
Maintenance Debridement in the Treatment of Difficult-to-Heal Chronic Wounds

Recommendations of an Expert Panel

This activity is supported by an educational grant from Healthpoint.

HEALTHPOINT[®]
A DFB COMPANY

This activity is sponsored by the North American Center for Continuing Medical Education. **NACME**

Continuing Education

Completion Time:

Estimated time to complete this complimentary activity is 1 hour.

Target Audience:

Physicians and nurses who treat patients with wounds

Learning Objectives:

Upon completion of this educational activity, participants should be able to:

- Describe the range of clinical and biological abnormalities in the wound bed that can inhibit wound healing
- Define initial and maintenance debridement and describe the primary goals of each
- Discuss the scientific and clinical rationale for initial and maintenance debridement
- Name two tools that can be used to assess the adequacy of debridement and wound bed preparation in general
- Describe how to implement debridement within the context of an effective wound care strategy
- List several different methods of debridement.

Method of Participation:

Participants must read the journal supplement then take, submit, and pass the post-test by June 30, 2009. Participants must completely fill out the answer/evaluation form and mail the answer/evaluation form to NACCME at 83 General Warren Blvd., Suite 100, Malvern, PA 19355 or fax it to 610-560-0502. Within 60 days, the participant will be advised of passing or failing the exam. A score of or above 70% will comprise a passing grade. A certificate will be awarded to participants who successfully complete the exam.

Accreditation:

MD/DO: This activity is sponsored by the North American Center for Continuing Medical Education (NACCME). NACCME is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians. NACCME designates this educational activity for a maximum of *1 AMA PRA Category 1 Credit™*. Physicians should only claim credit commensurate with the extent of their participation in the activity. This activity has been planned and produced in accordance with the ACCME Essential Areas and Policies.

Nurses: ANCC: NACCME is an approved provider of continuing nursing education by the Pennsylvania State Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation. This continuing nursing education activity was approved by the PA State Nurses Association for 1 contact hour (Provider Number 110-3-E-06). Provider approved by the California Board of Registered Nursing, Provider Number 13255, for 1 contact hour.

Off-Label/Unapproved Usage Disclosure:

This educational activity contains discussion of published and/or investigational uses of agents that are not indicated by the FDA.

NACCME does not recommend the use of any agent outside of the labeled indications. Please refer to the official prescribing information for each product for discussion of approved indications, contraindications, and warnings.

Faculty Disclosures:

All those with control over the content of continuing education programs sponsored by NACCME are expected to disclose whether they do or do not have any real or apparent conflict(s) of interest or other relationships related to the content of their presentation(s). It is not assumed that these relationships will have an adverse impact on presentations; they are simply noted here to fully inform participants.

Planner and Faculty: Dr. Falanga has disclosed that he is a consultant to Organogenesis.

Faculty: Dr. Brem has disclosed that he is a consultant to Organogenesis. Dr. Ennis has disclosed that he is a consultant to Celleration and Accelecare Wound Centers. Dr. Ayello has disclosed that she is a consultant to 3M Health Care, Smith & Nephew, Hill-Rom, KCI, IVIVI, Sage, Mölnlycke, Coloplast, and Ross. She is a member of the speakers' bureau for Smith & Nephew, KCI, Ross, and Hill-Rom. She has received other financial or material support from Gaymar, Healthpoint, NJHA (3M Health Care, Sage, and Healthpoint), LWW, Hollister, and WUWHS. Dr. Gould has disclosed that she has received grant/research support and is a scientific advisor to Diversified Clinical Services. She is a member of the speakers' bureau for KCI. Dr. Wolcott has disclosed that he has received grant/research support from NIH. He is a member of the speakers' bureau for Cubist and Pfizer.

Medical Writer: Amy Lindsay, PhD, has disclosed that she has no significant financial relationships.

Reviewer: Dr. Siegel has disclosed that he has no significant financial relationships.

Commercial Support: This activity is supported by an educational grant from Healthpoint.

Conflict of Interest Resolution/Content Validation:

In compliance with ACCME Standards for Commercial Support and NACCME's policy and procedure for resolving conflicts of interest, this continuing medical education activity was reviewed by Dr. Siegel in June 2008 for clinical content validity and to insure that the activity's materials are fair, balanced, and free of bias toward the commercial supporters of the activity, that activity materials represent a standard of practice within the profession in the United States, and that any studies cited in the materials upon which recommendations are made are scientifically objective and conform to research principles generally accepted by the scientific community.

Sponsor: This activity is sponsored by The North American Center for Continuing Medical Education.

Release Date:
June 30, 2008

Expiration Date:
June 30, 2009

Maintenance Debridement in the Treatment of Difficult-to-Heal Chronic Wounds

Recommendations of an Expert Panel

Vincent Falanga, MD;^{1,2} Harold Brem, MD;³ William J. Ennis, DO;⁴ Randall Wolcott, MD;⁵ Lisa J. Gould, MD, PhD;⁶ Elizabeth A. Ayello, PhD, RN⁷

From ¹Boston University, Departments of Dermatology and Biochemistry, Boston, MA; ²NIH Center of Biomedical Research Excellence, Roger Williams Medical Center, Providence, RI; ³Division of Wound Healing & Regenerative Medicine, Department of Surgery, New York University School of Medicine, New York, NY; ⁴University of Illinois, Chicago, IL; ⁵Southwest Regional Wound Care Center, Lubbock, TX; ⁶James A. Haley Veterans' Hospital, Department of Surgery, Tampa, FL; ⁷Excelsior College School of Nursing, Albany, NY

Abstract

Introduction: Maintenance debridement has been proposed as a therapeutic intervention to address the problem of chronic wounds characterized by an adequate wound bed but absent or slow healing. A panel of experts convened to address the rationale and method of maintenance debridement.

Purpose: The goals of the panel were to summarize the scientific rationale for maintenance debridement, discuss the biochemical and cellular abnormalities in the wound bed, and provide a working algorithm for how maintenance debridement should be used.

Methods: A multidisciplinary panel of wound healing and wound care experts comprising the fields of nursing, dermatology, internal medicine, and surgery was assembled to address maintenance debridement from different points of view and offer a unified approach.

Findings: The chronic wound contains a number of microbial, biochemical, and cellular features and abnormalities that prevent or slow its progression to healing despite a seemingly adequate wound bed. Under these circumstances, maintenance debridement is proposed as a way to remove tissues that are colonized with an excessive bacterial burden and diminish what can be described as a biochemical and cellular burden that impairs healing. A working clinical algorithm is proposed.

Conclusion: Maintenance debridement is a proactive way to "jump-start" the wound and keep it in a healing mode, even when traditional debridement may not appear necessary because of a seemingly "healthy" wound bed.

