

Medical management of chronic wound pain

Chronic wounds require complex care because they have a myriad of causes and complications. A painful, chronic wound often indicates that there is something wrong and that it needs to be corrected. Treating the cause of the chronicity should involve determining the correct diagnosis and initiating appropriate wound pain treatment. The chronic wound pain paradigm outlined in this article provides a foundation on which a structured approach to optimising pain management in chronic wounds can be built. By listening to the patient's perspective, optimal wound and pain management can be combined with successful outcomes.

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KEY WORDS

Chronic wound
Pain
Paradigm
Venous leg ulcers
Diabetic foot ulcers

The study of pain in chronic wounds is in its infancy and there is a paucity of evidence to guide the clinician. Chronic wounds require complex care because they have a myriad of causes and complications.

The 'preparing the wound bed management paradigm' (previously developed by Sibbald et al, 2003b) is a basis for the management of chronic wounds and has been used extensively by wound care specialists. It consists of four major principles:

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1. Treating the cause
2. Addressing patient-centred concerns.
3. Providing local wound care
4. Using advanced therapies when the wound is not healing at the expected rate.

We have integrated the management of chronic wound pain into this wound management paradigm (*Figure 1*). Treating the cause should involve determining the correct diagnosis and initiating appropriate wound pain treatment. Patient-centred concerns must focus on what the patient perceives as the disability caused by the pain, and their willingness to receive treatment. Local wound care needs to revolve around the three pillars of local wound care practice: debridement, bacterial balance/prolonged inflammation, and moisture balance.

The 'preparing the wound bed management paradigm' provides a foundation on which a structured approach to optimise pain management in chronic wounds can be built.

General approach to chronic wound pain Treat the cause

Treating the cause of a chronic wound may involve removing the source of the problem, for example with a venous ulcer oedema can be reduced with compression therapy. This will be

examined further when the types of chronic wounds are discussed more specifically.

Patient-centred concerns

Pain in chronic wounds is a major concern for patients and healthcare professionals (Sibbald, 1998; Neil and Munjas, 2000). In a recent survey of 210 long-term care nurses and nurses who spend 50% of their time in wound care, pain was identified as the third biggest issue after time to healing and limb preservation (Eager, 2005). The patients in this same survey identified pain and quality of life issues as their first concern. Pain control is often more important to patients than it is to healthcare professionals (Queen et al, 2005).

Local wound care

Dressing removal is usually cited as the time when the most pain occurs. Dried out dressings and adherent products are most likely to cause pain and trauma at dressing changes, with gauze removal being the most common cause of this pain. Newer products, such as soft silicone dressings, hydrogels, Hydrofiber® (ConvaTec, Ickenhams), and alginates are less likely to cause pain (Hollinworth and Collier, 2000).

Debridement

Wound debridement can be achieved through surgical, autolytic, enzymatic,

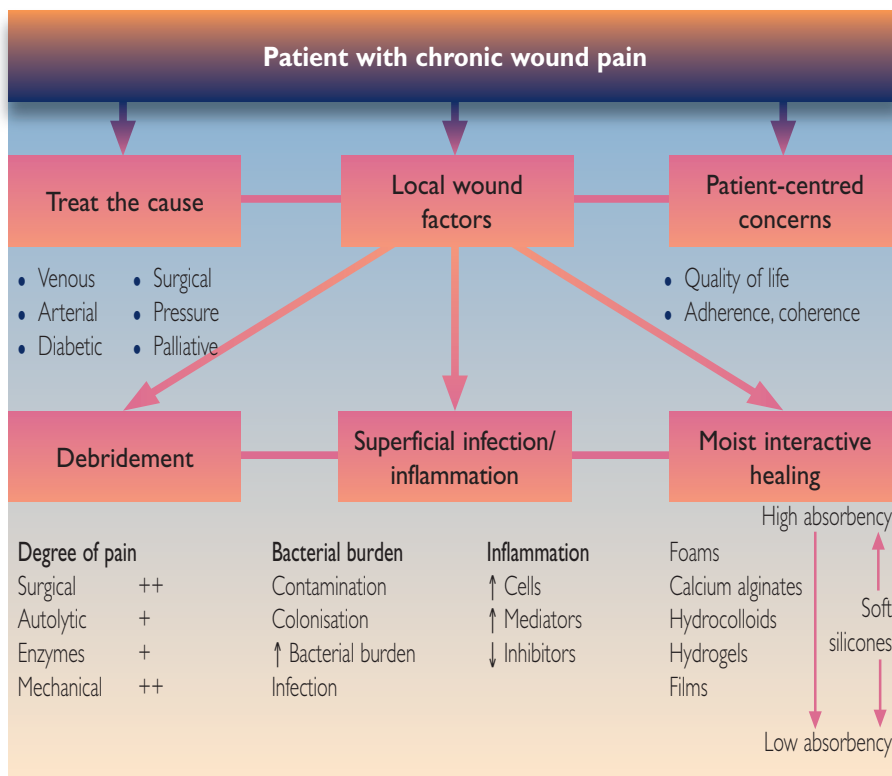


Figure 1. Chronic wound pain paradigm (Adapted from Reddy et al, 2003; Sibbald et al, 2003).

and/or mechanical means (Davies et al, 2005). A number of factors come into play when choosing an appropriate debridement method. Each method can have a negative or positive impact on wound pain. The more aggressive the debridement regimen (e.g. surgical and mechanical), the more potential pain for the patient. In the presence of neuropathy, surgical debridement may be painless, unless deeper structures such as bone are disrupted. Enzymatic and autolytic debridements are slower but are generally less painful, with autolytic methods being essentially pain free.

