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- Wound healing
- Biofilms in chronic wounds
- Wound-related pain

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Aims and intended learning outcomes

This article is aimed at exploring the key principles that underpin the safe and effective management of patients experiencing wound-related pain. It discusses pain mechanisms and seeks to explain why and how wounds are experienced as being painful. The practice of wound-related pain assessment is considered and the article concludes by considering how best to manage such pain.

After reading this article and completing the time out activities you should be able to:

- Identify the parts of the nervous system that are involved in the perception of wound-related pain.
- Differentiate between nociceptive and neuropathic pain.
- Consider the benefits of assessing and treating wound-related pain.
- Explore the range of interventions that can be used to minimise wound-related pain.

Introduction

The management of wound-related pain has received increasing attention over the years. In the past seven years, five guidance documents aimed at practitioners caring for patients with painful wounds have been issued (European Wound Management Association (EWMA) 2002, Wounds-UK and Mölnlycke Health Care 2004, World Union of Wound Healing Societies (WUWHS) 2004, 2007, Coloplast 2008).

This article aims to provide an overview of the clinical features, assessment and treatment of wound-related pain. It outlines the extent to which the management of both acute and chronic wound-related pain aids recovery from ill health and improves quality of life. The complex and subjective nature of the pain experience is discussed and a brief overview of the physiology of wound-related pain is presented. The article also explores the assessment and treatment of wound-related pain.

Types of wound-related pain

A range of terminologies has been used to describe the conditions under which a patient experiences wound-related pain. Figure 1 shows the WUWHS (2004) approach to classifying wound pain in terms of underlying causes. The approach highlights four main types of pain – operative, procedural, incident and background – and also acknowledges the role played by environmental and psychological factors in pain perception. Alternative terminologies for wound
pain are found in the Wound Pain Management Model, which uses the descriptors temporary, persistent and procedure-related to categorise types of pain. In this model, categories of pain type are not mutually exclusive and patients with chronic wounds may experience a number of different types of pain on different occasions (Price et al 2007).

**Acute wound-related pain**

Wounds are generally categorised as either acute or chronic. Acute wounds, such as incision wounds following surgery, thermal burn injuries or traumatic wounds, are initially extremely painful, but often become less painful as healing progresses. The rate of healing is dependent on many variables, including age, nutritional status and concomitant illness, and there is some evidence to suggest that pain-induced stress may be a barrier to wound healing (Soon and Acton 2006). Research conducted on acute wounds such as blisters and other types of superficial skin wounds suggests that psychological stress is associated with an increase in wound repair times, and that such delay is likely to be attributable to lower levels of pro-inflammatory cytokines secondary to depressed immune function (Soon and Acton 2006). Various physiological and behavioural responses are associated with acute pain. Pediani (2001) cited the adverse effects of poorly managed post-operative pain. These include:

- Decreased respiratory movement.
- Delayed mobilisation following surgery.
- Increased activity in the sympathetic nervous system.
- Changes in hormonal and metabolic activity.

Pediani (2001) also suggested that decreased blood flow, resulting from activity in the sympathetic nervous system, may be detrimental to healing because the deposition of collagen is adversely affected by a diminished oxygen supply to the wound. Choinière (2001) suggested that the inadequate management of procedure-related pain evokes an anticipatory conditioned response to pain. Although this was originally related to the management of burn pain, it could be applied to a number of painful wounds. Choinière (2001) also highlighted the link between pain management, psychological resilience and recovery, suggesting that psychological resilience is likely to decline and fatigue levels increase when the experience of discomfort and distress persists.

### FIGURE 1

**Causes of wound pain**

- **Operative**
  - Cutting or prolonged manipulation of tissue that usually requires an anaesthetic, for example debridement or major burns dressing.

- **Psychosocial factors**
  - For example, age, gender, culture, education, mental state – anxiety, depression, fear, loss or grief.

- **Procedural**
  - Routine or basic interventions, for example dressing removal, wound cleansing, dressing application.

- **Environmental factors**
  - For example, timing of the procedure, resources, setting - level of noise or positioning of the patient.

- **Incident**
  - Movement-related activities, for example friction, dressing slippage, coughing.

- **Background**
  - Persistent underlying pain as a result of wound aetiology or local wound factors, for example ischaemia or infection.

(Adapted with permission from the World Union of Wound Healing Societies 2004)
Chronic wound-related pain Chronic wounds are associated with prolonged healing, which is usually secondary to a combination of local, regional and systemic factors that prevent an ordered sequence of healing (White 2008a). Pressure ulcers and leg ulcers are common types of chronic wounds, both of which have been consistently documented as being associated with significant pain and discomfort (Hopkins et al 2006, Briggs et al 2007, Spilsbury et al 2007). Chronic wound-related pain is likely to have a complex aetiology (as discussed later) (White 2008b). Briggs (2006) highlighted the poor recognition of wound-related pain, which, it is suggested, is likely to be experienced by between half and three quarters of patients with leg ulcers. The experience of chronic pain may have a negative effect on quality of life, mental health, sleep quality and physical and social activity (Palfreyman 2008).

The American Pain Society’s (1999) assertion that pain is the fifth vital sign signifies the importance of pain as an indicator of bodily dysfunction or disease. In the context of wound care, there is general consensus that pain is a reliable indicator of localised wound infection and/or inflammation (Woo et al 2008). However, in chronic wounds, pain may be attributed to causes other than infection and inflammation. Grocott et al (2008) cited the example of leg ulcers and suggested that additional causes of wound pain include macerated or eczematous peri-wound skin, phlebitis or thrombosis, underlying bone pathologies, tight bandages and uncomfortable dressings.

Defining pain

It is widely acknowledged that the experience of pain is subjective and personal. This idea is supported by the explanation that environmental, social, psychological, developmental, emotional and cultural factors are important mediators of the pain experience (MacLellen 2006). The most widely cited definition of pain states that pain is ‘an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage’ (International Association for the Study of Pain (IASP) 1994).

The concept of pain as a psycho-physiological phenomenon developed from work undertaken by Melzack and Wall (1965), which led to the development of the Gate Control Theory, followed by a three-dimensional model of pain (Melzack and Wall 1982, Briggs 2006, MacLellen 2006).

The Gate Control Theory was important because it described the transmission and modulation or modification of nociceptive signals from the peripheral and central nervous system (Briggs 2006) and suggested that emotional and cognitive elements were important in the experience of pain.

The three-dimensional model of pain (Melzack and Wall 1982) identifies the sensory, affective (emotional) and cognitive dimensions of pain. The sensory dimension of the pain experience relates to the physical sensation of pain; the emotional part relates to how the experience of pain affects a person’s emotional state; and the cognitive dimension relates to how a person makes sense of or explains the pain experience (Briggs 2006).