Introduction

Difficult-to-heal and chronic wounds, such as venous, diabetic, pressure, and arterial insufficiency ulcers, affect millions of people in the United States. Several reviews of incidence/prevalence studies have been published. Venous ulcers are estimated to affect approximately 2.5 million people in the United States.^{1,2} Approximately 6% (0.882 to 1.2 million) of the 14.7 to 20 million people with diabetes in the United States are at risk of developing diabetic neuropathic ulcers over any given 3-year period.^{3,4} Pressure ulcers afflict an additional 1.3 to 3 million people,⁵ including an estimated 10%–18% of those in acute care and up to 28% of those in extended care facilities.⁶ The total direct annual cost incurred in the treatment of these wounds is estimated to be in the tens of billions of dollars.^{7–9}

Recent estimates suggest that complete wound closure is achieved in as low as 25%–50% of chronic wounds, specifically venous and diabetic ulcers, following up to 20 weeks of treatment.^{10–12} The delayed healing of these wounds has a significant negative

Address correspondence to: Vincent Falanga, MD, Roger Williams Medical Center, Dept. of Dermatology, 50 Maude Street, Providence, RI 02908; E-mail: vfallanga@bu.edu

impact on quality of life and is associated with the risk of serious and costly complications, such as recurring infections and limb amputation.^{2,13-15} These treatment failures are devastating to patients and add many more billions of dollars to the overall economic burden of difficult-to-heal and chronic wounds (approximately \$11 billion for amputations alone).^{8,13}

To promote greater uniformity of care and improve treatment outcomes, several professional societies and agencies have published evidence-based documents that address the care of chronic wounds. Most recently, these include the series of guidelines on the best care of chronic wounds published by the Wound Healing Society (WHS),^{3,5,16,17} the Centers for Medicare & Medicaid Services Expert Advisory Panel on the Usual Care of Chronic Wounds,¹⁸ and the Agency for Healthcare Research and Quality technology assessment on usual care in the management of chronic wounds.¹⁹ Other important guidelines, including those from the Wound, Ostomy, and Continence Nurses Society (WOCN), have been reviewed as well.¹⁹ These guidelines provide a helpful foundation for the development of effective treatment plans and discuss the importance of effective debridement in achieving the goals of therapy. Admittedly, guidelines are evidence-based only up to a point and are by necessity works in progress due to changes that occur in the understanding of the pathogenesis of disease processes and the role of therapeutic agents. Moreover, clinicians can benefit from additional guidance regarding the practical implementation of specific guideline recommendations within a comprehensive treatment strategy. The need for clearer recommendations for practical implementation of debridement

procedures has become more pressing in light of recent concerns that greater attention must be paid to the performance and documentation of debridement.²⁰

In November 2007, an expert panel was convened in Boston, Massachusetts, to discuss the principles of maintenance debridement as a critical element in a successful wound care strategy and develop practical recommendations for its implementation. The expert, multidisciplinary panel included clinicians from the following fields: nursing, dermatology, internal medicine, and surgery. The panel members have had extensive experience in the care of difficult-to-heal and chronic wounds and are actively involved in wound care research and/or education. The focus was on maintenance debridement because of a growing body of evidence that demonstrates the deleterious effects of an unchecked necrotic, cellular, and bacterial burden on the ability of wounds to heal.²¹⁻³¹ Moreover, a direct relationship has been demonstrated between healing rates and the efficiency and frequency of debridement.³²⁻³⁵ The panel members reviewed the available guidelines that have been published most recently^{3,16,17} and analyzed the scientific evidence for bacterial, biochemical, and cellular abnormalities in chronic wounds.²²⁻³¹ All authors of this manuscript were panel members.

MAINTENANCE DEBRIDEMENT IN THE CONTEXT OF WOUND BED PREPARATION: GLOBAL VIEW

Truly active and effective treatments for chronic wounds have emerged only recently, within the last 10 years. First, topically applied recombinant human platelet-derived growth factor (rhPDGF) was

approved for the treatment of diabetic neuropathic foot ulcers. Shortly thereafter, a living bilayered skin construct received regulatory approval for treating venous and diabetic ulcers.^{23,34,36} With the emergence of these sophisticated products, which were the result of many decades of research and technological advances, came the expectation that the clinical outcome for chronic wounds would be dramatically improved. However, panel members believe that three obstacles became apparent shortly after these and other advanced products were approved for clinical use. Admittedly, complete published data on these obstacles and how they affect clinical care in a measurable way are not available. Two rather predictable obstacles were reimbursement and acceptance by clinicians. The reimbursement issues have now been largely dealt with satisfactorily, at least for some tissue skin substitutes. Moreover, in response to educational efforts, clinical evidence, and guidelines, clinicians are now increasingly using these advanced therapeutic agents. A more interesting obstacle to the success of advanced therapies, however, was unexpected. It became clear that even experienced clinicians were not paying close enough attention to the state of the wound bed and what would be required for advanced products to display their optimal efficacy. This had become clear during the rhPDGF trials³⁴ and after the approval of living skin substitutes.²²

As a result, the concept of wound bed preparation was developed to address the overall state of the wound and the steps necessary to optimize both the endogenous process of healing and the effectiveness of advanced therapeutic agents. After its first formulation,²² the concept was expanded

The removal of nonfunctional cells within the wound bed and at the wound edge, removal of necrotic tissue, or elimination of biofilms may promote healing in wounds that are stuck in a nonhealing mode.

in other publications.^{23–25} The concept of wound bed preparation stimulated the field of chronic wound care in important and beneficial ways. It helped clinicians focus on control of wound exudate, removal of necrotic tissue, attention to biological events that occur during impaired healing, and the proper guidelines for conventional treatment. As part of wound bed preparation, another concept with important practical ramifications was born: maintenance debridement.^{22,23,37} This concept was based on early evidence accumulated from clinical trials,²² biochemical and cellular features of chronic wounds,^{25,28,30,31,38} and the determination of increased bacterial burden and biofilm presence.^{16,26,39} In light of these findings, maintenance debridement represents the need to restore the healing mode by a program of debriding the wound bed even when the wound bed may appear clinically adequate.

SCIENTIFIC RATIONALE FOR DEBRIDEMENT

The importance of debridement was observed as a secondary outcome in a randomized, double-blind, placebo-controlled, multicenter trial of the safety and efficacy of topically applied rhPDGF in the treatment of chronic diabetic ulcers.³⁴ In that trial, all ulcers were thoroughly debrided (wound bed

and callus) at the beginning of the study, and the investigators were allowed to perform additional debridements during follow-up when they deemed the procedure appropriate. The study achieved its primary goal by demonstrating that the rate of wound closure was higher among ulcers treated with rhPDGF than those treated with placebo. An important and unexpected finding, however, was that the centers performing the most frequent debridements had the highest wound closure rates in both the rhPDGF and placebo treatment groups. Similarly, the centers that performed the least frequent debridements had the lowest healing rates.³⁴ These findings suggest that debridement can lead to improved clinical outcomes. This conclusion gains considerable support from results showing that frequent debridement of chronic wounds can help reduce amputation rates.⁴⁰

As previously discussed, the need for initial and repeated debridement of a clearly necrotic or nonviable wound bed in diabetic neuropathic foot ulcers was quite evident from the findings of Steed et al.³⁴ Later, as less clinically evident abnormalities present in the wound bed became more apparent, the concept of maintenance debridement evolved.²² The importance of maintenance debridement gains further support from the

recognition that in difficult-to-heal and chronic wounds there are many biochemical, cellular, and bacteriological abnormalities that will interfere with healing and increase the risk of infection if not frequently or continually controlled.^{22–24} Therefore, wounds that clinically show a seemingly adequate wound bed may benefit from debridement if healing has stalled.³⁵

There are now promising tools that can be used to assess the effectiveness of debridement and wound bed preparation. Two such studies show a correlation between the efficacy of debridement and healing rates.^{32,33} The Debridement Performance Index,³² which evaluates the adequacy of debridement, has been shown to directly correlate with the likelihood of wound closure by Week 12. This was demonstrated in a validation study that found that the Debridement Performance Index, which is based on the removal of callus, undermined edges, and necrotic tissues, was an independent predictor of wound closure in diabetic foot ulcers that had been treated with either standard therapy or a bioengineered skin construct.³² More recently, the Wound Bed Score (WBS), which provides a broader and more global assessment of the wound and reflects the adequacy of wound bed preparation in general, has been shown to be a predictor of ultimate wound closure in venous ulcers.^{33,37} The WBS includes assessments of the presence of eschar, fibrotic tissue, and callus — all factors that reflect the adequacy or need for debridement.³⁷ Figure 1 shows how the WBS is determined. The WBS relies entirely on critical parameters that are important in wound closure and can be analyzed at the bedside without the need for laboratory tests. Indeed, the WBS was developed with the goal

that the clinician could assess the wound quickly using purely clinical inspection.^{33,37} While the WBS assesses 8 factors, as shown in Figure 1, three of these (eschar, fibrotic tissue, and callus) are factors that reflect the need for debridement. The authors recognize that a possible discrepancy may be erroneously detected when we recommend reliance on some of the clinical parameters used in scoring the wound (Figure 1) and at the same time state that the appearance of the wound bed is not necessarily indicative of a wound that is going to heal. However, functional parameters incorporated in the WBS in Figure 1, such as exudate and resurfacing epithelium, argue against this possible discrepancy.

