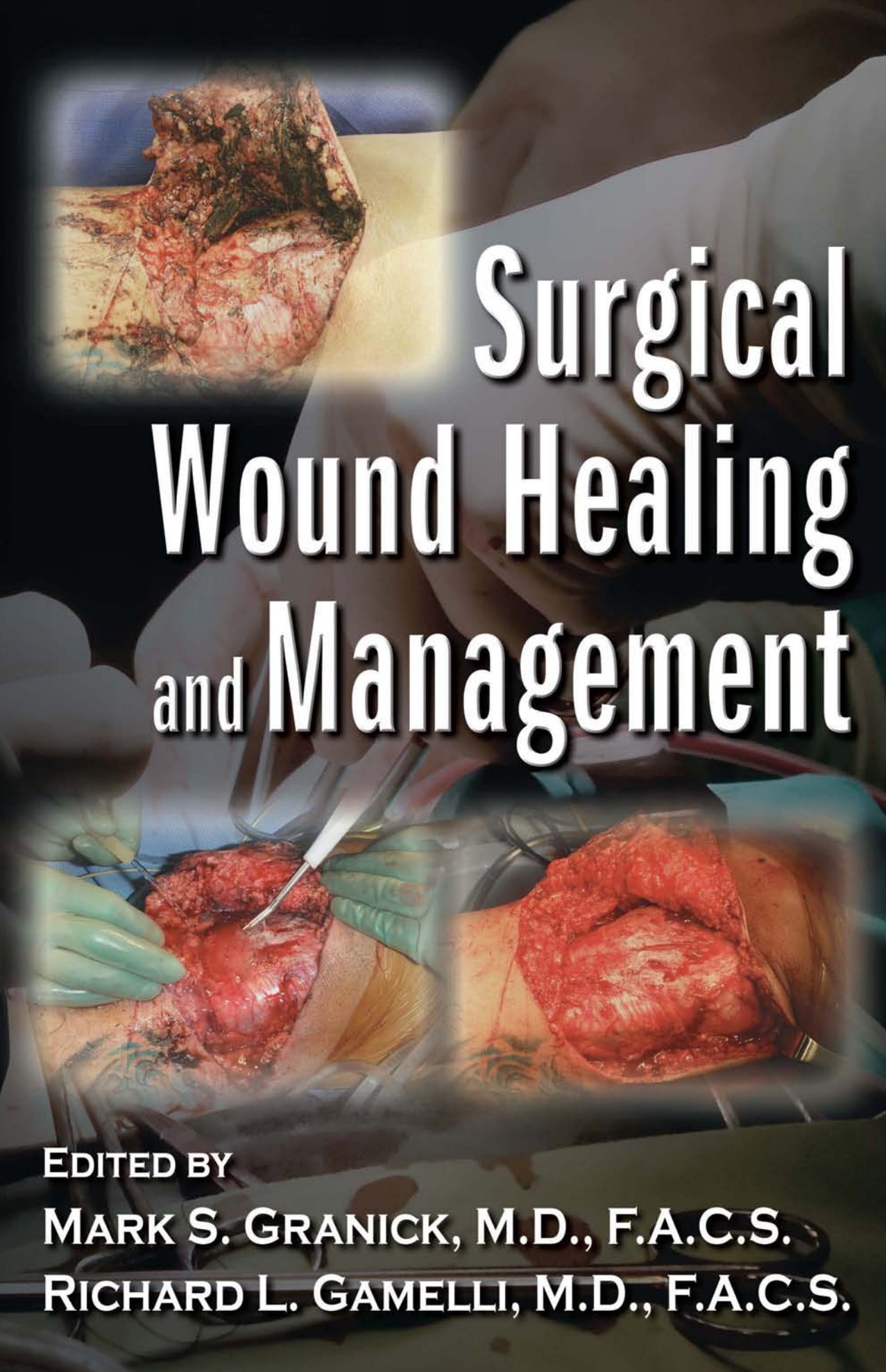

Surgical Wound Healing and Management

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Preface

Surgical wound management is largely neglected in medical textbooks. This book is an attempt by 14 senior international clinicians and scientists who represent the major surgical specialties to address this deficiency by focusing on a procedure that most surgeons had to learn for themselves during the course of their careers, namely surgical debridement. Once the wound has been appropriately debrided, it can then follow a pathway to closure that employs topical treatments or surgical reconstruction.

Surgical debridement was described by Jean Laray in 1840 to be one of the most important and significant discoveries in all of surgery, yet in the wound management community its significance is underestimated. More importantly, it is only now that we are beginning to understand the precise impact that effective surgical debridement has in minimizing wound infection and in encouraging the development of growth factors that ultimately facilitate wound healing. Furthermore, there are exciting improvements in surgical technology that facilitate wound debridement. Surgical wound debridement creates the conditions necessary for wound healing, whether reconstructive surgery or topical treatments are used to complete wound closure.

So, what is in surgical wound management? Greg Schultz, a scientist in the obstetrics/gynecology department from the University of Florida, begins by introducing the scientific and practical context behind debridement and surgical wound bed preparation. In acute wounds, an appropriately prepared surgical wound bed is required in order to successfully accomplish wound closure. In chronic wounds, surgical wound bed preparation rapidly recreates an acute wound, which can then proceed more effectively through the healing cascade. Mellick Chehade, an academic orthopedist from Adelaide, Australia and I describe the history of wound management and how the surgical community diverged from the nonsurgical wound community hundreds of years ago. We introduce a new concept in this field—a classification system of tissue type, wound personality, and debridement—that we hope will foster a new protocol on surgical debridement that all surgeons can use. Mayer Tenenhaus and Dhaval Bhavsar, plastic/burn surgeons from San Diego, California and Hans Oliver Rennekampff, a burn surgeon from Tübingen, Germany describe the nature of micro-organisms in the wound, and the role that surgical debridement plays in the control of bacteria. Richard L. Gamelli, a surgeon from Chicago, Illinois and my co-editor, describes the management of fasciitis and related wounds. Roy M. Kimble, an Australian pediatric burn surgeon, and S. L. A. Jeffery, a burn and military surgeon from the U.K., examine surgical treatment of burns. Peter V. Giannoudis and Michael Suk, orthopedic traumatologists from Leeds, U.K. and Jacksonville, Florida, respectively, describe how surgical debridement and wound management can minimize the ill-effects of traumatic wounds. Luc Téot, an internationally recognized wound expert from France, discusses debridement of surgical wounds. Giovanni Mosti, an Italian vascular surgeon, Joseph V. Boykin, a plastic surgeon from Richmond, Virginia, and Lucca Dalla Paola, an Italian endocrinologist, look at a range of chronic and otherwise infected wounds, and the contribution that surgical debridement and wound management can make to their treatment. John S. Davidson, an experienced orthopedic surgeon from the U.K., investigates debridement of infected orthopedic prostheses.

Surgical wound management, beginning with wound debridement, is the cornerstone of treatment for patients with both acute and chronic wounds. In spite of the critical role of surgery

in wound management, historical events have led to a separation between surgical and medical wound specialists, with the medical perspective widely published. This textbook seeks to overcome these differences by highlighting the role of surgery. It is time for the wound communities to integrate their practices and unite for the benefit of our patients.

Mark S. Granick
Richard L. Gamelli

Contents

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1 The Physiology of Wound Bed Preparation

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CONCEPT OF WOUND BED PREPARATION

The concept of wound bed preparation originally emerged as a result of the development of advanced wound-healing products such as exogenous growth factors and bio-engineered skin substitutes. It was recognized through careful clinical observation that chronic wounds must be properly prepared for these advanced products to be effective. This preparation included debridement of nonviable tissue and denatured extracellular matrix (ECM), control of bacterial burden and inflammation, establishing optimal moisture balance, and stimulation of epidermal cell migration at the wound edge.

Wound bed preparation eventually broadened into a basic approach to chronic wound management that aimed to "stimulate the endogenous process of wound repair without the need for advanced therapies" (1). Wound bed preparation is now established as a systematic approach for managing all types of chronic wounds, and wound care practitioners are broadening it further to adapt the principles for the management of acute wounds (2).

The development of wound care products such as bio-active wound dressings, bio-engineered skin substitutes, and exogenous growth factors was only possible through an increased understanding of the roles of cellular factors in regulating normal healing. The rationale for their development was that there was a simple molecular or cellular disorder underlying the failure of a wound to heal, and that if the wound was supplied with enough of the appropriate element, healing would take place. In fact, as we shall see, the physiology of the wound bed is far more complex than this: each element is part of an orchestrated sequence; it interacts with many other components and is only required at a specific stage in the process.

This greater understanding of the biology of normal wound healing, and a recognition of the molecular and cellular abnormalities that prevent wounds from healing has allowed wound care practitioners to move from an almost entirely empirical approach, to one based on analysis of the wound microenvironment and correction of the factors that prevent the occurrence of healing from occurring.

THE MOLECULAR AND CELLULAR PROCESSES INVOLVED IN HEALING

Most of the current understanding of wound management derives from studies of the healing process in acute wounds. Wounds caused by trauma or surgery generally progress through a healing process in which can be recognized four well-defined phases: (*i*) hemostasis (or coagulation), (*ii*) inflammation, (*iii*) repair (cell migration, proliferation, matrix repair, and epithelialization), (*iv*) and remodeling (or maturation) of the scar tissue (3). These stages overlap with the entire process and last for months (Fig. 1).

Coagulation/Hemostasis

Coagulation rapidly slows bleeding and prevents hemorrhaging from the wound but also provides to the wound surface various components that are essential for healing. Platelets aggregate at the site of injury and form a hemostatic plug. The coagulation process activates thrombin, which

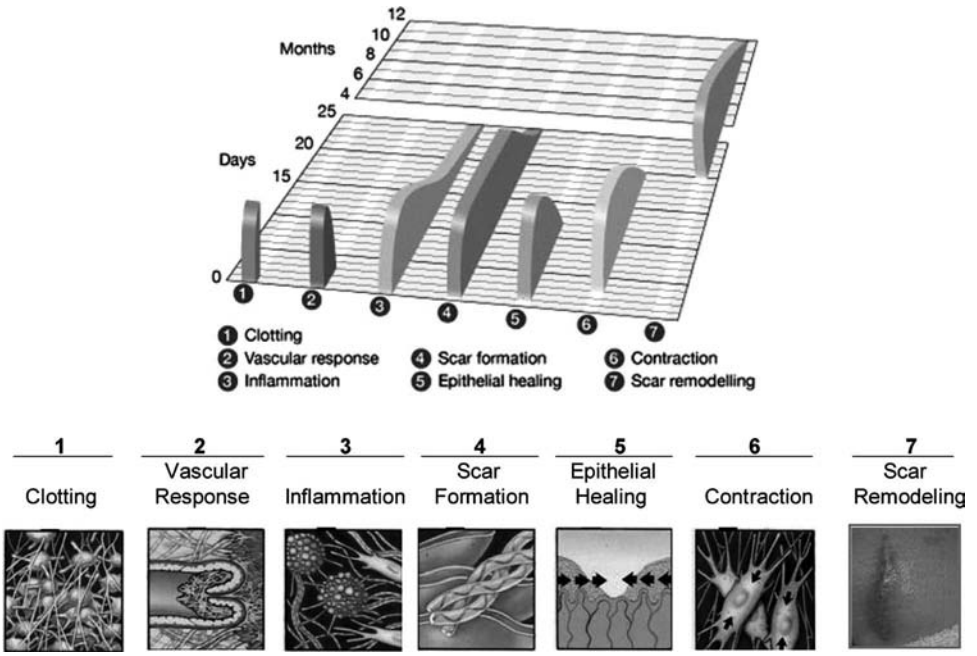

FIGURE 1
The sequence of molecular and cellular events in normal (acute) wound healing.

converts fibrinogen to fibrin, which then polymerizes to form a stable clot. The fibrin clot provides the provisional wound matrix into which the wound cells (fibroblasts, vascular endothelial cells, and epidermal cells) migrate. The aggregated platelets degranulate and release chemoattractants for inflammatory cells as well as a number of soluble proteins including platelet-derived growth factor (PDGF), insulin-like growth factor-1 (IGF-1), epidermal growth factor (EGF), fibroblast growth factor (FGF), and transforming growth factor-β (TGF-β). The function of these growth factors is to stimulate the growth and proliferation of wound cells such as keratinocytes and fibroblasts and to promote the migration into the wound of other cells such as macrophages (Table 1).

TABLE 1
Major Growth Factors and Their Function in Wound Healing

PDGF	Activates immune cells and fibroblasts
	Stimulates deposition of ECM and angiogenesis
	Stimulates synthesis of collagen, and TIMPs
	Suppresses synthesis of MMPs
IGF-1	Stimulates proliferation of keratinocytes, fibroblasts and endothelial cells
	Stimulates angiogenesis, collagen synthesis and deposition of ECM
EGF	Stimulates proliferation and migration of keratinocytes
	Stimulates deposition of ECM
FGF	Stimulates endothelial cells and proliferation and migration of keratinocytes
	Stimulates deposition of ECM
	Stimulates angiogenesis
TGF-β	Stimulates growth of fibroblasts and keratinocytes
	Stimulates TIMPs
	Suppresses synthesis of MMPs
	Stimulates deposition of ECM, particularly collagen

Abbreviations: ECM, extracellular matrix; EGF, epidermal growth factor; FGF, fibroblast growth factor; IGF, insulin-like growth factor; MMP, matrix metalloproteinases; PDGF, platelet-derived growth factor; TGF, transforming growth factor; TIMP, tissue inhibitor of matrix metalloproteinases.

Inflammatory Phase

During the inflammatory phase, initiated by blood clotting and platelet degranulation, there is vasodilation and increased capillary permeability, which give rise to the visible signs of inflammation: erythema, swelling (edema), and a rise in temperature in the injured tissue. At the molecular level, the release of growth factors from platelets is responsible for inducing vasodilatation and an increase in blood flow to the site of injury. Vascular permeability is also increased, enabling an influx of phagocytic cells (macrophages), polymorphonuclear granulocytes (neutrophils), mast cells and complement, and antibody.

Neutrophils are the first inflammatory cells to respond. Their primary role is to phagocytize and kill bacteria primarily by generating reactive oxygen molecules. They also release proteases that degrade and digest damaged components in the ECM so that ECM molecules (e.g., collagen) that are newly synthesized during the repair phase of healing can correctly interact with ECM components at the wound edge. Neutrophils also release inflammatory mediators such as tumor necrosis factor alpha (TNF-α) and interleukin-1 (IL-1) which recruit further inflammatory cells, fibroblasts, and epithelial cells.

Monocytes begin to migrate into the wound about 24 hours following injury and differentiate into tissue macrophages when exposed to the appropriate cytokines and when their integrin receptors contact the fibrin's provisional matrix. Tissue macrophages also have a major phagocytic role, and produce collagenases and elastase to break down devitalized tissue. This process is self-regulated by the production and secretion of inhibitors for these enzymes, including the tissue inhibitors of metalloproteases.

Macrophages mediate the transition from the inflammatory to proliferative phase by secreting additional growth factors and cytokines, including TNF-α, TGF-α, PDGF, IL-1 and -6, IGF-1, heparin-binding epidermal growth factor (HB-EGF), and basic FGF (bFGF) as well as TGF-β. Fibroblasts and keratinocytes drawn to the wound by these growth factors also release cytokines.

Cytokines are small polypeptides that have a range of actions essential to the wound-healing process (4). For example, the cytokines IL-1 and IL-6 stimulate the migration, proliferation, and differentiation of fibroblasts, while TNF-α stimulates the production of proteases [especially matrix metalloproteinases (MMPs)] and induces apoptosis in fibroblasts (Table 2). The significance of these will become clear in the section that follows on cell proliferation and matrix repair. Macrophages continue to stimulate inward migration of fibroblasts, epithelial cells, and vascular endothelial cells into the wound to form granulation tissue around five days after injury.

TABLE 2
The Role of Cytokines in the Wound-Healing Process

Proinflammatory cytokines	
TNF-α	Migration of PMN and apoptosis of cells
	MMP synthesis
IL-1	Fibroblast and keratinocyte chemotaxis
	MMP synthesis
IL-6	Fibroblast proliferation, protein synthesis
IL-8	Macrophage and PMN chemotaxis
	Maturation of keratinocytes
IFN-γ	Activation of macrophages and PMN
	Suppression of collagen synthesis and cross-linking
	MMP synthesis
Anti-inflammatory cytokines	
IL-4	Inhibition of TNF-α, IL-1, and IL-6 production
	Proliferation of fibroblasts
	Stimulates collagen synthesis
IL-10	Inhibition of TNF-α, IL-1, and IL-6 production
	Inhibition of macrophages and PMN

Abbreviations: IL, interleukin; IFN, interferon; MMP, matrix metalloproteinases; PMN, polymorphonuclear lymphocytes; TNF, tumor necrosis factor.

Cell Proliferation and Matrix Repair

The provisional fibrin matrix is populated with platelets and macrophages, which release growth factors that initiate activation of fibroblasts. Fibroblasts migrate into the wound using the fibrin matrix as a scaffold and proliferate until they become the most common cell type within about three to five days. As fibroblasts enter and populate the wound, they utilize MMPs to digest the provisional fibrin matrix and deposit large glycosaminoglycans (GAGs). At the same time, they deposit collagens onto the fibronectin and GAG scaffold in a disorganized fashion. Collagen types I and III are the main interstitial, fiber-forming collagens in ECM and in normal human dermis. Type III collagen and fibronectin are deposited by the fibroblasts within the first week, and later, type III collagen is replaced by type I (5). About 80% of dermal collagen is type I, which provides tensile strength to the skin (6). The collagen is cross-linked by lysyl oxidase, which is also secreted by fibroblasts. The initial scar matrix acts rather like a bridge over which the sheet of epidermal cells migrates. Once the initial layer of epithelial cells has formed, the keratinocytes proliferate and eventually form a multilayered stratified epidermis.

Cell proliferation and synthesis of new ECM increases the demand for energy in the wound, which is met by a substantial increase in vascularity of the injured area. Granulation tissue gradually builds up, consisting of a dense population of blood vessels, macrophages, and fibroblasts embedded within the loose ECM.

During the repair phase, the level of inflammatory cells in the wound decreases, and fibroblasts, endothelial cells, and keratinocytes take over the synthesis of growth factors (Table 3) to promote further cell migration, proliferation, formation of new capillaries, and synthesis of the components required for the ECM.

EPITHELIALIZATION AND REMODELING

At the edge of the wound, keratinocytes sense the ECM, proliferate, and begin to migrate from the basal membrane onto the newly formed surface. As they migrate, they become flat and elongated (7) and sometimes form long cytoplasmic extensions. At the ECM, they make contact with large fibers of type 1 collagen, attach, and migrate along them using specific integrin receptors (6). Collagenase is released from migrating keratinocytes to dissociate the cell from the dermal matrix and to allow locomotion over the provisional matrix (8). Keratinocytes also synthesize and secrete other MMPs: MMP-2 and -9, particularly when migrating (9, 10).

A simple model of this process is to think of the migratory cell putting forward an extension, which attaches to components of the provisional matrix. It then assembles and contracts its cytoskeleton and, as it moves forward, disengages itself by expressing proteases to degrade the matrix (11). These enzymes are clearly essential for the process of epithelialization, but MMPs can also interfere with the healing process if expressed at elevated levels in an uncontrolled fashion.

In the provisional wound matrix, collagen is deposited in a random orientation. As the keratinocytes migrate and settle over the provisional matrix, the process of controlled degradation, synthesis, and reorganization of molecules in the matrix normalizes the tissue structure and composition, leading to increased tensile strength and anchoring of the upper to the lower layers (12). The migrating keratinocytes do not divide until the epithelial layer is re-established. Following this, the keratinocytes and fibroblasts secrete laminin and type IV collagen to form the basement membrane and the keratinocytes then become columnar and divide to provide further layers to the epidermis.

TABLE 3
Source of Growth Factors During Cell Proliferation

Keratinocytes	TGF-β, TGF-α, IL-1
Fibroblasts	IGF-1, bFGF, TGF-β, PDGF, keratinocyte growth factor, connective tissue growth factor
Endothelial cells	bFGF, PDGF, vascular endothelial cell growth factor

Abbreviations: bFGF, basic fibroblast growth factor; IL, interleukin; PDGF, platelet-derived growth factor; TGF, transforming growth factor.

TABLE 4
Proteases Important in the Wound-Healing Process

MMP-1	Interstitial collagenase Fibroblast collagenase	Collagens: types I, II, III, VII, and X
MMP-2	72-kDa gelatinase Type IV collagenase	Collagens: types IV, V, VII, and X
MMP-3	Stromelysin-1	Collagens: types III, IV, IX, and X Gelatins: types I, III, IV, and V Fibronectin, laminin, and pro-collagenase
MMP-7	Matrilysin Uterine metalloproteinase	Gelatins: types I, III, IV, and V Casein, fibronectin, laminin, and pro-collagenase
MMP-8	Neutrophil collagenase	Collagens: types I, II, and III
MMP-9	92 kDa gelatinase Gelatinase B Type IV collagenase	Collagens: types IV and V Gelatins: types I and V α-1 protease inhibitor
MMP-10	Stromelysin-2	Collagens: types III, IV, V, IX, and X Gelatins: types I, III, and IV Fibronectin, laminin, and pro-collagenase
MMP-11	Stromelysin-3	Not determined
MMP-12	Macrophage metalloelastase	Soluble and insoluble elastin
MMP-14	Membrane type MMP-1	Pro-MMP-1, gelatin, fibronectin
MMP-15	Membrane type MMP-2	Pro-MMP-2, gelatin, fibronectin
Elastase	Neutrophil elastase	Elastin, fibronectin, laminin, TIMPs Collagens: types I, II, III, IV, VIII, IX, and XI Activates pro-collagenases, pro-gelatinases, and pro-stromelysins

Abbreviations: MMP, matrix metalloproteinases; TIMP, tissue inhibitor of matrix metalloproteinases.

This reorganization of the matrix is an important component of connective tissue repair. During this process, fibroblasts, especially myofibroblasts, in the granulation tissue attach to newly deposited collagen and contract to draw together the wound edges. This process is also regulated by proteases expressed by migrating keratinocytes at the leading edge of the epithelium and by proliferating keratinocytes lying just behind the wound edge, which restructure the basement membrane that is newly formed by the migrating keratinocytes (12).

Proteases are proteolytic enzymes that catalyze the breakdown of peptide bonds in proteins. Collagenase is just one member of a family of more than 20 MMPs. The MMPs, along with neutrophil elastase, can degrade most of the components of the ECM (13). They are secreted by neutrophils, macrophages and fibroblasts, epithelial cells, and endothelial cells. Collectively, these and other MMPs are involved in re-epithelialization, remodeling (14), and migration processes (Table 4). Proteolytic degradation of ECM is an essential part of wound repair and remodeling, but excessive levels of MMPs may degrade ECM, preventing cellular migration and attachment.

As the migrating epithelium moves forward over the initial scar matrix, it is replaced by new keratinocytes generated by proliferating keratinocytes that are located several millimeters behind the leading edge of the migrating cells. Eventually, the new epithelium stratifies and differentiates, while the provisional, randomly oriented basement membrane over which the epidermal cells have migrated is reformed to increase tensile strength. This initial remodeling process continues for several weeks after the initial wound closure and the scar may be red and raised during this period, due in part to the increased density of fibroblasts and capillaries. At the cellular level, a balance is reached between synthesis of ECM components and their degradation by proteases. Tensile strength finally reaches a maximum once the cross-linking of collagen fibrils is complete.

MOLECULAR PROCESSES IN THE NONHEALING WOUND

In nonhealing wounds, there is a failure of the injured tissue to progress through the expected phases of healing. While abnormalities can occur at any point, it is not always clear to the clinician where the abnormality has occurred. Improved understanding of the molecular pathophysiology and biology of chronic wounds enables clinicians to take a more rationale approach to wound management.

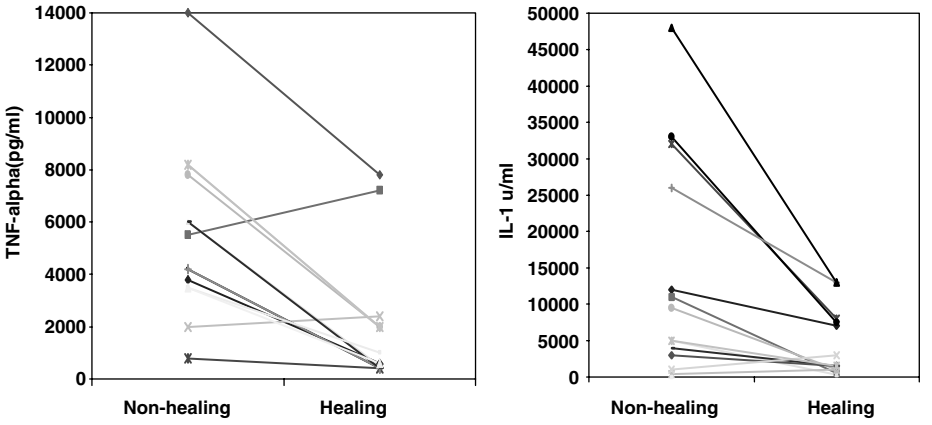

FIGURE 2
Levels of tumor necrosis factor-α and interleukin-1 as wounds progress to healing.
Source: From Ref. 15.

Trengove et al. (15) and Ulrich et al. (16) showed that the activity of TNF-α and IL-1 decreases consistently in venous ulcers as they progress from nonhealing to healing (Fig. 2). Conversely, levels of tissue inhibitors of metalloproteinase (TIMP-1) rise more than 10-fold as healing progresses (17). Thus, at a molecular level, nonhealing wounds tend to be stuck in a chronically proinflammatory cytokine status that reverses when the wounds begin to heal.

The fibroblast is a crucial component in the processes of deposition of ECM and remodeling. It deposits a collagen-rich matrix and secretes growth factors during the repair process. Any impairment to fibroblast function will therefore obstruct normal wound healing. Hehenberger et al. (18) and Loots et al. (19) observed that the proliferation of fibroblasts from chronic diabetic wounds was inhibited or disturbed. Earlier, Spanheimer (20) had observed reduced collagen production in fibroblasts from diabetic animals. It has also been seen, in vitro, that diabetic fibroblasts show a 75% reduction in their ability to migrate compared with normal fibroblasts, and also show a sevenfold reduction in production of vascular endothelial growth factor (VEGF) (21).

The traditional explanation for the failure of diabetic fibroblasts to migrate is that the cells have become unresponsive to the appropriate signals. This observation was based on studies which show that some fibroblasts in chronic wounds display phenotypic dysregulation and are therefore unresponsive to certain growth factors (22,23). One explanation is that they had become senescent (24–27). In vitro studies with fibroblasts from venous ulcers (24–26) also show that there is a decreased proliferative potential, and that there are other markers of senescence. One explanation for senescence could be that, during repeated attempts of wound repair, these cells undergo numerous cycles of replication and exhaust their replicative potential. It may also be that senescent cells are not responsive to the normal apoptosis mechanisms and cannot be easily eliminated.

However, senescence of fibroblasts does not fit all the observations. Some chronic wounds display hyperproliferation of cells at the margins, possibly because of suppression of differentiation and apoptosis within the keratinocyte and fibroblast cell populations (28). In one study, biopsies taken from the edge of chronic venous ulcers revealed that epidermal cells were in a heightened proliferative state, but the epidermal basement membrane lacked type IV basement membrane collagen, which is necessary if the epithelial cells are to attach and migrate (29).

It was initially assumed that failure to migrate was because of problems of synthesis of new cells, but these observations suggested that wound cells were present but did not have an appropriate structure over which to migrate. Attention turned to the role of proteases in wound healing.

Proteases are clearly central to the healing process. Proteolytic degradation of ECM is an essential part of wound repair and remodeling, permitting removal of damaged components, cell migration during wound re-epithelialization and revascularization, and finally, remodeling after new tissue has formed. Restructuring of the ECM is necessary to allow cells to adhere and form basement membrane. However, if the regulation of proteases is disrupted in some way,

TABLE 5
Inhibitors of Proteinases

TIMP-1	Inhibits all MMPs except MMP-14
TIMP-2	Inhibits all MMPs
TIMP-3	Inhibits all MMPs, binds pro-MMP-2 and pro-MMP-9
α1-protease inhibitor	Inhibits elastase

Abbreviations: MMP, matrix metalloproteinases; TIMP, tissue inhibitor of matrix metalloproteinases.

they may be produced to excessive levels and may corrupt the ECM, preventing migration and attachment of keratinocytes, and, eventually, destroying the newly formed tissue (30).

The activity of MMPs is partly regulated by a family of small TIMPs (Table 5). The natural inhibitor of neutrophil elastase is α1-protease inhibitor and abundant serum protein.

Successful wound healing requires a balance between proteinase and inhibitor levels in order to bring about controlled synthesis and degradation of ECM components. Ladwig et al. (31) showed that the ratio of MMP-9/TIMP-1 correlated inversely with the rate of healing of pressure ulcers (Fig. 3).

It is clear that there needs to be a coordinated expression of MMPs and TIMPs for successful re-epithelialization. Blocking key molecules of either group will prevent or delay wound healing. In addition to TIMPs, which are specific inhibitors of proteases, there are also a number of nonspecific protease inhibitors that, together, create a powerful antiprotease "shield" in the plasma and interstitial fluid to limit the activity of MMPs to the area under repair (32).

There is a substantial body of evidence that suggests that the temporal and spatial distribution of MMPs, serine proteases, and TIMPs is disrupted in nonhealing wounds.

Vaalamo et al. (33) in a study on normally healing acute wounds versus chronic venous ulcers found that the inhibitor TIMP-1 was only detectable in acute wounds. Keratinocytes bordering chronic wounds appear to express lower levels of TIMP-1 than normal; collagenase (MMP-1) was therefore able to act without regulation from its inhibitor (8). Agren et al. (34) noted that TIMP-3 expression is absent from the epidermis of chronic venous ulcers even though it is expressed at high levels in acute wounds.

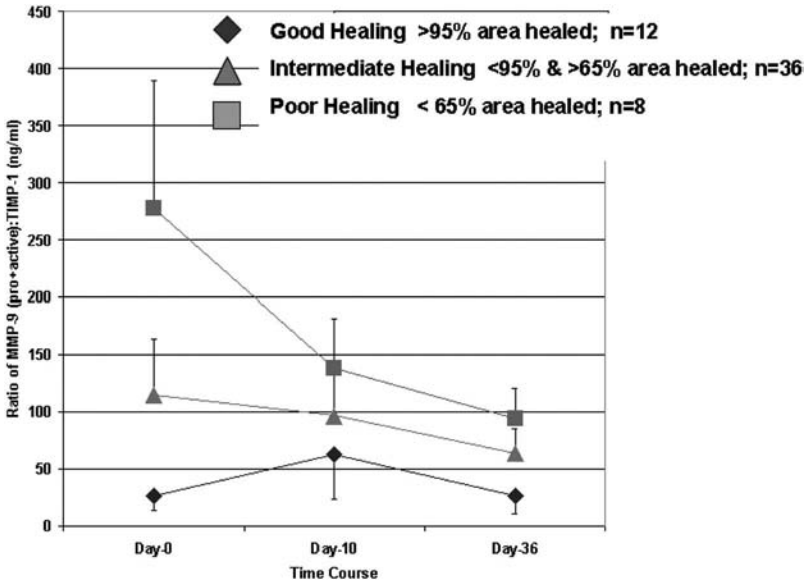

FIGURE 3
High ratio of matrix metalloproteinases-9/tissue inhibitors of metalloproteinase-1 correlates with poor healing of pressure ulcers.

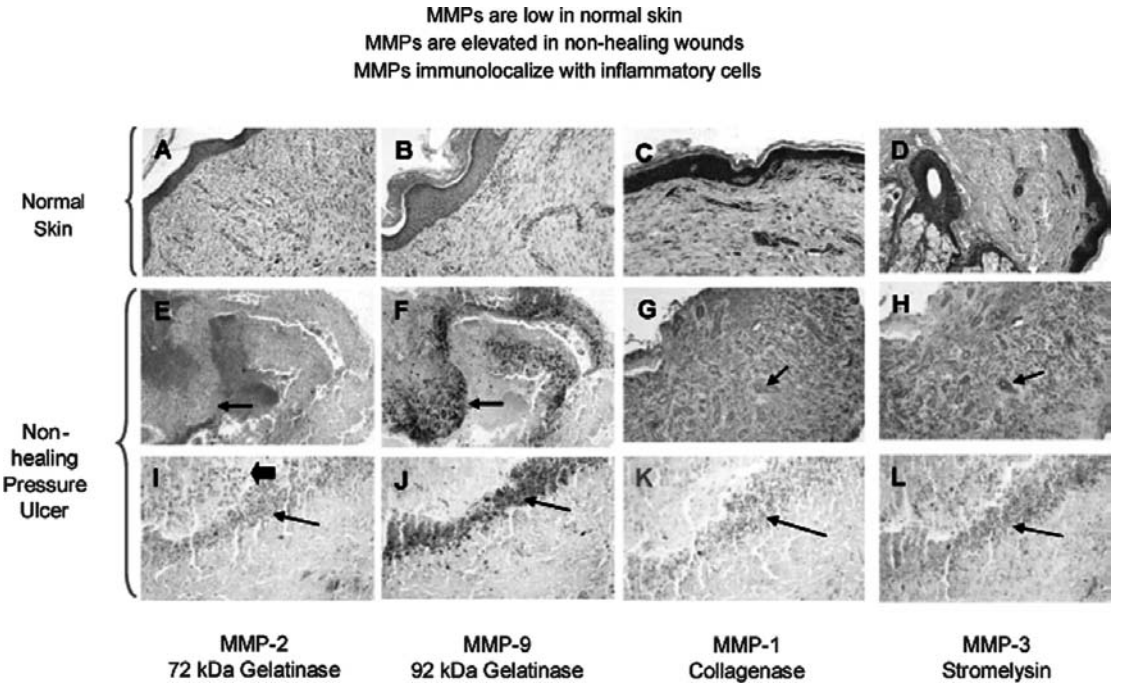

FIGURE 4
Matrix metalloproteinases in normal skin and nonhealing wounds. *Source:* From Ref. 64.

Elevated levels of MMPs in the granulation tissue of chronic pressure ulcers suggest that a highly proteolytic environment impedes healing (30). This observation is supported by a number of other studies, which show that levels of MMP-2 and -9 are higher in chronic wound fluid compared with surgical wound fluids or fluids from donor graft sites. Trengove et al. (15) reported that MMP activity was 30-fold higher in chronic wounds compared with acute. Wysocki et al. (35) found that levels of MMP-2 and -9 were higher in wound fluid from chronic leg ulcers than from acute (mastectomy) wounds. Tarnuzzer and Schultz (36) observed that levels of MMP activity in the early stages of healing were low in mastectomy fluids and did not change substantially in the seven days following surgery. In contrast, the average level of proteases in chronic wounds was 116-fold higher than in acute wounds and dropped only two weeks after the ulcers began to heal. Biopsies of chronic pressure ulcers showed that levels of MMPs were highly elevated compared with normal skin tissue (Fig. 4) (2).

Bullen et al. (17) found that TIMP levels were lower and MMP-9 levels were higher in chronic wound fluid and Yager et al. (32) showed that activity of MMP-2 and -9 in decubitus patients were 10 to 25 times those found in surgical wounds, while levels of TIMPs were lower. Nwomeh et al. (37) and Bullen et al. (17) also reported lower levels of TIMP-1 in fluid from leg and pressure ulcers than that found at peak levels in fluid from healing surgical wounds or open dermal wounds.

As is the case with chronic wounds such as venous ulcers and pressure ulcers, levels of proteases are disrupted in diabetic ulcers. Lobmann et al. (38) measured the concentrations of various MMPs and TIMPs in biopsy samples taken from diabetic foot ulcers and trauma wounds in nondiabetic patients. The concentrations of MMPs were significantly elevated in diabetic wounds compared with traumatic wounds in nondiabetics: MMP-1 (×65); MMP-2_{pro} (×3); MMP-2_{active} (×6); MMP-8 (×2); and MMP-9 (×14). At the same time, the expression of TIMP-2 in diabetic wounds was half that seen in nondiabetic lesions.

Loots et al. (39) found differences in the pattern of deposition of ECM molecules and the cellular infiltrate in diabetic wounds, compared with chronic venous ulcers and acute wounds.

TABLE 6
Levels of Proteases and Tissue Inhibitors in Acute and Chronic Wounds

Factor	Acute	Chronic	References
TIMP-1	Present	Absent	Vaalamo et al. (33)
Keratinocytes bordering chronic wounds		Express lower levels of TIMP-1	Saarialho-Kere et al. (8)
TIMP-3 in venous leg ulcers	High levels	Absent	Agren et al. (34)
MMPs in granulation tissue of pressure ulcers	Normal	High levels	Rogers et al. (30)
MMPs in wounds		30× acute levels	Trengove et al. (15)
MMP-2 and MMP-9 in wound fluid	Normal (mastectomy fluid)	Higher	Wysocki et al. (35)
Levels of protease activity	Normal (mastectomy fluid)	116× acute levels	Tarnuzzer and Schultz (36)
TIMP	Normal	Lower	Bullen et al. (17)
MMP-9	Normal	Higher	
MMP-2 and MMP-9	Normal in surgical wounds	12 to 25× in decubitus ulcers	Yager et al. (32)
TIMPs		Lower	
TIMP-1	Normal in surgical wounds	Lower in leg ulcers	Nwomeh et al. (37)
TIMP-1	Normal in dermal wounds	Higher in pressure ulcers	Bullen et al. (17)
MMPs in tissue	Low in normal tissue	Elevated in nonhealing wounds	Schultz et al. (2)
MMP-1 in diabetic foot ulcers		65× normal	Lobmann et al. (38)
MMP-2 in diabetic foot ulcers		6× normal	Lobmann et al. (38)
MMP-8 in diabetic foot ulcers		2× normal	Lobmann et al. (38)
MMP-9 in diabetic foot ulcers		14× normal	Lobmann et al. (38)
TIMP-2 in diabetic foot ulcers		Half normal levels	Lobmann et al. (38)

Abbreviations: MMP, matrix metalloproteinases; TIMP, tissue inhibitor of matrix metalloproteinases.

Extracellular matrix molecules, including fibronectin, chondroitin sulfate, and tenascin are expressed early in normal dermal wounds and reach a peak at three months before returning to prewounding levels; in chronic wounds, a prolonged presence of these molecules was noted. The chronic wounds also had a higher level of cellular infiltrates such as macrophages, B cells, and plasma cells. A summary of these observations can be seen in Table 6.

In the early stages of wound repair, neutrophil proteases participate in antimicrobial activity and in debridement of devitalized tissue. But in chronic wounds, it has been demonstrated that levels of neutrophil elastase activity are elevated (40). Elastase is very nonspecific in its actions and is capable of degrading fibronectin in the provisional matrix. The majority of proteases found in elevated levels in chronic wounds are primarily of neutrophil origin (41), including collagenase (MMP-8), gelatinase (MMP-9), neutrophil elastase, cathepsin G, and urokinase-type plasminogen activator (μPA).

Wysocki and Grinnell (42) found that fibronectin in diabetic ulcers was partially degraded and there was no fibronectin in pressure ulcer wound fluid. When intact fibronectin was added to pressure ulcer wound fluid, it was fragmented within 15 minutes (Fig. 5).

Herrick et al. (43) took sequential biopsies form the margins of venous leg ulcers during the course of healing and found that fibronectin was initially absent in the ulcer base but reappeared during healing (Fig. 6).

To summarize, all chronic wounds begin as acute wounds but fail to progress through the normal healing process and become locked in an extended inflammatory phase. In this phase, there are increased levels of proteases such as MMPs, elastase, plasmin, and thrombin, leading to deterioration of the structure of the provisional matrix and an inability of the wound cells to proliferate and migrate (Fig. 7) (44).

Specifically, there is an excess of two cytokines: TNF-α and IL-1β, high levels of a number of proteases, including MMP-2 and -9, along with correspondingly low levels of their regulators,

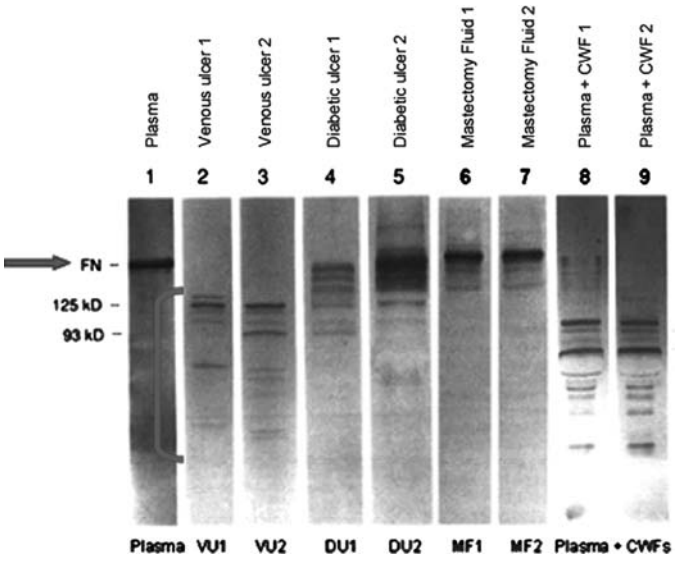

FIGURE 5
Action of chronic wound fluid on fibronectin. Fibronectin profile in plasma shows a single intact band at 250 kDa. In contrast, fibronectin is degraded to lower molecular weight fragments in venous stasis ulcers and in diabetic ulcers. *Source:* From Ref. 42.

the TIMPs which increases the ratio of proteases relative to that of their inhibitors (36). As the ECM is constantly being degraded, the tissue perceives that there is still injury and maintains the inflammatory cascade which continues to draw in neutrophils, macrophages, and other phagocytic cells. The massive influx of neutrophils release cytokines, reactive oxygen species, and inflammatory mediators, which injure host tissue in a continuous cycle (Fig. 8).

However, this begs the question: Why is this process perpetuated? What is happening at the wound bed to maintain this cycle of injury and attempted repair? There is a large body of

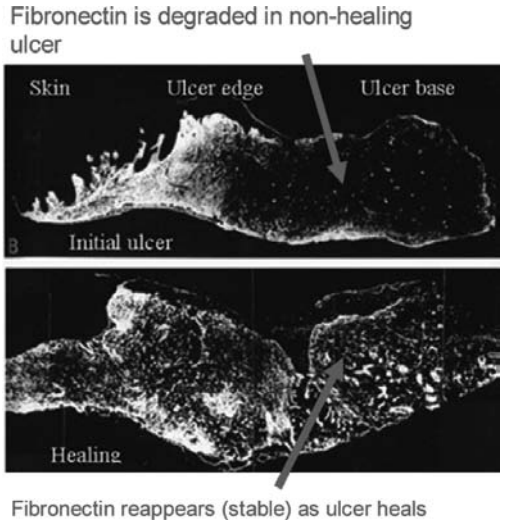

FIGURE 6
Fibronectin levels during healing. *Source:* From Ref. 43.

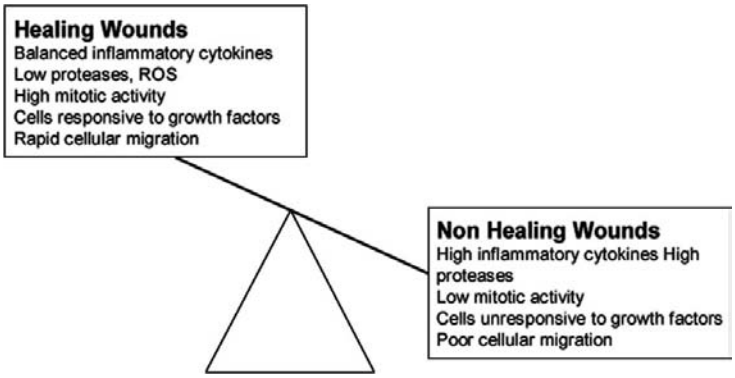

FIGURE 7
Cellular and molecular imbalance in nonhealing wounds. *Source:* From Ref. 44.

evidence to suggest that bacteria play an important role in maintaining a proinflammatory cycle in nonhealing wounds.

ROLE OF BACTERIA IN NONHEALING WOUNDS

Once a wound is created, either through surgery, trauma, or endogenous mechanisms there is a 100% probability of it being contaminated.

A number of studies have been carried out in an attempt to assess the impact of microbial load on wound healing. In 1964, Bendy et al. (45) reported that healing in decubitus ulcers was inhibited if the bacterial load was greater than 10^6 CFU/ml of wound fluid. Superficial wound swabs were used in this study but other studies, using tissue biopsy specimens, reported similar results in pressure ulcers and surgical wounds (46–48).

A substantial amount of data has shown that a bacterial load greater than 10^4 per gram of tissue is necessary to cause wound infection (49) while Elek (50) demonstrated that an average of 7.5×10^6 staphylococci is required to produce a pustule in normal human skin. Krizek et al. (1974) (51), in a study on 50 granulating wounds receiving skin grafts, showed that the average graft survival rate was 94% on wounds with a bacterial count of $<10^5$ bacteria per gram of tissue, but was only 19% when the bacterial count was above this level.

Similar data have been reported for wounds undergoing delayed closure. In an initial study on 40 wounds, a review of the bacterial counts performed at the time of delayed wound

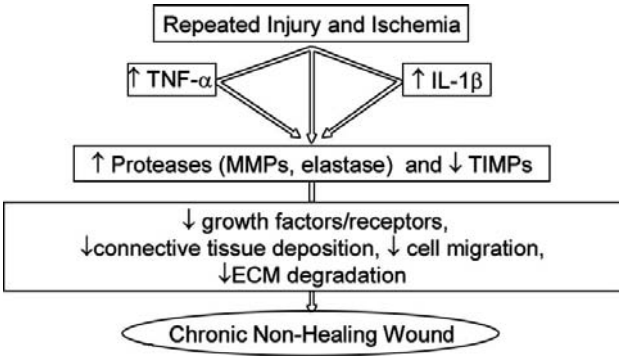

FIGURE 8
Hypothesis of chronic wound pathophysiology.

closure showed that 28 out of 30 wounds containing 10^5 or fewer bacteria per gram of tissue progressed to uncomplicated healing, whereas none of the 10 wound closures performed on wounds with a higher bacterial load were successful (48). These findings were confirmed in a later study on 93 wounds where 89 wounds with a bacterial count of $<10^5$ per gram of tissue progressed rapidly to uncomplicated healing (52).

Successful closure of pedicled flaps also depends on the bacterial load in the wound at the time of closure (53). In heavily contaminated wounds containing 10^6 bacteria per gram of tissue, the flap was not able to prevent bacterial proliferation and subsequently failed. But in minimally contaminated wounds containing up to 10^4 bacteria, both random and musculocutaneous flaps achieved wound healing and decreased the bacterial level in the wound. In an intermediate group containing 10^5 bacteria per gram of tissue, musculocutaneous flaps lowered the bacterial count and allowed wound closure, whereas the random flaps failed.

It is clear from the available data that bacteria in the wound—even in the absence of overt infection—can inhibit the normal wound-healing processes and prevent wound closure, whether it be by direct approximation, skin graft, pedicled flap, or spontaneous contraction and epithelialization.

All wounds are at risk of progressing to infection. Burn wounds and donor sites are highly susceptible for opportunistic colonization by endogenous and exogenous organisms. Surgical wounds are rarely at risk from exogenous sources of bacteria, but overwhelming evidence exists to implicate endogenous sources (49). Traumatic wounds have obvious sources of bacteria, both exogenous and endogenous. Even wounds that appear clean may harbor significant numbers of organisms. In a series of 80 emergency department wounds, 20% yielded at least 10^5 organisms per gram of tissue (54).

Time is also important. In the latter study, there was a strong correlation between the bacterial load and the time since injury. Patients with fewer than 10^2 bacteria per gram of tissue in their wounds were seen within a mean time since injury of 2.2 hours. Those who had been injured three hours previously had a bacterial load of 10^2 to 10^5 bacteria per gram of tissue, and patients who presented to the emergency department at a mean of 5.17 hours after injury had a bacterial load greater than 10^5 organisms per gram of tissue. Only those in the last group developed clinical infection that prevented primary healing.

It is not always possible to detect infection solely on the basis of clinical signs (55), particularly when the bacterial load is around 10^5 bacteria per gram of tissue. At this level of "critical colonization" bacteria replicate and prevent the wound from healing without displaying signs of frank infection. The evidence suggests that elevated MMP levels can occur in the absence of outright infection but where the bacterial load is still sufficient to stimulate an inflammatory response (15,44).

THE ROLE OF DEBRIDEMENT IN RESTORING NORMAL HEALING

In acute wounds, debridement is used to remove devitalized, damaged tissue and bacteria, and once this has been accomplished, there is a clean wound bed that is likely to heal with relative ease. Chronic wounds slowly and constantly accumulate abnormal cells that are not responsive to growth factors and impede the growth of healthier cells. Frequent maintenance debridement is therefore required to remove debris—including exudate—that may be impairing healing. While debridement is rarely required more than once in an acute wound, it is now clear that chronic wounds continue to generate a necrotic burden which requires regular removal if the wound is to heal (56).

The term "chronic" is generally used to refer to wounds that have not healed in six weeks. In some respects, all chronic wounds begin as acute wounds; however, the underlying pathology that accompanies the acute injury slows the healing process so much that other factors (infection, ischemia) begin to alter the molecular and cellular environment of the wound and healing cannot proceed. Burns can be considered chronic if scarring remains a problem for the patient. Surgical wounds can be chronic if they become infected. Debridement removes necrotic tissue that may provide nutrients for further bacterial growth and allows for a thorough investigation of the wound to remove pockets of infection.

Debridement also directly removes bacteria from the wound surface. Barret and Herndon (57) carried out quantitative bacteriological assessments of wound and biopsy samples taken from wounds that were excised 24 hours after burning and those that received delayed excision. Patients who received immediate excision had $<10^5$ bacteria per gram of tissue in biopsy samples, compared with $>10^5$ in the other group of patients. Patients in the first group suffered no infection or graft loss, compared with three in those receiving delayed excision. The pattern of colonization also differed between the two groups, with the conservatively managed group displaying a greater concentration of gram-negative species. Overall, greater bacterial colonization and higher rates of infection were correlated with topical treatment and late excision. Early excision of wounds, along with improvements in fluid resuscitation and general medical care, significantly reduced the incidence of infections following thermal injury (58,59). The pattern of infection also changed, with a difference in the organisms that were responsible for infection, and an increase in the time between injury and infection (60).

Debridement may be of benefit by removing senescent cells from the wound (61). The new granulation tissue that forms may initiate the healing cascade. Desiccated areas that may impede cellular migration can be removed and the environment of the chronic wound can be adjusted to one that more closely resembles an acute wound. With optimal support to the patient in order to maximize host defenses, debridement can be a powerful tool to kick-start the healing process.

However, even good debridement will not be effective if the patient is compromised. Adequate tissue perfusion is necessary for wounds to heal rapidly. Good blood perfusion allows oxygen, nutrients, and cells to be delivered to the wound and limits the opportunity for microorganisms to colonize. Acute wounds in otherwise healthy individuals usually have oxygen tensions of 60 to 90 mmHg whereas chronic nonhealing wounds are frequently hypoxic owing to poor blood perfusion, and oxygen tensions can be as low as 5 to 20 mmHg. Hypoxic conditions cause cell death and tissue necrosis which create ideal growing conditions for the growth of micro-organisms. Anaerobes are likely to proliferate in low oxygen tension conditions and continue to proliferate as the remaining oxygen is consumed by facultative bacteria. Arterial or venous insufficiency, trauma, blood loss, and edema all interfere with tissue perfusion and increase the likelihood of microbial proliferation.

Other chapters in this book will look in detail at the techniques and outcomes of various forms of debridement.

SUMMARY

In a chronic wound, the normal cellular processes are disrupted and the molecular and cellular environment is very different to that in an acute wound. In particular, levels of cytokines and proteases are much higher than in acute wounds, leading to degradation of the ECM and growth factors, and consequently, a failure of wound cells to migrate across the wound bed. Subinfective levels of bacteria can lock the wound in this cycle of repeated trauma and inflammation. Debridement is a highly effective method of removing bacteria and their nutrients from the wound bed, and restoring the environment to that of an acute wound.

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2 The Evolution of Surgical Wound Management: Toward a Common Language

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HISTORY OF SURGICAL DEBRIDEMENT

Wound-related issues are an increasingly prevalent problem in our aging population. There are strong economic and medical reasons for caring efficiently and effectively for wounds. Nevertheless, two parallel and divergent management systems for wound care have evolved: The medical specialists, largely driven by paramedical personnel, and the surgical specialists, populated by surgeons in numerous specialties. The medical approach consists in using a wide variety of topical dressings, oral and systemic medications, with the ultimate goal of secondary healing. The surgical approach is based on surgical intervention to prepare the wound and also to heal the wound. There is a historical basis for this paradoxical system for wound management. This chapter will review the development of wound management practices, the primary area of commonality, and will set forth a new model to support the surgical role in the process of treating wounds. Ultimately, our patients and the practice of medicine will benefit from the merging of these approaches into a more consistent and predictable algorithm.

HISTORY OF WOUND MANAGEMENT

The history of wound management is essentially the history of surgery. Prior to the mid 1800s, surgery was limited to the skin and extremities. The Egyptians as early as 2000 BC and the Greeks in 500 BC performed primarily incision and drainage procedures. Hippocrates wrote the first account of primary and secondary wound healing and defined the signs of suppuration (1). Hippocrates advocated keeping wounds dry. Galen was the most prolific of the Greek medical writers. He authored 22 volumes, 2.5 million words and two-third of his work is preserved. Galen essentially abandoned his experimental findings to emphasize his theories of wound treatment. He expostulated the theory of "laudable pus," that wounds needed to suppurate in order to heal secondarily (1,2). His writings were acquired by the Romans, the Byzantines, and the Islamics, and became the unchallenged basis of medical practice until Theodoric first dared to challenge them in AD 1266 (1). During the intervening 1200 years, virtually no progress was made in the management of wounds. It was difficult to introduce new thinking during the medieval era in the Western world. Most of the wound care was left to the clergy. However, in the twelfth century, the use of the hands for any practice other than oratory was considered undignified and the Council of Tours in AD 1163 (3) and the Tenth Lateran Council in 1215 (4) banned priests from performing surgery. The practice was confined to itinerant quacks and later taken up by barbers as they were one of the few trade people experienced with cutting implements. In 1316, Mundinus was the first to recognize that the knowledge of anatomy was required for surgery (4). Henri de Mondeville in 1306 was the first to advocate simple cleansing and primary closure of wounds (5). Almost all the literature dealing with wounds during and prior to the medieval times dealt with acute injuries. People rarely lived with chronic wounds at that time.

