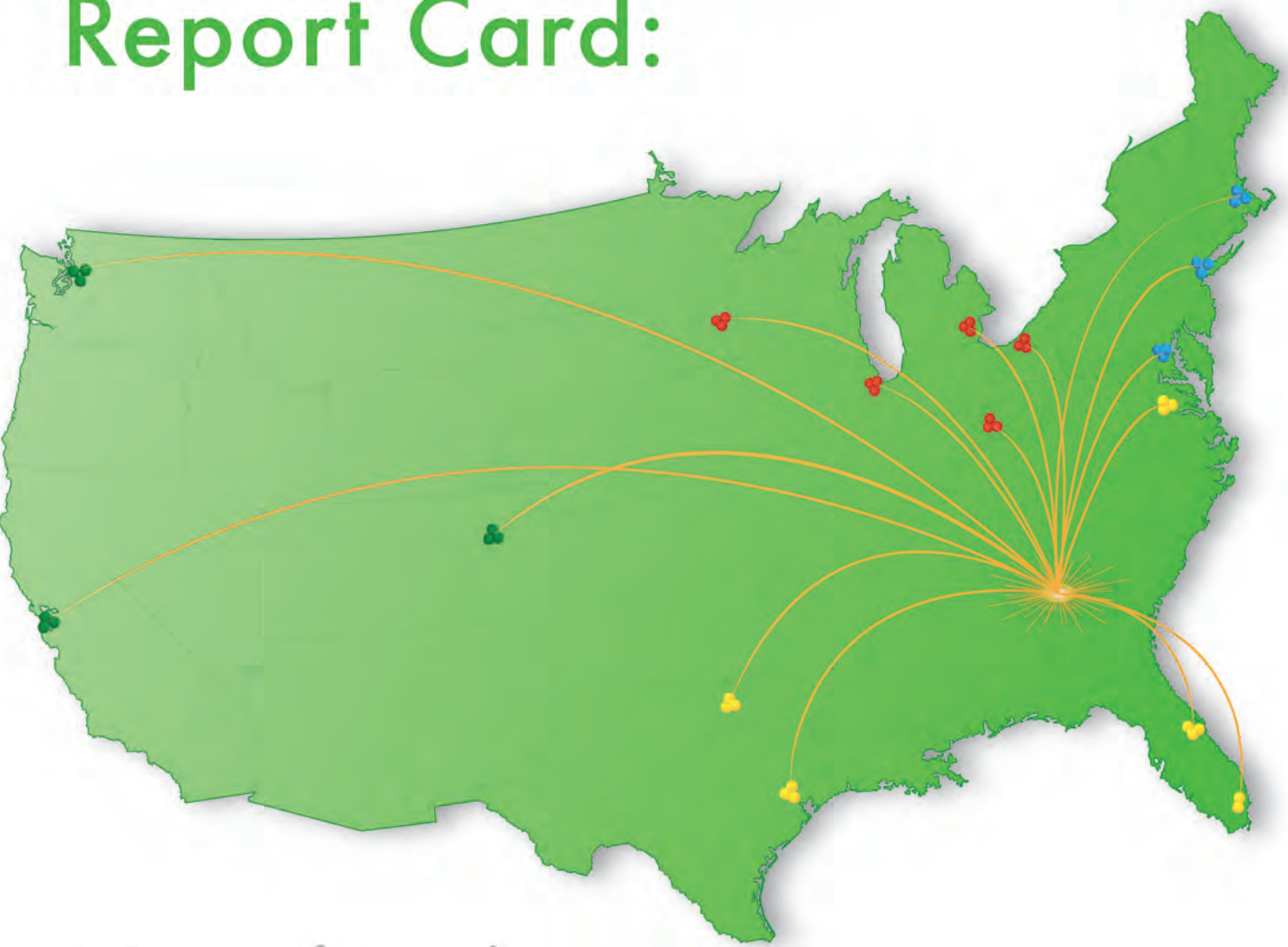
SUPPLEMENT TO APRIL 2009
OWM
OSTOMY WOUND MANAGEMENT

Clinical Practice Report Card:



A Survey of Wound Care Practices in the U.S.A.

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MÖLNLYCKE HEALTH CARE

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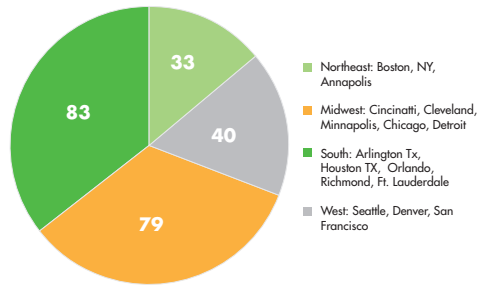
Figure 1

Number of wound care forum participants by region and location



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USA Report Card:
A tally of current wound care practices

Elizabeth A. Ayello, PhD, RN, ACNS-BC, ETN, FAPWCA, FAAN
R. Gary Sibbald, MD, FRCPC (Med, Derm), MACP, FAAD, MEd, FAPWCA

Treating and preventing wounds continue to be a challenge. Chronic wounds in particular have several known barriers that interfere with healing and require attention to achieve appropriate patient care outcomes. This includes debridement of necrotic tissue (debridement), identification and management of infection or inflammation (infection/inflammation), maintaining the right level of wound moisture (moisture balance), and attention to stalled wound edges (edge effect). The first letters of these four components comprise the acronym DIME.

Examining the bedside practices of wound care professionals can provide insight into the application of knowledge and challenges to the provision of best practices. In an attempt to determine attitudes and practices using the DIME woundbed preparation (WBP) paradigm, key opinion leader clinicians across the United States were invited to participate in one of 16 regional interactive wound care forums (WCF). Before attendance, each participant was asked to complete a survey developed by the authors (with the assistance of Mary McNeil and Valerie C. Johnson). The WCF were made possible through an unrestricted educational grant sponsored by Mölnlycke Health Care. (Locations of the forums are outlined in **Figure 1** on page 2.) A total of 215 clinicians attended the wound care forums, and 235 survey responses form the basis for the data. Not all respondents answered all questions. Participants who agreed to have their names included in this document can be found in the acknowledgments on page 2.

The following charts and graphs illustrate the survey respondents' answers to questions about types and numbers of patients seen by wound care teams, DIME components for desired clinical outcomes, and pain documentation. The following sections also detail some of the latest evidence that may improve best practices for each of the important elements of wound healing. To improve patient-centered care, a section has been devoted to improved wound-related pain management.

These data from more than 230 wound care community survey respondents and attendees at the sponsored wound care forums provide a snapshot of actual practices across the United States. The majority of survey responders were nurses caring primarily for adults and geriatric patients with pressure ulcers, venous ulcers or neuropathic/diabetic ulcers. For most, their practice setting was either a hospital or hospital wound care clinic. The clinician group was mainly comprised of individuals who spend most of their time involved in direct patient care. They confirmed that they were very busy wound healers and often members of wound-healing teams. They were willing to share their expert knowledge and represented a collective experience of caring for more than 5,000 patients weekly. We thank them for taking the time from their important work to provide such candid responses that add to our understanding of wound care. ■

Authors' note: *The following graphs (pages 4 to 6) represent the* ***number of responses*** *for each question, not the percentage of responses.*

Figure 2

Number of patients with wounds seen in an average week by 228 survey responents

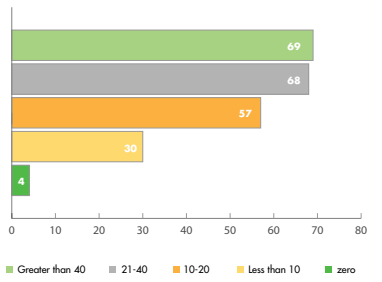


Figure 3

Patient population primarily treated by 230 respondents

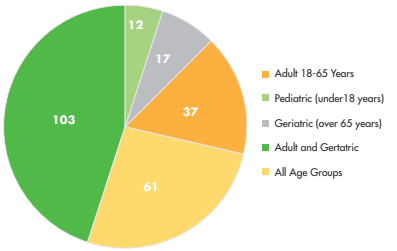


Figure 4

Number of patients with wounds seen in an average week in the workplace of 228 respondents

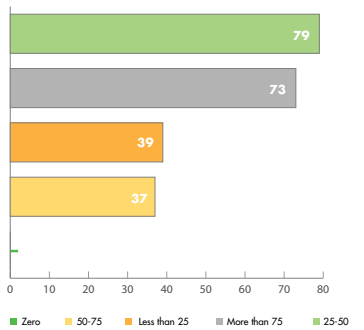


Figure 5

Most common wounds treated by survey respondents

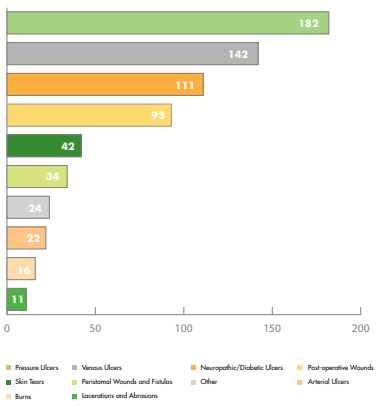


Figure 6

Number of respondents who regularly measure and document pain at dressing changes

