Chronic wounds arise when the normal healing process has stalled. Interventions based on patients’ individual history can set them on the path to healing.

How to address wound healing complications

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When patients are in good medical and nutritional condition, with normal blood flow, acute wounds should heal normally. In chronic wounds, however, healing is more problematic, as the underlying causes of the wound are more difficult to determine.

A thorough, holistic assessment of the patient, above and beyond that required of acute or uncomplicated wounds, is needed so the underlying causes can be understood. For more information on wound assessments, see parts 2 and 4 of this series (Brown, 2015a; Hampton, 2015a).

Even when known, the problems that delay healing are often complex and difficult to treat (Attinger and Bulan, 2001). Chronic wounds are defined as wounds that have failed to proceed through an orderly and timely healing process over a period of three months (Mustoe et al, 2006). These wounds have become stuck in one of the phases of wound healing, generally considered to be chronic inflammation (Guo and DiPietro, 2010). They are unlikely to heal without assistance.

There are many types of chronic wound, including leg ulcers, diabetic foot ulcers, pressure ulcers, dehisced surgical wounds, complicated burns and fungating wounds. Each type has its own particular traits and complications, although in all cases, the delayed healing can be linked to poor blood supply or infection, with poor nutrition also playing a key role (Attinger and Bulan, 2001). There are also wounds that will not heal without medical intervention, such as some arterial wounds, breast cancer or wounds with clinical infection.

Good nutrition is vital to optimise healing, so regular nutritional screening should be undertaken and the results acted upon. There is evidence that psychological stress and other behavioural factors can also affect wound healing (Gouina and Kiecolt-Glaser, 2011), and patients who experience the highest levels of depression and anxiety are four times more likely to be categorised in the delayed healing group, compared with individuals who report less distress (Cole-King and Harding, 2001).

Chronic inflammation
Inflammation is a necessary and healthy response to injury, delivering defensive materials (blood cells and fluid) to the body to combat infection and wound repair. However, chronic inflammation occurs when the body’s natural immune response is prolonged or exaggerated, resulting in chronic pain, swelling and tissue damage.

5 Practice points

1. Complicated wounds need a thorough, holistic assessment in greater depth than that required of acute or uncomplicated wounds.
2. Chronic or complicated wounds have become stuck along the healing pathway.
3. Address the underlying cause to bring chronic wounds back to an acute state.
4. Unrestrained protease activity is a major underlying abnormal response of non-healing wounds.
5. Treatment priorities for terminally ill patients with non-healing wounds are based on relieving distressing or painful symptoms.
affected site. It is also a process rather than a state. Acute inflammation defends damaged tissues against bacterial invasion, while delivering mediators to stimulate the wound-healing process (Majno and Joris, 1996).

The inflammatory process initiates repair and is arguably the most important stage of healing. In normal wound healing it is a carefully controlled balance of destructive processes that are necessary to remove damaged tissue and repair processes that lead to new tissue formation (Cullen et al, 2002). In non-healing wounds, the inflammatory process has become unbalanced. The wounds fail to progress through the normal phases of healing, and remain in a chronic inflammatory state (Loots et al, 1998).

Three key factors contribute to wounds becoming chronic or non-healing:
» Poor oxygenation (poor blood supply);
» Excessive proteolytic activity;
» Colonisation/infection.

**Wound oxygenation**

The oxygen supply to wound tissues depends on adequate blood supply. Vascular disruption and high oxygen consumption by metabolically active cells can deplete the micro-environment of the wound of oxygen, causing wound hypoxia. In normally healing wounds, this hypoxia is temporary and actually triggers healing; prolonged or chronic hypoxia, however, delays wound healing (Bishop, 2008).

A number of conditions – including advancing age, diabetes, pressure, arterial or venous disease, dead tissue and infection – can impair the circulation, leading to prolonged poor tissue oxygenation (Guo and DiPietro, 2010). If oxygenation is not restored, healing is impaired.

**Proteolytic activity**

Proteases are enzymes present in wound exudate. In acute wounds, they are essential for the healing process, but in chronic wounds or chronic inflammation, they become destructive to wound healing. A key feature of non-healing wounds – and a major consequence of the persistent inflammatory response at the wound site – is uncontrolled proteolytic activity, which overwhelms local tissue protective mechanisms. There is compelling evidence that uncontrolled protease activity is one of the major underlying abnormal responses of non-healing wounds (Barrick et al, 1999).