The importance of debridement is supported by new insights into the unique barriers to healing that exist in the chronic wound bed. In a recent study, biofilm-embedded bacteria were found in the majority of chronic wounds evaluated (60%) but only a small percentage (6%) of the acute wounds studied.^{26,41} A biofilm is a community of micro-organisms encased in a self-secreted matrix (coating) composed of bacterial extracellular polymeric substances (EPS) and commandeered host factors (fibrinogen), which tightly bind the biofilm to the wound bed.^{23,26} The biofilm gives the individual bacterium protection from biocides, antibiotics, and the host immune system, which makes it difficult to remove those bacteria by any means other than debridement.²⁶ Biofilms increase the opportunity for the transfer of genes encoding antimicrobial resistance between and among individual bacteria, which increases the potential for particularly tenacious and virulent infections.^{26,41} However, as also stated in a recent publication,²⁶ panel members realize that more work is required to fully elucidate the role of

Wound Bed Score (WBS)			
	Scores of 0	Scores of 1	Scores of 2
Black Eschar	0	1	2
Eczema/Dermatitis	0	1	2
Depth	0	1	2
Scarring (fibrosis/callus)	0	1	2
Color of wound bed	0	1	2
Oedema/Swelling	0	1	2
Resurfacing epithelium	0	1	2
Exudate Amount	0	1	2
Add scores for each column →			
TOTAL SCORE (Max=16)	Copyrighted. V.Falanga 2007		

Figure 1. The Wound Bed Score (WBS) accounts for assessments of the wound bed as well as surrounding tissue. Each of the parameters listed in Figure 1 receives a score of 0 to 2. The scores are added to obtain the total score (maximal is 16 = best score), which correlates directly with ultimate wound closure.

biofilms in delaying the healing of chronic wounds.

In addition, studies have shown that the borders of difficult-to-heal and chronic wounds are often populated by abnormal cells.^{23,27,28} These cells have a markedly diminished capacity for normal migration and proliferation, do not respond properly to growth factors, and may be incapable of participating in normal healing.^{22,28,38} These *in-vitro* findings require closer correlation with *in-vivo* results, but some studies have found an association between impaired healing *in vivo* and chronic wound fibroblast abnormalities *in vitro* (reviewed in Falanga²³). There is evidence that chronic wound fibroblasts within the wound bed have developed phenotypic abnormalities in signal transduction pathways required for proper responsiveness to growth

factors. These fibroblast populations are abnormal by a variety of criteria, including large and bizarre morphology, decreased proliferation and lifespan, and decreased responsiveness to certain growth factors, such as PDGF and TGF- β . Decreased receptors for growth factors and signal transduction abnormalities have been found.³⁸ More recently, venous ulcer fibroblasts have shown decreased expression of β ig-H3, a key protein that is normally induced by TGF- β and may play a role in wound healing.²⁸ These *in-vitro* studies have relied on comparison of fibroblasts cultured from chronic wounds with those derived from acute wounds created in the same patient.^{23,28,38} Cells just a few millimeters away from the edge of venous ulcers exhibit normal physiology and morphology and have a greater capacity to participate in normal healing.²⁷

Table 1. Definition of terms and goals of debridement

Initial Debridement: The removal of necrotic, damaged, and/or infected tissue from the wound bed. Removal of periwound callus is included in this definition. The debridement needs to be repeated as long as those clinical parameters persist or recur.

Maintenance Debridement: Ongoing debridement to help maintain the wound in a healing mode. The debridement intervention is dictated not only by clinically evident parameters but also by the need to achieve optimal wound bed preparation. Therefore, maintenance debridement is performed even in the face of a seemingly healthy wound bed if the wound is not showing evidence of closure.

Chronic wounds also have been shown to have elevated levels of matrix metalloproteinases, which have been associated with poor healing.²⁹⁻³¹ Debridement may help by removing abnormal cells and excess matrix metalloproteinases and help in the recruitment of normal cells to the wound.²²

DEFINITION OF TERMS

Generally, terms are defined before one addresses the issues that those terms represent. However, the panel thought it would be more helpful to first discuss the background that led to the evolution and formulation of maintenance debridement.

The distinction between initial (or repeated) debridement and maintenance debridement is not necessarily in the method used but rather in both the temporal sequence and the rationale. Thus, unlike the initial (and sometimes repeated) debridement, maintenance debridement may be required even in the context of an adequate wound bed when the wound shows continued impaired healing.^{22,23}

Traditionally, the term *debridement* has been used to address the removal of necrotic, damaged, or infected tissue.^{3,22} Therefore, debridement may

need to be repeated periodically (typically once or twice a week for chronic wounds) as long as the wound bed displays the features of necrotic, damaged, and/or infected tissue. This is what was done in previous studies in which debridement was found to correlate with faster wound closure. In those studies, the presence of a newly formed callus around the neuropathic diabetic foot ulcer could be considered evidence of damaged, and certainly reactive, tissue surrounding the wound.³⁴ As shown in Table 1, *initial debridement* refers to the first debridement performed on a wound following the initial evaluation by the clinician. This may or may not, however, be the first debridement performed during the life cycle of a particular wound. Patients with difficult-to-heal and chronic wounds may have many comorbidities and may transition between multiple clinicians and sites of care before complete healing is achieved. Nonetheless, the first time any clinician sees a patient for wound care, the wound should always be re-evaluated and an independent decision made about the need for debridement.

Maintenance debridement, on the other hand, can be defined as ongoing

wound debridement to keep the wound free of impediments to healing in addition to the removal of nonviable, damaged, and/or infected tissue.^{22,23} Maintenance debridement was first described in the context of a novel approach to make the wound more permissive to healing, the concept of *wound bed preparation*.^{22,23} The expert panel and authors of the present manuscript agree that a wound may need maintenance debridement to restore and sustain a healing mode. Thus, in a wound that is resistant to healing, the clinician must recognize that other factors that are not clinically detectable upon inspection may be present in the wound bed. Such factors include a high bacterial burden or biofilm, a disequilibrium of tissue metalloproteinases, and wound cells that have become phenotypically abnormal (the cellular burden).^{22,23} The panel suspects that, in time, some of these parameters may become more easily measurable and provide a greater rationale for therapeutic intervention.

