Skin Grafting

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To Nancy, Pat, Scot, and Kristi

Foreword

This is a practical book that will be of immense usefulness to the practicing surgeon and to the surgeon-in-training. Skin grafting is a fundamental surgical procedure, as necessary for closure of many broad wounds as suturing is for linear wounds. The ability to do it well is a *sine qua non* for the plastic surgeon, but skin grafting is also frequently used by, and essential for the patients of, other surgical specialists. Unfortunately, there has not been a major monograph published on this fundamental procedure for 20 years, so this fine new book on the subject by Drs. Rudolph, Fisher, and Ninnemann is sorely needed and doubly welcome.

There have been many developments in skin grafting during the past two decades, and you will find all the current knowledge here—concisely and clearly written, beautifully and profusely illustrated. To all who are interested in skin grafting, and nearly every surgeon should be, I commend this book as a nearly essential, at least highly desirable, part of your library.

Frank McDowell
Honolulu, Hawaii

Preface

In recent decades, numerous advances have taken place in our understanding of how skin grafts heal. We all owe a debt of gratitude to Drs. James Barrett Brown and Frank McDowell for their *Skin Grafting,* a classic text that established split skin grafting as a basic reconstructive technique throughout the world. Many skin-grafting techniques we use today remain unchanged from those first described by Drs. Brown and McDowell. But significant technical refinements have appeared, particularly in the motorized dermatome. We estimate that today a majority of skin grafts are cut with a power dermatome, and thus we have placed greater emphasis on this technique than might be found in other surgical texts. Furthermore, we now know more about how skin grafts heal, and correlation of this knowledge with improvements in techniques served as the stimulus for writing this book.

Skin grafting, like any other surgical maneuver, requires that the surgeon have an intimate knowledge of the behavior of biologic tissue in order to select the proper technique. Rather than placing biologic principles in the first half of the book and practical methods in the second half, we have combined biology and technique within each chapter. The text progresses chronologically, following the successive steps in skin grafting. We start with the biology of the wound and preparation of the bed, progress to graft selection and graft cutting, and thence to graft fixation, healing, maturation, and specialized techniques. The approaches we detail are intended to be helpful both to the experienced plastic surgeon who grafts skin frequently and to surgeons who do so only occasionally.

The techniques we describe extensively are those that we have found to be most useful, both in our hands and in those of other plastic surgeons. Alternative techniques and approaches certainly are available, and some surgeons may disagree with our choices,

particularly in methods of wound bed preparation. Such controversy is healthy and we encourage it, at the same time wishing to present the approaches that we have found most successful.

We share the frustration of many readers of medical books when they find that animal studies and human studies are hopelessly and invalidly mingled. What is true for animal skin may not always be true for human skin. Therefore, throughout the text we have separated the findings of clinical studies from those of animal studies; the term *experimental studies* flags discussions of animal experiments and their conclusions.

Special gratitude is expressed to Mr. Bud Lewis, our photographer, who devoted many hours not only to the initial photography but also to subsequent printing and editing. We are happy that Ms. Pat Viviano let us photograph her at work (Chapter 11). Ms. Julie Hernley receives our thanks for her drawings. Thanks are also due to Mr. Fred Belliveau of Little, Brown and Company for his encouragement and advice, and to Dr. David Frank for his review of the manuscript. Our patient secretaries—Mses. Karen Delaney, Nancy Ellers, Joanne Wehner, and Anne Ugartechea—all helped sort out the editorial changes that proliferated in the hands of three authors.

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Contents

1. Natural Healing of Wounds

A wound that needs a skin graft is a wound with a deficit of skin cover. We ordinarily think of wound healing in terms of an incision closed with sutures. Wounds may also have insufficient skin to permit immediate (primary) or even delayed (secondary) suture closures. Both kinds of wounds, those that can be sutured and those that need skin grafts, progress through the same phases of healing.

Phases of Wound Healing

The phases of wound healing consist of (1) the substrate or so-called lag phase, (2) the proliferative or fibroblastic phase, and (3) the remodeling or maturation phase.

