JWC International Consensus Document

Identifying and treating foot ulcers in patients with diabetes: saving feet, legs and lives

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Foreword

There has been a great deal of debate around diabetic foot ulcers (DFUs) and pressure ulcers (PUs) on the feet of patients with diabetes, in terms of how to define, detect, assess and treat them. The confusion and lack of evidence in differentiating between these two types of foot ulcers, particularly on the heel, can lead to misdiagnosis, which can increase both financial and patient-related costs.

To address and tackle those inconsistencies, the *Journal of Wound Care* (*JWC*) has published its first international consensus document, *Identifying and treating foot ulcers in patients with diabetes: saving feet, legs and lives*. The main objectives of this project were to:

- Provide information on the differences between a DFU and a PU in patients with diabetes
- Help reduce misdiagnosis by providing and discussing assessment guidelines
- Make a difference in practice through improved patient outcomes.

With this in mind, an international panel of ten key opinion leaders from Australia, England, Republic of Ireland, Malaysia, Poland, Portugal, Spain, United Arab Emirates and US met on 1 and 2 March 2018 in London. They discussed the definitions of a DFU and a PU, and concluded that one way to distinguish between them is knowing whether the patient is mobile (usually associated with DFUs) or immobile (normally related to PUs), although this should be considered along with simple assessments for ischaemia and neuropathy. To this end, and given the importance of an early and correct assessment, the mnemonic 'VIPS' was suggested:

- V: vascular (ischaemia)
- I: infection (local signs, odour, exudate, slough, inflammation, etc.)
- P: pressure (causes mobility or immobility)
- S: sensation (neuropathy).

The panel also agreed that another key point was that, if the health professional treating the ulcer is unable to perform a full diabetic foot assessment, it is crucial that the patient be referred to a health professional/department who can. As many members of the professional team would usually come across an ulcer—job titles varying throughout the world—a referral pathway focusing on the referee's skills rather than their specialties was suggested.

The importance of prevention and the need to follow clear management and treatment strategies, which will vary from centre to centre, were emphasised. The issues around education were also discussed, as well as future research needed. Finally, potential new technologies or alternative therapies that could help treat a DFU or a PU when standard care fails were summarised.

Given the international focus of this document, and the various levels of knowledge among the health professionals that come across a foot ulcer, it was highlighted that this document should be read and implemented in conjunction with the clinician's local guidelines.

We hope you enjoy this document and that it helps make a difference in practice.

Camila Fronzo and Rachel Webb

Introduction

In developed countries, it has been estimated that the overall incidence of non-healing wounds is approximately 1–2%.[1] Pressure ulcers (PUs) and diabetic foot ulcers (DFUs) are among the most prevalent chronic wounds in many countries.[2,3] They are a major global clinical and health economic challenge, which is expected to escalate as the population increases, poor lifestyle leads to increased diabetes and obesity and the population ages.[4–6]

International expert consensus guidelines recommend, in general terms, similar pathways for the prevention and management of PUs and DFUs.[7] Nevertheless, critical differences in the precise delivery of effective care lie within the guidelines, which, if not administered appropriately to the diagnosis, are likely to lead, at best, to slow healing. PUs and DFUs, despite describing clinically different indications, share commonalities in definition, for example, shear and friction, pressure and ischaemia.[8] However, they require quite different approaches to management. These differences can lead to patients being managed on the wrong pathway.

This consensus paper addresses these similarities and differences with two key objectives: first, to differentiate between PUs and DFUs with regard to their definition, causes, assessment, diagnosis, management and treatment, and second, to address confusion and lack of evidence when differentiating PUs and DFUs.

Prevalence

Approximately 451 million adults worldwide have diabetes, a figure projected to increase to 693 million by 2045 globally.[4] The prevalence of DFUs will also increase in line with this. The lifetime incidence of DFUs is reported to be 25%[9] and the global prevalence of DFUs in patients with diabetes is 6.3%,[10] with wide variation by country.[11–15] When PUs occur on the foot, those on the heel are the most common;[16,17] the overall PU prevalence in five European countries in 2008 was 18.3%,[16] while more than 2.5 million people in the US develop a PU annually,[18] where the prevalence across all settings is 12.3%.[16] More recent figures suggest the prevalence of PUs in Canada is 26%[19,20] and in Western Australia between 6.3% and 9.5%.[21]

Issues around misdiagnosis

Differentiating between a heel wound that is a PU rather than a DFU presents a diagnostic challenge for clinicians. Furthermore, the prognosis, complications and treatment pathways/responsibility of care for PUs and DFUs are different. Risk factors for PUs include diabetes and perfusion,[22–24] which should be considered in the formation of PU guidelines.[7,25–27] Pressure is a common factor in the formation of both a PU on the foot and DFU, and both are managed in fundamentally the same way by reducing or redistributing the pressure.[7,28] However, care pathways for PUs and DFUs are different, reflecting the specific characteristics of the wounds and skill sets required. It is critical to understand the patient clearly, to make an accurate diagnosis and to implement the management strategy appropriate to the wound, particularly where overlap in definitions exists.[8] Among nurses caring for DFUs, around 35% may have only minimal knowledge of the diabetic foot.[29] Furthermore, PUs and DFUs on the heel may be diagnosed differently, depending on the specialism of the health professional, leading to inappropriate care, particularly in the community setting.[8,30] In countries such as the US, where payment for care depends on the identity assigned to the wound, the correct diagnosis may make the difference between receiving, or not, certain types of management and products.[31,32] For example, Apligraf for PU treatment is not even mentioned for reimbursement in the US.[33]

Cost of misdiagnosis

Incorrect diagnosis leading to an inappropriate care pathway will to lead to financial and patient-related cost. Management of PUs in all health-care systems is costly,[34–38] and associated with higher mortality.[39–40] Complications in the diabetic foot are among the most serious and costly in patients with diabetes. A third of the total cost of managing diabetes is attributable to DFUs, and these are significantly higher after

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ulceration compared with patients with diabetes and no foot ulcers.[41] If not successfully treated, DFUs often lead to amputations, which involve lengthy stays in hospital.[42] In fact, amputation in the diabetic foot is preceded by a DFU in approximately 80% of cases.[43] The cost of a DFU is high in all health-care systems[34,44,45] and increases with severity. DFUs are widely recognised to have a major impact on patients' quality of life (QoL)[46,47] and impact on the wider family and friends. QoL is also adversely affected by PUs and any misdiagnosis is likely to exacerbate this.

It is clear that the costs of both PUs and DFUs are high and escalate with severity. Ensuring that the correct diagnosis is made and a care pathway, designed by appropriately-qualified and experienced health professionals, is followed, will help control patient-related and health-care-related costs of PUs and DFUs, and provide the greatest probability of success in healing the ulcer and avoiding complications.

This is a working document that addresses general principles and provides guidance designed to minimise the likelihood of misdiagnosis and inappropriate management of PUs and DFUs. It should be read and implemented in conjunction with local guidelines. It brings theory and practice together, and offers areas of reflection that allow the reader to review the information and then decide where and how to use it to underpin their own clinical area. The consensus will inform and enable opportunities for practice change.

Differentiation between DFUs and PUs

Diabetic foot ulcers (DFUs) and pressure ulcers (PUs) have been defined in detail by a number of expert panels, consensus documents and publications.[7,8,48,49] According to the International Working Group on the Diabetic Foot,[49] a DFU is defined as:

'A full-thickness wound below the ankle in a diabetic patient, irrespective of duration. Skin necrosis and gangrene are also included in the current system as ulcers.'