For surgical debridement, application of topical local anaesthetics, such as 4% topical lidocaine, amethocaine 4% gel or EMLA, 30–60 minutes before the procedure may be helpful. For deeper debridement, injected local anaesthetics should be used. Even though spinal cord injured patients are often insensate in the debridement area, injected local anaesthetics should be used to avoid autonomic dysreflexia that can cause catastrophic elevations in blood pressure or muscle spasms (Weaver et

al, 2006). If oral pain medications are used, the agents should be administered 30–90 minutes before debridement to obtain a therapeutic effect.

Non-pharmacological interventions are also important for debridement. These interventions include proper positioning before the procedure, and creating a relaxed environment for the patient by making them comfortable and giving them a sense of control by allowing them to call a time-out. Pain intensity should be measured with a pain intensity scale, and the roles and responsibilities for pain management should be clarified before debridement (Davies et al, 2005). Environmental pain control strategies include guided imagery, talking to a supportive person, warm sheets, and the ability to call an agreed time-out when the pain reaches a predetermined level.

Superficial infection/inflammation

Infection and inflammation can be painful in themselves (Gardner et al, 2001). Superficial infections may be treated with topical antimicrobials, while deeper infections require systemic agents (Krasner and Sibbald,

1999). Many topical preparations, both pharmacological and non-pharmacological (e.g. antibacterial dressings), exist to treat both infection and inflammation. Silver-containing dressings are both anti-inflammatory and antimicrobial (Wright et al, 2002). Acute inflammation is an important phase in normal healing and, therefore, treatment should be used only in cases of chronic inflammation (Bowler, 2002; 2003). Many of these preparations can be soothing or cooling (ointments/gels), or can create an appropriate environment to reduce or prevent pain (e.g. moist dressings).

Deep infection

It has been shown in numerous studies that chronic wound exudate has an abnormally high concentration of proteases (particularly matrix metalloproteinases, or MMPs) (Mast and Schultz, 1996). These increased proteases shift the wound healing balance into a continuing chronic inflammatory phase. Continued inflammation and tissue injury contributes to chronic wound pain and prevents the wound from transitioning to the proliferative stage. Increased pain in the area of an ulcer is a sign of possible deep infection. The mnemonic: STONES (Size increasing, Temperature elevated, O for probing to bone, New areas of breakdown, E for erythema, exudate, edema and Smell) and ulcers that probe to bone should be investigated for osteomyelitis.

Superficial bacterial burden can be identified with the mnemonic NERDS (Non-healing, Exudate, Red friable granulation, Debris on the surface or Smell). To identify bacterial damage, usually two or three of these symptoms need to be present. This can often be treated topically but if deep- or surrounding skin-invasion is present, the use of appropriate systemic antibiotics is required (Sibbald et al, 2003b). Exudate is often clear before it is frankly purulent. In the presence of exudate and odour, other signs of deep infection are used to clarify if bacterial damage involves the deep compartment as well. Treatment of bacterial invasion often leads to improvement in pain as the infection resolves.

Moist interactive healing

Moist wound healing has been demonstrated to result in faster healing, less scarring, and less pain (Rovee, 1991; Kannon and Garrett, 1995). The pain reduction has been attributed to the bathing of the exposed nerve ending in fluid, preventing dehydration of the nerve receptors.

Other dressing-related factors that can cause pain, include the dressing absorbency mechanism, dressing adherence, and the presence of contact irritants and allergens within the dressing.

The manner in which a dressing absorbs and manages exudate can also result in wound pain. Dressing adherence to the wound can occur if the dressing absorbs too aggressively, or if the primary dressing allows secondary products to adhere to the wound. Adhesion of primary and secondary products can result in the accidental traumatic removal of the primary contact layer, resulting in damage to the wound bed and pain (World Union of Wound Healing Societies [WUWHS], 2004).

Fibrous products (eg. alginates, Hydrofiber®) are excellent primary contact layers. In the presence of wound fluid, these fibres transform into gels to facilitate a moist interactive local wound bed environment and result in a soothing sensation. The retained moisture facilitates non-traumatic removal and generally provides pain relief. Some dressings are abrasive and can cause local friction with movement or adherence to the wound surface (e.g. some traditional gauze-based products). This can be avoided with the appropriate choice of primary dressings (e.g. gels/foams), or the utilisation of an atraumatic wound contact layer (e.g. soft silicone dressings) (Meuleneire, 2002).

Adhesive dressings, as the name suggests, can be traumatic to both the wound bed and peri-wound areas. Their removal can be both painful and damaging, depending on the force necessary to remove the dressing. Films and hydrocolloids should be removed with care and as

recommended by the manufacturer's instructions. To minimise adhesive trauma at dressing change, healthcare professionals are encouraged to stretch the dressing laterally, loosening the adhesive bond before exerting an upward, vertical force away from the skin surface.

Hydrocolloids are generally non-adherent where the contents have gelled and interacted with the surface of the wound bed. They form a soft, conforming gel in the presence of exudate (Fletcher, 2005). However, upon removal, care should be taken to ensure that there is no skin stripping where the adhesive is fixed to the peri-wound skin at the ulcer margin (Eaglestein, 1993; Baxter, 2000).

Many dressings and topical treatments contain allergens that can result in inflammation (i.e. allergic response). These local reactions can be uncomfortable, often resulting in itching or pain. Products with a high sensitisation potential (neomycin, bacitracin, lanolin, perfumes and natural rubber derivatives) should be avoided, particularly on patients with venous leg ulcers, where the sensitisation potential is greatest (Siegel, 2000).