The three major events leading to the development of modern surgery occurred in the fifteenth century (5). First, the capture of Constantinople by the Turks. Thousands of Christian refugees poured into Italy, bringing Greek and Arabic medical texts. Second, the development of the printing press, and finally, the start of gunpowder and the subsequent deployment of high-energy weapons in war. Most of the advances in surgery came about treating ballistic and sharp wartime injuries.

Another significant event of the fifteenth century was the chartering of the Barber's Company by King Edward IV in 1462 (4,5). Barbers were now allowed to legally practice surgery. They came into conflict with the Fellowship of Surgeons, a small select group of more educated practitioners. The 100 Years War and the War of the Roses created an increased demand for surgeons. Ultimately, the barbers prevailed and were united with the surgeons in 1540 as the Union of Barber–Surgeons Company (5). This was a critical occurrence because it led surgery down the path of the tradesman at the same time as the practice of medicine was carried out independently by a group of physicians who represented the most educated people of the time. Physicians, however, left the care of the wounded and those with ulcers to the barber-surgeons. This arrangement lasted until 1745 when the barbers and the surgeons legally separated (5,6). This multicentury separation between surgery and medicine persists even today. In the United Kingdom, for instance, surgical consultants are addressed as "Mr.," whereas the medical counterparts are addressed as "Dr." More subtly, the approach to problems, such as wounds, remains at great variance between the disciplines.

During the 300 years of the barber-surgeon, there were still many important contributions to surgical wound management. Ambrose Pare is perhaps the best known barber-surgeon. He apprenticed in a Parisian barbershop before becoming an army surgeon. In 1537, he reintroduced the concept of surgical ligatures to control bleeding as opposed to the 1000-year-old concept of cautery with boiling oil (5). Although there were some significant strides forward, the barber-surgeons were a superstitious lot. John Woodall, for example, published a "Treatise on Wounds" in 1672 (6), proposing a wound salve composed of earthworms, iron oxide, pig brain, powdered mummy, etc. to treat rapier wounds. However, the salve was to be applied onto the weapon that caused the wound, not on the patient. The patient was placed in a linen dressing and left alone. These patients did better and it was attributed to the weapon salve.

Until the breakup of the Barber-Surgeon Union, surgery was a nonacademic trade job learned by apprenticeship. John Hunter introduced academic study to the discipline of surgery in the late 1700s (5,6). Prior to the introduction of anesthesia in the 1840s "all invasive surgery depended on the swift hand, sharp knife, and cool nerve of the operator" (7). Another set of three critical occurrences allowed surgery to progress into the modern era. These include the advent of anesthesia, the introduction of antisepsis by Lister, and the subsequent technologic advances in instrumentation (5,6,8).

The contemporary concept of surgical wound debridement dates back to teachings of French surgeon Pierre Joseph Desault, around 1790 (1,9–12). Desault was a military surgeon from 1789–1793, and coined the term "debridement" to describe the process of freshening the edges of a war wound, cutting away all dead appearing tissue and primary closure. Desault then became a surgeon at the Hotel Dieu in Paris, where he developed the practice of bedside rounds, morbidity and mortality reports, clinical-pathological conferences, and discharge summaries. He taught the concept of debridement to numerous students, including Dominique Jean Larrey. Larrey was a career French military surgeon for 53 years (9). He developed the concept of the ambulance and field hospitals, and recognized the importance of debridement. He wrote in his "Memoirs of a Military Surgeon" in 1814 (9,12) that debridement was "one of the most important and significant discoveries in all of surgery." The term "debridement" first entered the English lexicon in the 1842 edition of Dunglison's Medical Dictionary, and was defined as "the removal of filaments in wound of abscess which prevented the discharge of pus" (1).

The work of these French military surgeons remained largely unrecognized until World War I. Col. H. M. W. Gray described the practice of wide excision of a wound with a ½-inch margin of normal tissue, with immediate primary closure (14). Depage (9,10,14), a Belgian surgeon reintroduced the term debridement. Dean Lewis and E. H. Pool, US military surgeons,

recognized the role of debridement and introduced it in the United States at the 70th annual American Medical Association meeting in 1919 (15,16). Lewis blamed the high rate of wound-related mortality to the virulent soil in Western Europe from years of fertilization, and the new high-energy explosives. He performed a radical en bloc excision into bleeding tissue. His report was a review of his experiences, but no data was presented. Lewis projected the use of debridement into civilian life. Pool likewise recommended aggressive debridement and primary suture. Drs. Macrae and Thompson commented on these presentations lamenting the lack of training in debridement in the United States. In fact, the practice of wide surgical debridement ultimately entered into surgical training and practice in the United States and Western Europe.

WOUND BED PREPARATION

As advanced ordnance created higher-energy war wounds, as the private sector acquired high-energy weapons, and as vehicles became faster and more pervasive, surgeons have had ample opportunity to apply the lessons of war to the civilian sector. In terms of chronic wounds, people have been living longer and suffering from some of the latter stages of chronic disease. Diabetic foot ulcers were rare prior to the advent of insulin. Paraplegic patients rarely lived for long periods with pressure ulcers, and end-stage venous disease is similarly a latter-day occurrence.

During the Golden Age of Surgery, that is, the twentieth century, safe anesthesia was available, antibacterial agents and eventually antibiotics were developed, and surgical instrumentation was refined. Most of the surgical community directed attention to surgical management of wounds with debridement as the primary surgery. Plastic surgeons created complex flaps, grafts, and other closure techniques. Microsurgery became clinically available in the 1980s. It became imperative to obtain a clean wound prior to attempting closure. Failure to obtain a clean surgical wound prior to closure inevitably led to postoperative infection, flap breakdown, or late recurrence of infection. This concept is thoroughly engrained into the surgical psyche. In essence, the surgeons were engaging in surgical wound bed preparation.

The medical wound management community on the other hand has been concentrating its care of wounds around a variety of topical modalities for the treatment of chronic wounds. Debridement can also be accomplished with topical therapies (17). Options include mechanical, autolytic, enzymatic, and biologic treatments. Each of these has its advantages and its role, but all of them have significant limitations. Mechanical debridement, such as "wet to dry" dressings, is essentially the process of ripping unhealthy tissue off of a wound. It is painful, nonspecific, and results in potentially worse postoperative scarring. Autolytic debridement capitalizes on the body's inherent ability to digest and rid itself of necrotic tissue. This process can be promoted with hydrocolloid dressings. However, it remains fairly nonspecific and uncontrolled. The potential of invasive infection is a risk. Industry has widely promoted enzymatic treatment of wound surfaces using papain/urea or collagenase-based enzymes. Enzymatic debridement is effective for minimal necrotic loads, but is labor intensive and very slow. Biologic therapy involves the placement of blowfly maggots onto a wound. The larvae digest necrotic tissue and spare living tissue. This is a very effective therapy, but it carries a marked lack of acceptance with patients and nursing staff. Surgery remains the gold standard of debridement. Yet, surgical intervention is barely mentioned in recent medically oriented wound management texts (18,19).

The topical treatment of wounds has undergone some exciting advances during the last several decades. The concept of moist wound healing has facilitated chronic wound management (20,21), and led to the development of numerous dressing materials to control surface moisture, such as films, hydrogels, foams, and alginates. The wound-dressing market is saturated with thousands of products. With the emergence of advanced therapies, such as growth factors and living skin equivalents, a standard was required as to when these expensive, but effective, products should be used. The notion of "wound bed preparation" accounted for the process of obtaining a wound suitable for the application of these products (22,23). An International Advisory Board was established to find an algorithm to describe this process. The

"TIME" acronym is the result. It breaks down the wound bed preparation process into its components in a reproducible way (24–28). The "T" refers to the removal of nonviable tissue and unhealthy tissue. "I" refers to the control of infection and reduction of bacterial load. "M" is the maintenance of moisture balance. The wound should be kept moist, but not overly exudative. The "E" deals with the advancing wound edge.

Just as advances in topical wound therapy brought about a review of the treatment process in the form of an International Advisory Board on Wound Bed Preparation, advances in surgical instrumentation brought a similar effort. The introduction of hydrodissection in 2002 spurred an international effort to re-evaluate the need for and the role of surgery in the debridement process. The Versajet™ (Smith & Nephew, Largo, FL) is a high-powered parallel cutting waterjet (29). This instrument affords the surgeon with heightened control and precision, allowing a tangential excision of any wound, not just a burn wound. This approach is a paradigm shift for surgeons. In order to sort out the role of this new technology and to review the role of surgery in wound management an International Advisory Board of Surgical Wound Management was convened in 2005. Participants represent the following surgical disciplines: plastic, orthopedic, vascular, general, burn, and trauma. This organization is dedicated to education and service with regard to the role of surgery in wound care. At a symposium presented at the Wound Healing Society 2005 (28), an attempt was made to use the TIME concept to create a common language between surgical- and medical-wound specialists. Optimal wound management for our patients lies in an interdisciplinary effort to understand the methodology of our colleagues and to utilize each other's expertise.

WOUND PERSONALITY

There has been an explosion in recent years in the research and development of products and approaches to wound healing. The clinician is now left with a huge range of options to assist in the management of wound-healing problems. For many, this has only created greater confusion. Before these new options can be used to full advantage, a clear understanding of both wound physiology and pathophysiology is required (17,30,31). Chapter 1 covers this topic in great detail. The commonly described three phases of wound healing (32)—inflammation, proliferation, and maturation—depend on a complex interplay of inflammatory mediators, nitric oxide, and cellular elements. The hemostasis that follows the injury leads to the release of cytokines and growth factors that initiate the inflammatory phase and ultimate healing of the wound. Debridement should therefore aim to create the "ideally injured tissue," whereby the wound healing cascade is optimized.

There are many wound types and etiologies which are managed by a variety of professionals and methodologies, but they all share a common goal—getting the wound to heal. Where necrotic, soiled, or infected tissues exist, some type of debridement is nearly always required. Debridement of a wound can be defined as the removal of necrotic tissue, foreign matter, and bacteria from an acute or chronic wound. Surgical debridement is very often the quickest and most appropriate means of achieving this objective.

Interestingly, even though we all "know" the benefits of debridement, there is actually very little in the way of "proof" from randomized control studies. When a surgeon reports that "the wound was debrided," we rarely know how the wound was debrided. How much tissue was removed? What was the quality of the tissue left behind? How much of the remaining tissue was still "compromised," and what type of tissue was it?

The training of surgeons from different countries and in different disciplines can vary significantly, yet they all perform "surgical debridement." Tools may include a sharp scalpel, scissors, curettes, rongeurs, burrs, rasps, saws, saline wash and a syringe (with or without a needle), pulsatile lavage (low or high pressure), the Versajet hydro-dissector (33–38) (with variable water-cutting pressures), or any combination of the aforementioned. Hemostasis may have been achieved with local pressure, a coagulant, or diathermy (bipolar or monopolar). Without some accurate description of the tissue being debrided and the tools used to perform the debridement, assessing the role and relative merits of different debridement strategies is practically impossible.

Advances in wound debridement and therefore wound healing can only occur through the systematic and critical evaluation of our practice. This requires a clear description of both how we

TABLE 1
Factors Determining Wound "Personality"

Patient	Surgeon	Environment	Wound
Age	Training/specialty	Facilities	Acute vs. chronic
Financial	Experience	Equipment	Crush
Social	Preference	Dressings	Penetrating
Emotional		Staff	Burn—thermal, electrical, friction, chemical
Reliability		Contaminants	
Intellectual			Cold injury
Medical: associated disease			Infection
■ Hematological			Vascular
■ Coagulopathy			■ Arterial
■ Sickle cell			■ Venous
■ Thrombocytosis			■ Microvascular
■ Cryoglobulinemia			Radiation
■ Hyperglycemia			Neoplasia
Race			
Smoking			
Nutrition			

debride and what we debride. We often talk about the "experienced debrider" and how "you cannot teach somebody how to debride" and "it only comes with years of experience." Although there is no doubt that practice improves skills, difficulties in communicating debridement techniques undoubtedly hinders the speed at which skills can be taught. This is yet another reason to develop a clear and common language with respect to surgical debridement.

In the ideal situation, following debridement, all the remaining tissue will be like the normal prewound tissue. Soft tissue will be supple and well-perfused. Bone will be hard, white, and bleed from its ends. In the ideal situation, all abnormal tissue can be removed. In many cases, however, this complete or radical debridement approach may result in sacrificing salvageable critical tissue. In many cases, even the "normal" tissue is compromised by some systemic or local process, such as diabetes, venous stasis, arterial vascular deficiency, smoking, or infection. However, not all physiologically compromised wounds are destined for failure and further necrosis.

It is the role of the practitioner to decide when to attempt tissue salvage and to implement strategies that will optimize the chances of success. This can only be achieved after a careful assessment of the wound "personality." This is a concept frequently referred to in the orthopedic literature when one refers to the "personality of the fracture," and was first used in reference to fractures of the tibia by Nicoll in 1964 (39). It is equally applicable to soft-tissue wounds, and in addition to the wound, takes into consideration factors related to the patient, the surgeon, and the environment. The list in Table 1 is comprehensive, but by no means exhaustive, and merely serves to illustrate the many variables that should be considered and might influence wound-management decisions.

SURGICAL DEBRIDEMENT CLASSIFICATION

An important consideration in the advancement of the art of surgical wound debridement is the need for language to accurately describe and communicate the process amongst clinicians. The word "debridement" is used very loosely and without qualification or quantification. A classification of wound debridement is required that is meaningful and has application to all surgical wounds, regardless of causality. It needs to be understood and accepted by all surgical disciplines. Ideally, it would be reproducible, simple to use, easy to document, and quantifiable. It needs to be able to both drive management decisions and facilitate correlation with treatment response.

Another mission for the International Advisory Board of Surgical Wound Management is to codify debridement practices. All wound types were considered by the group and included those of acute traumatic origin (lacerations and crush), burns (thermal, chemical, friction, and electrical), and chronic ulcers (diabetic, vascular, pressure, inflammatory, radiation, and the like).

The pathophysiology of a wound clearly differs depending on etiology. Jackson (40) described three concentric zones in the burn injury: the zone of coagulation, the zone of stasis, and the zone of hyperemia. The central coagulation zone defines the area of greatest damage and represents nonviable tissue. It is otherwise referred to as the zone of necrosis. The zone of stasis represents tissue damaged by the burn in which there is the potential for progressive dermal ischemia. Depending on the extent of this progression, the tissue in this zone has the potential to survive. The outermost zone is the area of hyperemia where the tissue has suffered minimal injury and is expected to be viable. This description has been very useful in both practical and research discussions of thermal injury.

Although, speaking of Jackson's zones has direct application in discussions on burns, the specific pathology cannot be directly applied to nonthermal wounds. Irrespective of etiology, however, all chronic wounds and most significant acute traumatic wounds can broadly be described using a similar zone concept. There will be a zone where the tissue is most severely affected and necrotic. Adjacent to this will be an area of abnormal or injured tissue where the tissue is viable but at increased risk of necrosis. Bordering this "potentially viable zone" will be a zone where the tissue is essentially normal or at minimal risk of necrosis.

Following this line of thought, a descriptive classification based on that used to describe tumor resection was proposed at the second annual meeting in Lisbon, Portugal, March 2006. All members agreed with the underlying concept of an alpha-numeric grading system based on the zone of injury. It describes three general wound zones: a zone of necrosis, a marginal zone, and a normal zone (Fig. 1). Based on these zones, wound debridement can be classified into one of five categories: nondebrided wound (*0*), incomplete (*1*), marginal (2), complete (3), or radical (4) (Table 2).

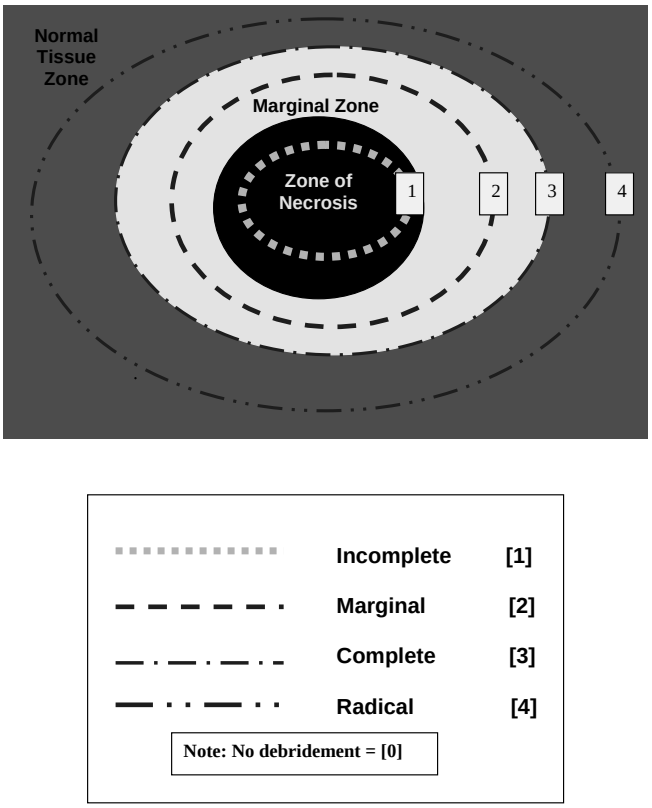

FIGURE 1
Wound zones and classifications.

TABLE 2
Classification of Debridement

Debridement	Nondebrided	Incomplete	Marginal	Complete	Radical
Code	0	1	2	3	4

Nondebrided (0)

The nondebrided wound has not progressed through the TIME sequence of wound bed preparation.

Incomplete (1)

This describes debridement in which nonviable, necrotic tissue (present at the time of debridement) has not been completely removed. This is nearly always inadequate and leads to complications of wound healing, including infection, an increase in wound size and delay, if not failure, of wound healing.

Marginal (2)

This describes complete removal of the necrotic/nonviable tissue. Some of the remaining tissue, however, is compromised in the region of injury or pathology. It is potentially viable. This "marginal tissue," therefore, has the potential to be "resuscitated." The more critical the tissue, the more likely it is that it will not be debrided with the hope that it can be salvaged. Depending on the nature and extent of the underlying injury or pathology and the "host condition" (e.g., elderly, diabetic, smoker, and the like), different strategies for resuscitation would be indicated. The strategies would involve optimizing systemic and local conditions, and may include improving intercurrent illnesses, controlling blood pressure, restoring perfusion, providing oxygen, correcting anemia and malnutrition, reducing edema, maintaining core and local temperature, ensuring adequate analgesia, and treatment of infection.

Complete (3)

This describes complete removal of injured/affected tissue—both nonviable and "potentially viable" to the border of normal tissue. It often follows serial debridements, after which clear demarcation of normal tissue from abnormal tissue is carried out. Recent innovations, such as the hydro-dissection device, may achieve this level of debridement with fewer surgical interventions (37,38).

Radical (4)

This is the same as "complete," but also includes a rim of clearly normal, unaffected tissue. Complete and radical debridements essentially result in clean acute wounds that can then be managed accordingly.

In the case where there is indecision regarding the zone of debridement, the lesser category is chosen. For example, if the surgeon is not sure whether the debrided tissue is marginal or necrotic, the debridement will be recorded as though there is still necrotic tissue; i.e.—incomplete.

TISSUE CLASSIFICATION

In addition to describing the extent of tissue debridement, it is also important to know the type of tissue being debrided. The affected tissue can be allocated into one or more of four groups: skin (S), subcutaneous connective tissue (C) (consisting of fat, vessels, and nerves), deep soft tissue (M) (consisting of muscle, fascia, tendon, and periosteum), and bone (B). This further facilitates the communication of debridement, in that often, the extent of tissue injury varies between layers as does the extent of wound debridement.

TABLE 3
Classification of Tissue Type

Tissue type	Skin	Subcutaneous (connective tissue)	Muscle/fascia	Bone
Code	S	C	M	B

By assigning a letter to the depth of the tissue involved (Table 3) and a numerical value to the extent of debridement (see Table 2), a simple alpha-numeric descriptive classification of wound debridement can be recorded. As an example, an open fracture in which there was comminution at the fracture site with periosteal stripping, muscle contusion and skin and subcutaneous tissue loss, may have had the skin and subcutaneous tissue radically debrided, the muscle cut back to bleeding, but slow to contract fibers, and the bone ends washed but not excised. This could be classified as S4, C4, M2, and B1. This is merely an example to illustrate a concept that even a simple system like this can convey much more information than we are used to communicating (Figs. 2–4).

COMPREHENSIVE SOFT-TISSUE WOUND CLASSIFICATION

Just as the debridement requires classification, so does the wound. Cierny has a classification that describes "the host" risk in patients with osteomyelitis. The host will be of either a minimal (A), moderate (B), or high risk (C), based on the presence of a systemic or nutritional disorder and smoking status (41). For open fractures, there are several wound classification systems in the literature, such as those proposed by Gustilo et al. (42,43), Tscherne (44), and Lange et al. (45). These take into consideration the size of the wound and the energy of the injury. They have some use in directing treatment and predicting treatment outcomes, but are far from being comprehensive and reproducible.

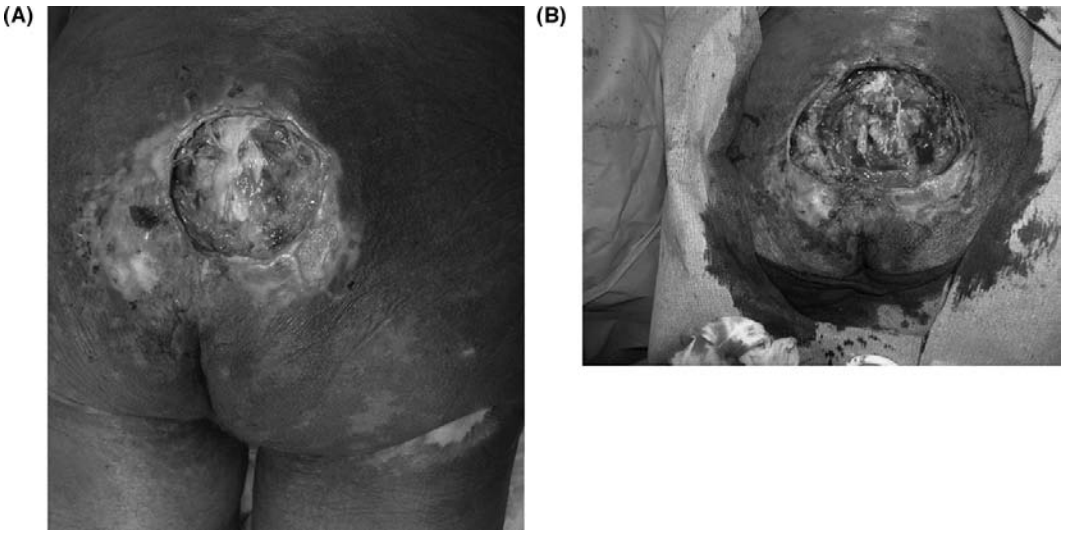

FIGURE 2
(**A**) This patient had a stage IV sacral pressure ulcer with extensive undermining and necrotic muscle and fascia. (**B**) The wound was debrided using a scalpel on the skin and a Versajet™ (Smith & Nephew, Largo, FL) on the underlying tissues. All undermined skin was excised, but all of the damaged skin was not removed (S2). The subcutaneous tissue was removed to a bleeding margin (C3). Muscle and fascia were removed to a bleeding surface (M3). Bone was not removed (B0). (*See color insert.*)

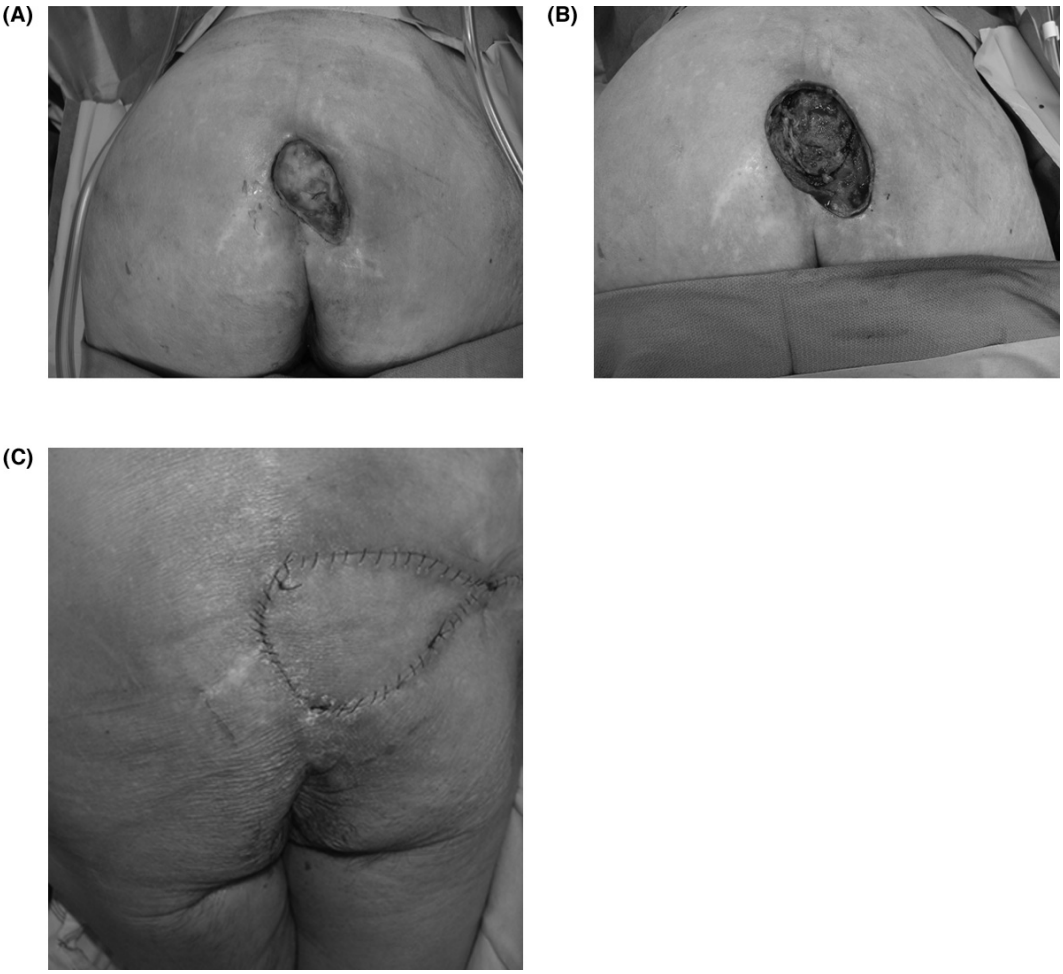

FIGURE 3
(**A**) Sacral ulcer. (**B**) Debridement performed by direct excision of skin, Versajet™ (Smith & Nephew, Hull, U.K.) removal of subcutaneous tissue, and osteotome removal of bone. Debridement score S4C3M0B3. (**C**) After immediate reconstruction with a gluteus maximus myocutaneous flap, the patient healed uneventfully. (*See color insert.*)

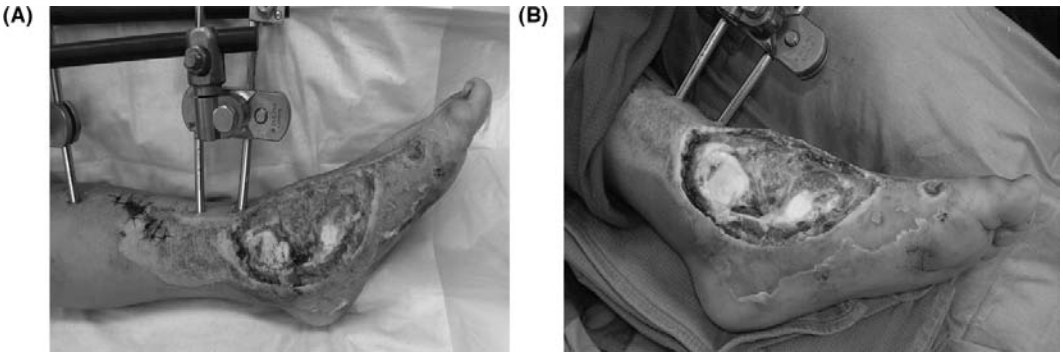

FIGURE 4
(**A**) Open ankle fracture following serial debridements (S2C2M2B2). (**B**) Open ankle fracture debrided with Versajet™ prior to definitive closure (S3C3M3B3). (*See color insert.*)

A comprehensive wound classification system is required that better captures information about the wound "personality." Too many classification systems are "comprehensive" in a descriptive manner, but the increased complexity merely adds to the difficulty in recording data and in the reliability of that data. They often add little to the predictive capacity or usefulness in directing management. Information collected should incorporate relevant data on the host, the wound etiology, the tissues involved, and the extent of the wound. The classification should be flexible and expandable to code complex wounds when required. It must also be relevant and applicable to all wound types. Such a classification should be able to direct specific treatment protocols and correlate closely with prognosis. It will be essential for future studies to assess the effect of wound-management strategies.

The International Advisory Board of Surgical Wound Management is working to develop these wound and debridement classifications to drive evidenced-based, best-practice wound management. The board is similarly interested in disseminating the experience and knowledge of its international team of experts representing numerous surgical specialties. The following chapters would clearly demonstrate the renewed interest in the surgical community to participate along with the medical-wound community to optimally manage our patients with both acute and chronic wounds.

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3 Diagnosis and Surgical Management in Wound Bacterial Burden

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INTRODUCTION

Carrel alluded to the concept of bioburden in 1921 (1), when he noted, "an abscess far removed from the site of healing can cause a delay in the healing process." Wounds are rarely a purely locoregional phenomenon; a dynamic balance exists between the patients' medical status and their ability to heal. Prompt and directed attention toward optimizing metabolic support, control of hyperglycemia, antimicrobial therapy, and tissue support are critical aspects in the successful care of patients and their wounds. The importance of optimizing nutrition, vascular status, hemodynamics, moisture control, pressure relief, immunologic status, and the control of edema cannot be overstated in the quest for improved and successful wound healing. It is well established that wound healing is impaired with infection, and this is one area that the clinician can directly intervene, treat, improve, and potentially prevent quite readily.

SKIN MICROBIAL FLORA

An individual's cutaneous flora is harbored as both transient and resident flora. Transient flora is the flora that is loosely associated with our appendages, and it is this flora that we are addressing with proper hand washing prior to meeting, greeting, and treating our patients. Resident flora on the other hand is that flora which is more intimately associated with our skin appendages, and rapidly re-establishes itself in most circumstances. It is likely that this symbiotic relationship protects us from pathogenic organisms. If one were to biopsy and quantitatively culture an adult's skin, one would find that the skin harbors on average 10^3-cfu/g tissue. Macerated skin or skin that is so dry that it has cracks and tears, tend to harbor higher bacterial counts.

BACTERIAL WOUND INFECTION

Colonization and Infection

Throughout the 1970s and 1980s, Dr. M. Robson (2) published several landmark studies on the objective characterization of what constitutes an infected wound and identified critical thresholds of bacterial load that impair graft take and wound closure. To summarize these briefly, he noted that if the bacterial count in the wound approximates quantitative counts of greater than 10^5 bacteria/g of tissue, healing is impaired. A quantitative count of less than 10^5 is considered contamination, whereas a count of greater than 105 is considered invasive infection. Several species of bacteria, like Streptococcus, will inhibit healing even when present in much smaller concentrations. Interestingly, the presence of a foreign body within the wound, such as an implant or a silk suture, may require an inoculum of only 100 micro-organisms to cause clinical infection. It has been estimated that on average it requires concentrations of 10^6- to 10^9-bacteria/g tissue to yield clinical evidence of pus.

All open wounds can be expected to harbor bacteria and other potential pathogens, and this flora would undergo significant changes over time. From a clinical perspective, it is imperative to distinguish a state of colonization from that of infection, as the treatment and management strategies would differ. To further complicate matters, the synergistic patterns of bacterial colonization often found in chronic and nonhealing wounds have also been implicated in impaired wound healing. As in the case of frank purulence, semiquantitative swab cultures may prove helpful in monitoring therapy and identifying predominant organisms. Ultimately, it is our clinical judgment derived from experience, an understanding of the pathophysiology and mechanism of injury, and an understanding of the principles of wound healing that guide our practice.

Bacterial Burden

Pathogenicity, the propensity toward impaired wound healing and resultant local and systemic inflammatory responses, is a function of the complex interaction of the host and the pathogen. The mere presence of bacteria in the wound is significantly unlikely to impair healing, and as such, wound colonization must be clearly distinguished from wound colonization and infection. The groundbreaking investigations by Robson et al. have shown that bacterial infection in wounds and the success of therapeutic surgical interventions like skin grafting is dependent upon the number of organisms present, their virulence, host resistance, quality of the wound bed, and the presence of foreign bodies.

Contamination describes the presence of nonmultiplying bacteria (3) on the surface of the wound. Colonization refers to the presence of multiplying bacteria within the wound, which do not induce host reaction or result in pathogenic effects. This relationship is commensal. Increasing bacterial counts within the wound can reach a critical point when the bacterial load activates local host defense mechanisms (4). A wound is classified as infected when the presence of multiplying bacteria within the wound results in regional and systemic effects mediated by bacterial toxins and host defense response mechanisms.

The presence of devitalized tissue in the wound is one of the major predisposing factors facilitating wound infection and inhibiting wound healing. Dead tissue provides nutrition for the increasing bacterial load and impairs local defense mechanisms. Wound debridement to remove this debris and devitalized tissue reduces the bacterial burden, freshens the wound margin, and promotes a state similar to that found in acute wound healing.

Effects of Bacterial Burden on Wound Healing

Invasive wound infection interferes with the normal wound healing process (5) by a variety of interactions. The body's defense mechanism of stimulated inflammatory cell production, cellular migration, and T lymphocyte activation results in the release of cytokines and inflammatory mediators like prostaglandin E2 and Thromboxane. Local neutrophilic response includes the release of free oxygen radicals and cytotoxic enzymes. This collective pattern of defense against micro-organisms injures the granulation tissue and the normal surrounding tissue. Edema and inflammation isolate the process, and may further inhibit systemic efforts at control.

Many species of micro-organisms produce toxins that are released into the wound, then into the circulation, effecting local tissue damage and a systemic inflammatory response. Certain bacteria produce fibrinolytic enzymes, for example, streptolysin, which might promote invasion and prevent successful wound closure. Biofilms produced from glycocalyx secretions of attached microcolonies of bacteria protect the bacteria from antimicrobials, further impairing therapeutic intervention and wound healing.

Clinical Signs of Wound Infection

It is essential that wound infection is recognized and diagnosed as early as possible, before systemic sequelae develop as wounds with uncontrolled infection, which might lead to toxemia, tissue loss, and death. The classic signs of inflammation (red, hot, swollen, painful) are generally present following acute infection with nonspecific bacteria. Chronic wounds may only manifest

subtle signs, such as impaired progression in the rate of wound healing. Malaise, diminished appetite, and lethargy, may also complicate.

Cellulitis is considered a sign of locally invasive infection, whereas purulent or serosanguinous discharge from wound indicates active infection. Friable or bleeding granulation tissue and discoloration of wound margins are also considered later cardinal signs of wound infection.

Clinical suspicion and specific therapeutic directed intervention can be bolstered by microbiological investigations. The goal of these investigations is to identify the organism (species and strain), quantify the biological burden (number per unit volume of tissue), and identify and monitor the organisms' sensitivity to therapeutic intervention (surgery and antimicrobials). Numerous techniques have been advocated, and it is essential to select an optimal culture method from an array of available techniques.

Wound Culture Methods

Quantitative Culture

Robson and Heggers (6) (1969) concluded in their study of 41 wounds, cultured with varying techniques, that quantitative culture of tissue biopsy method provided 100% sensitivity, 93.5% specificity, and 95.1% accuracy in predicting secondary closure of the wounds. This landmark study established quantitative wound culture as the gold standard in analyzing wound biological burden. Though later studies have not shown the same extremely high sensitivity and specificity, this diagnostic technique still maintains high acceptance.

Quantitative cultures identify and analyze clinical infection more accurately than swab culture. Recently, Chua et al. (7) (2005) studied 71 patients with implantable cardiac devices at the time of lead extraction. A swab culture and tissue biopsy culture specimens were taken from all the opened pockets. They observed that significantly more quantitative wound culture results were positive when compared with swab culture for implant pockets with clinical signs of infection. There was no difference in culture results for specimens taken from pockets without clinical infection.

Buchanan et al. (8) (1986) described a technique for burn wound biopsy and quantitative culture. The wound-care clinician obtains a piece of wound tissue with scalpel or punch biopsy; the specimen is accurately weighed and then homogenized in fixed volume of sterile normal saline. Serial dilutions (10^{-1}–10^{-5}) of homogenate are plated on various culture media as per organism of interest. Routinely, 48 hours after plating the wound solution, culture plates are evaluated for the number of colonies. Multiple specimens are obtained, and the results are averaged for large wounds and burn wounds to obtain precise bacteriological assessment.

This quantitative culture method requires adequate amount of wound tissue to accurately represent the microbiological environment of the wound. Our laboratory generally requires 3 g of tissue for this. 10^5cfu/g of tissue is the commonly established threshold for the majority of skin-based bacteria. Infectious load above this range is considered significant and requires appropriate intervention. In burn patients, bacterial burden greater than 10^5cfu/g of tissue is considered predictive of sepsis. Histologic evaluation of the specimen is also adjunctively carried out in some centers, particularly burn centers, where rapid identification of fascial extension and invasive fungal infections are critical components of care.

Quantitative tissue culture is an invasive procedure that may interfere with wound healing and cause additional pain. It requires a degree of expertise from both the clinician and the laboratory, and involves additional time and cost. As a result, these procedures are often not commonly available and clinicians continue to employ other techniques of obtaining specimen and bacterial culture.

Swab Culture

Surface swab culture is the most commonly utilized and simple technique for evaluating wound infection. It provides a specimen of surface micro-organisms, and in frankly purulent environments, the representative pathogen may often be identified. Topical swabs may not adequately reflect the invasive and bacterial attachment microenvironments, and as such, lack the high specificity and sensitivity that wound biopsy culture achieves, failing to distinguish between

contamination and infection. As a result of these shortcomings, there has been no consensus on the usefulness of this technique, though some continue to advocate its use in monitoring wound infections.

Levine et al. (1976) (9) established a linear relationship between bacterial counts obtained from swab culture and wound biopsy culture with a quantitative swab culture technique. They concluded that the success of the secondary closure of open wounds can be reliably predicted with wound swab culture. Bill et al. (10) (2001) revisited quantitative swab culture and compared it with tissue biopsy results. In their study, quantitative swab culture successfully identified 79% of infected wounds (diagnosed by wound biopsy). They concluded that swab culture still provides valuable additional information to aid wound care.

Many techniques have been described for obtaining quantitative swab culture. Perhaps, the best known is that described by Levine (1976), where the wound surface is cleansed of surface exudates with a moist saline gauze. A sterile culture swab is then pressed and rotated over an area of 1 cm^2 of wound to bring wound fluid and bacteria to surface. Many consider Levine's technique to be the best swabbing technique for obtaining quantitative swab culture.

Another variant of this technique is the Z-stroke technique, where after cleaning the wound with sterile saline swab, the whole surface of wound is swabbed in a Z shape when pressing down on the swab. This technique however provides less precise samples than Levine's technique.

Semi-Quantitative Culture

Semi-quantitative culture methods have been advocated to provide relatively precise bacteriological investigation at lesser cost, time, and effort compared with the more demanding quantitative tissue-culture sampling. These techniques are gradually receiving increasing recognition and acceptance amongst wound- and burn-care physicians. Maki et al. (11) (1977) first described the semi-quantitative bacteriologic culture method for assessing catheter infection. This technique can be applied to both wound biopsy specimens and wound surface swabs. There is no established cut-off point for identifying significant bacterial load for the prediction of sepsis and systemic effects, and there is no established method for semi-quantitative culture. As described by Buchanan et al., the wound biopsy specimen is processed similar to quantitative culture, but only one or two dilutions are plated on a single medium. The colony count is performed at 48 hours after plating, and the total colony count per unit (g) volume of tissue is calculated. In their study, Buchanan et al. achieved similar sensitivity, specificity, and positive/negative predictive values. They also noted significant reduction in labor and material usage. Many investigators have studied semi-quantitative culture from wound swabs and found the technique to be of value in wound care. A wound swab is taken by one of the techniques described earlier. The content of the swab is transferred to a sterile saline container, and this diluted wound exudates/saline solution is plated on a culture dish. There is no single widely accepted technique here.

Needle Aspiration

This technique is most applicable when there is fluid collection along the wound margin. Minuscule wound-tissue fragments can also be obtained by needle aspiration. The entry point for needle is chosen over intact normal skin. Currently, the sensitivity, specificity, and accuracy of needle aspiration biopsy culture are not established (12).

Newer techniques of bacteriologic diagnosis of infection and biological burden of wound involve the use of polymerase chain reaction (PCR) (13) and enzyme-linked immunosorbent assay (ELISA). A homogenized tissue culture specimen is analyzed for the presence of specific bacterial DNA or particular bacterial protein with PCR or ELISA techniques.

When faced with frank purulence and the presence of local (cellulitis) or systemic signs of invasion, this distinction is often not very difficult to make. In cases of invasive infection, the bacteria responsible are best identified by tissue biopsy and quantitative culturing obtained from the deepest affected area of debridement. The diagnosis and treatment of osteomyelitis often requires bone-derived biopsy and may be confirmed on histopathology and radiographic studies. The reality is that in many centers, though technically easy to perform, quantitative cultures may be difficult, if not impossible, to obtain.

SURGICAL TREATMENT OF THE WOUND

General Principle of Wound Debridement and Closure

The majority of acute lacerations seen in the emergency department can often be closed primarily, particularly if they present within three hours of injury and have not been incurred with complicating etiologies like crush or direct contamination. These wounds should undergo a gentle debridement, removal of any particulate contaminants, lavage, control of hemostasis, and tetanus treatment or prophylaxis as warranted. The face, so highly vascularized and with generally excellent dependent drainage and lymphatic circulations, may often tolerate lengthier periods prior to closure. It is the 20% of wounds which present in a delayed fashion or with elements of crush or ischemic injury or contaminated mechanism of injury that are most likely to be complicated by infection, that is, human bites or soil contamination. Meticulous debridement removes the tissues most likely to have high bacterial counts. In general, wound closure can be performed when quantitative cultures are $<10^5/g$. If the counts are $>10^5/g$, one should consider leaving the wound open and treating with appropriate wound care. Closure should be delayed till the counts are $<10^5/g$ tissue or there is significant clinical evidence of improvement.

Optimizing nutritional and local and systemic vascular status, improving and treating comorbidities and hemodynamics, and controlling infection, moisture balance, pressure, and edema cannot be overemphasized in the management of our patients and their wounds.

Antimicrobial Therapy

Wounds with evidence of heavy bioburden can usually be managed with topical agents, such as sustained-release antibacterial dressings (e.g., cadexomer iodine or sustained release silver). Topical antimicrobials alone will not suffice in cases of invasive infection (e.g., cellulitis and osteomyelitis). A patient manifesting signs or symptoms of sepsis, like anorexia, malaise, confusion, lethargy, fevers, and chills, resulting from the wound state, requires surgical debridement in addition to systemic antibiotics. The appropriate antibacterial is that to which the microbe is sensitive. Ideally, antibacterials should be administered prior to bacterial contamination so that an effective tissue concentration is obtained, termed surgical prophylaxis. The bloodstream is the preferred route of administration, and is ideally delivered within the first three hours of wounding. After that time, the efficacy of systemically delivered antimicrobials reaching the wound is significantly impaired.

Preoperatively, systemic antibiotics should be administered on an average of one hour prior to surgical incision to allow for satisfactory circulatory time to effect appropriate tissue concentrations.

Chronic wounds pose particular challenges for the clinician. In general, wounds that occur outside the hospital setting and are present for less than one month are usually sensitive to agents with suitable bioavailability, which address gram-positive organisms. Chronic wounds of longer duration often involve gram-negative and anaerobic pathogens.

Debridement

Debridement is defined as the removal of nonviable material, foreign bodies, and poorly healing tissue from a wound. Exudate and eschar, which impair visualization and epithelialization, are removed.

Chronic wounds behave as though they are trapped in the inflammatory and proliferative phases of wound healing. Bacterial load, elevated levels of activated proteases (matrix metalloproteases, MMPs), entrapped growth factors, and overproduction of fibronectin, result in a biochemical imbalance. Excessive and defective remodeling of the extracellular matrix, the failure of epithelialization, and the presence of surrounding cells which respond poorly to normal activating signals create a "senescent" state.

Surgical Debridement

Surgical debridement can efficiently facilitate the re-establishment of a more appropriate moisture and biochemical balance. Reducing the wound bioburden, decreasing the inhibitory load, and excising the senescent rim, converts the chronic wound state into one similar to that found in the acute wound. An important adjunctive benefit of surgical debridement is that it gives the clinician an opportunity to specifically and accurately examine the depth, extent, and quality of the wound. Accurate and reflective cultures can be obtained, underlying bony integrity can be assessed, hidden tracks and collections can be unroofed and drained, and the wound can be efficiently prepared for either surgical or nonsurgical options of closure or care.

Although surgeons recognize the importance of debridement, little data have been generated in randomized trials to support its use. One notable work by Dr. David L. Steed et al. published in 1996 (14), looked at the treatment of diabetic ulcers. In his study, it became evident that wound debridement, in and of itself, improved healing rates of these challenging ulcers. Elsewhere in this text, other forms of available clinical wound debridement modalities are discussed at greater depth, and as such I will only briefly discuss them as they relate to the context of this chapter.

Autolytic Debridement

Autolytic debridement describes the treatment of a wound with either occlusive or hydrocolloid type dressings. A moist wound-care environment is created where proteolytic enzymes and wound macrophages liquefy and debride devitalized tissue and eschar. This form of debridement is slow and not particularly aggressive, yet is relatively easy to perform. Dressings must be changed regularly to remove accumulated fluid and liquefaction and to reassess the wound and periwound state. This form of debridement is contraindicated in infected wounds.

Enzymatic Debridement

Enzymatic debridement employs endogenous or exogenous enzymes, like collagenase (derived from clostridia), elastase, or papain (papaya derived) proteolytic enzymes. Bacterial collagenase debrides type 1 and type 2 collagen, whereas papain digests fibrinous tissue sparing collagen. Papain is relatively ineffective when used alone, and as such, is often combined in preparation with urea. Urea acts as a denaturant (15), unfolding proteins and exposing cysteine residues, which can then bind the papain.

Biologic Debridement

Biologic debridement generally employs maggots, preferably sterile larvae that effect debridement and promote healing by several mechanisms. These include enzymatic debridement, decreasing bacterial counts, and motion stimulation of the wound bed.

Mechanical Debridement

Mechanical debridement generally refers to the use of a force to remove bacteria, foreign bodies, and necrotic debris. The methods include the use of wet-to-dry dressings, fluid irrigation systems, whirlpools, and pulsed lavage among others. Surgical debridement of all foreign material and nonviable tissue is often combined with these modalities to facilitate the removal of surface contaminants and bacteria.

Wet-to-dry dressings are one of the most commonly prescribed forms of mechanical debridement. They are generally easy to perform, but require frequent dressing changes. The moist gauze can often prove macerating to surrounding tissues, and many of the employed solutions can be cytotoxic. The dressing changes themselves can often prove painful for the sensate patient.

Numerous irrigation-based systems have been advocated and proven helpful as adjuncts in this process (16,17). These methods include the use of bulb syringe, gravity flow, and pulsatile lavage. Ultrasonication as a means to reduce bacterial load has also recently been advocated.

Pulsatile Lavage

Pulsatile lavage techniques are usually divided into low- and high-pressure systems. Generally, low-pressure systems exert between 1 and 15 psi, whereas high-pressure systems exert pressures between 35 and 70 psi.

The overall value of irrigation as an adjunct to mechanical debridement is still under debate, as the available literature is often difficult to directly compare and can often appears contradictory. In early works by Singleton et al. (18) and Peterson (19), they demonstrated that saline irrigation decreases the incidence of wound infection, and that the decrease was proportional to the amount of irrigation solution used. Conversely, several investigators have reported that low-pressure saline irrigation is ineffective in preventing wound infections (20–22). Recently, a significant volume of data has emerged, suggesting that the pulsatile irrigation streams delivered at high pressure and with a high flow effectively decrease the amount of bacteria, foreign bodies, and necrotic crushed tissue in wounds, at the same time as decreasing the incidence of the resultant wound infection (23–26).

In an animal model, Hamer et al. (26) showed that the wounds contaminated with bacteria alone were ineffectively debrided with gravity-flow irrigation. In contrast, bulb syringe irrigation was able to significantly reduce a pretreatment bacterial count of 6×10^6 to 9×10^5. This reduction in the bacterial count however did not prevent a rebound effect at day 3, and macroscopic examination of the wounds revealed gross infection.

Pulsating jet lavage with a pressure of $3\,g/mm^2$, 1200 cycles per minute over a period of 20s was able to significantly reduce the bacterial counts in contaminated wounds. The initial counts were reduced to 1×10^5, and most wounds remained clean over the treatment period. In a similar set of experiments, the wounds were contaminated with bacteria and foreign bodies (sterile sand). In these experiments, only pulsating jet lavage was capable of significantly reducing bacterial counts.

Saxe et al. (27) in a similar study used sequential quantitative wound biopsies to follow the efficacy of the treatment. Their study showed that high-pressure pulsatile lavage was more effective than conventional irrigation in lowering the bacterial counts in staphylococcal wound infection in guinea pigs. Interestingly, they also concluded that pulsatile lavage had no therapeutic effect on established postoperative infections.

Wound closure as an endpoint was studied comparing different mechanical cleansing techniques in an animal model with a wound infection by Nichter et al. (32). In this experiment, *Staphylococcus aureus*–inoculated wounds were treated by a variety of methods, including scrubbing and treatment with high-pressure irrigation and ultrasonication. They concluded that ultrasonication was the most effective modality of the examined techniques. However, ultrasonication reduced gross wound infection by 75% as compared with the controls; pressure irrigation lowered the infection rate by 25%.

Bhandari et al. (28), in a recent study, compared the effects of high- and low-pressure irrigation on wounds with exposed bone in an animal model. In this experiment, the wounds were contaminated with *S. aureus* and irrigated at various time points postinoculation. Quantitative bacterial analysis revealed that high-pressure lavage was effective in reducing bacterial counts up to eight hours of postinoculation, whereas low-pressure irrigation was only effective when administered up to four hours postinoculation.

Much concern has recently been afforded to the question of whether bacteria might be directed deeper into the wound and surrounding tissues with high-pressure lavage techniques. Hassinger et al. (29) studied this question comparing a low pressure (3 psi) system to a commercially available high-pressure lavage system in an in-vitro model. They found that bacteria were in fact driven into the tissue by high-pressure lavage. They concluded that high-pressure lavage should not be used in grossly bacteria-contaminated tissue and advocate surgical debridement with additional low-pressure irrigation. In another study performed by the same author on bone specimens, again bacteria were seen to have been propelled into deeper levels of bone with high-pressure lavage.

Although the clinical relevance of these investigations has yet to be definitively established, studies by Tabor et al. (30) using an in-vivo canine model and high-pressure lavage when compared with bulb syringe rinsing did not lead to a detectable bacteremia. As is the case with many of these studies, a potential limitation of this study was that this particular model might not accurately reflect the situation posed in a patient with multiple comorbidities, the immunocompromised, or the critically injured. It will be very interesting to see how other clinically utilized mechanical debridement modalities, like brush techniques, dermabrasion, ultrasonication, or water-jet debridement modalities affect the propagation of bacteria into deeper tissue planes.

Water-Jet Debridement

Versajet™ (Smith & Nephew, Largo, FL) is comprised of high-velocity sterile saline stream, which jets across an operating aperture. The flow pattern creates, by "Venturi" and "Bernoulli" effects, a tool which debrides and evacuates debrided tissue and exudates. Adjustable power setting allows for precise and controlled depth of debridement. Webb et al. (31), in a study of experimental cadaveric human wounds seeded with various sized metal particles, observed that Versajet removed 88% of particles compared with the 22% particle reduction by high-pressure pulsatile lavage. Clinical studies performed by the authors have demonstrated accurate control of the depth of debridement. Quantitative tissue cultures taken prior to and after Versajet debridement reveal effective reduction in bacterial counts, which correlated with successful application of skin grafts, biosynthetic integration, and flap closure. These data have been submitted for publication. Quantitative bacterial tissue culture counts performed before and after clinical wound debridement with the use of VersaJet has been demonstrated in clinical review. These data have also been submitted for publication.

