Abstract
Wounds have traditionally been classified as acute or chronic. While this classification is useful when attempting to estimate healing times, it might lead to an acceptance that some wounds will take longer to heal or might not heal at all. Chronic wounds can adversely affect patients’ quality of life, and the management of these wounds may involve significant healthcare resources and costs. Chronic wounds rarely develop in healthy individuals and are often associated with pre-existing conditions that complicate wound healing, such as diabetes mellitus and vascular disease. This article discusses how acute wounds and chronic wounds are differentiated. It details the phases of wound healing and identifies potential barriers to progression through these phases. Enhancing nurses’ understanding of chronic wounds will enable them to identify any potential barriers to wound healing early and remove or ameliorate them. While a holistic assessment should also include a thorough assessment of the wound itself, this is beyond the scope of this article.

Phases of wound healing
Most wounds heal in a predictable manner, progressing through the four phases of healing (McFarland and Smith 2014). However, factors such as clotting disorders, infection and conditions such as diabetes mellitus can adversely affect the wound at each stage, thus delaying healing. While evidence suggests that delays often occur in the inflammatory phase, all phases of healing can potentially be disrupted.

Why you should read this article:
- To refresh your knowledge of the normal stages of wound healing and how to differentiate between acute wounds and chronic wounds
- To understand the patient-related factors that can support and inhibit wound healing
- To support the early identification of chronic wounds using a holistic patient assessment, thus promoting effective wound healing

**Haemostasis**
During haemostasis, damaged blood vessels constrict to reduce blood loss, platelets form an initial plug and release serotonin to further reduce blood loss, and thrombin forms a blood clot (Vuolo 2009). This process should take place within minutes of wound formation. However, there are occasions when this does not occur, for example when the blood vessel damage is too significant to be occluded, if the patient has a clotting disorder such as haemophilia, and if the patient is taking anticoagulants such as warfarin sodium or aspirin and may require additional time for haemostasis and clotting to take place (Peate and Glencross 2015). During haemostasis, adhesive proteins are also released to promote platelet aggregation, and growth factors promote cell recruitment and early extracellular matrix (ECM) formation.

**Inflammation**
During inflammation, the body’s blood vessels become dilated and white blood cells such as leukocytes and macrophages are released into the damaged area in an attempt to destroy any invading bacteria and cleanse the wound (Wolcott et al 2008, Flanagan 2013). Growth factors are secreted to further enable ECM deposition, collagen synthesis and formation of new blood vessels (angiogenesis).

The presence of comorbidities such as diabetes, together with the medicines the patient is taking, can negatively affect this phase of healing. For example, anti-inflammatory drugs such as corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs) can compromise this stage of healing because the normal flow of blood observed during inflammation is impeded, thereby reducing the presence of white cells in the wound area and increasing the risk of infection (Peate and Glencross 2015). The management of inflammatory conditions such as Crohn’s disease, ulcerative colitis or rheumatoid arthritis can involve patients taking corticosteroids, which affect the inflammatory stage of healing, delaying the healing process or resulting in the wound becoming stuck in the inflammatory phase and increasing the risk of infection. Conditions such as diabetes and peripheral vascular disease, as well as impaired cardiac or pulmonary function, can also reduce perfusion to the wound bed, adversely affecting the inflammatory stage of healing and increasing the risk of infection (Sharp and Clark 2011).

**Proliferation**
The proliferative phase of healing involves the development of new granulation tissue, which can take time particularly if the patient has experienced extensive tissue loss. New blood vessels develop to provide the wound bed with a supply of nutrients and oxygen, from which fibroblasts (collagen-producing cells) develop collagen fibres and ground substance (Vuolo 2009). These collagen fibres along with myofibroblasts (large cells that secrete collagen but also have contractile properties) contract the wound margins before the wound develops a covering of epithelial cells, which then differentiate into durable epidermal tissue.

Delays in the proliferative phase can be caused by infection, where the wound effectively remains in the inflammatory stage of healing until the bacterial level is brought under control. The actions of the nurse can result in delays in the proliferative phase, for example if the wound dressing selected does not create the optimal environment for new tissue growth (Peate and Glencross 2015). If the wound environment is too moist, maceration (skin breakdown resulting from prolonged exposure to moisture) occurs and tissue is destroyed rather than being produced. Maceration can result in the wound returning to the inflammatory stage so that any fresh debris can be removed. Conversely, if the wound bed is too dry, cells cannot migrate easily across the wound and the development of new granulation tissue is delayed (Winter 1962). In addition, if the dressing adheres to the wound bed, trauma caused when the dressing is removed can return the wound to the inflammatory stage. Any trauma incurred by dressing removal can delay wound healing by up to five days (Peate and Glencross 2015).