Proteases are secreted by the cells involved in the repair process, such as fibroblasts and endothelial cells, and are also produced by immune cells stimulated by the inflammatory response or by infection. Bacterial proteases, present when bacteria are in a pathogenic state (Lebrun et al, 2009), add to the cocktail, stimulating a further immune response.

In the normal course of wound healing, there is an initial increase in matrix metalloproteases (MMPs), which peaks at about day three and starts to reduce by around day five. In non-healing wounds, however, not only do proteases reach higher levels than in healing wounds, but they persist for far longer, resulting in a highly destructive wound environment (Wounds International, 2011).

MMP activity decreases significantly as healing occurs in chronic leg ulcers, mirroring the processes observed in normally healing acute wounds. This supports the case for the addition of protease inhibitors in chronic wounds (Wounds International, 2011). A range of products can inhibit MMP activity and make the wound environment unsuitable for bacteria. These include honey, which reduces the pH; hydrofibres, which absorb bacteria and reduce the number of MMPs at the wound bed; and dressings with a bacterial binding action. For more information about dressings, see part 5 of this series (Hampton, 2015b).

**Colonisation/infection**

When skin is damaged, micro-organisms that are normally found on its surface gain access to the underlying tissues. The bacterial load determines whether the wound is classified as having contamination, colonisation, local infection/critical colonisation, and/or spreading invasive infection (Guo and DiPietro, 2010).

Bacteria can also stick together and develop a protective biofilm that shields them from the phagocytic activity of invading neutrophils (the most abundant circulating blood leukocytes). It is likely that the presence of biofilms containing *Pseudomonas aeruginosa* are the cause of many non-healing wounds (Fig 1). This mechanism may explain the failure of systemic antibiotics as a remedy for chronic wounds (Bjarnsholt et al, 2008).

**Wound types**

**Leg ulcers**

All patients with leg ulcers should be assessed for arterial disease before compression therapy is initiated (Fig 2). This can be most easily achieved by measuring their ankle/brachial pressure index (ABPI), which must be between 0.8 and 1.2 to qualify for compression therapy. If the result is outside this range, the patient is likely to have arterial disease and the advice of a vascular specialist nurse or GP should be sought.

In arterial ulcers, restoration of blood flow by revascularisation is the intervention most likely to lead to healing. If tests show that an ulcer is likely to be due to poor arterial flow, the patient should be referred to the vascular team, who will aim...
Oedema is caused by venous insufficiency, in which damaged venous valves allow backflow of blood, which pools in the legs instead of returning to the heart. This can cause capillary damage, allowing fluid to leak into the tissues.

The compression bandage fulfils the function of damaged valves, with graduated compression forcing the blood up the legs and reducing oedema.

The Lindsay Leg Club Foundation (www.legclub.org) aims to empower patients with leg problems through community-based leg clubs and can be helpful to patients with leg ulcers. They provide leg-ulcer management in a social environment; in addition to improving healing rates and reducing recurrence, they can reduce social isolation and stress levels, as well as improving patients’ general wellbeing.

Diabetic foot ulcers

Diabetic foot ulcers (Fig 4) are a serious complication of diabetes and precede 84% of all diabetes-related lower-leg amputations (Brem and Tomic-Canic, 2007). More than 100 known physiological factors contribute to wound-healing deficiencies in people with diabetes and, coupled with an impaired ability to fight infection, these patients become largely unable to mount an adequate inflammatory response.

Although a diabetic foot ulcer might look like a healing wound, it can become a portal for infection that can lead to sepsis and require limb amputation (Brem and Tomic-Canic, 2007). These ulcers often require debridement to stimulate healing (Saap and Falanga, 2002), but this should only be performed by clinicians who have been fully trained in debridement of the diabetic foot.

Infections are a serious complication in diabetic foot ulcers and increase the risk of amputation. Antimicrobial dressings can help to prevent colonisation and infection. A swab should be taken if an infection is suspected in a diabetic foot ulcer.

Restoration of oxygen levels (blood supply) to the leg is also vital, so shoes should be checked for pressure points and patients referred to a podiatrist for off-loading devices if necessary.

Blood glucose levels and nutritional status should also be checked, as these are of particular concern and can adversely affect the wound-healing process.