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4 Surgical Management of Necrotizing Fasciitis

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The term necrotizing fasciitis was first used in 1871 by the Confederate Army Surgeon, Joseph Jones (1). Other similar clinical syndromes have been variously termed nonclostridial gas gangrene, necrotizing cellulitis, necrotizing erysipelas, hemolytic streptococcal gangrene, or Fournier's gangrene, if it involves the perineal area or genitalia. Gradually, these have become incorporated into the single term, necrotizing soft-tissue infections, describing rapidly spreading infections requiring radical surgical debridement of affected skin and subcutaneous tissue. The incidence of necrotizing fasciitis is approximately 0.4 per 100,000, although it might have increased in the 1990s (2). The Centers for Disease Control and Prevention (CDC) estimates 10,000 to 15,000 cases of invasive group A streptococcus annually, with 5–10% of those being necrotizing fasciitis (3). Necrotizing fasciitis is more prevalent in adults, although it has been reported in children too (4). Mortality has been reported in up to 40% of patients, with most series ranging from 12% to 30% (3–5). Patients at increased risk include older patients and those with obesity, diabetes mellitus, alcoholism and intravenous drug abuse, peripheral vascular disease, immunosuppression, and recent surgery. The pathogenesis of these infections is thought to result from the entry of organisms through a compromised skin barrier with subsequent spread along subcutaneous fascial planes, sparing the muscle fascia. Infections involving the muscle and muscle fascia are usually clostridial myonecrosis, and have different clinical characteristics than necrotizing fasciitis and Fournier's gangrene. The rapidly spreading necrotizing soft-tissue infections cause thrombosis of penetrating vessels, which in turn causes necrosis of overlying tissues supplied by those vessels. Systemic spread of infection causes overwhelming sepsis or the toxic shock syndrome if associated with streptococcal exotoxin (6). Streptococcal toxic shock-like syndrome, although most common with group A streptococcus, can also occur with group B streptococcus and have a mortality of 30–60% in the first 72–96 hours (7). It has been theorized that the poor prognosis in some patients is related to an immunogenetic response as a causal factor for their increased morbidity and mortality from invasive streptococcal infections (7).

Necrotizing soft-tissue infections, although relatively rare, can be serious causes of morbidity and mortality. Prompt diagnosis, aggressive empiric antibiotics therapy, and radical surgical debridement are essential for the survival of these often gravely ill patients. Preoperative planning and new adjuncts in wound care can aid in the definitive closure of the often-massive soft-tissue defects, necessitated by the extent of surgical debridement. These patients may be best served with referral to a major burn center with experience in the surgical management of necrotizing fasciitis, and specialized wound care, typically best done by the burn team. Furthermore, these patients often have significat physiologic perturbations, require goal-directed therapy for restoration of their homeostasis and surgical intensive care unit support, and have significant rehabilitative needs.

DIAGNOSIS

Critical to the effective care of patients with necrotizing soft-tissue infections is early diagnosis and intervention. Early manifestations of the necrolytic process include erythema, often mistaken for cellulitis, or red, shiny, swollen skin that progresses to purplish or bluish areas with fluid-filled bullae or vesicles with a watery, thin, foul-smelling discharge, often termed "dishwater pus." Patients may have fever and an elevated white blood cell count, in addition to pain

out of proportion to the wound. Late manifestations may include insensate areas of the skin followed by frank gangrene, systemic shock, and coagulopathy. Admission blood leukocyte counts greater than 15.4×10^9/l and serum sodium values less than 135 mmol/l have been used to help differentiate necrotizing infections from simple cellulitis (8). Plain X-ray, computed tomography, or magnetic resonance imaging studies may show subcutaneous gas and inflammation along subcutaneous tissue planes, but waiting for radiologic studies may cause inappropriate delays in the necessary surgical management. Clinical examination and a high degree of suspicion are often enough to warrant operative intervention without confirmatory studies.

ANTIBIOTIC THERAPY

Prompt empiric broad-spectrum antibiotic therapy is an important adjunct to operative debridement. Historically, the term necrotizing fasciitis referred to single-organism hemolytic streptococcal infections. However, it is now quite apparent that the microbial spectrum of what has become the clinical syndrome referred to as necrotizing fasciitis often involves a polymicrobial, and may be a nonstreptococcal in origin infectious process. In a large series by Elliott et al., the organisms recovered from these wounds included streptococci, enterococci, staphylococci, *Escherichia coli*, *Proteus*, *Klebsiella*, *Enterobacter*, *Pseudomonas*, *Acinetobacter*, *Eikenella*, *Citrobacter*, *Peptostreptococcus*, *Bacteroides*, clostridia, and fungal species. Antibiotic regimens for coverage of gram-positive organisms may include a continuous infusion of high-dose penicillin or alternatively extended–spectrum penicillin derivatives and vancomycin in a penicillin-allergic patient. Gram-negative coverage is supplied with aminoglycosides, cephalosporins, or carbapenems, and anaerobic coverage with clindamycin or metronidazole (3). Antibiotic therapy can subsequently be tailored to specific cultures as they become available. Laboratory studies have used polymerase chain reaction (PCR) to confirm the presence of group A streptococcal exotoxin in culture-negative patients, but this may not have widespread clinical application (9).

SURGICAL INTERVENTION

Early surgical intervention has been shown to improve outcomes in patients with massive soft-tissue infections (4) (Fig. 1). The primary principle in operative debridement of necrotizing soft-tissue infections is expeditious removal of all necrotic or infected skin and subcutaneous tissue. Confirmatory findings include necrosis of the superficial fascia, thrombosis of superficial vessels, and foul-smelling drainage. Fluid and tissue cultures should be sent for immediate Gram stain and aerobic and anaerobic culture and sensitivities. Deep fascia and muscle should be inspected, and if a muscle is involved, this may signal a clostridial infection or streptococcal myonecrosis rather than necrotizing fasciitis. Invasive clostridial infection in the extremities may necessitate amputation in some dire circumstances, but is usually not required with necrotizing fasciitis. Despite the obvious need for swift radical excision, incisions may be planned along anatomic lines with an eye on facilitating eventual wound closure. Clearly viable skin should be preserved, if possible, to aid in future definitive wound coverage. Once hemostasis has been achieved, the wounds should be packed open. One approach for the initial dressings is to use a 50% betadine solution in saline for the initial dressing, followed by wet-to-wet dressings soaked in 5% mafenide acetate solution, changed at least twice daily. Although many have written about the need for multiple operative debridements to ensure the removal of all infected tissue, this thought should not cause the surgeon to leave suspect tissue in the wound, as effective debridement is a necessary component of the overall goal-directed therapy. It has become our practice to do the majority of the debridement at the initial setting. This requires careful preoperative preparation, including placement of central lines for large-volume resuscitation and rapid availability of blood and blood products. Early and frequent reminders of the critical nature of the disease should be communicated to the anesthesia team to encourage rapid aggressive resuscitation in the operating room, and if necessary, the use of rapid fluid infusion devices. When the operation is completed, the patient should be promptly returned to the intensive care unit with ongoing correction of hypothermia, acidosis, and coagulopathy, as necessary. Frequent wound examination is prudent, and any signs of ongoing spread of infection, including failure to respond to resuscitation, should

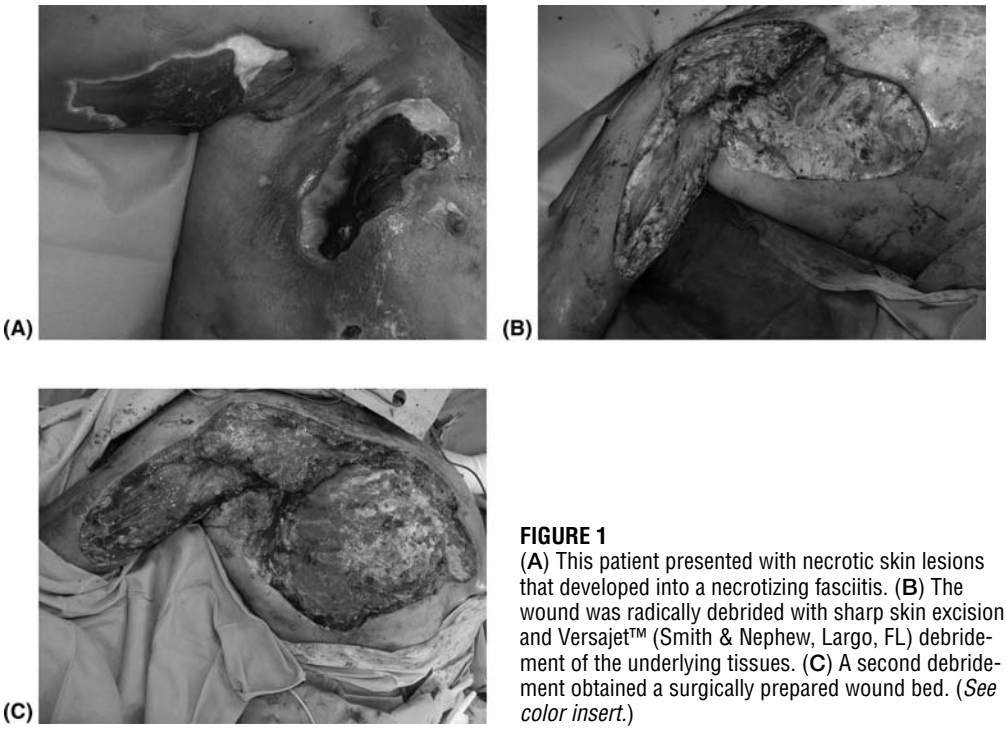

FIGURE 1
(**A**) This patient presented with necrotic skin lesions that developed into a necrotizing fasciitis. (**B**) The wound was radically debrided with sharp skin excision and Versajet™ (Smith & Nephew, Largo, FL) debridement of the underlying tissues. (**C**) A second debridement obtained a surgically prepared wound bed. (*See color insert.*)

prompt a return trip to the operating room for a second look operation. Bedside intervention may be necessary in the unstable patient, and can be accomplished with sharp debridement and portable electrocautery.

WOUND MANAGEMENT

The goal of postsurgical debridement wound management is to create a wound that is amenable to closure. The management of the postdebrided wound requires an understanding of the disrupted local physiology that is present in the wound bed that is incapable of supporting wound healing and closure. The International Advisory Board on Wound Bed Preparation has formulated the TIME principles (tissue, infection, moisture, and edge) of wound bed management, which provides a very useful conceptual framework for the plan of wound care (Table 1) (10). The wound bed early postsurgical debridement, although perhaps no longer a wound with ongoing invasive infection, is composed of a tissue bed that contains a defective matrix and cellular debris. The treatment plan must now move to one of the episodic or continuous debridement of residual necrotic debris as with sharp, autolytic, enzymatic, or mechanical debridement. It is likely that a complicated wound, which often results from the initial surgical management of a massive soft-tissue infection, would require elements of a combination of these approaches to achieve a tissue bed in which there has been a restoration of functional extracellular matrix proteins. The control of infection and inflammation in the wound bed would result to some degree with the debridement process, but would require a period of systemic antimicrobial therapy, and may be further facilitated by the use of topical antimicrobial agents guided by culture results. Monitoring of the microbial status throughout this phase of wound management can help in focusing on the selection of antimicrobial agents and whether superinfection of the wound has occurred. The control of infection leads to a decrease of the local inflammatory response and protease activity within the wound along with an increase in the wound-derived growth factors. The moisture content of the wound is a critical factor that is not well appreciated as one that should be actively managed. Desiccation impairs local epithelial cell migration,

TABLE 1
TIME (Tissue/Infection/Moisture/Edge)—The Principles of Wound Bed Management

Clinical observations	Proposed pathophysiology	WBP clinical actions	Effect of WBP actions	Clinical outcomes
Tissue non-viable or deficient	Defective matrix and cell debris impair hearing	Debridement or continuous autolytic, sharp surgical enzymatic mechanical, or biological agents	Restoration of wound base and functional extra-cellular matrix proteins	Viable wound base
Infection or inflammation	High bacterial counts or prolonged inflammation inflammatory cytokines protease activity growth factor activity	Remove infected foci topical/systemic antimicrobars anti-inflammatories protease inhibition	Low bacterial counts or controlled inflammation inflammatory cytokines protease activity growth factor activity	Bacterial balance and reduced inflammation
Moisture imbalance	Desiccation shows epithelial cell migration; excessive fluid causes maceration of wound margin	Apply moisture balancing dressings, compression negative pressure, or other methods of removing fluid	Restored epithelial cell migration desiccation avoided oedema, excessive fluid controlled, maceration avoided	Moisture balance
Edge of wound-non-advancing or undermined	Non-migrating keratinocytes; non-responsive wound cells and abnormalities in extracellular matrix or abnormal protease activity	Re-assess cause or consider corrective therapies debridement skin grafts biological agents adjunctive therapies Migrating keratinocytes and responsive wound cells Restoration of appropriate protease profile	Advancing edge of wound	

whereas excessive wound moisture content causes tissue maceration. Wound edges are nonadvancing, undermined, or contain nonmigrating keratinocytes and nonresponsive cells. Biochemically, such wound margins have abnormalities of extracellular matrix proteins and protease activities and do not promote closure via peripheral ingrowth, and would be problematic with the surgical wound closure. The wound edges must be actively approached with debridement and be a component of the overall wound management scheme. The notion that nurse-initiated wound-dressing changes will achieve the desired outcomes underestimates the challenges that these wounds present.

Standard wet-to-wet gauze dressings with or without topical antimicrobial agents may be used in the initial (24–48 hours) period following surgical debridement until the wound bed has stabilized and there are no signs of persistent invasive wound sepsis. To achieve a wound bed that is suitable for closure has become greatly facilitated by the use of the vacuum-assisted closure (VAC) device (Kinetic Concepts, Inc., San Antonio, TEXAS, USA). This system consists of foam dressing placed into the wound cavity with an overlying adhesive seal to which a controlled subatmospheric pressure can be applied. This approach has been shown to reduce chronic edema, increase local blood flow, enhance the formation of granulation tissue, and promote contraction of the wound edges (7). The use of a silver-impregnated cloth dressing as an underlay beneath the VAC, or alternatively, a prefabricated silver-containing foam, have been found to augment the wound response to VAC therapy. The dressing apparatus is changed every two to three days, but any signs of surrounding erythema, fevers, excessive pain, or bleeding should prompt removal of the dressing and examination of the wound. Some authors have used hyperbaric oxygen therapy to promote wound healing in patients with necrotizing infections, but true value of this therapy cannot be assessed based on the current published reports (3). The goal of this phase of wound management is a clean and well-vascularized wound bed. The

wound management techniques that are used are the means to achieving this end. Failure to appreciate these simple concepts does not allow a dressing management system to compensate for the lack of rational surgical decision making.

At the planned time of wound closure, the patient's overall physiologic status should have been optimized. This includes the resolution of their septic process, restoration of their nutritional health, correction of anemia, and prophylaxis of thromboembolic complications based on the patient's assessed risk profile. Systemic antibiotics should be administered, given that the wound bed likely harbors residual bacteria that may seed the blood stream during the process of wound manipulation. The wound closure may involve the use of local-tissue rearrangement techniques, application of split thickness skin grafts, or combinations of these approaches. What is critical to the success of the planned wound closure is that the wound bed preparation has resulted in the resolution of not only the initial necrolytic infection, but any subsequent microbial colonization and the removal of all necrotic debride. At the time of wound closure, the need for further surgical therapy of the wound bed must be determined. This may include excision of the wound margin perimeter to remove overhanging edges, fibrotic tissue, and marsupialization of wound edges to the wound bed. The status of the granulating bed should also be assessed. The areas of hypertrophic granulation should be debrided; a uniform wound surface should be created if possible; and the removal of adherent biofilm must be carried out. Various approaches can be used to achieve remodeling of the bed, and include the use of a surgical scrub brush, planning with a wide osteotome, bone curettes, and a water-cutting system (Versajet™ dissector; Smith & Nephew, Largo, FL) (Fig. 2). The latter is a particularly useful approach, and allows for a quite precise dissection of the bed and easy preservation of critical structures, at the same time as providing a clean surgical field, as the Versajet system evacuates the debrided tissue and blood. In these often-obese patients, redundant skin and subcutaneous tissue may allow primary closure of the wounds, particularly in those involving the groin and perineal areas. Wounds not amenable to primary closure typically require coverage with split-thickness autografts. Meshing of skin grafts allows easier conturing of the graft to an irregular wound bed than with sheet autografts. The upper limits of the expansion preferably should not exceed a 1:3

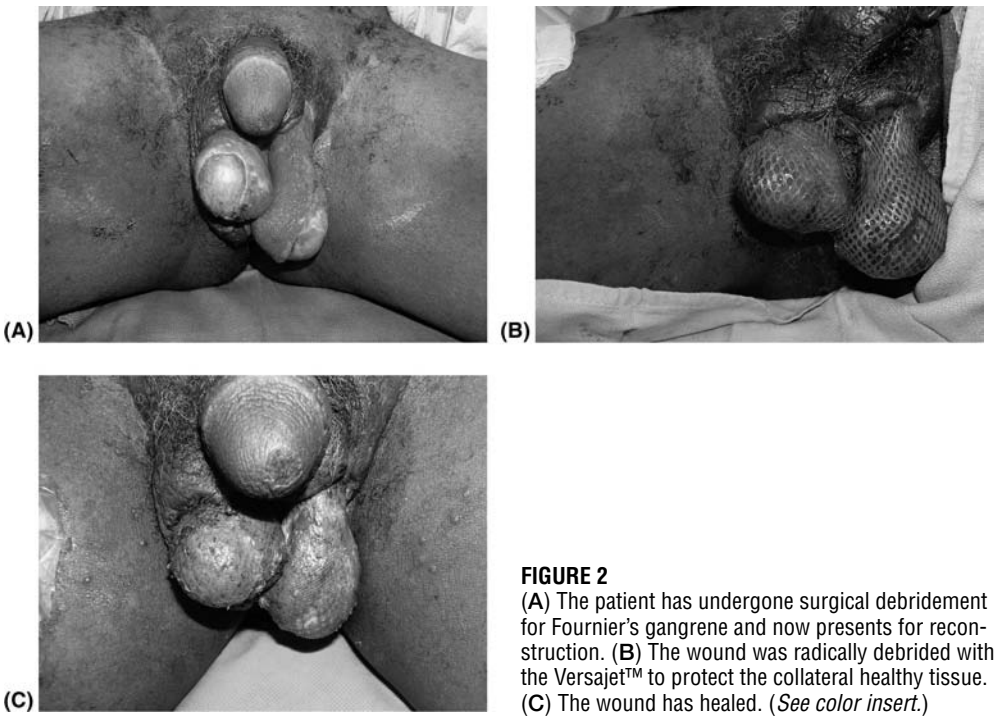

FIGURE 2
(**A**) The patient has undergone surgical debridement for Fournier's gangrene and now presents for reconstruction. (**B**) The wound was radically debrided with the Versajet™ to protect the collateral healthy tissue. (**C**) The wound has healed. (*See color insert.*)

expansion ratio. The fixation of the graft to the wound can be with staples or absorbable sutures. Graft fixation can be supplemented with the use of a tissue adhesive, such as fibrin glue. There is no unanimity of opinion as to whether or not the dressing should include topical antimicrobial agents, but is favored by some and has been a very useful approach. Five-percent mafenide acetate solution applied to postgraft necrotizing fasciitis wounds has been shown to increase the success of first-time wound closure (11,12). The closure procedure can be a combination of approaches and done in stages. A VAC dressing can be used to manage the components of the wound not amenable to closure and serve as the surgical dressing. Patients with large necrotizing soft-tissue infections typically have prolonged hospital courses with severe debilitation. Postoperatively, when the wound is determined to be healing satisfactorily, early physical and occupational therapy should be instituted to maximize functional recovery. Anticipating referral to rehabilitation facilities is mandatory to expedite these patients' complete recovery, as nearly half of the patients in a recent series needed further subacute care before returning home (12). In the long-term management of extremity wounds, it is often of help to employ custom-fit pressure garments to control limb edema.

CONCLUSION

Necrotizing soft-tissue infections, albeit somewhat rare, can be rapidly lethal. The mainstays of management are prompt diagnosis, aggressive use of empiric antibiotics, aggressive support of the patient's septic process, and most importantly, early radical debridement of affected tissue. New adjuncts in wound care may aid in the preparation of the wound bed and closure of the wound. The surgical management of these patients can be very challenging, and requires a carefully laid out plan that encompasses in a sequential fashion each phase of management with the goal of a healing and closed wound with maximal tissue conservation and restoration function.

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5 Tangential Debridement

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INTRODUCTION

Management of full thickness and deep dermal partial thickness burns usually involves debridement with split skin grafting. Debridement involves removal of devitalized, necrotic tissue from a wound in order to promote healing and prevent infection. Debridement is also vital to obtain a vascularized wound bed to accept the skin graft. Ideally, debridement will only remove dead tissue (eschar) with preservation of all living tissues. Theoretically the more dermis one can preserve the less the resulting scarring will be. This chapter will explore this hypothesis and the different techniques available to achieve optimum results.

HISTORY

As early as 1947, it was recognized that early excision of burn eschar and grafting resulted in better survival (1). Progress however was hampered by unacceptable blood loss and sepsis. The 1960s saw the advent of topical antimicrobial agents containing silver such as silver nitrate and silver sulfadiazine with a resulting dramatic increase in survival rate. It has been estimated that burn wound sepsis at this time was reduced from 60% to 28% (2). Prior to the 1970s, grafting was mainly performed after the burn eschar had started to lift from the burn wound. Eschar starts to lift at 10–14 days postburn and can be removed with the aid of a blade or electrocautery at this stage. Some surgeons preferred to wait for several weeks for all the eschar to lift totally leaving a granulating bed behind. During this waiting period many patients succumbed to sepsis, and those who survived inevitably developed unsightly hypertrophic scars and severe contractures. In an attempt to improve survival rates and reduce scarring, Jazekovic in 1970 (3) introduced tangential excision and early grafting. Trials over the next few years showed that healing time, hospital stay, and overall mortality can be reduced by following this technique (4,5) with the greatest improvements in survival rates for patients with large burns (6–8).

THEORY

Excision of burns eschar can either be performed by fascial excision or tangential excision. Fascial excision involves removing all tissue down to facia. This procedure is relatively straightforward, resulting in less bleeding than tangential excision, and producing a well-vascularized wound bed which usually readily accepts a skin graft. However, owing to the lack of supportive structures, including dermis and fat, fascial excision results in a significant cosmetic deformity. For this reason, fascial excision is usually reserved for massive burns where the burn has extended down into the fat layer. Tangential excision involves the excision of eschar in thin layers until viable dermis or underlying subcutaneous fat is reached. By using this technique, a maximum of viable tissue is preserved with the aim of achieving optimal functional and cosmetic results. The hypothesis is that preservation of dermis under a grafted area will reduce scar formation. The presence of necrotic tissue on a burn wound not only encourages bacterial proliferation, but could be an inhibitory factor in the wound healing process. Further, it might cause injury to the underlying tissue leading to a deepening of the burn. It has been shown in the porcine burn model that early tangential excision speeds up re-epithelialization of deep dermal partial thickness wounds and closure of full thickness burns (9).

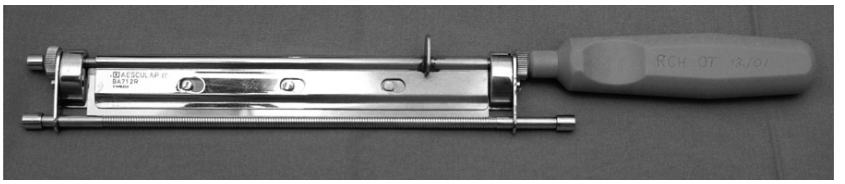

FIGURE 1
A Humby debridement knife (Down, Sheffield, U.K.).

A study in pigs looking at the rate of epithelialization of second-degree burn wounds showed that seven days postinjury, 75% of burn wounds completely epithelialize when tangentially debrided 24hours after burning, as opposed to 22% when tangentially debrided 96 hours after burning (10). Several studies in humans have shown that early tangential excision if performed in the first 24 hours after a burn leads to a decrease in the wound inflammatory response as shown by a decrease of the inflammatory mediators: interleukin-8 (IL-8), malondialdehyde (MDA), and myeloperoxidase (MPO). Levels of epidermal growth factor (EGF), basic fibroblast growth factor (bFGF), and platelet-derived growth factor (PDGF) were also reduced in the non-debrided wounds. Significantly, the depth of the final burn wound was reduced when early debridement was performed, suggesting that the removal of necrotic tissue with the contained inflammatory mediators prevents extension of the tissue damage (11,12). In another study, an increase in urine output has also been recorded during the shock phase with early debridement, with a more rapid healing time when compared with debridement at four to six days postburns (13). This later study shows that removal of the necrotic tissue limits the systemic inflammatory response. However, not all studies have supported the theory. A porcine study comparing the rates of infection and re-epithelialization in debrided and nondebrided second-degree burns, found that early postburn dermal debridement resulted in more infections and slower rates of re-epithelialization (14).

TANGENTIAL DEBRIDEMENT IN PRACTICE

Tangential Debridement with Knives

Tangential debridement has been traditionally performed with a dermatome [Humby (Down, Sheffield, U.K.) or Watson (Integra, Plainsboro, N.J.) knife] (Fig. 1) or a guarded knife (Goulian/Weck knife) (Fig. 2). These instruments can remove very thin slices of tissue (down to 0.006 in) at each passage.

Determining the Level of Excision

One of the challenges with tangential excision is how far down to debride. Debriding too far will result in the loss of valuable dermis, and conversely inadequate debridement will lead to graft loss. For small areas on limbs, or for areas on the head, neck, or trunk for which a tourniquet cannot be used, an acceptable wound bed is identified by active punctate bleeding (Fig. 3). The problem however arises where a tourniquet has been placed on an exsanguinated limb to minimize bleeding. In this situation, great experience is needed to recognize healthy tissue by its color and texture from devitalized tissue. Some surgeons choose to facilitate this process by not having the limb exsanguinated prior to inflation of the cuff. In this situation bleeding can still be observed, although at a much reduced level (15). Another technique used is to apply the dye methylene blue topically to the burn surface. The assumption is that the dye will only stain dead tissues which can be removed leaving the healthy unstained tissue behind (16). Alternatively, some surgeons have used intravenous fluorescein with the assumption that only living tissues will take up the stain. It is of special value in hand burns (17).

The percentage of body surface area which can undergo tangential debridement at one sitting depends on several factors. These factors include the stability of the patient, the adequacy of the anesthesia and the surgical team performing the procedure, and the utilization of tourniquets. For large burns several surgical teams can work simultaneously on different body areas.

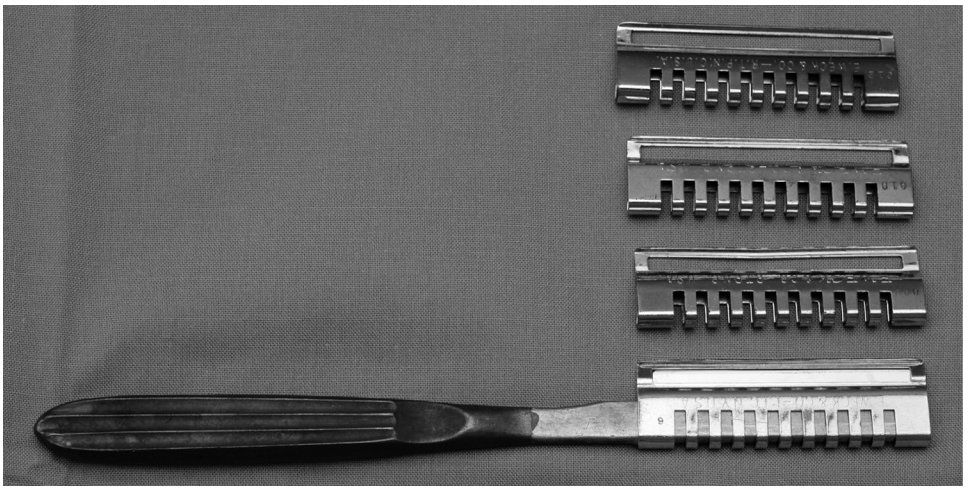

FIGURE 2
A Guillian/Weck knife with various guards which allow tissue to be cut between 0.006 and 0.012 inch.

This strategy will not only speed up the procedure, but allow larger areas to be debrided with less cooling owing to prolonged patient exposure. For large burns central venous access, an arterial line, a nasogastric tube, and a urinary catheter are usually required. Under optimal conditions, up to 20–40% total body surface area can be debrided at one sitting.

Control of Blood Loss

Blood loss during debridement is inevitable but should be kept to a minimum. Blood loss can vary greatly (18); however, anticipated blood losses are 0.75 ml/cm^2 of area of excision during 2–16 days postburn, or 0.40 ml/cm^2 if excision is performed during the first 24 hours (19). Strategies to minimize blood loss include: tourniquets, local pressure, topical thrombin, and subcutaneous adrenaline (epinephrine) and para-ornithin-8-vasopressin (POR-8). As stated before, the use of tourniquets on limbs can drastically reduce blood loss. Their use, followed by the application of firm supportive dressings, have been shown to reduce blood loss by as much as 80% (20). Subcutaneous infiltration of adrenaline has been used successfully to reduce blood loss (18). However, side effects of excess adrenaline administration include hypertension and paroxysmal tachycardia, especially in children. Interestingly, topical application of adrenaline has little effect on blood loss when used for wound debridement (21). Topical thrombin solution (1000 units in 1 ml of saline solution) has been used successfully. It is best applied to limbs before the tourniquet is released and a pressure bandage is applied for 10min for the thrombin to have maximal action. Larger bleeding points should be controlled with electrocautery (22). Subcutaneous infiltration of POR-8, a synthetic neurohypophyseal-like hormone has had success (23) but has lost popularity because of the potential complication of supraventricular tachycardia (24). Before applying a skin graft, major bleeding points should be controlled but split skin grafts are themselves hemostatic and can be applied early.

Other Instruments Developed for Tangential Debridement

Although Humby and Goulian/Weck knives cut in thin slices, it is inevitable that some viable tissue will be removed. This may mean that that some deep dermal partial thickness burns are converted to full thickness wounds. This is because it is difficult to take extremely fine cuts with these instruments, compounded with difficult skin contours and the variable depths of the eschar within the individual wound. In an attempt to minimize the amount of living tissue removed, and because these knives are often not suitable for difficult areas such as digits and

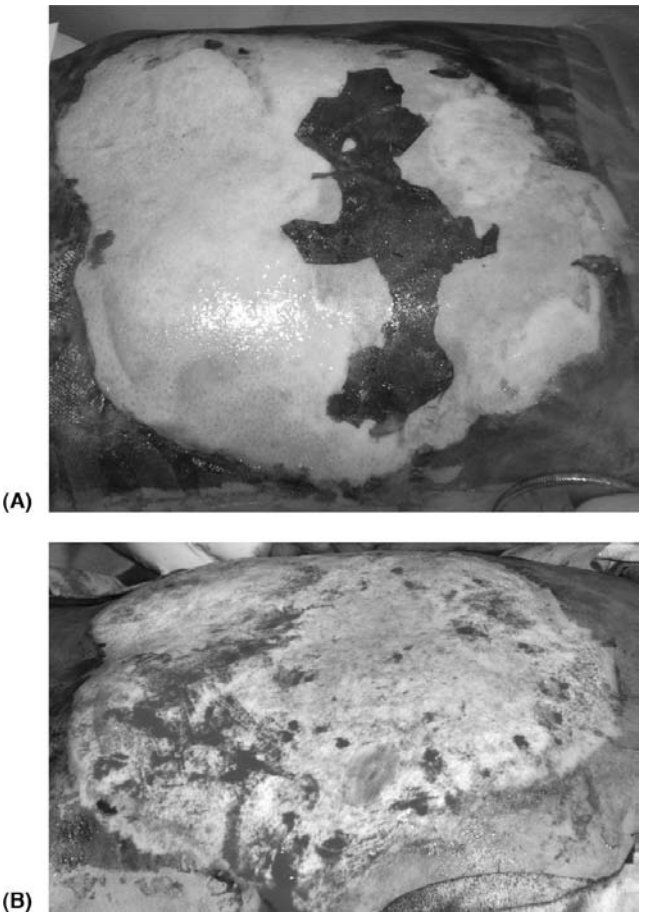

FIGURE 3
Deep dermal partial thickness burn to anterior chest before (**A**) and after (**B**) tangential excision with dermal preservation (note punctuate beeding). (*See color insert.*)

faces including ears, other debridement instruments have been developed. Table 1 shows a comparison of different debridement instruments.

Dermabrasion

Dermabrasion was first described as a debridement tool for burns in 1963 (25). It is a modification of tangential excision which removes tissue in even smaller increments and in smaller areas than one can with a knife. Advocates claim good results from this technique with less bleeding than with traditional debridement knives with easier access to difficult areas (26). In limited studies, dermabrasion has been shown to facilitate shorter wound healing times and a reduction in scar formation than burns treated with traditional tangential excision (27). The disadvantages of using dermabrasion are that it is generally slower than tangential debridement with a knife, and there is a spray of debrided tissue and fluid from the device which has obvious potential dangers to theatre staff.

Hydrosurgery

Another advance in the field of tangential excision is the Versajet™ Hydrosurgery System (Smith & Nephew, Largo, FL) Fig. 4. (28) The device produces a high powered jet of sterile saline which passes across an operating window and into an evacuation collector. This creates

TABLE 1
Comparison of Different Debridement Techniques in Burns

Technique	Speed	Bleeding	Dermal preservation	Fluid and tissue spray	Availability
Fascial excision	Fast	Less than tangential excision	Nil	Nil	No special equipment required
Debridement knives	Fast	0.40–0.75 ml/cm^2	Some	Nil	Debridement knives are available in all burn centers
Dermabrasion	Slow	Less than with knife	Maximum	Yes	Only available in some centers
Hydrosurgery	Slow, especially with early debridement	Less than with knife	Maximum	Yes	Only available in some centers
Laser (YAG)	Slow and mainly experimental	Less than with knife	Maximum	Vapor	Only available in some centers

a localized vacuum across the operating window by the venturi effect, enabling the target tissue to be held and cut, while debris is aspirated from the site. Owing to the ability to debride in small increments, hydrosurgery theoretically can preserve more dermis than conventional tangential techniques with a knife. This suggests that the use of hydrosurgery may be of great benefit in reducing scarring in patients. This may be more important in pediatric burns where hypertrophic scarring and scar contractures are common. Our own use of hydrosurgery has been very encouraging in pediatric burns, and its small cutting surface and fine control is especially useful for the "difficult to access areas" on hands, feet perineum, and head and neck.

Laser Debridement

The CO_2 laser has been used to debride full thickness burns since 1975 (29). The CO_2 laser is a photothermal laser with a marked coagulative effect. When used in a series of children with full thickness burns it proved to be very useful at minimizing blood loss, without interfering with graft take (30). Recently, the erbium:YAG laser has shown promising results on partial thickness burns. The erbium:YAG is a photomechanical laser and for this reason has a very limited coagulative effect on the target tissue. Because the beam produced has such a strong affinity for its target chromophore it only penetrates a short distance into the skin before its energy is fully absorbed (31).

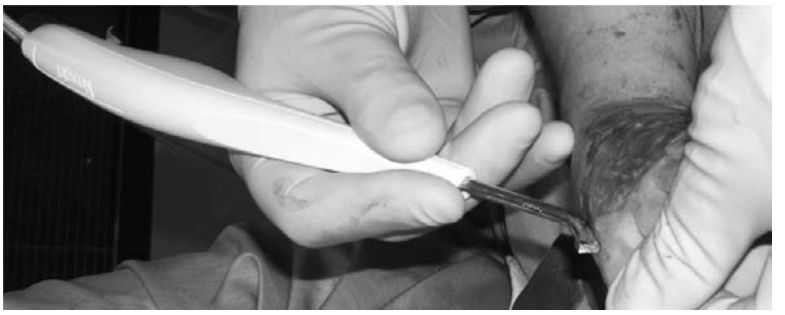

FIGURE 4
The Versajet™ Hydrosurgery System (Smith & Nephew, Largo, FL) being used to debride an ankle burn. (*See color insert.*)

FUTURE DEVELOPMENTS

In the future, we will see further development of the debriding instruments which will allow accurate rapid debridement of devitalized tissue with minimum blood loss. This will enable larger areas to be debrided at one sitting. Further studies are required to determine the optimum level to which debridement should be taken, possibly at a level prior to one where bleeding is observed.

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6 Debridement of Pediatric Burns

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INTRODUCTION

Why does the debridement of burns in children warrant a separate chapter in this book? There are many reasons. The thinner skin is less resistant to thermal (or any other) injury than thicker adult skin, with heat penetrating more deeply. Hair follicles and sweat glands are less well developed in children than in adults, and are more superficially located within the dermis. This means that mid- or deep-dermal burns have a reduced innate ability to re-epithelialize. Hypertrophic scarring is more common in children, and as these scars are stretched across joints by the growth of the child, contractures develop. A common place for scarring in children is the chest, after a pull-down scald. In girls, the development of thick, unyielding scarring over the developing breast can lead to disastrous consequences for the self-conscious teenager. Children are coerced into prolonged wearing of pressure garments and other scar therapy agents, all of which are costly. The amount of blood loss associated with burn debridement in children has a higher significance because of the relative physiologic immaturity of the younger pediatric patient. And finally, burns scarring obtained in childhood will stay with the patient for the rest of their life, possibly incurring life-long social and psychologic problems, perhaps even impacting on employment choices.

WHY DEBRIDE?

Infection is the enemy of the burn surgeon. Most surgeons rely on antimicrobial topical agents to try to prevent burn wounds from getting infected. Nearly all antimicrobial agents are hindered/bound by the proteinaceous material present in burn slough, and will not penetrate a thick eschar. Apart from providing a growth medium for the micro-organisms, the presence of necrotic tissue will prolong the inflammation associated with burn injury.

Temporary skin substitutes, such as Biobrane or Transcyte, have been shown to reduce pain scores, length of hospital stay, and time of healing (1,2). The frequency of dressing changes is greatly reduced with the use of these dressings.

These dressings are not infrequently associated with infection, particularly if there has been a delay between the time of burn and the application of the dressing. Thorough debridement of all loose tissue is essential to ensure good adherence of these expensive dressings. It is also absolutely essential that burns are debrided as thoroughly as possible before the application of keratinocytes.

WHERE TO DEBRIDE?

Burn debridement in children is an undertaking which should not be taken lightly. This should only be performed in burn units that have the facilities to provide the best level of care available in the country. This will vary from country to country, but if you know that your burn unit does not have the same level of expertise or equipment as a neighboring unit, it is your moral duty to refer this child onward, providing that the transfer will not be detrimental to the physical or psychological well-being of the child. Dilemmas may arise, for example, if this means separating children from their parents, perhaps who have been injured in the same accident.

WHEN TO DEBRIDE?

Generally, the sooner the better, as the presence of dead tissue has a negative impact on wound healing, and provides a medium for the growth of micro-organisms. Nevertheless, the safety of the patient should be paramount, and debridement must wait until the appropriate staff and other resources are available.

HOW TO DEBRIDE?

The choice of the debridement method to be used would depend on the depth of the burn, the amount of slough, and the experience of the surgeon. The depth of the burn is a notoriously difficult judgment to make, with even the most experienced surgeons often getting it wrong. The clinical assessment of the depth of a burn is only 65–80% accurate. The use of Laser Doppler Imaging can bring this accuracy up to 97% (3). Deeper scald injuries, commonly seen in children, can mislead the surgeon by their "pink" appearance, caused by the coagulated hemoglobin retained in capillaries. Very superficial burns will need nothing more than gentle mechanical cleansing of loose tissue with a wet gauze swab.

For over 30 years, since Zora Jancekovic described her experience of tangential excision, this technique has been the most commonly used method of burn excision. Instruments, such as a Watson or Goulian knife, are used, and thin slithers of tissue are removed until healthy tissue is reached. This technique has the advantage of being fast. The use of adrenaline infiltration and soaks and sterile tourniquets greatly reduce the blood loss associated with this technique. One important disadvantage is that there is a tendency to remove too much tissue. As the skin graft will still take perfectly well, nobody (except perhaps the surgeon) would ever know that salvageable, precious dermis has been thrown away and that a "perfect crime" had been committed. Histologic analysis of the debrided tissue would be illuminating.

Dermatome debridement (4) will lead to less loss of tissue than using a Watson or Humby knife, as the "slice" taken may be thinner. The "saw-tooth" effect around the edges of the debridement will also be lost, as the blade oscillates at a much higher rate than the human arm can manage.

Removal of necrotic tissue using a rotating burr or dermabrader allows even more precision in debridement. This technique is difficult, however, owing to the amount of blood which

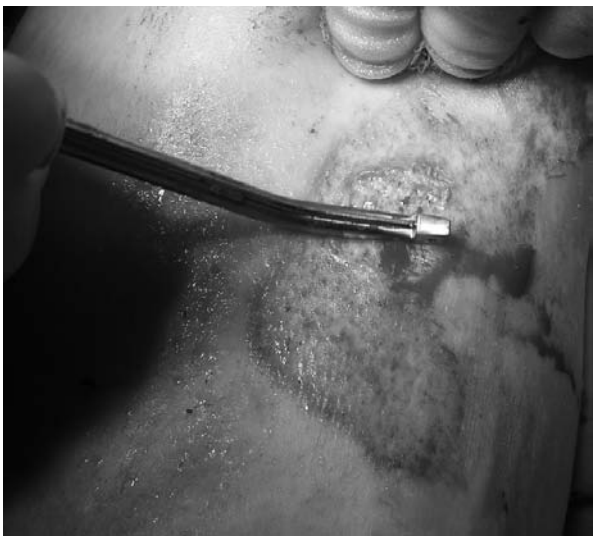

FIGURE 1
Versajet™ (Smith & Nephew, Largo, FL). (*See color insert.*)

is generated. Unchecked, this results in blood spraying throughout the operating theater. To combat this spray, many surgeons use a clear plastic sheet over the area to be treated. This soon becomes opaque because of the spray of blood. Debridement using sandpaper has also been described (5).

The most exciting improvement in surgical debridement recently has been the development of the Versajet™ hydrosurgery system (Smith & Nephew, Largo, FL) (Fig. 1). This allows the precise removal of unhealthy tissue, having the precision of dermabrasion but without the mess, and having the ease of blade excision but lessening the potential for excessive, unnecessary removal of healthy tissue. In many instances, debridement using the Versajet system would allow the retention of dermal elements that would be impossible, if not very difficult, to preserve with conventional blade excision. As the loss of dermis is one of the principal factors that contributes to poor scar outcome, the use of the Versajet to achieve surgical debridement is being associated with better cosmetic results following burn injury. It also greatly facilitates the use of adherent, occlusive dressings, such as Biobrane and Transcyte, which require a very close apposition of the dressing material with the wound bed. This intimate interface between the dressing and the wound bed is easily disrupted by the presence of slough or bacteria.

The fact that using the Versajet, which is held rather like a pencil, requires the action of fine hand muscles rather than the more proximal and coarse shoulder and elbow muscles used when debriding with a Watson knife, reinforces the precision achievable with this technique. The aperture of the Versajet handpiece is much smaller than any conventional surgical knife used in the debridement of burns (11–13), allowing access to awkward places, such as the web spaces of a child's hand, around the malleoli, or on the face. For use in larger burns, it is important to use warmed saline or water with the Versajet to avoid cooling the patient. The addition of adrenaline to the irrigation fluid does not seem to have any advantage.

Like all surgical techniques, there is a learning curve before the surgeon can get the best results. Care must be taken when debriding full-thickness burns with the Versajet system as the burned skin is usually much harder than the underlying fat, and the high-pressure fluid preferentially removes the softer tissue. This can lead to a "scalloping" of the underlying fat, and for this reason, I prefer to excise full thickness burns with a blade. When debriding deep dermal burns, grooving can occur if too much pressure is applied to the burn wound. This can be avoided by using a higher setting with less pressure on the wound. If grooving does occur, it can be remedied by changing the orientation of the Versajet handpiece so that it cuts tissue at 90° to the grooves. The handpiece can also be tilted to 45° to allow feathering of the edges.

Debridement using an ablative laser, such as a CO_2 laser, has been described but is rarely used clinically because of the practical limitations of using this technique in the operating theater.

Enzymatic debridement has been used in many parts of the world. Proteolytic and fibrinolytic enzymes produced by bacteria, such as *Bacillus subtilis* (e.g., Travase) (6,7), or Clostridia species (e.g., Novuxol) (8,9), are applied to sloughy burns. Proponents of this method of debridement claim that unaffected tissue is spared and that subsequent mechanical debridement is easier.

This technique has been troubled by bleeding, pain, wound infection, and bacteremia (10), not helped by the fact that the efficacy of these enzymes are reduced by the presence of commonly used antimicrobial agents, such as silver. These wounds therefore require close monitoring, and prophylactic topical antibiotics have even been proposed for patients undergoing enzymatic debridement. For these reasons, enzymatic techniques have yet to become standard practice in most burns units.

Fascial excision, using scalpel, cutting diathermy, or harmonic (ultrasonic) scalpel is still sometimes required for pediatric burn surgery. This technique does save time and blood loss, but at the expense of poor cosmesis. The harmonic scalpel has been criticized as not being as fast as cutting diathermy, but once the different technique needed to cut the tissue is quickly mastered, I find the harmonic scalpel just as quick as cutting diathermy. The harmonic scalpel appears to cause less damage to the wound bed.

SUMMARY

The management of pediatric burns is both challenging and rewarding. One of the biggest challenges is removing unhealthy tissue while preserving healthy tissue. The biggest reward is knowing that you have succeeded in producing the very best outcome for the child and for their family.

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7 Surgical Debridement of Open Fractures

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INTRODUCTION

Severe open fractures of long bones of the lower extremities still pose a considerable challenge to the treating surgeon. Despite the evolution of the methods of their treatment over the past years, the overall morbidity of these injuries remains significant, especially when severe open fractures are treated by less-experienced surgeons. The goals of the management of open fractures can be summarized as the prevention of deep sepsis, fracture healing, and ultimate complete functional recovery of the injured limb. These goals can only be achieved with close adherence to a strict protocol of treatment, consisting of tetanus prophylaxis, intravenous administration of appropriate broad-spectrum antibiotics, meticulous wound debridement, operative stabilization of the skeletal injury, and early soft-tissue coverage of the open wound (1–3).

HISTORICAL BACKGROUND

Hippocrates was the first to recognize the need for aggressive wound treatment with "iron" when healing did not progress (4). Ambroise Paré (1510–1590), a French army surgeon, advised the enlargement of the wound to facilitate the discharge of the purulent material (5). Desault, in the eighteenth century, adopted the term "debridement" to describe the enlargement and exploration of the wound in order to remove dead tissue and provide drainage. Sir Robert Jones, during World War I, popularized the practice of wound debridement and later on Trueta, during the Spanish Civil War, further added, apart from wound debridement, the splinting of the wounded extremities (5). During that time, traumatic wounds were left open to heal by secondary intention. World War II was characterized by the extensive use of local antiseptic agents to the open wounds. Later on, during Korean and Vietnam conflicts, the systematic use of antibiotics and delayed wound closure, after appropriate initial debridements, were adopted. This accumulated experience from the treatment of war wounds also influenced the establishment of the current principles of acute management of open fractures in a civilian setting.

MECHANISM OF DEEP CONTAMINATION

The mechanism of deep contamination in open fractures is rather complex. The breakage of skin barrier allows direct communication between the fracture hematoma and the outside environment, facilitating the inoculation of pathogens into the deep tissues. Specific environmental exposure is associated with the contamination by specific pathogens; farmyard- and soil-related injuries, for example, are associated with contamination by *Clostridium perfingens*, whereas open wounds having been exposed to the environment of a lake or a river carry the risk of infection by *Pseudomonas aeruginosa* or *Aeromonas hydrophila* (1). Open fractures represent high-velocity injuries that result in the release of a large amount of kinetic energy, according to the equation: $E_k = \frac{1}{2}mv^2$, where E_k stands for the kinetic energy released

TABLE 1
Wound Management at the Emergency Unit

- Gross assessment of the wound
  —History of injury
  —Visual inspection of the wound
  —Photograph
  —Removal of gross contamination
  —Sterile dressing
- Prevention of further contamination
- IV antibiotics
- Tetanus prophylaxis

during the impact, m is the mass, and v^2 represents the square of the velocity. This amount of energy is absorbed by the limb, creating a shock wave within the soft tissues. This shock wave is responsible not only for bony comminution, but also for a variable degree of soft-tissue disruption and stripping. Furthermore, it creates a momentary vacuum within the injured limb, which, in cases of loss of skin barrier, tends to absorb foreign material into the depths of the wound (6).

The presence of a crush component adds to the severity of the injury as it causes further soft-tissue devitalization, increasing the likelihood of septic complications.

The existence of foreign, contaminated material deep into the wound, particularly in the presence of contused and devitalized tissue, creates an optimal environment for bacterial proliferation that would potentially result in septic complications. Therefore, the goals of surgical debridement are the removal of foreign material and bacterial inoculum along with aggressive excision of devitalized tissue.

PRIORITIES IN WOUND MANAGEMENT AT THE EMERGENCY UNIT

The initial priorities in wound management of open fractures are summarized in Table 1. The initial evaluation of the soft-tissue trauma at the Emergency Unit is dictated by the patient's general condition. Open fractures usually occur in polytrauma patients, and the initial management should follow Advanced Trauma Life Support (ATLS) guidelines (6). The open wound should be dressed under sterile conditions, the limb should be immobilized appropriately, and any effort for further assessment and management of the injured limb, apart from the control of active bleeding, should be deferred for secondary survey (7). However, in patients in a stable condition, a gross assessment of the soft-tissue trauma can take place at the Emergency Department. Details from the history of injury could be very helpful in determining the extent of soft-tissue disruption and the degree of contamination. Inspection of the wound can reveal its dimensions and any crush or contusion component at the injury zone, indicative of large amount of absorbed energy and subsequent tissue devitalization and any gross contamination with foreign material (such as soil, etc.). All these should be carefully recorded. A Polaroid photograph of the wound is recommended for documentation of its basic characteristics (8) (Fig. 1A, B). However, grading of the open fracture according to the system proposed by Gustilo and Anderson (9,10) should be avoided at this stage, as it is based on the degree of osseous and deep soft-tissue involvement and not on the superficial characteristics of the wound. Formal wound debridement and irrigation with sterile saline solution should also be avoided. The environment of the Emergency Department is often colonized with nosocomial bacteria, and thus an attempt of wound cleansing could result in further increase of the bacterial load, particularly with resistant micro-organisms (6). Only easily accessible foreign bodies should be removed from the wound before the application of the sterile dressings. Nevertheless, irrigation of the open wound at the environment of the emergency room is advocated by some authors in case of heavily contaminated wounds (2). The practical value of obtaining specimen cultures from the open wound in the emergency room has been questioned, as they usually isolate superficial contaminants or normal skin flora and,

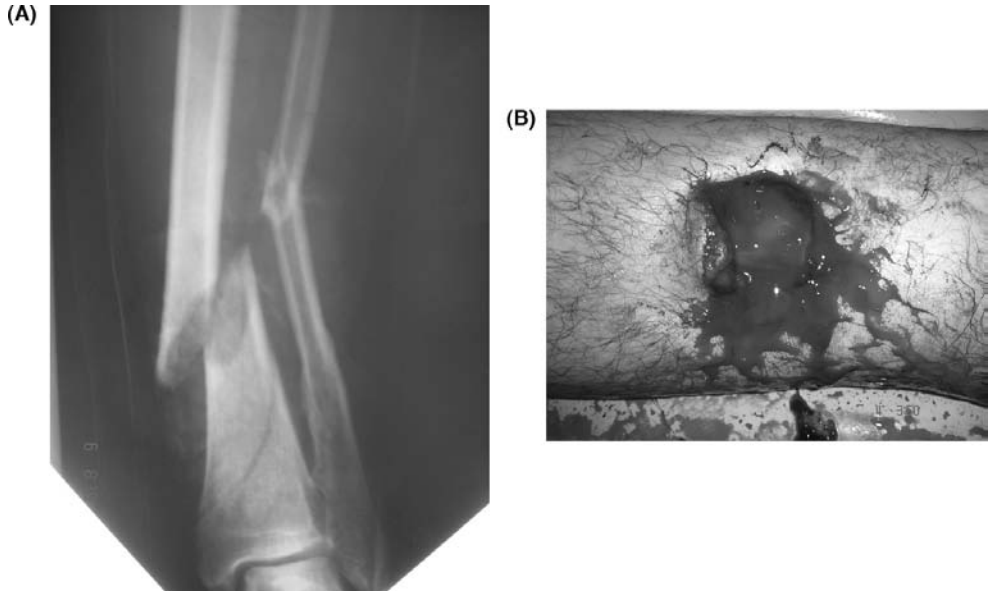

FIGURE 1
(**A**) Anteroposterior X-ray of open tibial fracture; (**B**) photographic documentation of an open fracture.

at the same time, carry the risk of causing wound contamination (6,11). Patzakis et al., in a prospective study of 1104 open fracture wounds, found that although 64.1% of them had positive initial cultures; eventually, only 7.0% of the open fractures became infected. Organisms isolated in the initial cultures were found in 66% of the infected cases (12). Furthermore, a positive postdebridement culture for *C. perfingens* was found to be an increased risk factor for the development of a clostridial infection (12). The high prevalence of microbial contamination of the open fracture wounds predisposes them to the development of infection (13). Thus, the role of prophylactic antibiotic therapy in the initial management of open fractures is well established (9–11,14).The risk of infection and the type of the offending micro-organisms depend on the severity of soft-tissue damage (2,9–11,15).

As the role of both gram-positive and gram-negative micro-organisms in causing deep infections in open fractures has been clearly established, it became apparent that most of the open fractures require combined antibiotic therapy (9,10,14). A second-generation cephalosporin for 48–72 hours seems to be enough for type I open fractures. For type II and type III open fractures, a combination of a second-generation cephalosporin with an aminoglycoside offers the best protection against most gram-positive cocci and gram-negative bacteria or mixed infections. Moreover, the addition of Penicillin G as a third antimicrobial agent is highly recommended for open fractures that have been exposed to soil or farmyard environment and those open injuries with a significant crush component or vascular compromise (14,16). The recommended duration of the aforementioned antibiotic prophylactic scheme is three days, as longer periods of antibiotic therapy have not been proven to offer better protection, where they carry the risk of creating resistant strains (2,9,11,12,14,17).

Some authors recommend the combined use of intravenous and local antibiotic therapy, as it has been shown to decrease the incidence of both acute and chronic infections (18,19). However, the early use of various antibiotic regimens is only a supplement to the meticulous and thorough surgical debridement of an open fracture.

Prophylaxis against tetanus should be considered for all open fractures and follow specific guidelines, according to the immunization status of each individual patient and the tetanus susceptibility of the wound (6).