The key elements are the location of the wound and the diagnosis of diabetes. The breadth of this definition means that a PU on the foot in a patient with diabetes is a DFU, as would be any foot wound in a patient with diabetes.[8] A DFU can occur on any part of the foot, including the plantar and dorsal surfaces. A DFU may be neuropathic, ischaemic or a combination of these two factors (known as neuroischaemic), but the three types of DFUs have overlapping pathophysiology.[48]

A PU is defined by the European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Ulcer Advisory Panel (NPUAP), and Pan Pacific Pressure Injury Alliance (PPPIA) as (Box 1):[7]

'A localised injury to the skin and or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear.'

The scope of this definition encompasses skin and tissue damage that results from pressure and/or shear and friction, irrespective of comorbidities. Nevertheless, there is scope for imprecision in the diagnosis and definition of a PU. The EPUAP definition warns us that:

'A number of contributing or confounding factors are also associated with PUs; the significance of these factors is yet to be elucidated.'

This implies that merely diagnosing a wound as a PU does not necessarily fully describe the ulcer and therefore the care that it should receive. The definition of PU also encompasses those that occur at the end of life, related to Skin Changes at Life's End or Kennedy Terminal Ulcers,[49,50] and PUs that are caused by medical devices such as respirator masks, intubation, catheters, splints, casts, and compression bandaging.[8,51]

Key points

- The degree of patient mobility status could be a characteristic that helps differentiate between a DFU and a PU. DFUs tend to be associated with mobility and PUs with immobility
- Neuropathy and peripheral arterial disease (PAD) are the key risk factors for developing a DFU
- The factors that underlie the ulcer are the targets for management and they must be clearly identified to develop an effective care plan
- A critical factor when managing a wound is accurate assessment and diagnosis.

Where heel PUs and DFUs are concerned, there is clear room for overlap in their definitions, if not their precise underlying causes. The consensus panel recognises, in addition to other diagnostic features, that the degree of patient mobility could be a defining characteristic. PUs tend to be associated with immobility; DFUs tend to be associated with mobility. This is not an absolute differentiator. Where a heel PU is related to friction and shear, the patient may have been able to move, resulting in friction. This may be deliberate movement, where the patient tries to reposition themselves, pushing with their heels. However, movement may be passive, where the patient is moved manually by health professionals as part of care. For example, passive friction and shear may be caused by articulating bed frames, used widely in EU hospitals to assist in patient handling, while reducing risk of injury to staff. Involuntary sliding movement of the heel up to 15 or 20cm, which is recognised as a risk for heel injury, occurs when these bed frames are articulated.[52] On the other hand,

mobility/weight bearing is more prominent in the development of a DFU, where repeated friction and pressure on the foot, as the result of patient walking (ambulating), can cause the trauma component of ulceration.

From the viewpoint of management of the wound, and the patient on the appropriate pathway, the critical factor is accurate assessment and diagnosis, rather than the precise terminology used. Guidelines followed to achieve accurate assessment may be expert consensus guidelines, but they should be used in conjunction with local or national guidelines. The name ascribed to the ulcer is a start point; the factors that underlie the ulcer are the targets for management and must be clearly identified to develop an effective care plan.

Box 1. Key definitions

EPUAP, NPUAP, PPPIA guidelines, 2014[7]

Pressure ulcer

A pressure ulcer is a localised injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear. A number of contributing or confounding factors are also associated with pressure ulcers; the significance of these factors is yet to be elucidated

IWGDF guidance, 2015[53]

Diabetic foot

Infection, ulceration or destruction of tissues of the foot associated with neuropathy and/or peripheral artery disease in the lower extremity of people with diabetes

Foot ulcer

Full-thickness lesion of the skin of the foot

Note: these are not comprehensive and the reader should always refer to local guidelines.

EPUAP—European Pressure Ulcer Advisory Panel; NPUAP—National Pressure Ulcer Advisory Panel; PPPIA–Pan Pacific Pressure Injury Alliance; IWGDF— International Working Group on the Diabetic Foot

Causes of PUs and DFUs

The pathophysiology of a DFU is complex and multifactorial (Fig 1). A patient with type 1 or type 2 diabetes may develop a number of underlying comorbidities that lead to an at-risk foot. At this stage, the foot does not have an active DFU, but is at high risk of forming one. Key factors in the risk of development of DFUs include:[43,53]

- Peripheral neuropathy, which reduces the ability to sense touch and pain and causes loss of protective sensation
- Foot deformity as a result of damage to the distal nervous system, which leads to small muscle wasting and muscle atrophy. The deformed foot (sometimes referred to as a Charcot deformity) is subject to increased pressure where bony prominences become more pronounced and the protective fat pads under the heels and metatarsal heads shift, exacerbating the harmful effects of pressure
- Autonomic neuropathy, causing loss of sweating that leads to dry skin and callus formation increases pressure locally, and the likelihood of the skin cracking. Autonomic neuropathy also causes increased peripheral blood flow and distended foot veins and a warm, dry foot. This can appear to be a healthy foot when, in fact, it is at risk
- Peripheral arterial disease (PAD) is present in nearly half of patients with diabetes,[54] leading to reduced blood supply and tissue ischaemia. PAD is more common in type 2 diabetics than in type 1[55]
- A history of previous DFU or amputation.

Older patients who have had diabetes for longer and male patients are at higher risk of DFU formation. When one or more of these underlying causes are overlaid with pressure and trauma from footwear or other sources, skin damage can lead to ulceration.[56] Infection is not regarded as a cause of DFUs, but a consequence of it.[43] Once an at-risk foot has skin damage, without the correct care, the wound can deteriorate rapidly as the tissue becomes hyperinflammatory, leading to the overexpression of

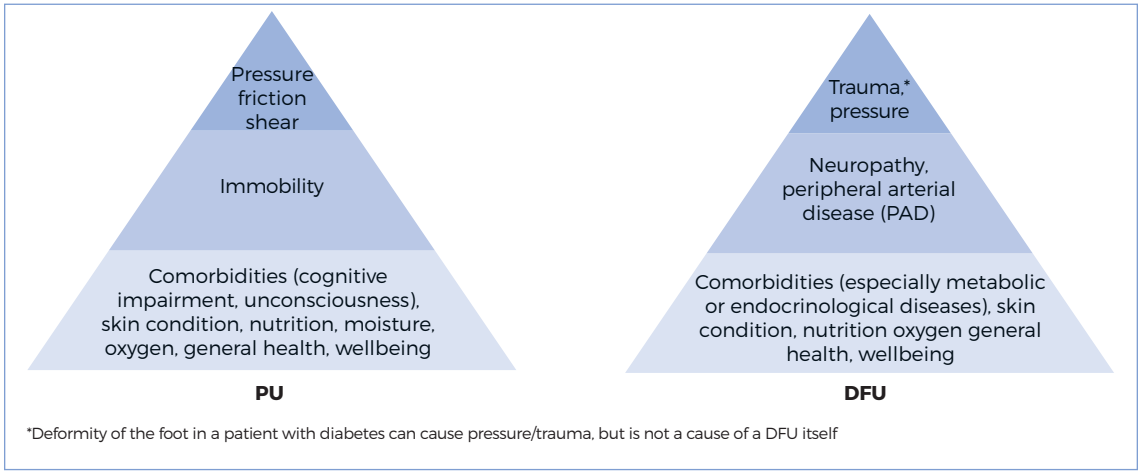


Fig 1. Cause hierarchy of pressure ulcers (PU) and diabetic foot ulcers (DFU)

powerful tissue-destructive proteinases and reactive oxygen species (ROS).[57–59] Amputation in the diabetic foot is preceded by a DFU in approximately 80% of cases.[43]

