Chronic Wound Pain: A Conceptual Model

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Dr Woo has disclosed that he is/was a recipient of grant/research funding from the Canadian Association of Wound Care; is/was a consultant/advisor to Coloplast, Mölnlycke, KCI, and the Registered Nurses of Ontario Association; and is/was a member of the speaker’s bureau for Johnson & Johnson, Coloplast, and Mölnlycke. Dr Sibbald has disclosed that he is/was a recipient of grant/research funding from Coloplast, Smith & Nephew, 3M, KCI, Mölnlycke, Cividien, and Johnson & Johnson; is/was a consultant/advisor to Coloplast, Smith & Nephew, 3M, KCI, Mölnlycke, Cividien, and Johnson & Johnson; and is/was a member of the speaker’s bureau for Coloplast, Smith & Nephew, 3M, KCI, Mölnlycke, Cividien, and Johnson & Johnson. All staff in a position to control the content of this CME activity have disclosed that they have no financial relationships with, or financial interests in, any commercial companies pertaining to this educational activity. Acknowledgment: This article has been partially supported by unconditional education grants from Coloplast and Mölnlycke Healthcare.

Lippincott CME Institute, Inc, has identified and resolved all faculty and staff conflicts of interest regarding this educational activity.

PURPOSE
To present the wound care practitioner with a model for the assessment and treatment of wound-related pain.

TARGET AUDIENCE
This continuing education activity is intended for physicians and nurses with an interest in wound care and related disorders.

OBJECTIVES
After reading this article and taking this test, the reader should be able to:
1. Discuss the pathophysiology of chronic pain and the wound pain model.
2. Describe the patient’s wound-related pain perspective.
3. Identify aspects of local wound care and their relationship to pain.

Unremitting and recalcitrant pain is disabling and devastating in patients with chronic wounds. To help clinicians manage pain, this article will discuss a wound pain model based on the wound bed preparation (WBP) paradigm. The key components include patient-centered concerns, cause(s) of the wounds, associated complications, and local wound factors.

Pain is a common concern in patients with various types of chronic wounds, including pressure ulcers, venous leg ulcers, diabetic neuropathic foot ulcers, and malignant

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ADVANCES IN SKIN & WOUND CARE • APRIL 2008
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These wounds are often recalcitrant to healing, apt to recur, and may not respond to currently established management strategies. The prevalence of chronic wounds is increasing as society continues to age. An average of 26% of hospitalized and community care patients have pressure ulcers across a continuum of health care settings. In a study of patients with leg ulcers, half of the affected population had a previous leg ulcer history spanning 5 to 10 years, and a third of affected individuals had a history exceeding 10 years. Another retrospective survey suggested that only about half of the patients had healed ulcers in a 5-year period, and the recidivism rate was as high as 60% to 70% among these individuals.

In persons with diabetes, 2% to 3% will develop a foot ulcer annually; their lifetime risk is as high as 25%, primarily caused by neuropathy or loss of protective sensation and potential coexisting vascular disease. In a 1-year period, 7.2% of patients with both diabetes and neuropathy will develop their first foot ulcer.

To achieve the best possible patient outcomes, management of these chronic wounds must address the local wound issues, patient-centered concerns, and systemic barriers, even when healing is not expected. Evidence indicates that pain is a primary patient-centered concern. Pain not only has a decisive impact on patients’ quality of life but also may affect wound healing through various mechanisms.

WOUND BED PREPARATION PARADIGM AND CHRONIC WOUNDS
To address the complexity of chronic wounds and pain management, an organized integrated approach to treatment is required. Sibbald et al articulated the WBP model (Figure 1) to manage chronic wounds and optimize achievable patient outcomes. The premise of the WBP model encourages the practitioner to correct the wound cause and address patient-centered concerns, including wound-related pain, before initiating local wound care.

WOUND PAIN
What Is Wound Pain?
Pain has been described as a complex paradigm that encapsulates 3 key dimensions: sensory-discriminative, affective-motivational, and cognitive-evaluative dimensions. Pain cannot be measured directly; it can only be established and determined by the person who is experiencing it. The International Association Study in Pain Task Force on Taxonomy defines pain as “an unpleasant sensory and emotional experience that is associated with actual or potential tissue damage or described in terms of such damage.” Accordingly, pain is more than an electrical impulse signaling a noxious sensation that is conducted from the peripheral receptors, then ascends through the spinal pain pathways, and ultimately projects to higher centers of the brain where pain perception is shaped. Pain is a function of a centrally processing mechanism modified by a broad array of excitatory and inhibitory inputs from ascending and descending spinal and supraspinal tracts.