Substrate (Lag) Phase

Despite popular references to a "lag" in wound healing, there is considerable physiologic activity within the wound during the first three to four days following injury. An inflammatory process develops, with dilation of blood vessels, exudation of fibrin, and accumulation of polymorphonuclear leukocytes throughout the wound [187]. Fibrin acts as a temporary adhesive and serves to hold the edges of the wound together. At the same time, epithelium migrates across the wound gap, so that within one to two days [80, 82] the strength of the wound relies on fibrin clot formation and epithelium. Because fibrin clots and epithelium have weak holding power, sutures must often be used in the beginning to maintain wound closure.

Proliferative (Fibroblastic) Phase

During the next 10 to 14 days, collagen synthesis becomes the dominant process in the wound. Fibroblasts are mobilized, their source probably being perivascular stem cells [94, 260]. The fibroblasts produce new collagen, first as a soluble tropocollagen and then as a more mature cross-linked collagen [187]. Synthesis proceeds at a rapid rate, leading to a gain in wound strength. The pace of collagen production slowly levels off, with total wound collagen reaching its peak and stabilizing by the end of the sixth week. Collagen synthesis

persists within a wound for several years and is balanced by simultaneous collagen breakdown.

Maturation (Remodeling) Phase

The third phase of wound healing, maturation or remodeling, is perhaps the most complex and least understood of all the stages of healing. As a wound matures, alterations in the surface characteristics of the wound scar accompany the gradual replacement of wound collagen. During the remodeling phase a wound gains strength largely by altering the physical weave of its collagen framework. The dynamic equilibrium between collagen degradation and synthesis within a wound may be illustrated by the example of seamen with scurvy who suffered breakdown of wounds many years old. The explanation for this biologic phenomenon is that when ascorbic acid is lacking, collagen synthesis ceases while collagen degradation continues. Thus, even old and well-healed scars broke open because of the lack of vitamin C in the seamen's diet [187].

A commonly seen example of the dynamic equilibrium between protein synthesis and protein degradation is in the healing of a recent facial wound in a young child. The parents are deeply concerned about the appearance of the reddened, raised scar, but with time it will become less conspicuous. Redness fades and the scar texture softens. The surgeon's confidence that a conspicuous wound will become less apparent after a time is based on the expectation that remodeling will occur as the final stage of wound healing.

The Wound with Skin Loss

When there is loss of skin within a wound, one may observe the three phases of wound healing that have been described. Open, fresh wounds such as burns and avulsions consist of subcutaneous fatty tissue, fascia, or muscle surrounded by a skin border. In the substrate (lag) phase a wound of this type first experiences an inflammatory reaction, with exudation of protein, local dilation of capil-

laries, and proliferation of inflammatory cells. Within a few days the wound fills with granulation tissue containing a myriad of new capillaries, fibroblasts, and newly produced primitive collagen fibrils. Such wound surfaces are termed *granulating* because in reflected light they present a cobblestone or granular appearance (Fig. 1-1). The production of a granulating wound base coincides with the fibroblastic phase of wound healing and is a response to inflammation and bacterial presence. Open wounds display additional natural processes, *wound contraction* and *epithelial migration*.

Wound Contraction

After four to five days the borders of the open wound begin to migrate toward the center [260]. A square or rectangular wound that is permitted to contract fully will result in a stellate (cruciate) scar (Fig. 1-2). Circular defects contract less efficiently than do quadrangular wounds, a fact recognized since medieval times [14, 139], yet still not explained fully.

Wound contraction may be defined as the drawing together of the edges of the wound by forces generated within that defect [260]. A clinician can determine whether or not a wound will heal by contraction by trying to move the wound edges together manually [187]. If the edges cannot be closed easily, wound contraction is unlikely to produce successful healing.

Numerous experiments have been performed to determine the source of the wound contracting force. Current interest is focused on two possible explanations: the "pull" theory and the "picture window" theory. According to advocates of the "pull" theory [1, 2, 53], the forces for wound contraction are generated throughout the substance of the wound. "Picture window" supporters [95, 264] base their beliefs on the observable expansion of a contracting wound

Fig. 1-1. Granulation tissue has a pebbly, cobblestone appearance. Healthy intact skin nearby generates a thin sheet of epithelium that spreads over granulation tissue.

