CHRONIC PROBLEM WOUNDS

Rudolph
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Preface

Patients with chronic problem wounds have traditionally been treated by professionals in many different fields, encompassing medicine, nursing, social work, and psychology. Even within the field of medicine, plastic surgeons, general surgeons, dermatologists, internists, orthopedic surgeons, neurosurgeons, psychiatrists, and physiatrists all get involved. In fact, people in every field of medicine or professional health care must deal with the patient with chronic problem wounds.

In part because of this diversity, much confusion about the causes and treatment of chronic problem wounds has developed. This has served as the impetus for this book—we wanted to set out a perspective and a plan of approach to problem wounds, drawing on both our knowledge of the biology of normal and abnormal wound healing, and our clinical experience. In a multiauthored book, some overlap is unavoidable and, in fact, desirable. Thus, leg ulcers are dealt with by specialists in different fields, with different perspectives on similar problems.

A common theme was needed, and this book is organized according to the time frame of how a wound should be approached clinically. Thus, the early chapters progress from basic wound healing to recognition and initial treatment, then to conservative management and support systems, and finally to definitive surgery where feasible. Because of our selection of a temporal sequence, some reiteration of information about dressing techniques and topical agents was necessary to reduce the need for constant referral to other chapters. Individual chapters on specific kinds of wounds follow this same sequence, with a brief outline of management at the end of each chapter.

We are plastic surgeons, and the book is written from the standpoint of those problems that we commonly see and treat. Thus, we have not included some chronic infection wound problems that usually can be treated and cured surgically once the diagnosis is made. We have also excluded some of the rare and exotic wounds that dermatologists and others may see. We have tried to

concentrate on wounds that we perceive as common and yet often difficult to deal with. Our goal has been to establish an overall approach to chronic problem wounds, modifying it as necessary based on the biology of the individual problem.

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R. R.
J. M. N.

From the sole of the foot even to the head, there is no soundness in it but bruises and sores and bleeding wounds; they are not pressed out, or bound up, or softened with oil.

Isaiah 1:6

Contents

1 Natural Wound Healing Processes

Ross Rudolph

Chronic problem wounds are those wounds in which simple medical or surgical treatment does not produce an easy resolution. Typically, chronic problem wounds are open skin ulcers or sinuses with loss of both epidermis and dermis. Complicating the fact that the wounds themselves are difficult to treat is the problem that patients with chronic wounds are often debilitated or have other systemic problems that inhibit both natural and surgically induced healing.

Nature has provided the body with mechanisms that close wounds [296, 333, 337]. Anyone attempting to provide optimum care for a patient with a chronic wound should be aware of the natural sequence of wound healing in wounds both with and without loss of skin, to take advantage of these natural mechanisms.

WOUNDS WITHOUT SKIN LOSS

Most studies in wound healing have been done on the classic fresh surgical or traumatic wound that has been closed by sutures. Because in some cases this type of surgical closure is either not feasible or does not work, some wounds become chronic problem wounds. Nevertheless, an understanding of the phases of natural wound healing is useful as it provides a basis for understanding the chronic problem wound [296].

Preparatory or "Lag" Phase

During the first 2 to 3 days after wounding, the wound undergoes a sequence of events that is basically inflammatory [95, 184]. Capillaries dilate and pour fluid into the wound [12]. This fluid contains fibrinogen, which converts to a thin fibrin clot that helps to hold the wound edges together. White blood cells (polymorphonuclear leukocytes) are also deposited in the wound during the inflammatory reaction. Further, vasoactive substances such as kinins and

prostaglandins are released into the wound [207, 296]. The epithelium grows rapidly across the wound surface [399], so that during this phase the wound is held together by sutures, by the fibrin glue, and by the small amount of strength contributed by the surface epithelium [133, 134].

Fibroblastic Phase

Within the next 2 weeks, the wound undergoes its most rapid gain in strength. Fibroblasts move into the wound, probably from multipotential stem cells in local small capillaries [147, 398]. These fibroblasts generate large amounts of collagen and ground substance, which make up a new fibrous connective tissue within the wound's fibrin meshwork [184, 296]. During this phase, the wound requires large amounts of nutrients. Adequate serum protein is essential. Vitamin C is needed to allow conversion of proline to hydroxyproline, a necessary event in collagen synthesis. Numerous other trace elements such as zinc are also essential. A wound in the fibroblastic phase often becomes quite red and thickened [184], owing to the presence of many active dilated capillaries and exuberant collagen deposition.