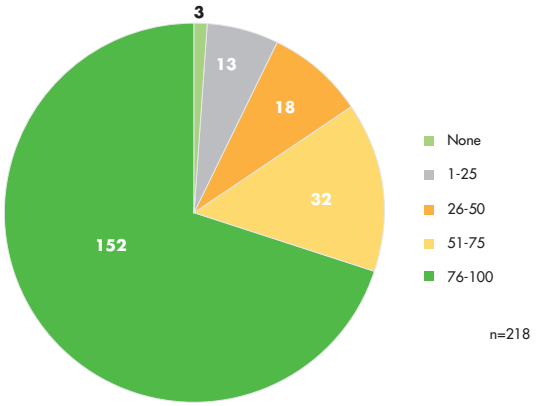


Figure 7

Number of respondents indicating choice of debridement based on the following factors

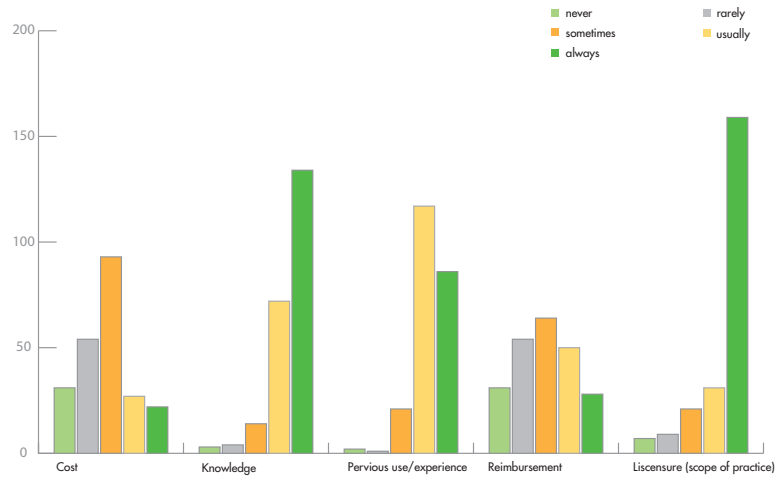


Figure 8

Number of respondents ranking dressing characteristics for their importance in maintaining moisture balance for moist wound healing

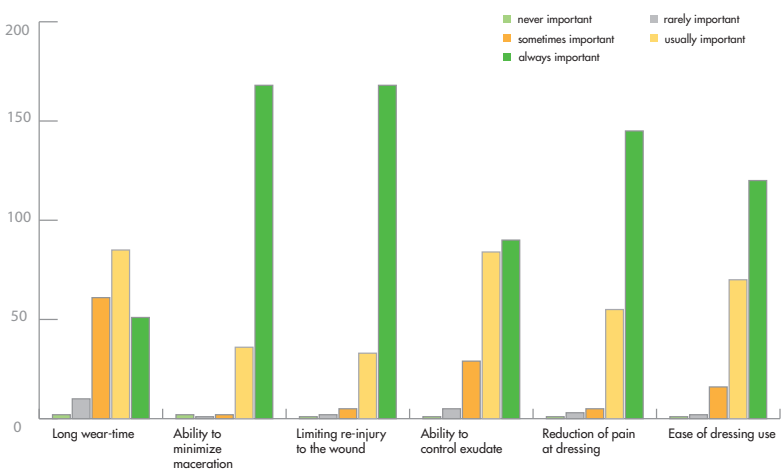


Fig. 6 — Pain Report Card

Pain is an impor tant patient-centered concern. Almost all respondents (88.40%) reported that they regularly measure and document pain at dressing changes, with almost thr ee-quarters repeating the assessment 76% to 100% of the time. Most respondents (77.98%) considered the dressings in their w ound care formulary to be helpful with the r eduction of w ound-related pain. **See clinical update on p. 7.**

Fig. 7 — Debridement Report Card

Debridement methods v ary depending b y wound type and characteristics. For diabetic foot ulcers, sharp debridement was most fr equently specified as the method of choice . For venous ulcers, sharp bedside debr idement or enzymes were most frequently mentioned, with some clinicians using honey. For pressure ulcers, debridement by sharp surgical methods or enzymes was frequently specified. As outlined in Figure 7, the scope of practice limitations as defined by licensure for sharp surgical debridement techniques was ranked as the highest importance (70.04%). This was followed by knowledge (59.03%) and previous use/experience (37.89%), with reimbursement (12.33%) and cost (9.69%) ha ving lower relevance. Debridement is dr iven by established protocol for 11.95% of r espondents. **See clinical update on p. 13.**

Fig. 8 — Moisture Balance Report Card

Participants provided insight into the impor tance of different wound-dressing characteristics for moisture balance and wound healing (Figure 8). The majority rank ed both the limiting of w ound bed re-injury on dressing removal (80.38%) and minimizing maceration and damage to sur rounding tissue (80.38%) as most important. This was followed closely by the reduction of pain (69.38%) and ease of dressing use (57.42%). The remaining dressing characteristics ranked were ability to control exudate odor (43.06%) and long wear time (24.40%). Over half (53.14%) felt the products in their for mularies met their desir ed wound-dressing characteristics more than 50% of the time. **See clinical update on p. 19.**

Figure 9a

Percentage of institution's wounds that are routinely cultured

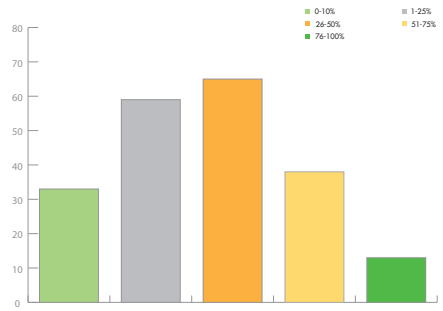


Figure 9b

Number of respondents indicating the method used most often to *diagnose* infection/inflammation of a wound

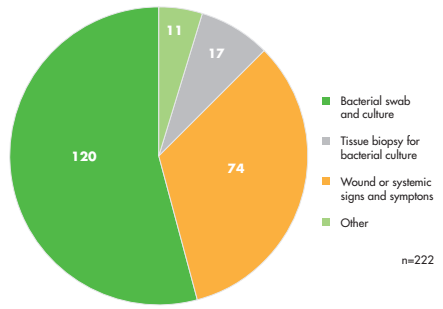
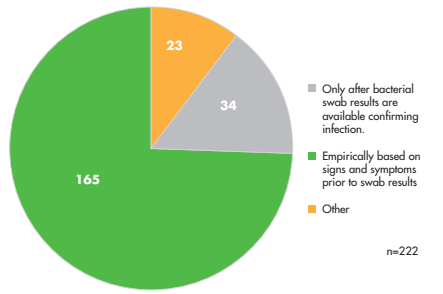


Figure 9c

Number of respondents indicating method for treating infection



Figs. 9a, 9b, 9c, 10, 11 — Infection/Inflammation Report Card

Considering wound infection/inflammation, there was a disconnect between what participants see as the diagnostic procedure and the institutional percentage of cultured wounds. The number of wounds routinely cultured is depicted in Figure 9a, with more than 50% of the wounds in their institutions routinely cultured 24.52 per cent of the time. While 33.33% of participants correctly identified that wound infection/inflammation is ***diagnosed*** by localized wound or systemic signs and symptoms, 54.05% stated that the diagnosis of infection was determined by bacterial swab and culture (Figure 9b). The majority of infections were ***treated*** empirically based on signs and symptoms before swab results (74.32%, Figure 9c). The six most-cited symptoms and signs that were identified for the diagnosis of infections included increasing pain, increasing exudate, erythema/redness, odor, elevated temperature, and wound failure to progress or heal.

Improved criteria are needed to accurately diagnose increased superficial bacterial burden, as only 41.15% of respondents were confident concerning their current diagnostic criteria. In contrast, 50.72% were more confident in their abilities to accurately diagnose ***deep*** infected wounds. When directly asked if a better method of diagnosing bacterial damage was needed, 79.33% said yes, with about half stating that they would always (19.23%) or usually (39.42%) incorporate a bedside diagnostic tool for detecting superficial increased bacterial burden or deep and surrounding skin infected wounds into their practice.

Commonly ordered treatments for wound-related bacterial damage and infection included silver-based dressings (57.14%), systemic antibiotics (46.22%), other topical agents (33.63%), or other treatments (34.48%) (see Figure 10). When these treatments are ranked as ***effective*** (Figure 11), silver-based dressings were ranked highest at 64.38%, followed by systemic antibiotics (61.29%). Participants overwhelmingly correctly reported that the treatment for wounds with increased superficial bacterial burden differs from the treatment of deep- or surrounding-skin wound-associated infection (91.82%). **See clinical update on p. 15.**

Figure 10

Number of respondents indicating frequency of therapies used for treating infected wounds

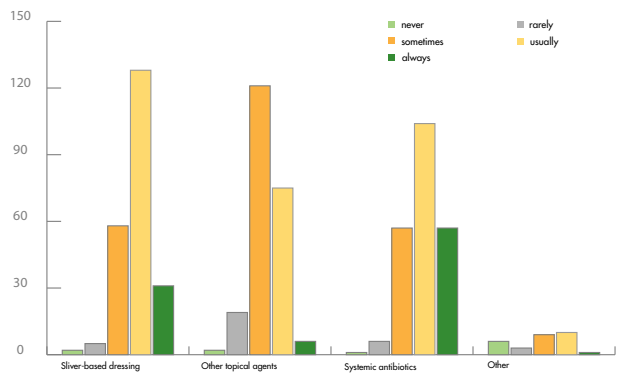
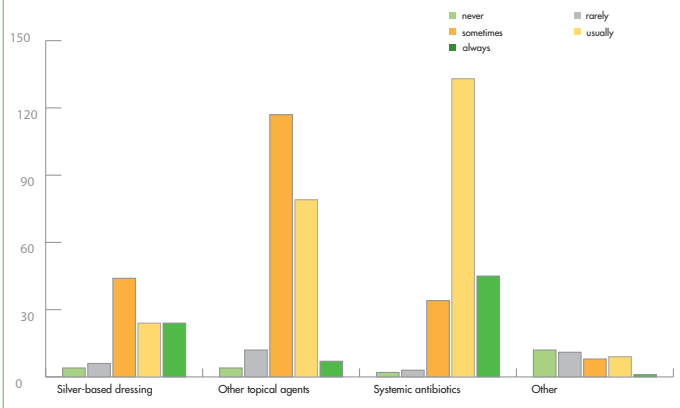