DEFINITIVE WOUND MANAGEMENT

The following issues should be considered when operative debridement of an open fracture is to be undertaken:

1. Timing of debridement
2. Technique of debridement
3. Optimal use of irrigation

Timing of Debridement

Early surgical debridement and skeletal stabilization are considered the gold standard of open-fracture management. The conventional time period of six hours from injury is used for the distinction between "early" and "delayed" treatment. Intuitively, delayed definitive treatment is expected to be related with an increased risk for septic complications. Kindsfater and Jonassen in a retrospective comparative study of grade II and III open tibial fractures found an increased risk of osteomyelitis in those fractures treated with late (>5 hr) debridement (20). On the contrary, other authors have failed to confirm the aforementioned results (21–24). Surgical delay of up to 13 hours has not been found to increase the incidence of septic complications of open fractures, on the conditions of thorough, and if necessary, repeated debridement and early commencement of antibiotic prophylaxis (25). It seems that the adequacy of the initial debridement rather than timing determines the final outcome (26). Nevertheless, surgical debridement of open fractures should be thought of as an urgent procedure, and can only be delayed in cases of other surgical priorities in polytrauma victims.

Technique of Debridement

Formal debridement requires experience. Ideally, it should be performed by a surgeon well-versed in the techniques of soft-tissue reconstruction. In some trauma centers, a cooperation of orthopedic and plastic surgical teams has been practiced with encouraging results in regard to the adequacy of the procedure (26,27).

A complete and adequate debridement should follow certain steps:

1. Enlargement of the initial wound
2. Resection of nonviable soft tissue
3. Removable of dead cortical bone

Enlargement of the Initial Wound

In open fractures, contamination and soft-tissue devitalization usually extends further off the margins of the initial wound, and casual inspection of the wound is not enough for its complete evaluation. Particularly in high-energy open fractures, the released energy creates a momentary vacuum that tends to absorb foreign material from the surrounding area into the depth of the wound (6). Subsequently, surgical extension of the initial wound is of paramount importance for both sufficient wound assessment and debridement. Extreme caution should be paid, during surgical enlargement of the initial wound, to preserve skin viability and allow subsequent skeletal stabilization. In a transverse or oblique wound, the extension can be performed by making two incisions, beginning on either edge of the wound, and advancing proximally and distally, respectively, in a z-plasty fashion (Fig. 2A–C). These two incisions should be placed in obtuse angles with respect to the initial wound to avoid necrosis of the tips of the skin flaps. The size of wound extension depends on the severity of the soft-tissue injury, and should allow the surgeon to have full access to the whole zone of injury. In grade I open fractures, the location of the fracture dictates the extent of wound enlargement. In the case of a small puncture wound over a subcutaneous bone (such as the anteromedial aspect of tibia), a very little extension is required, as muscle damage is minimal. However, in the thigh region, even a small puncture wound indicates considerable damage of underlying muscle, and a generous enlargement of the initial wound is necessary for a sufficient debridement.

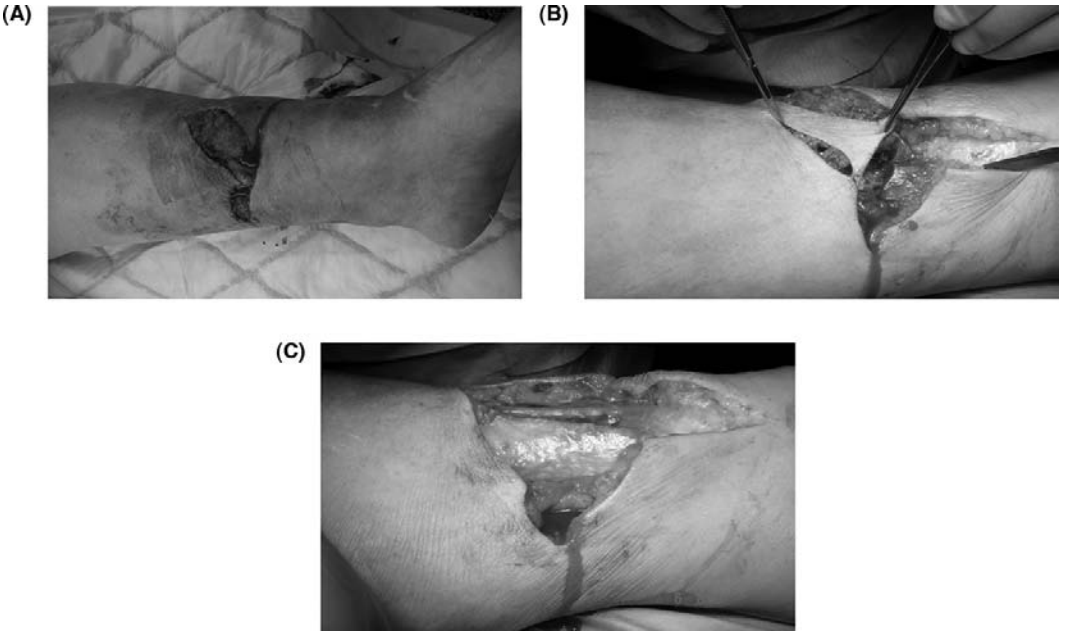

FIGURE 2
(**A**) Open tibial fracture; (**B**) surgical extension of the initial wound in z-plasty fashion; (**C**) exposure of the damage zone.

Resection of Nonviable Soft Tissue

The skin margins of the open wound are debrided first until bleeding tissue. Tourniquet should not be inflated, if possible, throughout the whole process of debridement, as skin and deep soft-tissue viability cannot be determined under tourniquet control. The subcutaneous fat tissue has poor vascularity, dependent mostly on perforating vessels through the underlying fascia. In degloving-type injuries, the avulsed ends of subcutaneous tissue have been rendered avascular and should be meticulously excised. The underlying fascia is then assessed, and all necrotic parts of it are also removed. With respect to tendons, the decision of sacrificing them is not an easy one, as these structures are important for the functional integrity of the injured limb. A completely severed tendon with gross contamination might have to be excised, especially when its respective musculotendinous unit is badly damaged. Otherwise, every effort should be made to remove the contamination, even as preserving the tendon itself. Throughout the process of debridement, the tendon and fascia tissues should be kept in an environment of optimal moisture by regularly irrigating the open wound, as desiccation of such structures would lead to necrosis and render their excision unavoidable. Evaluation of the muscles requires experience and is based on the "four Cs" (28): contractility, consistency, color, and capacity to bleed. Contractility is tested by pinching the muscle belly with a pair of toothed forceps, or by stimulating it with an electrocautery device on a low setting. Viable muscle fibers respond with contracture to either mechanical or electrical stimuli. Consistency is tested by touching or squeezing the muscle with a pair of forceps. Dead muscle disintegrates to touch and it is marked with an imprint when squeezed with a pointed instrument. Contractility and consistency are the most sensitive determinants of muscle viability. On the contrary, color and capacity to bleed are not considered reliable indicators of muscle viability, as local hematoma might render viable muscle discolored, and even dead muscle can produce profound arteriolar bleeding (28,29). All unequivocally dead muscle should be resected. Marginal muscle tissue (that responds weakly to mechanical or electrical stimuli and appears contused) can be left in place for a second wound

inspection within 24–48 hours. Special attention should be paid to muscle–tendon units, as they are very important for limb function. It is surprising that only 10% of the preserved muscle belly is associated with satisfactory function of its respective tendon. Subsequently, the preservation of musculotendinous units, whenever possible, without compromising the adequacy of debridement, should always be attempted (2).

Removal of Dead Cortical Bone

Cortical-bone fragments, deprived of all soft-tissue attachments, act as potential sequestra, providing a substrate for micro-organism growth and eventually leading to the development of deep infection (28–31). Retention of free diaphyseal or metaphyseal bone is risky as it is associated with a higher infection rate (3). The only exception to this rule concerns large osteochondral fragments that are vital for joint function. These should be left in place after being debrided first. It has been shown that the debridement of free bone segments, which are eventually preserved, is associated with decreased prevalence of infection (2).

Optimal Use of Irrigation

Irrigation aims at removing foreign material and contaminating bacteria from the deep soft tissues that potentially could cause deep sepsis. Although irrigation along with sufficient surgical debridement are considered the most important factors in reducing the prevalence of infection following open fractures, significant controversy surrounds some aspects of this procedure, particularly (*i*) optimal volume and type of fluids, (*ii*) delivery systems, and (*iii*) fluid additives.

Optimal Volume and Type of Fluids in Irrigation Systems

The volume of fluids used during irrigation plays an important role in the effectiveness of the procedure, as increased volume of irrigating fluids improves their wound cleansing capacity, at least to a certain point (32). However, the optimal volume of the irrigating fluid is unknown. Anglen recommends the use of at least 3l of fluids for type I open fractures. For types II and III open fractures, the recommended volumes of irrigation fluids are at least 6 and 9l, respectively (32). Most authors recommend either normal saline or Ringer's lactate as irrigating fluids. Museru et al., in a prospective randomized study, compared isotonic saline, distilled water, and boiled water, in the irrigation of open fractures. They concluded that the final outcome was unaffected by the type of irrigating fluid used (33).

Delivery Systems

These include high-pressure lavage (continuous or pulsatile), low-pressure lavage, gravity-flow irrigation, and bulb-syringe (BS) irrigation (Fig. 3A,B). High-pressure irrigation has been considered effective in eliminating the bacterial load in soft tissues (34). The addition of a pulsatile component on high-pressure irrigation was thought of further improving the effectiveness of the procedure. The basic principle of the function of high-pressure pulsatile lavage (HPPL) is

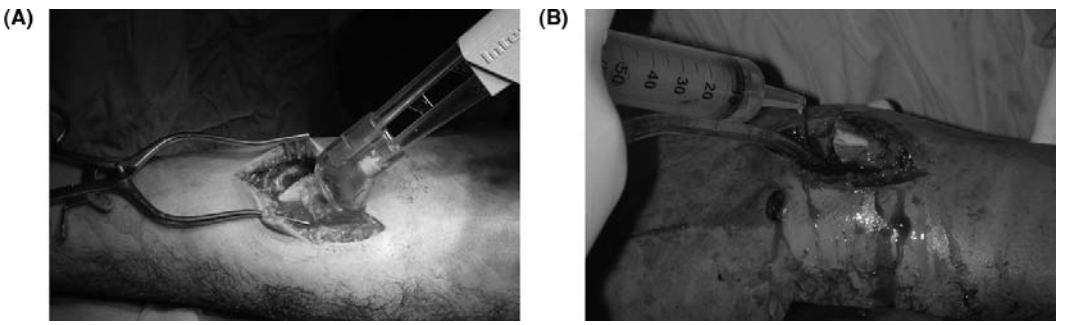

FIGURE 3
(**A**) High-pressure pulsatile lavage (HPPL); (**B**) bulb-syringe irrigation with concomitant use of suction.

the creation of alternating pulse compression and interpulse decompression phases of the soft tissues. During compression phase, the soft tissues are pushed away and displaced, whereas, during the decompression phase, they recoil owing to their elasticity. This recoil of the soft tissues facilitates the dislodgement of particulate debris and bacteria (35,36). In spite of the theoretical advantage of HPPL, its use has been associated with certain complications, such as further soft tissue and bone damage, deeper bacterial propagation within soft tissues, and delayed union or nonunion. Several studies have investigated the association of the various irrigation modalities with the aforementioned adverse effects.

Boyd and Wongworawat tried to quantify and compare the damages caused to fresh bovine muscle tissue by HPPL and low-pressure irrigation. The specimens that were subjected to HPPL suffered deeper soft-tissue penetration and disruption compared with those that were irrigated on a low-pressure setting. Moreover, cellular death was detected at twice as deep level with HPPL than low-pressure irrigation (37).

Draeger and Dahners, in a randomized experimental trial, compared the debridement efficacy and soft-tissue damage of three irrigation modalities, namely HPPL, suction irrigation, and BS irrigation. They concluded that HPPL caused considerably more significant soft-tissue damage compared with the two other irrigation methods. Surprisingly, it was found that both BS and suction irrigations were capable of removing more inorganic contaminant than HPPL. This study supports the concept that sharp debridement and suction irrigation are more efficient in removing particulate debris from deep soft tissues than HPPL (38) (Fig. 3).

Lee et al., in an intra-articular fracture model, found that both HPPL and BS irrigation were equally effective in removing particulate matter from metaphyseal cancellous bone. Furthermore, HPPL was not found to be associated with the inoculation of bacteria farther into metaphyseal cancellous bone compared with BS irrigation (39).

Bhandari et al. compared the effect of HPPL and low-pressure pulsatile lavage (LPPL) on microscopic bone architecture. They also investigated their capability of removing slime-producing bacteria from the surface of cortical bone. They found that although both high- and low-pressure irrigation resulted in a similar degree of periosteal stripping from cortical bone, low-pressure irrigation was associated with less damage to cortical bone micro-architecture. Both high- and low-pressure irrigation were equally effective in removing adherent bacteria at three-hour delay, but only HPPL could efficiently remove adherent bacteria at six-hour delay. These findings indicate that HPPL, although more damaging to both soft and osseous tissue, is more effective in removing the bacterial load of an open fracture in case of delayed treatment (40).

In another study, Bhandari et al., examining the effects of HPPL on contaminated human tibiae in an in-vitro model, found that high-pressure irrigation resulted not only in macroscopic damage to bone, but also was responsible for driving surface bacteria into the intramedullary canal (41).

A recent experimental study by Hassinger et al. compared the degree of bacterial propagation into contaminated soft tissue subjected to either HPPL or low-pressure gravity flow. Their results showed that HPPL was associated with deeper bacterial penetration and greater amount of bacterial retention within soft tissue compared with low-pressure gravity flow (42). The interference of the various irrigation systems with bone healing was addressed in several experimental studies.

Polzin et al. found that irrigation pressure above 50 psi might adversely affect new bone formation and subsequently fracture healing (43).

Adili et al., using an open, noncontaminated femoral diaphyseal fracture model in rats, found an early (three weeks) deleterious effect of HPPL on fracture healing. This effect was not apparent in the late phases (six weeks) of fracture healing. However, firm conclusion for clinical practice cannot be drawn from this experimental study, as the impact of HPPL on fracture healing in the setting of open wound contamination was not addressed (44).

The effect of irrigation on fracture healing in the context of contaminated open fractures was investigated experimentally by Caprice et al. (45). The authors of this study concluded that irrigation either delivered as HPPL or BS irrigation had a clear beneficial effect on fracture healing in the presence of contamination of open fractures (45).

Park et al., in their experimental study, incriminated the use of repeated irrigation and debridement of open fractures as a predisposing factor to delayed union or even atrophic nonunion (46).

In conclusion, it seems that high-pressure irrigation is not always appropriate in treating open wounds, especially when they are not heavily contaminated, and operative treatment is undertaken early. Concerns of further soft-tissue damage or delayed bone healing limit its role in favor of low-pressure irrigation methods. However, in heavily contaminated wounds, especially when operative treatment is delayed, HPPL seems to be more capable of removing the bacterial load of an open fracture.

Fluid Additives

Various additives have been used in the irrigation solutions in an effort to eliminate the bacterial load of an open wound and optimize wound healing. Anglen has classified the various irrigation additives into three classes: antiseptics, antibiotics, and surfactants (32).

Various antiseptics, such as povidone-iodine, chlorhexidine, and hydrogen peroxide, have been used in irrigation solutions as they offer a broad-spectrum activity against most of the bacteria, fungi, and viruses that colonize the wound. The efficacy of antiseptic solutions has been tested extensively and the relative results have been reported in several studies (47–50). Because of questionable capacity to reduce the infection rates of open fractures, potential toxicity to the cellular elements of bone (particularly osteoblasts and osteoclasts) and deleterious effect on wound healing, the use of antiseptic irrigation solutions is generally not recommended (32,51).

Bacitracin, polymyxin, and neomycin have been used as antibiotic additives in irrigation solutions in an attempt to increase the bacteriostatic and bactericidal capacity of the irrigation solution and eliminate septic complications (52). Nevertheless, their clinical efficacy has not been proved unequivocally, and concerns regarding the cost of their use, serious, though rare, toxicity, and promotion of bacterial resistance limit their role in optimal open wound care (53).

Examples of surfactants include liquid soap and benzalkonium chloride. The addition of detergents in irrigation solutions in the treatment of open wounds was once practiced by several surgeons (54,55). Their main advantages were attributed to the interference with the adherence of bacteria to surfaces and also to their capacity to emulsify and remove foreign material from the open wound (32). The introduction of antibiotics as an integral part of standard wound care has supplanted the use of the various soap solutions. Nevertheless, the observation that antibiotic irrigation solutions produce foaming led to the speculation that their efficacy may be attributed to a detergent-type action (51). This hypothesis has been tested experimentally, and it was found that soap solutions were more effective than antibiotic solutions in removing slime-producing staphylococcus from metallic surfaces (56,57). The efficacy of a sequential irrigation protocol consisting of benzalkonium chloride followed by castile soap and normal saline in reducing the rate of positive wound cultures has been tested experimentally (53,58). The results of these experimental studies indicated a potential therapeutic value of the sequential surfactant irrigation in treating infected orthopedic wounds, although further studies should be undertaken to clarify the exact role of the sequential irrigation protocol in clinical practice. At present, surfactant irrigation should be considered in highly contaminated wounds, particularly as first irrigation (32).

LATEST ADVANCES—FLUIDJET TECHNOLOGY FOR THE DEBRIDEMENT OF OPEN FRACTURES

Recent advances in hydrosurgery facilitated the development of a new system for the simultaneous irrigation and debridement of open fractures (59). The Versajet™ system (Smith & Nephew, Largo, FL) uses pressurized saline in a sterile circuit based on the Venturi effect: high-pressure waterjet is pushed through a suitable hose to the tip of a procedure-specific handpiece. The water executes a 180° turn and is forced out of a miniscule nozzle. The water jet passes parallel to the wound and is captured by an evacuator port 8–14 mm from the nozzle. The waterjet runs at a variable speed and pressure ranges from 265 to 670 mph and from 103 to 827 bar, respectively, depending on the 10 speed setting on the console. The surgeon can safely control the cutting, debriding, and aspiration effects by adjusting the console power settings and by angulating the hand piece (60). The flow carries the waterjet, ablated tissue, and debris into the evacuator port without the need for separate suction.

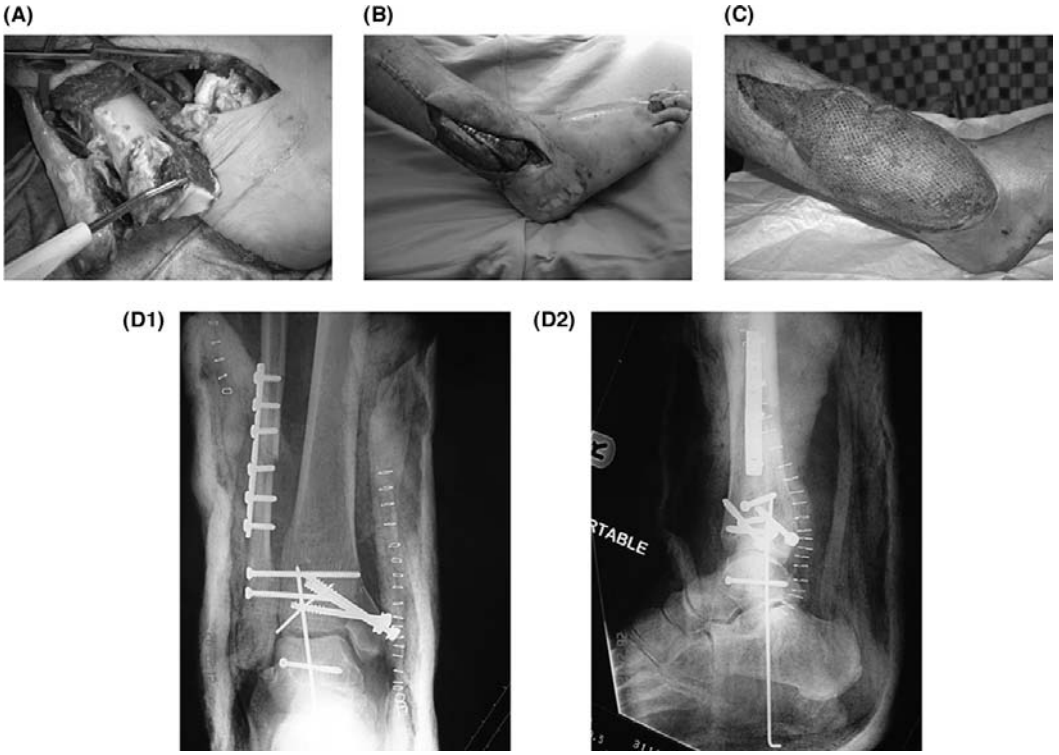

FIGURE 4
(**A**) Surgical debridement of a grade IIIb open distal tibial fracture using the Versajet system; (**B**) wound prior to free tissue muscle transfer; (**C**) wound coverage with a latissimus dorsi muscle flap and split skin graft; (**D**) radiographs taken following the stabilization of the fracture; D1: anteroposterior radiograph; D2: lateral radiograph. (*A–C: See color insert.*)

This new surgical tool has been used for the debridement of over 25 open-grade IIIB lower-limb fractures in our institution. The Versajet machine was used at a variable setting based on the demands of each case and the state of the soft-tissue envelope. In addition to the irrigation and removal of devitalized soft tissues, the Versajet was found to be very successful for the debridement of bone (small fragments requiring excision). Furthermore, the removal of foreign bodies and debris, in general, from the cancellous part of the bone and the intramedullary canal areas was very efficacious (Fig. 4A–D). The mean time of utilization was 4.5 min (range 4–9). There were no bleeding episodes observed during and after the surgical procedures. All the fractures were subsequently covered with free muscle-tissue transfer. All open fractures progressed to union. In this high-risk case series, the overall incidence of superficial infection and deep sepsis was 8% and 4%, respectively. Previous studies have reported a variable incidence of infectious compilations after open-grade IIIb tibial fractures ranging between 7% and 35% (61). The Versajet was found to be easy and friendly to use. It is a useful adjunct to the surgical debridement of open fractures, especially the ones associated with high-energy and heavily contaminated wounds.

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8 Debridement of Acute Traumatic Wounds (Avulsion, Crush, and High-Powered)

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INTRODUCTION

Open fractures, particularly those that have been caused by high-energy impact, or which involve crushing, pose considerable management problems to the surgeon. While stabilization and fixation are essential to restore function to the affected area, these processes also increase the risk of deep infection which may lead to amputation or even death. Immobilization with casts and traction is less invasive and carries a smaller risk of inducing infection, but may increase the risk of compartment syndrome and pulmonary embolism. While there is an overall accepted management protocol—prophylactic administration of antibiotics, meticulous debridement, copious irrigation, stabilization of the injury, early soft tissue coverage—each patient presents with a unique set of problems. The surgeon constantly has to balance one approach against another, trading off a possibly increased risk of infection for better stabilization, or earlier functionality, or balancing the need for early closure against the need for recovery of soft-tissue injuries.

One aspect of the care plan, however, is constant: the need for thorough, comprehensive debridement of the injured area to limit the development of infection. This should take place not just on admission, but possibly many times afterwards until the surgeon is confident that all necrotic tissue has been removed.

HIGH-ENERGY WOUNDS: OPEN FRACTURES (FIGS. 1 AND 2)

These are among the most severe wounds seen in emergency and orthopedic units and generally fall into the Grade III classification in the Gustilo classification scheme for open fractures (1). High-velocity guns, automobile accidents, falls from significant heights, and crushing by

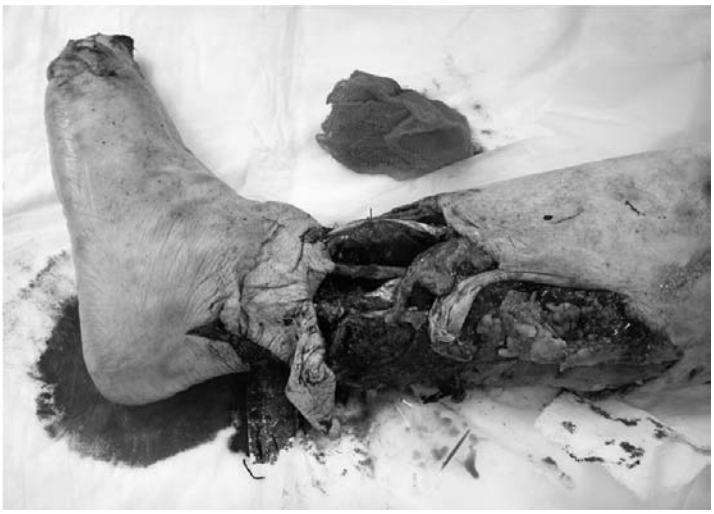

FIGURE 1
Grade IIIC distal tibia injury of 57-year-old involved in high velocity motorcycle accident.

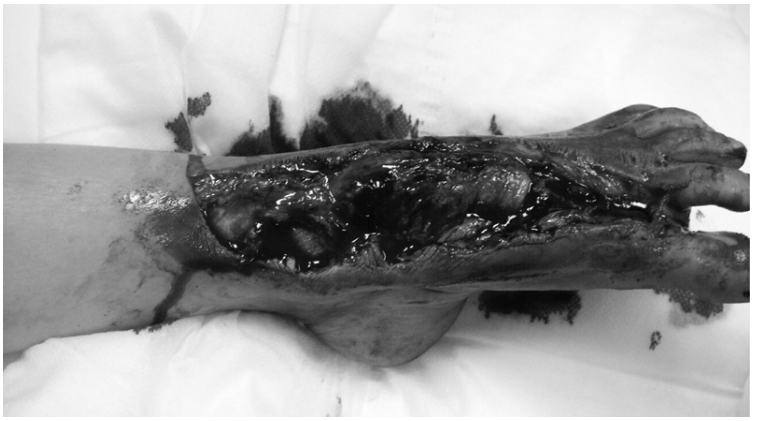

FIGURE 2
Grade IIIB foot injury of 9-year-old run over by an ice cream truck.

heavy machinery often produce open fractures with multiple displaced fracture fragments, extensive soft tissue damage, periosteal stripping, neurovascular injury, dislocations and multisystem trauma. The soft-tissue envelope may be severely contused or crushed and is commonly breached, allowing external contamination through the wound.

Gunshot wounds from high-energy weapons are not necessarily more serious than other forms of high impact wounds, but may initially appear to be more complex owing to the multiple effects on tissue and bone. Damage is caused owing to the transfer of kinetic energy from the projectile to body tissues. There are three mechanisms of tissue damage because of bullets: laceration and crushing, shock waves, and cavitation (2). Laceration and crushing are caused by the projectile displacing the tissues that lie in its track and are generally recognized as being the primary wounding mechanism produced by handguns (3). The degree and extent of laceration and crushing are related to the velocity and shape of the missile, the angle of impact and yaw (deviation from flight path), and the degree of tumbling of the projectile (2) (Figs. 3, 4).

Shock waves occur because of the compression of tissues that lie ahead of the bullet. They are only generated by high-velocity missiles with a speed of at least 2500 feet per second and are therefore rarely a factor in most handgun wounds, but are encountered in wounds caused by high-velocity rifles.

Cavitation occurs when the kinetic energy imparted to the tissues forces them forward and in a radial direction, with this displacement producing a temporary cavity in its wake. The temporary cavity lasts a few milliseconds and then collapses into the permanent cavity generated by the bullet (4). The wounding effect of the cavitation phenomenon is only significant at missile velocities exceeding 1000 feet per second and has been used to explain the fracturing of bone not in the direct path of a missile (5) but this is a controversial view which is not universally accepted (6).

The amount of kinetic energy possessed by the projectile is not the only factor that determines the extent of injury. Soft, elastic tissue does not significantly retard the projectile, which may therefore pass through the skin or organ with relatively little collateral damage. Bone, however, is much denser and causes rapid deceleration of a bullet and transfer of a large amount of kinetic energy leading to complete shattering of the bone at the point of impact. Cancellous bone usually suffers less damage than more compact cortical bone as kinetic energy can readily dissipate within its honeycomb structure (4).

In addition to the primary damage caused by the missile, bone fragments often function as secondary projectiles, further disrupting the tissue. These multiple mechanisms of injury are responsible for producing what can seem, at first sight, a very complex wound. However, the method of treatment is the same as that for other severe open fractures, including prophylaxis for possible contamination, coverage, and stabilization.

Crush injuries most commonly occur in automobile accidents, industrial or farm incidents, severe falls, and major incidents such as falling buildings. The severity of the damage is related

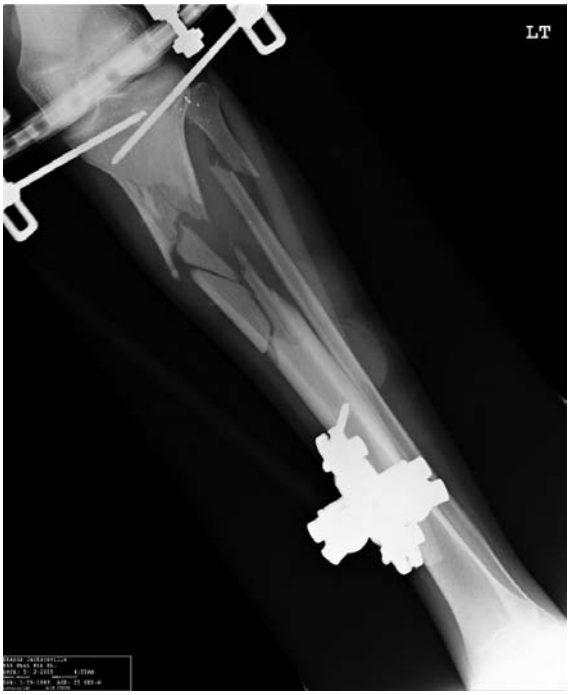

FIGURE 3
Anteroposterior radiograph of Grade IIIB proximal tibia injury after high velocity GSW (AK-47).

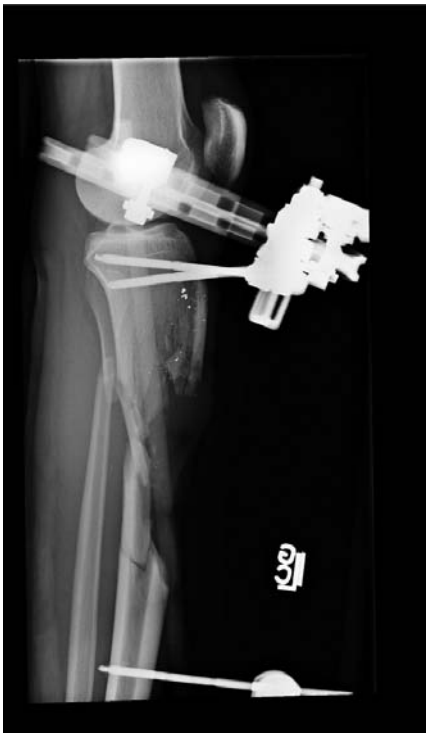

FIGURE 4
Lateral radiograph of Grade IIIB proximal tibia injury at high velocity GSW (AK-47).

to the amount of force applied, the length of time the force was applied, and the type of tissue or muscle that was compressed. Prolonged compression causes ischemia and anoxia of the tissues, eventually leading to necrosis or death of the tissue and muscles. Swelling increases pressure on the muscles and tissues and causes additional ischemia and anoxia. Crush injuries have a particularly poor prognosis owing to the threat of ischemia and the increased likelihood of developing sepsis.

A number of syndromes with significant complications can follow from severe crush injuries. Compartment syndrome is time-dependent and life-threatening and requires surgical intervention to perform a fasciotomy. Rhabdomyolysis can develop because of the breakdown of muscle and subsequent release of myoglobin, creatinine phosphokinase, and various inflammatory mediators. Finally, fat embolism syndrome (FES) and acute respiratory distress syndrome may occur following the release of intravasated debris. Fat emboli are believed to cause aggregates that obstruct the pulmonary microvasculature and cause ventilation perfusion mismatching (7).

GOALS OF TREATMENT

Owing to the severe and extensive nature of high-energy fractures, patients usually need to be treated according to Advanced Trauma Life Support Guidelines of the American College of Surgeons, as they may have associated life- or limb-threatening injuries, before attention is directed to the fracture site. Vascular injuries can cause muscle necrosis within six hours and immobilization or surgical intervention will be necessary to restore arterial or venous flow.

While low-velocity, low-energy injuries can often be treated nonoperatively with local wound care and outpatient management, soft tissues play a more important role in the management of high-energy fractures. Surgery is essential to correct displacements, restore vascularity, and repair tissue but may have to be delayed because of the condition of the soft tissue. Preliminary stabilization can be carried out using an external fixator to prevent contractures and maintain alignment, until the soft tissue is in a condition to accept definitive surgery. Control of infection is vital, as the development of deep infection is a major risk factor for amputation. Antibiotics are probably only required for grossly contaminated wounds, but as it is difficult to assess the degree of contamination most surgeons will use routine prophylaxis. High-energy injuries require aggressive irrigation and debridement and a search for foreign material.

DEBRIDEMENT OF HIGH-ENERGY FRACTURES (FIGS. 5–7)

The aim of debridement is to remove foreign bodies and contaminated material from the wound, and to excise devitalized tissue and bone. The presence of foreign bodies in any open wound

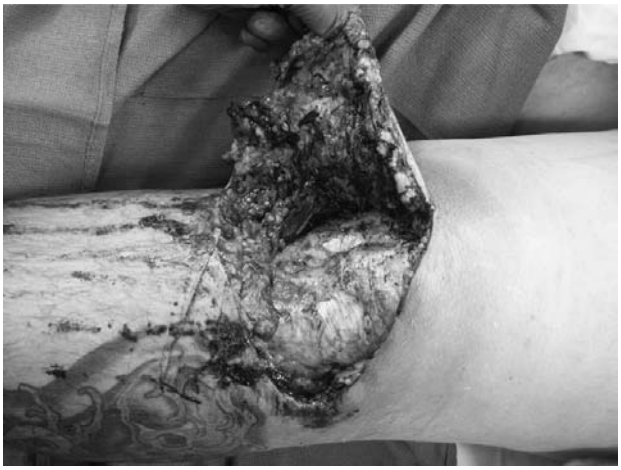

FIGURE 5
Soft tissue knee injury after "wood chipper projectile." (*See color insert.*)

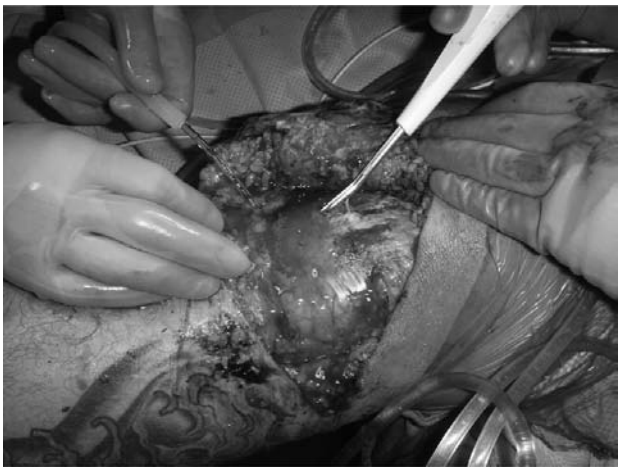

FIGURE 6
Versajet™ debridement and gravity irrigation. (*See color insert.*)

increases the risk for bacterial proliferation, but this risk is increased in severe fractures where the surrounding tissue is contused and devitalized.

TIMING OF DEBRIDEMENT

Surgical debridement of open fractures is a mainstay of treatment and it therefore seems logical to carry it out as soon as possible to minimize the risk of infection. However, owing to the contaminated environment of most emergency rooms, irrigation and immediate debridement is rarely recommended; it is preferable to wait until the patient can be taken into the sterile conditions of the operating theater.

Little research has been carried out on the subject of the timing of debridement, but a study by Kindsfater and Jonassen (8) suggests that the risk of infection increases significantly if debridement is carried out more than five hours postinjury. In a group of Grade II and III fractures, 7% (1/15) of fractures debrided within five hours became infected while 38% (12/32) of fractures debrided beyond five hours became infected. Increasing the length of time to debridement allows more time for colonization of the wound, and delay can also allow local spread of

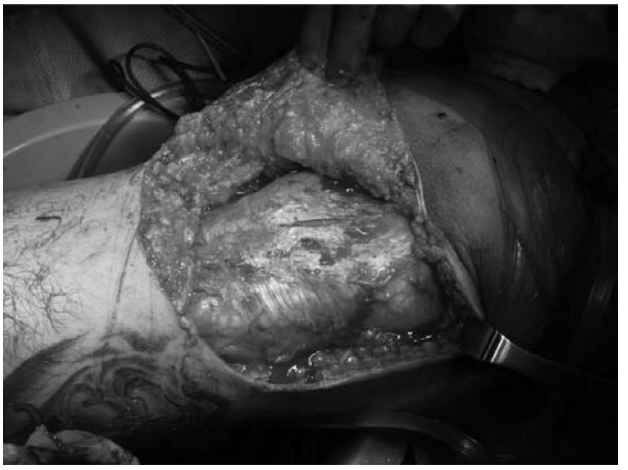

FIGURE 7
Final appearance after debridement and irrigation. (*See color insert.*)

contamination, making it more difficult to adequately debride the wound. Furthermore, increasing the length of time that the bone is exposed or stripped of soft tissue allows more time for the tissue to desiccate and prolongs the time that the bone has to survive with an impaired blood supply. Other authors however have not replicated these results and suggest instead that there is a window of up to 13 hours during which delay does not appear to increase the incidence of infection in open fractures (9–12).

EXTENT OF DEBRIDEMENT

High-energy fractures usually involve injury that extends beyond the margins of the visible wound; therefore, extension of the wound is the first step to assess the need and extent of debridement. While full access to the area of injury is required, this must be balanced against the need to preserve the viability of the skin as far as possible.

All soft tissues must be assessed for viability and should be removed if there are obvious signs of necrosis or lack of vascularity, or if salvage is clearly impossible. The greatest difficulty in assessing viability arises with the muscle, as dead muscle can still bleed if there is severe arterial injury and viable muscle may appear discolored if there is local hematoma (13). Contractility and consistency are more reliable indicators of the viability of the muscle and can easily be tested with a pair of forceps. As with all debridement, it is often difficult to assess the extent to which it should be carried out. Owing to the importance of muscle for limb function, muscle that responds weakly to mechanical or electrical stimuli should be left in place and assessed at subsequent debridements.

Necrotic bone or bone that is at risk should be debrided until bleeding edges are seen. There has been considerable discussion in the literature regarding the approach to debridement of devascularized cortical bone fragments. The argument for leaving them in situ is that mechanical integrity of the internal fixation and eventual limb length may be improved, but often at the cost of deep wound infection, which typically would occur in up to 25% of the patients. Removal of all necrotic bone prior to external fixation and wound coverage typically results in much lower infection rates of around 9% (14). With improved fixation techniques, and given the extremely serious consequences of deep bone infection, it is now generally accepted that all bone fragments should be removed. Where bone fragments are vital for joint function they can be left in place following meticulous debridement.

Tissue necrosis may not be obvious in the initial surgery but may become apparent on redebridement; therefore, staged surgical debridement should be planned every 24 to 48 hours with delayed wound closure until all compromised tissue is removed.

DEBRIDEMENT TECHNIQUES

The sharp knife and curette are standard debridement tools for the surgeon and are highly effective for the debridement of large areas and for the removal of hard eschar and callous. However, sharp debridement is difficult to carry out in confined spaces and can sometimes be an insensitive technique, resulting in the unwanted removal of healthy tissue.

Water jet dissection has been used in liver, kidney, and laparoscopic surgery for some time (15–18) but a new tool for tangential excision—the Versajet™ Hydrosurgery System (Smith & Nephew, Largo, FL)—has recently become more widely available as a method for excision of various open wounds (19). In this system, a jet of pressurized saline travels parallel to the wound surface across the operating window of the handpiece and then into a suction collector, along with the debrided tissue which is carried in by the Venturi effect.

The fluid jet is accelerated through a constricted opening with a corresponding decrease of pressure, which results in a suction effect that lifts and removes contaminants from the wound site without requiring external suction. This reduces spillage, maintains good visibility, and minimizes overload of the tissues with fluid. The suction effect also makes it possible to "hold" the tangential tissue as if by forceps while the high-pressure jet cuts the tissue.

Other surgeons who have used this innovative system report that it removes particles faster than conventional debridement with a knife (20). It is also reported that the Hydrojet system does not drive infected material further into the wound (21). The design of the handpiece allows for

more precise debridement, particularly of areas such as deep cavities and avoids the risk of over-aggressive excision of healthy tissue. Meticulous knife debridement still often results in an uneven wound surface, but with the fluid jet technique there is a consistent and reproducible degree of debridement. There also appears to be less bleeding, and therefore less use of electrocautery (22).

IRRIGATION

A number of irrigation techniques have been developed: continuous high-pressure lavage, high-pressure pulsatile lavage (HPPL), low-pressure lavage, gravity flow irrigation, and bulb syringe irrigation.

It was established very early on that the efficiency of wound irrigation is substantially improved by delivering the irrigant solution under continuous high pressure. Rodeheaver et al. (23) established in 1975 that irrigation of the wound with saline delivered at 15 psi removed 85% of bacteria and other contaminants from wounds. Sufficient bacteria can be removed to restore the wound to a noninfected state, so long as the pressure is high enough to mechanically disrupt micro-organisms.

It is generally accepted that HPPL produces significantly more microscopic damage than low-pressure pulsatile lavage (LPPL), resulting in larger and more numerous fissures and defects in the cortical bone (24). Both types of irrigation produced similar degrees of periosteal separation from the cortical bone surface and were equally effective in removing adherent bacteria at a delay of three hours, but only HPPL was able to remove adherent bacteria from bone at six hours' delay. The microscopic evidence of a deleterious effect of HPPL on bone does not however translate into a clinically relevant decline in bone strength after six weeks of fracture healing (25).

There have also been concerns that high-pressure water jets can cause further tissue and bone damage and can drive bacteria deeper into the soft tissues. Many studies have been carried out to assess the relative impact of high- and low-pressure irrigation on bacterial load and tissue integrity, and these are summarized in the preceding chapter of this book (chap. 7).

INFECTIONS OF OPEN FRACTURES

A major factor influencing fracture repair is the development of infection and is the most common and the most severe complication affecting attempted limb salvage (26). In many cases, osteomyelitis leads to amputation (8). Infection frequently develops despite prophylactic administration of antibiotics. Brueckmann and Roberts (27) noted infection rates of 24% in type III fractures where patients had been administered cephalosporin, plus penicillin for farm injuries.

The incidence of infection in open fractures varies with the amount of damage to the soft tissues with infection in type III fractures being far more common than in types I and II. The highest probability of infection is after high velocity, open injuries with skin necrosis or skin loss and following open reduction internal fixation (ORIF) with failed flap.

Overall, the incidence is around 25%, and higher if internal fixation is used (1). Following the refinement in classifying type III fractures (Table 1), Gustilo reported in 1990 that type IIIB fractures were most prone to develop infection, reporting rates of up to 50% in this type of wound. Increased wound sepsis and amputation occurs where the bone is exposed from loss of soft tissue and periosteal stripping, which usually takes place with massive contamination (28).

TABLE 1
Gustilo Classification of Type III Fractures: Worsening Prognosis from A to C

Type IIIA	Adequate soft-tissue coverage of a fractured bone with extensive soft tissue laceration or flaps, or high-energy trauma irrespective of size of wound.
Type IIIB	Extensive soft-tissue injury with periosteal stripping and bony exposure. Usually associated with massive contamination.
Type IIIC	Open fracture associated with arterial injury requiring repair.

Green et al. (29) stressed the importance of asepsis in the healing of tibial fractures, and reported that 51% of nonunion patients were draining at initial evaluation. Intuitively, it seems that many factors may contribute to the development of infection, such as the time to debridement, antibiotic therapy and so on, but the most important contributing factor has been found to be the severity of soft-tissue injury; fracture severity is the major significant risk factor for nonunion (30,31).

MANAGEMENT OF INFECTION USING A HYDROSURGERY SYSTEM AND A SILVER-COATED DRESSING

A novel approach to preventing infection in open fractures is currently under investigation by the author (32). Twelve grade II and grade IIIA/B open fractures were treated with hydrosurgical debridement using the Versajet high-pressure system described previously, as well as gravity irrigation on soft tissue and bone. Patients were all treated between six and eight hours after injury. Following debridement, a silver-impregnated barrier was placed directly on the bone and around external fixation pin sites. The barrier Acticoat® (Smith & Nephew, Largo, FL) consists of a nanocrytsalline silver layer with confirmed broad-spectrum antimicrobial properties, including methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant enterococci (VRE). Repeated debridement was carried out between 48 and 72 hours after injury.

The overlying skin was loosely closed over the silver dressing which was removed at subsequent debridements. The procedure was repeated until final stable fixation surgery was performed, at which point the silver dressing was removed from the bone and fresh dressing was placed over the incision as a postoperative dressing (Figs. 8–10).

Cultures were taken at repeated debridements but there was no bacterial growth after 72 hours. Neither was there any clinical evidence of superficial or deep infection four weeks after the final stable fixation. Where temporary stabilization was required using external fixation, there was no evidence of pin site infection. Although this was only a case series on 12 patients, the initial results of this novel approach looked encouraging.

MANAGEMENT OF INFECTED NONUNIONS (FIGS. 11 THROUGH 14)

Although infection rates can be high in certain types of wounds, chronic osteomyelitis and possible amputation can be avoided with early diagnosis and immediate treatment.

Management of infected nonunions of the tibia always begins with elimination of infection by further debridement of all necrotic tissue, antibiotic therapy, and appropriate intervention

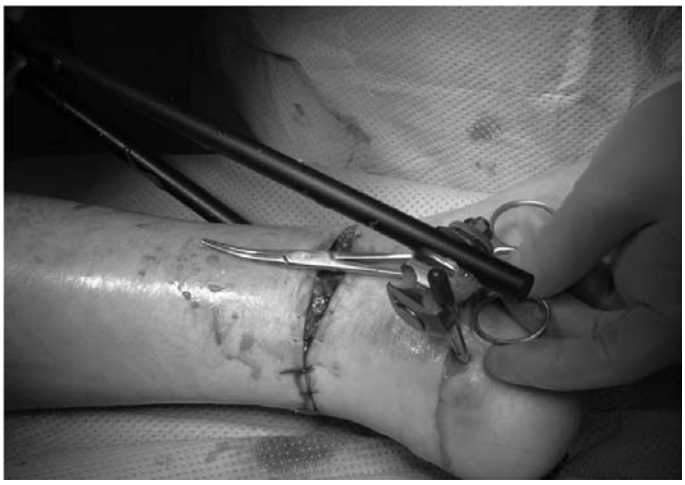

FIGURE 8
Grade IIIA distal tibial injury (clamp demonstrating length of undermined soft tissue).

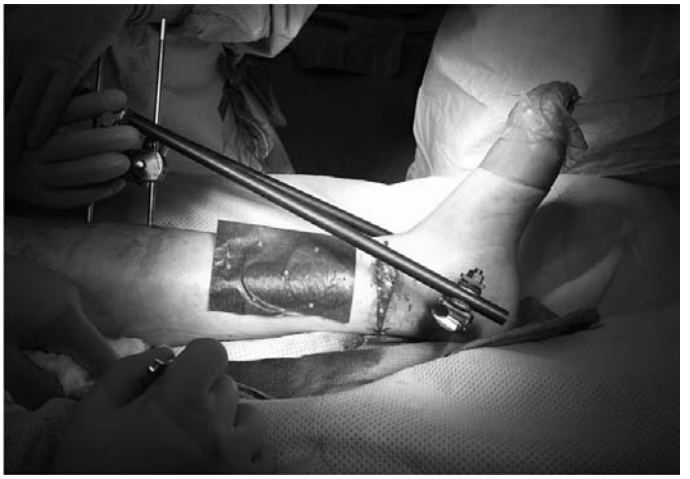

FIGURE 9
Size comparison of Acticoat™ sheet to wound opening.

such as secondary osseous reconstruction with cancellous bone grafts (29). Debridement must be thorough and comprehensive with the removal of all nonviable infected tissue: dead bone is a medium for bacterial colonization and inadequate debridement of devitalized bone is a risk factor for chronic infection (33,34).

While all surgeons would agree that radical excision of all devitalized soft tissue and bone is essential, there is difficulty in defining the border between dead and live tissues. Some surgeons have recommended using intravital dyes to help define the border (35) but in most cases, surgeons can use the presence of spot bleeding from cut surfaces—including bone—to be an obvious sign of vascularized and live tissue.

Some authors have not found antibiotics to be especially useful in the management of infected nonunions. Green et al. (29) in a review of 70 patients found that there was no difference in outcome between the group that received proper antibiotic therapy and those that did not.

There is also a question as to whether metal implants should remain. Damholt (33) reported that there was a stratum of devitalized tissue around metal implants which could hinder cleaning of the infected area. These were therefore removed and external fixation was used at some distance from the infected area.

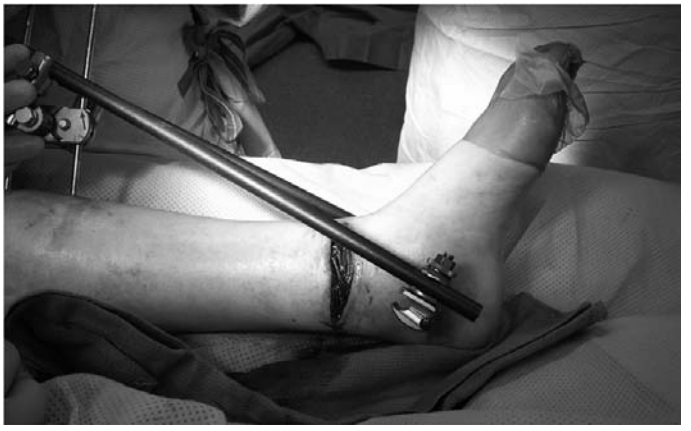

FIGURE 10
Placement of Acticoat™ sheet into entire soft tissue space (see Fig. 8).

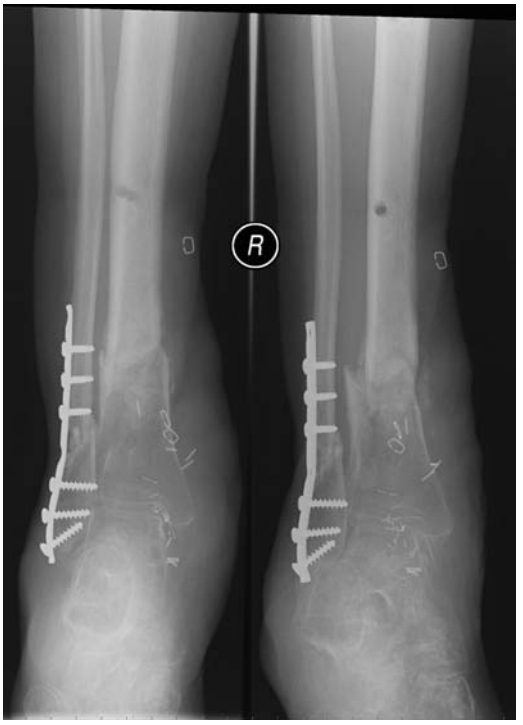

FIGURE 11
Anteroposterior and Mortise radiograph of 34-year-old with Grade IIIC distal tibial injury, one year after initial injury.

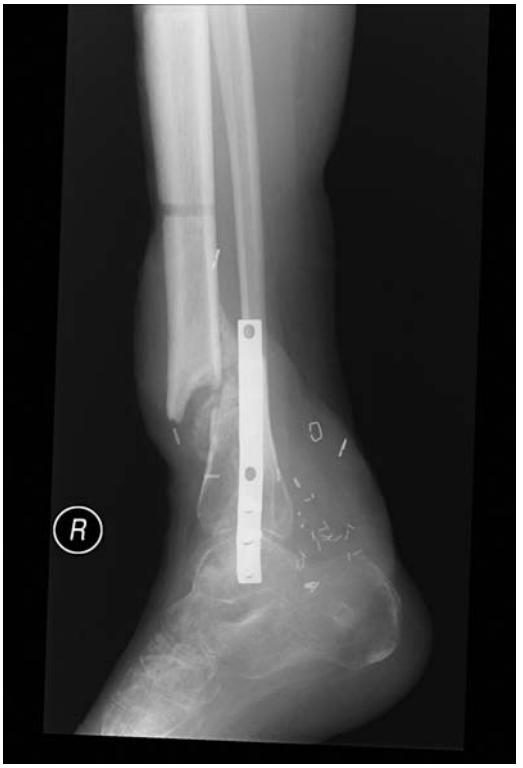

FIGURE 12
Lateral radiograph demonstrating infected nonunion.

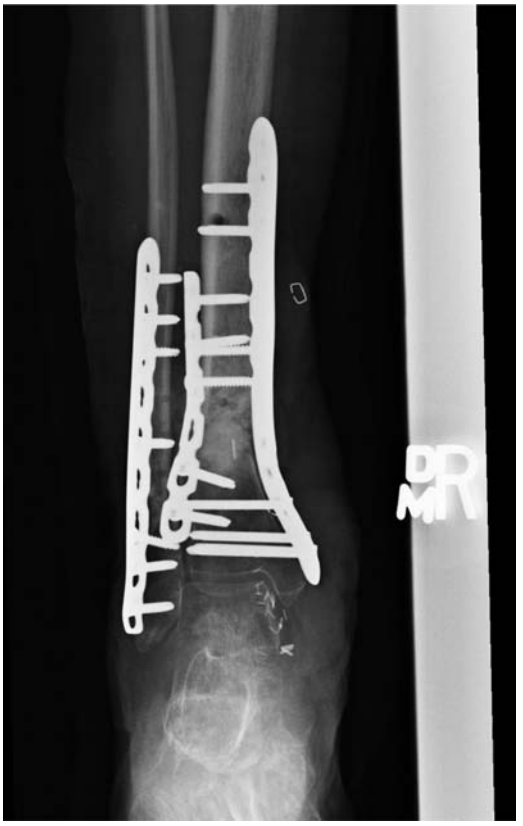

FIGURE 13
6 month post-operative anteroposterior radiograph after debridement, treatment of infection, bone grafting and open reduction internal fixation.