Wound pain from inappropriate local wound care can generally be corrected by substituting a well thought-out local wound care regimen. The elements to remember include the use of dressings appropriate for moisture balance, bacterial balance, controlled inflammation, and autolytic debridement. This wound care regimen should be patient, wound, and disease specific.

Moisture balance

The dressing removal procedure is frequently cited by patients as the time they experience the greatest amount of pain (Hollinworth, 2000; Puntillo et al, 2001; Fauerbach et al, 2002; Kammerlander and Eberlein 2002; Sanders, 2002; King, 2003). Pain at dressing change can be minimised by adopting strategies to reduce pain and trauma, and by choosing dressings with pain-reducing characteristics.

Some strategies to avoid pain with dressing changes include: soaking dressings with saline or even 4% topical lidocaine preparations, amethocaine 4% gel or EMLA before removal; using non-adhesive/non-adherent dressings; or using atraumatic dressing products (e.g. soft silicones). Many patients fear pain with dressing removal, so talking to the patient about their fears or concerns before the event is important. Having a plan for pain management in place before dressing changes helps to alleviate discomfort and anxiety. In addition, pain medications can be given an hour before dressing removal if pain is expected to occur.

Gauze dressings often stick to the wound and are painful. Soft silicone products have been recommended to help minimise pain and trauma on dressing removal (Hollinworth, 2000; Naylor, 2001). Hydrogels and alginates may also minimise pain in patients with mild to moderate exudate.

As with debridement, spinal cord injured patients may also experience pain and/or autonomic dysreflexia with dressing changes. These issues should also be addressed in this patient population.

Venous and leg ulcer pain

Treat the cause

Venous leg ulcers are the most common leg ulcers and increase in frequency with advancing age (Margolis et al, 2002). Although, historically, venous ulcers were thought to be relatively pain free, a significant number of patients will experience pain that impacts on their quality of life (Phillips, 1994; Ryan et al, 2003). Patients may have pain in the absence of an ulcer, and may experience prolonged pain once an ulcer has healed, due to the other components of the underlying venous disease. Venous disease consists of a spectrum of changes that slowly evolve over time. A variety of pain symptoms are associated. These range from discomfort and aching to deep, chronic pain. Acute, disabling pain may develop at each stage, and will impact on the plan of care for the patient.

Patients with early stages of venous disease (i.e. pitting oedema and prominent varicose veins) will often describe a dull aching or heaviness in their legs that worsens towards the end of the day, or after prolonged periods of standing, as the oedema or fluid accumulation is maximal towards the end of the day. Support stockings, walking, and leg elevation while sitting will often relieve these symptoms. Attention must also be paid to other factors contributing to their venous stasis disease, such as obesity, sedentary lifestyle and other co-morbid illnesses. Once venous ulcers have healed, oedema control is essential for pain management and patients are advised to wear support stockings for life to control the oedema and prevent recurrence of ulcers.

Co-existing arterial disease must be ruled out, either clinically or by Doppler measurement of the ankle brachial pressure index (ABPI). Use of inappropriate compression bandaging in a patient with arterial insufficiency can lead to worsening of the ulcer and

more serious complications. Ulcers related to arterial vascular disease have been traditionally considered to be painful; however, this feature alone is not adequate to differentiate a venous stasis ulcer from an ulcer resulting from arterial disease.

Long-standing pitting oedema leads to pigmentary changes, related to deposition of haemosiderin from extravasated red blood cells on the distal one-third to one-half of the legs. Over time, this pigmented area becomes sclerotic due to the leakage of fibrin. Lipodermatosclerosis consists of the triad of non-pitting oedema, sclerosis, and pigmentation. Pain is found in up to 43% of patients with lipodermatosclerosis, even without ulceration (Bruce, 2002). Lipodermatosclerosis may be further divided clinically into acute and chronic forms. The acute form consists of painful erythema mimicking an acute cellulitis with non-pitting oedema, in contrast to the chronic form, consisting of only sclerosis and pigmentation changes. Acute lipodermatosclerosis

is sometimes mistaken for cellulitis, especially if it is unilateral. However, venous stasis changes are rarely unilateral, though the disease may have some asymmetry in terms of severity.

Management of lipodermatosclerosis includes: compression bandaging or support stockings; topical medications, including topical steroids and lubricants for skin surface erythema and dryness; and oral medication, including non-steroidal anti-inflammatory drugs (NSAIDs) (for pain), or pentoxifylline (for the woody fibrosis).

Support stockings may be adequate to control the symptoms of lipodermatosclerosis; however, non-steroidal anti-inflammatory medications may be needed for both their analgesic and anti-inflammatory properties. Adverse effects to this class of drugs must, of course, be considered before their use, particularly in the elderly (gastrointestinal haemorrhage, decreased renal function and increased cardiovascular risk).

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 localised portion of an inflamed and tender vein. The pain is often aggravated by palpation or standing, and the involved area may be quite warm to touch. The long saphenous vein is most commonly involved.

Treatment options for superficial thrombophlebitis include compression, walking and NSAID therapy. The role of the newer Cox-2 inhibitors has not yet been established in the management of superficial phlebitis. When patients with superficial thrombophlebitis are screened, an association with deep vein thrombosis is uncommon and it is unnecessary to look for the latter unless other risk factors are present (Bounameaux and Reber-Wasem, 1997).