The pathway to PU formation comprises three well-documented key factors: pressure, friction and shear (Fig 1). A period of immobility is a fourth component. Patients may be bed-bound with comorbidities, elderly with end-stage conditions, immobile from spinal cord injury or during surgery. Moisture alone will not lead to PU formation,[7] but in combination with pressure, and/or friction and shear, it is associated with ulcer formation. Shear is recognised by the NPUAP as a primary cause of PUs.[60] Moisture increases friction between the skin and a surface, such as a bed sheet,[61] which causes tissue deformation when the different layers of skin move tangentially relative to each other as the patient moves. These forces may damage tissue directly[62] or cause injury to superficial skin structures when a patient moves on a bed surface.[63] Friction and shear predict the development of PUs in adult critical care patients.[64] Tissue shear forces may cause cell damage and death more rapidly, over a period of minutes, than pressure alone.[65] Pressure over bony prominences in an immobile patient directly damages deep tissue by compression and restriction of blood flow, leading to tissue death and ulceration. In contrast to shear forces, pressure acts over longer time periods, measured in hours.[66] Pressure over bony prominences may be three to five times higher than other tissues, and this is doubled by shear forces.[67,68] Pressure over bony prominences does not occur in isolation from shear forces and as tissue is deformed by compression, shear forces also form around the deformation. As with DFUs, the physical aetiology of PUs leads to uncontrolled expression of tissue-destructive hyperinflammation that breaks tissue down, resulting in ulceration.[57–59]

Risk factors for the development of heel PUs[69] include: a previous or current heel PU, indicative of reduced tissue tolerance; diabetes and peripheral neuropathy; stroke or cerebrovascular accident (CVA), restricting the patient's ability to move; paralysis; hip fracture and dragging injuries resulting from knee replacements; dementia and cognitive impairment; PAD reducing tolerance to mechanical forces; leg spasms, Parkinson's disease or tremors causing heel rubbing; agitated heels; leg oedema, which may compromise capillary flow and reduce tissue tolerance; and frequent sliding on the bed or chair. Also at risk of developing a PU are patients with diabetes, those undergoing surgical procedures longer than two

hours,[24] those admitted to a nursing home after transfer from hospital compared with transfer from the community,[23] patients at the end of life[22] and those using a medical device.[70–72]

Summary

There are similarities as well as important differences between a PU on the heel and a DFU.[8] The risk and causative factors coincide in several areas, including pressure, shear forces, and peripheral blood supply. Furthermore, heel PUs and DFUs may appear similar on clinical examination and assessment. A difference in causation is immobility/mobility. A patient with diabetes and a heel ulcer may not be recognised as having a DFU; clinically, the ulcer may be confused with a non-diabetic heel PU if the correct assessment is not conducted.

Assessment, referral and the multidisciplinary team

Correct assessment of the patient to identify the ulcer aetiology, independent of the terminology used to describe it, is critical to allocating the correct care pathway. An ulcer on the heel may be described as a PU, but if the patient has diabetes, it must be assessed as a DFU. This ensures that not only is the wound itself treated effectively, but also the underlying causes are clearly identified and managed and the correct guidance is given to the patient and their carer(s)/family. For example, a heel ulcer in a patient with diabetes, if managed as a PU, is highly unlikely to receive the required multidisciplinary team (MDT) approach which is recommenced for a DFU and will be at risk of complications, deterioration and amputation, all of which could have been avoided if the correct care pathway was followed.

Having identified the condition, the next step is referral to the health professional and/or team that is best qualified to manage the patient. The assessment should identify the key clinical and patient characteristics to be managed and indicates the skill sets required to address them. In the case of a DFU, referral to an MDT is the optimal pathway.

When a patient presents with a heel ulcer, the first step should be to exclude the possibility of diabetes and that it is therefore a DFU.[8] If necessary, this step can be taken in the absence of the patient's notes. Where no diagnosis of diabetes has been made, two clinical signs that differentiate between a PU and a DFU should be evaluated:

- Presence of diabetic peripheral neuropathy (DPN), leading to loss of protective sensation
- Reduced arterial blood supply (ischaemia).

Furthermore, mobility/immobility can help differentiate between a DFU and PU. If any of these signs (DPN, ischaemia, mobility) are present, then the patient should be directed to the DFU care pathway for further assessment. If these signs do not suggest that the patient has a DFU, the patient may follow the PU pathway. The following section provides guidance on simple tests for identifying the presence or absence of DPN and reduced blood supply in the patient's feet, and to assess mobility.

Before the ulcer is assessed, the patient history should be taken according to local practice.

Key points

- When a patient presents with a heel ulcer, assess diabetic status—an ulcer on the heel may be described as a PU, but if the patient also has diabetes, the ulcer must be assessed as a DFU
- In order to ensure that the patient is directed to the optimal care pathway, it is necessary to conduct simple tests, such as pulse palpation, toe touch test
- Pulse palpation—if the patient does not have a pulse, refer to a vascular specialist (or relevant health professional) for a full assessment
- Once ulcer aetiology is established, the next step is referral to the health professional and/or team that has the optimal approach to manage the patient

Assessment of diabetic peripheral neuropathy

Several tests are available for assessing the presence and severity of DPN. Diagnosis of DPN is made by determining presence or absence of sensation in the foot. The equipment required to conduct the tests varies between the simple and the highly complex, where access to power supplies is required.

Toe Touch Test: The simplest test, which requires no specialist equipment, is the Toe Touch Test or the Ipswich Touch Test (IpTT).[73,74] The sensitivity (78.3%) and specificity (93.9%) are high. The test is always at

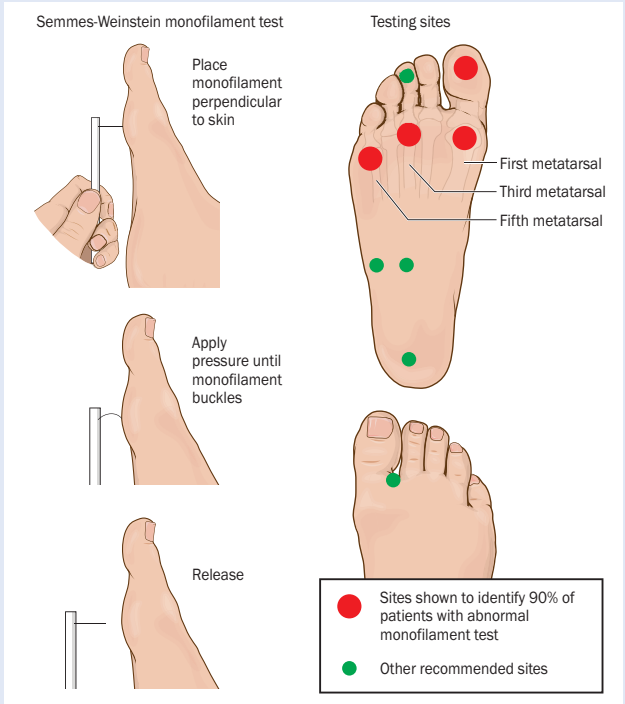
hand, simple to conduct, safe to do, quick and easy to perform, and easily learned. It can be administered effectively by family and non-specialist carers after training.

The test is conducted by lightly touching the tips of the first, third and fifth toes and the dorsum of the hallux of both feet with the index finger, and noting whether or not the patient can feel or sense the touch. It is important that the index finger touch is light, without pushing, prodding, tapping or poking, to avoid the patient feeling the test by sensing movement or force. To ensure the patient is unaware of the point of touch, he or she should be blindfolded or shielded from viewing the test. If the patient cannot feel the touch on two or more sites out of eight, a diagnosis of reduced sensation is made. If the test indicates potential DPN, the patient should be referred for monofilament testing, where available.

Nylon monofilament test: the next simplest test uses a monofilament nylon fibre, the Semmes-Weinstein monofilament, which bends or buckles when subjected to a force of 10g when pressed against a surface.[75] Different versions of the equipment used to conduct this test are available. The simplest is a short moulded plastic handle with the monofilament attached perpendicularly at one end. Other versions comprise a reusable handle with replaceable monofilaments.

