Wound bed preparation and the diabetic foot

Lynne Watret

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1 Patients with diabetes will often present with a compromised healing wound.

2 Wound bed preparation using the TIME acronym can help plan a rational approach to wound management.

3 TIME stands for: Tissue management; Infection/inflammation control; Moisture balance and Edges/epithelial advancement of the wound.

4 Wound bed preparation provides a framework that can be understood by taking a holistic view of the management of the patient with diabetes with a foot lesion.

KEY WORDS
- **Wound bed**
- **Compromised healing wound**
- **TIME**
- **Infection**
- **Management**

Introduction

The philosophy of wound bed preparation is now widely accepted as a valuable concept when implementing a wound management treatment plan. Wound bed preparation involves identifying individual patient issues, treating underlying causes whenever possible, and wound diagnosis. The philosophy also questions the differences between acute and chronic wound healing, leading to a greater understanding of the barriers that may compromise the healing process. Wound bed preparation, therefore, provides us with a rational strategy to plan the care of a patient with a complex wound.

Traditionally the acute wound healing cascade was used as an ideal healing model and the management of this type of wound was transferred to the chronic wound. However, the differences between acute and chronic wounds have been given greater credence when underlying patient co-morbidity problems have been taken into account (*Table 1*) as well as individual patient factors that may compromise wound healing (*Table 2*). Furthermore, on analysis of acute and chronic wound fluid there are profound differences that may promote healing in the acute wound in the 'healthy individual' but may result in imbalance which may result in barriers to healing for compromised patients (Kirstner et al, 2001; Schultz et al 2003; Ayello and Cuddigan, 2004; Keast et al, 2004). The International Working Group on the Diabetic Foot identified that impairment of neutrophils, abnormalities of growth factor production, and an increase in the amount of degrading enzymes present all result in a complex biological healing process in the person with diabetes.

A common feature of the compromised wound is that it appears to be arrested at the stage of inflammation and granulation formation (Moore, 2004). The presence of slough, necrosis and increased bacterial burden further compromise the wound's ability to heal. Barriers to healing are therefore preventing the wound progressing towards epithelialisation in a timely fashion.

The wound bed preparation paradigm has 'led to independence of chronic wounds from models of acute injury' (Falanga, 2003). Surgical debridement of underlying granulating tissue may convert the chronic wound to acute, however, due to compromised healing problems with diabetes, the patient's wound will still present the challenges of a compromised healing wound.

Wound bed preparation in practice – using TIME

In order for a diabetic foot lesion to progress to healing there must be well-vascularised granulation tissue to provide oxygen and nutrients for the proliferating epidermal cells to migrate and promote wound closure. 'Wound bed preparation provides us with a holistic structure to plan a rational approach to patient wound management, whilst within this framework the introduction of the TIME acronym focuses assessment and management on the wound bed' (Watret, 2004). TIME stands for Tissue management; Infection/inflammation control; Moisture balance and Edges/epithelial advancement of the wound (Schultz et al, 2003).

Tissue management

Each component will have an effect on the ability of the wound to move forward in the healing cascade. If, for example, sloughy tissue is the dominant factor (T) then healing will be delayed and increased risk of infection may result unless debridement takes place (Jones et al, 2004). Wet slough

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1 Aggressive surgical debridement can be used as a means to accelerate closure of diabetic foot ulcers.

2 Infection is a major problem in people with diabetes so the challenge is prevention or early diagnosis.

3 If the wound becomes critically colonised it can prevent healing progressing.

4 The diabetic foot ulcer tends to be polymicrobial and microbial interactions may create a more favourable environment for anaerobic bacteria.

5 Anaerobic bacteria and their metabolites may significantly impair the normal wound healing process.

may result in high levels of exudate and odour. Once the dominant factor is addressed and slough removed, the exudate and odour should diminish. Conversely if the slough or necrotic tissue is dry, unless it is hydrated to facilitate removal or sharp debridement takes place, the wound will not epithelialise due to the splinting effect of the dessicated tissue (*Figure 1*). There is also an increased risk of infection.