Another factor that complicates venous disease and venous ulcers is

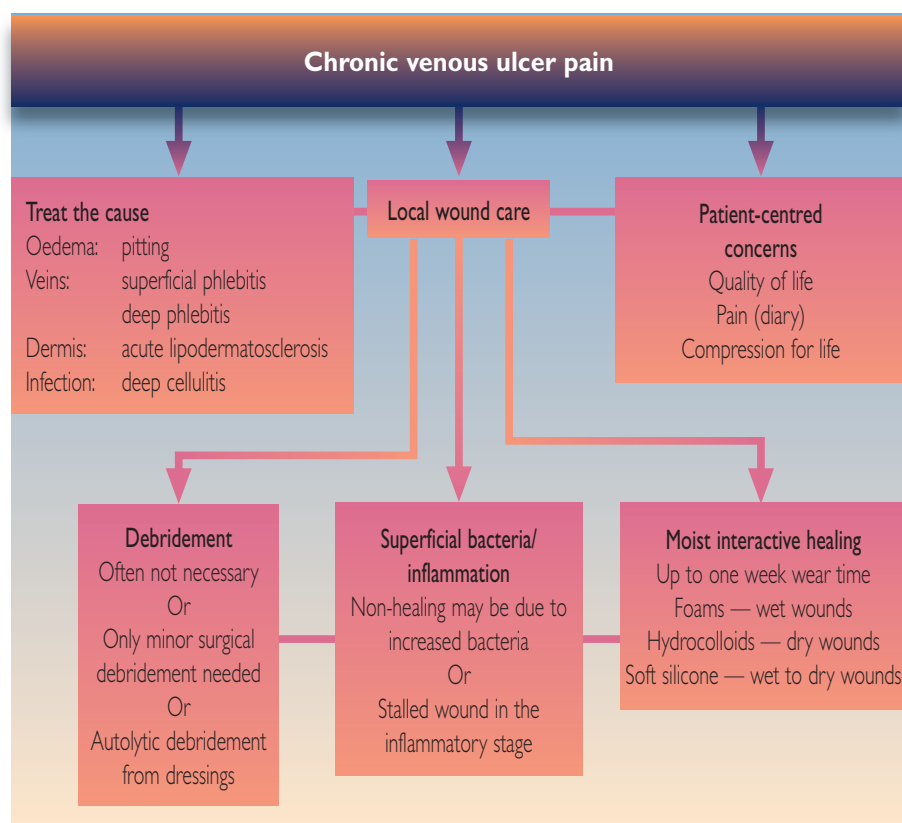


Figure 2. Venous ulcer pain paradigm. (Adapted from Ryan et al, 2003).

Table 1.
Differential diagnosis of venous pain

Diagnosis	Clinical/investigation	Treatment	Comments
Pitting oedema	Dull ache at end of day. Press thumb into skin and note degree of depression Grade 1+ to 4+	Compression bandaging, support stockings, walking, exercise. Improve calf muscle pump	Non-elastic stockings or compression bandaging may initially be preferred as they are less likely to cause pain at rest
Superficial phlebitis	Pain and tenderness along affected vein — usually saphenous	Compression, ambulation, NSAID therapy	Risk of associated underlying DVT is low, especially if affected area is below the knee
Deep vein thrombosis (DVT)	Acute, red, tender, swollen calf — almost too painful to touch. Doppler necessary to confirm diagnosis	ASA, unfractionated heparin, warfarin. Low-molecular weight heparin. Bed rest	Suspect a DVT in patients with a sudden increase in calf pain, with risk factors such as immobilisation, recent surgery, oral contraceptives, etc
Acute lipodermatosclerosis	Diffuse, purple-red swollen leg resembling cellulitis, aching and tenderness is common	Compression bandaging, support stockings, NSAIDs	Usually bilateral, although may be more prominent on one leg. Compression therapy essential
Chronic lipodermatosclerosis	Diffuse, brown sclerotic pigmentation with widespread chronic pain	Same as with acute lipodermatosclerosis, but with topical steroids and lubricants. Pentoxifylline	Support stockings may have to be custom made to accommodate for leg shape
Wound infection	Change in pain character associated with other clinical signs of infection	Topical antimicrobial agents and oral antibiotics, if indicated	Maintain bacterial balance, and watch for increase in pain, size, exudate, odour, or changes in granulation tissue as signs of infection
Cellulitis	Diffuse, bright red, hot leg, usually unilateral, associated with tenderness and often fever	Oral antibiotics, with IV antibiotics needed for severe episodes or with low host resistance	Venous ulcers may make individuals more prone to cellulitis
Atrophie blanche	Pain, stellate, white scar-like areas associated with pain at rest and standing	NSAID therapy. Other analgesics	May be seen in association with scars of healed ulcers, or may be an independent clinical feature
Acute contact dermatitis	Itching, burning red areas on leg corresponding to area of use of topical product	Remove the allergen. Apply topical steroids	Lanolin, colophony, latex, neomycin are some of the more likely agents involved

the presence of dermatitis, particularly if it surrounds the wound (Reichert-Penetrat et al, 1999; Patel et al, 2001). Therefore, products used on the distal legs of patients with venous stasis must be relatively free of potential contact allergens and irritants (Wilson et al, 1991). Adhesives, tulle or paraffin gauze, topical lubricants and emollients, topical antibiotics, and other wound and peri-wound products all contain agents that can lead to contact dermatitis (Osmundswen, 1982; Floyer and Wilkinson, 1988; Zaki et al, 1994; Lopez Saez et al, 1998; Gooptu and Powell, 1999; Dong et al, 2002). Patients will complain of burning and

itching, usually in the area of product application. Discomfort in association with eczematous changes, or a change in the type of pain, suggests irritation or allergic contact dermatitis (Anderson and Rajagopalan, 2001).