Figure 11

Number of respondents indicating the effectiveness of therapies used for treating infected wounds



Patient-Centered Concerns: Pain

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R. Gary Sibbald, MD, FRCPC (Med, Derm), MACP, FAAD, MEd, FAPWCA

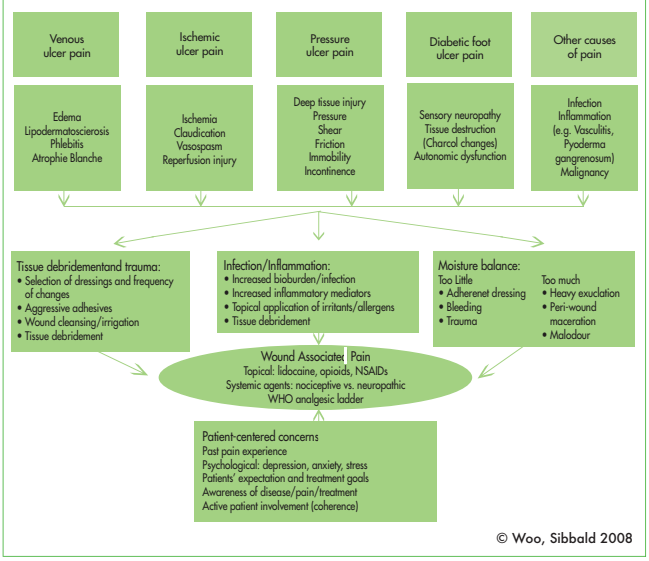
Pain is an under-recognized and undertreated component of chronic wound care. Persons with chronic wounds have both acute pain at the time of dressing change and persistent (chronic) pain between dressing changes. Affected individuals often experience a combination of stimulus-dependent nociceptive pain (gnawing, aching, tender, throbbing, or GATT), along with a spontaneous neuropathic component (burning, stinging, shooting, stabbing, or BSSS).[1]

The pain stimulus starts with free nerve endings that originate in the dermis and are distributed to the surface of the skin and its underlying structures. The perception of pain derives from the direct excitation of nociceptors in cutaneous free nerve terminals in response to trauma and tissue damage. Pain is a subjective, complex experience that involves the interplay of many physical sensory, emotional, motivational, social, interpersonal, and cognitive factors.[2] This conceptualization explicates the need to comprehend pain as more than a physiological phenomenon. The pain pathway involves a sensory electrical impulse that is conducted linearly from the peripheral pain receptors (nociceptors), ascends through the spinal pain pathways, and ultimately projects to higher centers in the brain where pain perception is constructed. Despite seemingly comparable levels of pain intensity, persons with pain experience have varying degrees of physical limitations, emotional distress and suffering.[3]

In studies[4–7] of chronic wounds, the majority of patients reported pain; many rated pain as moderate to the worst possible pain. Accumulating evidence confirms that unrelenting pain takes a central place in the experience of living with chronic wounds. Although the primary objective often is to heal the wound, patients with various types of chronic wounds consistently consider pain management the priority.[8–11] Prevailing opinion considers wound-associated pain to be a major source of stress and anxiety, contributing to psychological distress and eroding patients' quality of life.[12] Pain also may deleteriously affect wound healing through various mechanisms.[13]

Woo and Sibbald[1] developed a wound-associated-pain (WAP) model **(see Figure 1)** that incorporates several components contributing to the pain experience, including patient-centered concerns (eg, anxiety, depression, patient expectations), wound cause, and local wound care issues (tissue trauma, moisture balance, infection).

Figure 1: Chronic wound-associated pain (WAP) model: the wound, the cause, the patient



Pain Can Be Associated With the Cause of the Wound

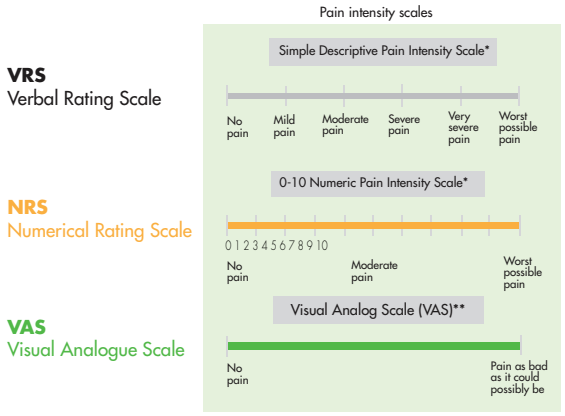
Patients with chronic wounds are vulnerable to the development of pathological pain due to conditions intrinsic to the underlying wound etiologies. The most common reasons for patients with venous disease to experience pain include acute lipodermatosclerosis, thrombophebitis, and swelling of the legs.[14,15] In a study of 758 patients with leg ulcers, Franks and Moffatt[16] demonstrated that the longer the duration of the ulcer, the higher the reported pain (P=0.022), suggesting a link between uncorrected acute pain and the emergence of chronic pain. Pressure-ulcer pain is often persistent unless pressure, shear, and friction are appropriately addressed. Even when patients have neuropathy, many patients experience spontaneous neuropathic pain in the absence of protective sensation.

Pain, Tissue Trauma, and Dressing Change

Although dressing changes are integral to wound management, associated local trauma may aggravate sensory fibers, causing pain. Up to 80% of nurses (n=225) noticed that patients experienced most pain during dressing change particularly during dressing removal.[17]

Patients often are concerned about pain upon dressing removal, which can occur when dressing materials have adhered to the wound bed due to dried-out materials, aggressive adhesives, abnormal granulation tissue with capillary loops growing into the product matrix, and the glue-like nature of dehydrated or crusted exudate.[18,19] Enzyme-rich exudate may spill onto the periwound skin, causing maceration and tissue erosion

Figure 2. Commonly Used Pain Assessment Tools



* If used as graphic scale, a 10-cm baseline is recommended
*** A 10-cm baseline is recommended for VAS scales.

Whatever scale is used-ensure same one used each time to provide true comparison

with a subsequent increased risk of trauma and pain.[20]

According to a review[21] of dressings and topical agents for secondary-intention healing of postsurgical wounds, sufficient evidence exists to show that patients experienced more pain with gauze than with advanced moisture-balanced dressings including foam, alginate and hydrocolloid dressings. Each time the dressing is removed, potential local trauma may evoke pain, perpetuating the inflammatory response with each dressing change. Despite the potential trauma to the wound base, gauze continues to be one of the most commonly used dressing materials, indicating a need to bridge research to practice.

Repeated application and removal of adhesive tapes and adhesive dressings can mechanically strip the stratum corneum on the skin surface from the epithelial cells. This can precipitate pain and skin damage.[22] In severe cases, erythema, edema and blistering have been observed.[23] Zillmer et al[24] evaluated potential skin damage from patches of adhesives based on hydrocolloid, polyurethane and soft silicone material. Dressings were applied to the forearms and peri-ulcer areas of 45 patients with open (n=29) and recently healed (n=16) venous ulcers. The adhesives were removed and replaced every second day for 14 days. After repeated removal and application of dressings, the investigators noticed a significant breach in the skin-barrier function (transepidermal water loss) or stratum corneum hydration (electrical conductance) with hydrocolloid dressings. Similar damage was not observed using other adhesives. Dressings that incorporated smaller amounts of adhesive or silicone coatings induced the least cutaneous irritation without compromising normal transepidermal water loss. Meaume et al[25] randomized 18 patients with Stage II pressure ulcers to either silicone dressing or polyurethane foam. Patients receiving silicone dressings displayed less wound edge and surrounding skin damage with decreased maceration and leakage. By limiting skin damage upon dressing removal, it is possible to minimize pain at dressing changes.

In considering pain as an outcome indicator, Dykes and Heggie[26] concluded that removal of a silicone dressing was less painful (n=24, $P<0.01$) compared to dressings that required higher peel force. However, results of studies on healthy volunteers with intact skin may not apply to patients with wounds and fragile periwound skin.

In addition to dressing removal, wound cleansing also is likely to evoke pain during the dressing change.[1,13,27]

Infection, Chronic Inflammation, and Wound Pain

Chronic wounds are invariably colonized by micro-organisms that are usually introduced by external contamination. Infection stimulates an inflammatory response, leading to a persistent influx of neutrophils. These polymorphic neutrophils not only compete for oxygen and nutrients, but also release damaging substances such as cytolytic enzymes, free oxygen radicals, and inflammatory mediators. Eventually, localized thrombosis and vasoconstricting metabolites lead to tissue hypoxia. These tissue-damaging events can promote a vicious cycle of ongoing bacterial proliferation and further tissue destruction. Early identification of wound superficial critical colonization and deep infection may facilitate healing. The diagnosis is best confirmed by documenting clinical signs and symptoms. Several researchers have proposed that the presence of unexpected or increased pain, along with other criteria, indicates a wound infection.[29,30] Gardner[30] reported that the specificity value for pain as a diagnostic criterion of wound infection was 1.00 (100%) in a small population of infected wounds. The mechanism linking infection to pain remains elusive but is thought to be via toll-like receptors (TLRs). TLRs are expressed by numerous types of immunoactive cells (leukocytes, and various nervous system cells). In response to infection, these receptors initiate a prompt responsive cascade of cytokine synthesis, facilitating the orchestration of the innate inflammatory and immune responses.