Pressure ulcers

Pressure ulcers (Fig 5) are caused by prolonged pressure, friction or shearing forces and the vast majority could be avoided if all the necessary prevention measures are in place. If a patient presents with a pressure injury, it is worth checking the necessary equipment is in place, and the people caring for the patient know how to use it and understand prevention techniques. These include regular repositioning and the use of pressure-relieving equipment, as appropriate, following a full risk assessment of the patient (National Institute for Health and Care Excellence, 2014).

Before dressings are selected, pressure on the affected area must be relieved and oxygen delivery to the skin optimised. Colin et al (1996) demonstrated a dramatic impairment of oxygen supply to the skin in the 90-degree laterally inclined position but not in the 30-degree laterally inclined position. Pressure should therefore be reduced by placing the patient in a 30-degree position, and appropriate dressings for the individual wound selected.

Choice of dressing will depend on grade and depth of the pressure ulcer and the condition of the tissue:

- Black [necrotic] tissue requires a wet dressing, such as a hydrogel and honey;
- Malodorous wounds require an antimicrobial dressing;
- Granulating tissue requires protection, for example, with foam dressings, hydrocolloids and silicone dressings.

When the wound is dry, there is rarely colonisation, but when wet, it can become colonised and require protection against infection. Some dressings reduce the potential for infection by debriding of the wound. These include hydrogel sheets, amorphous hydrogel, honey, and hydrofibres, while maggot therapy can also be used.

Once the wound is debrided, activated carbon cloth (ACC), hydrofibres, silicone or foams can be considered.

Topical negative pressure (TNP) therapy can also be effective. TNP therapy uses a sealed dressing connected to a vacuum pump, and draws out fluid while promoting bloodflow.
Surgical wounds
Surgical site infections, and wound and tissue dehiscence (rupture of the wound), are well-known post-operative complications, particularly following gastrointestinal surgery (Fig 6).

As with pressure ulcers, prevention is the priority. Patients should receive appropriate preoperative preparation for surgery and postoperative care, as described in the NICE (2008) guidelines.

The severity of surgical-wound complications range from mild cases needing local wound care and antibiotics, to serious cases requiring surgery and with a high mortality rate.

Infection is not always the cause of dehiscence. Other causes include haematoma, nutrition, vascular supply, oedema and patient interference. In most cases, such complications prolong hospitalisation, with a substantial increase in cost of care (Sørensen et al, 2005). TNP can be effective for these wounds as it encourages oxygen into the tissues and removes bacteria, although it is not always a suitable and cost-effective choice for every wound that has dehisced. For example it should not be used on wounds associated with cancer or wounds that connect to a body organ. TNP should not be used on exposed organs without special dressings produced specifically for this situation.

Fungating wounds
Fungating wounds (Fig 7) are a particular type of non-healing wound associated with advanced cancers. Since these wounds are unlikely to heal, nurses should focus on different management objectives. Care should be palliative, addressing uncomfortable and distressing symptoms, rather than offering the aggressive treatments that strive for an optimum healing environment. The first priority is to control pain, odour, bleeding and exudate; removal of necrotic tissue is a lower priority. For more information about wound pain assessments, see part three of this series (Brown, 2015b).

It is important to consider patients’ psychosocial needs, as well as their clinical condition, when suggesting treatments. For example, offering maggot therapy to someone with a potentially terminal disease could be a reminder of the process of death, while leakage or offensive odour may be causing significant psychological distress. Patients must be fully involved in any decisions about wound treatments and dressings, and their individual needs should be considered at all times.

Keeping the wound clean is important, as fungating wounds can be the seat of infection or colonisation. These wounds are mostly colonised by anaerobic bacteria, which creates a very offensive odour.

Discussion
All chronic wounds have some similarities in terms of depleted oxygen to the tissues and colonisation that can lead to clinical infection. These issues must be addressed if the wound is to have an opportunity of healing. However, colonisation, which can be addressed by antimicrobial dressings, is often mistaken for clinical infection, which requires antibiotics. This can lead to the unnecessary use of antibiotics.

Conclusion
Addressing the lack of oxygen to a wound, reducing the potential for clinical infection, applying the appropriate dressing and undertaking holistic patient assessment are all vital elements of preventing and healing chronic wounds. Reducing the occurrence of chronic wounds would save money for the NHS, free nurses to spend time on other essential care and, most importantly, improve outcomes for patients. NT

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