Caring for chronic wounds in the community

Annemarie Brown

Many chronic wounds are cared for by healthcare professionals in the community and can be challenging to manage. This article explains why wounds become chronic at a cellular level. It also discusses some of the contributory systemic factors that contribute to a wound becoming chronic, such as unrelieved pressure, unmanaged venous hypertension and poorly controlled diabetes. In addition, it explores local barriers to chronic wound healing, such as excessive exudate, high bacterial burden and the identification of local and systemic infection. Advice is given on how to identify biofilms in chronic wounds, together with suggestions on how to successfully remove these, based on latest guidance. The debridement of slough, a further barrier to healing, is also covered, together with the different methods available for community healthcare professionals to use.

KEYWORDS:
- Chronic wounds
- Barriers to healing
- Debridement
- Biofilm

It has been estimated that the cost of caring for chronic non-healing wounds is in the region of £3.2 billion per annum (Guest et al, 2015; Guest et al, 2017). Management of chronic wounds, which include leg ulcers, diabetic foot ulceration, pressure injuries and other slow-to-heal wounds tends to take place within primary care and presents a challenge for both healthcare professionals and patients alike (Dowsett et al, 2014; Murray, 2019).

From the healthcare professional’s perspective, they are complex to manage and for the patient, the lack of progress to healing is frustrating and impacts on quality of life in a negative way (Murray, 2019).

All wounds, whether healing by primary or secondary intention, pass through the same stages of healing: haemostasis, inflammation, proliferation, contraction and scarring (Martin, 2013). The purpose of the stages is to clean the wound, ensuring it is in the optimum condition for healing and finally rebuild lost tissue. The wound therefore passes through each phase, with some overlapping, before progressing onto the next.

Chronic wounds, however, heal differently from acute wounds, such as surgically created or trauma wounds, because they do not progress through the normal healing trajectory in an orderly way. Kantor and Margolis (2000) found that a reduction in wound surface of 20–40% after two to four weeks of treatment was a reliable indicator of healing in venous leg ulcers. For diabetic foot ulcers, a wound reduction of greater than 50% by four weeks was found to be predictive of healing (Sheehan et al, 2003; Lavery 2008; Synder et al, 2010). This is an approximate measure of healing, as wounds vary according to their aetiology, as well as patient factors, such as the maintenance of optimum blood glucose levels, and appropriate management, for example, optimum compression therapy or adequate pressure relief.

Acute wounds pass through these stages but at a much more accelerated and uneventful pace; although these may also become chronic as a result of intrinsic factors, such as medication or comorbidities (Murray, 2019).

More recently, these definitions of ‘acute’ and ‘chronic’ wounds have been challenged as being too simplistic and there are calls for wounds to be categorised as either ‘low risk uncomplicated wounds which are likely to heal’ (acute), or ‘high risk complicated wounds which are challenging to heal but where healing is possible’ (chronic) (Martin, 2013; Murray, 2019).

WHY DO WOUNDS BECOME CHRONIC?

Imbalance at the cellular level

Following injury or a breach in the integrity of the skin resulting in a wound, platelets are activated to form a blood clot. The platelets also release factors that attract immune cells from the general circulation into the wound and this process starts the inflammatory phase of healing (Krzyszczyk et al, 2018). Following this, neutrophils and monocytes appear which further develop into macrophages. The purpose of these cells is to stabilise the wound by removing any debris, bacteria and dead cells and prepare it for healing (Sindrilaru and Scharfetter-Kochanek, 2013). Once complete, the wound moves into the proliferative phase where new tissue is regenerated in the presence of endothelial cells, fibroblasts and keratinocytes. Finally, the wound moves into the remodelling phase when the new tissue, now granulation tissue, matures...
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regeneration of tissue, as they regulate the breakdown of collagen and growth factors, which, in turn, stimulates the development of new tissue and reepithelialisation (Caley, et al, 2015).

It is vital, therefore, that MMPs are activated and that levels decrease in a tightly controlled manner to ensure that the balance of tissue destruction versus tissue regeneration is maintained for healing to occur. Essentially, a wound that is stuck in the inflammatory phase will become a chronic, non-healing wound if the bacterial balance is not restored and the wound bed cleared of debris (Martin, 2013).