Atrophie blanche is one of the end stages of venous disease and consists of distinct white, sometimes star-shaped, depressed scar-like areas (Shornick et al, 1983). It is felt to develop spontaneously and is frequently associated with severe, sharp pain. Management of atrophie blanche pain can be challenging, and may require systemic analgesic medication.

The approach to venous ulcer pain must include an assessment of the cause of pain and the pain characteristics (constant or intermittent). Treatment can be local (eg. dressings, topical anaesthetic agents), regional (support rather than compression bandaging), or systemic (NSAIDs or other intermittent or long-acting pain medications).

Patient-centred concerns

Although venous ulcers have historically been described as painless, it is now known that a significant number of patients with venous ulcers will experience pain that impacts on their quality of life (Krasner, 1998).

Local wound care

There are many ways to debride a venous ulcer. Sharp surgical debridement is often not necessary and when performed may be quite painful. Topical anaesthetics, such as topical xylocaine (1–4%) preparations, EMLA or amethocaine 4% gel have been used clinically in appropriate settings before debridement to improve pain (Lok, 1999). Autolytic debriding agents are useful, particularly hydrogels or hydrocolloids, and may actually help reduce the pain. Mechanical debridement using wet-to-dry dressings is generally too painful to be tolerated, and more beneficial alternatives exist. Enzymatic debriding agents can be used for thick adherent eschar as an alternative to hydrocolloids or hydrogels.

Diabetic foot ulcer pain

Treat the cause

Persons with diabetic foot ulcers require not only adequate vasculature and infection control, but also pressure downloading or redistribution. In contrast to ischaemic pain, diabetic neuropathic pain is not generally associated with increased activity, but often appears during periods of diminished external sensory stimulation (e.g. at night). Because of its multifactorial nature, no single treatment has proven a panacea (Jensen and Larson, 2001; Simmons and Feldman, 2002). Neuropathic pain often responds incompletely to traditional anti-inflammatory and opioid pain medication (Gilron et al, 2005). Tricyclic antidepressants have been used successfully, especially for treatment of burning pain. Second generation tricyclics with high noradrenaline activity, such as nortriptylene or desipramine, are often the preferred choices. Newer anti-epileptic drugs, such as gabapentin or pregabalin, have also been used with success.

Although neuropathic pain may occur in these patients, pain is not common in the diabetic foot ulcer itself. When ulcer pain does occur, it may herald the onset of limb-threatening complications such as

critical ischaemia, deep infection, or Charcot arthropathy.

Critical ischaemia

Pulses are generally palpable in the foot with around 80 mmHg systolic pressure in the dorsalis pedis artery. In patients with diabetes and in elderly patients without diabetes, the ankle brachial pressure index (ABPI) may be falsely high because of calcified arteries. Therefore, further non-invasive tests, such as toe pressure or transcutaneous oxygen pressure, are necessary. Toe pressure over 50 mmHg, or transcutaneous oxygen pressure over 300 mmHg, are usually required to ensure sustained healing. In extreme cases, ischaemia can cause pain at rest. These patients require a consultation with a vascular surgeon for possible surgical reconstruction, or arterial dilation to improve flow (Gibbons et al, 1993; Akbari and LoGerfo, 1999; Akbari et al, 2000).

Deep infection

Wound infections, particularly those involving deep plantar spaces, can

cause pain even in the presence of severe neuropathy. Unfortunately, this pain is often overlooked since signs and symptoms of infection in the person with diabetes are often subtle. The presence of non-neuropathic pain in even a mildly erythematous or oedematous diabetic foot, even with a normal X-ray of the foot, requires assessment and, if necessary, treatment (Sibbald et al, 2003a). Peri-ulcer erythema greater than 2cm represents limb-threatening changes (Caputo et al, 1997). Patients may have new areas of breakdown and ulcer enlargement as their only indicators of deep infection. The extension or probing of the ulcer to bone, in concert with serial radiography or magnetic resonance imaging (MRI), may be an effective means to evaluate the potential extent of bony involvement and osteomyelitis (Grayson et al, 1995; Tomas et al, 2000).

Charcot arthropathy

Charcot arthropathy is characterised by pathologic fractures, joint dislocation and foot deformity in