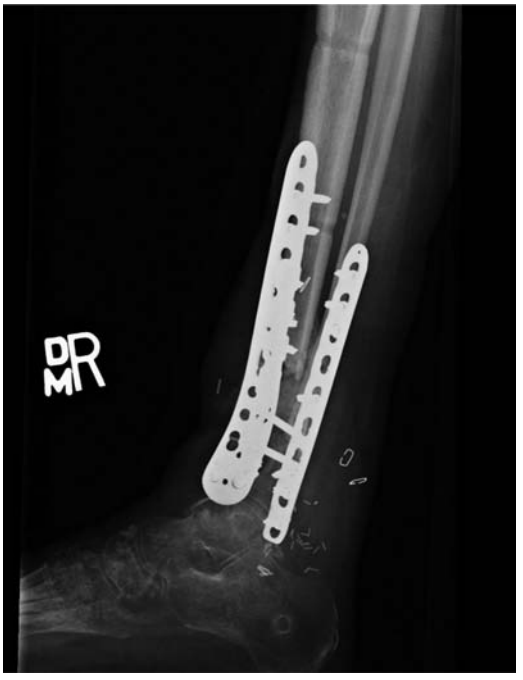

FIGURE 14
6 month post-operative lateral radiograph demonstrating healing nonunion.

CONCLUSION

Fractures caused by high-energy mechanisms (automobile, high falls, gunshot wounds, heavy crush, and the like) often result in significant soft-tissue damage as well as complex fractures. However, as with any open fracture, treatment consists first of all in life-saving measures for the individual, particularly if there are multiple injuries. This is followed by debridement and cleaning of the wound as soon as is practically possible, in order to minimize the risk of infection. The soft tissues must be allowed to recover before any attempt at surgical fixation is made. As infection of a fracture is the most serious, and possibly life-threatening, debridement must be meticulous and comprehensive and will almost certainly be carried out at least twice before fixation of the fracture. The author describes a recently introduced method of debridement using a water jet system, the Versajet Hydrosurgery System, and also describes a new way of using silver-coated dressings, placed on the bone, to limit infection.

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9 Wound Bed Preparation Prior to Flap Coverage

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INTRODUCTION

Preparation of a wound for flap coverage encompasses several aspects of surgical decision making. When addressing an acute or chronic wound, the surgeon must use a tailored approach based on experience, available technology, and patient characteristics. Meticulous debridement of the wound bed is essential to minimize the risk of wound breakdown or flap failure while viability of the flap is a vital part of ensuring successful flap transfer. Figure 1 suggests the decision-making process for operative wound evaluation and debridement where necessary.

PATIENT PREPARATION

Proper preparation of the patient prior to any surgery is essential to reconstructive success, as preventable circumstances may place the reconstruction in jeopardy. All good wound preparation begins with a detailed history, physical examination, and appropriate laboratory investigation.

It is important to identify patients with a medical history of disease processes that may inhibit wound healing. Common examples are poorly controlled diabetes, malnutrition, peripheral vascular disease, immunosuppression, and cardiovascular disease. Although these chronic illnesses can destabilize wounds, proper attention can minimize their adverse effects.

MEDICATIONS

Medications for concomitant diseases can also have an adverse effect on wound management and should be reviewed prior to surgery. Any medication that decreases the immune response, delays wound healing, or thins the blood should be discontinued and alternatives should be found. For example, corticosteroids inhibit wound healing by delaying the appearance of inflammatory cells, fibroblasts, deposition of ground substance, regeneration of capillaries, contraction, and epithelial migration through diminishing serum levels of TGF-B and IGF-1. Oral Vitamin A, 25,000 units twice daily, can be given to counter the inhibitory effects of corticosteroids.

LABORATORY INVESTIGATIONS

Laboratory investigations are important in the decision making process of the reconstructive surgeon. For instance, if a patient is malnourished and in a catabolic state, then it is unlikely that a debrided or reconstructed wound will heal. The incidence of malnutrition in hospitalized patients approaches 50% and accounts for a significantly increased complication rate following any type of surgery

Serum, albumin can be used to assess nutritional status in the chronically ill patient. Albumin has a normal value of 3.4 to 5.4 g/dl; a value of <3 g/dl generally reflects chronic severe malnutrition and is strongly correlated with poor wound healing and a high complication rate.

Pre-albumin is a superior nutritional screening and assessment tool compared to serum albumin. The normal value is 15 to 35 mg/dl and, with a short half life, it is an excellent barometer for short term nutritional status. A pre-albumin of <10 mg/dl is an indicator of malnutrition and poor wound healing potential. These individuals should receive nutritional supplementation, nutrition team consultation, and delay of surgery until improved.

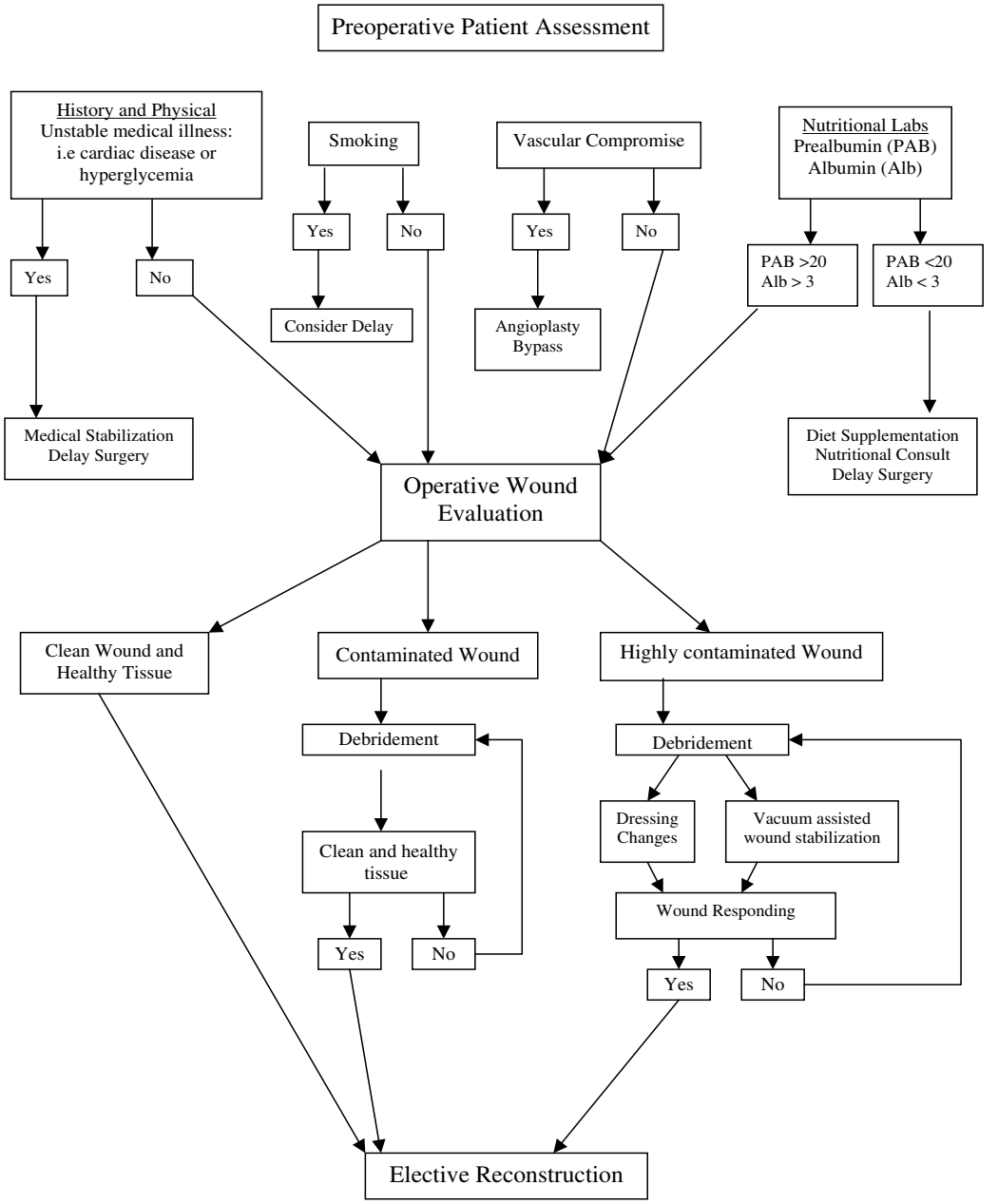

FIGURE 1
Wound care flowchart.

DEBRIDEMENT

In the past, debridement was a cautious process with the surgeon attempting to strike a balance between removing non-viable tissue and maintaining the integrity and coverage of vital structures within the wound. This often led to inadequate debridement and subsequent development of a chronic wound.

Residual necrotic tissue left in and around a wound will stall the inflammatory process and act as a nutritional source for bacteria, thus allowing organisms to proliferate and colonize, sometimes within the protection of a biofilm. As bacterial load increases, so does the potential for infection.

Quantitative cultures can be used to identify bacteria and infection within a wound. Ideally, the bacterial load should be less than 10^5 bacteria per gram of tissue; at levels above this, the wound is not likely to be ready for definitive coverage. Wound debridement directly reduces the bacterial load and disrupts the biofilm, allowing the body's cellular mechanisms to take over. Breidenbach determined that quantitative cultures provide an accurate assessment of bacterial loads, and can identify wounds at risk for infection

With the advent of vacuum dressings and microsurgery, more aggressive and extensive debridement can be undertaken with the knowledge that larger, more complex wounds can be closed with reliable methods. This has led to an evolution in the methods and endpoints of debridement and wound care in the last 10 years.

Aims of Debridement

Dissection techniques that result in further devascularization of bone and soft tissue should be avoided. An awareness of delicate soft tissue handling and atraumatic techniques helps to prevent adverse iatrogenic sequelae during debridement. Debridement should start in a logical fashion at the periphery of the wound and work systematically toward the center. Once finished, pulsatile lavage can be used to clean the wound.

The debridement of an acute wound such as an open Gustillo IIIB or C should occur once the patient is medically stable, within the first 48 hours of the injury (see Chapters 7 and 8). All non-viable tissue and bone should be removed back to bleeding tissue; all foreign material removed; and the wound stabilized for the next 24 to 48 hours. At the "second look" operation the wound can again be debrided and further prepared for coverage.

Mechanical Debridement

Mechanical debridement of wounds is the mainstay for preparing a wound bed for tissue coverage or expedited healing. Various instruments can be used including:

- Surgical scrub brush
- Curette, rongeur
- Scissors
- Scalpel
- Motorized burr (for bone)
- Hydrosurgery devices

The reconstructive surgeon must select the most appropriate tool for each type of wound in order to accomplish thorough debridement with minimal collateral damage.

Surgical Scrub Brush

This can be an excellent debridement tool but it will not be appropriate for the majority of acute wounds. It is useful for removing a thin layer of tissue that is not strongly adherent to the wound bed, such as granulation tissue prior to skin graft placement. The brush can remove this thin layer of tissue in a very efficient manner, particularly on larger wounds on the abdomen or thigh.

Rongeur, Curette

Orthopedic instruments such as the rongeur and curette have a specialized role in certain types of debridement. They are excellent for hypertrophic synovium, bone fragments, and small particles

such as embedded gravel or asphalt. The curette is also useful for preparing a wound for split thickness skin grafting. Granulation tissue of smaller wounds can be removed quite easily if the curette is used at a very low angle of attack in a scraping fashion.

Scissors

Scissors are of some use around a wound, however the size, shape, and variable sharpness of the instrument means it is not ideal for all types of debridement. Two types commonly used are the Mayo and Metzenbaum scissors. Mayo scissors should not be used for debridement due to their design and difficulty in use, while Metzenbaum scissors are useful around vessels, nerves, and fine structures.

Scalpel

The instrument of choice for the majority of debridement is the scalpel. A No. 15 blade scalpel is preferred for smaller wounds and those on extremities, while a larger No. 10 blade may be more appropriate on a trunk wound. The scalpel is of particular use around the periphery of the wound to create a fresh edge for grafting or suturing. One drawback is the requirement for frequent fresh blades, which is essential to minimize further trauma to the wound.

Another significant drawback to scalpel debridement is wound geometry. Typically, the scalpel blade must be placed at a very small angle to the surface in order to allow a shallow depth of tissue removal but it is difficult to use a small angle of attack with a deep wound while maintaining a shallow depth of tissue removal. Additionally, the surgeon may have to pick up the tissue to help guide the scalpel into the proper plane. The scalpel tends to stay in the appropriate plane of dissection for only small distances, creating a "skipped" and scalloped appearance to the wound bed. This may inadvertently leave small islands of non-viable tissue behind.

Hydrosurgery Devices

One of the newest and most promising tools for debridement is the Versajet™ (Smith & Nephew, Largo, FL). This is a high-powered hydrosurgery device which passes a high velocity stream of water across a specially designed head. The jet of water, based on the Bernoulli principle, cuts skin like a scalpel or dermatomeand removes debrided tissue with suction created by the jet of water. This device is useful for cutting small, thin, predictablelayers of tissue and removing the debrided tissue in one continuous process. Additionally the cutting head is able to lie flat on the wound, essentially lifting the tissue off and eliminating the need for forceps. The Versajet has a small head that can reach into smaller deeper wounds, and a water stream that provides a uniform depth, width, and length of tissue removal.

Hemostasis

When debriding extremities, it is important to use a tourniquet to avoid unnecessary blood loss. Hemostasis after tourniquet release is simple and provides the surgeon with a good picture of viable tissue. In the ischemic field it is easy to distinguish healthy from damaged tissue. Healthy tissue is bright and homogeneous in color whereas damaged tissues have color irregularities and often contain foreign material.

Hemostasis can be achieved with a combination of monopolar and bipolar electrocautery but this should be used judiciously and sparingly in light of the collateral damage which usually occurs. A dry wound bed can usually be achieved with a technique called "one-song hemostasis." This simply requires the user to hold pressure on the wound for the time it takes to sing or listen to one song on the radio. If working on an extremity the tourniquet should be loosened to remove the venous compression, which can worsen bleeding.

Wound Irrigation

In the acute setting, pulsatile lavage of a contaminated wound is an excellent way to reduce the load of foreign particulate matter. Modern methods of wound irrigation include gravity flow, bulb syringe, or pulsatile jet lavage. Of these, pulsatile lavage is superior in reducing bacterial contamination, removing necrotic tissue from crush wounds, and decontaminating wounds. (Pulsatile lavage was developed by military surgeons during the Vietnam War for decontaminating

blast injuries to the face. Since then, this system has evolved into a battery powered, closed system with built in suction and splash protection.) Dirt, asphalt, and other particulate matter are easily cleared from wounds with minimal risk of damaging viable structures. Three to nine liters of fluid (lactated Ringers' preferably) should be utilized for lavage following sharp debridement of the wound. The lavage system does not have the capacity to remove strongly adherent or attached necrotic tissue; significant debris that can be removed with sharp debridement, should be addressed prior to pulsatile lavage.

Maggots

Maggots were first observed to be effective wound debriders during the Napoleonic era by Baron Dominic Larrey, who reported that soldiers infected with maggots had very clean wounds and no systemic indications of sepsis. This was further confirmed by Confederate surgeon Zacharias during the Civil War who was the first to place them into necrotic wounds for the purpose of debridement. With the development of antibiotics and surgical techniques, maggot therapy was forgotten and largely abandoned by the 1940s.

In the past 15 years maggot debridement has been resurrected by wound care specialists. Maggots remove non-viable tissue by secreting enzymes that dissolve unwanted tissue and create a nutrient-rich medium as a food source. Thirty larvae, covered with a semi-permeable dressing, can consume one gram of tissue per day. Maggots must be replaced every 2 to 3 days but are an excellent alternative for the sensate, non-operable candidate who may experience tremendous pain with frequent dressing changes.

An additional advantage of maggot therapy is their ability to eliminate bacteria in chronic wounds where they are able to consume methicillin- and vancomycin-resistant bacteria. Finally, family, nurses, and other health care personnel can easily be trained to apply and care for these organisms to allow debridement for wounds to be carried out in an out-patient setting.

Tissue Viability (Clinical)

In many cases, it is difficult to assess the health of the skin-flap microcirculation despite the many subjective and objective techniques available. Adequate microcirculation is essential if the graft or flap is to survive, and a number of tests have been developed to assess viability before and after closure.

Intravenous fluorescein dye was proposed in the early 1980s as a test for the viability of surgically created skin flaps. In an early description of the technique, Zahr et al. noticed that flaps that took up the dye evenly went on to heal uneventfully, while flaps that were patchy consisted of non-viable skin. Where patchy fluorescence was observed, further debridement will prevent immediate post-operative skin necrosis.

Of all the techniques available at the time, this technique, using sodium fluorescein, provided the most reliable results. However, the pharmacokinetics of fluorescein prevented it from becoming fully established in clinical practice. The fluorescent dye indocyanine green (cardio green) has more favorable pharmacokinetics and has been proposed as an alternative method for predicting post-surgical skin necrosis.

In an experimental model, indocyanine green angiography (ICGA) was used to study post-operative changes in the microcirculation of a skin flap and to observe the hemodynamic imbalance within the flap. More recently, indocyanine green (ICG) fluoroscopy has been used to assess the perfusion index of flaps and to predict where necrosis may occur. In this animal study the authors conclude that differences between well-perfused and non-perfused areas of skin, as measured by ICG fluoroscopy, were highly statistically significant and could be used to accurately predict skin necrosis.

Timing to Wound Coverage with Vascularized Tissue

As has often been the case in medical history, war-time surgical experience provides a wealth of information based on a large number of cases with similar injuries treated in similar surroundings. Recent experience gained from treating personnel injured in the Iraq and Afghanistan conflicts has

provided some valuable insights into issues such as optimal wound closure time and flap creation and reconstruction processes.

Surgeons have always intuitively that early wound closure using healthy, well-vascularized autologous tissue is essential to a good outcome, and this is reflected in all the protocols for management of injured extremities. The rationale for early closure is based on an assumption that delayed closure leaves the way open for infection from the environment or other parts of the patient's body. The reality is that many patients requiring primary wound closure or grafting are physiologically too frail to withstand surgery, and they therefore require a period of intensive management before reconstruction or wound closure can be considered safe.

Another factor in theatres of war is that injured personnel can often not be treated immediately due to a lack of facilities or appropriately skilled clinicians at the time of injury. If delays in flap coverage are detrimental to outcome, then capable medical units should be placed closer to the area of military operations. In order to answer this question, Sherman et al. reviewed the available literature on timing of debridement of open fractures and found that results were not necessarily unanimous.

Byrd et al. found that flapcoverage patients treated within 6 days of injury experienced fewer infections, fewer re-hospitalizations and decreased time to union. Patients treated between 6 days and 6 weeks had the highest rate of infection and infection-related complications, higher even than those patients treated after 6 weeks. Similar results were reported by Cierny et al. and Godina. However, these and other studies do have limitations in that patients given flap coverage at a very late stage may have been treated with optimal medical care and could have been better stabilized by the time coverage was undertaken, thus confounding the effects of time as a single variable. Similarly, the results could have been biased due to patient selection for different types of care plans.

Sherman (2006) concluded that there is insufficient evidence to evaluate the specific influence of timing on soft-tissue coverage following high-energy lower extremity injury.

Kumar questioned whether early closure is the key to successful limb salvage, or is radical debridement and conversion to an acute wound the key step? He also speculated that widespread use of negative pressure wound suction has changed the nature of wound management such that delay in reconstruction is no longer detrimental. Vacuum-assisted closure (VAC) is a technique in which negative pressure is applied to the wound bed via a closed system. Edema fluid is removed from the extravascular space, improving blood supply during the inflammatory phase. The mechanical tension of the vacuum may also directly stimulate cellular proliferation of newly forming granulation tissue. VAC is often used as a dressing in order to anchor applied split-thickness skin grafts.

Kumar (2006) presented data for patients treated at the National Naval Medical Center (NMMC, Bethesda, Maryland) where, due to lengthy evacuation procedures, no patient received wound closure or reconstruction within 7 days of injury. Both pedicled and free flaps were used for reconstruction with an early total flap loss rate of 0% for free flaps and 1.4% for pedicled flaps. Partial flap loss was 0% for free flaps and 7% for pedicled flaps.

Maintaining vascularity to the tissue flap is a key factor in flap viability. The term "fillet flap" is commonly used to describe a spare piece of tissue (say from non-salvageable mutilated tissue) such as a pedicle flap or free flap. Classic fillet flaps are axial-pattern flaps harvested from amputated, discarded, or otherwise non-functioning parts. In some clinical circumstances, intact extremities must be converted into fillet flaps to facilitate complex reconstruction. In all cases, a major vascular axis must be identified that can contribute blood supply to the tissue that will be used. With pedicle tissue transfers, an axial vessel is selected (e.g., radial artery, ulnar artery) and the flap is dissected while preserving the arterial supply, dorsal veins, and lymphatic channels, until the flap is sufficiently mobile to be swung over to cover the defect.

Another new advance is the "perforator flap," in which the angiosomes that are distributed to skin territories can be followed to the feeder vessels, thus increasing the supply of cutaneous flaps and other composite flaps without sacrificing major vessels.

The advent of the sural flap has made it possible to avoid microsurgical reconstruction yet still provide adequate, well-vascularized cover, particularly in the distal third of the leg. Most authors describe good results with sural flaps, with low rates of flap necrosis (between 0% and

17%). However, a recent review of 70 sural flaps in two major institutions found that the rate of partial or complete flap necrosis was up to 36%. There were possible reasons for this including age, associated comorbidities, and social factors, which led Baumeister and colleagues to recommend a sural flap "delay procedure" to diminish failures.

The "delay phenomenon" is an observation that flap failure can often be reduced with delay between flap creation and re-attachment—a possible explanation for the observation that a long delay between flap creation and transfer often produced better outcomes than a moderate delay. The flap, including its neurovascular pedicle, is completely elevated, although the distal part of the skin island is only incised 50%, leaving the neurovascular bundle intact. The flap is left in this position for between 7 days and 1 month before flap transfer. This period of delay may allow reorientation of blood flow in an axial direction before flap transfer. The mechanism has been associated with increased vessel size, reorientation of vessels, increased number of vessels, and improved blood flow, all of which improve the chances of successful flap transfer. The effect was first reported in the 1920s and is still successfully applied today.

A disadvantage of this technique is that tissue quality at the time of transfer is different compared with newly constructed tissue: it is more edematous and has less elasticity. However, a decrease of flap failure rate is a justification for flap delay, particularly if there are other risk factors in the patient for flap necrosis.

CONCLUSION

Before flap transfer is initiated, adequate and thorough debridement of the wound bed must be carried out. Although a variety of methods are available for debridement, mechanical techniques—including a newly developed hydrosurgery system—are usually required. Techniques for constructing and developing tissue flaps have developed leading to improved vascularity, and vacuum-assisted closure techniques allow flap transfer to be delayed until the patient is in an optimal state of health.

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10 Surgical Debridement

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INTRODUCTION

Debridement should ideally be realized in an operative room where electrocoagulation, sterile drapes, and adequate anesthetic drugs are available. This surgical ambiance is needed especially when a surgical exploration has to be completed. In most of the cases, this step-by-step approach is the guarantee of a complete resection of necrosed areas, sloughy tissues, and undermined cavities hidden to a superficial evaluation. Debridement must be understood as a progressive evaluation of the lesions under the visual control of the professional, a person able to decide which tissue should be kept and which one should be removed. Debridement is adapted to the pathology, and the need for a complete removal of necrotic tissue is different in an extensive infection of the soft tissues like in Fournier gangrene than on a venous leg ulcer. Aggressive surgical debridement is considered as a means to accelerate closure of diabetic foot ulcers (DFU). In 1996, Smiths et al. (1) advised surgeons to debride the edges of the infected diabetic ulcers with large margins. Steed et al. (2) demonstrated in 1997 that surgical debridement of surrounding callus, necrotic tissues, and undermined ulcers' edges was associated with greater incidence of healing, even if this issue was considered as a secondary end point in this study designed primarily to analyze results of application of a skin substitute. Debridement is considered by most trauma surgeons as a standard of care. In chronic wounds, the presence of necrotic tissues is considered as one of the main reasons for wound-healing delay (Fig. 1). The appropriate extent of debridement is still a debate, especially as complementary techniques like powerful hydrojets and negative pressure therapy have been developed. The appropriate tool used to debride is a topic of debate, and the cutting, removing, destructing, washing, and aspirating properties of a technique/device have been compared and analyzed in order to choose the best indication in a defined condition (3).

What to do after surgical debridement is also a question the surgeon will have to answer. In and acute wound, as well as in some chronic wounds, the tendency has for a long period of time been to immediately close the wound. The development of recent technologies based on hydrojet debridement and negative pressure have slightly modified algorithms of care.

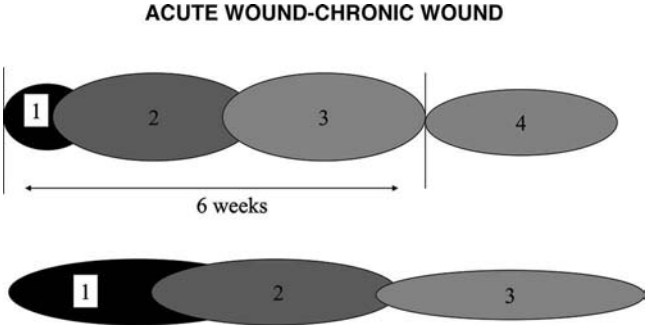

FIGURE 1
Schematic of the healing stages: (1) debridement; (2) granulation tissue formulation; (3) epidermisation; (4) maturation. The wound becomes chronic when debridement plus granulation tissue formation plus epidermization time exceeds 6 weeks. The lack of active debridement is one of the main causes.

EVALUATION OF THE EFFICACY: SCORING DEBRIDEMENT

Scoring systems to assess the quality of the debridement have already been published (4,5). Margolis et al. (5) designed a classification adapted to DFU ulcers. Three extents of debridement were defined: callus, ulcer's edge undermining, and wound bed necrotic tissue. A score of 0 to 2 was applied to each of these categories using the following criteria: 0 = debridement needed but not done, 1 = debridement needed and done, and 2 = debridement not needed. A total ranging from 0 to 6 could be defined, with the highest number being the optimal score. This instrument, the Debridement Performance Index, evaluates both the adequacy of debridement and whether the ulcer has been properly debrided. To initiate the validation of this scoring system and determine its predictive value for wound closure by week 12, the score was applied to 143 patients with DFUs who had been treated in a clinical trial involving either standard therapy or the application of a bioengineered skin construct. Each DFU was evaluated using sequential digital photographs and the Debridement Performance Index score was applied at day zero, before initiation of either treatment. Results showed that the lower the baseline Debridement Performance Index the lower the incidence of ultimate wound closure by week 12 and patients with a Debridement Performance Index between 3 and 6 were 2.4 times more likely to heal than those with a score of 0 to 2. After controlling for treatment, the Debridement Performance Index was found to be an independent predictor of wound closure.

DEBRIDEMENT TECHNIQUES

Debridement can be performed using different modes (Fig. 2). The surgeon will act in continuity with other professionals and should limit his action to what cannot be done by others (Fig. 3).

Surgical Excision

Surgical excision can be realized with or without anesthesia, depending on the pain level and the equipment of the operating room. An instrument set including scalpel, scissors, a gouge, and a coagulation system can be enough to realize a fruitful debridement. More specific tools have recently been developed in order to detach devitalized tissues strongly adherent to the adjacent structures. In the performance of the debridement procedure, sound surgical judgment is critical to balancing the need for adequate tissue resection and limb preservation versus the consideration of an amputation. A careful sharp surgical and complete parage can be performed in the operating room, in order to optimize tissue salvage. Options will vary depending on the availability of new technologies such as negative pressure therapy, or the capacity to cover the would using well-vascularized structures or other technologies. Cutting back along the would edges in order to refresh the local vascularization is usually performed. A precise evaluation of the vascular status of the lower limb is then critical. Preoperative

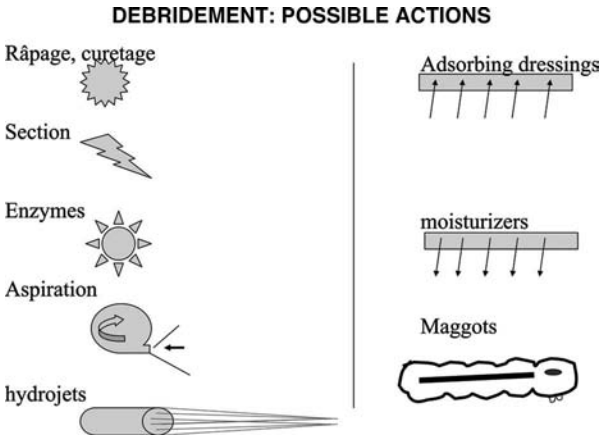

FIGURE 2
Different modes of debridement.

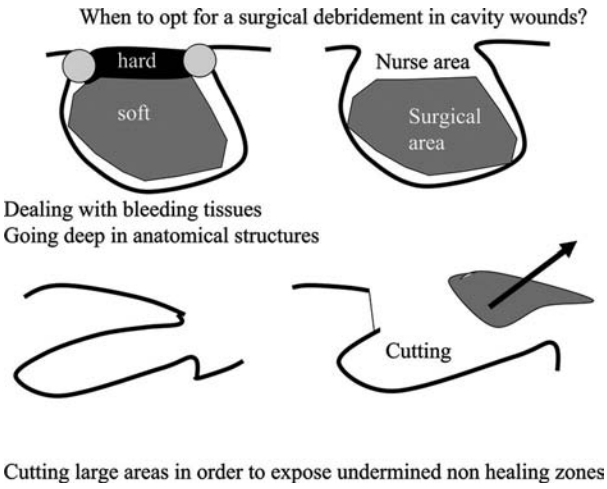

FIGURE 3
When to opt for a surgical debridement in cavity wounds?

prognosis of the vascular future of a tissue is one of the most difficult points: the operator has to keep in mind the general condition of the patient, the local tissue perfusion, and the degree of ischemia before giving a chance to an uncertain structure, the risk being reappearance of necrosis on the edges.

Necrotic tissues should be completely excised. Sloughy tissues will be removed, depending on their proximity to vital structures. Aponeuroses are protective structures for the underlying longitudinal muscles, neurovascular bundles, or tendons. When opened, infection can easily extend along the tendons, nerves, and vessels. Debridement outcomes have to be anticipated using decision analytic methods (7).

Washing

Washing is the standard technique for evacuation of germs and stimulation of angiogenesis. Undermined cavities, pouches, and sinuses have to be washed carefully. Washing using sterile water is the most common procedure. Cleansing can be performed using water projected from a simple syringe in order to remove devitalized tissues located in undermined areas. The pressure is mild and the risk of germ projections is limited. Other devices, like Jetox, combine the mechanical forces of the waterjet and oxygen from the wall unit.

More specific tools have recently been developed in order to detach devitalized tissues strongly adherent to the adjacent structures.

Hydrojets

More powerful hydrojet devices have recently been proposed. Debritom® (Medaxis, Santa Monica, CA) is a high-pressure device, developed in order to remove all devitalized areas using a very powerful jet, coming from a compressor, using a handpiece to concentrate pressure. This device needs a set of handpieces that can be sterilized and reused, limiting the cost. Tents have been developed in order to protect the environment from projections of germs. This technique is still under evaluation, but interesting preliminary series have evaluated the level of pain, which remains moderate, the numbers of stages needed to completely debride the wound, and the adaptability to an ambulatory use in the wards (Fig. 4) (8).

Versajet™ (Smith & Nephew, Largo, FL) was initially developed in Germany and more commonly used in the United States and Europe since 2004. Versajet is a unique device proposing a three-in-one combination of effects, debridement, aspiration, and removal of the sloughy and devascularized tissues (9). The handpiece is connected under a sterile manner to the aspiration machine. Based on the Venturi effect, removal of tissues is realized without any contamination of the surrounding tissues (Fig. 5). Cost of the machine and of the disposable device is

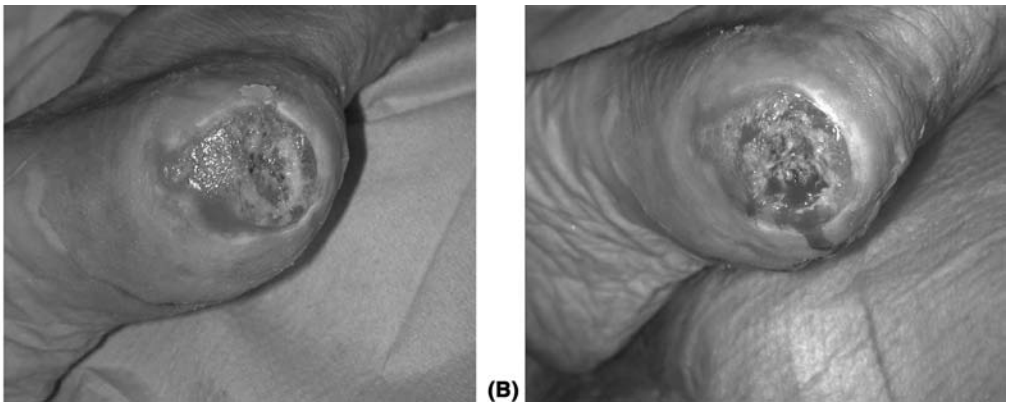

FIGURE 4
Heel pressure sore: (**A**) Before and (**B**) after a single application of Debritom™ high pressure waterjet.

high compared with the other systems. However, Granick et al. (11) have recently evaluated the cost efficacy of Versajet with a positive input on the global wound management costs. Selectivity of the debridement and reduced blood loss makes the technique attractive.

Pain provoked by hydrojet debridement varies following different situations: in DFUs, with reduced or absent sensation, Versajet is well tolerated. In trauma wounds or burns, pain can be severe and often require a general anesthesia. Versajet plus, a more powerful version with more capacity to remove hard tissues, looks promising.

Hydrojets should be used properly. Surgeons should keep in mind that fragile soft tissues cannot sustain excessive mechanical forces.

INDICATIONS FOR DEBRIDEMENT

Acute Wounds

Acute wounds should be washed, irrigated, and cleaned. Except in situations when an adherent coating is glued to the edges of the wound, tissues are soft and can be easily debrided earlier than if a wound is chronically exposed to devascularization of the edges, local inflammation, and fibrosis. For a long period of time, the need for an immediate closure after debridement was the rule. Since the appearance of negative pressure therapy (NPT), it is now possible to anticipate a secondary closure. Algorithms of trauma wound management should be expanded to include these newer modalities in the armamentarium of care options.

Trauma Wounds

Trauma wounds can expose hard tissues (bones) or soft tissues (tendons, vessels, nerves). The mode of action of the traumatic agent should be determined first. Penetration, desquamation injury, shearing or friction, compression, or explosion are different agents which must be precisely analyzed. A crush syndrome needs a different management than a high-velocity penetrating injury. An experienced opinion is often needed during surgical debridement in order to obtain a complete resection of all doubtful tissues.

Penetrating trajectories should quickly be explored and treated. Soft tissue color, adhesion to living structures, and resistance to traction have to be evaluated. Selectivity of debridement is a key factor, into which Versajet is a promising tool. Undermined cavities will be explored, washed, and drained. Long and sinuous subcutaneous cavities will be more difficult to manage. Fixation of large skin areas gliding over the underlying structures needs an adapted mechanical application combining sutures, stitches, and aspiration of cavities. The use of antiseptics is recommended to add an anti-infectious effect to water fluxes.

Negative pressure therapy (NPT) presents the advantage of exerting a permanent mechanical suction over the exposed surfaces, adding an effect of prevention of reinfection in this aspirated

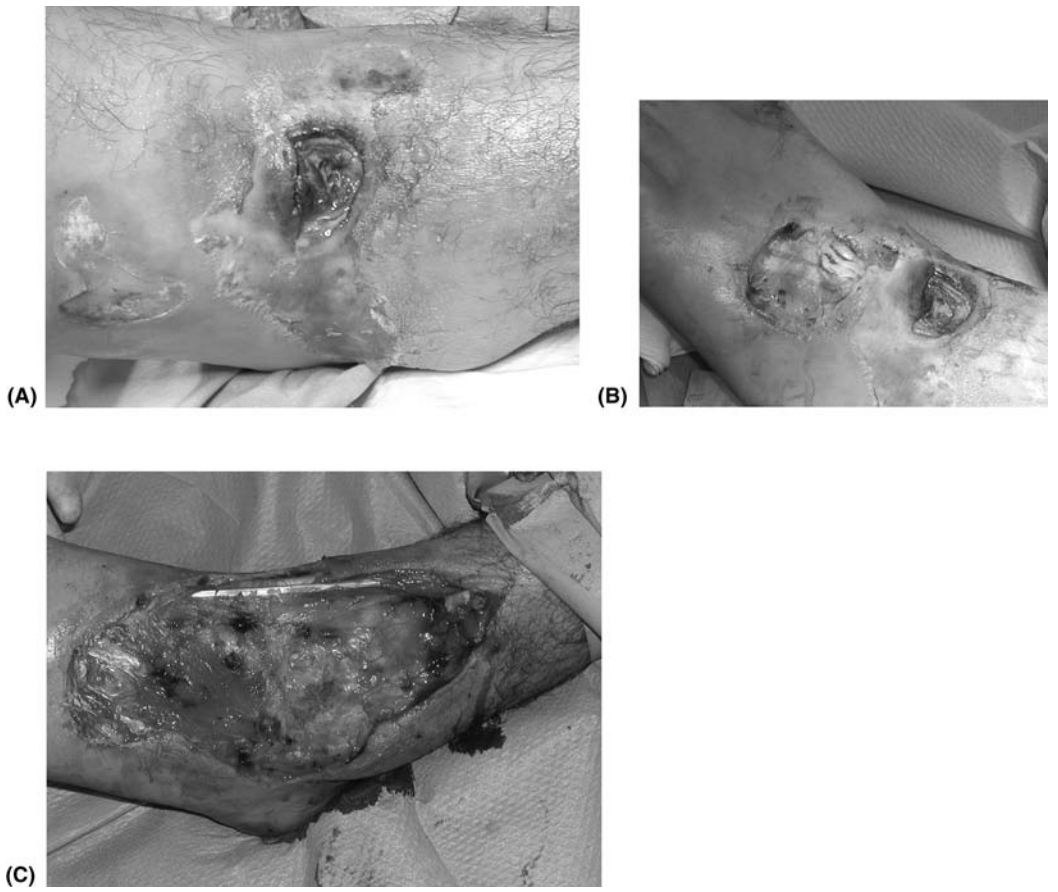

FIGURE 5
Electrical burns of the foot: (**A**) Before, (**B**) during, and (**C**) after one application of Versajet™ hydrosection.

area. NPT cannot be considered per se as a debriding agent, especially when tissues are locally infected. However, Armstrong and Lavery (12) demonstrated the effect of NPT when applied over previously debrided areas. In DFU, applying NPT over surgically debrided cavities will prevent reappearance of necrotic or infected tissues.

The same situation occurs in trauma surgery where NPT is applied just after the initial parage. In this case, the wound remains clean enough to prevent infection to occur. Some authors report large series of trauma cases exclusively treated by NPT, without the need for any flap to sever secondarily the wound. NPT seems to be a good healing complement when used immediately after surgical debridement, enhancing the stimulation of granulation tissue.

Burns

Burns are better managed by specialists. Burns are usually classified into three degrees: the first degree is a sun burn and needs adequate antalgics, the deep second- and third-degree can be source of partial or complete skin necrosis. Evaluation of the burn's depth can be difficult during the first days after injury, especially in second-degree burns. Deep second-degree burns will be excised and grafted (preferably) when the hands and the face are involved. (13). Other areas can be excised on demand. Third-degree burns will be rapidly excised and covered with skin grafts. The best treatment for burns and scalds depends on the depth of the skin necrosis. Epidermal and superficial dermal burn injuries (IIa) can heal spontaneously with conservative treatment without scar development, but deep dermal or full-thickness burns constitute an absolute indication for surgery.

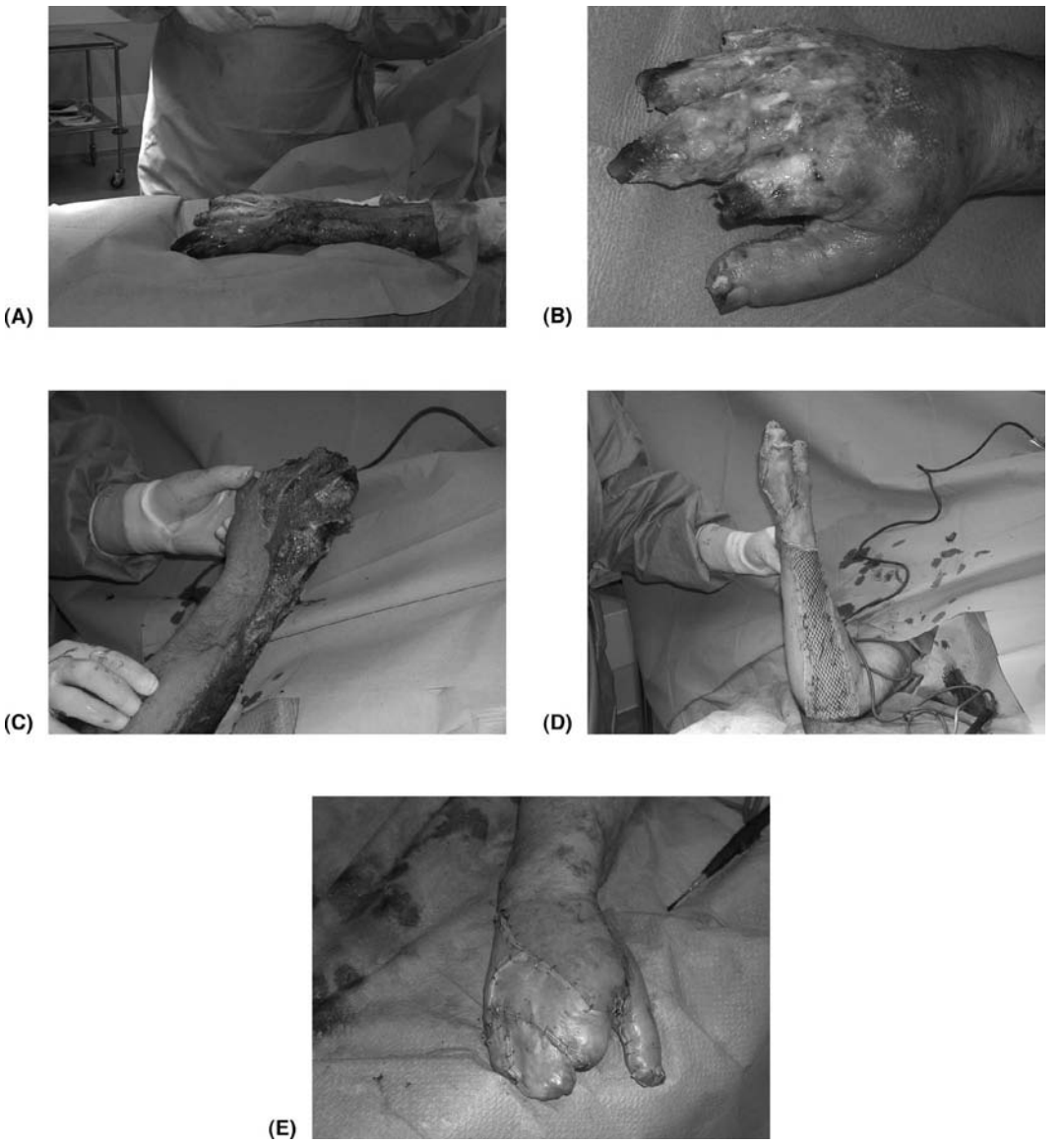

FIGURE 6

Excision is the debriding technique, and should be extended to the living well-vascularized structure, dermis, fat, or aponeurosis. Experience is needed when determining the extent of debridement, especially in chemical or electrical burns, the necrotic process being long and progressive. Versajet is a very selective technique in finding the precise plan of excision, owing to its capacity to exclusively debride dead tissues. Other techniques combine excision using scalpels, depth-regulated knives, and/or dermatomes (Fig. 6). Following a debridement, conservative treatment of superficial dermal burns involving wound coverage with biosynthetic dressings or nanocrystalline silver gauze dressings or use of special disinfecting ointments can be implemented.

Full-thickness or split-thickness skin grafts are used for wound closure. Allografts can be used for temporary wound closure. Autologous keratinocytes obtained from culture in specialized laboratories can be used for transplantation.

Some techniques are specific to immediate burns management: discharge incisions should be taught to all relevant emergency transportation teams. This technique consists in an

extensive linear cutting of the skin and subcutaneous tissues, leaving aponeurosis intact, in order to remove the excessive pressure caused by a circumferential third-degree burn. In circumferential burn injuries affecting the extremities or the trunk this rigid eschar has to be incised as it creates an ischemic situation. Incisions will be performed longitudinally at a distance from the lateroventral neurovascular bundles. On the hand, the dorsal aspect will be incised from the wrist to the digits, reaching the dorsolateral aspects of each side of the digit to the nails.

Bites

Bites usually mix different types of injuries, that is, degloving and penetrating lesions. One of the main difficulties is in evaluating the potential infectious risks of the biting animal salivar secretions (dog, cat, and so on, including humans). Prevention of infection (occurring in 10% to 50% of the cases) is the main objective of debridement. Removal of foreign bodies, extensive pulsed lavage, and drainage of collection of microbacteriological samples, are crucial for good management. The wound will be closed when seen early or left opened when seen later or in presence of infection (14). The use of adapted antibiotherapy remains the rule in case of bite, as proposed by Cummings from a meta-analysis of the literature (15).

Dermabrasions

Dermabrasions can be defined as mechanical lesions mimicking burns, and possibly leading to skin necrosis. Tattooing of foreign bodies should be carefully removed (16). Despite attempts at tissue conservation with dermabrasion, some of them result in third-degree lesions with destruction of the whole depth of the skin. Excision of necrosed skin will be required by skin grafting during the same procedure.

Chronic Wounds

Debridement in chronic wounds can be realized in different situations; when an extensive necrosis is present, debridement is a useful technique to prevent infection. When the wound has already been infected, debridement is the quickest way to reduce the bacterial number. However, chronic wounds are different in their origin and determining factor. A precise investigation of the tissue perfusion before debridement is strategically determinant; however, a poorly vascularized foot is a contraindication to wound debridement.

Diabetic Foot Ulcer

Tissues in DFUs can be either denerved in neuropathic lesions and/or devascularized when arteriopathy leads to chronic ischemia. Absence of sensation facilitates ambulatory surgery and appropriate debridement without anesthesia. When treating nonarteriopathic diabetic patients, debridement is more efficient and should be more selective. When osteoarthritis is present, bone resection should reach bleeding tissues on both sides. In arteriopathic patients, limits of the surgical resection and determination by visual evaluation may be difficult, and amputation can be the preferred technique. Negative pressure therapy has demonstrated its efficacy in preventing reinfection after an appropriate debridement in the neuropathic foot (17–20).

Pressure Ulcer

Pressure sores can be staged into four situations. In stage III necrosis of skin, subcutaneous tissue is present. In stage IV, bone is involved. Debridement should be adapted to the extent of necrosis and sloughy tissues, keeping in mind the shape of the wound, that it may be larger in depth, and the possible presence of undermined areas (21). The typology of the wound should be perfectly known by surgeons confronted with pressure sore debridement. Sacral pressure sore is usually flat and laterally undermined; trochanteric pressure sores are on the contrary deep, hidden from visual evaluation, and extending around the neck of the proximal femoral extremity toward the hip capsule. Heel pressure ulcers expose the calcaneal area, and may extend upwards to the Achilles tendon, downwards toward the plantar aponeurosis. Ischial pressure sores are deep lesions exposing the ischial bone, and are usually infected. These lesions are observed in seated paraplegic patients.

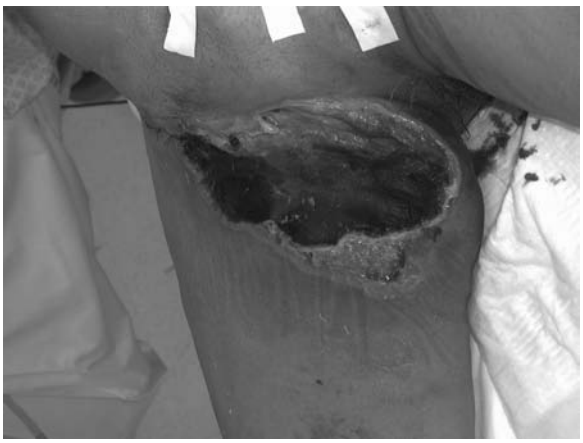

FIGURE 7
Extensive skin necrosis after a surgical groin area vascular approach. Necrosis will be removed surgically, exposing the femoral vessels. The procedure should be done with a vascular surgeon present.

Leg Ulcer

Venous leg ulcers are not usually candidates for surgical debridement. The chronicity of the lesions and their pathogenicity based on the recurrent presence of fibrin render these lesions more prone to be mechanically debrided by nurses. In some situations, a radical excision can be proposed. Bitsch et al. (22) recently reported a large series of extensive debridements followed by an immediate skin graft in large venous ulcers.

Arterial ulcers should not be debrided, owing to the high risk of creating new lesions at the margin of the resection, leading to amputation. These patients are candidates for revascularization techniques, evaluated on a vascular check-up including a Doppler and an arteriogram. Patients presenting with angiodermatitis can be candidates for a radical excision of the ulcer, mainly for pain reduction, followed by a skin graft.

Infected Wounds

Surgical debridement has demonstrated its usefulness in infected wounds, in acute situations as well as in chronic ulcers. Removal of sloughy, adherent, and infected areas, opening closed abscesses, and exposing large undermined areas by removing the skin cover remains the most rapid and efficient way to reduce locally the number of germs (Fig. 7). Blood passage of germs can be encountered in some cases, with the risk of bacteremia followed by septicemia, which is fatal in more than 50% of the cases. Diabetic foot ulcers remain problematic as most of the time the classical infection triad of rubor-calor-dolor are absent from the screen. In DFU, infection can be assessed only by the bone probe (23,24). This is why broad spectrum antibiotic therapy should be given before the surgical debriding procedure. Encountered germs are usually *Staphylococcus aureus* and *Pseudomonas aeruginosa.* Versajet is adapted to infected situations, as the risk of contamination is limited by the aspirating effect of the device.

Maggots can be proposed as a complementary technique to surgical debridement or as an alternative when tissue is poorly vascularized. Maggots have been extensively used in DFU with success, especially in arteriopathic ulcers (25,26).

Some infections are specific and the surgical procedure should be adapted to the pathology:

—Fournier's gangrene, observed after urinary surgical procedure is because of polymicrobial infection. Extension of infection along the subcutaneous tissue is marked by typical skin crepitations in the genital area. Survival is associated significantly with anorectal infection, chronic renal failure, the duration of symptoms before hospitalization, the extent of gangrene, and serum blood-urea-nitrogen and creatinine values on admission (27). The surgical procedure should be rapid in order to stop the progression of infection, by using an adapted skin resection, and by using large skin incisions to expose areas involved with the infectious process. The

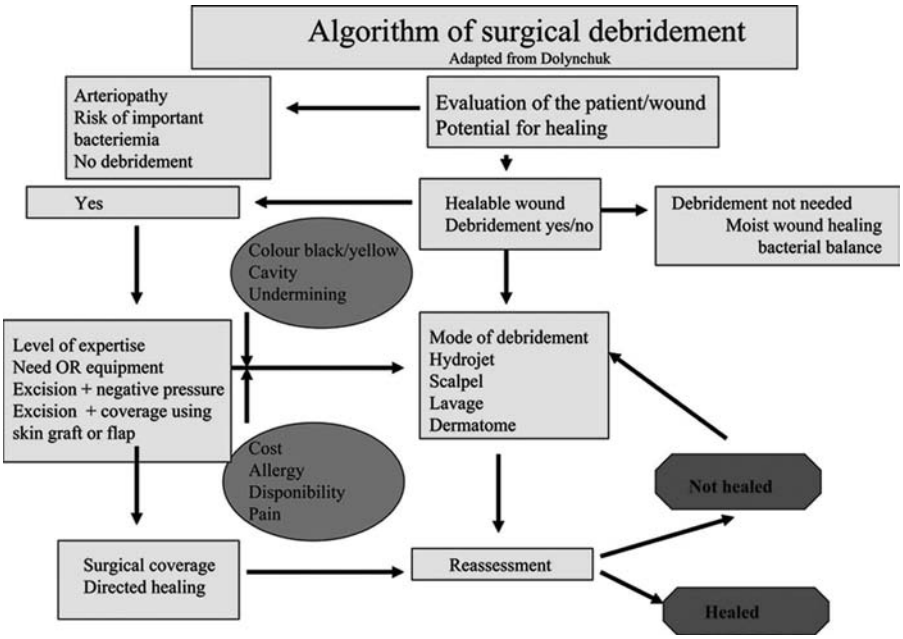

FIGURE 8
Dolynchuk algorithm of surgical debridement.

combination of effective systemic antimicrobial therapy and a series of daily surgical procedures can minimize the loss of critical anatomic structures in the genital area.

—Hidradenitis (acne inversa) is linked to an annexial chronic infection (apocrine sweat glands) observed in well-defined anatomic locations resulting in chronic wounds with abscesses, sinuses, and fibrosis. This pathology, whose cause remains unknown, is more common in females than in males. Genital, axillary, and fold areas are involved (Figs. 2–5). Debridement should remove multiple subcutaneous abscesses and expose large infected areas sometimes difficult to cover. Axillary involvement is present in 88% of the women and bilateral in 50% of cases. Inguinoperineal involvement was present in 87% of the men and bilateral in 92% of all patients. An algorithm for operative treatment was developed by Kagan et al. (28). Excision and primary closure was used for localized disease; wide excision with or without skin grafting was used for diffuse disease.

—Necrotizing fasciitis is linked to group A streptococcus which secrete a necrotizing substance, but a series of germs like *P. aeruginosa*, *Escherichia coli*, *Aeromonas hydrophila* leading to vascular thrombosis of skin and underlying tissues can cause a similar type of infection. Very extensive areas can be involved, leading to amputations and skin resection on different areas over the body surface. While septicemia is the cause of death in many of these cases, an effective resuscitation can result in a patient with a massive soft tissue defect that has limited surgical options for achieving a satisfacory outcome.