Local and systemic barriers to healing at wound level

There are many physiological local and systemic factors that can result in a wound becoming chronic (Table 1).

Some risk factors can be modified to prevent a non-healing wound, such as encouraging smoking cessation, optimising blood sugar levels, ensuring adequate nutritional status, managing anaemia and losing weight. However, the primary focus should be on accurate identification and correction of the causative and contributing factors (Martin, 2013).

Table 1: Effects of local and systemic factors on wound healing (adapted from Martin, 2013)

<table>
<thead>
<tr>
<th>Local wound factors</th>
<th>Effect on healing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inadequate tissue perfusion</td>
<td>Increased risk of infection</td>
</tr>
<tr>
<td>Non-viable tissue</td>
<td>Increased risk of infection</td>
</tr>
<tr>
<td>Wound infection</td>
<td>Prolongs inflammatory response</td>
</tr>
<tr>
<td>Excess MMPs</td>
<td>Unregulated breakdown of collagen and key growth factors</td>
</tr>
<tr>
<td></td>
<td>responsible for healing</td>
</tr>
<tr>
<td>Systemic factors</td>
<td>Effect on healing</td>
</tr>
<tr>
<td>Immune deficiency/suppression</td>
<td>Slow reproduction of key cells in healing process and their</td>
</tr>
<tr>
<td>caused by medication/</td>
<td>functions; nutritional deficiency</td>
</tr>
<tr>
<td>human immunodeficiency virus (HIV)/cancer</td>
<td></td>
</tr>
<tr>
<td>Systemic conditions, such as</td>
<td>Hyperglycaemia, increased risk of wound infection and wound</td>
</tr>
<tr>
<td>diabetes, anaemia</td>
<td>ischaemia. Low levels of oxygen may impact on granulation</td>
</tr>
<tr>
<td></td>
<td>tissue development in the latter stages of wound healing</td>
</tr>
<tr>
<td>Old age</td>
<td>Impaired immune response</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>Low levels of protein vitamins and trace minerals may result in</td>
</tr>
<tr>
<td></td>
<td>sub-optimum new tissue to fight off infection</td>
</tr>
<tr>
<td>Obesity</td>
<td>Increased risk of infection, wound dehiscence</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>Reduces oxygen levels and is associated with higher incidence</td>
</tr>
<tr>
<td></td>
<td>of wound complications</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>Detrimental effect on growth factors and the development of</td>
</tr>
<tr>
<td></td>
<td>collagen</td>
</tr>
<tr>
<td>Not treating the underlying cause</td>
<td>If this is not addressed, the wound will fail to heal and is at</td>
</tr>
<tr>
<td>of the wound appropriately</td>
<td>risk of becoming stuck in the inflammatory phase of healing as a</td>
</tr>
<tr>
<td></td>
<td>result of long duration, for example, increased bacterial burden</td>
</tr>
</tbody>
</table>

In chronic wounds, the proliferative and remodelling phases do not readily occur, and the wound remains stuck in the inflammatory phase, and is unable to regenerate new tissue and progress to healing (Frykberg and Banks, 2015).

Chronic wounds have also been found to contain excessive levels of proinflammatory cytokines, matrix metalloproteinases (MMPs) and sluggish cells, i.e. cells which do not respond appropriately to growth factors, which are signalling molecules between the different types of cells required for the wound healing process (Frykberg and Banks, 2015; Martin and Nunan, 2015).

MMPs are present in both acute and chronic wounds and are vital in the

and strengthens as the vascular and myofibroblasts die. Healing is complete with the formation of a collagen-rich scar (Zhao et al, 2016).

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Similarly, ensuring good glucose levels are achieved in patients with diabetic foot ulcers or correcting anaemia and poor nutritional status will help prevent the barriers to

Figure 1.
Diabetic foot ulcer showing no signs of healing.

Figure 2.
Chronic venous leg ulcer with poor quality granulation tissue and the presence of shiny biofilm.

Figure 2.
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NICE concluded that Debrisoft® is more clinically and cost effective than other debridement methods

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healing, as outlined in Table 1. Revascularisation of the limb when arterial circulation is compromised, for example, in the case of arterial leg ulceration or diabetic foot ulceration, may also be the only realistic approach to achieve wound healing.