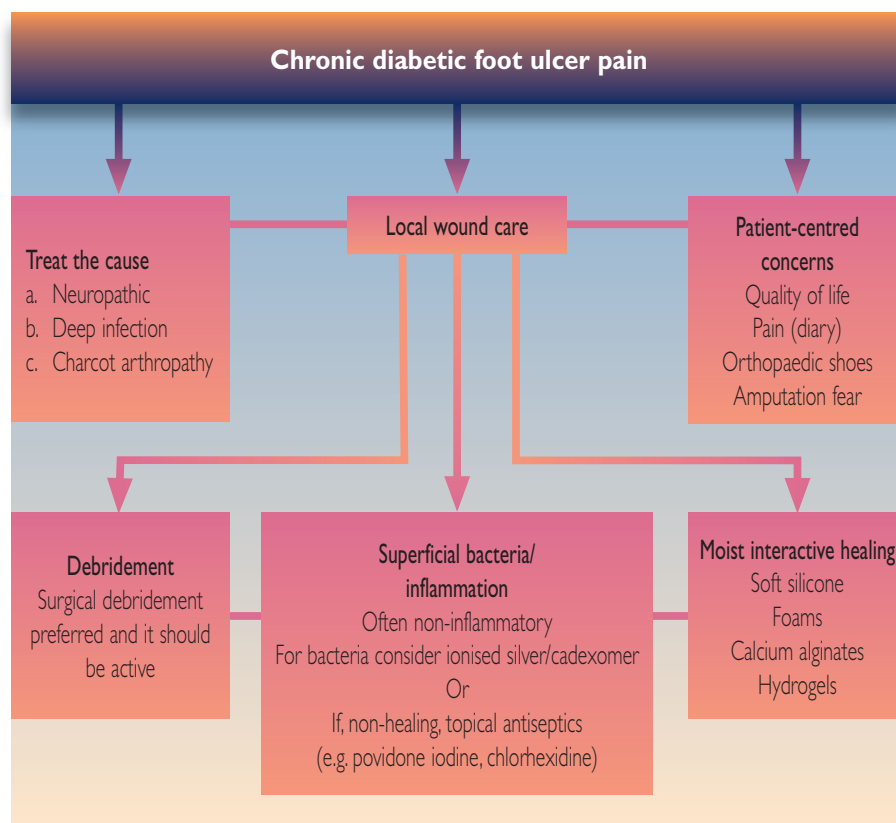


Figure 4. Diabetic foot ulcer pain paradigm (Adapted from Sibbald et al, 2003).

Table 2.
Diabetic foot ulcer pain

Neuropathic	Charcot foot	Deep infection			
<p>Neuropathic pain is due to irritation or damage of the nerve fibres</p> <p>There are 3 components to the neuropathy:</p> <p>Sensory — test with monofilaments ↓ sensation Autonomic — check for dry skin (DDx tinea) Motor — ↓ reflexes, claw toes</p> <p>Nerve irritation often causes burning (try tricyclics)</p> <p>Nerve damage often causes shooting or stabbing pain. Often tricyclics are not successful and gabapentin may be the treatment of choice</p>	<p>Recurrent minor trauma (not perceived due to neuropathy)</p> <p>↓</p> <p>Minor fractures</p> <p>↓</p> <p>Continuous trauma results in disintegration</p> <p>↓</p> <p>Contact cast (or other off-loading)</p> <p>↓</p> <p>Monitor: Swelling Warmth (skin temperature) Biochemical markers of bone formation</p>	<p>Deep infection needs to be distinguished from superficial infection. The following signs and symptoms are often useful.</p> <table border="1"> <tr> <td rowspan="2"> <p>Superficial (try topical treatment x 2 weeks)</p> </td> <td> <ul style="list-style-type: none"> • Non-healing • ↑ Exudate • Bright red granulation • Friable exuberant granulation • New areas of slough </td> </tr> <tr> <td> <p>Deep (need systemic treatment)</p> <ul style="list-style-type: none"> • Pain • New areas of breakdown • Dermal erythema > 2 cm • Warmth • Probe to bone </td> </tr> </table>	<p>Superficial (try topical treatment x 2 weeks)</p>	<ul style="list-style-type: none"> • Non-healing • ↑ Exudate • Bright red granulation • Friable exuberant granulation • New areas of slough 	<p>Deep (need systemic treatment)</p> <ul style="list-style-type: none"> • Pain • New areas of breakdown • Dermal erythema > 2 cm • Warmth • Probe to bone
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	<p>Deep (need systemic treatment)</p> <ul style="list-style-type: none"> • Pain • New areas of breakdown • Dermal erythema > 2 cm • Warmth • Probe to bone 				

patients with severe neuropathy (Sanders and Frykberg, 1991; Sanders and Frykberg, 1993; Armstrong et al; 1997; Armstrong and Lavery, 1998), and is aggravated by weight bearing. Pain is the presenting complaint in 75% of cases, along with a red, hot, swollen foot. The treatment of Charcot arthropathy requires pressure downloading or offloading until the skin surface temperature becomes normal (Armstrong and Lavery, 1997).

Patient-centred concerns

Pain in the neuropathic foot often reflects a deep infection or acute Charcot arthropathy, and places the patient at higher risk for amputation. Detection of diabetic foot problems often occurs late, and the human and economic cost of diabetic foot ulcers and amputation remains high (Wishner and Rubin, 2001). Individuals with diabetes need to be informed about the risks and complications, understand

the loss of protective sensation (LOPS), and be taught the problem-solving skills necessary to respond to health-related problems. Dressings and pressure offloading devices are often expensive and the healthcare professional may need to consult with appropriate agencies to facilitate access to such treatments for their patients.

Local wound care

Debridement is usually painless due to underlying neuropathy. If pain is present, ischaemia, deep infection or Charcot arthropathy should be considered. Although sharp surgical debridement is the procedure of choice (Steed, 1995), it should only be performed when blood supply is adequate for healing. Debridement should include removal of callus and friable bright red or exuberant granulation. Calcium alginate dressings post debridement will help control surface bleeding and prevent the

formation of a haemorrhagic crust. Autolytic, mechanical and enzymatic debridement are less effective.

Superficial infection may cause local pain or discomfort due to the release of pre-inflammatory mediators by the bacteria and the host, but it is much more likely that pain is indicative of infection in the deeper compartment. Signs and symptoms of increased bacterial burden have been reviewed with the mnemonic NERDS for superficial compartment increased bacterial burden, and STONES for the deep or surrounding skin infection.

Some patients with diabetes may have decreased inflammatory response that delays healing, particularly with poor diabetic control and a high glycosylated haemoglobin value. All persons with diabetes have a relative immune deficiency that decreases host resistance and results in an increased

damage from the effect of bacteria within the chronic wound.

Excess moisture in diabetic foot ulcers leads to macerated callus around the edge of the wound edge and may favour bacterial proliferation (Edmonds, 2006). This often results in increased pain. Moisture control is therefore crucial, and best achieved through moisture absorptive dressings, such as foams, Hydrofiber®, and calcium alginates, along with callus debridement. If the wound edges are macerated, protection around the outer rim can be accomplished through application of zinc oxide paste, petrolatum, film-forming liquid acrylates, windowed hydrocolloid or film dressings (Romanelli et al, 2003).