In the actively healing wound, collagen degradation occurs simultaneously with collagen synthesis [64, 65, 296]. The patient who is malnourished or who has had local tissue damage may be unable to synthesize new collagen rapidly enough to hold the wound together and counterbalance the collagen degradation [64]. The wound, rather than gaining strength, may lose strength and may even tear open, a situation known as *wound dehiscence*. This sequence can lead to a chronic problem wound in a patient who has had primary closure of a wound, which would not heal successfully in this second phase of wound healing [184].

Maturation Phase

The third phase of normal wound healing is maturation. This phase occurs in the next 6 to 12 months and involves simultaneous collagen degradation and synthesis, with the synthesis at a lower level than during the second phase of wound healing. The total collagen in the wound, as measured biochemically, remains approximately the same [296]. The surgeon who predicts that the reddened, raised, thickened, often itchy wound of the second phase of healing will gradually soften and mature takes advantage of this sequence. Thus the patient who has had a facial injury and requests plastic surgery consultation within the first few weeks is told that time must elapse to allow the wound to soften and mature as much as possible before definitive surgical correction.

Even in the third phase of wound healing, inadequate nutrition or poor circulation may lead to wound dehiscence. A classic example is found in the

book *Moby Dick*, in which sailors with scurvy had healed wounds many years old that broke open and turned into chronic wounds. In these unfortunate seamen, the lack of vitamin C in the diet led to a biochemical inability to synthesize new collagen. Yet degradation of collagen continued, and the wounds broke open to turn into chronic sores [64, 65, 296].

WOUNDS WITH LOSS OF SKIN

Wounds that have lost skin by a surgical excision or trauma, or wounds that have opened, undergo two other mechanisms of natural wound healing: *contraction* and *epithelization*.

Contraction

Contraction has been defined as the mechanism by which the edges of a wound are drawn together by forces generated within the wound (Fig. 1-1) [331, 398]. Often this mechanism is referred to as "granulating in." What actually occurs is that the open wound, first composed of fresh tissue such as fat or muscle, soon begins to fill with *granulation tissue*. Such granulation tissue is so named because in reflected light it has a pebbly, cobblestone appearance (See Plate 1). Healthy granulation tissue is bright pink. The multiple small nodules on the surface as well as the deep granulation tissue are composed of new fibroblasts, new capillaries, and new collagen. The development of granulation tissue within an open wound corresponds to the second phase of wound healing.

Excess granulation tissue may form a mound, so-called proud flesh. This excess tissue is usually edematous, gelatinous, and an unhealthy pale pink. Proud flesh can act as a mechanical barrier to both contraction and epithelization.

Within 3 to 4 days after wounding, the edges of the wound begin to be drawn together [398]. Such contraction occurs only if the surrounding tissues

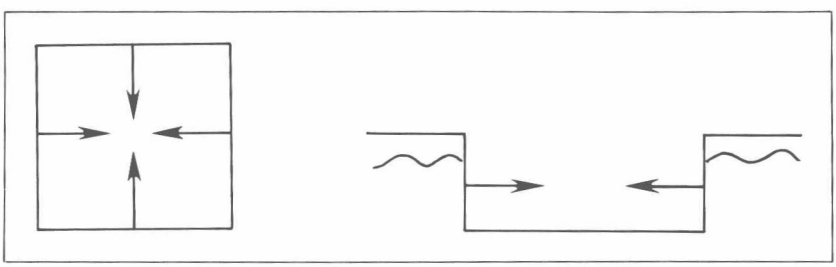

FIGURE 1-1. *Wound contraction pulls normal skin and subcutaneous tissue toward the center of the wound.*

are pliable enough to allow such movement. Thus a chronic wound surrounded by an old, fixed, rigid scar may not be able to contract, even though there is a healthy growth of granulation tissue. Wounds in certain areas of the body—the medial ankle, the pretibial region, and the forehead—cannot easily close by contraction since the tissues are so unyielding that they inhibit the forces of contraction. Chronic wounds that cannot heal by natural mechanisms often develop in these areas. The surgeon can tell if a wound will heal via the natural mechanism of wound contraction by seeing if he can oppose the wound edges without great force [296]. If he cannot bring the wound edges together, it is unlikely that wound contraction can close the wound.