Table 1 defines both initial debridement and maintenance debridement, as agreed upon by the panel. There was discussion among panel members about what constitutes a lack of wound closure or, perhaps even more challenging, inadequate wound closure. This has important ramifications when discussing at what point to institute maintenance debridement and when to terminate it (Figure 2). Improvement in the wound, specifically its depth and surface area, has long been used as a clinical parameter. However, short of no improvement at all, the critical issue is how much of an improvement is needed to regard the wound as closing in a timely manner. There was consensus that this should remain an active

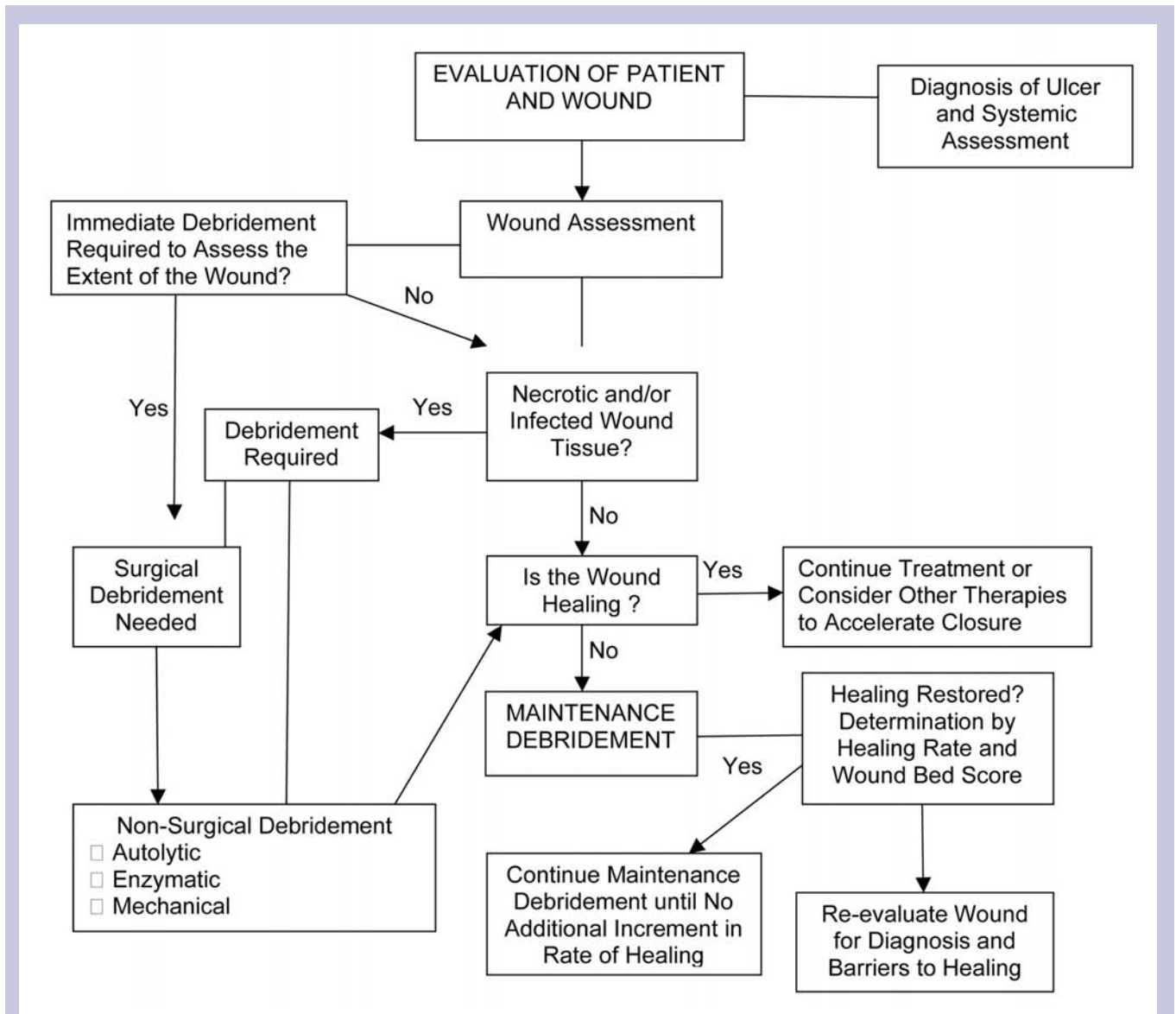


Figure 2. Algorithm for debridement in the care of chronic wounds. Copyright © 2008, V. Falanga.

area of investigation and testing. One possibility is to use the rate of 0.75 mm/week of wound edge advancement (to be discussed later), which is about 80% sensitive and specific for ultimate wound closure. However, an easier way that does not require calculating the healing rate is to use the WBS (Figure 1). Studies are needed to explore the validity of this recommendation.

GOALS OF INITIAL AND MAINTENANCE DEBRIDEMENT

Some chronic wound care guidelines have adopted these definitions of debridement and maintenance debridement, but in most cases, the two either have not been crisply separated or have been merged. For example, in some chronic wound care guidelines, debridement is listed as a key element of both infection control

and wound bed preparation. The WHS guidelines state that debridement “is required to remove the obvious necrotic tissue, excessive bacterial burden, and cellular burden of dead and senescent cells”^{3,5,16,17} and that maintenance debridement “is needed to maintain the appearance and readiness of the wound bed for healing.”^{3,5,16} These two statements may lead to confusion. Indeed, this discussion is meant

Table 2. Recommendations regarding surgical debridement

Extent of tissue removal

- Remove all apparent necrotic, infected tissue and clinically nonviable or infected bone.
- Carefully evaluate all exposed bone and remove if clinically indicated.
- Remove the callus surrounding the wound in diabetic neuropathic ulcers.

Biopsy material

- This may be required for diagnostic reasons.
- Biopsy tissue to determine quantitative bacteriology and the presence of *Streptococcus*.
- Cultures should be taken from the deep part of the wound to avoid sampling of surface colonization.

Aftercare

- Apply a postsurgical dressing to control bleeding and prevent infection.
- In follow-up, determine the need for repeated debridement (if necrotic or infected tissue recurs) or maintenance debridement if the wound does not improve.

emphasize that decisions about debridement can only be made after a thorough evaluation of all aspects of the patient's condition (including nutritional and vascular status). Therefore, debridement is most effectively implemented within the context of a therapeutic regimen that addresses all of the wound's needs. Some topical therapies may interfere with one another or with the use of certain therapeutic agents. For example, concomitant use of heavy metals and antiseptics, particularly those that are not slow-release, needs to be approached with caution in the context of living skin substitutes and growth factors.^{23,24}

The evaluation process involves close attention to systemic issues (ie, presence of diabetes, heart failure, use of immunosuppressive agents) as well as wound assessment. As shown in Figure 2, checkpoints prompt the clinician to make important decisions regarding the need for immediate surgical debridement, the type of debridement to adopt, and the need for maintenance debridement.

The recommendations for debridement and which type of debridement provided in this section stem from the opinions and experience of the expert panel. This is also by necessity, for there is general lack or paucity of clinical research trials addressing these basic wound care topics. Particularly, there are no large and reliable studies comparing different approaches and methods of debridement. The panel recognizes that this is a fertile area of investigation, which may have been lagging behind other evidence-based topics because of the lack of agreed upon endpoints.

Surgical/sharp debridement should be considered when there is obvious necrotic or infected tissue, clinically nonviable or infected bone,

to clarify those statements regarding maintenance debridement. Table 1 defines more clearly the goals of debridement, re-emphasizing the distinction between initial and maintenance debridement. This point is addressed further in the discussion on the algorithm for debridement (Figure 2) in the next section.