Nociceptive pain Nociceptive pain is common and occurs in everyday life. It is the type of pain
experienced when trapping a finger in the door, sustaining a paper cut or during a collision on the sports field. This type of pain subsides over time and is usually relieved by analgesia (Hollinworth 2005). Nociceptive pain is considered to be an appropriate physiological response to a noxious stimulus.

A nociceptor is a receptor that is preferentially sensitive to a noxious stimulus (IASP 1994). Nociceptors (sometimes referred to as pain receptors) are located at the distal end of sensory neurons, which transmit external information from the peripheral nervous system to the central nervous system via the spinal cord.

Following acute tissue injury or in the presence of an ongoing inflammatory state, nociceptors become physiologically sensitised. This results in a decreased nociceptor firing threshold and increases the responsiveness of sensory or afferent neurones (Wulf and Baron 2002). Hence, the presence of an inflammatory state within a wound will render a wound more painful and more susceptible to induced pain than in normal tissue (Clay and Chen 2005). This increased sensitivity may be experienced both in the wound itself (primary hyperalgasia) and in the surrounding skin (secondary hyperalgasia) (WUWHS 2004). The IASP (1994) defines hyperalgasia as an increased response to a stimulus, which is usually painful. The fact that hyperalgasia develops as a response to continued transmission and perception of pain signals, underlines the importance of good pain control when caring for patients who are experiencing pain.

Neuropathic pain
Neuropathic pain (sometimes referred to as neurogenic pain) has been defined as an inappropriate response caused by a primary lesion or dysfunction in the nervous centre (WUWHS 2004). The causes of neuropathic pain are less well understood (Clay and Chen 2005), but nerve damage as a result of trauma, infection, metabolic disorders or cancer are often implicated in this type of pain (WUWHS 2004). The symptoms associated with neuropathic pain are fairly well documented and include burning, tingling, electric or shooting sensations (Hollinworth 2005, MacLellan 2006).

Neuropathic wound-related pain is considered more difficult to manage than nociceptive pain and usually requires specific drugs such as low dose antidepressants and anticonvulsants to dampen the hyperactivity of damaged nerves (Briggs et al 2007).

Allodynia is a neuropathic condition in that it occurs in patients with lesions of the nervous system where touch, light pressure or moderate cold or warmth evoke pain (IASP 1994). It is different to hyperalgasia in that a pain response is elicited from a stimulus that does not normally provoke pain. In other words, the original modality of the stimulus is non-painful, but the response is painful (IASP 1994). Tests to determine the presence of wound-related allodynia include the use of gentle wound irrigation (Briggs et al 2007) or gentle stroking with cotton wool over the wound site (Bennett 2001).

The extent to which neuropathic pain is a feature of leg ulceration was explored by Briggs et al (2007) in a six-month cohort study of 96 patients with venous and non-venous leg ulcers. Pain symptoms were recorded using the Leeds assessment of neuropathic symptoms and signs (LANSS) pain scale (Bennett 2001), which has been used in a variety of clinical settings to discriminate between nociceptive and neuropathic pain symptoms. Briggs et al (2007) found that pain intensity ratings were statistically significantly higher in patients experiencing neuropathic pain compared with those experiencing nociceptive pain (P<0.001), and suggested that people with a leg ulcer who have neuropathic pain have more intense pain than those with an ulcer that is predominantly nociceptive.
**clinical pain assessment**

**Time out 5**

What sort of questions might you use to explore the pain experienced by some of the patients you encounter?

**Approaches to pain assessment**

Pain assessment is by no means straightforward. The choice of language, non-verbal communication and the style of conversation when discussing pain all need to take into consideration the cultural, economic, social, cognitive, demographic and environmental factors that might affect a patient’s willingness and ability to report and explain painful experiences (Closs et al 2004). Formal assessment and documentation of wound-related pain is recommended by a number of sources (Hollinworth 2005, Barrett 2007, WUWHS 2007). Formal pain assessment usually refers to the use of validated assessment tools and involves the use of pain scales designed to elicit information about pain intensity. Figure 2 (page 10) depicts examples of the four most commonly used scales: the verbal rating, numerical rating, visual analogue and pictorial rating scales.

Deciding which pain scale to use is best done in accordance with patient preference (Barrett 2007). Closs et al (2004) found that most pain assessment tools have been designed and tested on relatively young adults and, while such tools are often considered more effective than the use of general questioning to elicit information about pain, a broader, more wide-ranging assessment of pain may be necessary among older adults and, in particular, older adults with cognitive impairment. The view that older people may prefer a 0-10 numerical scale to other pain scales has been reported (Gibson et al 2004, WUWHS 2007). However, Closs et al (2004) suggested that the verbal rating scale (no pain, mild, moderate, severe) was found to be slightly easier to use than other scales among nursing home residents with some degree of cognitive impairment.

The WUWHS (2004) considered self-reported pain to be the gold standard in pain assessment and recommended that most patients should be deemed able to use a pain rating scale unless otherwise indicated. Pain assessment in patients who cannot communicate is difficult because proxy indicators such as vocalised signals and bodily movements become significant in terms of establishing the extent of the pain experience (Woo et al 2008). Pautex et al (2007) reported some success in the use of a pain assessment tool for patients with dementia. This tool is called the Doloplus-2 scale and features the use of observations of behaviour as a proxy measure for reported pain.

Wound-related pain may vary over time, necessitating frequent reassessment (WUWHS 2007). Pain related to wound dressing changes and procedures is often reported as the most distressing aspect of having a wound (Price et al 2008), and assessments of pain intensity should be taken before, during and after dressing changes or similar procedures (WUWHS 2004, 2007, Barrett 2007).

Some of the literature on pain management cites that a persistent pain score of four or more out of ten on a numerical rating scale (or equivalent) indicates uncontrolled pain necessitating the review of pain management strategies (WUWHS 2004, Mularski et al 2006). The most recent guidance from the WUWHS (2007) makes no reference to pain score thresholds and instead states that a change in the pain level may indicate a need to reassess the wound and consider new complications, the wound care procedure, wound dressing, analgesic choice or other pain management interventions.

**Time out 6**

Consider the concept that a pain intensity score of four or less suggests existing pain management strategies are likely to be acceptable or sufficient. How helpful do you find this idea? Do you agree with it?

**BOX 1**

**Rationale for assessing wound-related pain**

- The management of wound-related pain is often a high priority for patients.
- Pain management is more likely to be successful if performed in conjunction with regular and comprehensive pain assessment.
- Pain assessment information provides a baseline against which future pain assessment data may be compared.
- Pain assessment can help to identify factors that either help the patient to cope with pain, or factors that make the pain worse. This can inform the planning of painful activities such as wound dressing.
- The symptom of pain has important diagnostic potential.