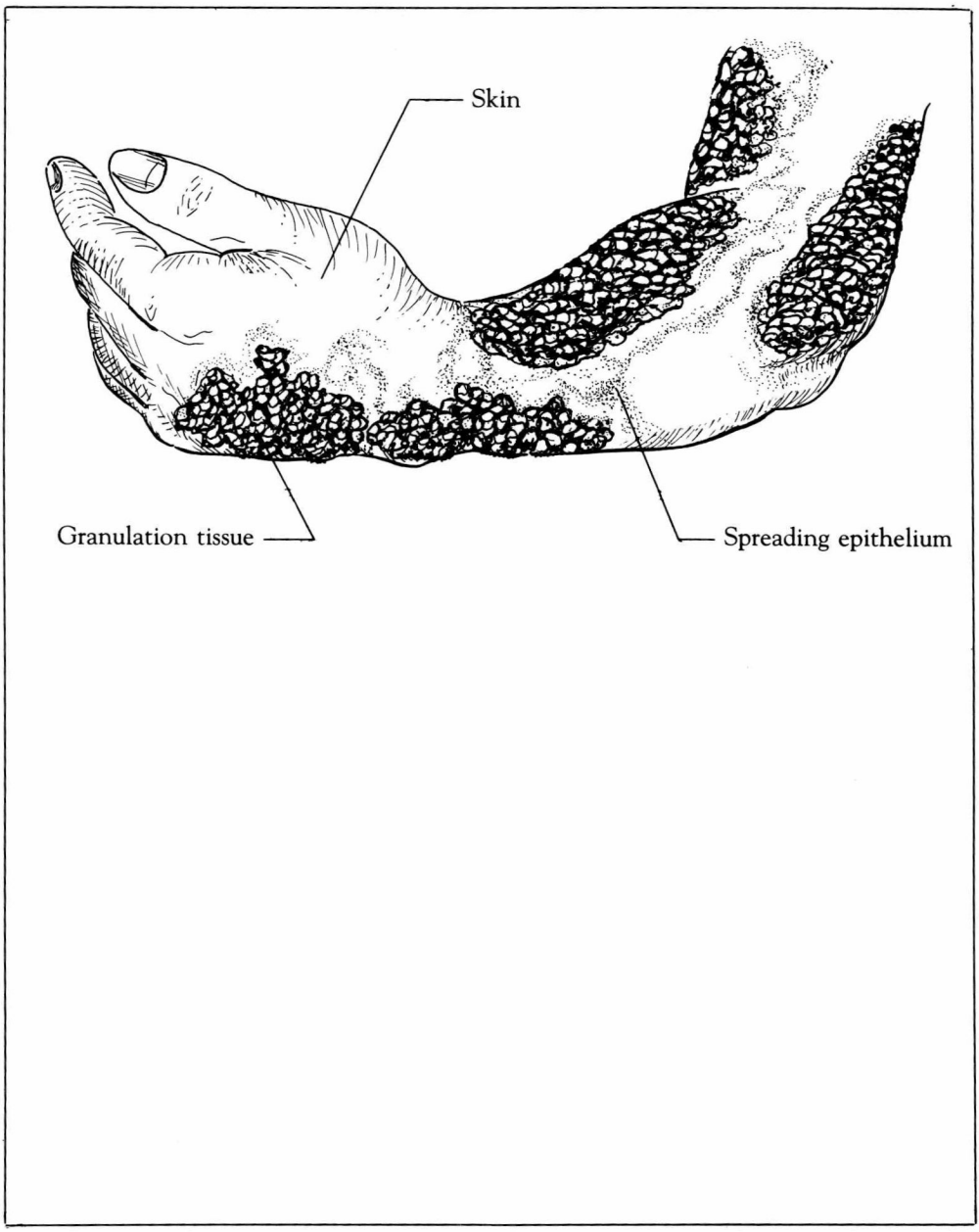
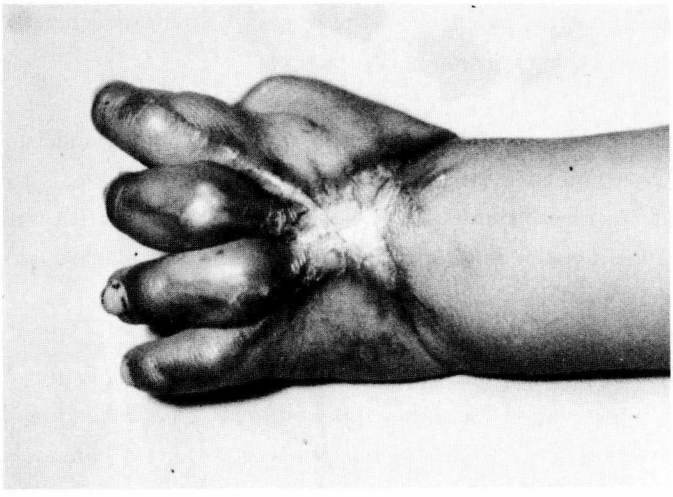