Social Debridement

In exceptional clinical situations, like lymph node necrosis in HIV patients in palliative care, some debridement techniques may be needed. The aim is not to heal the debrided area but to allow end of life to occur in socially acceptable conditions (odor) for the family and the patient.

CONCLUSION

Surgical debridement is a challenge with many traps. The procedures are multiple and should be known by all surgeons, whatever be their specialty. An algorithm derived from Dolynchuk's proposal in chronic wounds (Fig. 8) has to be used as a reminder. Effective surgical debridement requires judgment in analyzing the situation and determining the strategy. New technologies

such as hydrojets may bring simpler and more reliable progress in preventing the development of infections in the wound bed.

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11 Debridement of Decubitus Ulcers

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Decubitus ulcerations have been documented as a significant health care problem that affects all age groups and is observed across all health care settings. While this clinical problem is a common complication for the medical practitioner to address, the successful treatment of this condition requires an organized approach to the overall assessment of the patient and factors responsible for the ulcer being treated. The primary goal of decubitus ulcer debridement should be the conversion of this complex, necrotic, infected soft-tissue ulceration into a clean, healthy, acute (or recovering) wound that will respond predictably to clinical measures designed to promote wound bed preparation and possible wound closure. In this chapter, we will briefly discuss the etiology of this condition, its staging, the goals of decubitus ulcer patient treatment, wound bed preparation, and techniques of surgical and nonsurgical ulcer debridement.

BACKGROUND

Decubitus ulcers, also termed pressure ulcers or sores, are estimated to affect 1.3 to 3 million individuals in the United States (1). Decubitus ulcers pose a significant clinical problem and can be a source of significant patient morbidity and human suffering. The first clinical practice guidelines for the treatment of decubitus ulcer patients were published in 1994 by the Agency for Health Care Policy and Research (AHCPR), now known as the Agency for Healthcare Research and Quality (AHRQ) (1). Additional guideline formats have been published since that time. Discrepancies exist in the development of treatment protocols because of varying emphasis on the analytical methods used to assess these chronic wounds or the priorities of treatment that should be applied (2,3). The National Pressure Ulcer Advisory Panel (NPUAP) has reported incidence and prevalence findings for decubitus ulcer patients from various age groups and in various acute and chronic care settings (4). The higher prevalence of decubitus ulcers is seen in the elderly, acutely ill patients, and individuals with spinal cord injuries and paralysis. In the general acute care setting, incidence rates of 7–38% are reported with prevalence in this population between 10% and 17%. The most common anatomic sites for pressure ulcer development are the sacrum and heels. Critical care patients were observed with an incidence rate of 8% to 40%. Patients with hip fractures requiring immobilization and traction were reported with an incidence rate of about 20%. Critically ill infants and children were reported to have incidence rates of up to 15% in a pediatric intensive care unit and up to 20% in a neonatal intensive care unit. The highest incidence rates for decubitus ulcer formation were seen in terminally ill patients receiving palliative care or institutionalized cancer patients receiving palliative care that ranged from 13% to 85% (4).

Systemic risk assessment may be used to help identify individuals at risk of developing decubitus ulcers. The Braden Scale is the most frequently used risk assessment tool for identifying patients at risk for decubitus ulcer formation and is often used to develop patient care plans and preventive measures for this problem. Clinical programs with prevention strategies based on risk assessment tools like the Braden Scale, in the acute setting, have reported declines in hospital-acquired decubitus ulcers between 34% and 50% (4).

ETIOLOGY

Excessive pressure and shear forces on the skin surface overlying boney prominences are the key factors responsible for the formation of decubitus ulcers. Prolonged pressure occludes the skin microcirculation producing irreversible ischemia. Ischemic damage initially occurs in the fatty subcutaneous and muscular compartments while skin is relatively resistant to these same ischemic forces. This causes the development of a progressively enlarging cone of soft tissue, fascial and muscular necrosis between the skin (apex of the cone) and the boney prominence (base of the cone). Skin maceration may adversely contribute to the progression of decubitus ulcer formation by compromising the integrity of the epidermal layer. This excessive moisture, which may be secondary to urinary or fecal incontinence, diarrhea, or excessive body moisture, promotes the breakdown of the barrier features of the skin, weakening dermal integrity and ultimately causing localized ischemic necrosis that is enhanced by the effects of pressure. In these cases, the clinician is often confronted by localized cellulitis that is associated with a developing ulceration. In more advanced cases, deeper ulcerations that have been colonized with bacteria may convert to form abscesses within the muscular and fascial planes of the wound. These deeper infected areas of the decubitus ulcer may lead to the development of osteomyelitis at the boney base of the ulceration. In many cases, new onset osteomyelitis of the boney prominence will require surgical debridement by a trained specialist. Early assessment of the boney integrity of the base of the decubitus is necessary in chronic conditions associated with full-thickness soft-tissue necrosis.

Unfortunately, despite our reasonably good clinical understanding of the etiology of decubitus ulcer formation, this problem is not always preventable. In many cases, the patient who is observed with a decubitus ulcer initially presents with significant declining metabolic factors, advanced age, organ failure, poor nutrition with involuntary weight loss, and systemic illness that ultimately leads to decubitus ulcer formation. What is important in these settings is that the patient risk factors and the associated conditions responsible for skin breakdown are well documented so that effective measures to minimize and reverse the acuity of this pathology may be implemented promptly.

STAGING THE DECUBITUS ULCER

The decubitus ulcer is assessed by the location, size, depth, and degree of the tissue (and boney) necrosis associated with this wound. The current staging recommendations for decubitus ulcers have been developed by the National Pressure Ulcer Advisory Committee (NPUAC). Decubitus or pressure ulcers are normally described within one of four stages of progressive pathology:

Stage I: nonblanchable erythema of intact skin, considered the heralding lesion of impending skin ulceration. Discoloration of the skin, warmth, edema, induration, or hardness also may be indicators in individuals with darker skin.

Stage II: partial thickness skin loss involving epidermis, dermis, or both. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

Stage III: full-thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.

Stage IV: full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (i.e., tendon, joint capsule). Undermining and sinus tracts are also associated with Stage IV pressure ulcers.

Stages I and II ulcers are technically considered as partial thickness wounds while Stages III and IV are considered full-thickness wounds. These designations become useful when we begin to consider treatment options for debridement and wound bed preparation. Although not described as a "stage" of ulceration, the last category of decubitus ulcer formation is the eschar. An eschar is any necrotic covering of an ulcer. For the decubitus ulcer the eschar is the nonviable, intact skin or dermis that has been transformed into a necrotic, leathery covering that prevents the accurate staging of the ulcer that lay beneath. When eschar is found considerations for debridement should be made by the surgical specialist attending the patient. Usually, if the

eschar is dry (or mummified), well adherent, and without associated drainage, erythema, cellulitis, or crepitance it may often be simply kept dry to perform as a biologic dressing. If there are signs of infection (pain, redness, drainage) or expanding areas of necrosis a surgical consultation for debridement should be sought.

The boney prominences most affected by decubitus ulcers have remained consistent over many years of clinical practice with the sacrum (37%) and heel (30%) being the most frequently reported sites in 1999 (5). Following these locations, ulcerations were observed with the following frequency from this same survey: ischium (8%), elbow (6.9%), malleolus (6.1%), trochanter (5.1%), knee (3%), scapula (2.4%), and occiput (1.3%). The distribution of decubitus ulcer stages notes that the largest percentage (76%) of patients have partial thickness ulcers (i.e., Stages I and II). The remaining 24% of the decubitus ulcer patients are observed with necrotic full-thickness ulcers at Stage III (8%), Stage IV (6%), and necrotic eschar covering a decubitus ulceration (10%).

The presentation of the decubitus ulcer, in most cases, is a clearly defined wound associated with limited tissue necrosis in proximity to a boney prominence. Occasionally, the decubitus ulcer development may become complicated by the formation of extensive areas of infection and necrosis (often limb- or life-threatening) that lay outside of the original zones of soft-tissue pressure injury. Two particular examples of this situation are observed when an infected decubitus ulcer leads to the development of necrotizing faciitis and necrotizing cellulitis. In most of these cases, we observe these complex infectious wounds developing in patients with compromised immune response (immunosuppressive therapy, acquired immunodeficiency syndrome, renal failure, diabetes) or as complications of absent protective sensation (e.g., spinal cord injury, diabetic neuropathy, paralysis) where the decubitus development and deterioration have gone unchecked. While these situations do not appreciably alter our basic clinical approach to the need for thorough wound debridement, it underscores the importance of regular patient surveillance for decubitus ulcer formation in any setting where patients may be at risk for this problem.

TREATMENT OF THE DECUBITUS ULCER PATIENT

Several important aspects of clinical treatment are involved in the treatment of the decubitus ulcer patient. Evaluations include medical factors and social factors, as well as the availability of local resources for the patient within their community. However, in the development of a clinical plan of treatment within the medical center environment, aimed at reversing or preventing the progression of decubitus ulcer complications, two key areas for assessment and treatment are: (*i*) the evaluation of patient positioning and the use of support surface technology to reduce ulcer incidence or recurrence, and (*ii*) the implementation of adequate patient nutrition to satisfy metabolic requirements needed for early wound healing. Once these factors have been successfully implemented along with general wound bed preparation, more focused plans for decubitus ulcer debridement and ulcer closure may be reasonably undertaken.

The AHCPR guidelines provide evidence-based pressure ulcer strategies that are often found in care plans for ulcer prevention. In protecting skin against the effects of pressure, friction, and shear the following are recommended: (*i*) reduction of pressure over boney prominences, every two-hour turning schedules for patients in bed and every one hour repositioning for patients sitting in a chair (individuals should be encouraged to shift their weight every 15 minutes); (*ii*) avoid positioning directly on the greater trochanter; (*iii*) allow heels to float off the surface of the bed; (*iv*) increase mobility; (*v*) use pressure-reducing mattresses and overlays and ensure that "bottoming out" is prevented with appropriate mattress selection, and (*vi*) the avoidance of excessive moisture on the skin surface next to the mattress or cushion supporting the patient's weight. The effects of moisture (e.g., urine or fecal incontinence) have been demonstrated to be especially provocative in leading to skin breakdown and ulcer formation. Sustained skin wetness increases vulnerability to pressure-induced blood flow reduction. The effect appears to be mainly dependent on wetness, but urine constituents may exacerbate the effect. In addition, wetness-related skin cooling may play a role (6). Skin protectants and barriers should be used, bowel and bladder programs implemented when possible, and frequent, thorough skin cleansing with alcohol-free moisturizers should be encouraged.

Despite the broad acceptance of these guidelines for skin protection and patient "off-loading" to prevent ulcer formation, clinical research has demonstrated that specialized mattress technology must also be considered in insuring the prevention of ulcer recurrence or worsening of ulcers receiving treatment. Research studies of mattress surface technology have demonstrated that static pressure-reducing devices are superior to standard hospital mattresses in the prevention of recurrent ulcers and for use with patients observed at high risk for new ulcer formation (7). For patients with a large Stage III or Stage IV decubitus ulcer or multiple decubitus ulcers involving several turning surfaces low air-loss or air-fluidized mattresses are more beneficial than static pressure reduction devices (8). However, if the patient "bottoms out" (if there is less than one inch of mattress material between the bed and the pressure ulcer) the pressure-reducing mattress or device may be ineffective.

Inadequate nutritional supplementation is often a vexing problem with the individual with a decubitus ulcer. This is especially the case with elderly patients, patients with chronic, systemic illness or significant infection, multisystem trauma, malignancy, involuntary weight loss, obesity, or depression. Additional factors such as cigarette smoking, substance abuse, and absence of social support have also been demonstrated to promote impaired nutrition for healing in clinical settings (9). Similar to burns, a hypermetabolic, potentially catabolic state occurs in association with decubitus ulcers (10). Adequate nutritional intake is imperative to provide the necessary calories for the increased metabolic demand of this condition. For these reasons, the maintenance of an optimal nutritional status is an important clinical goal in treating the patient with a decubitus ulcer.

Clinical guidelines for nutrition will require early nutritional consultation, a review of protein supplements and vitamins available for use with the patient, the use of tube feeding or parenteral feeding and fluids, and the possible use of anabolic steroid supplementation in selected cases. In these cases, the goal of nutritional therapy should be the attainment of positive nitrogen balance that may be achieved with 30 to 35 calories/kg/day and 1.25 to 1.50 g of protein/kg/day (11).

DEBRIDEMENT AND WOUND BED PREPARATION

Following the assessment of the decubitus ulcer patient and staging of the ulcer, considerations for the possible role of debridement for wound bed preparation should be established. Wound bed preparation has been described as the management of a wound in order to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures (12). For the decubitus ulcer patient, surgical debridement has been considered a fundamental process for wound preparation as it promotes the conversion of the nonhealing chronic wound to an acute wound that may proceed with the normal sequential phases of wound healing.

However, the complex and unique environment of the decubitus ulcer dictates that surgical debridement alone will not adequately address all of the elements of this ulcer that establish its capacity to promote significant clinical morbidity. To effectively restore normal healing in the decubitus ulcer environment we must address several important pathological processes simultaneously. To accomplish this, we have used a clinical algorithm called the TIME principle which identifies four key elements of the chronic nonhealing wound and provides a systematic approach to the restoration of the "normal" biological wound environment that is associated with unimpaired healing (12). These elements are represented as: T (tissue nonviable or deficient)—the presence of nonviable or biologically deficient tissue prevents the normal wound matrix formation and requires the application of continuous or episodic debridement (by selected methods) to restore the normal biological balance of the wound base and the extracellular matrix proteins; I (infection or inflammation)—addresses the impairment and deterioration of the wound that is brought about by the presence of high bacterial counts or prolonged inflammation. This requires that the focus of infection be physically removed and that appropriate antimicrobial therapies be used to return the normal bacterial balance; M (moisture imbalance)—identified as a serious negative factor affecting wound closure. Excessive wound exudate, often associated with wound edema, desiccates epithelial cells, slowing their migration. Excessive exudates may also indicate an imbalance in the presence of wound proteases that

may degrade growth factors or suggest a deficiency of important cellular signals for wound healing. Mechanical fluid removal (e.g., vacuum appliance) or the application of specialized dressings may play an important role here in re-establishing moisture balance for optimal wound healing; E (edge of wound nonadvancing or undermined)—poor epidermal advancement or wound undermining is a physical aspect of the wound that directly affects matrix development and maturation. In these cases wound debridement, reconstructive surgery, or adjunctive wound therapies may be needed to correct these problems.

The process of wound bed preparation begins with the examination of the patient as a whole. Systemic illness, disease, and various drug therapies may significantly alter patient metabolism, nutritional requirements, immune response, and cutaneous microcirculation and may interfere with wound healing. This is well documented with steroid therapy, organ failure (renal, cardiac, gastrointestinal, pulmonary), autoimmune diseases, diabetes mellitus, collagen vascular diseases, and immunosuppressive drugs. These conditions must be considered risk factors for impaired wound healing and careful scrutiny of current therapies may often lead to adjustments in treatment that may facilitate improved wound response.

Wound oxygenation and tissue perfusion are also critical to normal wound healing and should be assessed whenever possible. When skin oxygenation is measured below 30–40 mm HgO_2 poor wound healing may be expected. To document this, the heated skin electrode of a transcutaneous oximeter may be applied to the intact skin surrounding a wound for baseline readings and readings with the patient breathing 100% oxygen by mask. Transcutaneous oxygen measurement (TCOM) has proven useful in assessing vascular perfusion and may be more accurate than Doppler studies in predicting the capacity for normal wound repair based on oxygen perfusion. Factors that adversely affect sympathetic vascular tone in cutaneous beds such as cold, pain, or stress should be eliminated. For these same reasons, decubitus ulcer patients with a history of cigarette smoking should be provided with medical treatment for cigarette addiction.

Consideration for the use of debridement of the decubitus ulcer goes beyond the task of the simple removal of necrotic, infected, or compromised tissue. Clinical and experimental research in the area of wound debridement suggests that debridement improves host defense mechanisms and reduces active infection (13). Infected soft tissue or bone will prevent wound healing in primarily closed wounds or with secondary closure. Only tissue with a low bacterial count ($\leq 10^5$/g tissue) and with no β-hemolytic streptococcus will proceed to closure. To accurately document soft tissue infection when clinically suspected, tissue biopsy or a validated quantitative swab technique should be employed (14). Wound debridement, especially when performed surgically, may significantly enhance wound healing and recovery (15). Wound debridement reduces dysfunctional cell populations that hinder wound recovery and promotes the release of tissue cytokines and growth factors that promote wound closure (16). In treating the chronic decubitus ulcer, we must also consider the possible presence of osteomyelitis as a contributing factor for the impairment in healing the clinically observed. In patients with ulcers with exposed bone at the base of the wound the clinician should expect that periosteal contamination has occurred and that localized debridement of the boney surface will be necessary. In patients with large, complex, chronic decubitus ulcers radiographic and imaging studies of the boney integrity must be included in the work up. Three-phase bone scintigraphy has an accuracy of 90% or greater for the diagnosis of osteomyelitis in an otherwise normal bone. Radiolabeled leukocyte scintigraphy is now considered the gold standard for clinical evaluation of complicating osteomyelitis away from the spine and is readily available in most health care centers. Magnetic resonance imaging (MRI) techniques also afford a selective and sensitive means of evaluating the periosteal surface and bone marrow for signs of acute/chronic infection. The use of these tools has greatly improved our ability to monitor the boney floor of the decubitus ulcer and validate the results of the surgical treatment selected (17,18).

As we have previously established, wound debridement is required to remove necrotic or compromised wound tissue, eliminate the excessive bacterial burden of the wound caused by infected tissue, and remove dysfunctional or senescent cells within the margin of wound necrosis. Often an initial debridement of the decubitus ulcer will need to be followed by a

regular schedule of follow-up debridements to maintain the readiness and cleanliness of the wound for healing. It should also be remembered that while there are several different techniques of debridement available, more than one debridement method may be required during the process of wound bed preparation. At the present time there are six effective methods of decubitus ulcer debridement: autolytic debridement, enzymatic debridement, mechanical debridement, biological debridement, surgical/sharp debridement, and hydrosurgical debridement.

Autolytic debridement of the decubitus ulcer promotes the removal of necrotic tissue by rehydration using hydrocolloid and hydrogel dressings keeping the wound moist and promoting devitalized tissue removal by the body's own enzymes. These wounds should be cleansed after this method to remove all necrotic tissue. If tissue autolysis is not apparent one to two weeks after the use of this method another debridement technique should be used. The primary use of this method should generally be restricted to Stage I or II ulcers that are not infected. Autolytic debridement is also not recommended for very deep ulcers with superficial necrosis or decubitus ulcers that require wound packing (19).

Enzymatic debridement of the decubitus ulcer is achieved by the topical application of exogenous enzymes to the ulcer bed for necrotic tissue removal. Preparations such as streptokinase or streptodornase or bacterial-derived collagenases are currently used for this technique. Clinical experience with this method had not been uniformly acceptable. One popular use of enzymatic agents is in the application of the enzyme to the scored surface of a decubitus eschar. The eschar-scoring process usually involves the use of a scalpel on the desiccated tissue and may be associated with complications of this procedure. Moist, flimsy tissue debris, and eschar are best suited for enzymatic methods. The application of these enzymes may also be painful during initial application. For these reasons, enzyme applications are frequently performed selectively on decubitus ulcers of varying stages and complexity. Once the eschar or film of necrotic tissue has been removed the enzyme should be discontinued (20).

Mechanical debridement of the decubitus ulcer involves methods designed to physically remove necrotic tissue by wet-to-dry dressings, wound irrigation, or hydrotherapy. Using this method, the rehydration of the wound facilitates the removal of the surface eschar by mechanical separation and removes surface debris. Clinically, this method may be painful and if the wound is dry could lead to damage of the newly formed granulation tissues. Pulsed lavage systems of irrigation or pressurized fluid streams may be quite effective in removing loose necrotic tissue as long as the pressure is not excessive. Antimicrobial agents are often added to the irrigant to enhance the reduction of micro-organisms colonizing the decubitus ulcer. Irrigation pressures between 4 and 15 psi are needed for effective debridement. Irrigation pressures below 4 psi may not be effective for wound cleansing and pressures greater than 15 psi may cause tissue injury and force colonizing surface bacteria into the ulcer bed. Hydrotherapy with whirlpool immersion is theoretically used to remove bacteria, debris, exudates, and necrotic tissue. Problems with this method of decubitus treatment include wound maceration, possible ulcer cross-infection, and the theoretical risk of fluid embolism. Mechanical debridement is a relatively slow process for the decubitus patient but is employed safely by many centers in conjunction with other methods of wound debridement (21).

Biological debridement is accomplished with the use of maggot therapy. The larvae of *Lucilia sericata* (greenbottle fly) digest necrotic tissue and pathogens. While this technique has not gained great popularity within this country, it is usually rapid and very selective. In one clinical study by Sherman (22), maggot therapy was documented to be more effective and efficient in debriding chronic pressure ulcers than were the prescribed conventional therapies.

Surgical/sharp debridement is performed with surgical instruments (e.g., scissors, forceps, scalpel, and hemostat) or laser to remove selected necrotic or compromised tissue from the decubitus ulcer. This method may also be employed to remove areas of undermined tissue or prepare a wound bed for reconstructive grafting or flap closure. Surgical debridement is the primary choice of treatment if large amounts of infected, necrotic tissue or bone are to be removed in a quick and efficient manner. The debridement of large amounts of tissue should be performed in an operating room by an experienced specialist. Special considerations that are required for surgical debridement are: pain control or anesthesia, sterile environment, demonstration of

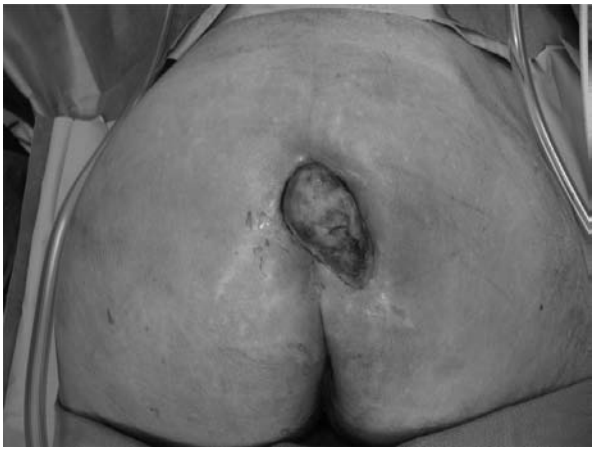

FIGURE 1
Chronic Stage IV sacral ulcer. *Source:* Courtesy of Mark S. Granick.

adequate vascular supply to the area being debrided, and absence of systemic sepsis or poorly controlled infection. Patients requiring systemic anticoagulation therapy will require special considerations for preparation for surgical debridement before undertaking this procedure. Risk assessment for the administration of anesthesia is necessary for severely ill patients considered for surgical debridement as well as the risks of intra- or postoperative hemorrhage or infection associated with the procedure. Surgical/sharp debridement is a complex undertaking that should involve a team approach. In performing this procedure, we must consider that we are creating an opportunity for a significant alteration in the decubitus ulcer that will prepare it for secondary closure or further reconstruction.

Hydrosurgery is the efficient debridement of necrotic and nonviable soft tissue by a newly developed surgical device called the Versajet™ (Smith & Nephew, Largo, FL). The VersaJet is a light weight handpiece that contains a Venturi-based irrigation and suction system moving saline irrigant through a 0.005-inch stream at up to 15,000 pounds per square inch (psi). As this supersonic stream of saline moves from a jet tube across an 8–14 mm operating window at the end of the hand-piece it is capable of holding tissue, cutting and scrubbing tissue planes, and removing necrotic debris and soft tissue which is easily and precisely pulled away from the healthy tissue margins within a wound. Nerves, vascular structures, healthy tendons, and ligaments are not injured by this debridement technique. For treatment

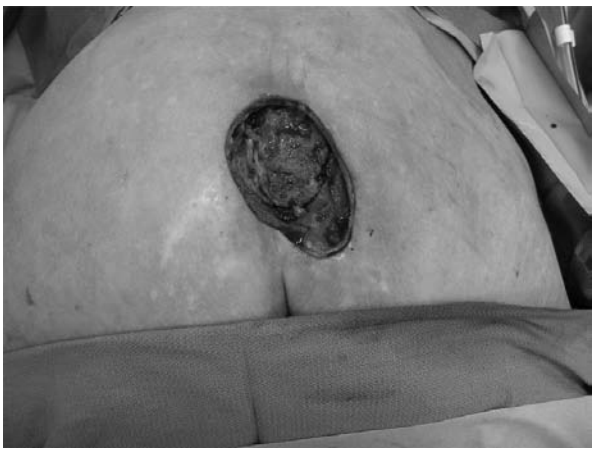

FIGURE 2
Wound edges were sharply debrided with a scalpel and the remaining wound was debrided with the Versajet™ (Smith & Nephew, Largo, FL.) *Source:* Courtesy of Mark S. Granick.

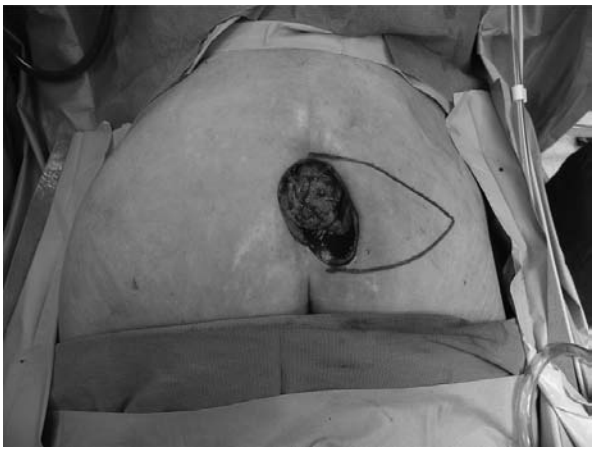

FIGURE 3
Immediate reconstruction with a gluteus maximus musculocutaneous flap. *Source:* Courtesy of Mark S. Granick.

of the decubitus ulcer, this tool appears superior to sharp or surgical debridement alone as a means of precisely removing the necrotic tissue from the wound and sparing the well-vascularized, healthy tissue with a minimal amount of bleeding or fluid spray. With experience, the surgical specialist may become well versed with the use of this very safe device for decubitus ulcer debridement (Figs. 1–4). With routine operations, it may function as a tool that may simultaneously mechanically and surgically debride necrotic soft tissue in a fraction of the time normally required for these procedures performed with traditional methods. Boney debridement must be performed with orthopedic instruments and the availability of a cautery device is advised as with any typical sharp debridement of a large volume of tissue. Clinical reports of the use of the Versajet with burn patients and leg ulcers document that this technique of tissue debridement offers more precise results and may accommodate difficult, complex wounds associated with undermining and sinus tract formation without ablating healthy intervening tissue beds (23,24). Our experience with the VersaJet also documents the ease of use of this device in the treatment of stage III and IV decubitus ulcer debridement (Figs. 5–7). Complex necrotizing wounds, often associated with decubitus

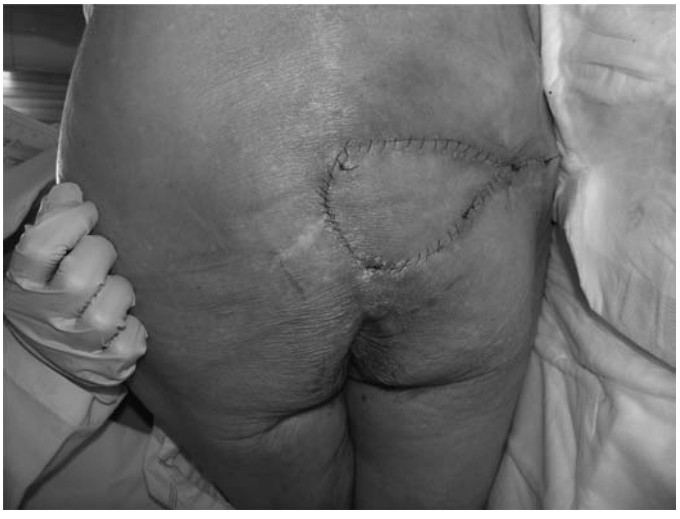

FIGURE 4
Healed reconstructed wound. *Source:* Courtesy of Mark S. Granick.

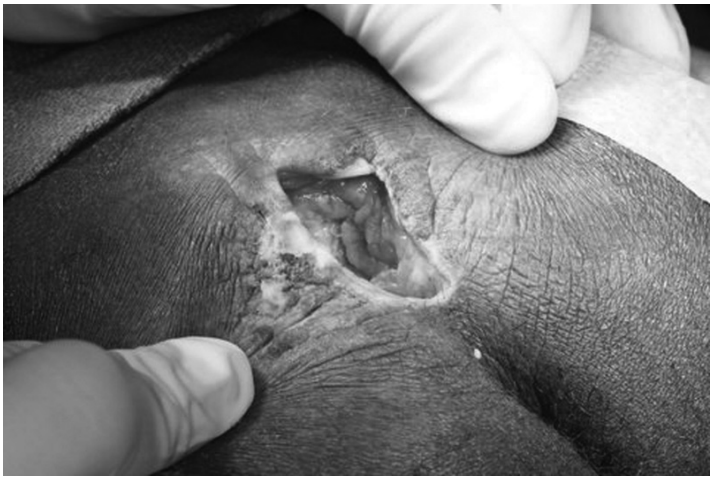

FIGURE 5
Chronic, infected Stage IV sacral decubitus ulcer.

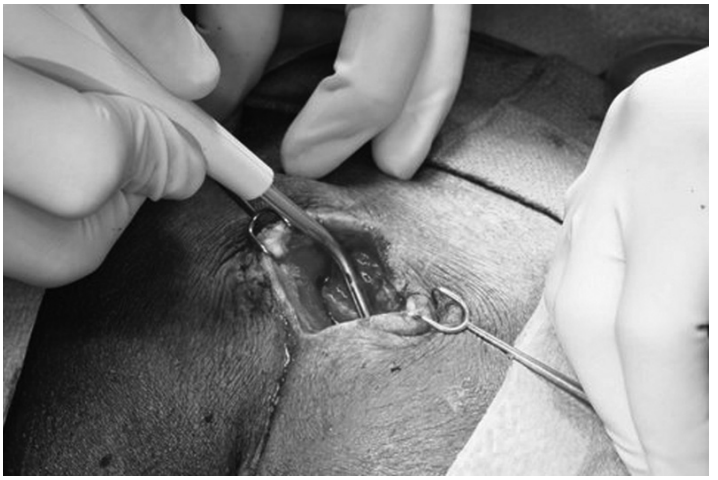

FIGURE 6
Chronic Stage IV sacral decubitus undergoing debridement with Versajet™ Hydrosurgery System™ (Smith & Nephew, Largo, FL).

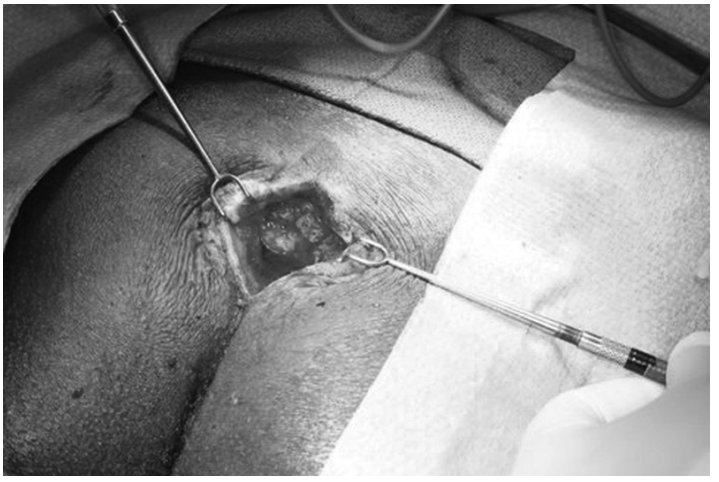

FIGURE 7
Stage IV sacral decubitus ulcer following Versajet™ debridement. (Smith & Nephew, Largo, FL.)

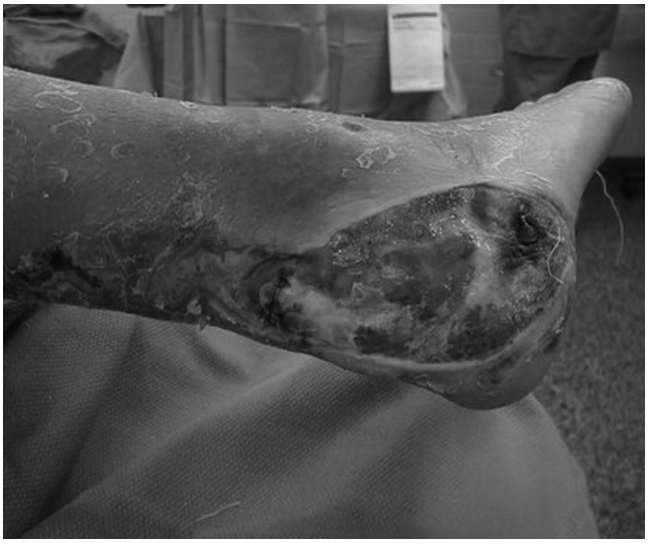

FIGURE 8
Complex, necrotizing cellulitis of left heel and lower leg that began as a necrotic, Stage IV decubitus ulceration of the heel in a patient with diabetic neuropathy of the lower extremities. (*See color insert.*)

formation, have also been observed with significant salvage of native soft tissue with the use this device (Figs. 8–11). In other cases of more complex infectious wounds of the trunk and buttock a single application of the Versajet hydrosurgery system has provided for efficient cleaning of a wound covered with eschar that subsequently experienced completed secondary healing without the need for further surgical treatment (Figs 12–14). Our experience has also demonstrated that moist dressings offer a synergistic effect with the VersaJet in those wounds requiring multiple debridements.

In conclusion, debridement of the decubitus ulcer should be accomplished as part of a comprehensive evaluation of the ulcer patient and should support the principles of wound bed preparation that are designed to accelerate endogenous healing. Adequate logistic preparation to provide appropriate support surfaces and nutrition is also critical for successful ulcer treat-

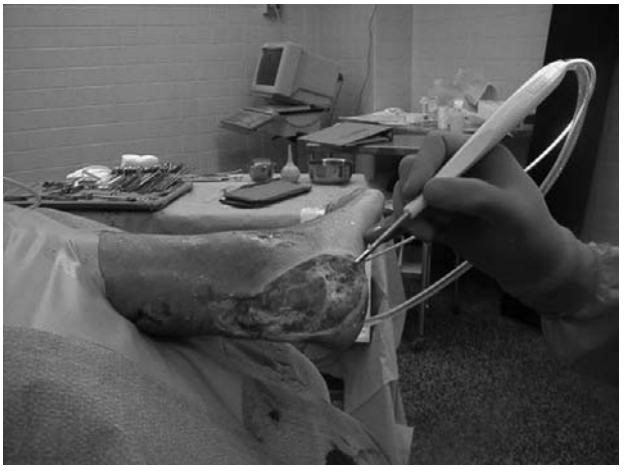

FIGURE 9
Versajet™ debridement of complex wound of necrotizing cellulitis of left heel. (*See color insert.*)

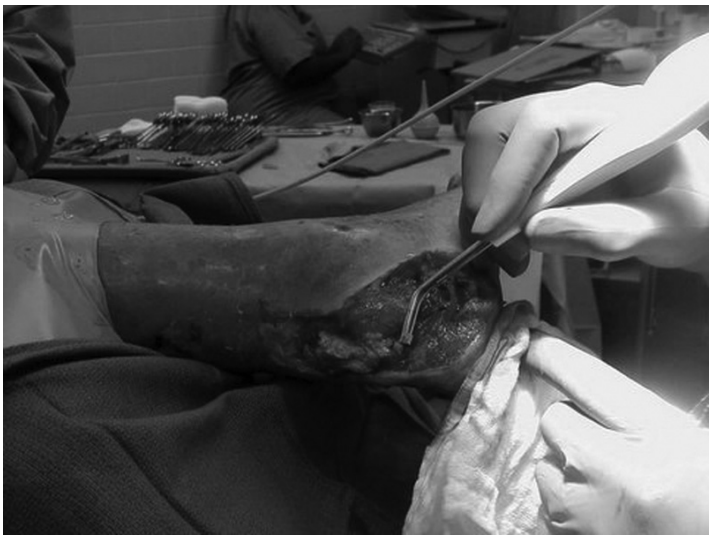

FIGURE 10
Versajet™ debridement of complex wound of necrotizing cellulitis of posterior lower leg. (*See color insert.*)

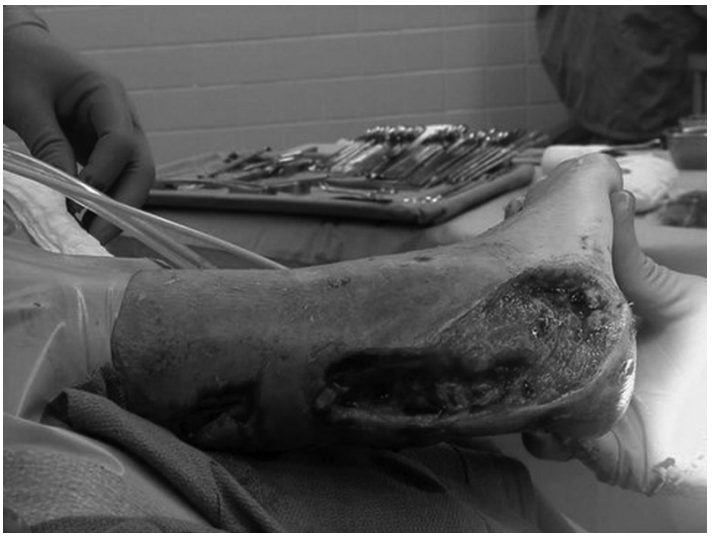

FIGURE 11
Wound of necrotizing cellulitis of left posterior heel and lower leg after completion of Versajet™ (Smith & Nephew, Largo, FL) debridement. Note precision of debridement and soft tissue sparing effects following the procedure. (*See color insert.*)

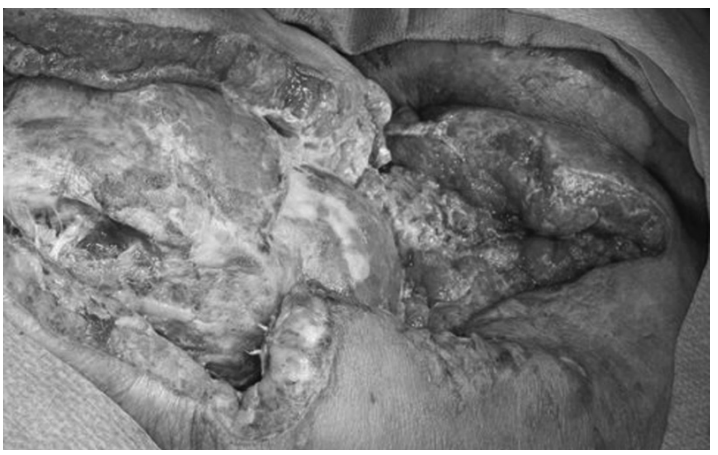

FIGURE 12
Complex, necrotizing faciitis of posterior buttock with extensive necrotic eschar coverage after initial surgical debridement of gangrenous skin, fascia, and abscessed tissues.

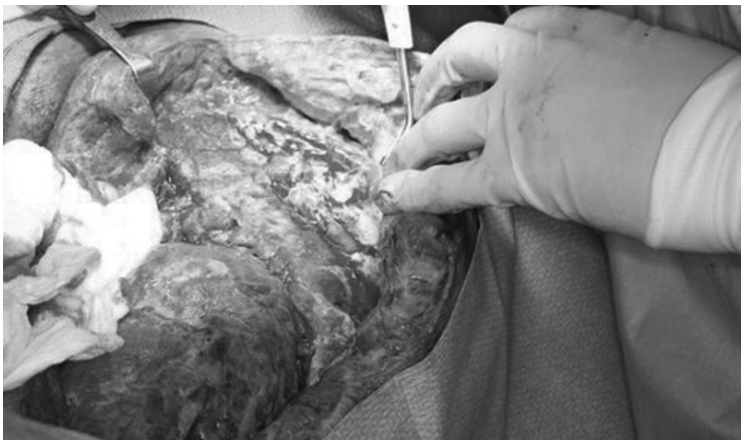

FIGURE 13
Versajet™ (Smith & Nephew, Largo, FL) debridement of complex buttock wound from necrotizing fasciitis.

ment. Surgical debridement should be considered the treatment of choice for Stage III and IV decubitus ulcers. However, as for any surgical procedure, the operative risks posed by the patient's illness and the complexity of the wound must be considered prior to the performance of this therapy. New fluid jet technology provided with the Versajet hydrosurgery system represents a significant novel advancement in our surgical approach to decubitus ulcer debridement. The Versajet system is designed to efficiently clean the ulcer bed while sparing viable tissue and combining the advantages of mechanical and surgical debridement methods in one device. With regard to ease and speed of use, efficiency of application, and decreased operative blood loss the

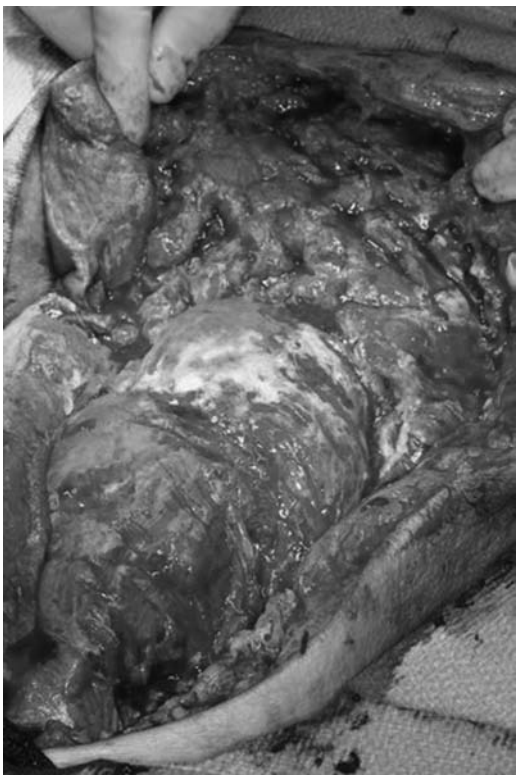

FIGURE 14
Area of complex necrotizing fasciitis of buttock and posterior right thigh after single session of Versajet™ (Smith & Nephew, Largo, FL) hydrosurgical debridement. Note thorough removal of debris and eschar with minimal trauma to underlying muscular tissues.

Versajet™ hydrosurgery system appears to offer significant advantage over our traditional sharp methods of surgical debridement.

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12 The Debridement of Chronic Vascular Leg Ulcers

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INTRODUCTION

Wound healing represents a complex process that involves several events; in acute wounds the healing process can be schematically divided into four phases: hemostasis, inflammation, proliferation, and remodeling (1).

In chronic, nonhealing wounds this process is completely disrupted because of many general and local causes; the underlying vascular disease, bacterial contamination and infections, diabetes and other metabolism disorders, anemia, tissue senescence, drugs, hypoxia, reduction of growth factors content, and increased proteolytic activity (2–9) can prevent the wound passage toward the next stage of the healing process.

Chronic wounds can be stuck in the inflammatory or proliferative phase (10–13); however, usually all chronic wounds tend to be characterized by nonresolving inflammation. This inability to resolve inflammation is believed to be the most significant delaying factor in the healing of chronic wounds (14).

In this case, a relevant tissue loss with tendons or bone exposition is possible; the wound may be covered by necrotic tissue or fibrin slough and other contaminants; the ulcer can be infected and undermined.

Necrotic tissue, fibrin, and tissue infection block the wound healing by prolongation and maintenance of the inflammatory phase and preventing the progress of the wound to the next stage of repair. Necrotic tissue, fibrin slough, and contaminants are, actually, a breeding ground for bacteria; they prolong the inflammatory reaction, mechanically prevent the ulcer contraction, and block the re-epithelialization (15).

When we face wounds at this stage, it is mandatory to eliminate all factors responsible of the healing blockade if we want to convert chronic wounds to healing wounds and hence promote the progress to the next stage of the healing process.

WOUND ASSESSMENT

Before dealing with wound management, we must always consider the underlying cause of the ulcer and ensure an adequate blood supply (or the correction of an impaired blood supply) to the leg and ulcer area. The great majority of ulcers are because of venous disease, arterial disease, or both (16).

This is a very important point and worthy to be underlined because dealing with ulcer management without worrying about the underlying disease leads us almost always to an unsuccessful outcome. In the case of venous ulcer, compression treatment must be applied together with local treatment. Compression therapy promotes ulcer healing through several effects: decrease of edema, softening of lipodermatosclerosis, decrease of venous volume, increase of venous velocity, blood shift into central compartments, reduction of venous refluxes, improvement of venous pumping, influence on arterial flow, improvement of microcirculation, and increase of lymph drainage (17). The effects of compression therapy on venous ulcer healing were evaluated in a review paper and a recommendation of grade A (the highest) was assigned to this therapy (18). From randomized controlled trials it turned out that compression increases ulcer healing compared with noncompressive treatment; high compression is better than low compression; multilayer bandages are better than single-layer systems. Compression therapy demonstrated the same healing rate when compared with venous surgery but is more effective in

preventing ulcer recurrence (19). Elastic stockings are also indicated but less effective (recommendation of grade B) (18).

For arterial disease, a simple screening method is represented by the Doppler ankle-brachial pressure index (Winsor Index) (20). It is generally accepted that an index ≥0.8 indicates the absence of significant arterial disease and does not contraindicate compression therapy. An index ≤0.5 indicates a severe arterial impairment and an unfavorable ulcer healing outcome unless arterial inflow can be increased by medical or surgical treatment.

ULCER DEBRIDEMENT

Debridement can be achieved by several methods—autolytic, enzymatic, biological, mechanical, and surgical (10,21,22). The first one permits a selective ulcer bed debridement, allowing the nonviable tissue removal and sparing the healthy one. The autolytic, enzymatic, biological, and mechanical methods can be carried out in outpatients because they do not need hospitalization; on the other hand, they require several days for the action to be completed. The surgical methods act in a very fast but nonselective way and can remove healthy tissue; they can also be very painful (23–25) and require hospitalization even if short-lasting (day surgery).

The more conservative and surgical methods can also be combined: the former method can be used to soften and loosen the necrotic tissues before surgical debridement or supplement the surgical debridement when we prefer to limit its aggressiveness to spare as much healthy tissue as possible.

Autolytic Debridement

Autolytic debridement (26–33) occurs spontaneously to a varying extent in all wounds and is because of macrophages and endogenous proteolytic enzymes, "which liquefy and spontaneously separate necrotic tissue and eschar from healthy tissue" (10).

The spontaneous autolytic debridement can be enhanced by moist dressings that create and maintain a moist environment capable of stimulating macrophages and endogenous proteolytic enzymes to carry out their action and promote tissue granulation.

The favorable effects of moist environment on wound healing were first described by Winter (34) on a swine experimental model and was repeated by Hinnman and Maibach (35) in humans.

They described a higher epithelialization rate for wounds occluded by a polyethylene film when compared with wounds freely exposed to air. Histological studies demonstrated that the dry environment causes further tissue necrosis probably because of the dehydration itself; it was also shown that epithelial cells can move faster in a moist environment when compared with a dry one (36). Subsequently, it was reported that a moist environment as created by semiocclusive or occlusive dressings does not induce bacterial growth or an increase in the infection rate but that, on the contrary, resulted in prevention by this peculiar condition (37).

Hydrogels are formed by insoluble polymers with a high concentration of water (up to 90%); sometimes they contain glycerine to delay water release; they can be manufactured as amorphous gel or compress and are the first choice in case of necrotic tissue or dry fibrin slough; they are unsuitable for wounds with high level of exudate and need to be changed within 24 to 72 hours. The amorphous gel needs a secondary dressing (e.g., film or hydrocolloids) in order to be maintained on the ulcer bed and avoid adsorption by the padding material of the bandage.

Hydrocolloids contain carboxymethylcellulose, other polysaccharides, pectin, and noncytotoxic adhesives. Their outer layer is formed by a waterproof polyurethane sheet that makes the dressing occlusive; they gel on contact with wound exudate and create a moist environment. When the adsorbing capacity of the dressing is lost it tends to detach and must be changed (usually after 24 to 48 hours in ulcers stuck in the inflammatory phase) (Fig. 1). They are indicated for mild to moderately exudating wounds. The hydrocolloids can also be used over granulation tissue and in the epithelialization phase. In this case, they can be changed even after one week depending on the exudate amount. Rarely, they cause allergic contact dermatitis (29).

Film dressings are formed by polyethylene and polyurethane with acrylic adhesive; they are permeable to water vapor and oxygen but impermeable to water and micro-organisms and

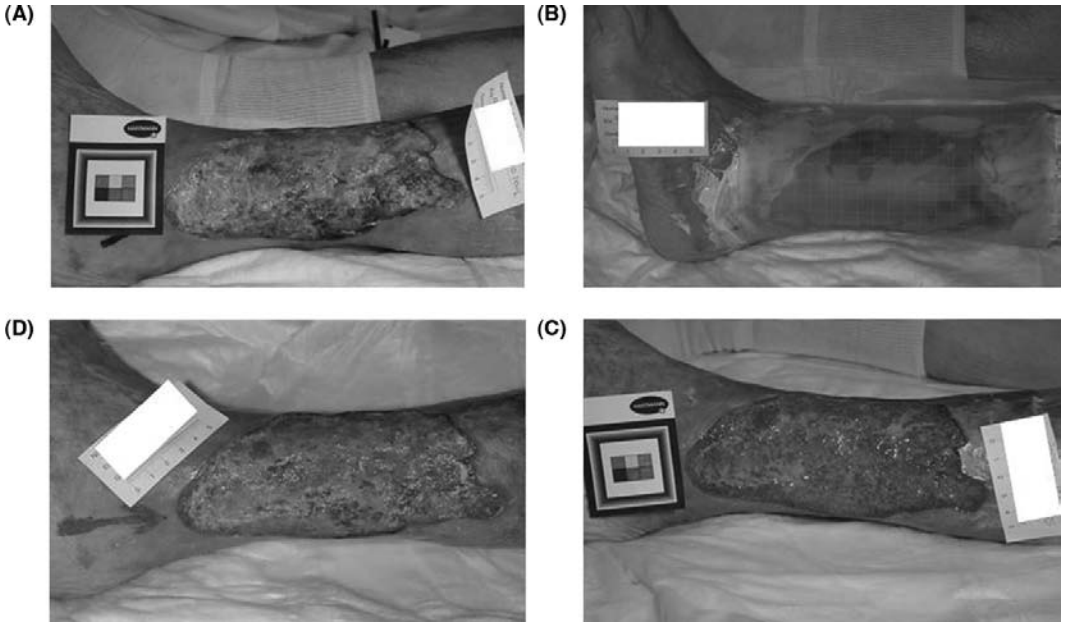

FIGURE 1
Large vasculitic ulcer: (**A**) covered by fibrin slough; (**B**) dressed with hydrocolloid; (**C**) the same after two days and (**D**) after eight days and four dressings. The ulcer bed is now debrided and is in the granulation phase.

do not have any absorbing ability. Their indication is restricted at the later stages of wound healing when there is not abundant exudate or as secondary dressing to keep other dressings (e.g., hydrogel) in contact with the wounds or to help in creating a moist environment.

Calcium alginate is formed by fibers of alginate (derived from brown seaweed) and tied together by calcium. They can have manuronic acid content (which gives a high gelling capacity) or galuronic acid content (which gives good fiber integrity). Calcium alginate exchanges calcium with the sodium ion in the exudate; calcium alginate is converted into sodium alginate causing the alginate to form a gel and promoting moist environment. The alginate gel adheres to the wound bed, trapping exudate and bacteria within its structure and promoting their removal during dressing change. The calcium donated to the wound fluid promotes a hemostatic effect. Calcium alginate is ideal in stimulating granulation tissue in exudative and infected wounds even when indented, undermined, or cavitary. Finally, the alginate gel can be removed without considerable trauma, allowing a poorly painful dressing change (Fig. 2).

Hydrofibers consist of sodium carboxymethylcellulose; they are able to absorb and lock considerable amount of exudate and bacteria into a cohesive gel and reduce the lateral wicking; they are indicated in moderate to heavily exudating wounds even if infected. Compared with alginate (31) they showed a longer wear time, a lower cost, better ease in application and removal but no statistical difference in numbers healed.

Polyacrylate is a new multilayered dressing pad containing superabsorbent polyacrylate activated with an appropriate volume of Ringer's solution. The polyacrylate has a greater affinity for protein-containing wound exudate than for salt-containing solutions (Ringer's solution); the wound exudate displaces the Ringer's solution from the superabsorbent pad. The Ringer's solution, in its turn, is given off continuously to the wound bed over hours (12 or 24 hours depending on the type of dressing used—if standard 12 hours or extended 24 hours). This permanent inflow of Ringer's solution softens and loosens necrotic tissue and, at the same time, superabsorbent polyacrylate absorbs and entraps bacteria, detritus, and toxins.