**Barriers to assessing and managing wound-related pain**

Research has identified failure by nurses to assess wound-related pain regularly and comprehensively (Hollinworth 2005, Briggs 2006, Barrett 2007). Information in this area is limited, but it has been suggested that nurses’ psychological
responses to patients’ pain may go some way to explaining why talking to patients about their pain is difficult (Nagy 1999, Wilson 2008). Nagy (1999) identified that almost all of a sample of 32 nurses who worked in a specialist burns unit used ‘distancing’ as a way of coping with the reality of being involved in inflicting pain on patients during dressing changes. Distancing was described not as an attempt to deny the existence of pain, but to lessen its effect on the nurse by placing an emotional and/or physical distance between the nurse and the patient’s pain (Nagy 1999).

Nurses’ psychological responses to patients’ pain were also explored in Wilson’s (2008) vignette-based part replication study of McCaffery and Ferrel’s (1992) exploration of the relationship between patients’ characteristics and nurses’ assessment and subsequent management of post-operative pain. Wilson’s (2008) findings lent direct support to the proposal that patient lifestyle and socioeconomic status can lead to the attribution of bias with respect to nurses’ pain assessment and instigation of pain management strategies. In addition, Wilson (2008) suggested that one explanation for a nurse giving sub-optimal doses of opiate pain relief to post-operative patients was that his or her previous experiences of pain management problems, for example inadequate drug prescriptions for pain relief or delay in reviewing prescriptions, may have resulted in the use of coping strategies in the form of denial or rationalisation. Wilson (2008) provides an example of this by suggesting that nurses may draw on myths about respiratory depression, addiction and physical dependence to rationalise the use of sub-optimal pain relief.

Pharmacological approaches

Analgesic medication is the first-line treatment for wound-related pain, and a number of options, with respect to type or class of analgesia and route of administration, exist. The option of long-acting and slow-release formulations should be considered for background pain and fast acting top-up analgesia should be considered for managing procedure-related pain (WUWHS 2004). Multimodal approaches to pain management may increase the effectiveness of pain management while removing the need for high doses of single analgesics (Coulling 2007).

The effectiveness of analgesia in managing wound-related pain varies between individuals and wound types. In an international survey of the pain experience of more than 2,000 patients with chronic wounds, 58% of patients reported the use of analgesia, with 82% of individuals suggesting that this type of pain relief was effective (Price et al 2008).

The World Health Organization (WHO 2009) has developed a three-step ladder for managing cancer pain (Figure 3), which is also recognised as a valuable approach to managing wound-related pain (EWMA 2002). The WHO (2009) pain relief ladder recommends a stepped approach to the selection of analgesia, with the additional consideration of co-analgesics or adjuvant medication to provide a comprehensive drug-based treatment where more complex pain symptoms exist. Examples of co-analgesics that may be used in the treatment of wound-related pain include the tricyclic antidepressants and anticonvulsants, which can be added to an analgesic regimen where evidence of neuropathic pain exists (WUWHS 2004).
As shown in Figure 3, the basic progression of analgesic medication is from non-opioids (step 1), to weak opioids, for example codeine (step 2), and finally to strong opioids such as morphine (step 3). Steps 2 and 3 of the ladder indicate that combinations of non-opioids and opioids should be considered. Although the model seems to imply that patients should be commenced at step 1 of the pain relief ladder, this is not always the case and individuals who experience high levels of pain will need to commence an analgesic regimen based on either step 2 or 3 of the ladder (WUWHS 2004).

Non-steroidal anti-inflammatory drugs (NSAIDs) dampen sensitivity and are particularly useful for controlling the throbbing or aching pain felt after a procedure such as a wound dressing (WUWHS 2004). Enoch et al (2006) suggested that NSAIDs may affect the inflammatory phase of wound healing adversely and may also be associated with a reduction in the tensile strength of the wound. These claims are unsupported by clinical data, and consensus guidelines on managing wound-related pain support their use while highlighting certain contraindications and cautions (EWMA 2002, WUWHS 2004). In addition, there is some evidence to suggest that the healing rate of chronic wounds (venous leg ulcers), treated with foam dressings containing a low dose of ibuprofen (112.5mg impregnated in a 15x15cm dressing released over a seven-day period), was similar to chronic wounds treated with the same dressing with no ibuprofen content (Gottrup et al 2008).

The research was in the form of a randomised, controlled, double-blind clinical investigation on the performance and safety of an ibuprofen-containing dressing (Biatin Ibu) (Gottrup et al 2008). The 122 patients recruited for the study had leg ulceration, were being treated with compression therapy and, before the study, were experiencing either moderate, lots or complete pain (categories on a five-point verbal rating scale). The authors reported that both the ibuprofen foam and the comparator foam dressing were associated with clinically significant pain reductions, with the ibuprofen foam having a slight advantage in this respect (Gottrup et al 2008).

The recommended time interval between the administration of the analgesia and the start of the wound dressing or procedure varies slightly. The WUWHS (2004) stated that a period of one to two hours before the procedure is appropriate when paracetamol and NSAIDs are being used. However, EWMA (2002) suggested that short-acting opioids, such as codeine, should be given up to one hour to take effect before commencing a wound-related procedure. Topical local anaesthetics can provide a degree of numbness for a short period of time while a particularly painful procedure is carried out (WUWHS 2004, Woo et al 2008). The most commonly reported use of topical local anaesthetic relates to eutectic mixture of local anaesthetics (EMLA) cream – lidocaine with prilocaine – before wound debridement in lower limb ulceration.

Briggs and Nelson (2003) undertook a systematic review of the literature with regard to the efficacy of this treatment when used before debridement of leg ulcerations. The review was based on six randomised controlled trials involving 137 patients. The authors concluded that while there was evidence that a local anaesthetic cream (EMLA) reduced the pain of debridement, there was insufficient evidence of the effect of this cream on side effects and healing. It is important to note that the British National Formulary (2008) states that EMLA should not be used on wounds.

**Non-pharmacological approaches**

Although analgesic treatments are considered important in terms of achieving pain relief during procedures such as dressing changes, they may not always be effective (Gibson et al 2004, Price et al 2008). Non-pharmacological approaches are targeted at the reduction of anxiety and stress and the improvement of personal coping skills (Woo et al 2008). Some publications on wound-related pain (EWMA 2002, Wounds-UK and Mölnlycke Health Care 2004, WUWHS 2004, 2007, Coloplast 2008) identify similar non-pharmacological pain relieving strategies for managing procedure-related pain. The most
frequently cited include distraction, relaxation techniques, music therapy, patient involvement, giving information and making use of patients calling for ‘time out’ to signal interruption to the procedure and time for rest.