The patient is introduced to the sensation by touching an area such as the hand or inside of the wrist. The monofilament is then applied to the tips and metatarsal heads of the first, third, and fifth toes[75] or the tips of the toes and the halluces (Fig 2).[76] The test should be conducted in such a way that the patient cannot see when the monofilament is applied to the skin to ensure fidelity. The monofilament is applied to the skin in a non-rhythmic pattern to rule out the possibility of the patient predicting when the test is being done. The patient should indicate if they can sense the monofilament. If the monofilament cannot be felt on any one site abnormal sensation in the foot has been

Fig 2. Monofilament test



detected. However, sensitivity increases when up to four plantar sites are tested. [75] Each monofilament must be rested for 24 hours after 10 applications[75,77] and replaced when bent or depending on the manufacturer, after 70–90 applications to ensure that the filament has not weakened.[78] It should be noted that different monofilaments perform differently.[77] Those that meet the requirement for buckling at 10g force should be used. In busy clinics, it may be necessary to have more than one monofilament available to account for the need to rest the device. A further test based on the principle of the Semmes-Weinstein hair is the von Frey's hairs test, which enables the practitioner to determine the threshold of touch sensation by using hairs that buckle at different forces.

Vibration perception threshold (VPT): the simplest-to-use vibration-related device for assessing loss of

sensation is a tuning fork with a specific frequency of vibration, 128 Hz. In one version of the test,[79] the tuning fork is set vibrating by striking it on the palm of the hand for 40 seconds. As with the monofilament test, it is then applied to the hand or wrist. The test on the foot is conducted on the dorsal surface of the great toe on the bony prominence just proximal to the nail bed. The patient indicates whether the vibration is sensed and when the vibration has subsided and stopped. The test is repeated on the same foot and then the other foot in a non-predictable sequence.

An alternative to the tuning fork method is a small, battery-powered, hand-held device, the VibraTip.[80] This device has been reviewed by the UK National Health Service body that develops guidance on new medical device technologies, the Medical Technologies Advisory Committee (MTAC), and is recommended for identifying peripheral neuropathy in the diabetic foot.[81] It is used in the same way as the tuning fork.

Other methods to determine diabetic peripheral neuropathy: simple manual and complex electromechanical devices are available to identify DPN.[82] Manual devices include the tactile circumferential discriminator, which detects the ability of the patient to discriminate two points applied close together on the skin, and a test that uses ball bearings of increasing diameters to identify which is the smallest that the patient can feel. A number of electromechanical devices are available to measure VPT. Examples are Biothesiometer, Neurothesiometer, Maxivibrometer, Vibrameter, Vibratron and the CASE IV system.[82] These require access to power and may be unsuitable for use in many locations.

Ankle reflexes: absence of ankle reflexes is associated with an increased risk of foot ulcer formation in patients with diabetes. [83] The test requires a tendon hammer, which is used to strike the Achilles tendon. The health professional performing the test dorsiflexes the foot to put the tendon on stretch before striking with a hammer. Absence of a reflex is abnormal and indicates the need for further assessment.

Vascular status assessment

Several tests are available for assessing the presence and severity of reduced blood supply, which is indicative of possible ischaemia. Initial assessment may be done using simple tests that require no or minimal equipment, or by equipment of increasing complexity and greater discriminatory potential. In order to ensure that the patient is directed to the optimal care pathway, it is necessary to conduct only simple tests. Where vascular issues and reduced blood supply are suspected, the patient should be referred for specialist vascular assessment. Simple tests that require no or minimal equipment include:

Pulse palpation:[84–86] Where other methods of identifying vascular issues and ischaemia are not available, palpation of dorsal pedal pulses allows initial screening and requires no equipment. In this test, the health professional assesses the pulse in the posterior and anterior tibial arteries by palpation. The posterior tibial pulse is palpated just behind the medial malleolus. The anterior tibial pulse should be palpated at the ankle, at the midpoint between the two malleoli, not more distally in the foot, where it lies deeper. The dorsal most prominence of the navicular bone is marked. Pulse palpation is evaluated by using two fingers, the index and middle fingers of the dominant hand. The posterior tibial is felt posterior to the medial malleolus of the tibia. For the dorsalis pedis, feel on the dorsum of the foot, lateral to the extensor tendon of the great toe.

Note: a diabetic foot with neuropathy and no ischaemia may present with a warm limb and bounding pulses.[83] In this case, do not rely only on pulse palpation for differentiating between a PU and a DFU, but use all assessment outcomes as a set to inform the decision. Furthermore, PAD can still be present, despite the presence of a palpable pulse.

Ankle-brachial pressure index (ABPI): ABPI involves the ratio of systolic pressures in the brachial artery at each elbow and systolic pressures in the posterior tibial and dorsalis pedis arteries at each

ankle. ABPI is calculated for each leg separately. ABPI is conducted with the patient in the supine position (lying down). Evidence states 10 minutes of supine rest as a minimum before pressure measurement is recommended, to allow equaling of the vascular beds which determine arterial pressure.[87,88] The sphygmomanometer cuff is placed around the ankle above the malleolus. The location may vary slightly from anywhere from just above the malleolus to 2.5cm above the malleolus, depending on which guidelines are followed. Where the ABPI is recorded ≤0.9,[89] the patient should be referred for further specialist vascular assessment, using more sensitive methods.

Patients with diabetes may have hardening of the arteries and medial arterial calcification (MAC) in the lower leg and foot, which reduces the compressibility of the arteries. The presence of MAC is known to reduce the compressibility of the vessel and can lead to false elevation of the ABPI. This makes ABPI interpretation in diabetes populations difficult. Health professionals should be aware that the ABPI should not be used as a stand-alone screening tool in diabetic populations, but in conjunction with other testing methods. Health professionals should consider using other non-invasive, vascular tools, such as hand-held Doppler auscultation alongside ABPI to aid accurate identification of PAD. Where the ABPI is measured as ≥1.3, further tests, such as a toe-brachial index (TBI), should be performed and if this is not possible, the patient should be referred for vascular assessment.

Note: diabetes involves the medium lumen and therefore the ABPI might not be accurate and a TBI is better.

Toe-brachial Index (TBI): TBI represents an alternative diagnostic tool in patients with diabetes and PAD. Digital arteries are usually less affected by calcifications, which provides insight into the microvascularity of the smaller vessels of the foot. TBI is obtained by dividing the toe systolic pressure by brachial systolic pressure. Since toe pressures are generally about 60% that of brachial pressures, prognosis is relatively good when toe systolic pressure is >50mmHg.[90] TBI>0.7 is considered within normal limits, TBI≤0.7 is an indication of PAD and TBI≥1 was an indictaion of distal arteries calcification.

Doppler ultrasound: assessing the sound waves from a hand-held Doppler can provide information about the condition of the arteries and blood flow. The Doppler machine provides an audible sound or visual tracing, which is created from the movement of blood in the vessel. PAD can change the sound and shape of the waveforms. A triphasic sound/waveform indicates healthy arterial flow. The third sound within the triphasic wave form comes from the dichrotic notch, which is formed from the elastic recoil within the artery. As the elasticity within the artery reduces, this can affect the production of this sound/wave; this represents a biphasic tone, which is an indication of arterial hardening, but not occlusive PAD. A monophasic wave form formed though a signal sound/wave is indicative of the presence of PAD.

The first test should be pulse palpation. Furthermore, ABPI could be false in patients with arterial calcification. If the patient does not have a pulse and has ulceration, refer, where possible, to a vascular specialist (or relevant health professional) for a full assessment.

Patient assessment

In most cases, the health professional who conducts the initial assessment of a patient with a heel ulcer is the 'wound care navigator' (WCN).[91] Referral to the WCN may have been made by a general practitioner or other primary care practitioner, a nurse, or the patient may have self-referred. The wound skill level of the WCN with respect to wound management may be high—as with a podiatrist, wound, ostomy and continence nurses (WOCN) in the US, tissue viability nurse (TVN) in the UK, TVN or advanced nurse practitioner in Ireland or a nurse with advanced wound care knowledge in other parts of Europe.