TenderWet®, therefore, can be used in infected and noninfected ulcers. According to our data (32), TenderWet 24 is capable of absorbing large amounts of exudate and can effectively and

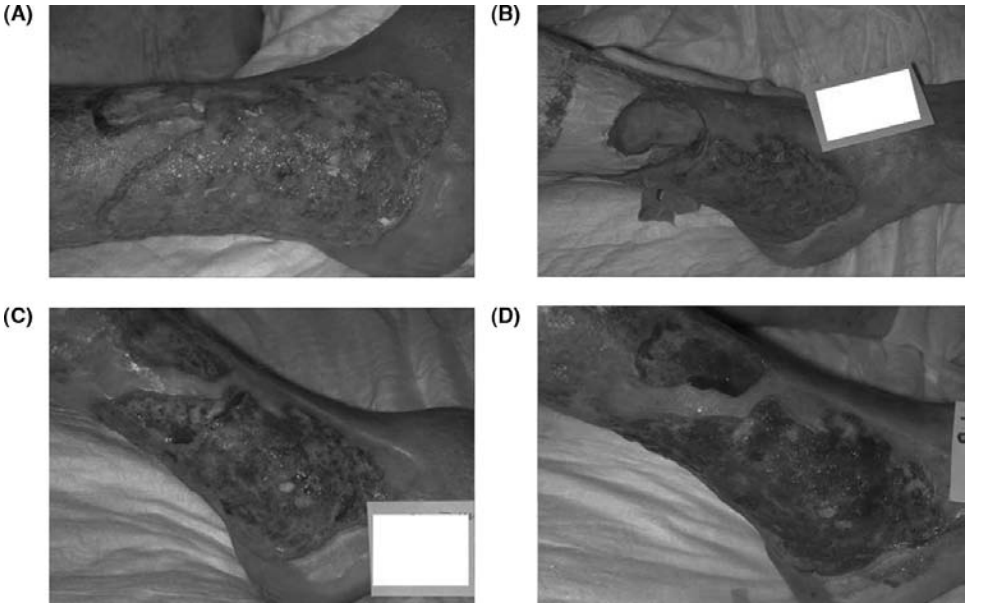

FIGURE 2
Large vasculitic ulcer: (**A**) covered by fibrin slough; (**B**) dressed with calcium alginate; (**C**) the same after four days and (**D**) after nine days and four dressings. The ulcer bed is now debrided and is in the granulation phase. (*See color insert.*)

quickly get rid of necrotic tissue, fibrin, and debris, promoting the appearance of the granulation tissue in a few days; it also has a noticeable effect in decreasing dramatically the bacterial burden, sometimes just after the first dressing, absorbing the bacteria and binding them inside the polyacrylate pad (Fig. 3).

The dressing is fairly atraumatic and slightly painful at removal if it is not moistened plentifully with saline solution; when removed, the ulcer bed is "clean" and further wound cleansing necessary with the other kinds of dressings is not required. This substantially contributes to the good compliance of the dressing as the mechanical ulcer bed cleansing during the change of dressing is usually the most painful event of all the procedures. When properly placed, TenderWet is well tolerated and does not cause any particular complication.

Infected Wounds: The Role of Silver

In an infected wound, the bacterial burden must be controlled, as bacterial cells produce several enzymes and toxins that can impede the healing process. We can choose between different approaches in order to manage bacteria in the wound: (*i*) employ sharp debridement to not only remove bacteria but also the necrotic tissue that represents the environment they populate; (*ii*) use occlusive dressings to prevent the penetration of exogenous bacteria into the wound; (*iii*) use topical antibiotics; in this case it is necessary to consider that antibiotics have a narrow spectrum as compared with the polymicrobial colonization that is usually seen in chronic wounds. They very rarely can reach an effective concentration as they are diluted by the wound exudate and can also induce sensitization reaction and bacterial resistance. For all these reasons using local antibiotics is not recommended and should be contraindicated; (*iv*) use topical antiseptic for infected wounds because of their effective broad spectrum against gram-positive, gram-negative, and also antibiotic-resistant bacteria. Nevertheless they have two major drawbacks: they are nonselectively cytotoxic and can also damage the host cells (fibroblasts and epithelial cells) necessary for wound healing and they have a very short duration of action.

Silver can play a major role in infected wounds because of its broad spectrum including highly resistant bacteria such as methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-

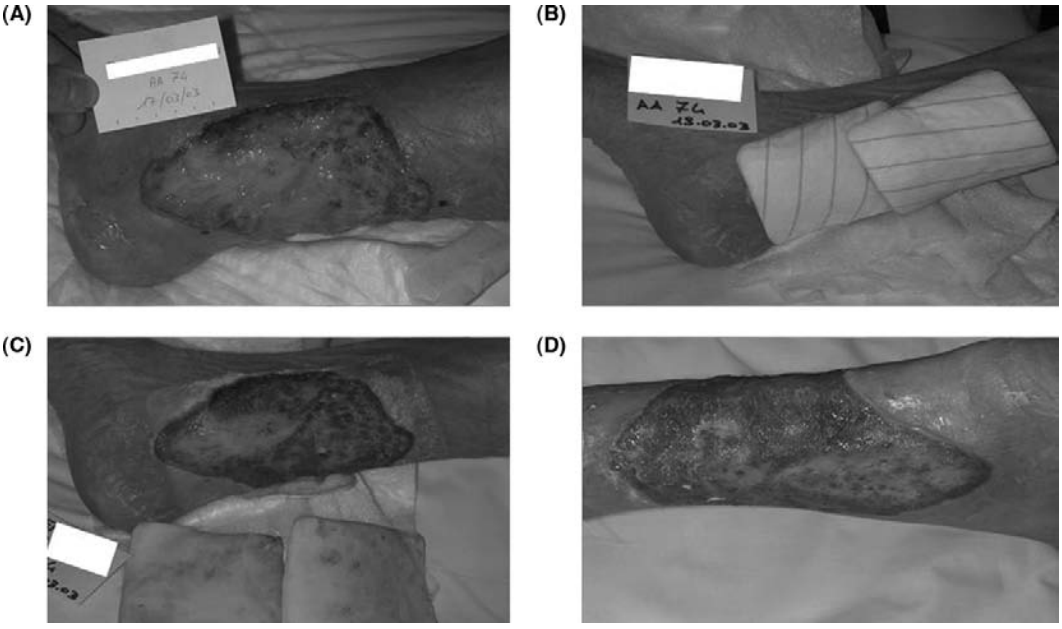

FIGURE 3
Arterial ulcer: (**A**) covered by fibrin slough; (**B**) dressed with polyacrylate; (**C**) the same after two days and (**D**) after seven days and seven dressings. The ulcer bed is now debrided and is in the granulation phase.

resistant *Enterococcus* (VRE) and *Pseudomonas aeruginosa*. Silver has a selective effect against bacterial cells and is apparently nontoxic to human cells. It can be released to the wound bed through a sustained release delivery system. Resistance to silver has never been shown.

Silver has been incorporated in a variety of dressings like alginates, foams, hydrocolloids, gauze, and hydrofibers. Silver exerts its antimicrobial activity only as silver cation, Ag^+, as elemental or metallic silver is not antimicrobial. Ag^+ has multiple binding sites on the bacterial cell and this explains why resistance to silver is very rare. The different dressings have different technologies to release Ag^+ and different amounts of silver but the difference of action among them is difficult to prove.

The silver-containing dressings can be schematically classified as (39):

- Those that release silver into the wound bed for antibacterial action;
- Those that absorb wound fluid and exudate containing infectious organisms into the dressing fabric, where the silver exerts its bactericidal action;
- Those that absorb wound exudate and also release silver into the wound bed; and
- Those that release silver sulfadiazine into the wound. The silver sulfadiazine molecule cleaves in the presence of the wound fluid to liberate silver and a sulfadiazine (sulphonamide) moiety; both have antibacterial activities. Sulfonamide may act against silver-resistant bacteria.

In each case, the dressings liberate more silver than is required for bactericidal action, which in tissue culture is estimated to be 10 to 40 parts per million (<60 ppm for particularly resistant bacteria).

Enzymatic Debridement

Enzymatic debridement (40–42) is carried out by using topical application of exogenous enzymes on the ulcer bed "where they work synergistically with endogenous enzymes to debride the surface" (10).

Several enzymes have been developed but only some are commercially available: bacterial collagenase, fibrinolysin/DNAase, and papain/urea. These enzymes inactivate fibrinogen and several coagulation factors, collagen types 1 and 2, and the necrotic tissue; they exert a selective debridement in saving the vital tissue—the most effective being the bacterial collagenase. Bacterial collagenase isolated from *Clostridium histolyticum* displays great specificity for the major collagen types in the skin (types 1 and type 2 collagen) and has been successfully used as an enzymatic debrider (10). None of the other proteases can digest collagen.

Papain/urea is a nonspecific proteolytic enzyme derived from *Carica papaya*. It can digest fibrinous material but not collagen; it can produce inflammatory response and induce pain. Enzymatic debridement is indicated for the removal of necrotic eschar without any surgical precedure and also if some score on the eschar is necessary before the application of the enzyme ointment.

The enzymes need to be changed frequently (three times per day) because they are inactivated by exudate inhibitors; this represents a limitation to their use because of the work overload for dedicated staff and the pain they cause to the patients during the dressing change. Rinsing of the wound with antiseptics containing silver, lead, or mercury must be avoided because they inactivate the enzymes. Finally, it is necessary to protect the periwound skin from the irritating effect of the enzymes.

Mechanical Debridement

In mechanical debridement, we use different techniques from the wound bed techniques, based on a nondiscriminatory physical force to remove necrotic tissue, fibrin slough, and contaminants from the simplest ones (wet-to-dry) to the more advanced (water jet or vacuum-assisted closure).

Wet-to-dry (43,44) is the simplest and oldest method that unselectively removes necrotic and healthy tissue; a gauze is applied wet on the wound and torn away when dried up, removing the embedded necrotic tissue. It must be repeated four to six times a day causing a considerable work overload for the nurses and an increasing cost compared with moist dressings. Furthermore, this method is painful and can damage newly formed tissues.

Equipment based on pulsed high-pressure water jet is useful in the removal of debris from the ulcer bed. Some devices used for this debridement technique can be painful when the water jet is driven into the ulcer surface at very high pressure (between 200 and 400 mbar), and can cause abundant vaporization and an aerosol effect that inhibits a good view of the treated area and represents a risk for the surgeon and his staff (inhaling devitalized tissue and contaminants that spread in the ambient). Furthermore, there is a possible concern regarding high-pressure irrigation that may drive bacteria and contaminants deep into the wound and adjacent tissues (45).

Another device combines a pulsed cleansing system with aspiration by means of a self-powered closed circuit, connected to an external aspirator with 1000 cc of sterile saline solution. It is mainly used by orthopedics to clean exposed fractures and also in wound debridement (46). This device seems to improve ulcer cleansing and promotes granulation tissue but more data are necessary.

Whirlpool is also used to loosen and remove the debris bacteria and necrotic tissue from the wound but it has to be used only in necrotic wounds at the inflammatory phase and is not suitable in wounds where granulating tissue and epithelial cells are reappearing because they may be removed as well. It may also spread the infection to other susceptible skin areas (47–49).

Ultrasound treatment was also used as a debridement procedure in wounds stuck at the inflammatory stage proving to be effective in debris removal and reduction of infection caused by bacteria (50,51).

The vacuum-assisted closure (VAC) (KCI, S. Antonio, Texas, U.S.A.) technique is widely used to induce wound debridement and promote granulation tissue after the first works by Argenta and Morykwas (52–55). In VAC therapy, a topical negative pressure (a suction force) is applied to the wound using a dressing interface and the wound is sealed with a semiocclusive film; this converts an open wound into a closed wound, provides a moist environment, and promotes healing. The negative pressure range varies from 50 to 200 mmHg, 125 being the most used.

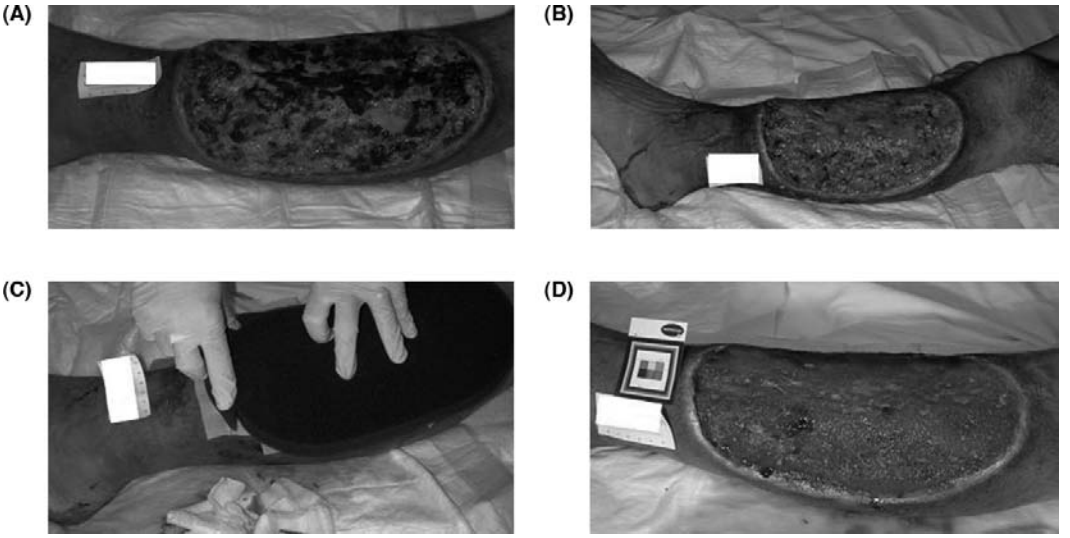

FIGURE 4
Large ulcer in a young, drug addicted, patient affected by hepatitis C and cryoglobulinemia. (**A**) Ulcer covered by necrotic tissue, deeply infected; (**B**) after removal of necrotic tissue by means of Versajet on the same day of admission to hospital; (**C**) vacuum-assisted closure (VAC) is applied immediately after Versajet™; (**D**) after four days and two VAC applications. The ulcer bed is now completely debrided and is in the granulation phase. (*See color insert.*)

VAC has proved to increase blood flow to the wound and the surrounding skin. As it was demonstrated that a continuous suction regimen could lead to an eventual decline in blood flow to baseline values after a few minutes, an intermittent pressure application was suggested. Other effects of this technique include: stimulation of granulation tissue, reduction of bacterial colonization, reduction of edema and interstitial fluid, and control of exudate. Several indications have been proposed for VAC therapy such as trauma, open tibial fractures, large skin/muscle loss, pressure ulcers, leg ulcers, poststernotomy infections, dehisced abdominal wounds, and skin graft fixation.

In particular, as regards venous leg ulcers, VAC can promote the granulation tissue but the results may vary over time. In mixed-origin or arterial ulcers the progression of the granulation tissue can be reduced because of the reduced arterial inflow. The techique can still be effective unless we deal with ulcers in critical ischemia where VAC therapy is not indicated. Finally, VAC can be used to secure the contact between the graft and the granulation tissue after a grafting procedure.

This technique cannot be applied on necrotic ulcers that are prepared with surgical debridement or other kinds of debridements. According to other authors (56), Versajet™ (Smith & Nephew, Largo, FL) is used for debridement purposes before applying VAC (Fig. 4).

Biological Debridement

Maggot debridement therapy (MDT) (57–60) was first introduced in the United States in 1931 and was almost abandoned after the advent of antibacterials. In the early 1990s, it was reintroduced first in the United States, and later in other countries such as Israel, United Kingdom, Germany, Sweden, Switzerland, Ukraine, and Thailand.

Today, sterile maggots of the green bottle fly, *Lucilia* (*Phaenicia*) *sericata*, are used for MDT; the maggots (up to 1000) are placed on the wound, covered with a semipermeable dressing, and left for one to three days. They secrete powerful enzymes that dissolve necrotic tissue and the biofilm that surrounds the bacteria without any damage to the healthy tissues. This therapy has proved effective in decreasing the bacterial overload in infected wounds, including MRSA. The

fluid derived by the digestion of bacteria and necrotic tissue forms a nutrient-rich liquid in which the larvae can live without the risk of drying and dying.

Maggot debridement therapy could be used for any kind of necrotic, sloughy wound on the skin even if deeply infected, independently of the underlying diseases and has been proven to be an effective method for cleaning chronic wounds and initiating granulation. It has been used for outpatients and also for hospitalized ones. This debridement is painless but the patient can feel the sensation of larvae movement.

Surgical Debridement

When a wound is largely covered by necrotic tissue or fibrin slough, when tendons and or bones are exposed and a considerable loss of tissue has taken place, and especially when the peri-ulcer soft tissue is contaminated, surgical debridement should be considered as more conservative procedures can imply several days or even weeks to get the ulcer bed debrided.

By definition, surgical debridement consists in the removal of all the foreign material or nonviable tissue from and around the ulcer until healthy, well-vascularized tissue is exposed on the wound edges and base; forceps, scalpels, scissors, curettes, and rongeurs are necessary. Surgical debridement can remove large amounts of tissue and is nonselective as healthy tissue can be removed as well; nevertheless, in selected cases it is considered the gold standard as it reduces the risk of wound complications and promotes the healing process.

Surgical debridement causes pain except for the patients suffering from neuropathy (diabetic foot) and it can be performed in the surgical theater in the case of large and painful ulcers when an extensive removal of the necrotic or infected tissue is required. In this case, a general anesthesia is usually necessary. In case of smaller or less painful ulcers, a more conservative and repeated debridement can be considered, trying to spare as much healthy tissue as possible; here, the debridement can be performed at the bedside or in a procedure room.

Bleeding could occur and can usually be stopped with a gentle pressure even if cautery or suture ligation is sometimes necessary. At the end of the surgical procedure, a compression bandage can help to stop bleeding and prevent its recurrence. With surgical debridement a chronic wound is transformed into an acute one hopefully progressing through the normal phases of wound healing.

Ulcer Debridement by Means of Versajet

Versajet (Smith&Nephew) is based on fluidjet technology which uses the Venturi effect: a high-pressure waterjet is pushed through a suitable hose to the tip of a procedure-specific handpiece; the "working end" is placed here between a jet nozzle and a collector which is 8 or 14 mm in length.

In this area, the waterjet runs at a very high speed and pressure (from 265 to 670 mph and from 103 to 827 bar depending on the 10-step speed setting on the console) and parallel to the axis of the device and is collected by the device, creating a localized vacuum. This results in capture and excision of the unwanted tissue centered in the working end and its aspiration at the collection point. This operating method prevents water splash, vaporization and aerosol effect, and allows good visibility during the entire procedure.

The operator can regulate the excising effect of the waterjet by adjusting its pressure and velocity (10 power settings) and modifying the handpiece direction and pressure. Higher waterjet speed, handpiece orthogonal direction (respect to the ulcer bed), and handpiece pressure (on the treating surface), the greater is the excising effect on the unwanted tissue, so that it is possible also to excise hard, contaminated tissues, and even bone. While reducing the power setting, steering the handpiece, and exerting a light pressure on it, the main action of the device is suction, irrigation, and scrubbing of the target tissue.

All vascular leg ulcers can be submitted to hydrosurgical debridement when an ulcer cleansing as quick and as complete as possible is required; we (61–63) used Versajet in 205 patients affected by vascular leg ulcers according to the following inclusion criteria:

- When 70% or more of the ulcer surface was covered by necrotic tissue or thick fibrin slough and in case of abundant and/or malodorous exudate and/or greenish material (Figs 5,6).

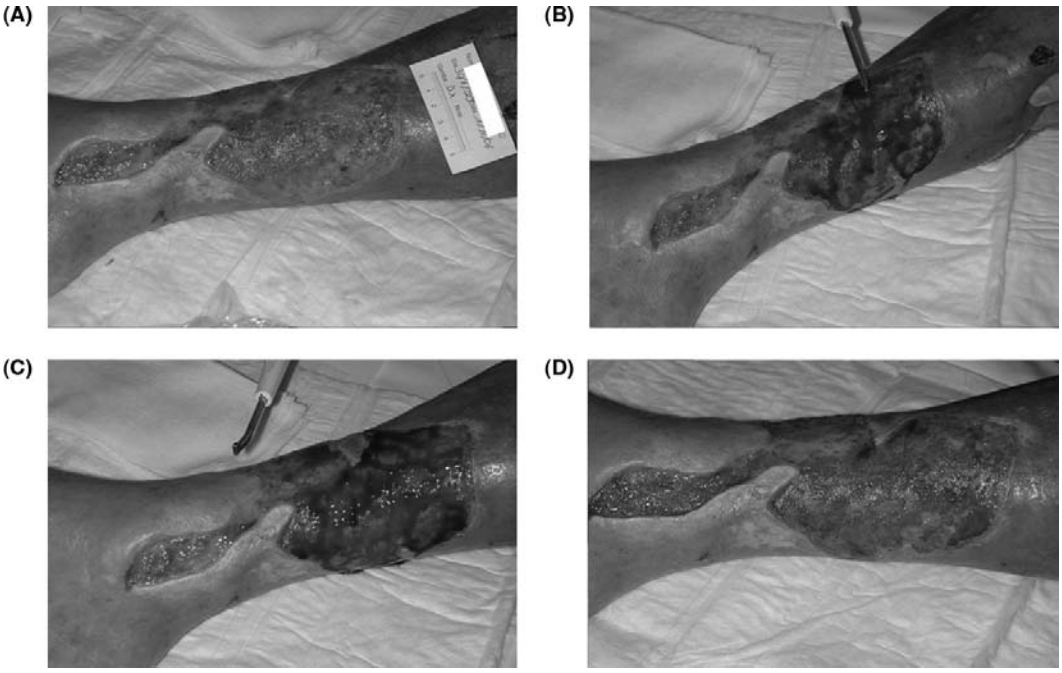

FIGURE 5
Ischemic ulcer in a patient affected by peripheral arterial occlusive disease already submitted to surgical revascularization. (**A**) Fibrin slough firmly stuck to ulcer bed; (**B,C**) debridement with Versajet; (**D**) the wound 30 minutes after the end of the procedure.

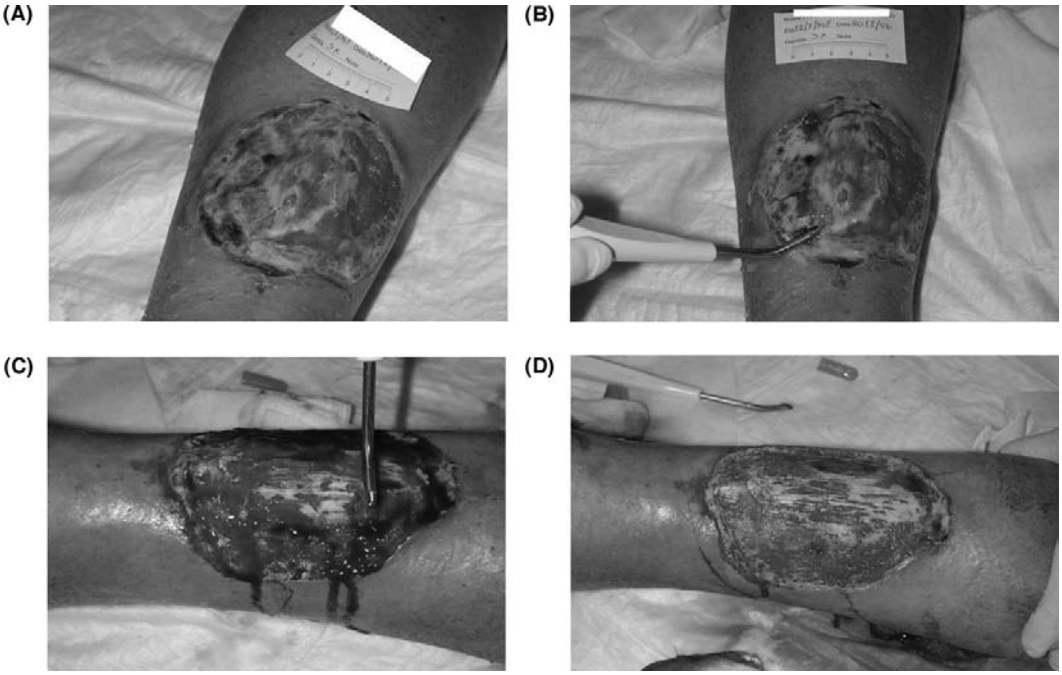

FIGURE 6
Large post-traumatic wound in a patient affected by post-thrombotic syndrome. (**A**) Necrotic tissue stuck to fascia and Achilles tendon; (**B,C**) Versajet debridement; (**D**) the wound 30 minutes after the end of the procedure.

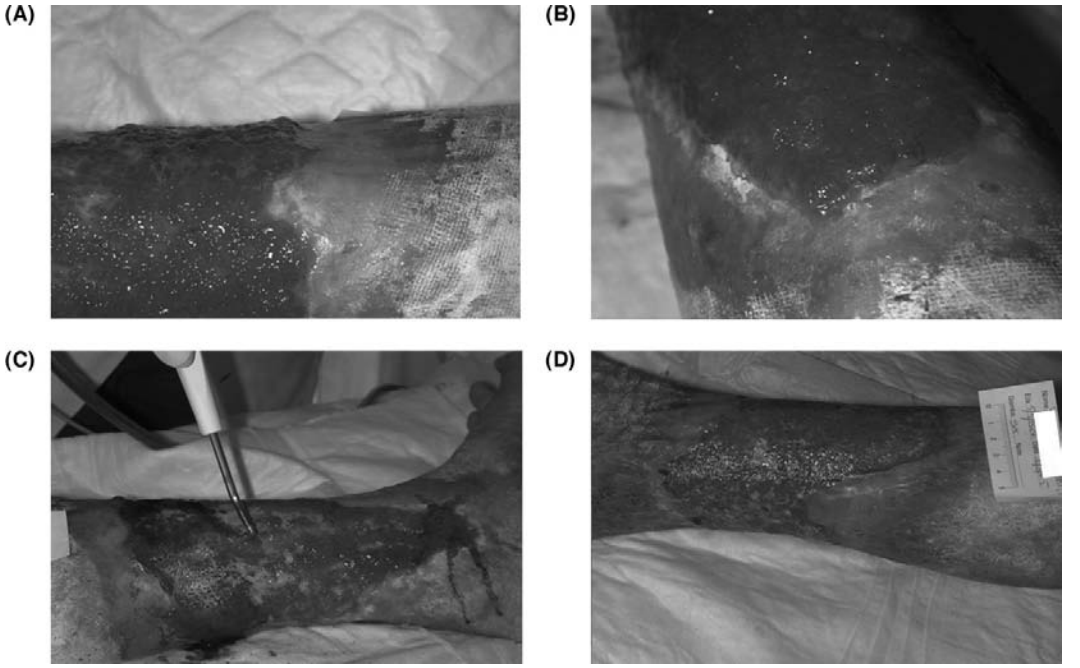

FIGURE 7
Vasculitis ulcer in a patient affected by scleroderma. (**A**) Ulcer with tendon exposition; (**B**,**C**) debridement with Versajet; (**D**) the wound 30 minutes after the end of the procedure; only the necrotic part of the tendon was removed and the vital part was saved.

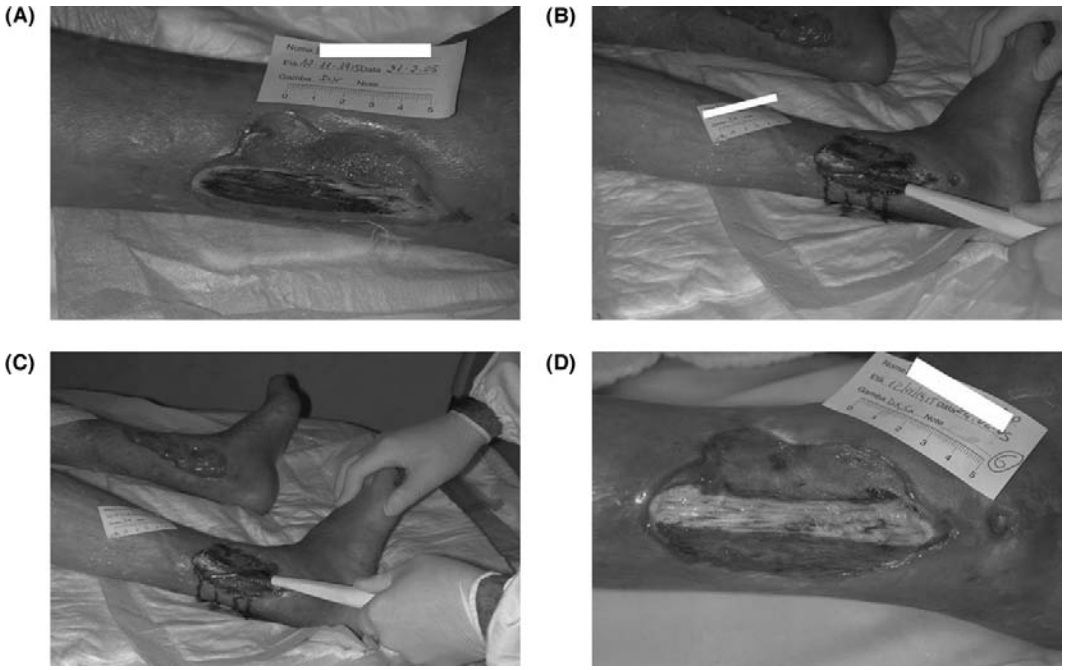

FIGURE 8
Large ulcer in a patient affected by venous insufficiency both superficial and deep. (**A**) Ulcer with hypertrophic granulation tissue; (**B**) different views of the same ulcer; (**C**) debridement with Versajet; (**D**) wound bed with a flat edge after the procedure.

- Presence of hypertrophic granulation tissue (Fig. 7).
- Exposed tendons or bone (Fig. 8).

There was no contraindication set to the use of the Versajet but by using it so extensively a possible contraindication to sharp debridement was accomplished in patients with ischemic ulcers: arterial disease at stages III and IV, vasculitis, and diabetic microangiopathy. In these cases, the excising effect on the ulcer edge can induce a necrotizing reaction that makes the ulcer larger. So far, it was not possible to use Versajet in an outpatient setting because of regulatory and reimbursement limitations; it would theoretically be possible when these problems are solved. The ulcer debridement with Versajet can be carried out in the surgical theater or in the ward.

The choice has to be made based on the ulcer size, level of pain, and wound infection. In case of large and painful and/or infected wounds, the patient has to be moved to the surgical theater because the local anesthesia is not suitable in preventing pain and a general anesthesia is preferable in order to guarantee an absolute absence of pain. In this condition, Versajet can be used at high setting to remove necrotic tissue, fibrin slough, and contaminants in a single step.

To treat smaller ulcers (<150 cm^2), especially if not too painful or not infected, the debridement can be performed in the ward under local anesthesia. Large ulcers can be debrided in the ward only when not painful (simultaneous neuropathy) or deeply infected or when a small area (in a large ulcer) has to be treated. When the procedure is carried out in the ward, it can be complementary and/or an alternative to the traditional moist wound dressing but it is necessary to be aware of the pain to the patient, which should influence the selected debridement method.

A careful evaluation of the baseline patient pain and tolerance to pain must be performed in order to avoid painful procedures (as sometimes happened in patients). If there is doubt, moving the patient to the surgical theater and a debridement under general anesthesia is preferable. In the patients selected for debridement in the ward, local anesthesia must be used to achieve a painless procedure or, at least, to cause a tolerable pain. To decrease the pain level, a more gentle debridement at low settings (<4), steering the handpiece, and decreasing the pressure is necessary.

Lidocaine/prilocaine ointment (LPO) or local xilocaine infiltration can be used for local anesthesia depending on the baseline pain. The combination of transcutaneous electrical nerve stimulator (TENS) and LPO can be useful in getting better anesthesia than the LPO alone because TENS strengthens the ointment effects. According to our experience, although used in more painful ulcers, this combination is able to reduce pain at the same level as LPO alone even if many cases are necessary for statistical analysis.

In the great majority of our patients treated in the ward, the pain caused by Versajet was well tolerated (considering its powerful action) when used at the recommended pump speed setting; however, a more extensive use of anesthesia is necessary in order to further minimize pain. Pain is always a major concern with every debriding procedure even for simple ulcer cleansing with gauze and saline solution. We report similar pain level for Versajet or debridement with gauze and saline solution and dressing change and (in most of the patients in control group) even if one considers that 51% of the patients treated by Versajet were given local anesthesia while the patients treated with moist dressings were given no anesthesia.

Considering the "global exposure" to pain, Versajet, by inducing a quick debridement and reducing the number of required procedures, decreases the cumulative pain level when compared with the longer debridement time necessary for moist dressings. In case of pain under local anesthesia, the procedure can be more long lasting as we have to adjust the debridement method continuously to the patient's level of pain. Also in this case a one-step procedure in a short time is usually enough to get a good debridement. Sometimes we have to get rid of hard necrotic tissue and/or fibrin slough firmly stuck to the ulcer and a sharp debridement in a single step, although possible, can cause an unacceptable removal of healthy tissue. In this case, it is preferable to perform daily, less aggressive, treatments with Versajet and, meanwhile, use moist dressings to prepare the ulcer bed for the next treatment.

The combined use of moist dressings and Versajet is synergistic. Versajet mechanically removes the unwanted tissue making the following lytic action of the moist dressings easier; the dressings soften the necrotic tissue making the following Versajet debridement action more complete. This attitude allows a more gentle debridement and spares the healthy tissue as much as possible.

In conclusion, even in the ward Versajet seems to be a valuable device for debridement of the ulcer stuck in the inflammatory stage in a single or a few steps. It proved to be extremely effective in reducing the bacterial burden and it also seems to be effective in changing the exudate composition. All these beneficial effects can be achieved with minimal pain and bleeding even in patients on anticoagulants.

The major drawback of debridement modality with Versajet is the high cost of the unit and of the disposable handpiece, especially if extending its use as an alternative or complementary procedure to moist dressings in the ward and in the outpatient. But, from a pharmacoeconomic point of view, it shortens the hospitalization and the healing time, promoting a quicker ulcer debridement and hence allowing for global cost saving in wound management.

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13 Debridement of Infected Orthopedic Prostheses

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INTRODUCTION

The implantation of any orthopedic prosthesis represents a major undertaking, the aim of which is to relieve pain and regain previously lost musculoskeletal function to degeneration or trauma. While the continued advent of biomechanical materials improves the longevity of these implants, infection remains a major nemesis compromising their function and longevity.

Despite the use of antibiotics and advances in surgical technique, the incidence of implant-associated infection in joint replacement remains between 0.5% and 2%, owing to the ubiquity of pathogenic organisms. Such an infected implant would then require further surgical debridement as a systemic antibiotic is ineffective in eradicating infection fully.

PATHOGENESIS

Host Factors

There are various host factors that increase the incidence of infection. These include systemic compromises to the immune system such as diabetes, and the presence of intercurrent infections during implantation such as chronic skin conditions or poor nutrition. In addition, the increasing age of the average orthopedic patient undergoing this type of surgery results in candidates with decreased lymphocyte efficiency secondary to thymic atrophy.

The use of immunosuppressant therapy required for autoimmune diseases such as rheumatoid arthritis increases the opportunity for infection to take hold. Other common medications that have been shown to decrease the efficacy of the immune system include corticosteroids and nonsteroidal anti-inflamatory drugs (1).

Tissue necrosis is always present to some extent following surgery. This coupled with hematoma formation and the presence of a large implanted foreign body provides an ideal bed for deep infection. Pathogenic bacteria are either deposited at the time of surgery or tract deeply from an infected surgical wound.

Bacterial Factors

The vast majority of implant-associated infections are because of skin commensals such as *Staphylococcus aureus* or *Staphylococcus epidermidis*. Together these generally account for more than 50% of the deep periprosthetic infections. In conjunction with other gram-positive bacteria, approximately 75% of the causal organisms are accounted for deep periprosthetic infections (2). Common bacteria implicated in periprosthetic infections are shown in Table 1.

The severity of the periprosthetic infection relies on both the host response and the virulence of the causal organism. In general, organisms that create a glycocalyx biofilm or are resistant to multiple antibiotics are particularly virulent (3) (Table 2).

Environmental Factors

The operation theater is the primary source of contamination of wounds.

Back in 1867 Lister identified airborne bacteria (4). Once a quantitative assessment of air contamination was established using slit lamp techniques (5), ventilation systems in theaters

TABLE 1
Common Organisms Associated with Periprosthetic Infections

Category	Organism
Gram-positive	*Staphylococcus aureus* *Staphylococcus epidermidis* *Streptococcus viridans* *Enterococcus*
Gram-negative	*Escherichia coli* *Proteus mirabilis* *Pseudomonas aeroginosa* *Salmonella* spp. *Klebsiella* spp.
Anaerobic	*Peptococcus* *Mycobacterium* *Clostridium bifermentans*

could demonstrate a significant reduction in surgical site infection (6). Charnley demonstrated a reduction in infection rates in hip replacements from 8.9% (theater exhaust ventilation) to 3.7% using plenum ventilation and a further reduction to 1.3% when multiple-filtered air enclosures are used with 300 air changes per hour (7,8). Ultraclean air theaters should show less than 10 colony-forming units per cubic meter and will reduce infection rates to 0.6% (9).

Theater personnel are a known source of bacterial contamination. This comes not just from nasal carriage but also by the shedding of skin squames. Each squame carries four to ten viable bacteria (10,11) and the human body sheds 3000 to 62,000 bacteria per minute (12). The use of impermeable body exhaust suits have been shown to further reduce infection rates in joint replacement to 0.3% (13).

Even with ultraclean air and body exhaust suits, contamination of wounds is inevitable and if the bacterial load is sufficient, and/or the host response is weak, then infection will ensue.

DIAGNOSIS

Acute Prosthetic Joint Infection

These either occur early in the postoperative period (first four weeks) or later and more rarely owing to hematogenous spread from a distant source of sepsis. They are usually characterized by sudden onset and the patients are often unwell with local and systemic symptoms.

In the acute setting, inflammatory markers such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) along with a full blood count (FBC) can be useful for supportive

TABLE 2
Common Organisms Classified by Virulence

Category	Organism
Less virulent	Are not antibiotic-resistant and do not produce a glycocalyx biofilm *Staphylococcus aureus* *Staphylococcus epidermidis* *Streptococcus viridans*
More virulent	Are antibiotic-resistant and produce a glycocalyx biofilm Methecillin-resistant *Staphylococcus aureus* (MRSA) *Staphylococcus epidermidis* Gram-negative bacilli Group D *Streptococcus* *Enterococcus*

evidence but cannot be used in isolation. The CRP is normally raised postoperatively, peaking at day 3 and falling back to normal levels three weeks postsurgery (14).

Obtaining specimens of joint fluid for culture are essential prior to any antimicrobial therapy. If the bacteria is cultured and antibiotic sensitivities are known, then the outcome of the surgery is significantly enhanced.

Chronic Prosthetic Joint Infection

The diagnosis of a chronic infected prosthesis is often challenging. They often present several months if not years following surgery. Patients complain of constant pain, especially "start-up" pain suggesting loosening of implant. Malaise, anemia of chronic disease, and occasionally sinus formation (15) are pathognomonic of the underlying infection.

Pyrexia is often not a useful indication of chronic infection as the vast majority remain afebrile (16). An estimation of the ESR and CRP may give an indication of concurrent infection although this is often not specific. The use of these markers are frequently unhelpful for diagnosis. The CRP is not always raised, and less than 50% of the confirmed infections have a raised ESR (17). Despite having limited use in the acute diagnostic phase, these parameters may be used in monitoring the treatment progress of periprosthetic infections.

Standard radiographic imaging may indicate radiolucent lines, focal osteolysis, or periosteal reaction and these may be associated with aseptic loosing of the prosthesis. However, any evidence of gross boney destruction or irregular periosteal reaction is often very suggestive of a periprosthetic infection (18).

Nuclear medicine scans such as three-phase bone scan can also be used and are very sensitive, but are nonspecific as aseptic loosening will have a similar result (19). Radiolabeled white cell scans profess a much higher sensitivity (90%) and specificity (85%), although these are labor-intensive, time consuming to perform, and expensive (20). In practical terms though, they are not very useful tools if the implant has been in situ for less than two years as there is a higher false positive rate.

Traditionally ultrasound, computed tomography, and magnetic resonance scans have a minimal role. However, the authors have been able to perform a magnetic resonance scan on a nonmetallic-infected total knee arthroplasty with good effect (21).

Aspiration of the prosthesis in question under sterile conditions in an operating theater after cessation of any antibiotics for at least two weeks represents the best way of obtaining laboratory diagnosis. It would require at least two samples to get a 96% accuracy of diagnosis (22). The aspirations are cultured in pediatric blood culture bottles and a separate specimen is sent for synovial fluid analysis looking specifically for primitive polymorphonuclear leukocytes. These primitive polymorphs are highly suggestive of an infection.

PRINCIPLES OF MANAGEMENT

The classification of periprosthetic infection was described by Coventry to apply to an infected total hip arthroplasty (23). However, this classification has been extended to include the principles of management and can be applied to various other prostheses (24). This is outlined in Table 3.

The principles of this management plan can be explained by the presence of biofilm produced by the virulent organism (25). The organism is protected in the biofilm which is characterized by cells that are irreversibly attached to each other, embedded in a matrix of extracellular polymeric substances (26,27). The immune system is able to gradually penetrate an immature biofilm within the first two weeks of infection (28) but is ineffective once the biofilm is established. It is for this reason that implant removal becomes necessary.

Surgical Management

The aim of surgical intervention is to eradicate infection and provide the patient with an infection-free stable functional prosthesis. We must be aware that in addition to eradicating infection, we should endeavor not to introduce a new infection into the surgical bed. The patient

TABLE 3
Classification and Management Principles of Infected Prostheses

Category	Symptoms	Management
Positive intraoperative cultures	Patient is often asymptomatic	Antibiotic therapy for six weeks
Early infection	Symptomatic infection within the first month of surgery Retain fixed components Polyethylene may be exchanged if possible Antibiotic therapy	Debride and washout prosthesis
Late chronic infection	Symptomatic infection after the first month of surgery Remove all fixed components as either a single- or two-stage procedure Antibiotic therapy	Debride and washout prosthesis
Acute hematogenous infection	Previously normal implant Acute infection following hematogenous spread Polyethylene may be exchanged if possible Remove fixed components if loose by either a single- or two-stage procedure Antibiotic therapy	Debride and washout prosthesis Retain fixed components

is made well aware that this is limb salvage surgery and not all cases will achieve the final surgical aim.

Patient Optimization

Once diagnosis has been achieved and surgical management has been agreed upon, the patient will need to be optimized prior to the procedure. All intercurrent medical problems are optimized to reduce the length of inpatient stay for reasons other than the postsurgical recovery. Any immunosuppressants are withheld if possible for as long as possible. In addition, any intercurrent infections are treated prior to surgery.

Operating Environment

The operating environment is vital to reduce any new bacterial contamination. Reducing bacterial contamination of the wound by limiting dispersal from the operating staff is essential. The number and movement of staff should be kept to a minimum. The usage of disposable exhaust suits, laminar airflow, and perioperative antibiotics all combine to reduce the total incidence of bacterial contamination (29).

Surgeon Asepsis

Although commonly taken for granted, surgical asepsis including hand asepsis has an important role. In addition to the basic surgical scrub, hands are cleaned in 70% isopropyl alcohol prior to surgical glove usage. This is based on the fact that while the usage of an iodine scrub eliminates up to 89% of bacteria, the addition of an alcoholic disinfectant can increase this level of asepsis to 97% (30) as illustrated in Table 4.

TABLE 4
Percentage of Asepsis 3 Hours Following Surgical Handwashing with Various Antiseptics

Antiseptic type	Percentage asepsis after 3 hours
Soap	12.3
Povidone iodine	89.4
70% ethanol	90.9
70% isopropyl alcohol	93.8
0.5% chlorhexidine with 70% ethanol	96.2
0.5% chlorhexidine with 70% isopropyl alcohol	96.9
4% chlorhexedine with 70% isopropyl alcohol	97.4

TABLE 5
Percentage Accuracy of Infection Based on the Number of Positive Tissue Cultures

Number of positive cultures	Percentage accuracy of true infection
1	10.6
2	41.0
>2	96.0

Surgical Draping

An alcohol-based antiseptic is used in the preparation of the skin in line with the amount of bacterial elimination (30) as shown in Table 4. In addition to this, the surgical field is draped with disposable impermeable drapes. A betadine impregnated plastic adhesive drape is then stuck onto the site of the surgical incision. This is because more than 50% of deep infections are a result of contamination by skin commensals, commonly *S. aureus* and *S. epidermidis* (2). Disposable surgical drapes are impermeable to both shedded squames from the operating surgeon or patient, as compared with woven reusable drapes (31). In addition, the usage of the plastic adhesive further limits the possibility of bacterial contamination from skin commensals (32).

Surgical Technique

It is common for the previous scar to be utilized while revising an infected prosthesis. However, if there is any sign of frank infection or of sinus formation, this region of skin should be avoided and debrided thoroughly. Good tissue care is essential to avoid the presence of dead or necrotic tissue postoperatively as well as hematoma formation that would result in a good culture medium for subsequent infections.

Tissue Samples

Tissue samples as well as fluid are taken from various sites prior to debridement. Each sample is taken with either a clean syringe or needle (for fluid) or clean scalpel and forceps for tissue (using separate instruments for each sample). Each specimen is labeled and processed individually to prevent cross-contamination. Samples are sent for both microbiological and histological examination. At least three positive cultures and histological results would be required to confirm infection and not due to the possibility of cross-contamination (22) as shown in Table 5.

Debridement

The complete debridement of infected tissue is essential in either a single stage or a two-staged revision of an infected prosthesis. All biofilms produced by the bacteria need to be removed. This includes various corners, regions close to neurovascular bundles, and within any boney cavities. This is often very difficult to excise or remove fully, owing to the adherent nature of the biofilm (26).

Sharp dissection with a scalpel is often excessive and imprecise, leading to concomitant injury to various normal structures. This macroscopic debridement is crude and invariably leads to increased subsequent scar tissue formation—a significant factor in postoperative stiffness, and poor functional outcome. Pulse lavage is useful in removing any loose material but quite ineffective against the adherent biofilm. It is thought that it may even drive bacteria into the soft tissues.

Versajet™ (Smith & Nephew, Largo, FL) (33) is a very effective tool both in debriding biofilm and various cavities while preserving adjacent normal structures (34). There is a great deal of control and accuracy in using this instrument for soft tissue debridement. By drawing up the infected synovium, it has the capacity for controlled removal of this surface. Normal underlying tissues are less readily removed unless higher settings are applied. Thus, a more

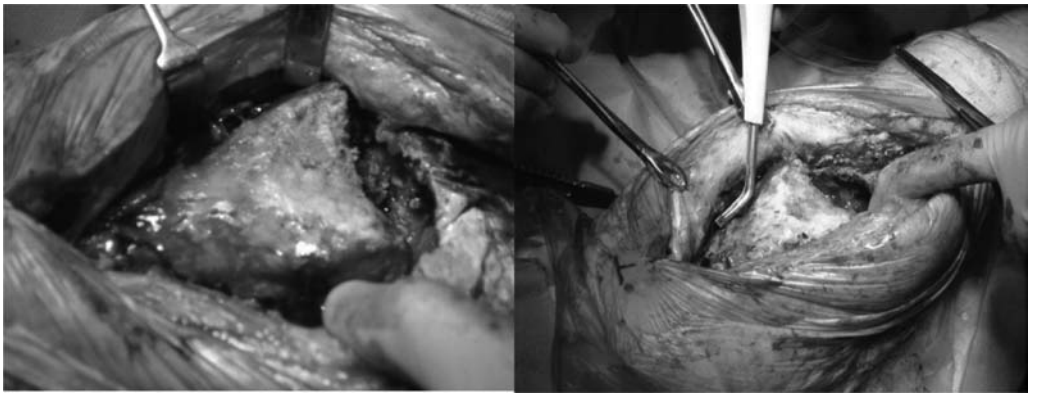

FIGURE 1
Before and after debridement with Versajet™ (Smith & Nephew, Largo, FL). (*See color insert.*)

thorough and less aggressive removal of infected material takes place (Fig. 1). The infected prosthesis and any remaining cement are removed fully unless debriding for an acute infection as explained previously.

Staging

In the acute setting (within four weeks postoperatively or acute hematogenous infection), open debridement and exchange of polyethylene is advocated (Fig. 2).

In chronic infection, a single-stage procedure involves removing the implant and all foreign material. Debridement of all the soft tissues and reimplantation of the prosthesis are all performed during the same operation.

A two-stage procedure requires an interval of several weeks or months between the initial removal and debridement and subsequent reimplantation. During this time an antibiotic

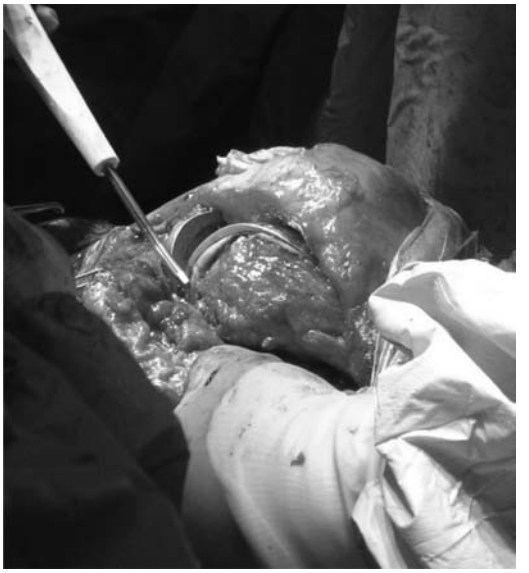

FIGURE 2
Debridement of an acutely infected knee replacement with Versajet™. Note that polyethylene has been removed for exchange.

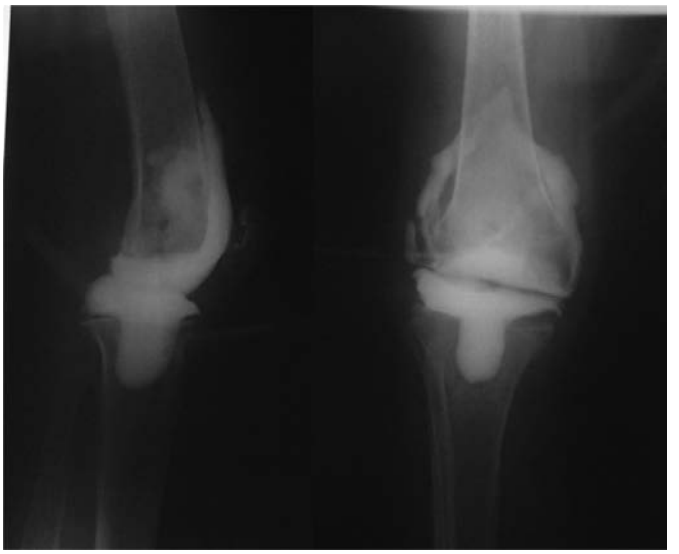

FIGURE 3
An antibiotic impregnated cement spacer in a two-stage revision for infection.

impregnated bone cement spacer acts as an interposition arthroplasty. This helps prevent soft tissue contracture and provides high dose local antimicrobial therapy. The choice between performing a single-stage or two-stage revision for an infected prosthesis remains controversial.

Advocates of the single-stage procedure quote the advantage of a cost-effective single procedure compared with two in an already frail individual (35). Success rates of up to 64% have been quoted in the Endo-Klinik, Hamburg for single-stage total knee arthroplasties, rising to 81% following a second revision in unsuccessful cases (36,37). The success of the more commonly performed two-stage procedure range from 12% to 85% (34,38–41) (Fig. 3).

A multifactorial approach is prudent in determining the choice between a single-stage and a two-stage procedure. Patient factors unconducive toward a single-stage procedure include major problems with soft tissue quality such as the presence of draining sinuses or overt evidence of concurrent immunosuppression. Other factors include the inadequacy of surgical debridement as well as the virulence of the organism encountered.

Reimplantation

Once debridement is completed, and a decision is made regarding a single-stage or two-stage procedure, reimplantation can be performed. The surgical wound is scrubbed again with antiseptic and the limb is redraped. Surgeons rescrub and use new surgical gowns. Reimplantation is performed as a clean procedure. Antibiotic impregnated cement is used to fix the prosthesis or used as a spacer in the two-stage procedure. The elution of the antibiotic from the cement provides an added barrier toward reinfection (42) (Fig. 4).

DISCUSSION

With the use of sound basic principles of diagnosis and initial and surgical management, the debridement of infected orthopedic prosthesis can be undertaken with a reasonable degree of success, giving your patient a functional quality of life.

However, the cost, both human and financial, of treating these cases is huge. The misery and suffering prior to and following surgery is enormous, with failure resulting often with amputation. In our institution, we have been able to carry out single-stage procedures on these difficult cases using the VersaJet system. This has significantly reduced the amount

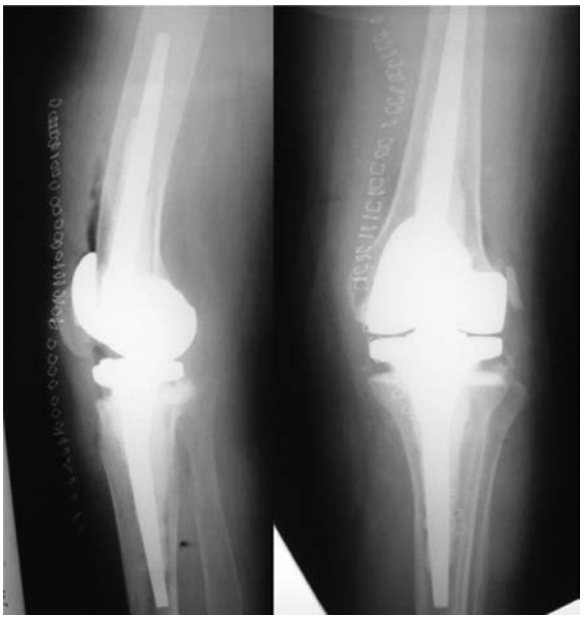

FIGURE 4
Reimplantation of a rotating hinge knee prosthesis.

of surgery a patient has to endure and reduced the cost of management significantly. Anecdotally, they also seem to get a better functional outcome probably because of a combination of factors including a more selective debridement, less scar tissue formation, and earlier rehabilitation.

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14 Surgical Debridement of Diabetic Foot Ulcers

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INTRODUCTION

Surgical debridement is the physical removal of necrotic material, foreign bodies, and infected tissue from the bed of an acute or chronic ulcer, with an evident secondary reduction in bacterial load. This treatment helps to obtain a more suitable environment for restoring the physiologic processes of tissue repair.

