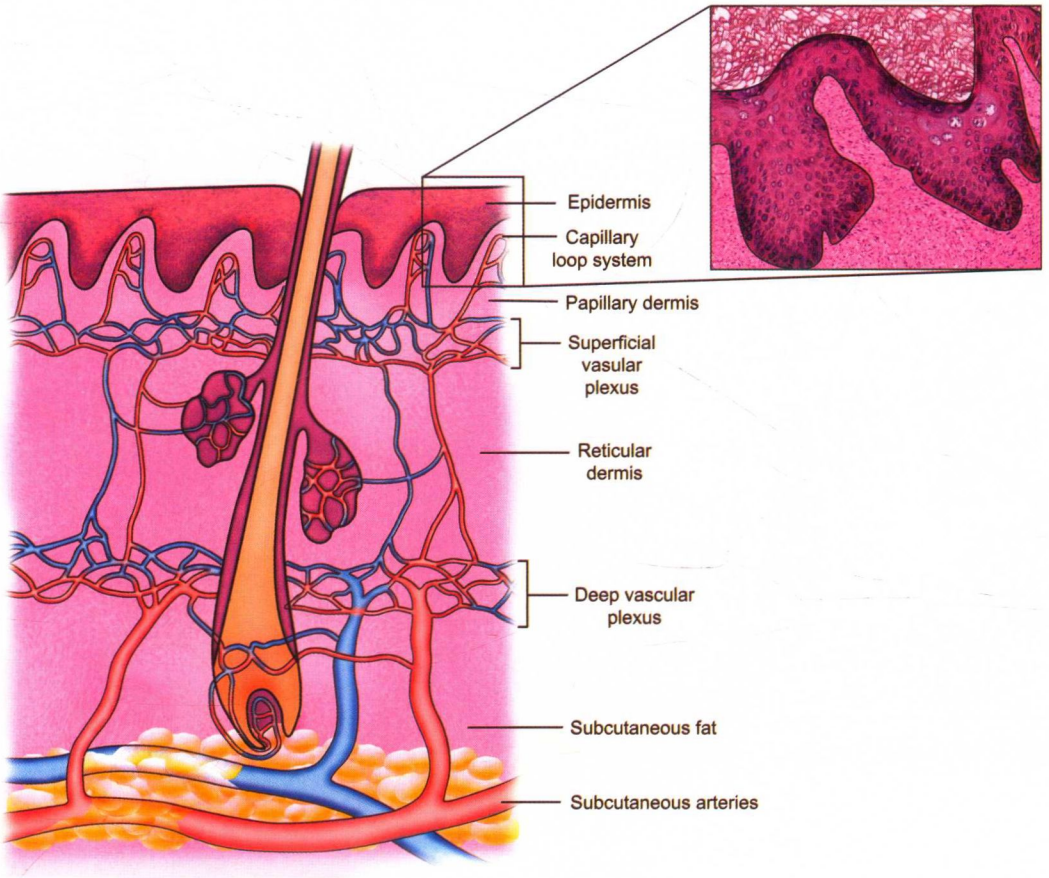

Figure 1-2. Vascular anatomy of the skin. Inset shows the papillary dermal plexus.

nerve endings located in the skin, and these are responsible for the perception of pain, temperature, proprioception, pressure, and itching (**Figure 1-3**). Meissner's corpuscles are encapsulated receptors that mediate touch and are located superficially in the dermal papillae. The Vater–Pacini corpuscles mediate pressure and are located at the fascial level. Many nonencapsulated nerve endings lie in the end organs of the skin, hair follicles, and dermal papillae. The efferent fibers are primarily autonomic in nature and innervate the smooth muscle of the blood vessels, glomus bodies, sweat glands, and the erector pili muscles that are associated with hair follicles.

Pilosebaceous Units

Most of the skin throughout the body is covered with hair. Some areas have only a fine or vellus hair, whereas other areas such as the scalp and beard in males are covered with thick hair of varying degrees of coarseness. The rate of growth of the hair varies with the area of the body. In males, some scalp follicles are programmed to respond to testosterone by becoming quiescent, resulting in male pattern baldness. Each hair follicle, whether containing a fine or a coarse hair, is contained within a pilosebaceous unit (**see Figure 1-4**). The pilosebaceous unit consists of the hair, hair follicle, and associated erector pili muscle and the sebaceous gland and sensory end organ. Pilosebaceous units are absent in mucosa and on the palms and soles. The presence of hair must be considered when designing a flap with consideration given to its ultimate location. A prime example of this would be to use a paramedian forehead flap for nasal defects. If the flap extends into the scalp, then hair bearing tissue will be transposed onto the nose. and this must be addressed.