Melzack and Wall proposed a “gate” mechanism located in the dorsal horn of the spinal cord, where pain transmission can be modulated. According to this theory, the smaller pain fibers are believed to inhibit substantia gelatinosa interneurons opening the “pain gate” to allow signal propagation, whereas activation of larger nonpain fibers can do the opposite to close the gate. Higher functions, such as cognitive interpretation, anxiety, stress, and emotions that are ascribed to pain, can modulate pain experience through the release of endogenous opioid peptides (eg, enkephalins, dynorphins, β-endorphins) and other neurotransmitters (eg, norepinephrine, serotonin). This would explain why a similar stimulation can evoke a very different response in different individuals under different circumstances. It has been demonstrated that appreciation of, expression of, and tolerance to pain can be influenced by knowledge/understanding, beliefs, attitudes, and expectations acquired through social interactions and experiences.
previous pain experiences. To elucidate the integrated experience of pain, Melzack introduced the term “neuromatrix” to describe interactions between complex circuitry and networks that modulate the perception of pain. Figure 2 illustrates pain mechanisms and dimensions.

According to a conceptual framework developed by Krasner, wound-related pain is complex, integrating the experience of noncyclic acute wound pain, cyclic acute wound pain, and chronic wound pain. Chronic wound pain is the background pain that occurs in the absence of any manipulation and is usually associated with the underlying etiology of the wound. Chronic pain may be continuous and persist throughout the day or may be intermittent and occur in waves. Cyclic wound pain is periodic acute wound pain induced by regular and repetitive treatment or interventions, often during recurring dressing change. Noncyclic wound pain is provoked by more sporadic procedures, for example, sharp debridement, that occurs either as a 1-time procedure or at infrequent and usually variable intervals.

**Is Wound Pain a Common Problem?**

Studies of patients with venous leg ulcers indicated that more than 80% of patients reported acute or chronic wound pain, with half of them rating pain as moderate to the worst possible pain. Patients gave vivid descriptions of the pain experiences even after the leg ulcers were healed. Dallam and colleagues reported that 59% of patients with pressure ulcers experienced some type of pain in a hospital setting. A substantial number of the subjects perceived pain to be severe. In another study of patients with pressure ulcers, Szer and Bourguignon reported that as many as 84% of their subjects experienced pressure ulcer-associated pain at rest, whereas 88% acknowledged cyclic acute wound pain at dressing change. Consistent with previous findings, Meaume et al reported that 79.97% of a large sample of chronic wound patients (n = 2936) experienced moderate to severe pain during dressing change. Seventy-seven percent of these patients experienced spontaneous pain. Other studies documented that pain did not follow any temporal patterns, with approximately 80% of patients with pressure ulcers experiencing constant pain despite the use of analgesics. Proctor and Hirdes performed an analysis based on a minimum data set involving 3195 nursing home residents. Data substantiated the significant association between pressure ulcers and daily pain experience (odds ratio, 3.5; 95% CI, 1.81–6.76).

Despite the commonly held belief that most patients with diabetic foot ulcers (DFUs) do not experience pain because...
of loss of protective sensation, as many as 50% of patients reported they experienced varying degrees of painful symptoms at rest, and approximately 40% experienced moderate to extreme pain climbing stairs or walking on uneven surfaces. Results of these studies validate not only the various types of chronic wound pain, but also the severity and pervasiveness of wound-related pain.

Unfortunately, pain is inconsistently assessed and documented by health care providers. Lorimer et al. reviewed 66 nursing records of venous ulcer patients receiving home care. Only 15% contained any documentation of pain, and the assessment was not standardized. Pain was not part of a regular assessment of leg ulcers in up to 55% of community nurses. Husband sought to explore the care provided for venous ulcer patients in the community. They interviewed 39 patients and 33 nurses, and observed that nurses focused their primary attention on local wound care and underlying etiology, but the patients perceived pain management to be a much higher priority. Patients often express feelings of frustration at the failure of health care providers to mitigate wound pain. To highlight the importance of the pain issue, the National Pressure Ulcer Advisory Panel stated that pressure ulcers (wounds) contribute to the occurrence of considerable human suffering. The European Wound Management Association and the World Union of Wound Healing Societies have both produced position and consensus documents to address the importance of wound-associated pain.

**How Should Wound Pain Be Approached?**

Pain management is fundamental to the overall management of patients with wounds. As Figure 3 illustrates, the key components of the total wound pain model presented are as follows:

- **Patient-centered concerns:** What is the meaning of pain? How does pain affect patient’s quality of life?
- **Cause(s) of the wounds:** Is pain related to the underlying pathology of the wound?
- **Associated complications:** Is pain exacerbated or mitigated by associated complications and their treatment?
- **Local wound factors:** Is there recurring trauma to the wound? Is there an undiagnosed infection or inflammatory complication? Is there too much or too little moisture?