In complex wounds it is generally not possible to remove the underlying pathogenic abnormalities and consequently necrotic burden continues to accumulate. Aggressive surgical debridement can be used as a means to accelerate closure of diabetic foot ulcers (Saap and Falanga, 2002). Regular maintenance debridement may also be necessary to ensure that a build up of slough does not recur (Ayello and Cuddigan, 2004). Recurrence of slough may also indicate poor perfusion to the area and a vascular referral may be of value if this has not already been initiated.

Infection/inflammation control

Infection (I) is a major problem in patients with diabetes. Greater difficulty arises in wound diagnosis in that signs and symptoms of infection may be absent or reduced in many (Edmonds et al, 2004). The challenge in the diabetic foot is prevention or early diagnosis of infection (*Table 3*). A glossary of associated terms is provided in *Table 4*.

All wounds are colonised with bacteria and in a healthy individual this may not result in clinical infection. However, with an impaired host response infection could occur without a significant change to the wound population. If the wound moves from colonised to critically colonised it may not cause systemic infection but can prevent healing progressing. Critical colonisation (Kingsley, 2001) may respond to topical antimicrobials that reduce the bacterial burden (Jones et al, 2004). The wound may then progress to healing. If critical colonisation persists in the presence of an inappropriate host response, clinical infection may result. This situation may occur rapidly in a patient with diabetes and a clinically effective response to infection is essential, as infection may be life threatening.

Table 1. Additional underlying co-morbidity problems that may compromise healing in a person with diabetes

- Increasing age
- Increased obesity
- Cardiovascular disease
- Peripheral vascular disease
- Renal disease

Table 2. Individual patient factors that may compromise wound healing

- Social isolation/depression
- Low self-esteem
- Alcohol misuse
- Smoking
- Unemployment/inability to work
- Poor glycaemic control

The diabetic foot ulcer tends to be polymicrobial in nature. The longer a wound is in existence the greater the change in the microbial flora, resulting in a 'microbial soup' of different organism types (Bowler et al, 2004). Polymicrobial interactions may well play a crucial role in wound healing – for example, less invasive micro-organisms can be synergistic with more virulent forms (Bowler et al, 2004). A further example of synergy, cited by Percival and Bowler (2004), is that 'as the aerobic bacteria grow they consume oxygen and create a more favourable environment for anaerobic bacteria'. He also argues that 'some anaerobes may impair the host immune response to improve the survival chances for both themselves and other co-habiting organisms'.

Wound management strategies often overlook the potential role of anaerobic organisms in chronic wounds, instead focusing on aerobic organisms. Anaerobic bacteria and their metabolites may interfere with aspects of the wound healing process. Unfortunately, identifying anaerobic bacteria to provide meaningful results is not always possible in the clinical situation (Bowler et al, 2001).

Biofilms are being discussed more frequently in chronic wound management

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1 Bacteria proliferate forming micro colonies that attach to the wound bed and secrete a sugary coating (glycocalyx) that protects them from antimicrobial agents and antibiotics.

2 It is often difficult to establish what the causative micro-organisms are.

3 Management of bacterial burden involves wound irrigation, maintenance debridement and use of topical antimicrobials.

4 If moisture balance is viewed as the dominant factor in the chronic wound due to excess exudate production, it should not result in a dressing being used simply to absorb excess exudate.

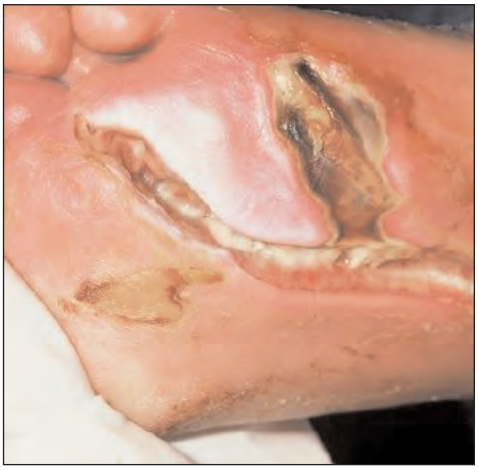

Figure 1. Slough is a dominant factor providing a barrier to healing.

(Percival and Bowler, 2004; Edwards and Harding, 2004). Biofilms occur when bacteria proliferate and form micro colonies that become attached to the wound bed and secrete a sugary coating (glycocalyx) that protects the micro-organisms from antimicrobial agents and antibiotics, which in turn can contribute to delayed healing.