Distraction as a mode of pain management can be explained in the context of the Gate Control Theory (Melzack and Wall 1965). The technique takes the patient’s attention away from the pain experience as he or she attends to different sensory information. Research on the use of distraction with respect to wound dressing-related pain has been undertaken by Hoffman et al (2004), who reported the use of video games for adolescent patients undergoing the removal of staples from skin grafts.

Research on the effect of more psychologically based interventions to manage wound pain has also been carried out by Gibson et al (2004), who undertook a pilot study of an educational intervention to manage the pain associated with wound care in an outpatient setting. The pilot study focused on the use of a structured educational intervention to reduce procedure-related pain, and five patients who had a history of dressing-related pain were invited to take part. Patients were provided with information about their procedure and were encouraged to explore ways in which their comfort levels might be improved during the procedure. Following on from this, a plan of treatment was compiled that identified pharmacological and non-pharmacological interventions. Pain and distress intensity ratings were recorded during the procedure. Four out of the five patients said that the education had been helpful, but the intervention resulted in less pain and distress (compared with not having an educational intervention) for only three of the five patients (Gibson et al 2004).

This small study identified some of the difficulties in researching interventions to reduce wound-related pain. In particular, it was noted that the status of the wound, for example whether improving or deteriorating, was likely to be the greatest determinant of wound pain and that this may override any psychologically-based pain management intervention (Gibson et al 2004).

Evidence for the extent to which patients perceive the effectiveness of non-pharmacological interventions in reducing pain during dressing changes is limited. In a survey of more than 2,000 patients with chronic wounds, only a small number of patients identified that practices such as being careful and gentle, not rushing the dressing, and listening and communicating with patients could potentially help to minimise dressing-related pain (Price et al 2008). However, given that such practices are relatively simple, their continued use by practitioners seems justified on both professional and moral grounds.

**Clinical pain assessment**

Think about some of the patients you encounter in your clinical practice. What sort of techniques and strategies do you use to distract, relax or involve a patient while undertaking dressing changes? Are there any strategies that you could use in the future that you had not previously considered?

**Dressing selection and wound care practices**

Published guidance on the management of wound-related pain identifies the extent to which wound dressings and treatments have the potential to cause wound bed and peri-wound tissue trauma and pain (EWMA 2002, WUWHS 2004). Price et al (2008) reported that 40% of patients perceived that pain during dressing changes was the worst part of living with a wound. Wound care practices that have been found to trigger painful sensations include (Hollinworth 2005, 2006):

- Exposure of the wound to air on dressing removal.
- The use of cool irrigation fluid.
- Dressing removal following adherence to the wound.
- Inappropriate dressing choices.
- Tightly packed cavity wounds.
- Wound swabbing and uncomfortable primary dressings causing stinging or drawing sensations.
- Retention or compression bandaging.

Dressings that adhere to the wound, causing pain on removal and trauma to the wound bed, should not be used (WUWHS 2004, Hollinworth 2006), as they can cause damage to delicate healing tissue in the wound and surrounding skin. Developments in wound dressings have resulted in the proliferation of low and non-adherent dressing materials designed to minimise wound trauma and pain. The performance of low and non-adherent dressings has been found to vary between products and wound types (Briggs et al 2008). In particular, soft silicone adhesive
clinical pain assessment

Any technique that features physical contact with the wound has the potential to cause pain. Activities such as wound cleansing, dressing removal and placement, and debridement are likely to be problematic in terms of wound care. Wound cleansing is not always necessary, but if it is then cleansing solutions should be warmed to body temperature as patients may find cool irrigation solutions painful (Hollinworth 2006).

The peri-wound area can become painful secondary to maceration, excoriation, contact sensitivities arising from dressing materials and epidermal stripping caused by the removal of adhesive dressings (Hollinworth 2003, 2006).

technology dressings have been found to be less painful before, during and after dressing change when compared with advanced dressings with traditional adhesives (WUWHS 2007). However, such dressings have been noted to be costly when compared with other products, and minor dissatisfaction has been noted with respect to the ability of the dressings to stay in place without the use of additional adhesive tape (Briggs et al 2008).

References


Maceration of the skin surrounding the wound that has a dressing applied suggests that the dressing has inadequate fluid handling properties and signals the need for dressing review. Skin barrier products offer some topical protection from excessive moisture and dressing adhesives, but care should be taken to follow the manufacturer’s instructions, and regular skin observations should determine the extent to which the skin protector is effective (Hollinworth and Stansfield 2008). The importance of peri-wound skin protection in controlling wound-related pain is highlighted in the most recent WUWHS (2007) guidance. It states that ‘practitioners should select an appropriate dressing to minimise wound-related pain based on wear time, moisture, balance, healing potential and peri-wound maceration’ (WUWHS 2007).

Conclusion

Pain is a distressing feature of having a wound. Practitioners caring for patients with wound-related pain need a sound knowledge base, good communication skills, a willingness to collaborate with colleagues and an understanding of the patient’s concerns to achieve the best outcomes in terms of pain minimisation and pain relief NS.
Biofilms: possible strategies for suppression in chronic wounds


Summary
Biofilms can delay wound healing significantly. The aim of this article is to highlight strategies that could be used to treat chronic wounds containing biofilms. Antibiofilm agents, their modes of action and efficacy in suppressing biofilms are discussed. The article was originally published in Nursing Standard in 2009, volume 23, number 32, pages 64-72.

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Keywords
Biofilms, chronic wound infections, microbial bioburden, wound healing

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The biofilm concept
As 99% of micro-organisms found in their native habitats persist in a biofilm state, this mode of microbial growth is applied to the wound environment (Percival and Rogers 2005, Ngo et al 2007, James et al 2008). Planktonic bacterium, coaggregates (clusters) of bacteria, or fragments of biofilm (clusters of bacteria encased within an EPS) often attach to a surface, multiply and grow (Percival et al 2007, Thomas 2008). Medical biofilms Up to 80% of human infections are thought to be related to pathogenic biofilms (Anon 1997). However, clinicians have begun to appreciate that many persistent infections are caused by biofilms. Many chronic infections influenced or induced by a biofilm, for example prostatitis, endocarditis and osteomyelitis, have been found to persist indefinitely (Percival and Bowler 2004b). It seems plausible, therefore, to hypothesise that biofilms have a fundamental role in chronic wound infections. Implications of biofilms As bacteria in the biofilm multiply they continually produce 'pheromones', called quorum-sensing molecules (Box 1). These are significant in the formation of a biofilm and in the development of the bacterial community. Changes in a chronic wound biofilm, induced by quorum-sensing molecules, enhance the recalcitrance of the biofilm to antimicrobials (Sauer et al 2002). With an array of physical and metabolic defences, the biofilm has an enhanced resistance to antimicrobials including antibiotics,
clinical wound healing

Many mechanisms inherent to the biofilm, including physical and chemical heterogeneity, interspecies co-operation, and intercellular structure, contribute to this enhanced recalcitrance to antimicrobials (Xu et al 2000, Fux et al 2005, Burmølle et al 2006, Shen et al 2006, Chang et al 2007).