**PATIENT-CENTERED CONCERNS**

Unremitting and recalcitrant wound-related pain is disabling and devastating. Eager surveyed patients with wounds in long-term-care facilities and elucidated that pain was their first and foremost concern. Puntillo and colleagues reported that adolescents had related pain as the greatest discomfort among 5 commonly performed hospital procedures. A number of studies, including patients with venous leg ulcers and pressure ulcers, significantly correlated wound-related pain to diminished quality of life. High levels of wound-related pain also hinder interpersonal relationships and decrease psychosocial adjustment at home. The greater the pain interference, the more problems the person experienced at home.

Using a qualitative study methodology (hermeneutic phenomenological model), Krasner identified the multitudinous aspects of pain and suffering and its negative impact on the quality of life among patients with painful venous leg ulcers. Four major themes emerged linking pain and quality of life: feeling frustrated, interfering with work, the need to make significant life changes, and finding less satisfaction in new activities. A pressure ulcer study also considered wound-related pain considered as all encompassing, permeating all domains of peoples’ lives. Activities important to maintain daily functioning, such as working, walking, standing, and stair climbing can often exacerbate wound pain. To avoid pain, people with chronic wounds limited mobility and social activities and complained of living a restricted life. Because of excessive pain, a proportion of patients (31%) were not even able to attend wound care clinics. It is not surprising to find that patients with venous leg ulcers often described pain as the worst aspect of having an ulcer.

Pain can be fatigue as it often occurs or may be exacerbated at night. Noonan and Burge reported that up to 73% of patients with a venous leg ulcer reported sleep disturbance.

There is an exquisite connection between pain and emotional well-being in patients with a chronic wound. Patients often expressed a sense of imprisonment, powerlessness, anger, sadness, and/or despair. A group of older adult patients stated eloquently that “it is a time of hopelessness, when the painful wound controls their existence.” Negative mood and depression are both common. Pain and anxiety are frequently reported in people with burns during dressing change. In a study of 24 patients requiring burn dressing change, pain was positively correlated to anxiety immediately after burn dressing changes. Using a hierarchical regression model, Aaron and colleagues demonstrated that anxiety is a significant predictor of procedural pain during dressing change and contributed to 40% of the variance of reported pain in burn patients. Anxiety and depression were significantly associated with increased pain in 190 patients with leg ulcers and diabetic neuropathy.
Patients inflicted with chronic wound pain may display maladaptive coping styles and relational distortions. Roth et al.⁸⁰ demonstrated that pain was correlated to catastrophic thinking in patients with chronic wounds. Chronic persistent pain may affect how people appraise their personal situation with a propensity to think the worst will happen, thereby raising anxiety, worry, and pain sensitivity.⁸⁰,⁸¹

As a source of psychological stress, wound-related pain may promote the production of glucocorticoids that reduce the inflammatory response, inhibit the regeneration of endothelial cells, and delay collagen synthesis.⁸² Stress-induced cytokine and neuroendocrine activity will activate sympathetic outflow leading to vasoconstriction and subsequent compromised tissue-oxygenation levels.⁸³,⁸⁴ Thus, pain can have a significant adverse impact on wound healing.⁸⁵ Although causality cannot be inferred, patients with venous leg ulcers who were randomized to a nurse-managed support group achieved a 76.8% reduction in mean ulcer size (10.3–2.39 cm²) with a significant reduction in pain levels over 12 weeks (4.04 to 3.09, P = .001).⁸⁶ In contrast, the mean ulcer size was merely reduced by 11.8% in the control group (7.63–6.8 cm²) without any significant changes in pain scores. Following the improvement of pain, patients who participated in the support group also experienced improvement in mood, sleep, and normal work.⁸⁶ Although the results were not always consistent, pain was more severe in larger and deeper
wounds. Johnson examined the key determinants for the healing of venous and mixed venous/arterial ulcers in 155 patients. With the use of a hierarchical multiple regression analysis, pain with mobility was found to be a negative predictor for wound healing ($\beta = .46; P < .0001$). To validate the relationship between chronic pain and wound healing, McGuire et al$^9$ studied 17 women who underwent gastric bypass surgery. Patient pain ratings over 4 weeks after surgery were significantly associated with delayed healing of a 2.0-mm punch biopsy wound site.

Health care providers should be cognizant of the potential and devastating effect of pain on an individual and provide emotional support as part of comprehensive wound care. Using an experimental design, Jørgensen et al$^{10}$ demonstrated that subjective evaluation of well-being can significantly be improved, along with the reduction of pain, using a foam dressing containing ibuprofen ($P < .0001$).

Explanation for why pain occurs reflects ontological values and belief systems espoused by patients. However, myths and misconceptions about pain are commonly held by patients and even health care providers. These misconceptions can have serious ramifications, hinder optimal pain management, and should be addressed with sensitivity (Table 1).

### PAIN AND WOUND CAUSES

Free nerve endings originate in the dermis and are distributed to the surface of the skin and its underlying structures. Perception of pain derives from the direct excitation of nociceptors in cutaneous free nerve terminals in response to trauma and tissue damage incurred by mechanical forces (pressure, friction, and shear), chemical irritation, vascular compromise (venous hypertension and arterial insufficiency), infectious agents, or inflammation (Figure 3).