Pressure ulcer pain

Treat the cause

Pain is the end result of tissue damage and pressure ulcer formation, but the pain may also contribute to further immobility. Spinal cord injured, elderly, and cognitively-impaired patients with pressure ulcers present special challenges in pain management. Up to 80% of nursing home residents with pressure ulcers have significant pain that is under treated (American Geriatrics Society [AGS], 2002; Reddy et al, 2003a). Although age alone is not a risk factor for increased pain, age has been identified as a key risk factor for inadequate pain management (Cobbs et al, 1999; AGS, 2002).

Pressure ulcers develop from unrelieved pressure, often over bony prominences. This pressure often exceeds capillary closing pressure, resulting in tissue ischaemia. Tissue ischaemia itself leads to pain through the release of inflammatory mediators. Pressure relief and reduction provided by support surfaces are components in the treatment of the cause of the ulcers. These surfaces often off-load pressure from bony prominences, but they may also aid in pain management. Seating and positioning assessments by trained professionals are an important part of pain management in persons with pressure ulcers. All institutionalised patients should be on pressure

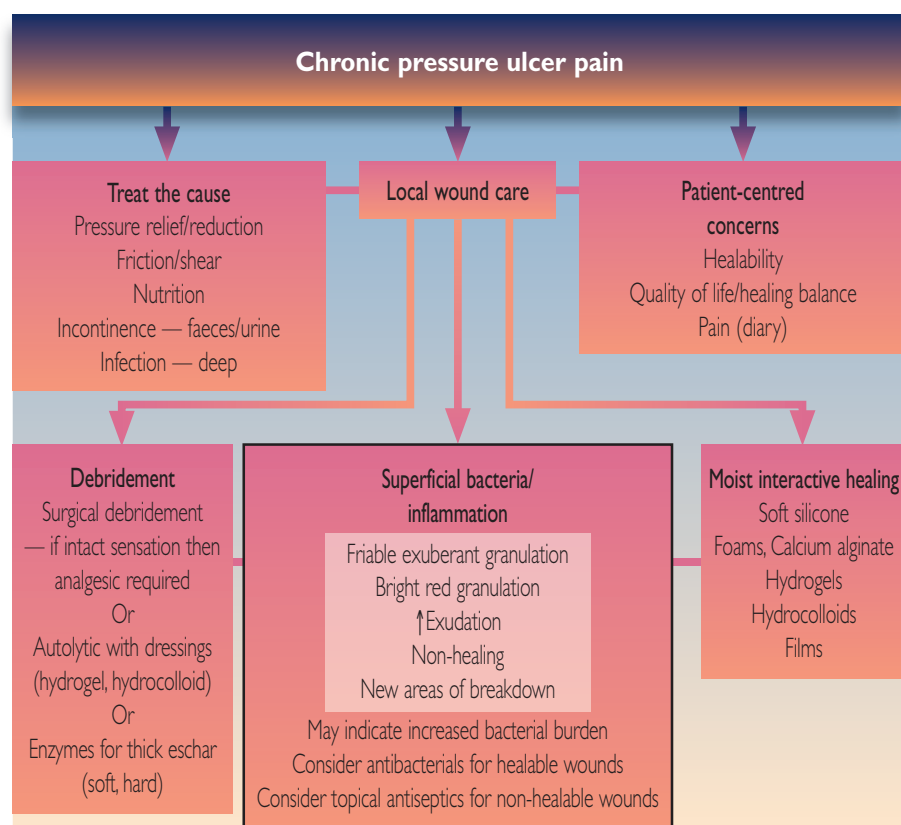


Figure 5. Pressure ulcer pain paradigm (Adapted from Reddy et al, 2003b).

reduction foam mattresses and seating surfaces to prevent pressure ulcers and to provide comfort.

Nutritional support is also important in pressure ulcer healing and pain management. Healing of pressure ulcers is compromised by moisture from incontinence of stools and urine, friction and shear, decreased mobility and poor nutritional status (Green et al, 1999; Cunha et al, 2000; Thomas, 2001). Poor nutrition leads to decreased muscle mass and wasting of protective subcutaneous fat, and patients with significant muscle wasting tend to experience greater pain. Nutritional demands for protein and calorie intake are actually greater in patients with pressure ulcers, and nutritional consultation is important for optimal management (Russell, 2001; Westergren et al, 2001; Todorovic, 2002).

Pressure ulcers may also cause damage or irritation to peripheral nerves, leading to neuropathic pain. The burning pain of nerve

irritation may respond to tricyclic antidepressants, while the stabbing pain of nerve damage may require anticonvulsants, such as gabapentin or pregabalin.

Regrettably, paralysis does not mean freedom from pain. Patients with spinal cord injuries may experience spasms, leading to damaging friction and shear in locations such as the heels, which may result in skin breakdown. The anti-spasmodic, baclofen, can be used to help control spasms; however, a referral to a specialist in physical medicine and rehabilitation may be beneficial for optimal patient care.