Fig. 1-2. Severe wound contracture due to untreated loss of tissue from a child's hand. Contraction forces can be sufficiently strong to dislocate joints.



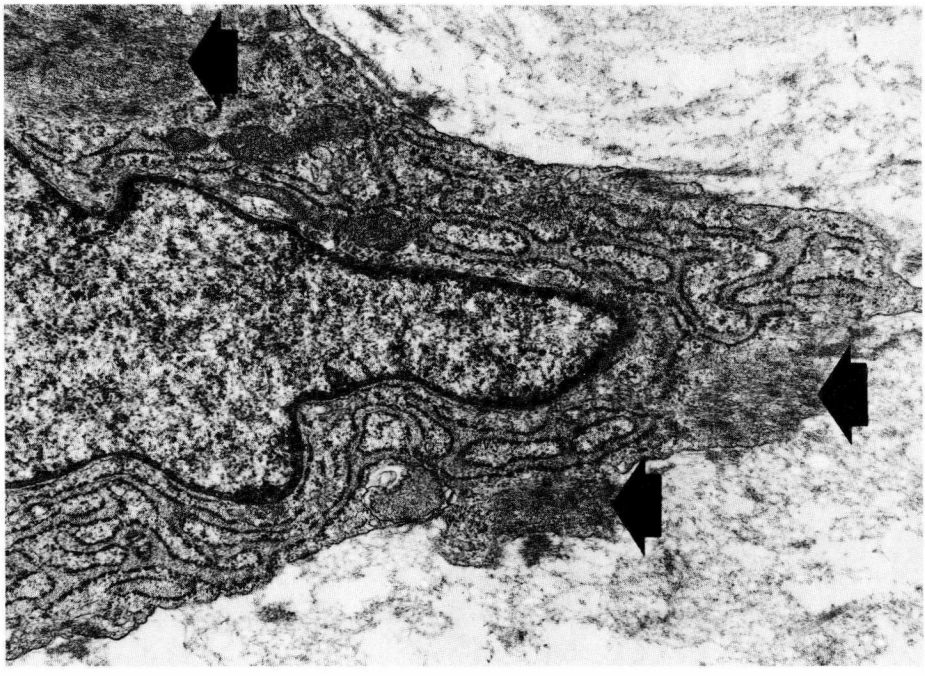
when wound margins are excised, which suggests that contraction originates at the perimeter of the wound. Current evidence favors the "pull" theory of wound contraction.

The biologic basis of wound contraction is not yet fully understood. Collagen, the main structural protein of granulation tissue, is not in itself a contractile protein [97]. Elastin, which is contractile, is not a prominent component in granulation tissue. All available evidence suggests that contraction must be *cellular* in origin.