Patients with chronic wounds are vulnerable to the development of neuropathic pain due to protracted and recurring noxious stimulation. Franks and Moffatt$^{11}$ demonstrated that large (>10 cm) and chronic (>6 months) wounds were associated with elevated pain levels ($P < .05$). Chronic wounds (eg, pressure ulcers) are often inducted with inflammatory cells and proinflammatory cytokines within a dense fibrin matrix at the ulcer surface.$^{12}$ Persistent inflammation and associated cell injury may activate peripheral and central nociceptors, characterized by spontaneous firing of nociceptors (spontaneous pain), exaggerated or prolonged responsiveness to noxious inputs (hyperalgesia), reduced pain threshold (allodynia), and spread of pain to uninjured tissue such as wound margins (widened receptive fields).$^{13}$ The mechanism is complex, involving the remodeling of synaptic contacts between the neurons in the spinal dorsal cord and should be addressed with sensitivity (Table 1).

### Table 1. PAIN: MYTHS AND TRUTHS

<table>
<thead>
<tr>
<th>Myths/ Misconceptions</th>
<th>Truths</th>
</tr>
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<tbody>
<tr>
<td>Pain is an auspicious sign indicating active healing with new nerves and tissues beginning to sprout.$^7$</td>
<td>Pain can impair wound healing.</td>
</tr>
<tr>
<td>Health care providers should know when patients are in pain. Many patients (41%) do not like to bother others with their complaints of pain.$^3$</td>
<td>Persons with chronic pain do not always exhibit physiological signs typical of acute pain.$^9$</td>
</tr>
<tr>
<td>People will report that they are in pain or ask for help.</td>
<td>Patients need to be empowered to express their pain.</td>
</tr>
<tr>
<td>Wound pain is transient and does not have a lingering effect after dressing change.</td>
<td>Pain often persists after dressing change.$^9$</td>
</tr>
<tr>
<td>Painkillers are bad for the body.$^5,70$</td>
<td>All pain medications have adverse effects and may require adjustment based on renal and hepatic function. Most medications, when used appropriately, are not harmful to the body.$^9$</td>
</tr>
<tr>
<td>People can get addicted to painkillers.$^9$</td>
<td>Less than 1% of patients develop addiction, and this is seldom a problem if patients have debilitating pain.$^9$</td>
</tr>
<tr>
<td>Pain is inevitable, and there is no effective way to manage pain.$^5,66$</td>
<td>Multiple modalities are available to manage pain including topical treatment.$^9$</td>
</tr>
<tr>
<td>Pain indicates something terribly wrong with the wound.</td>
<td>Increased pain above ordinary levels may be indicative of infection, persistent abnormal inflammation, trauma, or a failure to correct the wound cause.</td>
</tr>
<tr>
<td>Pain assessment tools are time consuming. Patients always have difficulty reporting pain using a scale.</td>
<td>Many validated pain instruments (MPQ, NRS, VAS, FACES) can be easily used in routine practice to assess pain.$^4$</td>
</tr>
<tr>
<td>Older adults, especially those with cognitive problems, do not have pain.</td>
<td>Pain is not different in older adults, even if they have cognitive impairment (watch for body language).</td>
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</table>

MPQ = McGill pain questionnaire; NRS = numerical rating scale; VAS = vascular analogue scale; FACES = faces scale.
Nevertheless, central sensitization or “wind-up” phenomena lend credence to the prevalence of noncyclic wound pain that is out of proportion to the stimulus at dressing change and pain that is produced when the area surrounding the wound is cleansed.

It is also postulated that as peripheral nerves regenerate in chronic wounds, the nociceptive nerve terminals transmit immature sprouts of neurons that have been shown to be hypersensitive to both noxious and nonnoxious stimuli. Increased pain has also been linked to infection. Nerve endings are irritated by enzymes and proinflammatory mediators that are produced as part of the host and bacterial response to wound tissue infection.

Venous Leg Ulcer-Associated Pain

Patients with early stages of venous disease often describe a dull aching or heaviness in their legs due to pitting edema and prominent varicose veins. The sense of heaviness usually worsens toward the end of the day or after prolonged periods of standing that exacerbates edema or fluid accumulation. Lipodermatosclerosis is a common presentation in patients with venous disease, and it is characterized by a triad of nonpitting edema, sclerosis, and pigmentation. Even in the absence of an ulcer, pain has been found in up to 43% of patients with lipodermatosclerosis. Excruciating erythema may accompany acute lipodermatosclerosis, and this may often be mistaken as cellulitis. Superficial or deep phlebitis must also be considered when patients describe a new pain or a change in pre-existing pain. Superficial phlebitis tends to present as bruise-like pain over a localized portion of a red, inflamed, and tender vein. The pain is often aggravated by palpation or standing, and the involved area may be quite warm to touch. Another factor that complicates venous disease is the presence of dermatitis, particularly the periwound skin. Dressings, bandages, and even compression hosiery can cause local irritation and may aggravate stasis eczema. Excessive exposure to wound exudate can result in enzymatic damage of the skin, increasing the risk of allergic contact dermatitis. Patients may complain of a burning pain and itching sensation. Scratching and rubbing of the area may cause further trauma and pain. Atrophic blanche is a sign often seen with the end stages of venous disease. It consists of a distinct white, sometimes star-shaped, depressed white atrophic scar-like area. Although the exact mechanism remains elusive, atrophic blanche is frequently associated with severe, sharp, localized pain.