Assessment of Pain

Unfortunately, healthcare providers do not consistently assess and document pain. Lorimer et al[31] reviewed 66 nursing records of venous ulcer patients receiving home care. Only 15% of the records contained any documentation of pain, and the assessments were not standardized. Up to 55% of surveyed community nurses said they did not regularly assess pain in leg ulcer patients. Husband[9] sought to explore the community

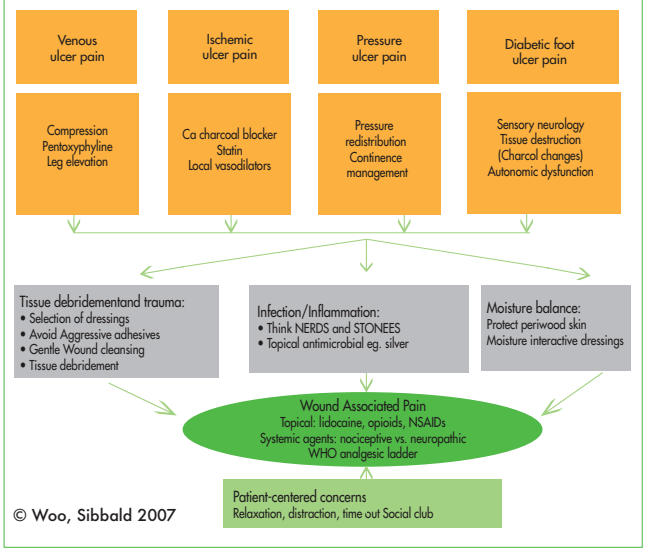
care provided for patients with venous ulcers. None of the interviewed 33 community nurses expected patients with venous leg ulcers to have significant pain.

Patients often express feelings of frustration at the failure of healthcare providers to mitigate wound pain. Pain intensity can be documented by a wide variety of self-reported pain scales. The selection of a specific pain scale must take into account a patient's age, language, educational level, sensory impairment, developmental stage and cognitive status. Ferrell et al evaluated five commonly used self-report pain-rating scales among people residing in nursing homes.[32] Although most subjects (83%) were able to indicate the presence of pain using any one of the scales, no one tool was deemed universally useful for all patients.

Regarding alternative approaches to pain measurement and documentation, studies have shown that the observation of nonverbal indicators encompassing a wide range of vocalized signals and bodily movements may provide a means of assessing pain in patients (eg, neonates or the cognitively impaired) who are not able to verbalize their pain.[33] However, many of the behavioral mannerisms may have idiosyncratic meaning to individuals, and only abrupt alterations in the identified behaviors may suggest pain.[34] Nevertheless, once chosen, the same measurement scale should be used for subsequent assessments to ensure consistent ongoing comparison. Changes in pain levels may indicate a need to reassess the choice and timing of analgesics and/or other interventions used in pain management.[35]

In any case, all pain assessments should be well-documented to facilitate the continuity of patient care. The key elements of a holistic pain assessment may include the pain type (nociceptive, neuropathic or mixed), duration of pain (chronic versus acute), severity/intensity, impact of pain on the patient, expected levels of pain relief, and identification of treatment-related adverse effects in order to reduce their impact.[36] The personal interpretation of the pain experience, anxiety, and anticipation of pain also should be addressed.

Figure 3: Chronic wound-associated pain (WAP) model: the wound, the cause, the patient



Pain Assessment Tools

Several validated pain assessment tools are available for clinical practice **(see Figure 2).**

The visual analogue scale (VAS) is the most commonly used instrument to evaluate pain with ratio-scale property. It usually consists of a continuous 100-mm vertical or horizontal line between of "no pain" and "worst pain" to represent the two extremes of pain intensity. To help patients conjure the worst scenario, the worst pain is often described as "the most intense pain imaginable" or "the worst pain experienced," providing the individual has had some horrific pain experience with which to compare. Patients are asked to mark on the VAS line nearest the descriptor that best represents their pain intensity.

The numeric rating scale (NRS) is similar to VAS, except that the number calibration 0 through 10 is displayed on the line in order and at equal intervals. The NRS has low verbal and high numeric features.[37,38] To compensate for this, anchor words and a color spectrum also may be incorporated. Patients are asked to select the number that best describes the intensity of their pain at the present time. In general, pain is classified as mild (1 to 3), moderate (4 to 7), or intense (8 to 10) by their corresponding numeric values. Because the NRS is concrete and easy to interpret, it is preferred by individuals across all age groups.[39,40] Clinicians prefer the NRS because it offers an immediate score for a dynamic concept like pain without extra calibration and calculation.[41] The minimum clinically significant VAS/NRS pain score reported in studies not related to wound care was 1.4.[42,43] Up to 87.9% of venous leg ulcer patients were able to assess pain using the NRS, compared to 75% using VAS.

The VAS and NRS are one-dimensional simple instruments that are easy to administer and score. They have been used in a variety of patient populations, including geriatric patients.

The verbal rating scale (VRS) is an ordinal pain scale made up of word descriptors of pain: none, mild, moderate, and severe. Patients may find the wording too limited and often describe pain in their own terms, such as "a little bit," "some," "bad but not severe," or "somewhere between mild and moderate."

For persons with low verbal comprehension, the Faces descriptive scale (FDS) has been introduced to document pain. The FDS

Table 1: Pharmacological Pain Management		
	Neuropathic pain	Nociceptive pain
1st line of use	TCA antidepressants: amitriptyline, nortriptylene, desipramine	ASA, NSAIDs, or acetaminophen
2nd line of use	Anticonvulsants: gabapentin/pregabalin	Weak opioids, eg codeine
3rd line of use	SSRI antidepressant: duloxetine, venlafaxine Anticonvulsants: carbamazepine, sodium valproate	Strong opioids, eg morphine

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consists of a series of faces, each depicting different facial expressions from smiling to crying, that represents increasing degrees of pain. The FDS has been criticized because it may be difficult to distinguish and determine if pain or emotional state is being measured. To enhance the validity of the scale, the affective qualities including the smile and tears have been removed.

Pain has several dimensions that include both its severity and quality. To capture the complexity of pain, multidimensional tools may be considered. The Short Form McGill Pain Questionnaire (SF-MPQ) is one of the most commonly used multidimensional pain measures. It contains 11 word descriptors for the sensory qualities of a pain experience and four related to the affective dimension. Word descriptors on the SF-MPQ have been used to evaluate neuropathic pain in numerous studies. Nociceptive pain (response to injury) often is described as GATT, while neuropathic pain is more likely to be BSSS. The SF-MPQ has been used to evaluate the characteristics of pain during wound care.

Pharmacological approach to wound-associated pain

The treatment of pain can be conceptualized holistically using the wound bed preparation model modified to include the potential causes of wound-related pain **(see Figure 3).**

Using an experimental design, Jorgensen et al[44] demonstrated that subjective evaluation of well-being can significantly be improved with the reduction of pain ($P<0.0001$). Wound-related pain is often underestimated and undertreated. Hofman et al[45] reported that only half of patients with venous leg ulcer and severe pain received appropriate morphine-based analgesia. Clinicians' knowledge of pain management is often deficient. Nevertheless, pharmacotherapy continues to be the key strategy to manage wound-related pain. Depending on the severity of pain, the choice of pharmacological agents for pain should adhere to the recommendations by the World Health Organization (WHO) **(see Table 1)**. Most patients with mild-to-moderate pain respond favorably to oral acetylsalicylic acid, non-steroidal anti-inflammatory drugs (NSAIDs) or acetaminophen. Caution must be exercised in people older than 65 years, for whom oral NSAIDs may be associated with an increased risk of GI bleeds or renal compromise, or the aggravation of congestive heart failure. As pain intensifies, weaker and then stronger opioid analgesics should be utilized. Neuropathic pain is common in patients with chronic wounds due to damage and irritation to nerves.

Specific agents should be selected to address neuropathic pain. Titrate the medication while monitoring therapeutic responses (using a standardized tool) and consider potential adverse effects when working to achieve the therapeutic objective.

Briggs and Nelson[46] reviewed literature that pertains to topical interventions for pain in patients with venous leg ulcers. Strong evidence corroborates the use of an EMLA cream (a eutectic mixture of lidocaine and prilocaine) for the debridement of venous leg ulcers. None of the reviewed trials evaluated persistent wound pain.

Since that meta-analysis was published, new evidence has emerged from randomized controlled trials that demonstrates the effectiveness of ibuprofen-releasing foam for treating persistent and temporary acute wound pain with promising results.[47-49] This dressing is not licensed for use in the United States, but has been released in Canada and Europe. The use of topical morphine offers another option for pain management. Although pain improvement was indicated in several studies of patients with chronic wounds, this formulation is not commercially available; the lack of pharmacokinetic data has precluded the routine clinical use of this compound at this time.[50-53]

Non-Pharmacological Approach to Wound-Associated Pain

Common non-pharmacological pain management strategies aim to reduce the potential effect of psychological factors such as anxiety and stress that may aggravate the pain experience. These techniques include relaxation, music therapy, touch therapy, visual stimulation, hypnosis, stress-reducing strategies, guided imagery, behavioral and cognitive therapy, and distraction.[54-59] However, little evidence supports their relative effectiveness in the chronic wound population.[60] Other complementary therapies for pain control include transcutaneous electrical

nerve stimulation (TENS), acupuncture, massage, laser therapy, and thermal therapy. These techniques' clinical utility in managing wound-related pain is dubious due to the paucity of wound pain-related research concerning these adjunctive therapies.