Random Pattern Flaps

Local or random pattern flaps are the most common type of flap used in facial reconstruction for

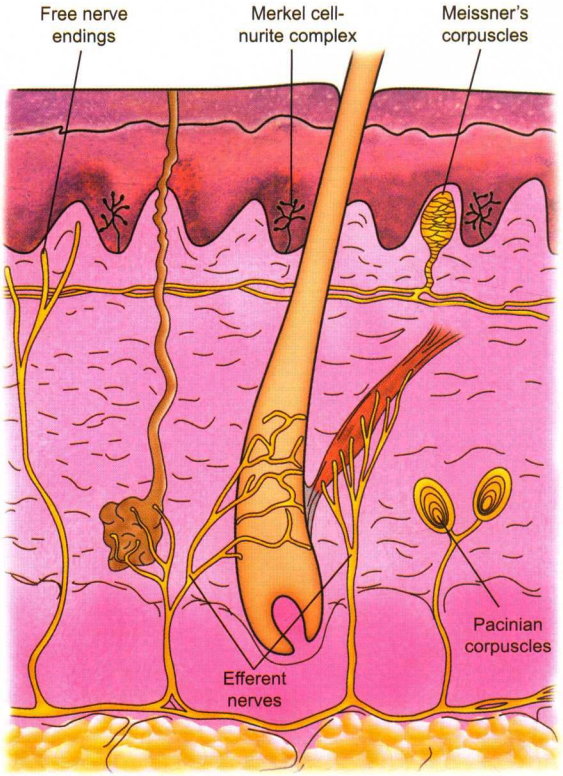

Figure 1-3. Neural anatomy of the skin with specialized cutaneous nerve endings.

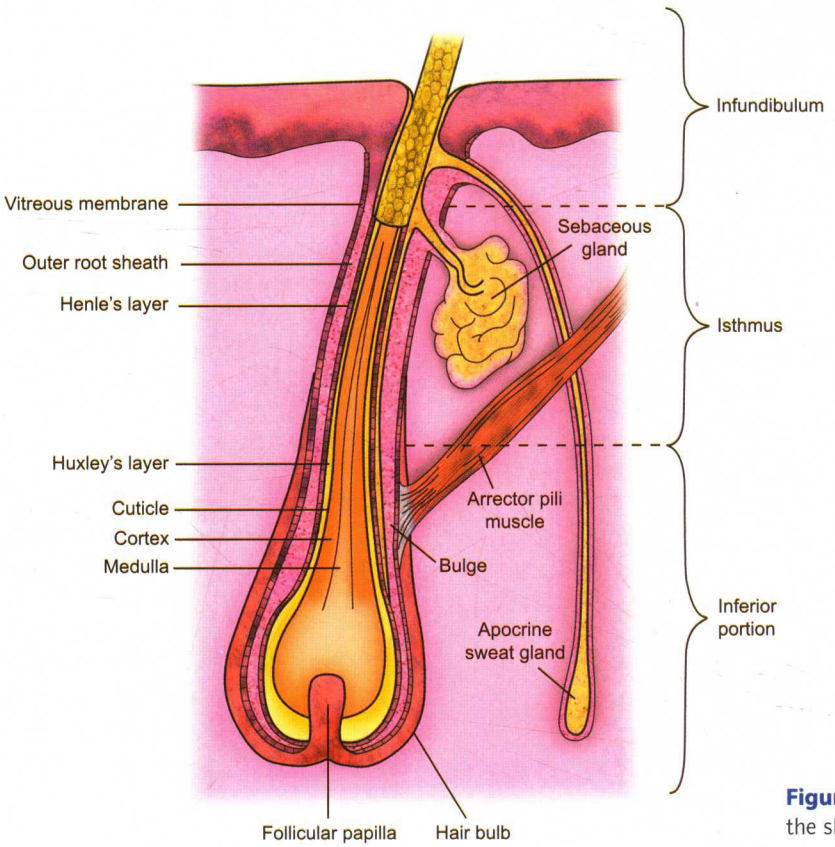

Figure 1-4. Pilosebaceous unit of the skin.