MODIFIABLE WOUND-RELATED BARRIERS TO HEALING

Effective exudate management

In acute wounds, exudate has an important and beneficial role in wound healing. It develops from serum and contains electrolytes, glucose, cytokines, leukocytes, MMPs, macrophages and other microorganisms (Cutting 2004; White and Cutting, 2006). It creates a moist environment for optimal healing by providing a medium for cells and growth factors to migrate, and facilitates autolytic debridement of dead tissue and bacteria (Cutting, 2004; White and Cutting, 2006; Martin, 2013). In the inflammatory phase, there is generally a copious volume of exudate, however as the wound passes through the healing process, the volume of exudate gradually reduces to provide the optimum amount of moisture to facilitate healing.

In chronic wounds, exudate contains excessive levels of MMPs and other destructive compounds, which will degrade soft tissue and prevent new tissue developing (Cutting, 2004). Exudate in chronic wounds tends to be copious and the composition, therefore, is considered a ‘wounding agent’, which is detrimental to healing (Trengrove et al, 2000; Bianchi, 2012). Unless the imbalance of uncontrolled destruction of tissue and poor regeneration of new tissue is addressed, the wound stalls in the inflammatory phase and healing can no longer be predicted (Barrett, 2016). To achieve this, healthcare professionals should be familiar with the composition and mode of action of dressings, which form an important component in maintaining this balance.

When there is an excessive volume of exudate present, superabsorbent dressings, such as Cutimed® Sorbact® (Essity), DryMax Extra® (Aspen Medical), Vliwasorb® (L&R), or Cutimed® Sorbion Sachet S® (Essity), which have increased fluid-handling capacity and may be helpful in managing high exudate volume, will provide an optimum wound environment (Jones and Barraud, 2013). Foam dressings, although available in several absorbencies, may not have sufficient fluid-handling capability on their own to manage excessive levels of exudate. However, the additional use of secondary dressings, such as hydrofibers (sodium carboxymethylcellulose), for example, Aquacel® (ConvaTec) or alginate-based dressings, for example, Kaltostat® (Convatec) may help to manage the exudate. These can also be used with superabsorbent dressings to enhance fluid handling. In addition, these dressings have been found to modulate and reduce activity of MMPs and other destructive compounds found in chronic wounds by binding them within the dressing matrix, rendering them inactive (Rayment et al, 2008). Figure 3 is an example of poor exudate management.

MANAGING WOUND MARGINS

With highly exuding wounds, there is a risk of further skin breakdown around the wound site and eventual extension of the wound itself. As stated earlier, the exudate in chronic wounds is corrosive and, if left in contact with intact skin for long periods, the surrounding skin will become macerated, excoriated and painful for the patient (Brown 2016). Maceration presents as a white, ‘soggy’ discolouration around the wound as a result of saturated keratocytes and eventually the top layer of skin will be removed (Thompson and Stephen-Haynes, 2007). To avoid this, a dressing with enough fluid-handling capacity should be selected, changed at appropriate intervals according to exudate volume, and the use of skin barriers products, such as the Cavilon® range (3M), Cutimed Protect Film or Cream® (Essity) or LBF Barrier Cream® (CliniMed) will help prevent this occurring (Brown 2016; 2017). Figure 4 shows a wound with a high bacterial burden and maceration to the wound due to inadequate fluid handling.

OPTIMISING THE WOUND BED — CHRONIC WOUNDS

Debridement

In normal wound healing by secondary intention, any non-viable
tissue, slough and bacteria will have been removed by the actions of morphonuclear leukocytes, proteases and oxidants during the inflammatory phase of wound repair. Low levels of bacteria in any open wound will inevitably be present. However, if the levels are low, the wound will be classed as ‘colonised’, meaning that there is no systemic response from the patient and healing will not be delayed (Edwards-Jones and Flanagan, 2013; Swanson et al, 2015).