Most microbial metabolism and bacterial divisions occur in cells located near the biofilm surface. These active bacterial cells are thought to reproduce constantly and disperse from the biofilm at a high rate. These metabolically active cells have been shown to be the most vulnerable sub-population in the biofilm to antimicrobials, particularly antibiotics and host defences (Costerton et al 2003).

Contrary to this, micro-organisms, specifically bacteria found deep in the extracellular matrix of the biofilm, have been found to be protected from external perturbations, are less metabolically active and more recalcitrant to antimicrobial management practices (Xu et al 2000, Lewis 2007). In addition, microbial cells found deep within the biofilm have the ability to reconstitute the community of the biofilm extremely quickly, particularly during periods of extreme stress (Lewis 2007).

Based on the above findings, the management of a biofilm community is significantly more challenging than that traditionally used in planktonic-based wound care.

**BOX 1**

**Glossary**

- Colonisation – the presence of bacteria in increasing numbers in the wound without inducing a host reaction. It is not possible to differentiate clinically between contamination and colonisation.
- Contamination – the presence of bacteria in a wound without inducing a host reaction.
- Matrix metalloproteases – proteases that may be endogenous or exogenous in origin and break down protein, for example collagen.
- Planktonic – free-floating micro-organisms not attached to a surface.
- Proinflammatory cytokines – protein molecules derived from the immune system that amplify the inflammatory response.
- Quorum sensing – measuring the bacterial population concentration through the expression of bacterial signalling molecules. When a ‘quorum’ is reached, specific biological activities, for example expression of virulence factors are activated.
- Sessile – micro-organisms attached to a surface.

**Theory and practice of wound biofilms**

When skin is broken a relatively ‘immature’ wound is formed and the primary bacterial defence barrier is initially compromised (Niyonsaba et al 2006). The host’s primary objective is to prevent the bacteria that have contaminated the wound from increasing in number and inducing infection. Typically, the host easily fend(s) off potential pathogens through inflammation (proinflammatory cytokines, matrix metalloproteases, phagocytosis and degranulation of neutrophils). However, a number of host factors promote the establishment of a chronic wound and infection, including poor perfusion, malnutrition, foreign body presence, pressure, repetitive trauma, hyperglycaemia and white blood cell dysfunction.

If a biofilm becomes established in a wound, it will be difficult to suppress, particularly in an immunocompromised individual (Costerton et al 2003). Consequently, the micro-organisms and their extracellular components within the biofilm will prolong the state of acute inflammation indefinitely, delaying the normal healing process. Clinicians often focus on the number of culturable bacteria that are present in the wound as this frequently correlates with the degree of immune stimulation (classic signs of acute infection) seen in the patient (Dow 2001). However, many clinical biofilm bacteria cannot be cultured and so these bacteria can also be overlooked using traditional microbiological techniques (Costerton et al 2003).

Sharp debridement of devitalised tissue promotes healing by removing tissue that not only supports microbial proliferation (the biofilm) but also reduces the efficacy of topical therapies. Consequently, debridement should be performed at weekly intervals in chronic wounds containing devitalised tissue and biofilm (Wolcott et al 2009), although this should only be carried out by those who are qualified to carry out this skill. Debridement is thought not only to remove micro-organisms but also to expose deeper host defences, therefore enhancing their efficacy. However, debridement alone in the authors’ opinion is not sufficient to manage the majority of chronic wounds because of the nature of biofilm removal and re-establishment. Other concomitant strategies, such as antimicrobials, should be considered as an adjunct therapy (Schultz et al 2004).

Some studies have shown that antimicrobial agents sometimes work effectively to suppress the metabolically active cells in a biofilm (Flemming et al 2009). However, it is important to acknowledge that no single strategy has yet proven to be consistently effective at suppressing the entire biofilm. For example, infection often
reoccurs following the administration of a course of antibiotics. This is because antibiotics are only effective in transiently suppressing rapidly growing cells, which are generally located on the outermost surface of the biofilm. Recalcitrant microbial cells found deep in the biofilm will persist.

Shortcomings exist in the use of single or sequential treatment strategies. In the authors’ experience, concurrent management strategies will increase the likelihood of prolonging suppression of the biofilm. Such extensive suppression of the biofilm is essential to foster sufficient healing (Costerton et al 2003). There are no visible clinical signs to indicate the presence of a biofilm, however if other causes of delayed wound healing such as smoking and poor nutrition have been ruled out, then a chronic infection (biofilm) is usually suspected.

Management strategies

Many commercially available wound dressings are not inherently antimicrobial. However, some wound dressings have been shown to help reduce the bacterial load at the wound surface through sequestration (binding) of bacteria (Mertz and Eaglstein 1984, White et al 2006). Consequently, wound dressings should be selected carefully to help reduce the risk of biofilm growth and therefore further proliferation on and in the wound.

The modes of action of various antimicrobial agents differ. However, their effects are similar, in that they impair the metabolism or integrity of micro-organisms by stopping or substantially reducing cell division (microbiostatic) or by killing micro-organisms directly (microbicidal). The most recently designed antibiofilm agents often work without impairing microbial growth, reproduction or cell integrity but by breaking up the biofilm, removing essential nutrients or metal ions, or interfering with microbial community interactions such as quorum sensing (Singh et al 2002, Costerton et al 2003, Kaneko et al 2007). These agents are not yet available as they are experimental developments. Once approved by the regulatory authorities they will provide a more effective approach to wound biofilm management. However, they will not be a ‘one dose’ or ‘one application’ only approach but will need to be repeated or sustained in line with current antibiotic and antiseptic use.

Systemic antibiotic treatment is used in chronic wounds in situations where there is significant deep tissue wound infection or a risk of septicaemia. However, systemic antibiotics have been shown to have a low efficacy rate when biofilms are present (Moss et al 1990, Marr et al 1997). The use of antibiotics is problematic in ischaemic wounds. This is because the therapeutic concentrations that are routinely used may not reach the site of infection at the therapeutic dose. However, systemic antibiotics have been shown to contribute to the clinical management regimen of many wound biofilms by suppressing the cells at the outermost region of the biofilm (Xu et al 2000).

Topical antiseptics can also help to reduce the wound bioburden and biofilm, particularly where there is a concurrent infection or an increased risk of infection.