An aggressive surgical approach to ulcers, recognized in the treatment of acute lesions or burns, can also be beneficial in the treatment of chronic lesions. One of the most important motives for using debridement is, in fact, to transform a chronic lesion into an acute wound that has more suitable characteristics for healing. The frequency of the debridement of a lesion depends on various factors, such as the time the lesion has been present, whether the surface and deep tissues and bone are infected, and whether concomitant vascular disease is present. Steed et al. noted the positive effect of surgical debridement performed routinely on chronic lesions compared with its sporadic use (1).

Routine debridement seems to effectively remove factors that inhibit tissue repair (proteases, collagenase, elastase), promoting instead the production of growth factors for tissue repair (1).

The importance of the removal from the ulcer of factors inhibiting tissue-repair accounts, for example, for the rationale underlying treatments, such as negative pressure wound therapy (NPWT) (Fig. 1); this type of treatment is capable of positively guiding tissue repair toward healing by means of a constant suction effect produced by the negative pressure, facilitating the removal of proteases and bacteria from the bed of the lesion, and allowing contraction of the lesion and finally neoangiogenetic activity (2,3).

Diabetic foot disease is a major health problem that concerns 15% of the 200 million patients with diabetes worldwide. Major amputation, above or below the knee, is a feared complication of diabetes. More than 60% of nontraumatic amputations in the western world are performed in the diabetic population. Major amputations increase morbidity and mortality and reduce a patient's quality of life. The treatment of foot complications amounts to one of the main items in the consumption of economic and health resources by the diabetic population.

A history of ulceration is frequently present in the majority of patients who undergo an amputation. It is clear that an effective treatment allows a reduction of major amputations.

In 1989, the St. Vincent Declaration cited a reduction by 50% of the number of major amputations in five consecutive years among its most important objective. This was in addition to improving the quality of care worldwide for patients affected by diabetes mellitus (4). Although some population figures of amputation have not shown a fall in the number of diabetic patients (5,6), reports from Sweden (7), Denmark (8), Italy (9–11) and United Kingdom (12) have shown a reduction in major amputations.

It has been estimated that 15% of diabetics encounter a foot ulcer at some point in their lives (13). The incidence and prevalence of the diabetic ulcer in literature varies according to the population and the method of survey used. Studies carried out in the United Kingdom have highlighted a prevalence of this lesion varying between 5.3% and 7.4% in the diabetic population (14,15). In the United States, Ramsey et al. highlighted a cumulative incidence of 5.8% of ulcerated lesions in the diabetic population discharged from hospital over three years (16). In Sweden, a yearly incidence of 3.6% of ulceration was recorded (17), whereas in Holland, a yearly

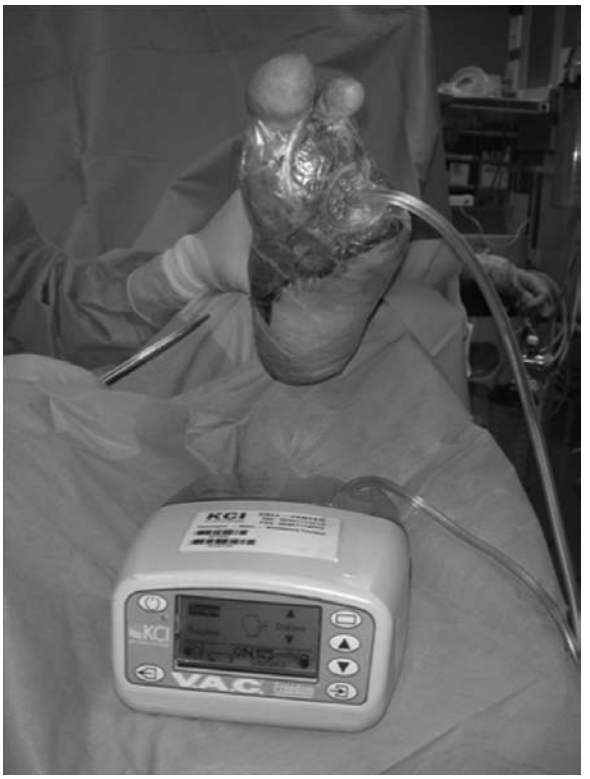

FIGURE 1
Negative pressure wound therapy.

incidence of 2.1% of ulcers in the diabetic population type II was shown (18). In a large community survey in the United Kingdom, the annual incidence of foot ulceration was slightly more than 2.0% among all patients with diabetes (19) and between 5.0% and 7.5% among diabetic patients with peripheral neuropathy (20).

More than 60% of nontraumatic amputations in the Western world are performed in the diabetic population. The incidence of major amputations within the diabetic population varies from 0.5 to 5 per 1000 diabetic patients (21,22).

In the general population, rates vary between countries, racial groups, and within countries. They can exceed 20 per 100,000 people (5,23–27). Ulcerations, and above all, amputations are made worse by incorrect prognosis (28,29). Morbidity and mortality rates are higher in the amputated population. Mortality during surgical amputation is high: 9% in a Dutch study (30), 10–15% in the United Kingdom (31). A recent retrospective work by Aulivola et al. has shown the rate of mortality within 30 days after major amputation (above or below the knee) reaching 10% (32). In a follow-up study of an amputated population, Faglia et al. have shown a three-year survival rate of 50% (33).

During the last 20 years, a progressive increase in physiopathological knowledge and treatment methods for diabetic foot have been witnessed. The percentage growth of limb salvation in patients treated in multidisciplinary units is linked to the improvements of treatment technique of an acutely infected foot, neuropathic foot, and of the critical ischemic conditions of a neuroischemic foot. Some notable physiopathologic knowledge concerning the development of the ulcer has been important in putting into effect a therapeutic behavior which has in turn shown itself to be particularly effective.

Over recent years, we have seen a significant increase in knowledge about the physiopathologic pathways of this complication. This has been associated with an improvement in

the diagnostic techniques, above all, a standardized conservative therapeutic approach that allows limb salvage in a high percentage of cases. This target has been achieved in specialized centers. An important prelude to diabetic foot treatment is the differing diagnosis of neuropathic and neuroischemic foot. This differentiation is useful for an effective treatment.

Ulceration in the neuropathic foot arises from biomechanic stress and high pressure that involves the plantar surface of toes and metatarsal heads. The treatment of a neuropathic plantar ulcer must provide, through weight-bearing relief, correction of pathologic pressures. Surgical treatment of deformities, with or without ulcerations, is an effective therapy. A neuropathic ulcer that is not adequately treated can become a chronic ulcer that does not heal. The presence of neuropathy reduces or completely eliminates pain, so that the ulcer can be tolerated by the patient for a long time. However, an ulcer that does not heal for many months has a high probability of causing osteomyelitis, where treatment with antibiotics is not useful and usually requires a surgical procedure. Charcot neuroarthropathy is a particular complication of neuropathy that can lead to fragmentation or destruction of joints and bones. A well-timed diagnosis of Charcot neuroarthropathy is essential to avoid deformities of chronic evolution.

Ulceration in a neuroischemic foot is caused by minor traumas, often conflicting with shoes. In recent years, it has been established that peripheral vascular disease is, in the diabetic population, the main risk factor of amputation. If peripheral vascular disease is ignored, surgical treatment of the lesion, without revascularization, cannot be successful. In diabetic patients, peripheral vascular disease is especially distal, but often fully involves the femoral, popliteal, and tibial axes. Tibial arteries can be successfully treated either with open surgical procedures or with endovascular procedures.

Infection is a serious complication of the diabetic foot, especially if neuroischemic: phlegmons or necrotizing fascitis represent not only a limb-threatening problem, but also a life-threatening one. In this case, emergency surgery is needed.

Prevention is the main objective. Prevention programs must be carried out to point out risk factors, lowering amputation incidence. Surgical debridement has a primary role in the topical treatment of ulcers of the diabetic foot. It is clear that the type of treatment would differ according to the therapist, the type of lesion, and the health center treating the patient. In short, although it is always defined as surgical debridement, we should distinguish between the simple removal of the perilesional callus of a plantar neuropathic ulcer and the surgical debridement of a phlegmon; the former can be treated on an outpatient basis by nonmedical personnel, whereas the latter would require emergency surgery by skilled medical staff.

ASSESSMENT OF THE LESION

Although the pathophysiologic principles that generally govern tissue repair also apply to diabetic foot, a number of unique diagnostic and therapeutic guidelines must be acknowledged in this specific context. The risks are known of progression to gangrene, resulting in amputation, of an ulcer that is not correctly assessed and treated. This justifies a more aggressive approach to the diagnosis and treatment of vascular problems and infections.

After an evaluation of the history (Table 1), the lesion should be evaluated, determining its size and depth, and photographs should be taken before any debridement procedure. This procedure takes place between diagnosis and treatment, as it may demonstrate the real extent of damage to deep tissues and bone.

TABLE 1
General Points to be Assessed Prior to Surgical Debridement

- Course of the lesion
- Bleeding history
- Drug history
- Previous surgical procedures
- Clinical and radiological documentation

A vascular assessment must be performed prior to surgery. Vascular assessment, as we perform it, comprises a clinical assessment to detect peripheral pulses, Doppler echocardiography, and monitoring of transcutaneous oximetry (TcPO2) (9,11).

The infection of the surrounding tissues should be determined, and if present, would influence therapeutic choice [e.g., regarding the type of antibiotic treatment, surgical debridement, type of care (inpatient or outpatient)] and the prognosis. One of the most widely used practices is marking the area affected by cellulitis with a dermographic pen and subsequent checks on whether the area has regressed, remained stable, or progressed. If the local signs of infection exceed the limits previously defined, 24 hours after the start of the treatment, the treatment would then probably have been inadequate.

The possible involvement of deep structures (tendons or capsules) or bone should always be taken into consideration when assessing the depth of the lesion; the use of a blunt probe would help in doing this. The procedure known as probing to bone is considered essential in the literature and is often sufficient to diagnose osteomyelitis (34). As the involvement of the tendon compartments means a significant risk of the proximal progression of the infection, these should be inspected carefully; if the progression of infection is strongly suspected, these areas should be incised and exposed.

Despite the importance of the clinical examination for the diagnosis of osteomyelitis, X rays with standard projections should always be performed. It should, however, be kept in mind that in order to yield a positive X ray, a focus of osteomyelitis must cause the loss of approximately 50% of the bone mass over about three weeks. More complex examinations, such as magnetic resonance or leukocyte scintigraphy of the bone are unnecessary in the majority of cases. Such examinations should consequently be reserved for doubtful cases which are difficult to diagnose, for example, Charcot's neuroarthropathy, or in which the osteomyelitis is not localized, but believed to be diffuse, for example, with the involvement of the tarsal bones and ankle bone, or in osteomyelitis of the heel.

GENERAL TECHNICAL CONSIDERATIONS

Debridement should be performed in an operating theater if the extent of the lesion requires locoregional anesthesia or if there is a risk of hemorrhage. The procedure should be performed without the aid of a tourniquet to effectively assess the bleeding and the damage to the soft tissues.

The fundamental purpose of surgical debridement should be the complete removal of infected and necrotic tissue from the edges and bed of the lesion. The treatment should therefore be a balance between the necessary level of aggressiveness, to remove anything that may impede tissue repair, and the need to leave healthy tissue intact. The excision of damaged tissue should, in any case, always be complete, and should not stop until it reaches macroscopically healthy, bleeding tissue. Cleansing is important after debridement, preferably by high-pressure irrigation (35). This procedure allows deeper decontamination of both necrotic tissue and the bacterial load owing specifically to the pressure of the irrigation (Fig. 2).

There are various surgical instruments suitable for surgical debridement, and they depend on the preferences of the doctor performing the treatment. Usually bistouries, curettes, scissors, and rongeurs are used (Luer, Liston) (Fig. 3).

New instruments for surgical debridement have recently been launched in the market. These instruments use ultrasound (Fig. 4) or very-high-pressure water jets (Fig. 5) for the selective debridement of necrotic tissue. This instrument (Versajet™, Smith & Nephew, Largo, FL) is an innovative hydrosurgery system that can rapidly and selectively remove necrotic tissue from an ulcer. The high-speed water jet, projected through an operating window, creates a cutting and aspiration effect on necrotic tissue, so that it can be cleaned far more rapidly and effectively than with traditional systems (bistouries). The waterjet works by means of the Venturi effect. Our data show a reduction in cleansing times, in necessity of the operating theater to obtain adequate debridement of the ulcer bed, and finally in healing times (36).

If the skin is permanently damaged by the infection or by primary ischemia or ischemia secondary to the infection, it should also be removed surgically, because both proteases and

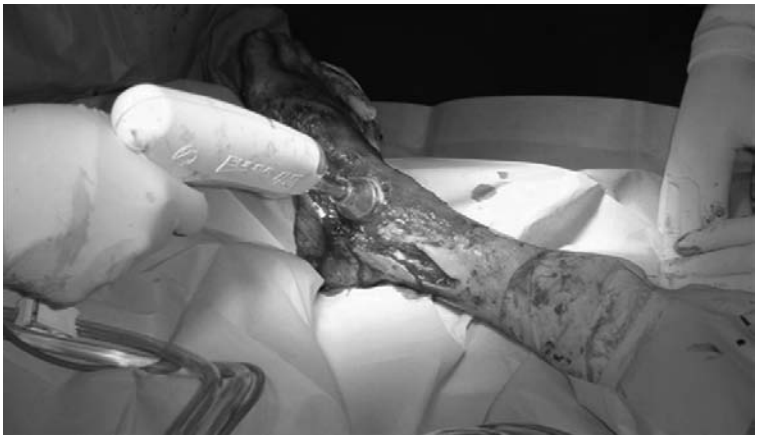

FIGURE 2
High-pressure irrigation.

bacteria accumulate around the edges and underneath the infection, and would cause necrosis to progress and impede tissue repair. If the margin between healthy and necrotic skin is clearly delineated, the skin should be incised along that margin. If, however, there is no clear delineation, a concentric technique should be used to remove the damaged skin, starting from the center of the lesion and gradually growing wider, until healthy, bleeding tissue is reached. The presence of venous thrombi at the edges of the lesion indicates that the microcirculation is completely compromised and is a clear sign of the need for excision of the tissue. The condition of the subcutis needs to be assessed after the removal of the skin. Samples for microbiological assessment should be selected during this phase. We usually use simple gauze dressings soaked in antiseptic after surgical debridement. A new local antiseptic based on superoxidized water has recently proved to be superior to iodopovidone in the treatment of infected ulcers

FIGURE 3
Instruments for surgical debridement.

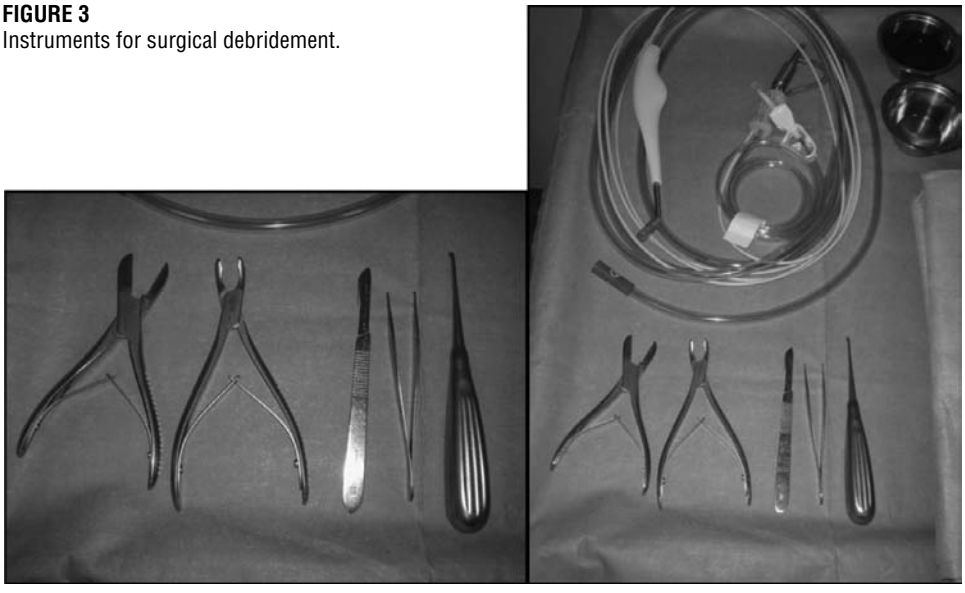

FIGURE 4
Ultrasound apparatus for surgical debridement.

(37). We then use silver-coated dressings and semi-occlusion with polyurethane foam (38,39) or NPWT (40).

Given the physiologic reduction in the vascularization of the subcutis, the degree of bleeding is not always a reliable indicator that the healthy tissue has been reached. Healthy adipose tissue is yellow, whereas the presence of grayish nonelastic tissue is a clear sign of damage. Undermined areas must be removed completely, as they jeopardize the survival of the skin. Small vessels must be coagulated with bipolar forceps to minimize damage to the surrounding tissues. Vessels measuring more than 3 mm in diameter must be ligated with non-reabsorbable suture thread. A moist dressing is often necessary to prevent the adipose tissue from drying out.

The fascia is whitish, shiny, and hard. If it appears to be in a good condition, it must be left intact if possible. If it is infected, it appears soft and frayed. If it is necrotic, it should be removed until healthy tissue is reached. The same treatment should be applied to muscle.

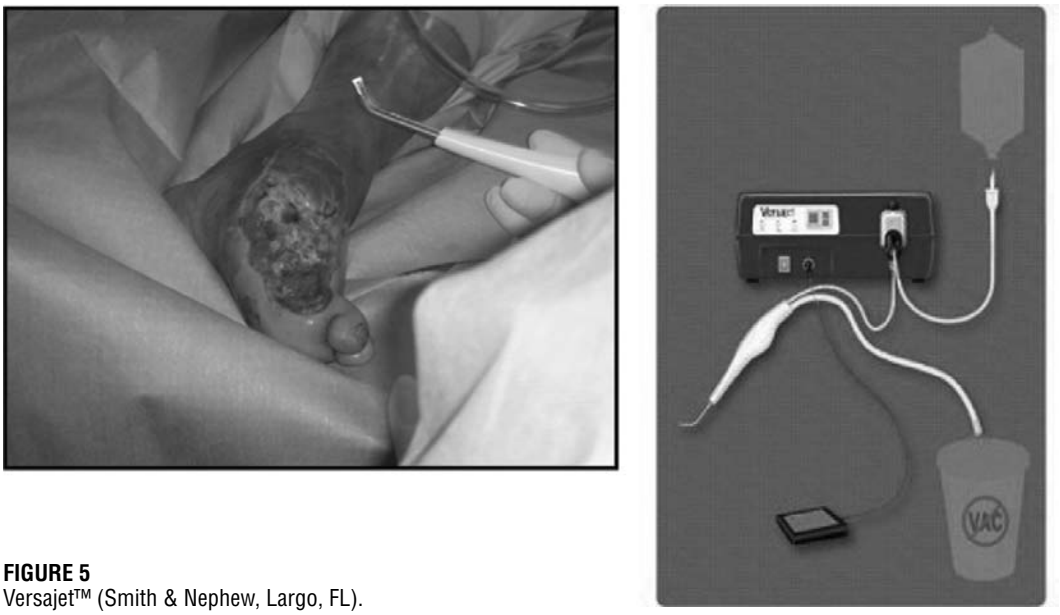

FIGURE 5
Versajet™ (Smith & Nephew, Largo, FL).

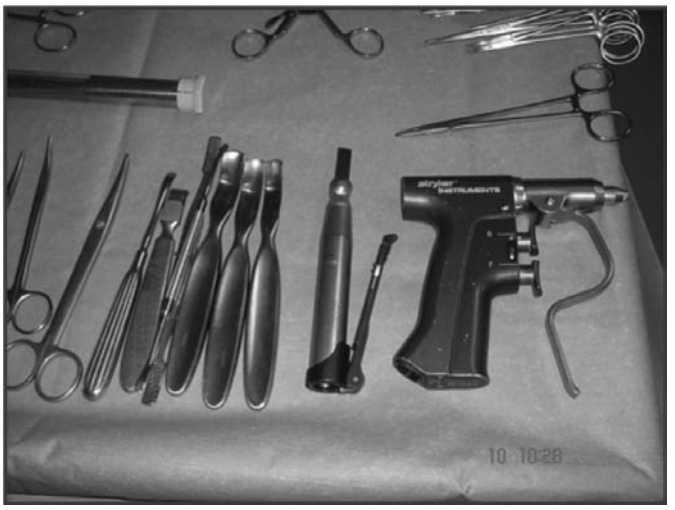

FIGURE 6
Oscillating saw.

The destruction of tendons is a "vexed question" as it involves a functional deficit. If they appear microscopically to be unaffected by the infection, they should be left intact by maintaining the paratenon which supplies their nutrition. It is then necessary to use moist dressings to prevent drying out and subsequent bacterial superinfection.

The Achilles tendon is often exposed in posterior lesions of the distal part of the lower leg. It has an abundant blood supply from the posterior tibial artery and the interosseous artery. If partial necrosis of the tendon has occurred, debridement must be performed until healthy tissue is reached. Subsequently, a moist dressing should be applied, or NPWT implemented after protection of the decontaminated tissues with greasy gauze. Once granulation tissue covering the tendon has been obtained, free skin-graft techniques can be employed.

A further key point is the decontamination of the foci of osteomyelitis. Nonbleeding malacic bone should be removed. The instruments needed for this procedure are rongeurs (Luer rongeurs, Liston forceps) and an oscillating saw (Fig. 6).

The best method of decontaminating the infected parts of small bones (phalanges, metatarsal bones) is to resect thin bone sections until healthy, bleeding bone is reached. The tissue should also be irrigated abundantly with physiologic saline to limit the high temperature caused by using the oscillating saw.

When dealing with osteomyelitis, the problems related to surgical treatment with the removal of bone segments should not be given too much consideration: all the infected bone should consequently be removed. The possible correction of the biomechanic imbalance resulting from the radical treatment of osteomyelitis should be addressed after the infection, and any ischemia should controlled. Microbiologic cultures of both clinically infected and clinically healthy bone remain essential to determine the limit of the infection. Surgical removal of the bone reduces the duration of treatment with antibiotics and reduces the risk of side effects (pseudomembranous colitis, appearance of resistant micro-organisms, and allergic reactions). When it is relatively certain that the focus of osteomyelitis has been eradicated, it is sufficient to continue antibiotic treatment for three weeks after surgery.

In cases where the deep compartments of the midfoot are involved (compartment syndrome), any involvement of the proximal structures around the ankle should be controlled (subastragalar and tibiotarsal joint).

The pathways along which the infection progresses, which can generally be identified in the flexor and extensor compartment and the fibular compartment, must also be controlled. A small incision above the compartment to be investigated is sufficient, with subsequent probing to the

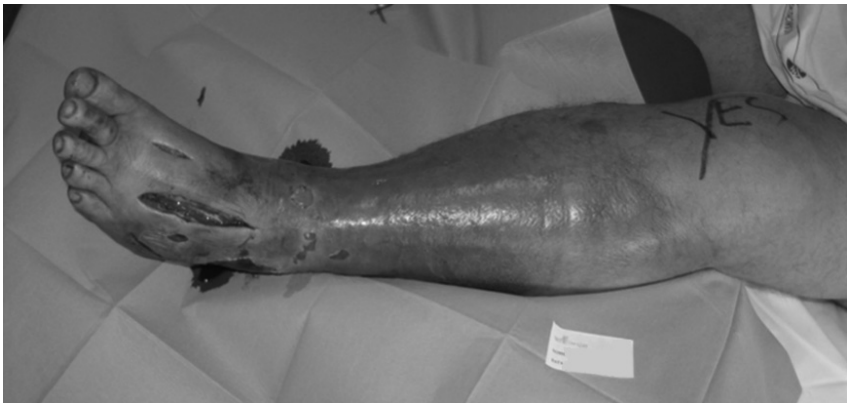

FIGURE 7
Necrotizing fasciitis.

bone of the deep spaces and gentle squeezing to detect any exudate or pus. Once the location of the infection has been identified, the incision must be extended beyond the most proximal location, with the removal of the paratenon and necrotic tendon. The proximal investigation must be stopped only when macroscopically healthy tissue is reached. In some cases, it is necessary to continue beyond the foot, and beyond the ankle, reaching the deep compartments of the lower leg, as far as the popliteal fossa. In the majority of cases, this can be ascribed to a picture of necrotizing fasciitis (Fig. 7). These procedures should often be repeated after 24–48 hours. This treatment is the only way of saving the limb.

DEBRIDEMENT OF NEUROPATHIC ULCERS

Neuropathic patients develop ulcers owing to pathological plantar pressure. A pathologic excess load is perceived immediately in a foot with normal sensitivity; in a neuropathic patient, however, repeated trauma from surrounding objects (inappropriate footwear, blunt objects, surfaces) is not perceived. The ulcer thus develops in relation to the lack of perception of the repeated trauma associated with walking. The clinical characteristics of neuropathic ulcer are therefore as follows: development over an area subject to an excess load, presence of a callus around the ulcer, and absence of pain. As the ulcer causes pressure, a plantar neuropathic ulcer tends to be undermined with small openings toward the skin surface concealing the actual involvement of deeper tissues (Fig. 8).

If the neuropathy is not accompanied by ischemia, the ulcer is more likely to become chronic. The risk of the ulcer worsening in terms of both the progressive destruction of the deep tissues and the progression of the infection is also related to the concomitant presence of ischemia. An ideal assessment should therefore always rule out arterial occlusive disease. The points to be addressed during the clinical examination of a neuropathic ulcer are listed in Table 2.

Appropriate debridement should follow an assessment of the ulcer. The debridement should remove the whole of the callus surrounding the lesion and all nonvital tissue until healthy, bleeding edges are revealed. It is then necessary to perform a careful probing to bone maneuver to establish whether deep structures, such as tendons, joint capsules or bone, are involved (Fig. 9).

Although there is as yet no agreement on the classification system for ulcers, the University of Texas classification, which takes into account the concomitant presence of infection and/or ischemia in addition to the size and depth of the ulcer itself, seems to be the most suitable for staging ulcers and determining their prognosis (41).

After a clinical assessment of the ulcer, careful debridement and the exclusion of the involvement of deep tissues and bones, the most appropriate treatment should be chosen for relieving pressure on the ulcer.

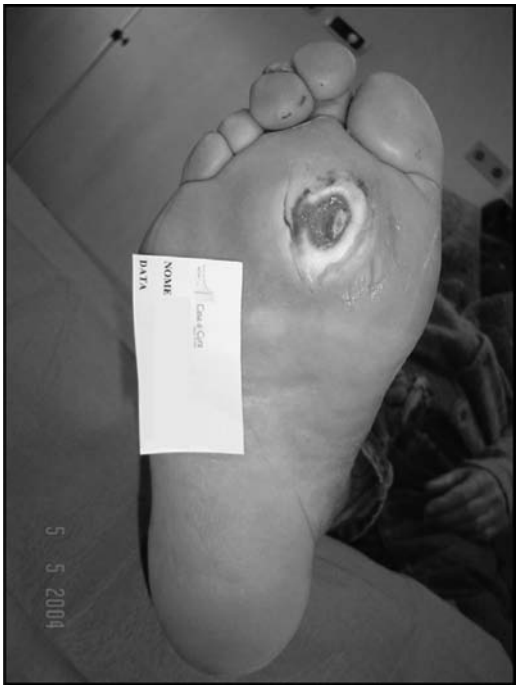

FIGURE 8
Plantar neuropathic ulcer.

The literature clearly shows that relieving pressure is the cornerstone of the treatment of a plantar neuropathic ulcer. There are many techniques for relieving pressure on an uncomplicated ulcer, and they include the total contact cast, therapeutic footwear, and walkers (42). The use of a nonremovable glass fiber cast has recently been shown by Caravaggi et al. to be associated with faster healing of plantar neuropathic ulcers than therapeutic footwear (43).

Conservative treatment is clearly the first step in the treatment of uncomplicated neuropathic ulcer. There are, however, clinical conditions that render ulcers resistant to conservative treatment, and in which surgery becomes the treatment of choice. Aggressive treatment of the local problem is, in fact, very often considered to have the greatest chance of saving the limb. Armstrong and Frykberg provided a classification for surgery of the diabetic foot which gives the types of treatment a score related to the risk of amputation (44).

The surgical indications for the treatment of plantar neuropathic ulcer are essentially as follows: (*i*) the concomitant presence of an underlying focus of osteomyelitis, (*ii*) the presence of a plantar exostosis which means that there is a high risk of the recurrence of the lesion, and (*iii*) an ulcer that has become chronic and is resistant to conservative treatment.

TABLE 2
Clinical Examination of a Patient with a Plantar Neuropathic Ulcer

1. The patient's medical history
2. When it appeared
3. Previous treatments
4. Assessment of the location, extent, and depth of the ulcer
5. Assessment of the edges for exposure of undermining
6. Assessment of the bed of the ulcer and any exudation
7. Assessment of the perilesional skin
8. Assessment of any arterial occlusive disease

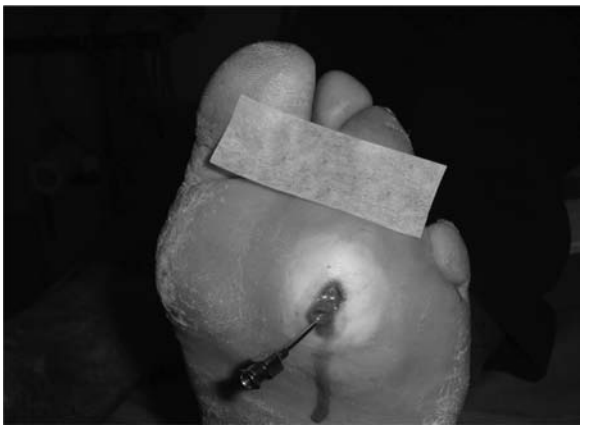

FIGURE 9
Manovra probe to bone.

Surgery can achieve two important results in these situations: first, healing the ulcer in a significantly shorter time, and second, surgically correcting the pathologic excess load by the anatomic correction of the exostosis (45). Piaggesi et al. demonstrated that the surgical treatment of the lesion (ulcerectomy), accompanied by the modification of the pathologic excess load (exostectomy), in a population of diabetic patients with a plantar neuropathic ulcer obtained significantly shorter healing times and a lower percentage of recurrent ulcers than conservative treatment (46).

A prerequisite for ulcerectomy is a vascular assessment and a microbiologic assessment for targeted antibiotic treatment. It is also necessary to establish whether the bone is involved (e.g., a metatarsal head) so as to plan the type of surgery to be performed on the ulcer.

Surgery (Fig. 10) is usually performed under locoregional anesthesia. We usually carry out excision of the whole thickness of the ulcer from the outset; if there is clinical and/or radiologic evidence of bone involvement, the bone in question is exposed and decontaminated. The commonest locations in the front part of the foot are the metatarsal heads, the tips of the toes, and the interphalangeal joints; in the middle part of the foot, the cuboid or medial cuneiform bone; and in the back part of the foot, the calcaneal tubercle.

The commonest location in the front part of the foot, involving a metatarsal head, should be exposed after excision of nonvital tissues, with resection as far as the passage between the diaphysis and epiphysis with the oscillating saw and subsequent removal of the distal segment. The base of the phalanx is removed only if there is clinical or radiologic evidence of osteomyelitis. The bone segment removed is usually sent for microbiologic and histologic testing. Before suturing, we use high-pressure irrigation with physiologic saline and antiseptic; we then perform careful hemostasis to avoid the risk of hematoma formation, and we introduce a suction drain or gauze which we remove after 24 hours. We suture the surgical access with a nylon or prolene 3-0 or 4-0 monofilament. We usually avoid suturing the subcutis with reabsorbable sutures owing to the relative risk of ischemia and superinfection.

This procedure is undoubtedly the simplest to perform, and we reserve more complex covering techniques, such as rotation or advancement flaps for larger ulcers. The involvement of several metatarsal heads or the presence of a very large plantar ulcer may require, depending on the case, the use of more complex surgical techniques, such as panmetatarsal realignment (Fig. 11), or more aggressive techniques, such as minor amputation.

The surgical treatment of an ulcer nevertheless involves a significant risk of recurrence of a transfer ulcer in the other metatarsal heads. In this case, the recurrent ulcer will be treated differently depending on whether the adjacent metatarsal head is already affected by osteomyelitis. If there is no osteomyelitis, conservative treatment can be carried out after debridement with a half-leg cast, and the balance can be restored by osteotomy to raise and/or lengthen the Achilles

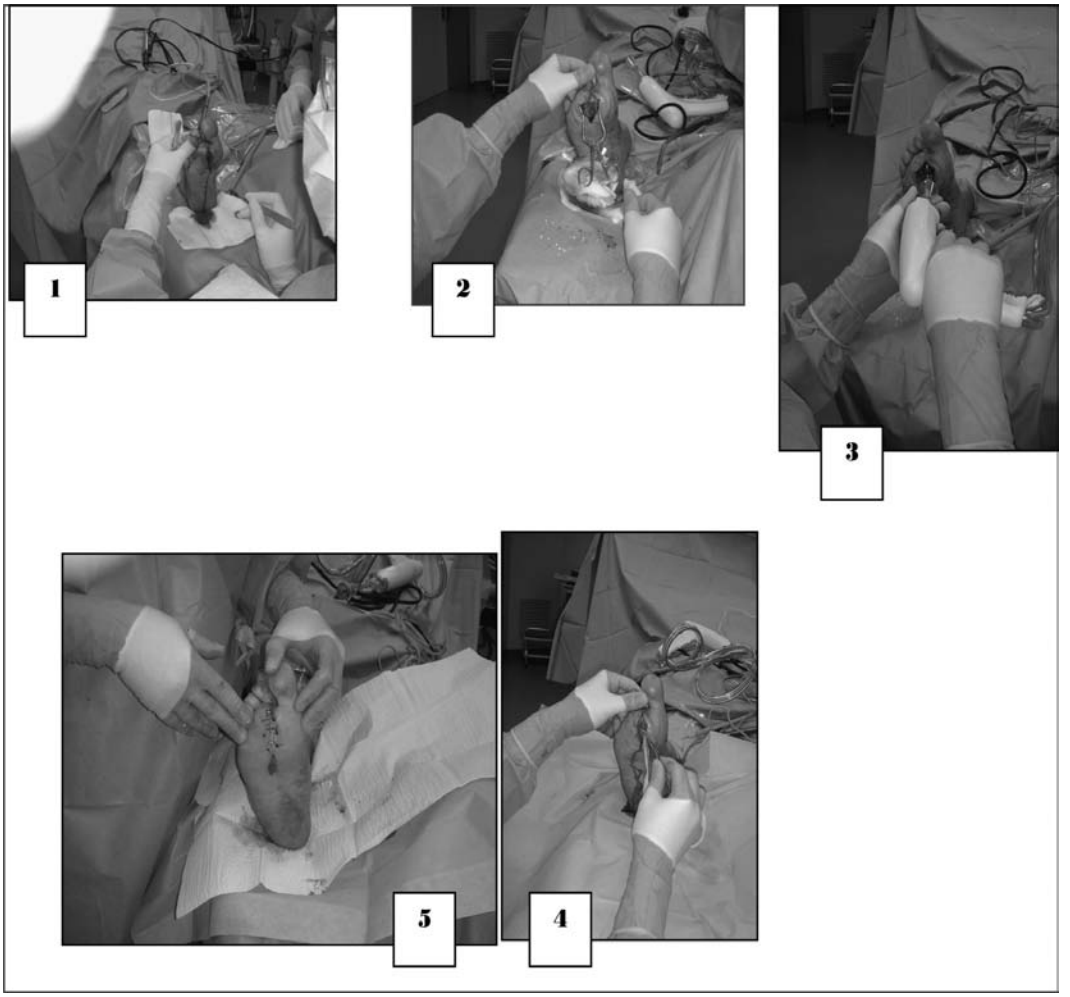

FIGURE 10
Surgical treatment of a plantar neuropathic ulcer—debridement.

tendon, which can be considered soon after. If osteomyelitis is present, the treatment may comprise a panmetatarsal realignment or a transmetatarsal amputation.

After the lesions have healed, the patient must be sent for a follow up, involving treatment with an orthotic device with a moulded plantar base and footwear with a rigid sole.

Debridement of the Infected Foot

Infection of the soft tissues, the progressive involvement of the deep tissues, and the development of foci of osteomyelitis (Fig. 12) are the factors that separate conservative treatment from more aggressive destructive surgery. Osteomyelitis can, in turn, be present at the initial examination, indicating that the infection has become stable and chronic. The location of bone infection in the neuropathic foot usually involves the metatarsal heads on a plantar level, the proximal interphalangeal joints of the toes on a dorsal level, and the tips of the toes on a distal level (47). Clinically, the overlying ulcers are torpid, chronic, and nonpainful, with hyperkeratotic edges; chronic exudation from the bed should usually be evident, and probing to bone should be positive.

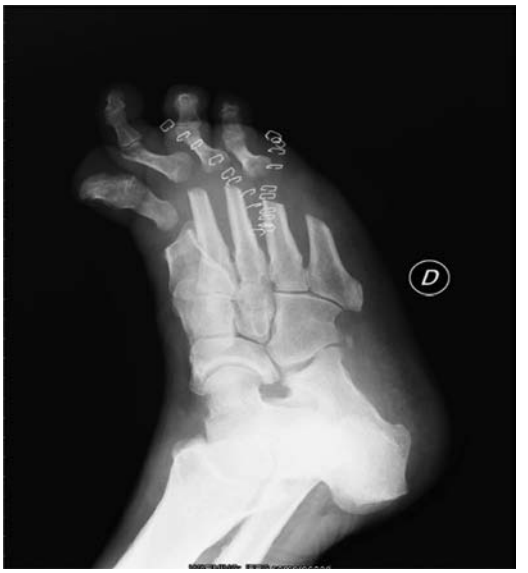

FIGURE 11
Panmetatarsal realignment below.

This requires careful therapeutic planning, which, after the exclusion of an ischemic component, should rely on the microbiologic result from deep samples obtained by scraping or biopsy, broad-spectrum antibiotic cover, and surgical treatment of the focus of infection (ulcerectomy + sequestrectomy) (48). This local treatment is clearly necessary if medium-sized bones (metatarsals or bones of the middle or back part of the foot) are involved. The eradication of distal foci affecting the toes has been reported using prolonged antibiotic treatment. Table 3 shows the advantages of the surgery of osteomyelitis.

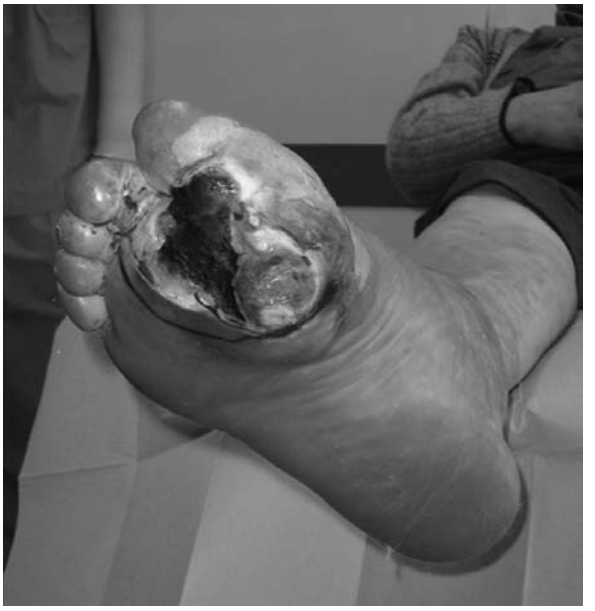

FIGURE 12
Inflamed diabetic foot ulcer.

TABLE 3
Advantages of the Surgery of Osteomyelitis

- Removal of the infected bone fragment
- Decontamination of soft tissues
- Transformation of a chronic lesion to an acute lesion with activation of wound healing processes
- Better microbiological definition of tissue removed
- Shorter antibiotic treatment times
- Better definition and inspection of deep compartments

Infections that do not pose an immediate risk to the limb are defined as non-limb-threatening, and are generally characterized by the absence of the signs of systemic toxicity; generally, perilesional cellulitis >2 cm is not present, and there are no deep abscesses, osteomyelitis, or gangrene present. On the other hand, infections defined as limb-threatening are characterized by extensive cellulitis, deep abscesses, osteomyelitis, or gangrene. Ischemia may mean that even superficial infections are included in this risk group (47). Lipsky produced a more specific classification of infection which is shown in Table 4 (48).

An infected ulcer may not be associated with significant signs of local or systemic symptoms in diabetic patients. Although the role of antibiotic therapy in the treatment of clinically uninfected ulcer has not yet been entirely clarified, the current evidence does not support the use of antibiotic therapy. In the majority of clinical papers in the literature, antibiotic treatment does not improve the outcome of uninfected ulcers (49). A clinical follow-up is, in any case, necessary, and should comprise the monitoring of local conditions for the early detection of local symptoms and/or signs of infection (47). All ulcers are colonized by potentially pathogenic bacteria; however, the diagnosis of infection is clinically, rather than microbiologically, based. The presence of purulent secretions or of two or more signs of inflammation (cellulitis, raised skin temperature, or edema) should be used to diagnose an infection. Antibiotic treatment should be started promptly in a clinical picture of non-limb-threatening infection. For mild and moderate infections, an oral antibiotic is used. Oral treatment is less costly, simpler to manage, and usually sufficient in this type of patient. Parenteral therapy may be used only in selected cases (intestinal absorption problems, gastrointestinal allergies, isolation of bacteria resistant to oral antibiotics). The chosen antibiotic should reach good serum levels and provide cover against gram-positive micro-organisms.

TABLE 4
Classification of the Clinical Characteristics of Infection of the Diabetic Foot

Clinical signs of infection	Severity
Ulcer with no exudation or other signs of inflammation	Not infected
Clinical signs of inflammation (exudation or erythema, pain, tension, increased skin temperature, edema, not extending more than 2 cm around the lesion and infection limited to the superficial tissues. No local complications or systemic signs.	Mild
Infection in a patient without systemic complications and who is metabolically stable in the presence of one or more of these signs: cellulitis extending more than 2 cm from the edge of the lesion; lymphangitis; subfascial progression; abscesses located in deep tissues; gangrene; involvement of muscles, tendons, joints, or bone.	Moderate
Infection in patients with systemic signs of toxicity or who are metabolically unstable (fever, malaise, tachycardia, hypotension, confusion, vomiting, leucocytosis, acidosis, hyperglycemia).	Severe

TABLE 5
Characteristics of Surgical Debridement

- Removal of necrotic tissue and part of the bacterial load
- Drainage of accumulated exudate
- Assessment of the extent and depth of the lesion
- Use of deep-tissue samples for more reliable microbiologic results
- Conversion of a chronic lesion into an acute lesion
- Platelet activation for control of bleeding with release of growth factors stimulating the proliferative phase of wound healing

The best method of cleansing the ulcer is to use high-pressure irrigation with at least 2l of saline solution. Microbiologic samples should be collected in the operating theater. It is important to identify a possible infection, as it correlates significantly with the risk of developing osteomyelitis (50). Antiseptic dressings must be maintained after debridement, and changed every 12–24 hours. The antibiotic cover must be broad-spectrum, pending microbiologic cultures (51).

An acute foot is, in the majority of cases, characterized by infections that require assessment and immediate treatment, usually in hospital. The infection may be related to progressive destruction of the soft tissues, involvement of bone, the need for surgery and, in some cases, amputation (49,52). When dealing with a diabetic foot with an acute lesion, it should be kept in mind that immediate treatment is absolutely essential in many cases. From this point of view, it is often necessary to carry out surgical debridement urgently, without considering factors limiting the metabolic compensation, or the patient's nutritional status or vascular condition (53–55). Surgical debridement in this specific context has advantages over other forms of debridement (Table 5). Less severe pictures can certainly be treated on an outpatient basis or at the patient's home, without the need for anesthesiologic support. In more extensive, deep infections, particularly where bone is involved, surgery in an operating theater is required to obtain adequate debridement and drainage. Special attention should be paid to relieving pressure during the postoperative period.

A decision on surgery, antibiotics, and support treatment should be made on the basis of a careful general and local examination of the patient (Table 6) (48).

In addition to being a negative prognostic factor, infection alone can also trigger ischemia via inflammatory and thrombotic mechanisms involving the terminal arterioles in the toes (increase in oxygen consumption, edema, angiitis, and septic thrombosis). Local treatment is therefore indicated for cleansing the tissues, reducing the mass of infection, and improving local circulation.

From a clinical point of view, the acute phase of a diabetic foot infection can be divided into four main phases: cellulitis, abscess, necrotizing fasciitis, and gangrene. Whereas cellulitis is usually treated with antibiotics, the other three conditions require surgery.

TABLE 6
Recommendations for the Examination of an Infected Foot Ulcer

- Describe the ulcer and check for the production of any pus and its location
- Check for the various clinical signs of inflammation
- Determine whether infection is present, seeking to identify its causes
- Examine soft tissues, demonstrating crepitation, and any abscess formation or fistulization
- Determine whether probing to bone is positive for any lesion or fistula
- Measure the lesion (diameters and depths)
- Palpation of arterial pulses; use Doppler if necessary
- Peripheral neurological assessment
- Lavage and debridement of the lesion; remove any foreign body and dry
- Microbiologic cultures (with curettage and aspiration)
- Request standard X-rays

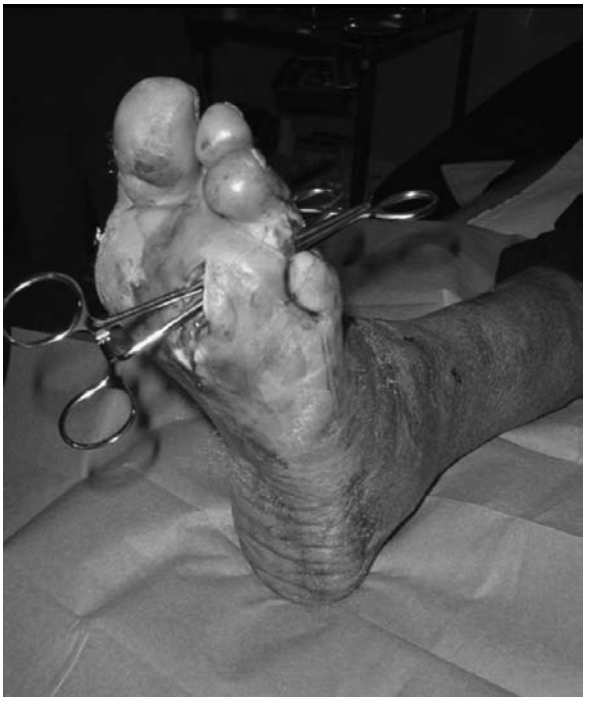

FIGURE 13
Scissors in foot.

Abscess

If purulent drainage from an ulcer is observed; if, during probing to bone, the deepest compartments are reached; and if fluctuation of the subcutis or deep soft tissues is noted, the formation of an abscess should be suspected. In this event, the suspected area should be incised, gently detaching the affected tissues to allow evacuation of the purulent accumulation (Fig. 13). Combined incisions via the plantar and dorsal routes are sometimes necessary to reach deep locations. Pressing on a proximal point as distant as the site of the incision may help to evacuate the pus completely. All necrotic and infected tissue must be removed, until healthy, bleeding tissue is reached.

Poorly vascularized tissue, such as tendons, joint capsules, and joint cartilage, should be decontaminated with great care. Once the liquefied material has been removed, the area needs to be irrigated with sterile physiologic saline. Table 4 gives further instructions on how to proceed with surgical debridement. From a microbiologic point of view, many micro-organisms may contaminate the ulcer surface and only deep samples can thus isolate species reliably to guide antibiotic therapy. When dealing with an infection with abscess formation, the following should be kept in mind:

- Assessment of the location and extent of the abscess to determine the best way of obtaining reliable cultures
- Assessment of the surgical access to obtain sufficient drainage and the debridement of infected tissues
- Need to determine the possible involvement of the bones

When assessing vascular condition, all necrotic tissue, including the affected bone segments, should be removed. Sometimes, minor amputations are necessary (toe, metatarsal) to reach healthy tissue. Drains should be positioned for daily irrigation with antiseptic solutions or antibiotic. Follow-up of the lesion would confirm the type of surgical approach, which could be applied to resolve the problem.

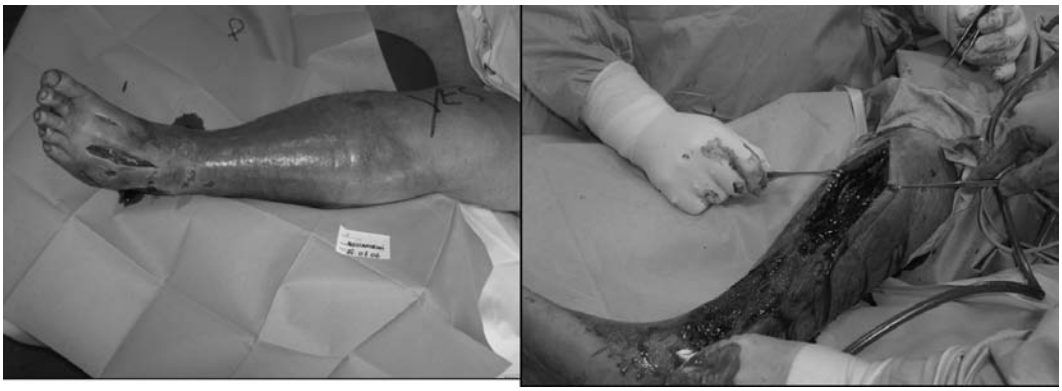

FIGURE 14

Necrotizing Fasciitis

Necrotizing fasciitis is life threatening. It may appear spontaneously, particularly in patients with diabetes and/or occlusive vascular disease. The commonest anerobic pathogen isolated is Peptostreptococcus, but Clostridium and Bacteroides may also be involved, as may *Staphylococcus aureus* and *Streptococcus pyogenes*. Infection starts rapidly within 24–72 hours of surgery or minor trauma, spreading extensively through the deep layers of the fascia, with necrosis of the subcutaneous tissue (Fig. 14). The extensive detachment and destruction of the more superficial tissues can be demonstrated below the cutis. The fascia appears gray and necrotic, but the muscles are not involved. The treatment of choice is extensive surgical debridement of the affected tissues, until healthy, bleeding tissue is reached. It is important to reach healthy tissue close to the limit of tissue destruction. Hyperbaric oxygen therapy might be used. It is often necessary to repeat debridement after 24/48 hours. Broad-spectrum antibiotic therapy should be started as soon as possible without waiting for the results of microbiologic cultures.

Gangrene

Gangrene is a fairly frequent complication of both neuropathic ulcer and neuroischemic ulcer (Fig. 15). It often involves one or more toes in neuropathic foot without affecting the whole foot. If the arteries of the toes are involved, gangrene would occur very rapidly (septic vasculitis).

If an infection has spread and is progressing, involving the vessels of the middle part of the foot, the situation may worsen with the appearance of gangrene and the involvement of the whole foot, when major amputation is indicated. An ulcer in a diabetic patient with occlusive vascular disease will generally develop far more rapidly and destructively into gangrene. The surgical approach in both situations comprises debridement, which should be as extensive as possible to remove all nonvital tissue. In the event of an occlusive vascular process, we recommend the

TABLE 7
Drainage of an Abscess

- As an inpatient for a mild or moderate infection
- Strict bed rest and immobilization
- Abscesses of the plantar fascia often involve multiple layers of sedimentation which must be debrided
- Length-wise incisions are preferable, particularly in ischemic patients
- Fistulas must be opened sufficiently
- Always probe tendon sheaths as proximally as possible

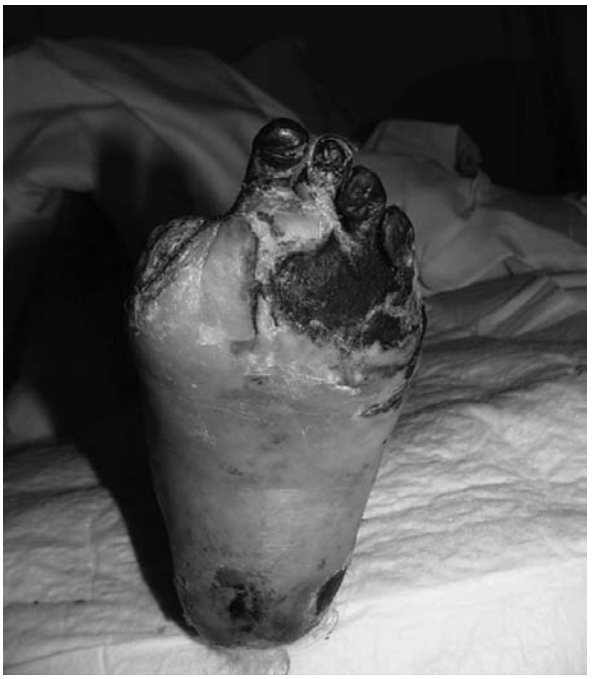

FIGURE 15

surgical removal of the necrotic tissue (avoiding any permanent surgical approach), followed by angiography to assess the feasibility of revascularization surgery. Once the acute phase has been resolved, the most practicable and acceptable surgical approach should be chosen as per the patient's clinical condition.

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Surgery

about the book . . .

Wound bed preparation can be defined as the process of removing dead tissue, debris, and contaminants from an injured site to facilitate healing and provide a more expedient means to patient recovery. This reference explores the critical role of surgery in wound bed preparation and management and provides a sound knowledge of wound mechanisms, physiology, and metabolic control. Founded on the expertise of internationally-recognized authorities, this source illustrates the many techniques utilized by surgeons to design optimal healing environments, maximize the efficacy of existing treatment modalities, and extract bacteria from a variety of wound situations resulting from burns, trauma, and disease.

Describing the factors that influence surgical wound healing and the risk of surgical site infection, this source provides in-depth examinations of a variety of clinical situations with numerous full-color photographs...contains extensive coverage of techniques for the debridement of leg and foot ulcers, infected orthopedic prostheses, surgical wounds, and pressure ulcers...discusses new approaches for treating difficult-to-heal wounds...helps clinicians correct chronic wounds more quickly by treating the cause of the patient's non-healing wound and applying evidence-based, medical and surgical techniques...provides an in-depth history of surgical wound care and proposes a new classification system for debrievement of both chronic and acute wounds...and outlines how to obtain a surgically clean wound in preparation for surgical closure.

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