**Maturation**
During the maturation phase, connective tissue develops strength, blood supply reduces and any scarring contracts, becoming paler. The composition of the ECM also changes to restore pre-injury tensile strength (McFarland and Smith 2014). This stage represents the end of the healing process, although tissue remodelling can continue for some time after the wound bed has been fully covered with epithelial tissue.

While the wound develops strength, it remains vulnerable. Although the nurse might no longer be involved in the direct care of the wound at this stage, the patient may still require support including advice about ongoing self-management; for example, skin dryness can cause itching and result in the patient scratching the freshly closed wound causing tissue breakdown. Sensation may also be altered as the nerve endings may have been damaged during the formation of the wound, resulting in the patient not recognising potential further tissue damage from trauma or exposure to sunlight. Such trauma can damage the newly formed epithelial tissue, resulting in wound breakdown (Vuolo 2009).

**Acute wounds and chronic wounds**
To effectively treat a wound, it is important that the nurse understands at the outset whether a wound is acute or chronic. Wounds have commonly been defined as acute where they progress through the four phases of healing within the predicted timeframes. Acute wounds tend to be the result of trauma, such as lacerations, abrasions or intentional wounding such as surgery (Vuolo 2009). Acute wounds occur suddenly and progress predictably and quickly through the phases of wound healing. These wounds typically
occur in healthy individuals, although it should be noted that any acute wound has the potential to become chronic (Doughty and Sparks-DeFriese 2012, Benbow 2016).

Chronic wounds are commonly defined as those that fail to heal within a specified timeframe. However, this timeframe is not consistent within the literature, ranging from four weeks to several months; it has been suggested that some wounds may take up to 13 months to heal (Frykberg and Banks 2015, Li et al 2017). It should also be noted that not all chronic wounds will heal.

Chronic wounds display some common characteristics and are often associated with an underlying pathophysiology that compromises healing, such as vascular insufficiency or infection (Barrett 2017, Han and Ceilley 2017). The common characteristics of chronic wounds are detailed in Box 1.

**Prevalence of chronic wounds**

The prevalence of chronic wounds has been identified as 1.47 per 1,000 of the population (Hall et al 2014). Guest et al (2017) identified that, of the wounds managed by the NHS in 2012-13, 40% were acute wounds, 48% were chronic wounds and 12% of the wounds were not classified.

Guest et al (2017) estimated that clinical commissioning groups or health boards spent an average of £9.7 million on managing acute wounds and £15.2 million on managing chronic wounds in 2012-13. Most of the total cost (78%) was incurred in the community, while wound care products accounted for 12-15% and staff costs 28% of the total cost. Guest et al (2017) also identified that chronic wounds required considerably more healthcare resources than acute wounds, including 162% more community nurse and GP visits, 100% more drug prescriptions and 178% more wound care products per patient.

**Types of chronic wound**

Some wound types are considered chronic irrespective of the stage of healing and are commonly referred to as ulcers, for example pressure ulcers, leg ulcers and diabetic foot ulcers (Doughty and Sparks-DeFriese 2012). In these wounds, the underlying pathophysiology produces repeated and sustained insults to the tissue that eventually result in damage. Failure to address the cause of these repeated insults can result in a cycle of repeated damage, while prompt intervention may promote early healing.

**Pressure ulcers**

Significant pressure from surfaces such as mattresses and seating cushions in patients with compromised mobility can compress the tissues and blood vessels in areas of the body such as the sacrum and heels, reducing or preventing blood flow and causing tissue damage because of limited perfusion. If the pressure is not removed entirely, or at least frequently, the tissues will begin to die (Vuolo 2009). Similarly, if the source of pressure is not removed, the wound cannot heal because of suboptimal perfusion.

Alongside pressure, repetitive friction such as that caused by the patient’s skin rubbing against bed sheets can damage the skin. Shear forces resulting from suboptimal patient positioning or inappropriate use of equipment, such as raising the head of the bed, can also result in the ‘stretching’ and ‘pulling’ of the skin’s underlying structures and the eventual development of a pressure ulcer.