In the authors’ opinion, the efficacy of antimicrobial agents can be substantially enhanced when combined with other management strategies, specifically debridement in conjunction with an appropriate wound dressing. Antimicrobial agents It is plausible to suggest that antimicrobial agents could act prophylactically to suppress biofilm development. However, some non-selective antimicrobials may be detrimental to wound healing by harming the host’s cells, for example alcohols, hydrogen peroxide, carbolic acid, sodium hypochlorite and acetic acid. Selective antimicrobials such as molecular iodine and ionic silver are more suitable for chronic wounds.

Iodine Iodine has been used for many years as

<table>
<thead>
<tr>
<th>Agent</th>
<th>Mode of action</th>
<th>Usage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactoferrin</td>
<td>Blocks the attachment of planktonic bacteria to a surface. Blocking the initial attachment of bacteria stops the first step in biofilm formation (Singh et al 2002)</td>
<td>Bovine lactoferrin is a protein used in the meat-packing industry to protect exposed meat from bacterial biofilm formation</td>
</tr>
<tr>
<td>Xylitol</td>
<td>Interferes with biofilm formation (Katsuyama et al 2005)</td>
<td>Xylitol is a five-carbon alcohol sugar used in chewing gum. It has been shown to reduce the incidence of dental caries (Burt 2006)</td>
</tr>
<tr>
<td>Gallium</td>
<td>Interferes with bacterial iron metabolism pathways (Kaneko et al 2007)</td>
<td>Gallium nitrate is a Food and Drug Administration (FDA) approved drug that can be used intravenously (DHSS, UK)</td>
</tr>
<tr>
<td>Dispersin B</td>
<td>Targets the extracellular polymeric substance of some types of biofilm and works to degrade the community structure of the biofilm (Iloh et al 2005)</td>
<td>Dispersin B is a bacterial enzyme but is not presently used commercially</td>
</tr>
</tbody>
</table>
clinical wound healing

A wound antiseptic (Cooper 2007). However, high doses of iodine can be detrimental to host healing (Kramer 1999, Wilson et al 2005). Cadexomer iodine can be used to suppress biofilms without causing significant host cell damage (Akiyama et al 2004).

Ionic silver Ionic silver is a beneficial antimicrobial for use in wound care, particularly for biofilm-based management strategies. Ionic silver has a broad range of efficacy against many micro-organisms (Russell and Hugo 1994, Lansdown et al 1997). In addition, a number of silver dressings have been shown to prevent biofilm formation in vitro (Percival et al 2007) and also have in vivo benefits (Fong and Wood 2006).

Honey In vitro evidence shows that honey is effective against a range of multi-resistant organisms including meticillin-resistant Staphylococcus aureus, vancomycin-resistant enterococci and multi-resistant Gram-negative organisms including Pseudomonas aeruginosa (George and Cutting 2007), and can surpass the use of antibiotics or antiseptics in previously unresponsive wounds (Dunford et al 2000). Irish et al (2006) demonstrated the effectiveness of honey in preventing biofilm formation. Okhiri et al (2004) found disruption of pseudomonal biofilms (in vitro) following application of honey. Honey also has potential as an antibiofilm agent (Table 1).

Antibiofilm agents The use of antibiotic agents are considered by many to be beneficial to wound management as they have been shown to be less cytotoxic than many traditional antimicrobials. These include lactoferrin (Singh et al 2002), xylitol (Katsuyama et al 2005), gallium (Kaneko et al 2007) and Dispersin B (Lu and Collins 2007). Evidence regarding the benefits of such agents to wound healing is

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slowly beginning to gain momentum. Agents that have been used as antibiofilm agents in wound care can be found in Table 1. These agents are still being evaluated and are not yet available in clinical practice.

Conclusion

The aim for the wound care provider is to establish the best management strategies possible for each individual patient. To achieve this, clinicians need to be familiar with antimicrobial and antibiofilm agents. The basis of managing biofilms is frequent removal of the biofilm from the surface of the wound. This can be achieved with either sharp debridement (curette), or formal surgical debridement. As biofilms reconstitute quickly following invasive debridement, suppressing their regrowth and reconstitution using multiple antimicrobial management strategies is warranted, including wound cleansers, topical antimicrobials and advanced wound dressings. Such strategies may work synergistically to help suppress and reduce the regrowth of the biofilm. A ‘rotating’ regimen of selective antimicrobials may be advantageous in biofilm-based wound management. Systemic antibiotics may also help to suppress further the biofilm.

For positive clinical outcomes it is important that all concurrent barriers to healing should be addressed. This will help to augment the host’s defences which, when working optimally, provide the best strategies to help manage a wound. Consequently, biofilm-based management is becoming fundamental to non-healing wounds NS

References continued


Aims and intended learning outcomes

The aim of this article is to introduce and define the concept of delayed wound healing, with a focus on venous leg ulceration, and to describe the causes of delayed healing and interventions to overcome it.

After reading this article, you should be able to:

- Understand what healing rates might be expected with ‘standard treatment’.
- Relate delayed or prolonged healing to treatment, medical, biological, psychological and lifestyle causes.
- Be aware of guidelines available to aid prediction of wound chronicity.
- Appreciate the evidence in support of interventions to overcome delayed healing.

Introduction

Many wounds that heal by secondary intent, such as leg ulcers, diabetic foot ulcers and pressure ulcers, have historically been referred to as ‘chronic’, but a wound is not always inherently ‘chronic’ simply because of its aetiology (Harding 2000).

The label ‘chronic’ is applied to wounds in which compromised healing is anticipated, usually because of complex underlying pathologies such as diabetes (King 2001), vascular disease (Grey et al 2006) and oedema (Williams 2009), malignancy (Izadi and Ganchi 2005), malnutrition (Graue et al 2008) or morbid obesity (Fife et al 2008).

The published literature contains many references to such wounds, referring to them as ‘stunned’ (Ennis and Meneses 2000), recalcitrant (Thomson 2000) or ‘difficult to manage’ (Ballard and Baxter 2000). Consideration must be given to the diagnosis, and to what degree or rate of healing is anticipated, if any, before any judgement of delayed healing can be made (Harding 2000, Cardinal et al 2008).

We are now urged always to refer to such wounds as ‘hard to heal’ (Gottrup et al 2010).

The management of chronic wounds in the UK has been estimated to cost over £1 billion a year (Bennett et al 2004), with an additional cost to patients of reduced quality of life (Charles 2004). What then constitutes a ‘chronic’ wound and what determines ‘delayed’ healing?