Pain management may be efficacious if other personal and social factors, such as anxiety and depression, that may cause variability in pain perception are addressed. Patients with chronic wounds often expressed feelings of powerlessness, depression, and social isolation. Only a small proportion of patients are cognizant of factors contributing to chronic wounds and strategies to improve their conditions.[61] Inadequate information and healthcare provider misconceptions regarding pain pose barriers to effective pain management.[62] These misconceptions often seriously hinder optimal pain management and should be addressed while maintaining sensitivity to patient beliefs. Patients are reluctant to report pain and take medications due to fear of addiction and side effects from analgesia. Culturally, some individuals believe that "good patients" do not complain about pain and that healthcare providers are too busy to manage their pain.[62,63] Some individuals perceive pain as unavoidable and integral to growing old, perpetuating a sense of helplessness and hopelessness about pain. Effective communication and education let healthcare professionals and patients establish credible therapeutic relationships. Caregivers need to reinforce the belief that chronic wound patients do not have to live with persistent or temporary pain and foster their active participation in the assessment, treatment, and coping behaviors.[64]

A therapeutic relationship between healthcare provider and patient can enhance treatment adherence to optimize patient outcomes.[65,66] In a study of 56 patients with venous leg ulcers, Edwards et al[67] evaluated the impact of a community leg club model of care on wound pain and healing.[67] Subjects were randomized to receive individual home visits from community nurses (the control group) or to pay weekly visits to a nurse-managed leg club (the intervention group). The leg club offers a setting in which patients can receive ulcer-management advice and information via social interaction with their peers and expert nurses. Patients who attended the leg club expressed significant reductions in the amount of pain experienced ($Z=3.02$, $P=0.001$) after 12 weeks. The control group's pain ratings did not improve. Education is another strategy to empower patients and to improve wound-related-pain control.[67] In a pilot study, five patients with chronic wounds found dressing pain more manageable after receiving educational information.[88]

Patient Bill of Rights

Each person with pain needs to have a sense of control and empowerment **(see Patient Bill of Rights above)**.

Patient Bill of Rights

- Check
- Cause
- Consequences
- Control
- Comfort
- Chart
- Call timeouts

Healthcare providers should check with patients for the presence of pain and recheck at regular intervals. The first step to treatment is to determine the components causing pain **(see Figure 1)**. Clinicians must also realize that pain impedes on quality of life and actitivities of daily living for patients, and that these factors need to be acknowledged and, where possible, addressed as part of a treatment plan.

Patients also are entitled to call timeouts when procedures are painful, and to receive medication to provide comfort from pain and suffering for both persistent pain and the acute pain at dressing change. The pain relief toolkit should include both pharmacological and non-pharmacological treatment options.

Caregivers need to chart the character and intensity of wound-related pain to monitor changes and recommend treatment. Standardized validated pain assessment tools should continually monitor the treatment outcome with the results charted to document change.

Conclusion

Pain is a common symptom for persons with chronic wounds. The totality of the pain experience continues to generate conceptual and methodological discourse due to the vicissitudes of this affliction. However, pain is whatever the patient says it is. The troublesome pain symptom may evolve from one or more sources, including wound etiologies and local wound care such as surgical debridement procedures or dressing change. Sub-optimal local components of the wound base that may be associated with pain include unwanted debris, bacterial damage from superficial critical colonization or deep compartment infections, abnormal inflammation, or moisture imbalance (either excess or paucity). It is crucial to remember that chronic wound-related pain is linked to abnormal wound characteristics but also to human suffering. Pain has a tremendous impact on an individual's quality of life, emotional state, and sense of well-being. To improve the lives of individuals with chronic wound-related pain, new pain-related knowledge must be translated into the treatment of the affected patients' physical and emotional states. ■

References available in convenient downloadable format online at www.o-wm.com/supplements/0409molnlyckecharts.pdf

Healing Chronic Wounds:

DIM Before DIME Can Help

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The wound bed preparation (WBP) model was created as a practical clinical guide for the optimal treatment of chronic wounds (**see Figure 1**).[1-3] Clinicialns should always put patients first (addressing patient-centered concerns), then treat the cause using the most advantageous local wound care. The initial components of local care are debridement, infection/inflammation, moisture balance (DIM). The E (edge effect) in DIME often presents clinically as a cliff-like wound edge of a healable wound that is stalled or not healing at the expected rate.[4] Active agents (biological, growth factors, skin substitutes, adjunctive therapies) are effective only if the wound bed has been appropriately prepared following all the steps in **Figure 1**.

Starting the Wound Healing Journey

Accurate wound diagnosis and correcting the cause are important first steps in the wound healing journey. For example, to approach a diabetic foot ulcer, think VIPS: Vascular supply is adequate; infections (both superficial increased bacterial burden and deep- or surrounding-skin infection) are controlled; plantar pressure is redistributed; and, if the wound is healable, sharp surgical debridement is performed.

Venous leg ulcers require bandaging for healing and stockings for maintaining the wound and preventing recurrences. High-compression bandaging is indicated for ankle-brachial pressure index (ABPI) >0.8; modified/lower-compression should be considered for ABPI between 0.6 and 0.8. Pressure ulcers would benefit from an assessment of lying (eg, bed and mattress) and seating surfaces (eg, wheelchair) to ensure appropriate pressure management. Other issues to address may include friction and shear, excess moisture (urinary and fecal incontinence), malnutrition, and immobility.

To following factors must be considered to determine the wound healability **(see Table 1):** whether the cause is treatable, blood supply adequacy, and if co-existing conditions or drugs prevent or delay healing.[3]

Moist interactive healing is contraindicated in nonhealable wounds. The care plan should include conservative debridement without cutting into living tissue and causing bleeding; bacterial reduction; and moisture reduction. When healing is not immediately possible, such as in cases of uncontrolled deep infection or where bacterial burden was more of a concern than tissue toxicity (maintenance or non-healable wounds), antiseptics including povidone iodine, chlorhexidine and their derivatives are good treatment options. Each of these agents has relatively low tissue toxicity compared to other commonly used antiseptic agents (sodium hypochlorite, various aniline dyes, quaternary ammonia compounds or hydrogen peroxide). The following articles will review the DIME components individually. ■

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Figure 1

Wound Bed Preparation and DIME

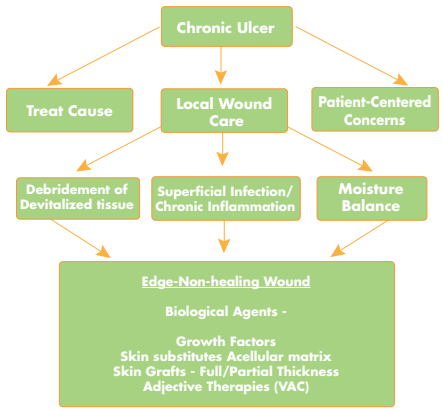

Table 2: Determination of Realistic Outcomes

Wound prognosis	Treat the cause	Blood supply	Co-existing medical condition/drugs
Healable	Yes	Adequate	Does not prevent healing
Maintenance	No	Adequate	+/- prevent healing
Nonhealable	No	May inhibit healing	May inhibit healing

D — Debridement

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Debridement is a crucial step to in preparing the wound bed; it removes devitalized and damaged tissue, such as firm eschar or slough, that may act as a pro-inflammatory nidus or growth media for bacteria.[1] Potential additional benefits of debridement — such as removing senescent sells that are deficient in cellular activities — have generated much debate.[2] Debridement disturbs bacterial biofilms, rendering bacteria more susceptible to antimicrobial treatment; tapers sharp slopes from the wound edge to facilitate keratinocyte migration; de-roofs skin over undermined edges to expose the wound margins; and creates an acute wound within a chronic wound to trigger the cellular cascade that facilitates healing.

The decision to perform debridement should depend on whether the wound is considered healable, nonhealable, or maintenance. Aggressive debridement to the vascular layer is not recommended in nonhealable wounds in which causative factors that preclude healing cannot be addressed. Under judicious deliberation, conservatively debriding nonhealable wounds — trimming loose-hanging fibrin to reduce necrotic mass and associated odor — may be appropriate. The purpose of conservative debridement is to enhance quality of life and decrease the risk of infection but not to facilitate healing. For healable wounds, different debridement methods have been described: mechanical (wet-to-dry dressing), enzymatic, autolytic (endogenous enzymes from dressings), biological (maggots), and sharp/surgical. Where appropriate, the choice of debridement method depends on the urgency, resources or expertise available, and patient preference[2] **(see Table 1)**.

Sharp Surgical Debridement

Sharp surgical debridement is the most expeditious method to remove unwanted tissue. It employs sharp instruments such as scissors, scalpels, and curette at the bedside or in the operating room, when extensive debridement and subsequent blood loss is anticipated. In persons with diabetes, sharp surgical debridement of hyperkeratosis and callus can reduce overall peak plantar pressure by as much as 29%.[3] A study of platelet derived growth factor in the treatment of neuropathic foot ulcers revealed that 83% of patients achieved healing with active surgical debridement, in contrast to the 20% healing rate in patients with suboptimal debridement.[4] The results strongly suggest "wound debridement is a vital adjunct in the care of patients with chronic diabetic (neuropathic) foot ulcers."[4] Although other research findings corroborate the therapeutic value of sharp debridement (especially for diabetic neutrophic foot ulcer), this debridement method may not always be feasible due to pain, bleeding potential, cost, and lack of clinician expertise.