**Leg ulcers**

Leg ulcers can be arterial or venous. Tissue damage can be caused by a lack of arterial blood supply or the ineffective removal of venous blood from the lower limb, resulting in the build-up of fluid (oedema).

Arterial ulcers develop as a result of atherosclerosis, or the destruction of blood vessels through trauma or disease such as smoking-related conditions, diabetes, and hypertension, which result in hardening and occlusion of the arteries and, in turn, a lack of perfusion in the tissues, leading to breakdown (Doughty 2012, Peate and Glencross 2015).

Venous ulceration, venous stasis can be the result of incompetent valves in the veins and a weakening of the blood vessel walls. This allows blood to ‘pool’ in the vessels of the lower limb, while

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**Box 1. Common characteristics of chronic wounds**

- Prolonged or excessive inflammatory stage
- High levels of proteases (enzymes contained in wound exudate that maintain the balance between tissue synthesis and degradation) and cytokines (small proteins that include growth factors)
- Cellular senescence (where the cells are not actively dividing, but are alive and metabolically active)
- Persistent infection
- Lack of initial bleeding at the time of wound formation (possibly the result of scar tissue from previous injury or peripheral vascular disease), which can impede fibrin production and release of growth factors

(Adapted from Frykberg and Banks 2015, Morton and Phillips 2016)

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**Key points**

- Following the formation of a wound, healing occurs as a cascade of events in four overlapping phases: haemostasis; inflammation; proliferation; and maturation (Frykberg and Banks 2015, Morton and Phillips 2016)

- Wounds have commonly been defined as acute where they progress through the four phases of healing within the predicted timeframes, or chronic where this timely progression is interrupted

- Undertaking a detailed assessment of the patient, in addition to the wound itself, will assist the nurse in identifying points in the healing cascade where the risk of delayed healing is likely to occur

Understanding the causes of pressure ulcers will enable nurses to promote healing in a timely manner. However, if preventive techniques such as appropriate use of equipment and careful patient positioning are not undertaken, the damage is unlikely to heal and will be perpetuated (Healthcare Improvement Scotland 2009, National Institute for Health and Care Excellence (NICE) 2014, Peate and Glencross 2015).

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deoxygennated blood is not returned to the heart. The pooling of blood causes the superficial blood vessels to become distended, which leads to deep vein thrombosis, inflammation and a build-up of waste products within the tissues (Vuolo 2009, Peate and Glencross 2015). Eventually, the tissues of the lower limb can break down spontaneously and any traumatic damage is unlikely to heal unless the venous stasis is corrected. Early identification of clinical signs suggesting venous disease in the lower limb – for example, pain that is relieved on elevation of the limb, oedema, brown-coloured staining of the skin, induration, itching and dilated blood vessels at the instep – can prevent venous insufficiency and promote wound healing.

Irrespective of the aetiology of a leg ulcer, it will not heal unless the underlying cause is identified and ameliorated. However, in some clinical areas, a leg ulcer is not considered as such or even assessed until it has been present for some time, even though proactive intervention from a nurse might have prevented the ulcer from developing (Vuolo 2009, Muldoon 2013). This is despite NICE (2017) guidance recommending that any lower limb wound present for two weeks should be considered a leg ulcer. The importance of early assessment and treatment of leg ulcers is supported by Gohel et al’s (2018) study into the use of early endovenous ablation (a minimally invasive treatment to cauterise varicose veins), which demonstrated faster healing times and a reduction in recurrence of venous ulcers.

Diabetic foot ulcers

Perfusion and pressure have a significant role in the development of diabetic foot ulcers, and any resulting wound will not heal in a timely manner unless these aspects are addressed (Sharp and Clark 2011). Suboptimal perfusion develops as a result of diabetes-associated atherosclerosis, which reduces the blood supply and, if not corrected, can result in ischaemia and nerve death (Vuolo 2009). Because of this nerve death, patients may lack sensation in their feet and not respond to painful stimuli (sensory neuropathy). When the motor nerves specifically begin to die (motor neuropathy), the muscles in the foot begin to atrophy, which can result in structural changes to the foot. The pressure points of the foot change, and, as the person redistributes their weight, the resultant pressure can damage the softer tissues (Vuolo 2009, Apelqvist 2013).