In 1971, electron microscopy was first used to identify the cells within granulation tissue that appeared to be responsible for wound contraction [75]. Studies of the pharmacologic and immunologic characteristics of those contractile cells, which are called *myofibroblasts* (Fig. 1-3), [75, 140, 217], have shown their similarity to both fibroblasts and smooth muscle cells [75, 140, 171, 217]. Myofibroblasts are found in conditions characterized by active contraction; for example, in Dupuytren's contracture [76], in the contracting fibrous capsule around silicone breast implants [206a, 217], in hypertrophic burn scars [9], in fibrosed intrinsic hand muscles [138], and in the cirrhotic liver, as well as in many other tissues with active contraction [97]. Thus, myofibroblasts are probably the cells that cause wound contraction [75, 137, 168]. They are not permanent inhabitants of wounds and seem to disappear once wound contraction has ceased [209].

Wound contraction, a natural wound healing process, must be carefully differentiated from surface wound contracture. The term *contracture* refers specifically to an abnormal process characterized by a fixed deformity that is the result of excessive wound contraction (Fig. 1-2). Surface contractures may occur across joints or within the mobile portions of the face, and they are predominantly a cutaneous

Fig. 1-3. Electron micrograph of myofibroblast from a human contracting wound. Cell contains bundles of contractile microfilaments (*arrows*). Wound contraction is probably due to myofibroblast function. (×25,000.)



phenomenon, although analogous conditions probably can be present in other organs, such as the cirrhotic liver [97].

Epithelial Migration

The second natural process of open wound healing involves migration of epidermis (epithelium) from the cutaneous perimeter [261]. Epithelialization can be recognized clinically by the thin silver-gray margin that forms at the border of a granulating surface (Fig. 1-1). Epithelium continues to spread over a granulating surface until it meets adjacent epithelium.

Histologically, migratory epithelium is remarkably neoplastic in appearance [80, 82], although it returns to a normal appearance once epithelialization is completed. In rare instances a type of squamous cell carcinoma known as Marjolin's ulceration is found years later in wounds that healed by slow epithelialization. Migrating epithelium generates large quantities of collagenase, which might explain its invasive tendencies [65, 66, 96, 125].

Unfortunately, migrating epithelium gains no firm connection to the underlying connective tissue as it spreads across a granulating wound. Normal human skin has rete pegs, which are closely interlocked ridges of dermis and epidermis. Migrating epidermis does not regain these interlocking structures. Hence, wounds that heal by epithelialization lack resistance to shearing forces, and new epidermis may be easily torn from the underlying tissue. Such wounds are therefore unstable and often break down repeatedly.

Clinical Advantages of Natural Wound Healing

Nineteenth-century surgeons, to whom skin grafting was unknown, had no choice but to wait for open wounds to heal by contraction and epithelialization. Surgeons today can decide whether they will wait for wounds to heal by natural processes or whether they will facilitate healing surgically. Natural healing will be chosen when

skin losses are limited in extent, or when the wounds are located away from joints or in zones where scars will not be conspicuous. On occasion, the resulting scar will be no different from, or may be even better than, one achieved by grafting.

Wounds that heal by epithelialization are surface injuries that have experienced total loss of epidermis and a partial loss of dermis. Examples include traumatic abrasions, partial thickness burns, and split skin graft donor sites. Epithelial derivatives that remain within the dermis proliferate and migrate to cover the dermal surface, ultimately blending with epidermis migrating toward the center from the wound borders. Skin with abundant epidermal accessory organs, such as facial bearded zones, will heal much faster than will skin with scarce epithelial structures, such as the leg.

When epithelialization becomes apparent, the wound should be dressed with a nonadherent dressing such as Xeroform-impregnated gauze, which will allow the natural healing processes of contraction and epithelialization to proceed. No dry dressings or wet-to-dry dressings should be applied to wounds healing by epithelialization, because during their removal fragile epithelium may be torn from the surface. Nonadherent gauze (lubricated gauze) dressings are less harsh to fragile wound surfaces.

Application of external heat may accelerate epithelial proliferation [83, 261]. A 60-watt or 100-watt reading lamp held no closer than 2 feet from the wound surface three or four times a day will be beneficial to the wound in limiting moisture accumulation and in promoting epithelialization. One word of caution: Patients must be willing and able to complain if the heat becomes excessive. Therefore, heat lamps ought not be used on paraplegic patients or others who do not have cutaneous sensation. A heat lamp should never be