Mechanical Debridement

Mechanical debridement requires physical forces to remove or dislodge wound debris. The wet-to-dry dressing technique is probably one of the most popular forms of mechanical debridement. As the wet (saline) dressing dries up, necrotic materials that are adhered to the structure of the dressing fabric are pulled and removed with force. This creates pain and trauma, hindering the healing process. Hydrotherapies such as whirlpool, pulsatile lavage, and irrigation are available but warrant extra precautions to limit cross-contamination and aerosolizing bacteria droplets. The force of irrigation should be limited to between 8 to 12 psi to avoid excessive trauma to wound surface.[5]

Autolytic, Enzymatic, and Biological Debridement

Autolytic debridement removes non-viable tissue through promoting the activities of phagocytic cells and endogenous enzymes. Smith reviewed randomized controlled clinical trials (RCTs) pertaining to debridement of DFUs.[6] Combined results from three RCTs suggest hydrogels are superior to gauze or standard care to assist tissue autolytic debridement and healing in DFUs. Enzymatic debridement adds exogenous agents such as proteolytic enzymes, including collagenase (this is the only enzyme available in the US), streptokinase, streptodornase, trypsin-chymotrypsin, papain/urea, and plant enzymes (eg, fig, pineapple) to accelerate the debridement process.[7-10] De-sloughing may be hastened by lowering wound pH, but using organic and inorganic acids and other chemical agents is discouraged in light of the potential for pain and tissue toxicity.

Biological debridement — inoculating larva that feed on necrotic tissue and exudate in a wound — is gaining popularity. Larva secretions contain proteolytic enzymes, antibacterial substances, and growth factors that purportedly facilitate wound healing. However, there is no evidence to indicate that maggot therapy is superior to other forms of debridement.[11] Despite the potential benefits of maggot therapy, this therapy is not innocuous, and local painful reactions are not uncommon.[12,13]

Table 1: Key Factors for Deciding Method of Debridement

Factor	Surgical	Enzymatic	Autolytic	Biological	Mechanical
Potential pain	+++++	++	+	+++	++++
Speed	+	+++	+++++	++	++++
Tissue selectivity	+++	+	++++	++	+++++
Exudate	+	++++	+++	+++++	++
Potential infection	+	++++	+++++	++	+++
Cost	+++++	++	+	+++	++++

+ indicates lowest and +++++ highest

Emerging evidence suggests that novel debridement methods may improve practice for selected patients. These methods include applying acoustic energy or ultrasonic sound waves. However, after reviewing the relevant literature, Ramundo and Gray[14] concluded that the evidence does not sufficiently substantiate the claim that ultrasonic devices adequately remove necrotic tissue from chronic wound surfaces. Introducing a high-pressure jet of sterile saline (Versajet Hydrosurgery System, Smith & Nephew, Largo, FL) excises and aspirates the unwanted tissue, but the depth of tissue removal is controlled by the direction and force of the jet stream, which could cause undesirable damage or remove healthy granulation in the wound base. However, used properly, this technology is effective and has been demonstrated to curtail debridement time significantly.[15] Cost and the potential need for an operating room (instead of the ambulatory clinical setting) hinders acceptance of these modalities into some wound care practices.[16] There is also a need for studies comparing efficacy and cost-effectiveness of these newer therapies with other types of debridement. ■

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I — Infection and Inflammation

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Wound-related bacterial damage and infection can result in poor wound healing and other adverse patient outcomes. Several studies suggest wound infection is one of the major risk factors that heralds amputations in patients with diabetic foot ulcers. Length of hospital stay and mortality are significantly increased in surgical patients who had wound infection: the mortality rate is more than 50% in patients who have bacteremia secondary to pressure ulcers.[1]

What Is Wound Infection?

All chronic wounds contain bacteria, so bacteria's presence on a surface swab does not define infection. In fact, the mean number of bacterial species per chronic ulcer has been found to range from 1.6 to 4.4.[2] Bacterial presence is not necessarily pathological but may actually facilitate normal healing.[3] Critical to wound healing is whether bacterial balance is maintained (contamination or colonization) or bacterial damage (critical colonization or infection) has occurred. In brief, all chronic wounds are contaminated, with bacteria on their surfaces. As the attached bacteria proliferate, colonization is established. With a decrease in host resistance, bacteria can cause local tissue damage in the superficial wound compartment. This phenomenon is referred in the literature as critical colonization, increased bacterial burden, covert infection, or localized infection. When the bacteria invade and damage the surrounding and deeper structures in the wound base, the classical signs and symptoms of infection are produced.

The risk of infection is determined by the number and nature of invading bacteria as well as host resistance as outlined in the following equation:

$$\text{Infection} = \frac{\text{Number of organisms} \times \text{Organism virulence}}{\text{Host resistance}}$$

In this equation, host resistance is the most important factor; it refers to the host's immune response to resist bacterial invasion and ability to prevent bacterial damage.[2]

How to Diagnose Wound Infection

The assessment of infection in a chronic wound is a clinical skill and the decision to prescribe antibiotics or apply topical antimicrobial agents should be based primarily on clinical presentations **(see Table 1)**. The clinical value of a culture is to identify multiresistant bacteria such as methicillin-resistant *Staphylococcus aureus* (MRSA) and determine the sensitivity of bacterial organisms associated with clinical infection.

To minimize contaminants, a swab specimen should be obtained using the recommended Levine's technique.[4] First, the wounds should be irrigated with normal saline until excess visible debris has been washed away. This is followed by rotating the swab tip in a 1-cm^2 area of the cleanest part of the wound, preferably in an area of granulation. A certain amount of pressure is used to extract tissue exudate for successful culturing. The swab then is rotated 360° and placed in the transport media.[4] The swabs then should be sent to local laboratory for processing in a timely fashion. In a study of patients with predominant diabetic foot ulcers, Gardner et al[5] examined the qualitative bacteriology analysis and reported a high concordance rate of 78% between swabs (obtained with Levine's method) and deep-tissue biopsies.

Signs Associated With Wound Infection: NERDS and STONEES

Wound bacterial damage could be divided into superficial and deep component. Validated signs of increased surface bacterial burden and deep infection are represented by the letters in the mnemonics NERDS and STONEES, respectively **(see Tables 2 and 3)**. No one individual sign or symptom accurately confirms the diagnosis of wound infection, but a combination of two or three of these signs can signal diagnosis in each level.[3,6] By focusing on salient clinical signs to separate superficial and deep compartment involvement, the clinician can consider therapeutic options that are most appropriate and cost-effective.

Osteomyelitis

Osteomyelitis should be suspected if ulcers probe to bone (sensitivity equals 38% to 87%; specificity equals 85% to 91%; positive predictive value equals 53% to 89%; negative predictive value equals 56% to 98%).[7-9] Additional bloodwork (eg, erythrocyte sedimentation rate and C-reactive protein) and radiographic assessment (eg, bone scan and X-ray) may be beneficial, although false positives are common.

Treating Wound Infection

Superficial colonization: The superficial compartment extends approximately 1 mm to 3 mm below the wound surface. Some bacteria will critically colonize the superficial, relatively hypoxic wound environment, but not all species have the virulence to invade the deep compartment. Cleansing solutions, saline, and

Table 8: Topical Agents

Agent	Vehicle	S. aureus	Streptococcus	Pseudomonas	Anaerobe	Comments
- Gentamicin sulphate - Cream/ointment	Alcohol cream base or petrolatum ointment	√	√	√		- Good broad spectrum vs. gram negatives - Topical use may ↑ resistance
- Metronidazole gel/cream/solution	Wax- glycerin cream and carbogel 940/propylene glycol gel				√	- Good anerobe coverage and wound deodorizer
- Mupirocin 2% cream ointment - Nasal ointment	Polyethylene glycol (ointment) Paraffin/glycerine (nasal ointment)	√	√			- Good for MRSA - Excellent topical penetration - Used predominantly perirectal, nasal colonization
- Polymyxin B sulphate - Bacitracin zinc	White petrolatum ointment	√	√	√	√	- Broad spectrum - Low cost
- Polymyxin B sulphate - Bacitracin zinc-neomycin	White petrolatum ointment	√	√	√	√	- Neomycin is a potent sensitizer and may cross react with other aminoglycosides in 40% of cases
- Polymyxin/ Gramicidin	Cream	√	√	√		- Broad spectrum coverage
- Silver sulfadiazine (SSD)	Water-miscible cream	√	√	√	√	- Do not use in sulpha sensitive individuals/ - Short half life Neutropenia is possible - may leave pseudoeschar on wounds

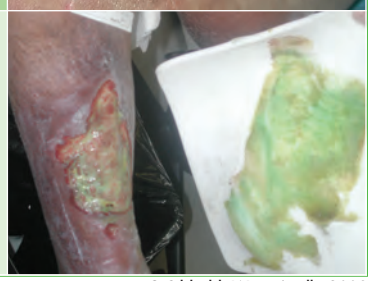
References and tables 1, 4, 5, 6, and 7 are available in convenient downloadable PDF format: www.o-wm.com/supplements/0409molnlyckecharts.pdf

water are usually recommended to remove surface debris and becteria because of their low tissue toxicity. New topical antimicrobial products containing chlorhexidine and iodine are readily available and less toxic **(see Tables 5 and 6)**. Silver dressings are popular, but silver requires moisture to be activated and released into the tissue to exert its antimicrobial effect **(see Table 7** for other considerations**)**. Alternatively, bacteria can be entrapped and sequestered in the micro-architecture of a dressing, where they may be inactivated.