'Biofilms are resistant to antibiotics administered orally, intravenously or topically' (Wysocki, 2002).

This provides a major challenge in the prevention of infection in vulnerable patients.

Quorum sensing is a method of communication used by bacteria that allows them to coordinate their group behaviour and 'form a slimy biofilm' (Wysocki, 2002). D. McCulloch and N.P. Bannister at the Smith and Nephew Research Centre (personal communication, 2004) argue that at low bacterial cell numbers there is a low concentration of auto-inducer molecules and the bacteria remain dormant, helping them to evade the immune system. As the bacterial burden increases, the auto-inducer concentration in the local environment reaches a threshold at which point the bacteria are sufficient in numbers to mount an attack on the host and switch on key virulence factors. The key to controlling this virulent attack would be to block the quorum sensing system, thereby tricking the bacteria into behaving as if they had insufficient numbers to successfully mount an attack. Further research is required in this area.

Despite a greater understanding of the potential action of bacteria on wounds, it is often difficult to establish what the causative micro-organisms are and therefore 'foot infection must be diagnosed primarily on clinical grounds' (Armstrong et al, 1995). Management of bacterial burden involves wound irrigation (Sibbald et al, 2003), maintenance debridement (Falanga, 2003) and use of topical antimicrobials (Edwards and Harding, 2004; Murray, 2004; Wright et al, 1999). Antibiotics continue to be required in the presence of clinical infection.

Table 3. Indicators of infection in diabetic foot ulcers (adapted from Edmonds et al, 2004)

- Ulcer base yellowish/grey
- Blue discolouration of surrounding tissue
- Fluctuance or crepitus on palpation
- Purulent exudate
- Sloughing of ulcer and surrounding tissue
- Sinuses with undermined or exposed bone
- Odour
- Wound breakdown
- Delayed healing

Moisture balance

If moisture balance (M) is viewed as the dominant factor in the chronic wound due to excess exudate production, it should not result in a dressing being used simply to absorb excess exudate. We should instead consider asking the question 'Why is the wound continuing to exude?'. Exudate in chronic wound healing is inextricably linked with the presence of slough and bacterial burden.

Gray and White (2004) state that assessment of the wound should involve the wound exudate continuum which takes into account not only the volume of exudate but the viscosity, which may be an indicator of the presence or absence of infection. Keast et al (2004) state that the 'Conversion of exudate to seropurulent and then to purulent drainage suggests bacterial proliferation or progression to wound infection'. The presence of tracking, with pockets of undermining slough, may be the cause of the continued high level of exudate.

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1 Once the underlying reasons for the exudate are addressed, moisture balance may be restored.

2 Build up of callus or necrotic tissue may prevent wound edges from contracting and so should be frequently removed by a podiatrist to encourage epithelialisation and restoration of the skin barrier.

3 The use of the wound bed paradigm is a valuable concept in managing the patient with diabetes with a foot lesion.

In this case it may be beneficial to cleanse and drain the wound with hydrogel or have further sharp/surgical debridement. Thorough systematic assessment is therefore required to identify the reason for the exudate. Once the underlying reasons are addressed moisture balance may be restored.

The same may be said of odour, which may be masked by the use of a 'simple' charcoal dressing. This will not remove the underlying reason for the odour, namely presence of sloughy, necrotic tissue, bacterial burden or need for more frequent dressing changes due to excess exudate.

Edges/epithelial advancement

Edges (E) are a particularly important area in the management of the diabetic foot. 'There must be a source of viable epidermal cells capable of undergoing repeated cycles of cell division' (Dodds and Hayes, 2004) to promote epithelialisation and restoration of the skin barrier (*Figure* 2). In the diabetic foot lesion the wound edges may be prevented from contracting due to a build up of callus or necrotic tissue, and frequent removal by the podiatrist is essential to encourage epithelialisation. Maceration around the wound edges may also delay healing and cause further tissue breakdown due to the destructive components of chronic wound exudate (Rodgers and Watret, 2003). There is therefore a need for the appropriate use of dressings to absorb excess exudate whilst preventing maceration to surrounding tissue.