It is essential to emphasize that complete and timely wound closure is the overriding objective of all aspects of wound care, including debridement, because the latter correlates with wound closure rates.³²⁻³⁴ One can acknowledge that circumstances exist in which patient comfort and wound stabilization, rather than complete wound closure, may be the primary goals of wound care. However, this approach should apply to only a small percentage of cases, such as in those patients for whom treatment is simply not possible or practical because of their unique and

underlying comorbidities.⁴² Terminal or serious illness, *per se*, generally is not considered sufficient cause to abandon efforts to promote healing. On the other hand, the exclusive emphasis on complete wound closure for all types of wounds, rather than additional focus on decreased wound size, pain control, and quality-of-life improvements, may have made it more difficult for new therapies to reach regulatory approval.

IMPLEMENTATION OF DEBRIDEMENT WITHIN THE CONTEXT OF AN EFFECTIVE WOUND CARE STRATEGY

The role of debridement within a comprehensive wound care strategy is highlighted in the algorithm shown in Figure 2, which was agreed upon by panel members. Discussion of all components of effective wound care is beyond the scope of this article. However, it is important to

undermining or tunneling, or callus (Table 2). The panel considered undermining of a wound to be a critical problem that affects both the proper evaluation of the extent of the wound and the possibility that undermining creates a dead space immediately beneath the surrounding skin that might facilitate bacterial overgrowth. However, it also was recognized that in certain clinical situations, eg, in pressure ulcers and when the undermining is not judged to be extensive, one need not immediately proceed to surgical/sharp debridement. The disadvantages of surgical debridement are its invasiveness and that healthy tissue is removed. An important consideration for surgical debridement also is based on proper wound assessment. This situation typically arises in the context of diabetic foot ulcers where the extent of the ulceration, tunneling, and the possibility of an abscess may not be clinically evident and must be explored immediately (Figure 2). The patient's vascular status, however, must always be considered before surgical/sharp debridement is performed.

Practical recommendations for surgical/sharp debridement are summarized in Table 2.

All exposed bone should be evaluated and removed if clinically indicated. It is also particularly important to remove callus in diabetic neuropathic ulcers due to the possibility that the callus causes additional pressure to the wound edges.

During surgical debridement, it may be appropriate to obtain tissue for histological and microbial evaluation. This is usually done to exclude a malignancy, to help in the diagnosis of an inflammatory ulcer (ie, due to vasculitis), or to rule out an unusual wound infection. A rare but important difficulty is encountered when faced with pyoderma gangrenosum,

an inflammatory condition that can be idiopathic or most commonly associated with rheumatoid arthritis and ulcerative colitis. If this condition is suspected (biopsy is not diagnostic), dermatological consultation is helpful. Pyoderma gangrenosum, through a process known as pathergy, can worsen and enlarge dramatically upon surgical debridement.⁴³

A critical checkpoint in Figure 2 is the decision of whether the wound is not healing, even when the wound bed seems adequate and free of obviously necrotic and infected tissue. If the wound shows signs of healing, such as decreased depth and overall size or wound edges that have migrated over the wound bed, one may opt to continue the treatment strategy that is being carried out at that time. However, additional and/or more advanced treatment modalities may be used at this point to further accelerate wound closure.

In the absence of satisfactory signs of healing, even when the wound bed seems adequate, maintenance debridement should be strongly considered. Falanga^{22,23} previously discussed the rationale for initiating maintenance debridement at that point. Biochemical and cellular abnormalities, as well as excessive bacterial burden, may cause impaired healing and may be helped by removing what falsely appears to be normal granulation tissue. For maintenance debridement, nonsurgical means may be tried first, but one should also consider surgical tissue removal if no improvement is noted.

Initial debridement. Initial debridement may be surgical/sharp, mechanical, enzymatic, biological (larval therapy), or autolytic, depending on the condition of the wound. The merits of each approach are discussed briefly in Table 3 and in

review articles.^{37,44-46} A key issue for initial debridement is when to choose surgical/sharp debridement, which requires specific personnel, facilities, and expertise, over other available techniques.

The previous section discussed surgical debridement. As indicated in Figure 2, depending on the clinical findings and circumstances, the clinician may decide to use nonsurgical means of debridement. These other types of debridement and their advantages and disadvantages also are discussed in Table 3 and reviewed elsewhere.⁴⁴⁻⁴⁹ It must be appreciated that overlaps exist in the way different debriding interventions work. For example, absorbent agents (including dressings) may overlap with slow-release antiseptic preparations that incorporate an antiseptic within a release vehicle (Table 3). Another difficulty is how to discuss a feature of a debriding agent that comes into play immediately before or after debridement begins. For example, occlusive dressings often are said to cause painless debridement. Yet, their removal can be associated with considerable pain. For this reason, the last column in Table 3 refers to an overall assessment of the patient's comfort rather than pain alone. Yet another difficulty encountered by the panel in defining debriding agents and in formulating Table 3 was whether to include slow-release antiseptics. However, clinical studies have shown that effective debridement can be achieved with this approach.⁵⁰

There are specific advantages and disadvantages to all classes of debriding approaches, as summarized in Table 3. Absorbent agents, such as beads (less commonly used now) and newer absorbing dressings tend to be slow in action. Autolytic debridement,

Table 3. Selection of debridement types and methods

TYPE OF DEBRIDEMENT	EXAMPLES	SPEED OF TISSUE REMOVAL	PRESERVATION OF HEALTHY TISSUE	PATIENT'S COMFORT
Absorbent Agents	Dextranomer beads, some absorbent dressings	□	□□□□	□□□
Antiseptics	Slow-release agents, cadexomer iodine	□	□□□	□□
Autolytic	Occlusive dressings	□	□□□□	□□□□
Biological	Larval therapy	□□□	□□□	□
Chemical	Zinc chloride	□□	□	□
Enzymatic	Collagenase, papain	□□□	□□□□	□□□□
Mechanical	Wet-to-dry, negative pressure, pulsed lavage	□□□	□□	□
Surgical/sharp	Scalpel, curette, hydro-surgery	□□□□□	□	□

□ = Minimal or none; □□□□□ = Maximal

generally achieved by the use of occlusive or semioclusive wound dressings, is also slow. Antiseptics, in a slow-release form, can be thought of as debriding agents because they remove exudate and can reduce the bacterial burden that is causing inflammation within the wound. Biological debridement using maggots is being used increasingly, especially in some European countries; the method is less popular in the United States. This type of larval debridement therapy can be quite effective and relatively rapid in removing an eschar and producing good granulation tissue. However, proper biological debridement requires considerable experience for application, attention to proper disposal of dressings removed from the wound (full of larvae), and careful avoidance of larvae-induced damage to the surrounding skin. Chemical debridement is rarely used. Zinc chloride, once used for *in-vivo* tissue fixation for the treatment of skin cancer by Mohs surgery, can be

quite painful but also effective.⁴⁷ Enzymatic debridement, either with collagenase or papain, has become popular for ulcers that do not require immediate tissue removal. There are differences in how enzymes work. Thus, collagenase is more specific than papain in that it cleaves the denatured portion of collagen.⁴⁸ Mechanical debridement by wet-to-dry dressing removal is painful and removes healthy tissues that are healing. Most clinicians now feel that this type of debridement is not justified. In the past, this type of debridement has been used when necrotic tissue needed to be removed and surgical debridement was not possible or practical. Mechanical debridement can also be accomplished by the use of negative pressure devices; this has the advantage of constantly removing exudate from the wound.^{37,44-46}

In summary, there are many types of nonsurgical debridement. Table 3 lists some of their characteristics, but it is up to the clinician to determine

what might work best in a particular clinical situation and treatment setting. Often, the choice of debridement is dictated by the experience of the clinician with that method. This is certainly the case with larval therapy. In exudative wounds, slow-release antiseptics or negative pressure devices may be ideal. Enzymatic debridement agents are very useful for long-term usage and certainly for maintenance debridement.⁴⁸

Because of the practical challenges associated with performing surgical/sharp debridement in certain clinical settings, such as long-term nursing facilities, it may be appropriate in many circumstances to treat many with a short course (1 week) of an enzymatic debriding agent before considering surgical/sharp debridement (Table 3). This approach allows the clinician to gauge the potential efficacy of this more conservative approach and to determine if the wound is progressing toward healing. However, the

safety and welfare of the patient must not be compromised because of difficulties with the clinical setting; the clinician needs to follow the optimal principles of wound care. Different types of enzymatic agents are available, and some are able to digest thick eschars in certain cases. Collagenase- and papain-based enzymatic agents are available for this purpose.⁴⁸ Enzymatic treatment, which can be considered a milder and slower approach to debridement, is not appropriate when advancing necrosis is present.