Suboptimally managed diabetes can also result in autonomic neuropathy (affecting the body’s non-voluntary, non-sensory nervous systems such as blood pressure regulation), which can result in increased blood flow to the lower limb, causing osteopenia (weakened bones) and subsequently Charcot’s foot (change in foot shape resulting from neuropathy and weakened bones), again with associated pressure damage (Vuolo 2009, Peate and Glencross 2015).

**Person-centred assessment**

Undertaking a detailed holistic assessment of the patient, in addition to assessing the wound itself, will assist the nurse in identifying points in the healing cascade where the risk of delayed healing is likely to occur. Wound assessment is a vital element of wound care practice (Greatrex-White and Moxey 2015). While various wound assessment tools are available – for example, the TIME (tissue, infection and inflammation, moisture balance and edge advancement) framework (Schultz et al 2004) or the Applied Wound Management framework (Gray et al 2005) – there is no consensus regarding the optimal core components of such tools. Li et al (2017) stated that the presence of pre-existing conditions such as diabetes or malnourishment means that it is possible to predict that some wounds are more likely to become chronic. Because early identification of factors that affect wound health can optimise a wound’s healing potential, a holistic assessment of the patient is as important as a thorough assessment of the wound itself (Frykberg and Banks 2015, Benbow 2016, Morton and Phillips 2016, Barrett 2017, Gupta et al 2017, Li et al 2017).

**Age**

Advancing age causes changes to the structure and function of the skin, such as reduced cell proliferation and decreased dermal vasculature, which can affect wound healing (Chamanga 2018). This results in reduced peak cutaneous blood circulation, which impedes the supply of oxygen and nutrients to the skin (Doughty and Sparks-DeFriese 2012, Gould et al 2015). Similarly, changes in the tissue at the epidermal-dermal junction, such as a flattening of the papillae, lead to increased susceptibility to skin tears as a result of shearing (Doughty and Sparks-DeFriese 2012). This is of particular concern for nurses involved in the application or removal of adhesive wound dressings because the shearing forces exerted on dressing removal can damage or ‘strip’ the skin (Reevell et al 2016).

The age of the wound itself is a factor that should be considered early in any treatment plan. Bosanquet and Harding (2014) stated that the chronicity of the wound has an adverse effect on its potential for healing, with most of the markers of chronicity (Box 1) being considered both as a consequence and a cause of wound chronicity (Frykberg and Banks 2015).

**Pre-existing conditions**

The effect of diabetes on wound healing is well documented (Driver et al 2012). Microvascular and macrovascular changes adversely affect tissue oxygenation, resulting in peripheral neuropathy and undetected tissue damage, as well as increased blood glucose levels, which have a negative effect on an individual’s immune response (Sharp and Clark 2011, Chamanga 2018, Murray et al 2018). Any condition that adversely affects the blood supply to the peripheral tissues requires careful assessment as venous and arterial insufficiency contribute to the development of lower limb ulceration. Li et al (2017) stated that, regardless of the wound’s aetiology, the condition of the patient’s vasculature is crucial for wound healing. Within the capillary beds, an effective oxygen supply
and adequate diffusion is required to enable the transfer of oxygen from the blood supply to the tissue cells, which in turn supports wound healing (Han and Ceilley 2017). Additionally, because oxygenation of the tissues is crucial, any patient with a wound should be assessed for anaemia (Nazarko 2005).

Other conditions such as thyroid disorders, cancer and inflammatory conditions should also be considered as part of the assessment because they can affect wound healing, although the details are beyond the scope of this article.

**Medicines**

A thorough examination of any medicines that the patient is taking should be included as part of any patient assessment, since these can have a significant effect on healing. For example, corticosteroids and NSAIDs suppress the inflammatory response as well as interfering with collagen synthesis and epidermal regeneration (Doughty and Sparks-DeFriese 2012). Chemotherapy medicines interfere with cell migration and may result in neutropenia and associated susceptibility to infection (Chamanga 2018). Various other medicines including anticoagulants, antibiotics, antihypertensives, tricyclic antidepressants, antipsychotics and diuretics are known to cause skin-related side effects such as photosensitivity and eruptions of skin lesions or pustules (Wysocki 2012). For example, the widely used anticoagulant warfarin can cause warfarin-induced skin necrosis (Fantus 2015).