The exact mechanism by which wounds contract has only recently become understood. Collagen, the main structural component of granulation tissue, is not a contractile protein [150]. The amount of elastin in granulation tissue is minimal; the contraction mechanism clearly must be a cellular one. In 1971, Gabbiani and associates described cells in granulating, contracting tissue that combined the electron microscopic appearance of fibroblasts and smooth muscle cells [123, 124, 246]. Pharmacologically, strips of granulation tissue responded to drugs with stimulation and relaxation as would strips of smooth muscle. Finally, immunofluorescent staining of granulation tissue with anti-smooth-muscle antibodies (to actin) showed that these cells had many smooth muscle characteristics. These contractile cells, known as *myofibroblasts*, have been found in a variety of tissues characterized by contraction [150, 246, 269, 331, 333, 338, 346]. Burn scars [33], the contracting fascia of Dupuytren's contracture in the hand [124], the scar tissue of hepatic cirrhosis [333], and the contracting tissues around silicone breast implants [335] all contain myofibroblasts. Myofibroblasts are found anywhere wound contraction is active and are probably the cells that cause wound contraction [242, 246, 269]. Myofibroblasts are distributed throughout contracting granulating wounds, which confirms the "pull" theory of wound contraction [2, 3, 331]. In both human and experimental animal studies, the myofibroblast population drops off once the stimulus to contraction has resolved [338].

The exact origin of the myofibroblasts is not known but is probably multipotential cells around capillaries. Tissues in which granulation tissue growth is limited by lack of blood supply or by damage such as seen after therapeutic radiation might be expected to have a smaller myofibroblast population or a slower onset of myofibroblast development. In experimental chronic wounds produced by radiation or Adriamycin, delayed onset of myofibroblast development is the apparent cause of slowed healing [339, 342, 343].

The natural mechanism of wound contraction proceeds most rapidly if the wound is clean and free of infection. Wound contraction can be slowed and even halted by infection [36] or by the presence of large amounts of necrotic tissue. Wound contraction may also be stopped by a thick adherent eschar of

dead skin or by a rigid dressing that holds the wound open [398]. Although most scabs and patches of necrotic skin will be undermined and lifted off by the contracting wound, an occasional thick, leathery eschar may resist this process and keep the wound open. Such a thick eschar should be removed surgically to speed natural wound contraction.

Epithelization

The second mechanism of natural wound healing is *epithelization*, by which epidermal structures spread across the surface of a open wound [399, 419] (Fig. 1-2). In a wound that contains epithelial remnants, such as a superficial abrasion or second-degree burn, the residual structures produce epithelium that will spread over the wound surface [85, 347, 419]. In full-thickness skin losses such as deep pressure ulcers or third-degree burns, only the epithelium at the edge of the wound is available to cover the open wound. The epithelium grows until it meets other epithelium, after which it ceases to spread. Epithelization is not merely migration of cells from nearby hypertrophic epidermis; it also involves cell mitosis and differentiation of new cells [405].

As the epithelium spreads across the underlying connective tissue, it has an almost neoplastic appearance and the ability to invade both fresh and chronic granulation tissue [133, 134]. Wounds such as burns and leg ulcers that healed 30 to 40 years earlier by slow epithelization may develop Marjolin's ulcer, an uncommon type of slow-growing squamous cell carcinoma [41, 412]. This tendency to malignancy demonstrates that epithelium may retain its invasive properties for many years. The healed scar may be an immunologically privileged site, which may allow development of an aggressive neoplasm [41].

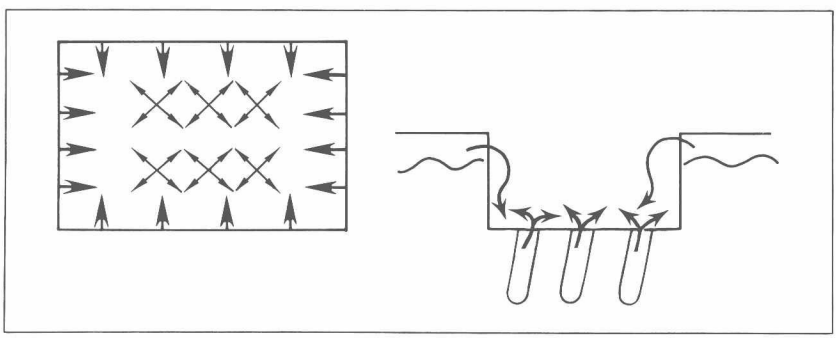

FIGURE 1-2. *Epithelization occurs by spread of epidermis from skin around the wound. Epidermal structures such as hair follicles, sweat glands, and sebaceous glands participate in epithelization if present in the wound base.*