For deep- and surrounding-wound infection, systemic agents **(see Table 4)** are usually recommended, as topical agents are not able to penetrate into the deep compartment. An effective topical antimicrobial **(see Table 8)** may still be considered to eliminate the bacteria that percolate to the superficial compartment where the circulation may be less than optimal. Lastly, if the infection is promulgated systemically, systemic agents must be considered. Systematic reviews[11] of trials to evaluate the effectiveness of antimicrobial treatment for diabetic foot ulcer fail to support the superiority of any intravenous or oral antibiotic regimen over others. Prophylactic antibiotics have not been demonstrated to facilitate wound healing.[10]

Table 2: NERDS

NERDS: *Any three or more indicate critical colonization; use topical therapy including silver dressings*

		Definition/defining features	
N	Nonhealing wound	• Wounds that are not 20% to 40% smaller in 4 weeks according to patient's history or existing documentation • Bacteria compete for nutrients and oxygen essential for wound healing activities. Bacterial exotoxins and endotoxins are generated and diffused into the wound milieu impairing normal cellular functions • Bacterial toxins have been proposed to adversely affect the normal collagen deposition and cross-linking potentially contributing to surgical wound dehiscence	
E	Exudative wound	• An increase in wound exudate can be indicative of bacterial pro-inflammatory damage and leads to periwound maceration • More than 50% of the dressing was tainted with exudate • Bacterial leukocidins, phospholipases, and toxins rapidly destroy neutrophils, producing watery exudate • Infections consisting primarily of Gram-positive organisms typically have purulent exudate	
R	Red and bleeding wound	• The wound bed tissue is bright red with exuberant granulation tissue • Bacterial stimulation of vascular endothelial growth factor (VEGF), resulting in excess formation of weak blood vessels, and tissue is easy to bleed even with gentle manipulation • A poor quality of collagen matrix formation from reduced fibroblast activity and ongoing thrombosis of larger vessels in infected wounds renders the granulation less resilient to trauma	
D	Debris	• The presence of discolored granulation tissue, slough and necrotic/nonviable tissue	
S	Smell from the wound	• Unpleasant or sweet, sickening odor • Putrid discharge is attributed to products of bacterial metabolism including volatile fatty acids, (propionic, butyric, valeric, isobutyric and isovaleric acids), volatile sulphur compounds, putrescine, and cadaverine	

Download references and tables 1, 4, 5, 6, and 7: www.o-wm.com/supplements/0409molnlyckecharts.pdf

Table 3: STONEES

STONEES: *Any three or more indicate deep infection, which requires oral or parenteral agent*

		Definition	
S	Size is bigger	• Wound size is increasing • Size as measured by the longest length and the widest width at right angles to the longest length. Only very deep wounds and most pressure ulcers Stage III and Stage IV need to have depth measured with a probe • Bacteria produce specific proteolytic enzymes such as invasins that attack protein and extracellular matrix components	
T	Temperature increased	• Increased periwound margin temperature by more than 3° F difference between two mirror-image sites • Temperature differences may be affected by the presence of deep structural damage, giving false elevation or impaired circulation, masking the presence of infection	
O	Os (probes to or exposed bone)	• Wounds that had exposed bone or that probed to bone	
N	New areas of breakdown	• New areas of breakdown or satellite lesions	
E	Erythema, edema	• Reddened skin in the periwound area • Presence of swelling in the periwound wound area • Increased amount of drainage	
E	Exudate	• Reddened skin in the periwound area • Presence of swelling in the periwound wound area • Increased amount of drainage	
S	Smell	• Unpleasant or sweet, sickening odor • Clinicians need to differentiate the smell of bacterial damage from the odor associated with the interaction of exudate with different dressing materials, particularly some hydrocolloids	

M — Moisture Balance

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Avoiding too much or too little moisture in the wound is essential to all phases of wound healing. The surface of a chronic wound should encourage cellular proliferation and migration. However, excess moisture can be deleterious to wounds. Numerous studies have established that the presence of a moist wound environment

- accelerates the re-epithelialization process,
- facilitates the action of growth factors,
- increases keratinocyte and fibroblast proliferation,
- enhances collagen synthesis, and
- promotes angiogenesis and early wound contraction.

Increased exudate may be produced during the inflammatory phase of wound healing due to alteration in capillary permeability, vasodilatation, and migration of the inflammatory cells. Exudate is slowly discharged from injured cells in the bloodstream through small pores in the capillary walls. Chronic wound exudate contains high levels of oxidative enzymes, leukocytes, cytokines, and proteases (eg, matrix metalloproteinases). This enzyme-rich and caustic exudate may spill over to wound margins causing maceration or epidermal erosion (ie, loss of part of the epidermis, but maintenance of an epidermal base), and pain.[1] As a result, the excess exudate damages periwound skin and retards healing.

Therefore, it is important to remove excess exudate from the wound with an absorbent dressing. A plethora of dressings have been developed to maintain moisture balance. Major categories of dressings include foams, alginates, hydrofibers, hydrogels, and hydrocolloids.

Foams

Polyurethane foam dressings are designed to wick up a large volume of exudate. These dressings are non-bioresorbable, with cells arranged like a honeycomb with an air-filled center and polyurethane wall. For convenience, foams can be divided into first-, second- and third-generation products.

First-generation foams are characterized by uniform pore size (like a sieve) that cannot lock in fluid. When they are compressed or oversaturated, the fluid is released back to the wound surface or spilled over to the periwound skin, causing maceration.

A second-generation foam has variable pore sizes; these foams have superior fluid management and trap moisture in the smaller pores, while certain amount of fluid is donated back to the wound from the larger cells, maintaining a moist wound environment. This type of foam is more likely to retain fluid under compression and is much less likely to cause periwound maceration. The fluid-handling capacity of various foams also can be affected by the polyurethane film backing's ability to transfer moisture vapor out of the dressing, but form a barrier to bacterial contamination (called moisture vapor transmission rate). The surface of the foam in contact with the wound may be plain (no coating) or contain acrylic adhesives or soft silicones to promote adherence to the wound base and surrounding skin.

Third-generation foams have appeared on the marketplace; first- or second-generation foam dressings are now used as the delivery vehicle for silver and other local agents to treat the superficial wound compartment.

Depending on the level of wound exudate, foams have a wear time of 1 to 7 days. Most foams can be cut to the wound size if maceration is a problem (except for boarder products). Additional slits also can be created on the upper surface to allow extra wound fluid to be wicked into a superabsorbent product or abdominal (ABD) pad.

Alginate and Hydrofibers

Alginate and hydrofibers are also able to handle copious exudate while their gelling effect keeps the wound base moist.

Alginates. Alginates are derived from kelp or brown seaweed and converted to a fiber when combined with calcium. Manufacturing techniques can produce the fibers as a wafer or pad that has lateral wicking or as a rope that presents more vertical wicking of wound fluid. Depending on the species of kelp from which the alginate is extracted, there may be higher concentrations of either manuronic acid or galuronic acid. A high concentration of manuronic acid in calcium alginate renders the dressing material more gel-like, while a higher concentration of galuronic acid increases tensile strength. Calcium alginate often is used post-debridement for its hemostatic property. The hemostatic cascade is triggered as the calcium ion is released from the dressing in exchange with sodium present in the wound fluid. As a natural product, calcium alginate is biodegradable and can disappear completely between dressing changes. If the wound is producing a scant amount of exudate, the alginate fiber may adhere to the wound base, and a hydrogel may be a more appropriate choice. Compared with foams, calcium alginates are less absorptive, but they are able to act as autolytic debriders. Dressings with alginates usually are changed daily to three times weekly.

Hydrofibers. Hydrofibers are carboxymethylcellulose strands available in pads or packing materials. They have a water-hat-

Table 1: Comparison of Dressing Materials

Recommended Dressings & Topical Agents for Diabetic Foot Ulcers	Debridement	Bacterial Balance	Moisture Balance	Comments
Foam (Mepilex, Mepilex Border, Biatain, Allevyn)	None	No direct effect	Absorbent ++++	• Not bioresorbable • Fluid exchange • Variable adherent contact surface
Hydrofiber (Aquacel)	None	May trap bacteria	Absorbent ++	• Not bioresorbable • Fluid lock
Calcium Alginate (Melgisorb, Kaltostat)	Autolytic	No direct effect	Absorbent +++	• Bioresorbable • Local hemostasis
Hydrogel (Normlgel, Hypergel, Intrasite gel, Duoderm gel)	Autolytic	No direct effect	Absorbent +	• Good for autolytic debridement with dry wounds
Films (Mefilm, Tegiderm, Opsite)				

ing component (hydrophobic methylcellulose) that gives the dressing its tensile strength as well as a water-loving component (hydrophilic carboxy) that acts as a fluid lockAs the dressing absorbs fluid, the hydrofiber consistency converts to a gel, weakening its tensile strength. Hydrofibers with lyocel stitching are preferred for undermining and packing into deep sinuses and tunnels,as this non-bioresorbable material may break and be left behind in these dead spaces. Similar to calcium alginates and hydrogel, dressing changes are generally made daily to three times a week.