Patient-centred concerns

The patient and healthcare professional should work together to define common goals of care, and to define their respective roles and responsibilities in the therapeutic relationship. Persons with pressure ulcers and healthcare professionals may decide that it is not reasonable to set healing as a goal for the patient, given declining

Table 3.
Pressure ulcer pain management strategies

Cause of pain	Definition	Treatment	Comments
Pressure	A force applied to the skin and underlying tissues which inhibits blood flow when it ↓ pressures within the capillary. Capillary closing pressure (CCP) = 32 mmHg (average value)	<p>Special surface: Pressure reduction: ↓ pressure, but not necessarily below CCP, eg. high density foam, standard hospital mattresses synthetic (not sheepskin heel booties) Pressure relief: ↓ pressure below CCP, eg. dynamic flow beds, turning schedules critical</p>	<p>Consider: Pressure mapping Wheelchair assessment</p>
Friction	Created by movement of the patient over a surface	<p>Heel booties, eg. sheepskin booties (do not reduce pressure but may decrease friction) Careful with patient transfers (use turning sheet) Hydrocolloid over bony prominences</p>	<p>On its own, friction does not usually cause ulcers. However, friction in addition to pressure greatly increases risk of ulcer development</p>
Shear	Adjacent body surfaces slide across each other (as happens when a patient slides down a bed)	<p>Keep head of bed ≤ 30 degrees. Tilt seating, rather than recline (may need some recline if slumping is a problem)</p>	<p>Again, more dangerous when combined with pressure</p>
Moisture	Irritation and maceration from sweating, stools, urine, wound drainage	<p>Prevention: If urine incontinent — bladder training; urinary catheter (in and out rather than permanent, if possible) Change undergarment pads frequently Local wound care to keep surrounding skin from maceration Absorbent surface next to skin Treatment: Barriers to wound edge (eg. zinc oxide, petrolatum, occlusive dressings) May need anti-diarrhoea medications If surrounding dermatitis, may need steroid cream/ointment</p>	<p>Can also use film-forming topical acrylate liquids Watch for secondary yeast, especially in folds</p>
Peri-ulcer irritation	Can be due to dermatitis, maceration, and/or infection (eg. candida)	<p>To reduce peri-ulcer irritation, apply either vaseline, zinc oxide, film-forming liquid (eg. acrylate), occlusive dressings (picture frame technique) and, if candida suspected, hydrocortisone in canestan cream, twice daily</p>	<p>If hydrocortisone in canestan is being easily wiped off with body positioning and movement, then cover with zinc oxide</p>
Nutrition	Both pain and healing may be exacerbated if a patient has low muscle mass	<p>Major risks: low overall dietary intake, especially protein. Consult dietician. Consider supplements</p>	<p>No particular evidence that certain vitamins produce healing. Check albumin and total lymphocyte count</p>
Deep infection	Infection requiring systemic agents	<p>Probe to bone — osteomyelitis unless proven otherwise, may need long-term antibiotics</p>	<p>Probe ulcer. Make sure it does not probe to bone and there are no sinus tracts or fistulas</p>
Neuropathic/radicular	Nerve irritation, damage	<p>Consider tricyclic antidepressants (nortryptiline), anticonvulsants (gabapentin)</p>	<p>Consider referral to anaesthesia for nerve blocks</p>
Spasms	Involuntary muscle movement	<p>May need baclofen</p>	<p>Consider referral to psychiatry</p>

health status or other related factors, such as treatment side-effects and availability. Instead, the focus may be on pain control, management of infection, exudate, odour, and improved quality of life. Non-healable

wounds may require the use of topical antiseptics with low tissue toxicity and sensitisation potential.

Local wound care

Although they may not be able

to effectively communicate their discomfort, severely demented patients who are bed-bound and develop pressure ulcers still feel pain with debridement. It is important to use simple pain assessment scales

(such as a faces tool), or to watch for non-verbal pain clues, such as agitation or facial grimacing. As previously discussed, patients with paralysis may still experience pain with debridement. It is important to discuss any unpleasant experience with each patient, as sensations differ between individuals. Some patients with spinal cord paralysis will require local anaesthetic prior to debridement to prevent autonomic dysreflexia. Severe muscle spasms with previous interventions are a clue that local anaesthetic use may be beneficial.

Conclusion

Pain is what the patient says it is. Optimal patient care must combine environmental and non-pharmacological interventions with appropriate pharmacological interventions, through an appropriate analysis of patient-centred concerns (Jacox et al, 1994; Ferrell, 1996). Assessment of pain must include the treatment of the cause and in this article we have discussed venous, diabetic foot and pressure ulcer related diagnoses and treatment. Local wound care may be slightly different in the three different causes of chronic wounds, but they all must consider tissue debridement, superficial increased bacterial burden or prolonged inflammation and moisture balance as components of optimal wound bed preparation. A painful, chronic wound often indicates that there is something wrong and it needs to be corrected. Patients need to be empowered to discuss pain with healthcare professionals in a non-threatening environment of co-operation. Advanced knowledge of the principles of pain management for healthcare professionals needs to be coupled with the ability to prescribe the appropriate pharmacological agents, and an interprofessional team to optimise care. By listening to the patient's perspective, optimal wound and pain management can be combined with successful outcomes. Even though ulcer healing is not always possible, we can improve the activities of daily living and quality of life of most of our patients. **WUK**

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Key Points

- ▶▶ The study of pain in chronic wounds is in its infancy and there is a paucity of evidence to guide the clinician.
- ▶▶ Treating the cause should involve determining the correct diagnosis and initiating appropriate wound pain treatment.
- ▶▶ Patient-centred concerns must focus on what the patient perceives as the disability caused by pain, and their willingness to receive treatment.
- ▶▶ Local wound care needs to revolve around the three pillars of local wound care practice: debridement, bacterial balance/prolonged inflammation, and moisture balance.
- ▶▶ It is not always possible to heal a chronic wound, but the activities of daily living and quality of life of patients can be improved through optimal wound and pain management.

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