A squamous cell carcinoma in a chronic unhealed wound can be clinically difficult to detect. Thus a biopsy should be considered in any chronically unhealed wound, especially one that is atypical or does not respond to treatment. If squamous cell carcinoma is found, aggressive treatment may be indicated. Occasionally, the biopsy may not show carcinoma, but rather an atypical pseudoepitheliomatous hyperplasia. This entity may behave in an invasive manner, and some authors [197] have recommended that it be treated like a true carcinoma. (See also Chap. 18.)

As the epithelium grows across the wound surface, there is little development of rete pegs, which normally interlock epidermis and dermis. Wounds that heal by epithelization are thus prone to peeling of the epithelium layer by trauma. Removal of adhesive tape from a recently epithelized wound may tear off much of the new epithelium.

The epithelization rate of a wound can be speeded by application of heat, as by a heat lamp [135, 399], and by increased tissue oxygenation [160, 188, 293]. By maintaining hydration, wet dressings promote more rapid epithelization than does air exposure [149, 328, 419, 420]. In experiments in rats, Salomon, Diegelmann, and Cohen [347] showed increased epidermal mitotic activity in wounds with the application of scarlet red, a commonly used topical agent. However, total wound healing time was not decreased. Clinically, covering wounds and using a gentle heat lamp are the best ways to promote epithelization of a clean wound. Infection, necrotic tissue [185], and proud flesh all decrease the speed of epithelization.

In chronic wounds in which wound contraction cannot draw the normal wound edges together, such as the ankle ulcer of chronic venous stasis, prolonged bed rest may produce healing via epithelization. Regrettably, these healed wounds are fragile, since the epithelium may be easily traumatized once the patient becomes ambulatory.

USE OF NATURAL WOUND HEALING

Natural healing processes often spontaneously heal wounds [18]. Thus, a small full-thickness burn on the thigh in an elderly lady can be treated conservatively and allowed to heal. Debridement of the burn eschar will lead to a granulating wound. This wound can then be allowed to contract and epithelize and may produce a small scar that is at least as good as would have been obtained surgically [29]. Another example is a small fingertip injury with the cut angled dorsally. In this wound, natural contraction will pull the remaining skin into good position and produce an adequately healed wound. Epithelization is essential in healing of second-degree burns, abrasions, and split-thickness skin graft donor sites.

Natural wound healing can also lead to bad results. In a large burn of the anterior neck, the process of wound contraction is detrimental since it pulls

the chin down into a distorted position. A significant loss of tissue on the back of the hand can cause significant disability since the inexorable pull of the wound contraction distorts the hand and can dislocate joints [333]. The outline at the end of this chapter lists situations in which the natural wound healing processes of contraction and epithelization may be sufficient, and those which require surgical intervention because natural healing would be unsatisfactory.

Spontaneous Slowing of Natural Wound Healing

The natural healing of open wounds does not continue indefinitely. Large open wounds such as pressure ulcers initially progress toward self-closure via contraction and epithelization. If the wound is kept clean, this self-healing may continue over the next few months to closure. Often, however, the wound contraction and epithelization will slow down and stop [54, 58]. Sometimes this can be attributed to constant pressure, infection, or a rigid eschar or dressing, but more often the cessation appears to be a spontaneous one whose etiology is not clear. Possibly as the scar in an open wound becomes more dense, its blood supply decreases to the point at which the spontaneous processes are inhibited (See Plate 2). Guttman [152] histologically studied scar tissue around chronic pressure ulcers and found arterial wall thickening with narrowing and occlusion of the vessel lumens. Such vascular constriction may decrease blood supply, leading to decreased healing.

Local blood flow can be correlated by photoplethysmography with a chronic wound's ability to heal. Lee et al. [224] identified pulsatile blood flow in ulcers that went on to heal spontaneously, whereas flat wave forms indicating poor blood flow were seen in ulcers that did not heal. This research tool may be able to predict which wounds will heal via natural wound healing mechanisms.

Knowledge of the natural healing processes may allow the physician to direct wound management in an optimal way. Such treatment requires that the wound be in its best possible situation—free of necrotic tissue and of infection.