Pain can be caused by compression therapy. Briggs and Closs indicated that only 56% of patients in their study were able to tolerate full compression bandaging, with pain being the most common reason for nonadherence. Elastic compression systems exert high pressure at rest and may not be tolerated until adequate pain control has been achieved. Nonelastic systems exert their main effect with high pressure only with muscle contraction (during movement), and they may be tolerated at rest where there is a lower pressure against a fixed resistance.

Arterial Disease and Ischemic Ulcer Pain

The first and most important step in any patient with arterial disease is to determine if the blood supply is adequate for healing. For leg and foot ulcers, a palpable pulse indicates that the blood supply is adequate for healing. If the pulse is not palpable, Doppler examination of the ankle brachial pressure index is necessary to determine the ability to heal.

Intermittent claudication is a cardinal symptom of peripheral arterial disease in the lower extremities. It arises when blood flow is insufficient to meet the metabolic demands of leg muscles during physical activities. With the accumulation of lactate and other toxic metabolic products, patients may experience leg pain, aching, cramping, or numbness in the calf, buttock, hip, thigh, or arch of the foot. Ultimately, as arterial disease continues to progress, pain may occur even at rest. In patients with a history of claudication, Aquino et al demonstrated that the 10-year cumulative risk of ischemic rest pain was 30%, and ischemic ulcers developed in 23% of 1244 subjects.

Ischemia causes tissue damage and, surprisingly, restoration of blood flow (reperfusion injury) causes even more damage. The main putative mechanisms involve leukocyte-endothelium interactions, reactive oxygen species, and the complement system. Tissue damage due to ischemia and reperfusion injury has been proposed to be responsible for intense and persistent skeletal muscle contraction or spasm. This leads to a vicious cycle of pain-spasm-pain.

Pressure Ulcer Pain

Pressure ulcers develop from unrelieved pressure that exceeds capillary closing pressure (approximately 32 mm Hg), resulting in tissue ischemia. Pain is the end result of tissue damage through the release of inflammatory mediators. Irritation can occur with accumulation of moisture from incontinence of stools and urine or because of friction and shear forces. Pain was often described as burning, stabbing, stinging, tugging,
Pain could be exacerbated by pressure-relieving equipment and treatment-related activities, such as repositioning in bed or sitting up. The majority of patients with pressure ulcers are immobile or have limited mobility. These patients may develop pain from contractures or muscle spasticity and spasms that clinicians should not overlook.

**Diabetic Foot Ulcer and Pain**

Up to 75% of persons with DFUs reported some pain, and 57% had pain while walking or standing or during the night. Painful diabetic neuropathy is a challenging complication of diabetes mellitus. Neuropathic pain is often spontaneous without an identified stimulus. It presents as a chronic and/or persistent pain invoking both physical and emotional pain responses. Persistent neuropathic pain is thought to be caused by nerve irritation and damage to the nerve receptors as a result of metabolic changes to the vasa nervorum, affecting nerve conduction and/or microvascular changes compromising perfusion to the peripheral nerves. The pain is usually described as a constant burning or tingling sensation in the foot involving the entire foot or lower leg in a “stocking-and-glove” distribution and may include unpleasant shooting, stabbing sensations.

Charcot foot is a progressive neuropathic arthropathy that affects the bony structures of the feet. Charcot arthropathy is characterized by pathologic fractures, joint dislocation, and foot deformity in patients with severe neuropathy. Pain is the presenting complaint in 75% of cases along with a red, hot, swollen foot. Deep tissue wound infection (usually osteomyelitis) or deep inflammation associated with a Charcot joint can cause pain even in the presence of severe neuropathy. The presence of nonneuropathic pain in even a mildly erythematous or edematous diabetic foot requires urgent assessment and immediate treatment to prevent limb amputation.

**Inflammatory and Miscellaneous Causes of Wound Pain**

Pyoderma gangrenosum is an inflammatory skin disease resulting in painful, enlarged, ulcerated plaques. The prominent features of active pyoderma gangrenosum are well-demarcated, irregular ulcers with heaped-up inflammatory, purple to deep red undermined edges and a boggy, necrotic central wound base.