Summary

For a variety of reasons, some wounds take longer than anticipated to heal, or do not heal at all. Delayed or impaired healing may occur with wounds such as leg ulcers, but can also sometimes be seen with acute traumatic wounds such as pre-tibial lacerations. This article, using leg ulcers as an example of ‘chronic’ wounds, provides a guide to delayed healing and how it can be anticipated, avoided and managed. The article was originally published in Primary Health Care in 2008, volume 18, number 2, pages 40-46.

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Keywords

Chronic wound, ulcer, delayed healing

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Time out 1

What are the differences between acute and chronic wounds in terms of time to heal? How might wound chronicity be predicted?
Delayed healing

The term ‘delayed healing’ has been defined as ‘healing that takes longer than anticipated, given appropriate therapy’ (Ballard and Baxter 2000). Wounds can heal within an ‘acceptable’ and predictable time given appropriate treatment. Thus a venous leg ulcer, treated with a moist wound dressing and graduated compression, that does not show signs of a healing response in four weeks, may be described as ‘delayed’ or compromised (Phillips et al 2000). This definition demands that ‘appropriate’ therapy is provided, and this will vary according to the aetiology and condition of the wound.

Predictive leg ulcer healing rates have been published (Margolis et al 2004, Flanagan 2003) and validated (Cardinal et al 2008). These provide a benchmark whereby ‘normal’ healing rates, as measured by reduction in wound area over time, can be compared with those achieved in individual patients. This, by definition, requires regular assessment including wound measurement (Langemo et al 2008). Diabetic foot ulcers require the control of blood glucose, pressure offloading and debridement if they are to heal (Cavanagh et al 2005); predictive healing rates have been published (Sheehan et al 2003). Similarly, pressure ulcers need pressure relief as well as attention to general medical condition if they are to heal (Thomas 2006).

It is not only chronic wounds that can be classed as delayed; acute traumatic wounds can also fall into this category. A good example is the pre-tibial laceration (Warren et al 1991); these wounds often become static and fail to respond to treatment (Dunkin et al 2003). In such cases, the patient’s underlying medical condition can be one of the main reasons (Wood and Lees 1994).

Anticipating a delay in healing

To predict, or anticipate, the chronicity of a wound at an early stage is clearly of great clinical benefit as it allows timely intervention with cost-effective treatments. To that end, attempts have been made to list various indicators (Boyd et al 2004, Gohel et al 2005). These authors have established an expert working group to consider factors that might adversely affect wound healing. Their findings can be summarised as follows:

- Local factors – for example, foreign bodies, scar tissue, peri-ulcer maceration, incontinence, undermining.
- Regional factors – venous or arterial disease, perfusion, neuropathy, lymphoedema, oedema.

These factors may be ranked as ‘severe chronicity’, ‘high to moderate chronicity’, ‘mild chronicity’, and, ‘unlikely to become chronic’.

Systematic factors – diabetes, malnutrition, chemotherapy, pain, rheumatoid disease, psychological issues, smoking.

Avoiding delayed healing

In effect, avoiding delayed healing constitutes ‘optimising’ healing. The positive steps begin with detailed and regular holistic assessment. Treatment should be evidence-based and meet with current gold standards, such as an optimum moist wound environment and appropriate compression for venous ulcers. In the case of compression, correct application is essential as incorrectly applied bandaging or hosiery can prove ineffective and lead to tissue damage (Beldon 2008).

Where local delaying factors exist, steps must be taken to overcome them by, for example, debriding slough and necrotic tissue, removing foreign bodies and avoiding maceration and consequent wound enlargement (Cutting and White 2002a, 2002b).

Regional factors may be more difficult to overcome. Venous disease and lymphoedema can often be counteracted by compression (Stephen-Haynes 2006), while arterial disease may require bypass grafting. Neuropathy, a factor in diabetic foot ulceration, cannot be treated and requires long-term protective measures for patients ‘at risk’ of ulceration (Cavanagh et al 2005).

Systemic factors, once recognised, can be addressed with appropriate treatment. For example, diabetes can be managed by blood glucose control and pain by analgesia, although not all such factors are manageable (for example, malignancy, smoking and alcohol misuse).

Biological factors

Abnormalities in the cellular and biochemical environment, although not directly observable, can delay or prevent wound healing (Douglass 2003). Clues to biological abnormalities can be gained by observing the tissues in the wound-bed and surrounding skin (Harker and Moore 2004). These aspects can be monitored, assessed and
Microbial factors in delayed wound healing

> Wound infection or critical colonisation (Kingsley 2001).
> Bacteria consume the vital nutrients needed for tissue regrowth.
> Increased exudate volume (Cutting 2004) and purulent exudate.
> Toxins from bacteria damage cells (Cooper 2003).
> Proteases damage the extracellular matrix and other vital tissue components.
> Odour: malodour is attributed to anaerobic bacteria.
> Biofilm formation inhibits healing (James et al 2007).
> Slough/necrosis provides an ideal environment for bacterial growth.

The extracellular matrix (ECM) is essentially the dermal component of the skin. It comprises fibrous proteins such as collagens and elastin, and protein-carbohydrate complexes known as glycosaminoglycans (GAGs). The most important GAG is hyaluronan, previously known as hyaluronic acid. The ECM provides the skin with its strength and resilience, as well as forming a foundation for the regrowth of epidermis and a scaffold matrix for the growth of blood vessels and nerves. In wounds that heal by secondary intent, the replacement of ECM is central to tissue repair. In chronic wounds, many factors conspire to prevent these processes occurring in an orderly fashion, thus impairing the healing process (Baum and Arpey 2005).

**Perfusion**

The blood supply to the wound bed and surrounding tissues brings vital oxygen, nutrients, white cell subpopulations, drugs (such as antibiotics) and hormones. Where perfusion is poor, tissue ischaemia and an increased risk of infection are barriers to healing (Hunt et al 2000). Low tissue oxygen levels compromise healing because of, for example, microvascular disease in diabetic foot ulceration, arterial ulceration accompanied by atherosclerosis, venous hypertension and lipodermatosclerosis in venous ulceration, and, in pressure ulcers, vascular occlusion and ischaemia.

Pulse oximetry has been successfully applied to patients with leg ulcers; this is a measure of arterial oxygen and, consequently, tissue perfusion (Bianchi 2008). Impaired perfusion can be alleviated in some patients by exercise, limb position and/or compression therapy (this improves venous return, reduces oedema and thereby raises arterial perfusion). If these fail, or are inappropriate, surgery for arterial grafting or endarterectomy is indicated.

**Overcoming delayed healing**

Having established the chronicity of a wound, either prospectively using the indicators listed above or retrospectively from the patient’s history, the next question is how to counteract these factors. The clinician can differentiate the severe, moderate and mild factors, and appreciate...
why each is ranked as such. By using the venous ulcer as a paradigm, practitioners can illustrate how each factor may be addressed with current therapeutic interventions.