Chronic wounds, however, are known to have a high level of bacteria present, and as this level increases, the inflammatory response releases excessive amounts of cytotoxic enzymes, oxygen-free radicals and inflammatory mediators, which damage the wound bed (Edwards-Jones and Flanagan, 2013). This is called ‘critical colonisation’ and starts the cycle of destruction of tissue and poor regeneration of new tissue, with the wound becoming stuck in the inflammatory phase.

To reduce the bacterial burden within the wound, any necrotic or sloughy tissue must be debrided. Methods include autolytic, which uses wound dressings to provide a moist environment that enhances the action of proteolytic enzymes, enabling necrotic/sloughy tissue to liquefy and separate from the wound bed (Flanagan, 2013). Other methods include:

- Mechanical debridement using disposable debridement pads, such as Debrisoft™ (L&R), Prontosan Debridement Pad™ (B Braun), or UCS™ (medi UK)
- Sharp debridement, which can only be performed by qualified healthcare professionals (Timmons and Morris, 2018).

Further methods of debridement, such as hydrosurgical debridement, which uses pressurised water or saline, is an effective, safe and speedy alternative to sharp debridement but is usually only performed in specialist wound care centres (Flanagan, 2013). Figures 5 and 6 show wounds that would benefit from debridement.

**BIOFILM**

If persistent slough formation continues in a static wound despite ongoing regular debridement, it should be assumed that the wound bed contains biofilm. Biofilm is formed by a mixture of different microbes.
embedded in the wound in a slimy matrix, which is difficult to remove from the wound bed (Wounds UK, 2017). The microbes communicate and work together and thrive within the wound environment and develop resistance both to antibiotics and antimicrobials, as they are protected by the slimy matrix (Siaw-Sakyi, 2018). In clinical practice, it is often not possible to visualise a biofilm within the wound, however the following are signs where the presence of a biofilm should be suspected:

- Delayed healing despite appropriate wound management and all other contributory factors have been addressed (for example, compression therapy in venous leg ulcers)
- Failure to respond to antibiotic and antimicrobial therapy
- Inconclusive wound culture results
- Signs of a shiny gelatinous mass, which is removed from the surface but reforms in 1–2 days
- Persistent signs of low-grade local infection or critical colonisation, such as increased exudate, stalled healing, alternating periods of improvement and deterioration
- Poor quality or abnormal-looking granulation tissue
- Increased exudate due to increased vascular permeability as a result of the inflammatory response

(Adapted from Wounds UK, 2017)

Managing biofilm

Biofilm can be disrupted by vigorous and regular debridement, but are not removed by routine cleansing or irrigation methods (Atiyeh et al, 2009; Wolcott et al, 2009; Bianchi et al, 2016; Fletcher et al, 2016; Siaw-Sakyi, 2018). In the author’s clinical experience, using debridement pads is generally the easiest method in the absence of healthcare professionals trained in sharp debridement. Unfortunately, biofilm will return within a few days post-debridement, however the period between debridement and re-forming has been found to be a brief window of opportunity where the bacteria are no longer resistant to topical antiseptic or antimicrobial treatments (Wolcott et al, 2009).

A best practice statement on the management of biofilm has developed a simple regimen to treating suspected biofilm formation which is named the Two-week challenge (Wounds UK, 2017).

<table>
<thead>
<tr>
<th>Table 2: Two-week challenge (Wounds UK, 2017)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Week one</strong></td>
</tr>
<tr>
<td>1. Change dressings a minimum of three times during the week and at each dressing change</td>
</tr>
<tr>
<td>2. Disrupt and remove the biofilm mechanically, preferably with a debridement pad</td>
</tr>
<tr>
<td>3. Reduce microbial load by applying a topical antimicrobial dressing, such as honey, silver or iodine and/or use a topical antiseptic preparation such as Octenidine dihydrochloride or polyhexamethylenebiguanide (PHMB) (for example, Octenilin Wound Gel™ or Prontosan Wound Gel™) with a suitable dressing</td>
</tr>
<tr>
<td>4. Maintain this for one week</td>
</tr>
</tbody>
</table>

**Improved?**

If the wound has improved and is showing signs of healing after two weeks, discontinue the use of antiseptics and revert to standard care. Regular debridement may still be required, particularly if the wound starts to stall again. In wounds at high risk of infection, the continued use of antiseptic or antimicrobial treatments may be justified, as per local guidelines.