Maintenance debridement. In the face of impaired or slow healing, the clinician should strongly consider instituting maintenance debridement to improve wound bed preparation.^{22,23,37} Although surgical debridement may be used even for maintenance debridement, a more gentle approach is often used (Table 3). A critical issue with regard to maintenance debridement is deciding how long to continue it and when to debride again if not choosing a method that is ongoing (enzymes, chemicals, etc.). The only way to ensure that the goal of maintenance debridement (keeping the wound free of barriers to healing) is being met is to evaluate the wound for evidence of healing. Although this is not a primary focus of our discussion, the actual healing rate, representing the speed at which wound edges move inward within the first 3 to 4 weeks of therapy, has been shown to be a valid endpoint.⁵¹ Using the healing rate in clinical trials could lead to more efficient screening of experimental treatments and could accelerate the regulatory approval of new and useful therapies. In previous reports, it has been shown that the healing rate, measurable at 4 weeks of therapy, is approximately 80% sensi-

Clinical inspection alone cannot determine whether the wound bed is adequate from a microbial, biochemical, and cellular standpoint. Hence, one must rely on functional parameters that show evidence of healing.

tive and specific for predicting ultimate wound closure in venous (by 24 weeks; n = 136) and diabetic (by 12 weeks; n = 96) ulcers.³⁶ Interestingly, in the same studies, it was found that a critical threshold of the healing rate for predicting wound closure was 0.75 mm/week of inward movement of the wound edges for both types of venous and diabetic neuropathic wounds. The implication is that there is a certain keratinocyte speed limit common to chronic wounds.^{23,51}

The goal of maintenance debridement is to keep the wound progressing toward healing with minimal interruption, but there may be cost-effectiveness benefits as well. A proactive strategy may reduce the need for more aggressive (and potentially more traumatic and costly) debridement in response to wound deterioration.

Some wounds may need only mild, ongoing debridement, while others may require more potent treatments. The available options are described in Table 3.

During maintenance debridement, the methods used from Table 3 may vary during follow-up, depending on the clinical findings and necessities. The effectiveness of maintenance debridement should be monitored regularly and adjusted as needed to ensure that the wound continues to progress toward healing. The WBS, as in Figure 1, can be useful in this regard, because optimal wound bed

preparation should be the result of an effective maintenance debridement strategy.^{36,37} Absence of healing, slow healing, or an increase in the size of the wound is sufficient to signal that the patient should be re-evaluated. If no explanation can be found for the failure to heal (ie, infection or excessive bacterial burden, systemic factors, lack of compliance with offloading), a more aggressive method of debridement may be chosen (Table 3). Of course, debridement is only part of wound bed preparation. It may be necessary to intervene with other treatment modalities to jumpstart the healing process.

It is important to keep in mind that once maintenance debridement has been instituted, it should not be discontinued just because the wound “looks good”; the ultimate goal is to achieve complete closure. A wound bed that “looks good” to a clinician (even an experienced one) does not necessarily mean that it is adequate from a microbial, biochemical, and cellular standpoint. In discussing and constructing the algorithm shown in Figure 2, the panel also considered at what point maintenance debridement should be discontinued when the wound is showing evidence of healing. Hence, as emphasized with the approach of wound bed preparation,^{22,23,37} one needs to rely on functional parameters that show evidence of healing. However, this needs further

A growing body of evidence shows that effective and frequent debridement can improve healing rates.

study. As shown in Figure 2, the panel proposes that maintenance debridement should be continued as long as further increments in healing occur, which could be characterized as an increasing healing rate or an improvement in the WBS (Figure 1).

CONCLUSION AND FUTURE NEEDS

This report relied on the collective expertise of a group of experts from different fields to arrive at an agreement with regard to maintenance debridement and a clinical algorithm that clinicians can use when evaluating a chronic wound. The clinical and scientific rationale for maintenance debridement was discussed based on the panel's understanding of bacterial burden and biofilms, biochemical abnormalities, and phenotypic changes in cells persisting (cellular burden) in from the wound bed and at the edge of nonhealing wounds. As a result, an important conclusion is that maintenance debridement may be required when the wound is not healing and even when the wound bed appears to be satisfactory upon clinical inspection. Implicit in these conclusions is that there can be a strong dissociation between the clinical appearance of the wound bed and satisfactory healing. Clinical

research, combined with *in-vitro* studies and biomarkers, may provide stronger evidence for these statements and recommendations.

The efficacy of debridement strategies could be improved by the development of simple markers or assessment tools that could be used at the bedside to determine quickly if additional debridement is necessary. Therefore, an important consensus statement from this expert panel is that, at least in chronic wounds, the effectiveness of debridement can only be determined by how the debridement improves wound healing. The recommendations made in this report, which are to use the healing rate and the WBS, follow this premise. However, the panel realizes that additional and larger trials will be needed to fully determine the validity of these recommendations regarding debridement and which assessments of debridement (ie, WBS or healing rate) most accurately predict healing. There is also a great and increasing demand for on-site educational programs focusing on the practical implementation of best practice paradigms. There is a particular need for a greater understanding of the strengths, limitations, and most effective use of different methods of debridement. Comparative studies of

different types of debridement, perhaps using the algorithm proposed in this report, could bring about a cogent approach to these difficult issues regarding basic wound care.

The human and economic impact of difficult-to-heal and chronic wounds, such as venous, diabetic, pressure, and arterial insufficiency ulcers, is large and growing. The overall healing rates for these wounds can be improved with the aid of clear, evidence-based guidelines for comprehensive care.^{3,5,16-19} These guidelines outline the importance of an effective debridement strategy in preventing infection and preparing the wound bed to heal.

A growing body of evidence shows that effective and frequent debridement can improve healing rates. Debridement aids healing by removing and keeping the wound free of necrotic tissue and excessive bacteria as well as the dead and senescent cells that can interfere with healing.^{22,23,37} An effective debridement strategy will consist of an initial debridement combined with a customized maintenance debridement plan. Maintenance debridement is likely to be most effective as a proactive approach to prevent the build-up of necrotic or denatured tissues and cellular abnormalities.

There are still many unmet needs in the treatment of difficult-to-heal and chronic wounds. Rates of complete wound closure remain unacceptably low,¹⁰ and far too many patients suffer from complications resulting from nonhealing wounds. The conscientious implementation

ACKNOWLEDGMENTS

Dr. Falanga's work was supported in part by NIH grant DK067836, the Wound Biotechnology Foundation, and Award Number P20RR018757 from the National Center For Research Resources. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Center For Research Resources or the National Institutes of Health.

of an effective maintenance debridement strategy within a comprehensive wound care plan may help in significantly improving these outcomes.