**Leg ulcers that fail to heal**

Leg ulcers are a major health problem in the UK, with a prevalence of between 1.1 and 3.0 per 1,000 (Callam *et al* 1985). Although most are associated with venous disease (RCN 1998, SIGN 1998), nurses need to recognise rarer lesions of the lower leg (Box 2). These often need different treatments from leg ulcers and referral to a specialist. Guidelines suggest that patients should be referred for biopsy if the appearance of the ulcer is atypical, or if there is deterioration or failure to progress after 12 weeks of active treatment (SIGN 1998).

**Box 2**

**Ulceration of the lower limb: common differential diagnoses**

- Many differential diagnoses for ulceration of the lower limb exist (Tillman 2004). All can delay or impair healing and need to be excluded at the outset and throughout the treatment period:
  - Venous leg ulcer
  - Arterial ulcer
  - Complex or mixed venous/arterial aetiology ulcer
  - Pyoderma gangrenosum (Bull 1997)
  - Necrobiosis lipoidica
  - Rheumatoid ulcer (Seitz *et al* 2010)
  - Malignant ulcer (Marjolin’s ulcer) (Enoch *et al* 2004)
  - Bullous pemphigoid

**Figure 1**

**Percentage of venous leg ulcers that heal over time with compression therapy: meta-analysis data**

![Graph showing percentage of venous leg ulcers healed over time with compression therapy](image)

(Rippon *et al* 2006)

Published data on healing rates vary. Typically, the figure given is the percentage healed in 12 weeks. While this may at first sight seem impressive, it is the percentage not healed that will present the clinical challenge to the practitioner. This group will also be the most expensive to manage, as treatment can often be protracted for many months or years. Figure 1 shows the percentage of venous ulcers healed, taken from data from 27 published clinical trials of compression therapy. This graph shows that more than 40% of ulcers are unhealed at 20 weeks and more than 20% still unhealed at 70 weeks (Rippon *et al* 2006).

**Therapy**

Exposed bone and tendon may be treated by either hydration with a hydrogel (Scardillo and Seeley 1996), occlusive dressings (Omidi and Nahass 1996) or cultured epidermal grafts (Bolivar-Flores and Kuri-Harcuch 1999), as re-epithelialisation will not occur where there is dry tissue. Lipodermatosclerosis and related atrophie blanche present major hurdles to healing, and treatment is either invasive, by excision and grafting (Schmeller and Gaber 2000), or conservative by compression (Kirsner *et al* 1993).

Malignancy, as basal cell or squamous cell carcinoma (Marjolin’s ulcer), occurs in more than 2% of leg ulcers (Yang *et al* 1996). The older a leg ulcer, the greater the likelihood of malignancy; where ulcers are of greater than 12 weeks’ duration (Figure 2), or malignancy is suspected on other criteria, urgent referral is essential (Enoch *et al* 2004).

The condition of the skin surrounding the ulcer can have an impact on healing. Where skin becomes exposed to chronic wound fluid, maceration and the consequent deterioration/enlargement of the wound is a risk (White and Cutting 2003). Optimal moisture control is central to good wound care (Bishop *et al* 2003). The simple precautions of protecting the peri-wound skin, use of emollients for dry skin, topical corticosteroids where indicated, avoidance of contact allergens, modern dressings to manage exudate, control of bioburden where indicated, carefully selected dressing/bandage wear time, suitable compression, leg elevation and management of infection are all important in this respect (Nelson *et al* 2005).

With respect to bioburden control it is important to recognise those wounds which require treatment with antimicrobials and avoid unnecessary treatment in those which do not (White *et al* 2006). Inappropriate use of antimicrobial dressings such as silver- or iodine-containing products, or antibiotics, is wasteful and must be avoided. The current...
best practice guidelines for topical antimicrobial are intended to clarify how they may be used to best effect (Wounds UK 2010).

The underlying pathology, venous disease, responds to compression therapy (Nelson et al 2005). This has justifiably become the mainstay of treatment and should be the first choice in every case.

Wound and patient assessment

The key to managing wounds is to assess systematically and frequently, so that appropriate treatments can be implemented in a timely fashion and any deterioration recognised early. The tools exist to predict delayed healing (Flanagan 2003), and to assess the wound systematically. In the latter case, the two systems in widespread use are AWM and TIME (Gray et al 2006, Schultz et al 2003). Accurate diagnosis is a prerequisite to effective treatment. Leg ulcers should be diagnosed according to the defined criteria, and a Doppler ankle-brachial pressure index is essential (RCN 1998, SIGN 1998). Recently, pulse oximetry has been shown to be valuable in assessing the vascular status of patients with leg ulcers before compression therapy (Bianchi and Douglas 2002, Bianchi 2008). The systemic factors involved in chronicity of venous leg ulcers include:

- Diabetes (venous leg ulcers, as opposed to foot ulcers, in patients with diabetes).
- Pain.
- Psychosocial factors.
- Smoking.
- Drug or alcohol misuse.

Time out 3

Having established the factors contributing to chronicity in a patient with venous leg ulceration, decide which may easily be addressed and which may not.

Each of these factors can be addressed. Pain will delay healing and have an impact on quality of life (Mangwendeza 2002). Published guidelines on pain assessment and management strategies exist (EWMA 2002, White and Harding 2006). Pain is often associated with the dressing change procedures, for example, with trauma on removal of a dressing (Romanelli and Dini 2006). The means to overcome this are now well known and must be adopted (Hofman 2006). Among the psychosocial factors is patient concordance, which can be addressed through the application of several measures, including education, communication, pain management and a social model (Goode 2004, Moffatt 2004a, Briggs 2005, Seymour 2005, Price 2006).

Interventions to address biological factors in venous leg ulceration

The use of the ‘new generation’ of what might be best termed biological treatments is in its infancy. Various compounds and living tissues for topical use or replacement therapy exist, and clinical data are being accumulated, for example, specific growth factors, available in topical formulation, for application to the ulcer bed (Enoch et al 2006). Tissue replacements for dermal and epidermal components are available. These are either autograft or allograft materials (Owen et al 2006). Extracellular matrix components have been developed for topical use on hard to heal wounds, and biological chemicals such as GAGs, hyaluronan (Colletta et al 2003) and integrins have been evaluated in clinical trials (Mirastschijski et al 2004). Control of inflammation is achieved by using agents designed to inhibit MMP activity (Moore 2004). In general, these are more expensive than orthodox dressings, and health economic data are needed to support their use in specific cases of delayed healing.

Clinical setting of care

The precautions and guidance set out here are not restricted to secondary care. Community-based practitioners, although they do not have direct

FIGURE 2

Chronic leg ulcer
Alternative to Doppler in leg ulcer
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