Table 1. VIPS foot assessment

V—vascular/ ischaemia	Pulse palpation and if possible ankle-brachial pressure index (ABPI)
I—infection/ biofilm/ inflammation	Visual signs, redness, swelling, slough, smell, reported pain
P—pressure	Is it caused by mobility (*likely diabetic foot ulcer*) or immobility (*likely pressure ulcer*)?
S—sensation (neuropathy)	Touch the toes and, if possible, monofilament test

The extent of patient management undertaken by the WCN should be in line with their skill level, with referral further through the health-care system, according to the patient's clinical needs. Minimally, the WCN should be trained to conduct the initial steps required to assess the patient and to conduct the tests required, based on the ulcer characteristics. Local or national guidelines should be consulted to ensure that optimal care is delivered. In general, the steps are:

1 Record patient history: including: patient characteristics, such as age and sex, relevant medical history, current medications and previous ulceration or amputation. The health professional should specifically ask about diabetes; the patient may disclose that they have diabetes or may not know, if it is undiagnosed. A family history of diabetes, especially type 2, is important. Record the duration and type of diabetes if it is known. Record the lower limb condition—hairs, temperature, colour, skin conditions, such as hyperceratosis. Also, note how the ulcer is being managed at the point of presentation, for example, whether the patient is using offloading of any sort.

2 Assess the wound characteristics: including location (plantar, heel, metatarsal head(s), instep, dorsal, lateral), size, depth, including presence of underlying function, edge and periwound appearance, exudate type, visual appearance, pain, presence of infection and surrounding cellulitis and redness. Skin condition (whether it is dry, atrophic, and/or there are fissures/ cracks) and temperature (a dramatic drop in skin temperature from proximal to distal along the lower limb) can be a sign of poor blood flow. Assess the foot for callus and deformity, which increase local pressure, for example, hammer toes, prominent metatarsal heads and Charcot deformity. Amputation should also be recorded. Assess between the nails and toes for signs of fungal infection. PUs on the foot are usually located on the heel.

When assessing a wound, the acronym MEASURE may be useful.[92] The acronym stands for:

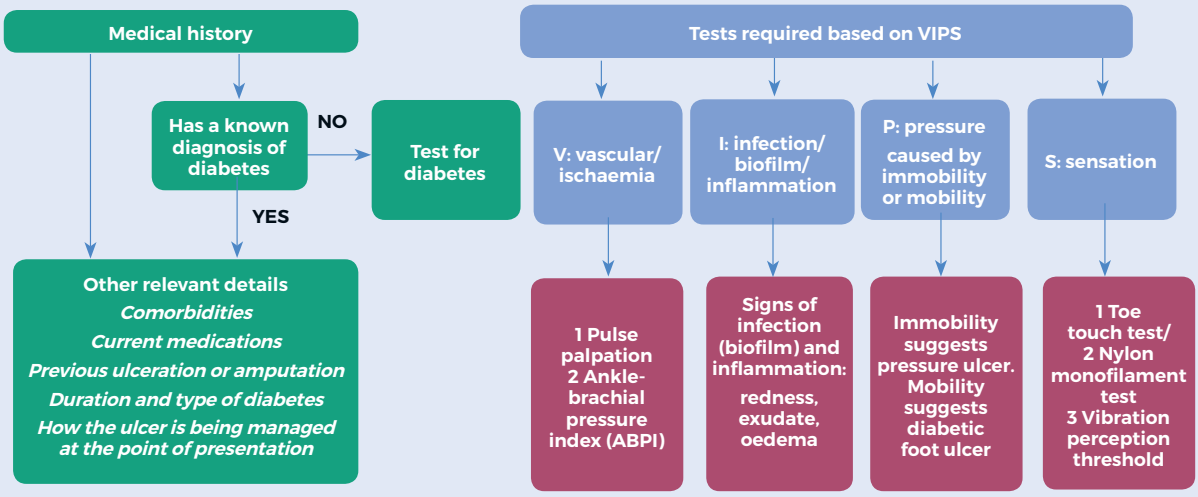
M: measure size
E: exudate amount (none, scant, moderate, heavy) and characteristics (serous, sanguinous, pustular, or combinations)
A: appearance, necrotic (black), fibrin (firm yellow), slough (soft yellow), or granulation tissue (pink and healthy versus red and friable—easy bleeding, unhealthy)
S: suffering pain
U: undermining, measured in centimetres and position in the ulcer recorded
R: re-evaluate
E: edge (hyperkeratotic, macerated, normal).

3 Identify the degree of patient mobility: if the patient is bedbound or relatively immobile, it is likely that the ulcer is a PU, whereas if a patient is reasonably mobile, it is more likely that the ulcer is a DFU. Attention should be made to the activity and mobility elements of the risk assessment tool used in the specific practice setting.

4 Assess DPN and/or blood supply using the test best suited to the equipment and skills available: this may be a simple test requiring no equipment, such as pulse palpation and the Toe Touch Test.

5 Refer the patient to the appropriate care pathway based on the overall outcomes of the assessment: these pathways should be described by local guidelines. Note: if the patient identifies as diabetic, this may be

Fig 3. Assessments required to help determine the origin of the ulcer and relevant treatment pathway



enough to lead to a referral; however, a full assessment will aid with referral urgency.

The consensus panel decided that a useful guide to the key aspects required for the assessment of an ulcer on the foot of a diabetic patient is the mnemonic 'VIPS' (Table 1, Fig 3):

- V: vascular/ischaemia
- I: infection/biofilm/inflammation
- P: pressure
- S: sensation/neuropathy.

Grading systems

The effective care of the ulcer depends on clear and accurate diagnosis and description of the condition. Where the skill level is appropriate, management may be conducted by the WCN, or the patient may be referred to an appropriate health professional/service.

Many grading systems for PUs and DFUs have been published by expert groups or institutes. The health-care provider may have developed a local grading system, which should be used if available. Where a local or national grading system is not available, a grading system developed by expert consensus or other developer should be used. Grading systems assume a level of skill to recognise and differentiate the scoring parameters and must be administered by appropriately-qualified staff.

PU grading systems

The most widely used grading system for PUs is that prepared by the NPUAP, EPUAP and PPPIA.[7] The term 'pressure ulcer' has recently been subject to review. The NPUAP in the US has proposed adoption of a new term, pressure injury (PI). This document continues to use the term pressure ulcer. The NPUAP, EPUAP and PPPIA grading system is based on the depth of the PU and the extent of tissue involvement, and assigns a PU to 'categories' or 'stages' as shown in Box 2.

DFU grading systems

A number of grading systems for DFUs exist. The most commonly-used systems are SINBAD, Wagner, University of Texas, Wound Ischaemia and Foot Infection (WIfI) and PEDIS. In general, the grading of

DFUs is based on the size of the ulcer and the presence or absence of DPN, PAD and infection, although the detail of how this is achieved by each system varies. Where they exist, clinicians should use local grading systems. Where no local system is available, one of the existing systems should be adopted, according to local preference.

SINBAD[93] is an acronym for: site; ischaemia; neuropathy; bacterial infection; area; and depth. Each parameter is allocated a score of either '0' or '1' according to the system shown in Box 3 and the total score for the DFU is calculated. Higher scores indicate greater severity.

University of Texas: assesses the DFU on two parameters and provides an alphanumeric score that is a combination of the two, as shown in Box 3.