References

- Phillips T, Stanton B, Provan A, Lew R. A study of the impact of leg ulcers on quality of life: financial, social, and psychologic implications. *J Am Acad Dermatol*. 1994;31(1):49–53.
- Brem H, Kirsner RS, Falanga V. Protocol for the successful treatment of venous ulcers. *Am J Surg*. 2004;188(1A Suppl):1–8.
- Steed DL, Attinger C, Colaizzi T, et al. Guidelines for the treatment of diabetic ulcers. *Wound Repair Regen*. 2006;14(6):680–692.
- Centers for Disease Control and Prevention. Diabetes Data & Trends. Available at: www.cdc.gov/diabetes/statistics/prev/national/figpersons.htm. Accessed February 14, 2006.
- Whitney J, Phillips L, Aslam R, et al. Guidelines for the treatment of pressure ulcers. *Wound Repair Regen*. 2006;14(6):663–679.
- Pressure ulcers in America: prevalence, incidence, and implications for the future. An executive summary of the National Pressure Ulcer Advisory Panel monograph. *Adv Skin Wound Care*. 2001;14(4):208–215.
- Olin JW, Beusterien KM, Childs MB, Seavey C, McHugh L, Griffiths RI. Medical costs of treating venous stasis ulcers: evidence from a retrospective cohort study. *Vasc Med*. 1999;4(1):1–7.
- Gordois A, Scuffham P, Shearer A, Oglesby A, Tobian JA. The health care costs of diabetic peripheral neuropathy in the US. *Diabetes Care*. 2003;26(6):1790–1795.
- Kumar RN, Gupchup GV, Dodd MA, et al. Direct health care costs of 4 common skin ulcers in New Mexico Medicaid fee-for-service patients. *Adv Skin Wound Care*. 2004;17(3):143–149.
- Margolis DJ, Allen-Taylor L, Hoffstad O, Berlin JA. Healing diabetic neuropathic foot ulcers: are we getting better? *Diabet Med*. 2005;22(2):172–176.
- Franks PJ, Moffatt CJ. Health related quality of life in patients with venous ulceration: use of the Nottingham health profile. *Qual Life Res*. 2001;10(8):693–700.
- Thomas DR, Diebold MR, Eggemeyer LM. A controlled, randomized, comparative study of a radiant heat bandage on the healing of stage 3–4 pressure ulcers: a pilot study. *J Am Med Dir Assoc*. 2005;6(1):46–49.
- Cali TJ, Bruce M. Pressure ulcer treatment: examining selected costs of therapeutic failure. *Adv Wound Care*. 1999;12(Suppl 2):8–11.
- Iglesias CP, Birks Y, Nelson EA, Scanlon E, Cullum NA. Quality of life of people with venous leg ulcers: a comparison of the discriminative and responsive characteristics of two generic and a disease specific instruments. *Qual Life Res*. 2005;14(7):1705–1718.
- Shukla D, Tripathi AK, Agrawal S, Ansari MA, Rastogi A, Shukla VK. Pain in acute and chronic wounds: a descriptive study. *Ostomy Wound Manage*. 2005;51(11):47–51.
- Robson MC, Cooper DM, Aslam R, et al. Guidelines for the treatment of venous ulcers. *Wound Repair Regen*. 2006;14(6):649–662.
- Hopf HW, Ueno C, Aslam R, et al. Guidelines for the treatment of arterial insufficiency ulcers. *Wound Repair Regen*. 2006;14(6):693–710.
- Centers for Medicare & Medicaid Services. Medicare Coverage Database. Available at: <http://www.cms.hhs.gov/mcd/viewmcd.asp?where=index&mid=28>. Accessed March 17, 2008.
- Lau J, Tatsioni A, Balk E, et al. *Usual Care in the Management of Chronic Wounds: A Review of the Recent Literature*. Rockville, Md: Agency for Healthcare Research and Quality; 2005.
- Office of Inspector General. Medicare payments for surgical debridement services in 2004. Office of Inspector General, 2007. Available at: <http://oig.hhs.gov/oei/reports/oei-02-05-00390.pdf>. Accessed May 5, 2008.
- Moore J, Jensen P. Assessing the role and impact of enzymatic debridement. *Podiatry Today*. 2004;17(7):54–61.
- Falanga V. Classifications for wound preparation and stimulation of chronic wounds. *Wound Repair Regen*. 2000;8(5):347–352.
- Falanga V. The chronic wound: impaired healing and solutions in the context of wound bed preparation. *Blood Cells Mol Dis*. 2004;32(1):88–94.
- Falanga V. Wound healing and its impairment in the diabetic foot. *Lancet*. 2005;366(9498):1736–1743.
- Schultz GS, Sibbald RG, Falanga V, et al. Wound bed preparation: a systematic approach to wound management. *Wound Repair Regen*. 2003;11(Suppl 1):S1–S28.
- James GA, Swogger E, Wolcott R, et al. Biofilms in chronic wounds. *Wound Repair Regen*. 2008;16(1):37–44.
- Brem H, Stojadinovic O, Diegelmann RF, et al. Molecular markers in patients with chronic wounds to guide surgical debridement. *Mol Med*. 2007;13(1–2):30–39.
- Cha J, Kwak T, Butmarc J, et al. Fibroblasts from non-healing human chronic wounds show decreased expression of beta ig-h3, a TGF-beta inducible protein. *J Dermatol Sci*. 2008;50(1):15–23.
- Wysocki AB. Wound fluids and the pathogenesis of chronic wounds. *J Wound Ostomy Continence Nurs*. 1996;23(6):283–290.
- Ladwig GP, Robson MC, Liu R, Kuhn MA, Muir DF, Schultz GS. Ratios of activated matrix metalloproteinase-9 to tissue inhibitor of matrix metalloproteinase-1 in wound fluids are inversely correlated with healing of pressure ulcers. *Wound Repair Regen*. 2002;10(1):26–37.
- Mwaura B, Mahendran B, Hynes N, et al. The impact of differential expression of extracellular matrix metalloproteinase inducer, matrix metalloproteinase-2, tissue inhibitor of matrix metalloproteinase-2 and PDGF-AA on the chronicity of venous leg ulcers. *Eur J Vasc Endovasc Surg*. 2006;31(3):306–310.
- Saap LJ, Falanga V. Debridement performance index and its correlation with complete closure of diabetic foot ulcers. *Wound Repair Regen*. 2002;10(6):354–359.
- Falanga V, Saap LJ, Ozonoff A. Wound bed score and its correlation with healing of chronic wounds. *Dermatol Ther*. 2006;19(6):383–390.
- Steed DL, Donohoe D, Webster MW, Lindsley L. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. *J Am Coll Surg*. 1996;183(1):61–64.
- Ennis WJ, Meneses P. Issues impacting wound healing at a local level: the stunned wound. *Ostomy Wound Manage*. 2000;46(1A Suppl):39S–48S.
- Falanga V, Sabolinski ML. Prognostic factors for healing of venous and diabetic ulcers. *WOUNDS*. 2000;12(5 Suppl A):42A–46A.
- Panuncialman J, Falanga V. The science of wound bed preparation. *Clin Plast Surg*. 2007;34(4):621–632.
- Kim BC, Kim HT, Park SH, et al. Fibroblasts from chronic wounds show altered TGF-beta signaling and decreased TGF-beta Type II receptor expression. *J Cell Physiol*. 2003;195(3):331–336.
- Stoodley P, Sauer K, Davies DG, Costerton JW. Biofilms as complex differentiated communities. *Annu Rev Microbiol*. 2002;56:187–209.
- Wolcott RD, Rhoads DD. A study of biofilm-based wound management in subjects with critical limb ischaemia. *J Wound Care*. 2008;17(4):145–155.
- Donlan RM, Costerton JW. Biofilms: survival mechanisms of clinically relevant microorganisms. *Clin Microbiol Rev*. 2002;15(2):167–193.
- Ferris FD, Al Khateib AA, Fromantin I, et al. Palliative wound care: managing chronic wounds across life's continuum: a consensus statement from the International Palliative Wound Care Initiative. *J Palliat Med*. 2007;10(1):37–39.
- Panuncialman J, Falanga V. Basic approach to inflammatory ulcers. *Dermatol Ther*. 2006;19(6):365–376.
- Kirshen C, Woo K, Ayello EA, Sibbald RG. Debridement: a vital component of wound bed preparation. *Adv Skin Wound Care*. 2006;19(9):506–517.
- Ayello EA, Cuddigan JE. Debridement: controlling the necrotic/cellular burden. *Adv Skin Wound Care*. 2004;17(2):66–75.
- Steed DL. Debridement. *Am J Surg*. 2004;187(5A):71S–74S.
- Falanga V, Iriondo M. Zinc chloride paste for the debridement of chronic leg ulcers. *J Dermatol Surg Oncol*. 1990;16(7):658–661.
- Falanga V. Wound bed preparation and the role of enzymes: a case for multiple actions of therapeutic agents. *WOUNDS*. 2002;14(2):47–57.
- Bradley M, Cullum N, Sheldon T. The debridement of chronic wounds: a systematic review. *Health Technol Assess*. 1999;3(17 Pt 1):1–78.
- Brem H, Balledux J, Sukkariet T, Carson P, Falanga V. Healing of venous ulcers of long duration with a bilayered living skin substitute: results from a general surgery and dermatology department. *Dermatol Surg*. 2001;27(11):915–919.
- Donohue K, Falanga V. Healing rate as a prognostic indicator of complete healing: a reappraisal. *WOUNDS*. 2003;15(3):71–76.