Hydrogels

Hydrogels are usually indicated for relatively dry wounds or autolytic debridement. The major ingredient is water (70% to 90%) that donates moisture into the wound base. Hydrogels have different backbones that include saline (eg, Hypergel, Normlgel), propylene glycol (eg, Intrasite), hydrocolloids (eg, Duoderm gel), alginate (eg, Nugel) and collagen (eg, Woun'Dres). These materials, available in amorphous and wafer formats, are excellent autolytic debriders and preserve moisture balance largely by donating moisture to the wound surface.Their "tack" provides the ability to adhere to the applied surface, increasing viscosity compared to water. In amorphous form, the dressings help stimulate granulation tissue; in wafer form, they can maintain surface moisture to support re-epithelialization.

Hydrocolloids

Hydrocolloid dressings consist of carboxymethylcellulose as the filler, water-absorptive components such as gelatin and pectin (commercial gelatin desserts), and an adhesive.These dressings can promote moist interactive healing and autolytic debr idement with an antibacterial effect (low pH).[3] Hydrocolloids are designed for wear times of 1 to 7 days, and for this reason their absorbency is lower than foams or calcium alginates, but similar to that of hydrogels. Common concerns include odor, strong peel force to remove the dressing, and residue left on the skin surface, as certain hydrocolloids have a tendency to melt into the skin. Occasional allergies have been reported from some colophony-related adhesives (Pentylin H) associated with some hydrocolloid dressings.[4] The utility of this class of dressing has improved by using a polyurethane film or foam-like material for the backing.

Films

Film dressings often are used for local protection.The choice of a nonadherent (no adhesive) versus a film with adhesi ve backing should be determined by the fragility of the skin. Adhesives may be responsible for skin tears due to the tear for ce on dressing removal. Caution is required with patients who are on steroid therapy and have a recently healed wound that only attains 20% of the original tensile strength of skin. Film materials are semi-occlusive with various degree of permeability that allows water molecules to pass through the dressing and evaporate into the ambient environment at a variable rate, depending on the water-vapor transmission rate. These dressings are designed to handle minimal amounts of fluid.

Evidence Base for Moist Interactive Healing

Despite the availability of different types of dressings, previous systemic reviews of random, controlled trials have not revealed the benefit of one dressing class over another in various chronic wound types.[5,6] Palfreyman et al[6] found no significant difference between adv anced moistur e-controlling dressings and normal saline gauze. However, saline gauze usually requires frequent dressing changes to maintain moistur e balance, increasing the likelihood of trauma and pain, as well as higher nursing costs. Alternative dressings may remain on the wound for longer periods to avoid frequent removal.[7]

Moist interactive healing is contraindicated in nonhealable wounds. Instead, local wound care involves conservative debridement of nonviable slough without causing bleeding, bacterial reduction, and moisture reduction. Nonhealable wounds are best treated with antiseptics when healing is not immediately achievable (uncontrolled deep infection) or when bacterial burden is more of a concern than tissue toxicity (maintenance or nonhealable wounds). ■

Wound Edge Effect

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Normal wound healing follows an orderly sequential trajectory that requires an integration of complex physiological and molecular events. These events include cell recruitment and migration; connective-tissue cell proliferation; and extracellular matrix synthesis and deposition.[1] As a rule of thumb, if a healable wound is not at least 30% smaller at week 4, despite optimal local wound care, it is unlikely to heal by week 12.[2]

Some of the mechanisms responsible for stalled wound healing remain controversial. The edge of the wound often fails to advance when keratinocyte migration is challenged, preventing wound closure. The epidermal edge may have a steep, cliff-like appearance (the edge effect), rather than the tapered edge of advancing purple-pink epithelium that slopes into the mature granulation base of a healing wound. This physiologic occurrence is different from epibole, in which epithelium has grown over a deficient space. Tomic-Canic et al[3] explain that hyperproliferation of keratinocytes and their impaired migration in chronic wounds may be due to overproduction of a molecule called C-myc. Many chronic wounds also demonstrate markedly increased activity of inflammatory cells and associated mediators such as matrix metalloproteinases (MMPs).[4] Wound healing stalls because degradation of extracellular matrix and growth factors occurs more rapidly than their syntheses, hindering the wound from progressing toward the proliferative phase and, ultimately, re-epithelialization. Harding et al[5] portend that the longer a wound remains in the inflammatory phase, the more cellular defects are detected, with potential delayed healing.

Wound healing can be improved by locally manipulating growth factors, extracellular matrix (ECM), and the signaling pathways that regulate migration, proliferation, differentiation, and metabolism of cells. Active wound therapies — including biological agents, growth factors, autologous epidermis, allograft, and adjunct therapies — may promote the advancement of the wound edge as outlined in **Tables 1 through 3** for venous leg ulcers, diabetic foot ulcers and pressure ulcers. ■

Table 1: Advanced Therapies for Venous Disease

Advanced Therapy	RCT Meta-analysis	Results
Oasis (Healthpoint, Fort Worth, TX)	Yes[6]	Complete healing
Apligraf (Organogenesis, Canton, MA)	Yes[7,8]	Complete healing
Therapeutic ultrasound	Yes[9]	Faster healing
Promogran (Systagenix, Somerville, NJ)	Yes[10,11]	Decreased wound size
Electrical therapy	Yes[12]	Decreased wound size and reduced pain
Ultrasounds	Yes[13,14]	Decreased wound size; faster healing
Light therapy	Yes[29]	Decreased wound size

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Table 2: Advanced Therapies for Diabetic Neurotropic Foot Ulcers

Advanced Therapy	RCT Meta-analysis	Results
Oasis (Healthpoint, Fort Worth, TX)	Yes[15]	Complete healing equal to PDGF
Growth factors (PDGF)	Yes[16,17]	Complete healing
Apligraf (Organogenesis, Canton, MA)	Yes[18-20]	Complete healing
Dermagraft (Advanced BioHealing, Westport, CT)	Yes[21-23]	Complete healing
Hyperbaric oxygen therapy (HBO)	Yes[24]	Prevention of amputations
Therapeutic ultrasound	Yes[25]	Complete healing
Negative-pressure wound therapy	Yes[26]	Decreased wound size

Table 3: Advanced Therapies for Pressure Ulcers

Advanced Therapy	RCT Meta-analysis	Results
Negative-pressure wound therapy (NPWT)	Yes[27,28]	Decreased wound size
Light therapy	Yes[30]	Decreased wound size

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Health Care Systems:
What are the Current Problems?

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Wound care forums sponsored by Mölnlycke in 2008 occurred during a year of great payment change for US hospitals. As of Oct. 1, 2008, the Centers for Medicare and Medicaid Services no longer reimburses for specific hospital-acquired conditions, such as pressure ulcers and surgical site infections. [1] The wound care community also saw dwindling reimbursement for treatment modalities such as enzymatic debriders for Medicare Part D in January 2008. In September 2008, the Food and Drug Administration issued a notice of withdrawal of all topical papain-urea.[2]

U.S. Challenges

We asked members of the US wound care community what they thought was the biggest obstacle (aside from workload) to improving outcomes for patients with chronic wounds. The themes identified were categorized in three groups: patient-centered concerns, professional issues, and payor regulations/costs.

Patient-Centered Concerns

The most frequently cited problem was patient adherence to and participation in care. Second was the severity and comorbidities of patients' illnesses; some patients lack "good body habits," have chronic disease, and lack healthy lifestyles. Factors include peripheral vascular disease, diabetes in which glucose levels are not regulated, continued smoking, and not offloading the foot or pressure area. Some clinicians expressed problems with patients' inabilities to manage their care plans. They note that many patients have "lost their appetites, and food tastes more like bad-tasting medicine" and that "patients cannot break through the mental barrier about eating," which "may easily end in the death of the patient."

Due to its psychosocial effects on wound healing, pain was another prevalent concern. Other concerns included short stay lengths, early discharge before adequate teaching could occur, problems accessing follow-up with wound specialists, and not qualifying for home care or home dressing change assistance. Patients' lack of insurance and inability to pay for or finance their required care was another often-voiced concern.

Professional

The most frequently expressed concerns in this area were professionals' low levels of wound knowledge and corresponding need for more education; need for consistency of care by all caregivers; improvement in continuity of care across care settings; lack of cooperation among the multiprofessional team; and not having all the best products to treat patients. Wound care professionals' collective frustration is exemplified by statements such as, "It takes forever to get management to add new products to the formulary," and "We need products to combat the chronic (non-responsive) cells in wounds."

Communication and cooperation are needed for better team care, as evidenced by these sample statements: "Sometimes I am called only after conditions have deteriorated in the wound bed," or "Why are physicians not ordering the latest techniques or not changing the wound care when current treatment has stalled the healing process?"

Others remarked about problems with the time required for nursing care, lack of access to desired therapies, and discharge of patients before verifying product effectiveness or before "finding treatments that are easy to use and effective with minimal dressing changes and disturbance of the wound bed."

Payor Regulations/Costs

The major issue was obtaining insurance coverage for products in the required quantities. Other problems: Insurance companies and payers don't allow some therapies outside the hospital, and patients can't always procure dressings. For example, says one participant, "cost of products to heal wounds for patients is unaffordable post-discharge."

Respondents were clear in their wish to have universal healthcare coverage and adequate coverage for wound care products in the US. They also wanted pain-free dressing application and removal, as well as a greater "availability of products and services recommended for the patient." The wish-list also included "better computerized documentation and outcome measurements, wound care teams in more settings" and "more emphasis on preventing wounds."

Summary

The concerns and needs of respondents mirror the World Union of Wound Healing Societies' (WUWHS) Guidelines for Wound Healing Policy, which can be found on the WUWHS Web site.[3] Comments from the global wound community are welcome. Despite the challenges, US wound care clinicians are doing an incredible job and have earned high marks on this report card for the overall care they are rendering to patients. ■

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