Box 2. Pressure ulcer grading system, NPUAP, EPUAP and PPPIA[7]

	Definition	Appearance
Category/stage I	Non-blanchable erythema: intact skin with non-blanchable, redness of a localised area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its colour may differ from the surrounding area	The area may be painful, firm, soft, warmer or cooler, as compared with adjacent tissue. Category/stage I may be difficult to detect in individuals with dark skin tones. May indicate 'at risk' individuals (a heralding sign of risk)
Category/stage II	Partial-thickness skin loss: partial thickness loss of dermis presenting as a shallow open ulcer with a red-pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister	Presents as a shiny or dry shallow ulcer without slough or bruising.* This category/stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration or excoriation. *Bruising indicates suspected deep tissue injury
Category/stage III:	Full-thickness skin loss: full-thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon or muscle are not exposed. Slough may be present, but does not obscure the depth of tissue loss. May include undermining and tunnelling	The depth varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and category/stage III PUs can be shallow. In contrast, areas of significant adiposity can develop extremely deep PUs. Bone/tendon is not visible or directly palpable
Category/stage IV	Full-thickness tissue loss: full-thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunnelling	The depth varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and these ulcers can be shallow. Can extend into muscle and/or supporting structures (e.g. fascia, tendon or joint capsule) making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable
Unstageable	Depth unknown: full-thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, grey, green or brown) and/or eschar (tan, brown or black) in the wound bed	Until enough slough and/or eschar is removed to expose the base, the true depth, and category/stage, cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as 'the body's natural (biological) cover' and should not be removed
Suspected deep tissue injury (DTI)	Depth unknown: purple or maroon localised area of discoloured intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler, as compared with adjacent tissue	May be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment

NPUAP—National Pressure Ulcer Advisory Panel; EPUAP— European Pressure Ulcer Advisory Panel; PPPIA—Pan Pacific Pressure Injury Alliance

Box 3. Summary of diabetic foot ulcer (DFU) grading systems

SINBAD

Category	Definition	Score	Category	Definition	Score
Site	Forefoot	0	Bacterial infection	None	0
	Midfoot or hind foot	1		Present	1
Ischaemia	Pedal blood flow intact: at least one pulse palpable	0	Area	Ulcer <1cm^2	0
	Clinical evidence of reduced pedal blood flow	1		Ulcer ≥1cm^2	1
Neuropathy	Protective sensation intact	0	Depth	Confined to skin and subcutaneous tissue	0
	Protective sensation lost	1		Reaching muscle, tendon or deeper	1
TOTAL POSSIBLE SCORE					**6**

University of Texas

Grades	Description	Stage	Description
0	Pre- or post-ulcerative or healed wound	A	No infection or ischaemia
1	Superficial wound not involving tendon, capsule or bone	B	Infection present
2	Wound penetrating to tendon or capsule	C	Ischaemia present
3	Wound penetrating to bone or joint	D	Infection and ischaemia present

Wagner

Grade	Description
0	Intact skin
1	Superficial ulcer of skin or subcutaneous tissue
2	Ulcers extend into tendon, bone, or capsule
3	Deep ulcer with osteomyelitis, or abscess
4	Gangrene of toes or forefoot
5	Midfoot or hindfoot gangrene

Wagner[94,95] uses six definitions that incrementally describe a DFU by the degree of severity (Box 3).

Wound Ischaemia and Foot Infection (WIfI):[96] developed to assess patients with critical limb ischaemia. WIfI assesses the wound, ischaemia, and foot infection and assigns a score to the ulcer. The WIfI system correlates well with outcomes for wound healing and amputation.[97] The scoring system is shown in Box 4.

Box 4: Description of the WIfI grading system, which assesses the wound and the presence of ischaemia and infection

WIfI Wound (W)

Grade	Ulcer	Gangrene
0	No ulcer, ischaemic rest pain	No
1	Small shallow ulcer on distal leg or foot, no exposed bone, unless limited to distal phalanx. Salvageable with simple digital amputation	No
2	Deeper ulcer with exposed bone, joint or tendon; generally not involving the heel; shallow heel ulcer, without calcaneal involvement. major tissue loss salvageable with multiple (≥3) digital amputations or standard transmetatarsal amputation±skin coverage	Limited to digits
3	Extensive, deep ulcer involving forefoot and/or midfoot; deep, full-thickness heel ulcer±calcaneal involvement. Extensive tissue loss salvageable only with a complex foot reconstruction or non-traditional TMA (Chopart or Lisfranc); flap coverage or complex wound management needed for large soft tissue defect	Extensive gangrene involving forefoot and/or midfoot; full-thickness heel necrosis, calcaneal involvement

WIFU Ischaemia (I)

Grade	ABI	Ankle systolic pressure	TP, $TcPO_2$
0	≥0.80	>100mm Hg	≥60mmHg
1	0.6–0.79	70–100mmHg	40–59mmHg
2	0.4–0.59	50–70mmHg	30–39mmHg
3	≤0.39	<50mmHg	<30mmHg

WIFI Infection grade (FI—foot infection)

Grade	Symptoms
0	No symptoms or signs of infection
1	Local infection involving only skin, subcutaneous (SQ) tissue
2	Local infection with erythema >2cm, or involving structures deeper than skin, SQ (eg, abscess, osteomyelitis)
3	Local infection with signs of systemic inflammatory response syndrome SIRS

PEDIS: developed by the IWGDF to use strict criteria that are applicable worldwide.[98] It was created primarily for use in research and, as such, is unlikely to be used widely in the management of DFU, outside research.[98]

Risk assessment

Risk assessment estimates the level of risk that a patient will develop a new ulcer or a recurrent ulcer and, in the case of DFU, progress to amputation. It is recommended that risk assessment be conducted for all patients who currently do not have an ulcer, or who have a healed ulcer, in order to identify where prevention strategies should be focused. Where local guidelines are available for conducting risk assessment, these should be used. Where local or national guidelines are not available, there are a number of risk assessment tools or instruments that may be used.

Table 2. International Working Group on the Diabetic Foot (IWGDF) guidance on attendance at foot protection services, based on risk category

Risk Category	Characteristics	Frequency
0	No diabetic peripheral neuropathy (DPN)	Once a year
1	DPN	Once every 6 months
2	DPN with peripheral artery disease and/or a foot deformity	Once every 3–6 months
3	DPN and a history of foot ulcer or lower-extremity amputation	Once every 1–3 months

Pressure ulcer risk assessment

Many risk assessment tools have been developed for PU. Examples include the Braden Scale and Waterlow.[99] PU risk assessment should be a combination of a structured assessment based on a tool and clinical judgement.[7] Good clinical judgement requires experience in risk assessment; if there is any doubt, at least one other person should carry out the assessment. Risk assessment should be conducted and documented as soon as possible after a patient is referred or presents, and no later than six hours after arrival. It should be repeated, especially if there is a change in the patient's condition, or for PUs. PUs in hospital inpatients should be assessed daily, if possible.[100]

Factors reported to increase the risk of PU formation include poor skin condition, an existing PU, immobility, poor nutritional status, higher or lower than average BMI, female sex, greater age, incontinence and increased skin moisture, comorbidities such as cachexia and organ failure, PAD, anaemia, motor and sensory impairment, spinal injury, and diabetes. Most risk assessment tools are based on these risk factors and assign a score to the patient which identifies the risk category for PU formation in the patient. A risk factor for heel PU in particular is a degree of mobility that allows the patient to move themselves, for example, on a bed, or the presence of leg spasms, Parkinson's disease or tremors causing heel rubbing, agitated heels, and frequent rubbing by sliding on a bed or chair. Articulated bed frames can also increase risk of heel PU.

Skin inspection is a critical step in PU prevention and should be performed regularly. The skin should be inspected within six hours of admission to a hospital and daily thereafter. All skin sites susceptible to PU formation should be assessed for pain or discomfort reported by the patient and the skin should be checked for:

- Skin integrity in areas of pressure
- Colour changes or discolouration. Non-blanchable erythema may present as colour changes or discolouration, particularly in darker skin tones or types
- Variations in heat, firmness and moisture (for example, because of incontinence, oedema, dry or inflamed skin).