Post-Test Questions

1) What are the recent estimates of the percentage of chronic wounds that achieve complete closure after 20 weeks of treatment?

- a) 80%–90%
- b) 5%–10%
- c) 25%–50%
- d) 100%

2) What factors within the wound bed can contribute to poor healing?

- a) The presence of clinically necrotic and infected tissue
- b) Excessive wound exudate
- c) The presence of biofilm within the wound
- d) Abnormal and unresponsive cells at the wound edge
- e) All of the above
- f) none of the above

3) What was the effect of debridement frequency in a clinical trial of the safety and efficacy of recombinant human platelet-derived growth factor in the treatment of chronic diabetic ulcers?

- a) There was no effect
- b) Centers performing the most frequent debridements had the highest healing rates
- c) Centers performing the least frequent debridements had the lowest healing rates
- d) b and c

4) True or false: There is no evidence that more frequent debridement can help reduce amputation rates.

- a) True
- b) False

5) True or false: Wounds that show a clinically adequate wound bed but are not progressing toward healing may benefit from debridement.

- a) True
- b) False

6) Approximately what percentage of chronic wounds contain biofilm-embedded bacteria?

- a) 100%
- b) 5%
- c) 60%
- d) 0%

7) What is not a method of debridement?

- a) Mechanical
- b) Magnetic
- c) Surgical
- d) Enzymatic
- e) Biological
- f) None of the above

8) What is the primary goal of maintenance debridement?

- a) To keep the wound free of barriers to healing
- b) To maintain the size of the wound
- c) To control itching
- d) To determine the cause of the wound

9) How long should maintenance debridement be continued?

- a) As long as it is effective and the wound is progressing toward healing
- b) Until the wound is clear of necrotic tissue
- c) Until there is no sign of infection
- d) All of the above
- e) None of the above

10) What is a sign that the wound should be re-evaluated?

- a) Nonhealing
- b) Slow healing
- c) Increase in the size of the wound
- d) All of the above
- e) None of the above

Answer and Evaluation Form

Supplement to June 2008 *Ostomy Wound Management*

Please print clearly: Certificate will be mailed to this address within 6–8 weeks.

Name: _____ Degree: _____ Position/Title: _____

Organization/Institution: _____

Department: _____

Mailing Address for Certificate: CHECK ONE: _____ WORK _____ HOME

Street: _____

City: _____ State: _____ Zip Code: _____

E-mail Address: _____

Phone: _____ Fax: _____

<p>Answers: Refer to post-test. Circle ALL that apply for each question.</p> <p>1.) A B C D</p> <p>2.) A B C D E F</p> <p>3.) A B C D</p> <p>4.) A B</p> <p>5.) A B</p> <p>6.) A B C D</p> <p>7.) A B C D E F</p> <p>8.) A B C D</p> <p>9.) A B C D E</p> <p>10.) A B C D E</p>	<p>Please answer the following questions by circling the appropriate rating: 5 = Strongly Agree 4 = Agree 3 = Neutral 2 = Disagree 1 = Strongly Disagree</p> <table style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 75%;">The stated learning objectives were met</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> <tr> <td>Faculty was knowledgeable on the subject matter</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> <tr> <td>Content was objective</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> <tr> <td>Content was balanced</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> <tr> <td>Met my educational needs</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> <tr> <td>Content was scientifically rigorous</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> <tr> <td>Content avoided commercial bias or influence</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> <tr> <td>Content was timely and related to my practice</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> <tr> <td>Content will assist me in enhancing patient care</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> <tr> <td>Information presented will improve my practice/patient outcomes</td> <td style="text-align: right;">5</td> <td style="text-align: right;">4</td> <td style="text-align: right;">3</td> <td style="text-align: right;">2</td> <td style="text-align: right;">1</td> </tr> </table>	The stated learning objectives were met	5	4	3	2	1	Faculty was knowledgeable on the subject matter	5	4	3	2	1	Content was objective	5	4	3	2	1	Content was balanced	5	4	3	2	1	Met my educational needs	5	4	3	2	1	Content was scientifically rigorous	5	4	3	2	1	Content avoided commercial bias or influence	5	4	3	2	1	Content was timely and related to my practice	5	4	3	2	1	Content will assist me in enhancing patient care	5	4	3	2	1	Information presented will improve my practice/patient outcomes	5	4	3	2	1
The stated learning objectives were met	5	4	3	2	1																																																								
Faculty was knowledgeable on the subject matter	5	4	3	2	1																																																								
Content was objective	5	4	3	2	1																																																								
Content was balanced	5	4	3	2	1																																																								
Met my educational needs	5	4	3	2	1																																																								
Content was scientifically rigorous	5	4	3	2	1																																																								
Content avoided commercial bias or influence	5	4	3	2	1																																																								
Content was timely and related to my practice	5	4	3	2	1																																																								
Content will assist me in enhancing patient care	5	4	3	2	1																																																								
Information presented will improve my practice/patient outcomes	5	4	3	2	1																																																								

What other topics would be of interest to you? _____

All tests/evaluation forms must be received by June 30, 2009.

Return via fax or mail to: NACCME-Princeton CME
 300 Rike Drive, Suite A
 Millstone Township, NJ 08535
 Fax: 610-560-0502



300 Rike Drive, Suite A
Millstone Township, NJ 08535