**Not improved?**

Reassess the patient and the wound and amend the treatment plan if necessary. The antimicrobial/antiseptic agent which has been used for the two weeks should be changed to an
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alternative and a further two-week challenge started.

Deteriorated?
Reassess the patient and the wound and amend the treatment plan if necessary. The antimicrobial/antiseptic agent which has been used for the previous two weeks should be changed to an alternative, if still appropriate, and a further two-week challenge started. If following this there is still no improvement, referral to a specialist may be appropriate (Wounds UK, 2017).

**WOUND INFECTION IN CHRONIC WOUNDS**

Chronic wounds are particularly at risk of developing infection due to the presence of slough, bacteria and biofilm (Brown, 2018). Table 3 gives a list of subtle and more obvious signs of local infection.

If several of these signs are present, it should be assumed that the wound has a local infection and interventions are required to reduce the bacterial burden (Brown, 2018).

The use of topical antiseptic solutions, for example, Oectenilin Wound Gel® (Schulke) or Prontosan Wound Gel® (B Braun), combined with antimicrobial wound dressings containing silver, iodine, honey or polyhexamethylene biguanide (PHMB) for an initial two-week period is recommended. Trust guidelines or formularies should be consulted for appropriate duration, re-assessment and usage. Wound swabbing is not necessary for local infection diagnosis (Brown, 2018). Figure 8 shows a wound tipping from high bacterial burden into local infection.

If the signs of spreading infection are present, such as increasing erythema, lymphangitis, crepitus, wound breakdown and the patient begins to feel unwell, wound swabbing and systemic antibiotic therapy may be required.

**CONCLUSION**

Managing chronic or non-healing wounds presents challenges and frustrations for both healthcare professionals and patients alike. This article has discussed some of the most common complications found in chronic wounds and has offered practical suggestions on how to manage them. Unfortunately, despite eliminating these problems, some wounds will still not progress to healing and, in these cases, the focus will be on managing symptoms, such as excessive exudate or malodour to enable the patient to lead as normal a life as possible. JCN

**REFERENCES**


**Practice point**

It is thought that at least 78% of chronic wounds contain biofilm (Malone et al, 2017), which contributes to chronicity in some patients.

**Table 3: Signs of local wound infection (adapted from Brown, 2018)**

<table>
<thead>
<tr>
<th>Subtle signs</th>
<th>Obvious signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence of hypergranulation</td>
<td>Erythema</td>
</tr>
<tr>
<td>Delicate, bleeding tissue</td>
<td>Local warmth</td>
</tr>
<tr>
<td>Epithelial bridging and pocketing in</td>
<td>Swelling</td>
</tr>
<tr>
<td>granulation tissue</td>
<td></td>
</tr>
<tr>
<td>Wound breakdown or extension for no</td>
<td>Purulent discharge</td>
</tr>
<tr>
<td>apparent reason</td>
<td></td>
</tr>
<tr>
<td>Delayed wound healing</td>
<td>Delayed healing</td>
</tr>
<tr>
<td>New or increased pain</td>
<td>New or increased pain</td>
</tr>
<tr>
<td>Increasing malodour</td>
<td>Increasing malodour</td>
</tr>
</tbody>
</table>

It is thought that at least 78% of chronic wounds contain biofilm (Malone et al, 2017), which contributes to chronicity in some patients.
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KEY POINTS

- It has been estimated that the cost of caring for chronic non-healing wounds is in the region of £3.2 billion per annum.

- Chronic wounds, however, heal differently from acute wounds, such as surgically created or trauma wounds, because they do not progress through the normal healing trajectory in an orderly way.

- In chronic wounds, exudate contains excessive levels of MMPs and other destructive compounds, which will degrade soft tissue and prevent new tissue developing.

- A moist wound bed provides the optimum environment for wound healing; however, a balance between excessive moisture and the wound bed becoming dried out is required.

- To reduce the bacterial burden within the wound, any necrotic or sloughy tissue must be debrided.

- Biofilm can be disrupted by vigorous and regular debridement, but is not removed by routine cleansing or irrigation methods.

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