Use finger palpation to determine whether erythema or discolouration (identified by skin assessment) is blanchable. A simple test to assess redness is to place a transparent plastic disc over the skin as it is depressed. Blanchable redness is identified by the skin losing redness which returns when the pressure is released and blood reperfusion occurs. Non-blanchable redness does not lose its red colouration and indicates development of inflammation in the skin. Where available, diascopy may be used to evaluate skin.[7]

Developments in skin assessment include new methods to assess pathological change in at-risk skin. Where skin changes that may lead to PU formation have started, the tissue becomes inflamed. Early inflammatory changes include extravascular fluid accumulation in the matrix of skin which are not visible to the naked eye. This fluid is called sub-epidermal moisture (SEM). The assessment method measures an electrical property of skin—impedance—which changes with SEM.[101,102] A device to measure SEM is commercially available and can detect potentially damaging skin changes up to five days before they are visible to the eye. Where available, the use of this device should be considered.

DFU risk assessment

All patients diagnosed with diabetes who develop peripheral neuropathy are at risk of DFU formation and should be managed according to local, national or international guidelines. The IWGDF has issued guidance on prevention of DFU based on assessing risk posed by DPN, foot deformity, PAD and history of foot ulceration. The associated screening frequency is recommended (Table 2). Risk assessment for progression of ulceration to amputation is covered by the WIfI assessment tool.

Every patient with diabetes and an ulcer should have a health professional perform a simple assessment in order to determine if a vascular assessment is required. For example, if a nurse was able to feel a pulse palpation, that should be enough to rule out the possibility of an ischaemic condition. However, if the wound fails to heal or there is any doubt, the patient should be referred for further non-invasive testing e.g. TBI and ABPI. Boxes 5 and 6 are examples of case study assessments of patients with diabetes and foot ulceration.

Referral

Following assessment and identification of the most likely type of ulcer, the patient should be referred as early as possible to the appropriate care pathway. It is important that the correct clinical procedures and competencies are brought to bear on the wound. Often, those will not necessarily reside in a specific health professional. The pathway that provides the optimal care, irrespective of the person who delivers the care, should be followed.

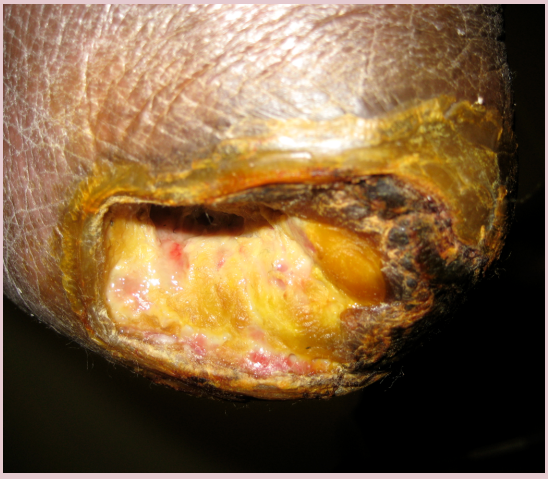
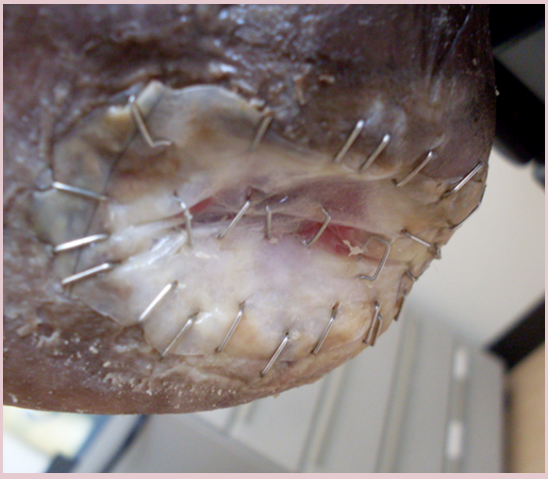
Procedures and competencies potentially required for a PU are infection management, nutrition management, debridement, pressure relief, friction and shear management, medicines, and surgery.

In the case of a DFU, the guidelines recommend referral to an MDT in order to manage the complexity of a patient with diabetes and a wound. Patients managed by an MDT have better outcomes than those not managed in this way.[91] Guidelines recommend that referral should take place promptly and within 24 hours of identification of a DFU.[28] Evidence suggests that this rapid referral does not happen in the majority

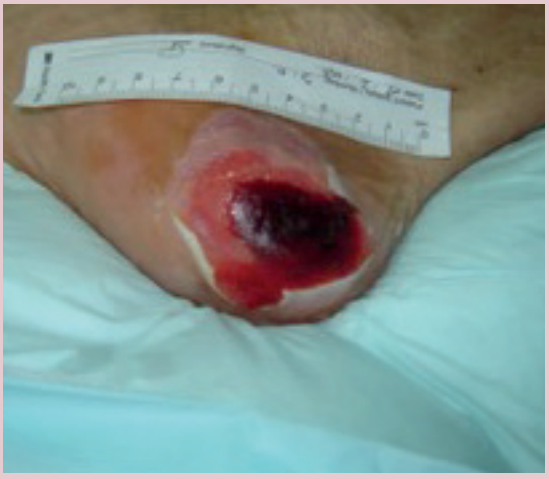
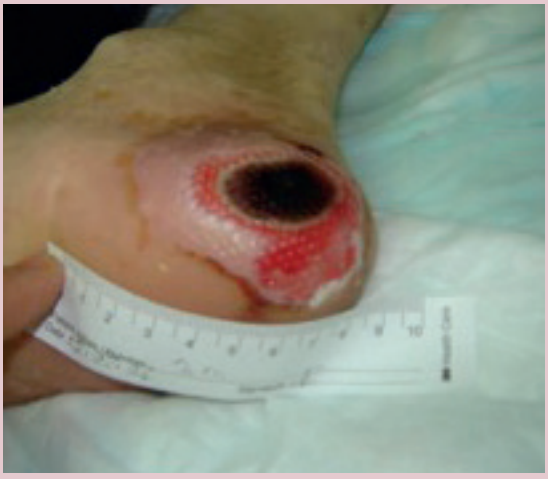
Table 3. Key assessment criteria for foot ulcers

Assessment step	Most likely to be DFU	Most likely to be PU
History	Patient self-identifies as diabetic	Patient self-identifies as not diabetic
Mobility	High/moderate mobility	Low mobility
Peripheral neuropathy	Present	Absent
Reduced blood supply	Present	Absent
Foot deformity	Present	Absent
Previous DFU or amputation	Present	Absent
Pain assessment	None	Pain reported

Box 5. Example of assessment and treatment for a diabetic foot ulcer

Diabetic foot ulcer at initial presentation	After acellular dermal matrix placement
	