An additional challenge for nurses caring for older people is the increased potential for the development of long-term conditions and comorbidities, and the associated polypharmacy, which can also affect wound healing (Gould et al 2015). For example, a patient might be taking an anti-inflammatory drug for a long-term rheumatological condition such as rheumatoid arthritis, alongside a chemotherapy drug such as methotrexate.

**Nutritional status**

Early assessment of nutritional status is essential to promote optimal wound healing. Protein and calories are required to promote anabolism (the synthesis of complex molecules, for example synthesising glucose), collagen synthesis and healing (Gupta et al 2017). Recommended daily intakes of protein – 45g per day for females and 56g per day for males (Public Health England 2016) – are based on the expected intake for healthy individuals; however, during wound repair an individual's protein requirements can increase (Gould et al 2015).

Vitamins A, B and C, and zinc, are important elements required for tissue repair and regeneration, while vitamin K is required for haemostasis (Stotts 2012a, Han and Ceilley 2017). People who are obese can experience malnutrition through vitamin, mineral and protein deficiencies, which can present additional challenges for wound healing, such as inadequate vascular supply of the adipose tissue (Nazarko 2005, Gupta et al 2017).

**Smoking**

Nicotine decreases capillary blood flow resulting in impaired oxygen supply to the tissues. This hardens and reduces the elasticity of the tissue because of a decrease in the levels of collagen and elastin within the dermis (Whitney 2012).

**Infection**

An explanation of the complex effects of infection on chronic wound healing is beyond the scope of this article. However, it should be noted that chronic wounds often have a high bacterial burden, which may adversely affect wound healing (Edwards-Jones and Flanagan 2013, Leaper et al 2015). Identifying infection in chronic wounds can be challenging and, as a result, it can be over-diagnosed and under-diagnosed (Edwards-Jones and Flanagan 2013). Therefore, nurses should always consider infection as a potential factor when a wound that was previously progressing through the healing phases becomes static. Taking a wound swab will assist in identifying the organisms present (Cutting and Harding 1994). Nurses should consider the application of topical antiseptics as a first-line treatment for wound infection since they are non-selective, have a broad spectrum of activity and inhibit the growth of microorganisms (Stotts 2012b).

**Socioeconomic factors**

Consideration should be given to the socioeconomic factors that can affect wound healing. For example, chronic venous insufficiency is believed to have higher prevalence in occupations where standing for long periods increases calf venous pressure, although the evidence is contradictory (Murray et al 2018). In the UK, access to healthcare resources such as wound dressings is free at the point of delivery; however, this is not the case in all countries, which can result in the cost of dressings being borne by the patient, or the provision of treatment being reliant on the patient having up-to-date medical insurance (Murray et al 2018).

**Psychosocial factors**

The psychosocial effects of chronic wounds include emotional distress because of pain and withdrawal from social and work activities, and must be considered within a holistic patient assessment (Pragnell and Neilson 2010, Dowsett and von Hallern 2017). Kapp et al's (2018) study of 25 adult patients with chronic wounds found that they all experienced adverse psychosocial effects, such as an inability to exercise or socialise as they had previously, disrupted employment and increased financial pressures resulting from wound dressing and care costs. Other studies have detailed the emotional implications of chronic wounds, particularly where wound pain and pain during dressing changes negatively affected patients' psychological well-being (Pragnell and Neilson 2010, Upton et al 2012). Kapp et al (2018) also identified that patients with chronic wounds could become frustrated and distrustful of healthcare staff because of the complexity and variation in their opinions regarding effective treatments. As a result of these factors, nurses may encounter an understandable reluctance on the part of patients with chronic wounds to continue with
treatment (Upton et al 2012, Kapp et al 2018). To improve concordance with treatment interventions, nurses should actively involve patients in any decisions about their care, as well as providing relevant information about the treatment options available, for the benefit of the various types of wound dressings (Gupta et al 2017).

Conclusion

The nursing care of chronic wounds is complex, partly because a range of patient-related factors can impede the wound-healing process, including age, nutritional status and pre-existing conditions. Furthermore, the increasing ageing population with multiple comorbidities can also complicate healing in chronic wounds. For nurses, knowledge of the factors that can affect each wound-healing phase, alongside a holistic person-centred assessment, can enable the early identification of chronic wounds and promote wound healing.

References


NICE, London.


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