Conclusion

Wound bed preparation provides a framework that may be universally understood by taking a holistic view of the patient with a compromised healing wound. The use of the TIME acronym promotes a structured, focused view on the wound within the wound bed preparation framework. The use of the wound bed preparation paradigm is a valuable concept in the management of the patient with diabetes with a foot lesion. ■

Table 4. Glossary of terms associated with bioburden

Bacterial effect	Definition
Anaerobic bacteria	Wounds with a sufficiently hypoxic environment are susceptible to colonisation with a wide variety of anaerobic bacteria (Bowler, 2001). With good microbial techniques, anaerobes have been isolated in 95% of diabetic wounds (Gerding, 1995).
Colonisation	Presence of multiplying bacteria with no overt host response. Continued exposure of devitalised tissue associated with a slowly-healing chronic wound is likely to facilitate the colonisation and establishment of a wide variety of endogenous micro-organisms (Bowler, 2001).
Critical colonisation/ local infection	Inability of the host to maintain a balance of organisms at the wound bed whereby 'wound healing may be delayed in the absence of typical clinical features of infection' (Edwards and Harding, 2004).
Infection	Overpowering of host response, presence of multiplication and increased virulence of organisms with spreading cellular damage, provoking systemic response.
Synergy	Interdependent action of bacterial strains that may affect the pathogenicity of the organisms. They have the capacity to have a direct effect on wound healing through the production of destructive enzymes and toxins. Mixed communities of organisms may also indirectly affect healing by promoting a chronic inflammatory state.
Biofilm	Biofilms occur when bacteria proliferate and form micro colonies that become attached to the wound bed and secrete a sugary coating (glycocalyx), which in turn helps protect the micro-organisms from antimicrobial agents. The presence of water channels allows the transfer of nutrients and waste products between colonies.
Quorum sensing	Production of signaling molecules within a bacterial population that enables bacteria to communicate with each other and initiate a response to their surrounding environment once a critical population density has been reached (Percival and Bowler, 2004).
Host response	The ability of an individual's (host) immune response and ability to respond to bacteria during good health. When illness occurs the host response may be impaired (resulting in immunocompetency failure).

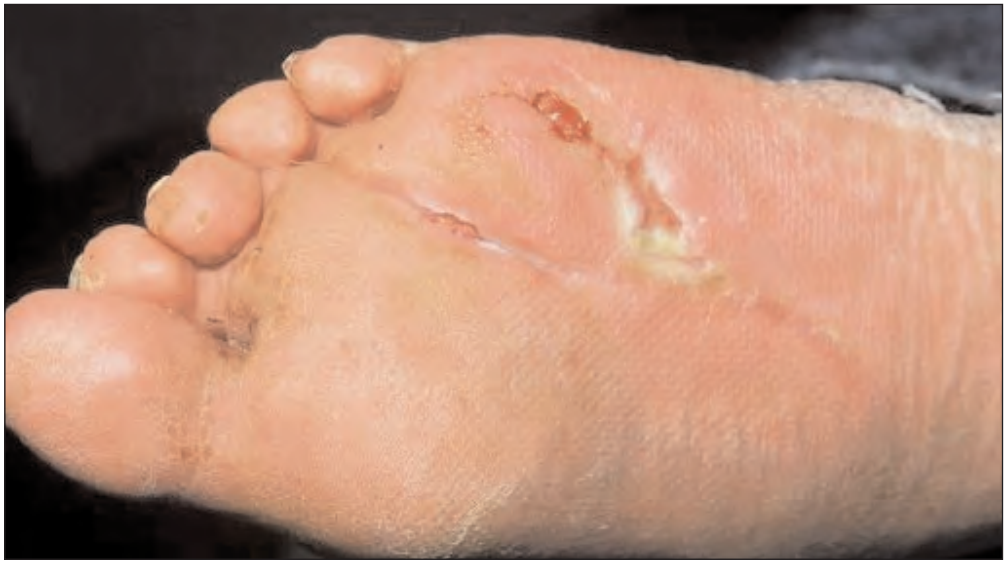

Figure 2. Frequent removal of callus or necrotic tissue by a podiatrist is required to encourage epitelialisation.

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