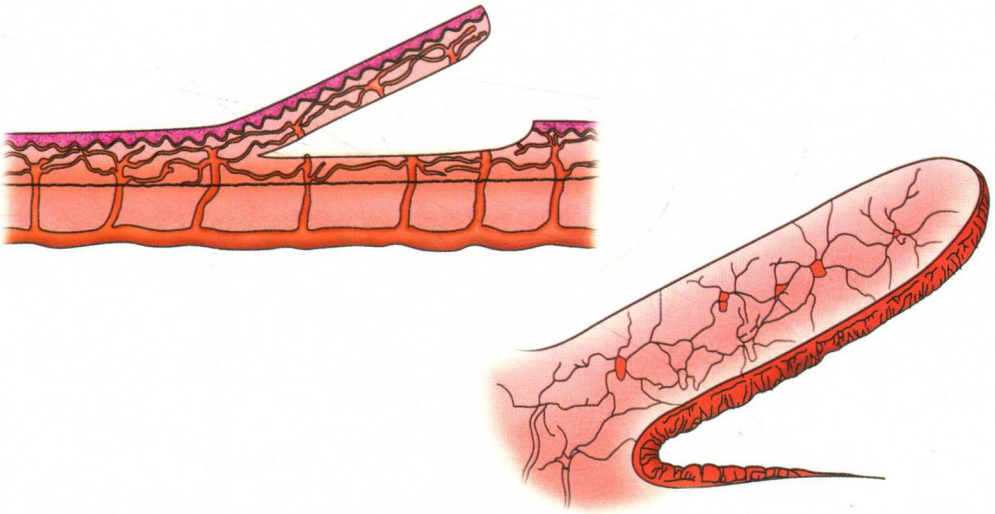

Figure 1-5. Random pattern flaps. Random pattern flaps are elevated in the subcutaneous plane and the musculocutaneous perforating vessels are divided leaving only the most proximal perforating vessels intact to supply circulation tothe flap.

small- to moderate-sized defects. Such flaps are often called local flaps because tissue that is local or adjacent to the defect is used. This local or adjacent tissue often provides a superior color/texture match. The term "random pattern" is used to describe the vascular supply to the flap. These flaps rely on the musculocutaneous perforating branches for their blood supply as well as the anastomotic connections to the subdermal and subpapillary plexi (**see Figure 1-5**). Because these flaps are raised at the level of the subcutaneous plane, the musculocutaneous perforating vessels are divided, and only the most proximal perforating vessels remain intact to supply microcirculation of the subdermal and subpapillary plexi. It was long felt that survival of the distal end of the flap was based on a ratio of flap length to flap width, with a ratio of 21 being the maximum allowable. However, whether the flap survives is dependent on the capillary filling pressure at the distal end of the flap. This pressure gradient must overcome the pressure that is exerted by any tension on the flap and the loss of pressure that occurs as the blood flows toward the distal end of the arcade and through any network of anastomosing vessels to arrive at the capillary loops in the dermal papillae. If this critical pressure is exceeded, regardless of flap length, the flap will show signs of demarcation and subsequent necrosis. This may be manifest as a superficial epidermolysis at the distal end of the flap or a full thickness loss if vascularity is sufficiently compromised.

Axial Pattern Flaps

Segmental vessels that lie deep to the muscle are usually termed "vessels" and give rise to direct cutaneous arteries (**Figure 1-6**). Flaps that are based on these direct cutaneous arteries are termed axial pattern flaps. The pedicle of the flap contains the artery and vein that supply the circulation for the flap. The artery is accompanied by several veins known as the venae comitantes. When the entire segmental artery and veins are elevated below the muscle, it is called a muculocutaneous flap. When the flap is elevated in the subcutaneous plane at the level of the direct cutaneous arteries, it is an axial pattern flap. These flaps are generally reserved for larger facial defects that require more tissue or perhaps a component that must be turned on itself as in the case of a forehead flap for nasal reconstruction. Survival of an axial pattern flap is dependent on maintaining an intact artery and vein or vascular pedicle. The distal end of the axial pattern flap is random in its vascular supply, and the same factors that affect the survival of the random pattern flap come into play here as well. Adequate venous outflow is essential to prevent venous congestion which is the most common cause of flap loss in