Pain is intense and often out of proportion to the appearance of the lesion. Pyoderma gangrenosum may be idiopathic but occurs with systemic diseases in about 50% of the cases (rheumatoid arthritis, inflammatory bowel disease, or myeloproliferative disorders). In susceptible people, even minimal trauma or irritation to the skin can result in the production of pyoderma gangrenosum lesions, a phenomenon referred to as pathergy.

Vasculitis is due to deposits of immune complexes in areas of slower blood flow in the lower leg, peripheral cooling, reduplication of the basement membrane, and gravitational forces (Table 3). The lesions often occur in crops associated with local pain. About 50% of cases of cutaneous vasculitis have systemic involvement with joint, liver, and kidney being more common, and involvement of the heart, lungs, brain, or gastrointestinal systems less frequent. Etiologic associations include infections, systemic disease, and malignancy.

Pain may also be associated with malignancy-associated cutaneous ulcers. The pain can result from the tumor pressing on nerve endings and adjacent visceral organs, mediators released by tumor cells that irritate nerve endings, ischemia, inflammation, or increased swelling.

**LOCAL WOUND CARE**

**Tissue Trauma With Dressing Change**

Wound dressing change involves removal of soiled dressings, cleansing of the wound bed, and reaplication or packing of new dressing materials. The purpose of local wound care is to promote wound closure by removing harmful debris, reducing bacterial burden, containing the exudate, and protecting the wound from mechanical injury. Although dressing changes are integral to wound management, aggravation of sensory fibers can occur. Approximately 80% of the 5850 patients in a study described pain as moderate to severe during dressing changes. A significant difference

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**Table 2. COMMON VASCULAR ASSESSMENTS AND PARAMETERS**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>Palpable pulse</td>
<td>80 mm Hg</td>
<td>Difficult to palpate in patients with edema</td>
</tr>
<tr>
<td>Ankle brachial ratio</td>
<td>&gt;0.5 and &lt;1.2</td>
<td>False elevation in patients with calcified vessels</td>
</tr>
<tr>
<td>Transcutaneous oxygen tension</td>
<td>&gt;30 mm Hg</td>
<td>Expensive equipment and is labor intensive</td>
</tr>
<tr>
<td>Toe pressure</td>
<td>&gt;55 mm Hg</td>
<td>The large toe is of a small caliber without a fully developed adventitial layer to facilitate circumferential calcium deposits</td>
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</table>
was detected in pain intensity before, during, and after dressing change in nonsurgical patients. On a scale from 0 (no pain) to 10 (the worst possible pain), the mean pain levels were 3.0, 4.4, and 2.7 before, during, and after dressing change, respectively. Pain during dressing change was significantly higher ($F = 6.57$, $P = .013$). Words such as tender, sharp, stinging, aching, and stabbing were frequently used to describe pain during dressing change.

These sensations represent a combination of nociceptive (gnawing, aching, tender, throbbing) and neuropathic (burning, stinging, shooting, and stabbing) pain. Vermeulen et al. asked patients and health care providers to rank their preferences for an ideal dressing material. Minimal pain during dressing changes was rated consistently by patients as the most important attribute even before the hypothetical ability of the dressing to accelerate wound healing.  

Of interest, during dressing change, dressing removal was indicated to be the most painful procedure in persons with chronic wounds. Up to 80% of nurses noticed that patients experienced more severe pain ($P < .01$), and dressings adhered more to the wound bed and surrounding skin with saline gauze dressing compared with an alternate choice of a hydrocolloid dressing. A review of dressings and topical agents for postsurgical wounds related that patients experienced more pain with gauze compared with advanced moisture balance dressings including foam, alginate, and hydrocolloid dressings. Each time a dressing was removed, trauma was the major problem. This trauma may perpetuate the inflammatory response with each dressing change. It has been demonstrated that repeated application and removal of adhesive tapes and dressings can instigate skin damage by stripping the stratum corneum.

Dressings that require less peel force on removal may invoke less pain. With a high peel force, the epidermis is pulled away from the dermis, causing erythema, edema, and blistering. In a study of 4200 wounds in 1891 nursing home residents, pain and trauma associated with dressing removal was a major concern for both patients and health care providers. Persons on long-term steroid therapy, those with a defect in type VII collagen–associated epidermolysis bullosa, and the older adult population all have delicate fragile skin that is particularly susceptible to skin damage. Pain is often exacerbated if local wound care product creates allergic contact or irritant dermatitis, or if it removes part of the skin barrier.

### Table 3. CLINICAL FEATURE OF PYODERMA GANGRENOsum AND VASCULITIS

<table>
<thead>
<tr>
<th>Condition</th>
<th>Clinical picture</th>
<th>Features</th>
</tr>
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<tbody>
<tr>
<td>Pyoderma gangrenosum</td>
<td><img src="https://example.com/table3_image1.png" alt="Image" /></td>
<td>A typical skin lesion is circumscribed by an irregular ulcer with raised, rolled, erythematous borders, and undermined edges with a necrotic base.</td>
</tr>
<tr>
<td>Vasculitis</td>
<td><img src="https://example.com/table3_image2.png" alt="Image" /></td>
<td>Vasculitis lesions appear on the distal extremities. They often occur in crops with palpable purpura, the most characteristic lesion, but the morphology can vary from fixed urticaria to vesicles, nodules, or frank necrosis.</td>
</tr>
</tbody>
</table>

Vasculitis lesions represent a combination of nociceptive (gnawing, aching, tender, throbbing) and neuropathic (burning, stinging, shooting, and stabbing) pain. Vermeulen et al. asked patients and health care providers to rank their preferences for an ideal dressing material. Minimal pain during dressing changes was rated consistently by patients as the most important attribute even before the hypothetical ability of the dressing to accelerate wound healing.  

Of interest, during dressing change, dressing removal was indicated to be the most painful procedure in persons with acute wounds and wound cleansing in persons with chronic wounds. Up to 80% of nurses noticed that patients experienced most pain during dressing change, especially when the dressing was removed. In a pilot study of 12 patients, pain was measured at baseline, dressing removal, cleansing, application of new dressing, and 5 minutes after dressing changes. Wilcoxon signed rank test indicated that pain intensity was significantly higher at dressing removal ($P < .016$) and cleansing ($P < .023$) compared with baseline levels.

Removal is painful when dressings adhere to the wound bed because of dried-out materials, aggressive adhesives, granulation tissue and capillary loops growing into the product matrix, and the glue-like nature of dehydrated or crusted exudate. Enzyme-rich exudate may spill onto the wound margins, causing maceration or tissue erosions with an increased risk of trauma. Despite availability of advanced dressings, 40% of nurses with special interest in wound care were oblivious to products specifically designed to prevent pain and trauma. Mahe and colleagues reported that less than 50% of wounds on medical units were covered with appropriate materials. In fact, gauze dressings continued to be the most commonly used wound contact layer in a study of 412 hospitalized adults. Up to 38% of surveyed nurses (n = 692) stated that wet-to-dry dressings were their choice to treat clean granulating chronic wounds. In a randomized controlled trial, pressure ulcer patients experienced more severe pain ($P < .01$), and dressings adhered more to the wound bed and surrounding skin with saline gauze dressing compared with an alternate choice of a hydrocolloid dressing.

A review of dressings and topical agents for postsurgical wounds related that patients experienced more pain with gauze compared with advanced moisture balance dressings including foam, alginate, and hydrocolloid dressings. Each time a dressing was removed, trauma was the major problem. This trauma may perpetuate the inflammatory response with each dressing change. It has been demonstrated that repeated application and removal of adhesive tapes and dressings can instigate skin damage by stripping the stratum corneum.
Wound cleansing has also been identified to be painful following dressing removal. Application of cold cleaning solutions to wounds can be unpleasant. Strong antiseptics are cytotoxic and may cause stinging and pain. Routine practice of physical manipulation using forceps and gauze to wipe across wound beds can cause tissue damage. In the study reported by Stotts et al., packing, irrigation, or debridement increased pain significantly during the procedure compared with dressing change alone (P = .0001). In conclusion, cyclic procedural wound pain constitutes both physiological and psychological stresses.

Infection (Superficial or Deep) and Chronic Inflammation

Chronic wounds are invariably colonized by microorganisms usually introduced from external contamination. Infection stimulates an inflammatory response leading to a persistent influx of neutrophils. These polymorphs 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 vasocostricting 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 infection may facilitate healing. The diagnosis of superficial increased bacterial burden or deep infection in a chronic wound is best confirmed and supported by documenting clinical signs and symptoms.

Cutting and Harding proposed that the presence of unexpected pain or tenderness along with other criteria is indicative of infection in granulating wounds. Based on these criteria, Cutting and Harding compared wound swab results with their clinical assessment. Thirty-nine (97.5%) of 40 wound assessments were consistent with bacteriologic findings. Agreement between 2 nurses (2 x 40 assessments) and the researcher was only 47.5% (38/80). Although Cutting and Harding used pain as a criterion for diagnosing infection, the usefulness of this criterion was not completely apparent form the data presented.

Gardner et al. evaluated the validity of a checklist of 12 clinical signs and symptoms to identify localized chronic wound infection in a study of 36 patients. All of the patients in this study with noninfected wounds did not exhibit increasing pain as an indicator so that pain has a specificity value of 1.00 (100%). However, only 36% of the infected wounds exhibited this symptom. The percentage agreement between 2 nurse assessors on the assessment of increasing pain was 96% (κ coefficient = 0.73) in a sample of patients with diabetes and foot ulcers. In another study of 115 patients, interobserver agreement on tenderness yielded a κ value of 0.72 with 79% agreement when pain or tenderness was absent and only 13% when present. Gardner also demonstrated that perception of increasing pain was not significantly different between patients with or without diabetes in the presence of wound infection.