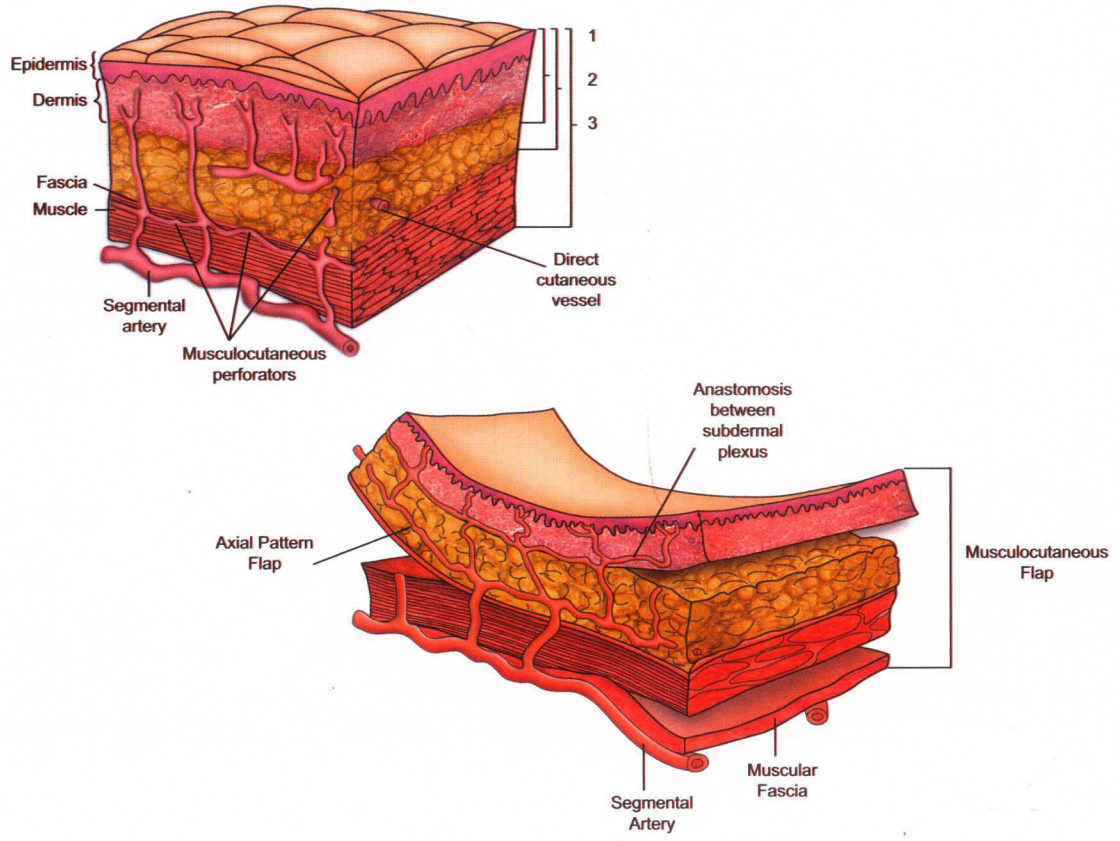

Figure 1-6. Axial pattern flaps. Axial pattern flaps are based on a segmental artery and vein that are elevated as a part of the flap known as the pedicle.

these flaps. Venous outflow is usually compromised by overskeletonizing or thinning of the pedicle with resulting injury to the venous outflow channels or venae comitantes.

Factors Affecting Flap Survival

There are a number of factors that impact flap survival. These factors occur both at the macroscopic and microscopic or cellular level and represent a balance between the perfusion and the metabolic demands of the tissues. Factors at the macroscopic level impacting viability include flap design issues and mechanical factors. At the cellular level, factors such as tissue injury response, microcirculatory regulation, and comorbid disease states greatly impact the ultimate success of the reconstruction. The principles of design of local skin flaps are covered in Chapter 4 and is not discussed in detail here. However, it is intuitive to conclude that a poorly designed flap (e.g., inattention to flap length to width) will have a poorer survival rate than a well-designed flap. Mechanical factors include such considerations as tension on the flap and may be related to a flawed design or to poor execution (e.g., inadequate undermining of the donor site). Necrosis ensues when the capillary filling pressure of the capillary loops is exceeded by the extramural pressure generated by the tension of the flap.

Tissue Injury Response

As the flap heals into its recipient site, many changes occur over time (**Figure 1-7**). These changes are well documented as a typical wound healing response; this area is covered in greater detail in Chapter 2.

Inflammatory Phase

After the tissue is injured during transposition to its new location, there are a number of factors at the cellular level that impact flap survival. Upon injury,

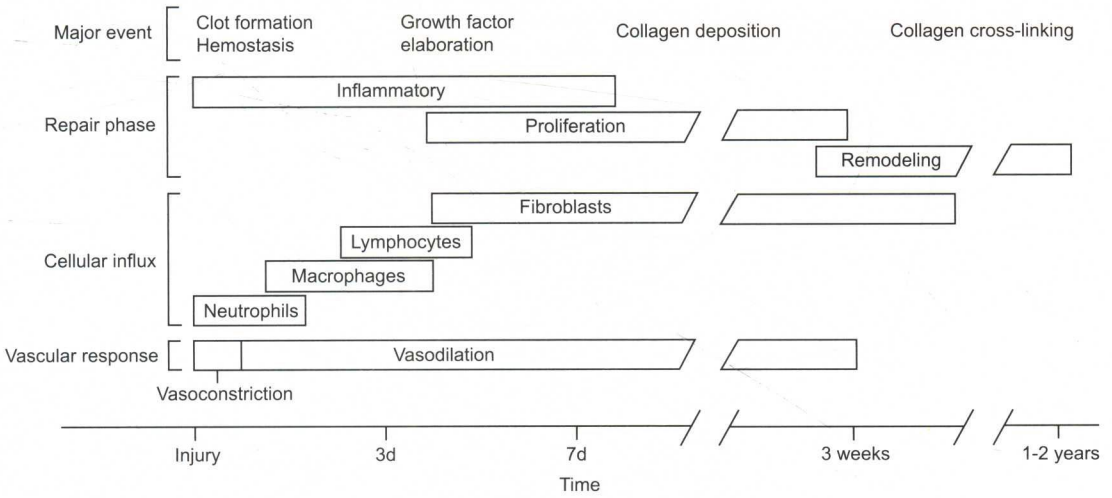

Figure 1-7. Timeline for wound healing and tissue repair. (Modified from Lorenz HP, Longaker MT: Wounds: biology, pathology, and management.)

the tissue immediately begins a vasoconstrictive response that is aimed first at controlling blood loss. During injury of the endothelial cells, a number of vasoactive compounds are released and the coagulation cascade is initiated. Platelet adhesion and aggregation follows from the initiation of the coagulation cascade. These activated platelets release a number of vasoactive compounds including serotonin, histamine, proteases, and thromboxane A2 (**Figure 1-8**).

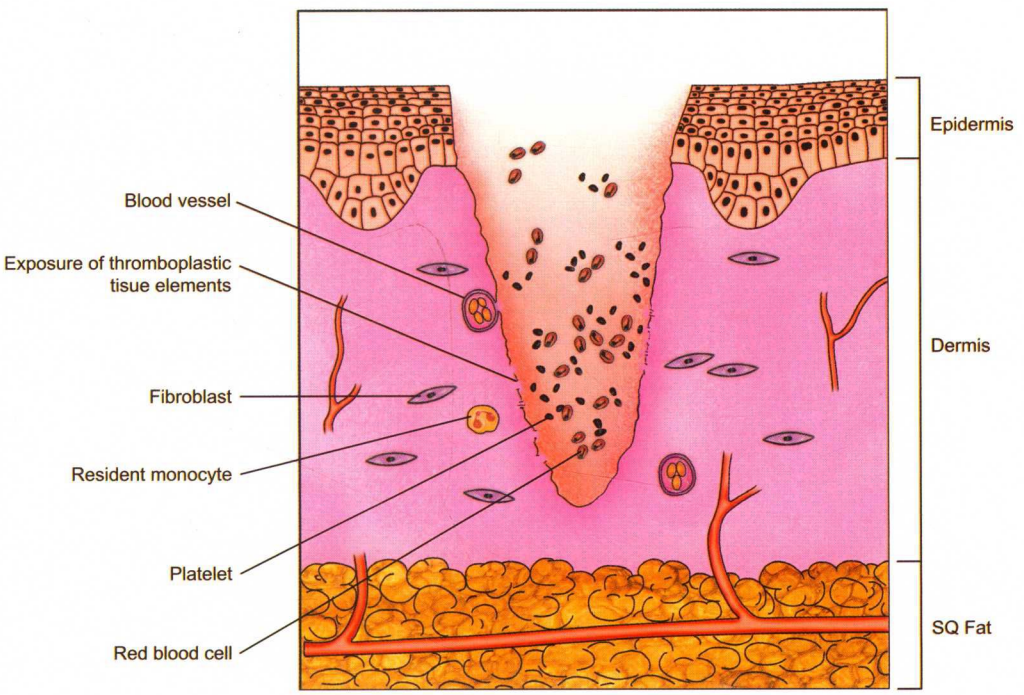

Figure 1-8. Wound-healing response initial phase. Hemostasis and release of vasoactive compounds is the initial step in the response of tissues to injury.