Bacterial damage can be divided into superficial and deep compartments. Superficial bacterial damage can be documented through the mnemonic, NERDS: Nonhealing, Exudate that has increased, Red friable granulation, Debris on the surface, and Smell. Superficial infection can be treated topically, whereas deep infection often requires systemic treatment. STONEES identifies the criteria for deep infection and refers to the increased wound Size, increased surrounding skin Temperature, the probing or exposed bone (Os), New or satellite areas of breakdown, Erythema or Edema, Exudate, and Smell (Figure 4). Features to differentiate infection from inflammation are highlighted in Figure 5.

Increasing scientific evidence suggests that cells in stalled chronic wounds are altered phenotypically with an increase in senescent cells that are less responsive to cellular signaling, decreased growth factors, and other diminished cellular responses. There may also be impaired cell activation if bacterial damage is superficial, deep, or both. These 2 paradigms have been studied in some publications and require further validation and modification, but they serve as a theoretical framework to study the effects of various antimicrobial agents. Alternatively, noninfectious processes can produce an inflammatory response that can mimic infection. Features to differentiate infection from inflammation are highlighted in Figure 5.

Figure 4.
NERDS AND STONES

<table>
<thead>
<tr>
<th>Superficial: Treat topically</th>
</tr>
</thead>
<tbody>
<tr>
<td>N.E.R.D.S.</td>
</tr>
<tr>
<td>Nonhealing</td>
</tr>
<tr>
<td>Exudate</td>
</tr>
<tr>
<td>Red friable tissue + bleeding</td>
</tr>
<tr>
<td>Debris</td>
</tr>
<tr>
<td>Smell</td>
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</table>

<table>
<thead>
<tr>
<th>Deep: Treat systemically</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.T.O.N.E.S.</td>
</tr>
<tr>
<td>Size is bigger</td>
</tr>
<tr>
<td>Temperature increase</td>
</tr>
<tr>
<td>Os or Bone (probes, exposed)</td>
</tr>
<tr>
<td>New breakdown</td>
</tr>
<tr>
<td>Exudate, Erythema, Edema</td>
</tr>
<tr>
<td>Smell</td>
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Image for Figure 4.
migration and insufficient angiogenesis to support complete closure with an imbalance favoring tissue destruction modulated by matrix metalloproteases (MMPs). Matrix metalloproteinases are important as part of the inflammatory stage of wound healing, but they become destructive to the wound matrix when a prolonged normal inflammatory stage predominates in a stalled chronic wound. MMPs are a family of structurally related zinc-dependent neutral endopeptidases that are capable of degrading collagen and other components of the extracellular matrix such as fibronectin, vitronectin, elastin, and proteoglycan core proteins. There are currently 24 major known human members of MMP gene family, and they are classified based on their molecular structure and substrate specificity. MMPs are produced by various cells including keratinocytes, fibroblasts, endothelial cells, neutrophils, and macrophages. The proteolytic property of the MMP is important during wound healing to remove debris and facilitate cell migration. Excessive accumulation and activation of MMPs can suppress cell proliferation and angiogenesis due to destruction of growth factors as well as matrix proteins that provide necessary substrates for cell migration and integrity of the tissue.

Wound fluids from chronic ulcers have been shown to be proteolytic partly because of an overexpression and activation of MMPs and partly deficient level of the tissue inhibitors of MMPs. MMPs are also produced by bacteria present in chronic wounds, and these MMPs may cause the characteristic tissue damage observed in critical colonization and infection. To substantiate the proteolytic theory, the levels of MMP-2 and MMP-9 in chronic wound fluid are 5 to 10 times higher than in acute wound fluid extracted from wounds healing at the expected rate.

Moisture Balance

An ideal dressing should be able to maintain a moist wound environment preventing the dressing material from sticking to the wound and causing trauma upon its removal. A highly exudative wound would require an absorptive dressing to ensure drainage containment. Leakage of wound fluid that contains bacteria and inflammatory proteases can increase the risk for damage to the periwound skin. In a randomized controlled trial comparing 2 foam dressings, patients registered higher levels of background pain with macerated periwound areas. The use of a film barrier to protect the periwound area is an effective strategy to promote patient comfort. In contrast, a dry wound should be managed with a hydrating agent because a proteinaceous exudate can become a glue-like substance when dried.

CONCLUSION

Pain is a common symptom for persons with chronic wounds. McCaffery and Pasero remind us that pain is whatever the patient says it is. In this article, the authors modified the WBP paradigm to delineate the various components of wound-related pain. The troublesome pain symptom may evolve from 1 or more components, including cause of the wound, patient-centered concerns, or local wound care, including surgical procedures or dressing change. Optimal pain treatment may improve patient outcomes including wound healing and the physical and emotional state of persons with chronic wounds.

REFERENCES