Medical history; *Age/gender*: 55 y/o male; *HPI*: wound started as a blister and gradually got bigger, patient hoped the wound would heal on its own; *Wound duration*: 8 months; *Previous ulceration/amputation*: No; *Pain to the area*: No (VAS Scale 0/10); Current wound management: self management with over-the-counter dressings from pharmacy; *Previous medical history*: type 2 diabetes (12 years), hypertension, hypercholesterolemia; *Allergies*: PCN, codeine; *Medicines*: lisinopril, atorvastatin, novolog, fuorsemide, lantis: *Glycaemic control*: $HgA1_c$ 8.6 (6 weeks prior); *Ambulatory/mobility status*: able to ambulate with cane assistance, and to change and control his body position	***Physical Exam Wound (VIPS)*** *Location:* left lateral heel; *Size:* 5.2cm x 4.3cm x 0.5cm *Base:* 90% fibrotic, 10% granular; *Margins:* hyperkeratotic, 40% eschar noted; *Tracking*: none; *Probing:* to periosteum; *Undermining*: 0.8cm along dorsal border; *Odour*: no *Exudate:* mild serosanginous exudate on dressing. No active drainage from wound **Vascular** *DP/PT pulses:* non palpable; *DP pulse biphasic*: PT pulse monophsic via Doppler; *ABPI:* 0.9; $TCPO_2$: 42mmHg **Infection:** No periwound oedema or erythema. =No purulence. No active drainage. No fluctuance. No odour. No slough. Wound deep and probes to periosteum. Possible bone infection **Pressure** Primarily from shoe gear; patient's foot measured as size 11, but wearing size 10 shoes **Sensation** *SWM* 0/10; *vibratory sensation:* diminished; *Toe Touch Test:* 2/8
Tests/Referrals **Vascular** Given that patient's pulses were non palpable and ABPI was 0.9, vascular was consulted, who ordered a transcutaneous oxygen pressure to assess wound's potential for healing; $TCPO_2$ was 42mmHg, indicating good potential to heal without need for vascular surgery. Radiographs or MRI (since the wound probed to periosteum. Radiographs and MRI ordered to rule out osteomyelitis). Both radiographs and MRI were negative for osteomyelitis; Nutritional consult; Endocrinology consult; Pedorthic consult	***Staging and Treatment*** **Staging for DFU:** Wagner's Grade 2 ulcer; UTSA – Grade 3A; WIfI – stage 2 Given that patient had no contraindications to healing, wound surgically debrided and acellular dermal matrix applied. Patient was offloaded with an instant total contact cast with extra padding around the heel Patient healed in 10 weeks and progressed on to a well-fitted diabetic shoe with custom diabetic inserts.

Box 6. Example of assessment and treatment for a pressure ulcer

Pressure ulcer at initial presentation	After two weeks' treatment
	
Medical history; *Age/gender:* 75 y/o male; *HPI:* began in a flictena, with blood content that broke. Caregiver had hoped that the blood would be absorbed and the skin healed; *Wound duration:* 2 weeks; *Previous ulceration/ amputation:* No; *Pain to the area:* yes (VAS Scale 3/10); *Current wound management:* pads wrapping the heel to protect; *Previous medical history:* type 2 diabetes (2 years); Alzheimer's disease, hypertension; Fall off the bed having been transported to the hospital where he was diagnosed cranioencephalic trauma and performed drainage of subdural haemorrhage *Allergies:* No; *Medicines:* memantine, furosemide, melperone hydrochloride; *Glycaemic control:* HgA1C 6.5 (8 weeks prior); *Ambulatory/mobility status:* partially dependent on daily living activities; agitation, difficulties to control his body position and does not comply with the indications for repositioning	***Physical exam wound (VIPS)*** *Location:* right lateral heel; *Size:* 5.4 cm x 4.8cm *Base:* 65% necrotic, 5% fibrotic, 20% granular, 10% epithelial; *Margins:* macerated; *Tracking:* none; *Probing:* No; *Undermining:* No; *Odour:* no; *Exudate*: mild serosanginous exudate on dressing; *DP/PT pulses:* palpable; *DP pulse biphasic:* *PT pulse;* biphasic via doppler; *ABPI:* 10; *$TCPO_2$:* 60mmHg; *Infection:* No periwound oedema / erythema. No purulence. No active drainage. No fluctuance. No odour. *Pressure:* Agitated for some periods, but most of the time immobile. Does not collaborate on repositioning SWM 8/10; Vibratory sensation: normal; *Toe Touch Test:* 6/8
Tests/referrals ***Vascular*** Palpable pedis and tibial pulses. No oedema in the limb and full pulse Normal skin temperature on the feet, no colour alterations. ABPI was 1,0.	***Staging and treatment*** *Staging for PU:* unstageable pressure ulcer – dark eschar; *During hospitalisation:* ECG = 13 (O4 + V3 + M6); fed orally from conventional soft hospital diet, dysphagia to liquids, does not always ingest the whole meal; *Dependent during hospitalisation, raise to highchair; during the day; Score 11 on the Braden scale – High Risk* *Treatment:* Specific heel silicone multi-layered foam dressing and fluidised positioners to raise calcaneous from bed and prevent foot drop. Biological separation of the necrotic tissue and the granulation tissue, with favorable evolution in 14 days. He was discharged to the home after 20 days of hospitalisation. Lesion healed after 3 months.

of cases.[103–105] Correcting referral intervals is likely to be a major contributor to shortening the time-to-heal for a DFU and avoiding progression to amputation. Procedures and competencies in an MDT for a DFU are wound care, infection management, nutrition management, debridement, pressure relief, friction and shear management, and surgery. Table 3 shows the key assessment factors for PUs and DFUs.

Emergency referral

Emergency referral is required when the patient suffers from a severe infection, according to Infectious Diseases Society of America (IDSA) guidelines and there is a high risk to the patient's life. This is especially important where there is deep soft-tissue infection, necrotising soft-tissue infection, acute limb ischaemia and osteomyelitis with systemic signs (fever, tachycardia, tachypnea, leucocytosis. etc).

The multidisciplinary team

An MDT, which may also be known as an interdisciplinary team, is a group of specialists with all the skill sets appropriate to the management of a specified condition. An example is a surgical team, comprising theatre staff, nursing, anaesthesiology, surgeons, ICU practitioners and so on. The critical point is that, whatever structure is in place, the patient should receive the best multidisciplinary care for the wound. A characteristic of an MDT is effective communication to ensure delivery of integrated care to the patient. Patients managed by an MDT tend to have better outcomes than those not managed by one.[91] The constitution of an MDT varies worldwide[91,106] and generally they are associated with the acute care setting rather than the community. Nevertheless, a patient in the community who meets the guidelines for management by an MDT, perhaps because of a change in status of the wound, should be referred to one. In the case of DFU, the IWGDF recommends an MDT should have three levels of the following structure and skills:

- Level 1: general practitioner, podiatry, diabetic nurse
- Level 2: diabetologist, surgeon (general, orthopaedic or foot), vascular specialist, endovascular interventionist, podiatrist and diabetic nurse, in collaboration with a shoe-maker, orthotist or prosthetist
- Level 3: a level 2 foot centre that specialises in diabetic foot care, with multiple experts from several disciplines, each specialised in this area working together, that acts as a tertiary reference centre.

The ASEAN guidelines recommend the following competencies in a DFU MDT: surgery for diabetic foot problems; diabetology; diabetes nursing; podiatry; tissue viability or wound management; specialist competencies including vascular surgery, radiology, clinical microbiology, nephrology and cardiology.

Many DFUs in Europe are overseen by podiatrists who make the clinical decision to refer the patient to the full MDT. Some countries stipulate that patients are managed by physicians who make the decisions on the care plan and referrals. MDTs with responsibility for the management of any chronic or acute wound are being set up in Malaysia.

To treat patients with a diabetic foot ulcer successfully, quality parameters of the facility's structure, treatment procedures and the patient outcome are needed. Structural quality is based on the qualifications of staff and the facility's spatial conditions, as well as a minimum of equipment. The application of available guidelines and documentation systems, as well as the establishment of a team approach between the facility's staff and other experts involved (vascular specialist or surgeon, orthopaedic surgeon, radiologist, podiatrist, orthopaedic shoemaker etc.), are the requirements of procedural quality. Outcome quality includes: wound healing rate and time, rate of amputation (major and minor), vascular intervention (bypass surgery, percutaneous transluminal angioplasty), death rate, clinical admission. In Germany, a certification system was established in 2005; there is a clear link and rules for responsibility, from general practitioner (GP) to diabetologist and

finally specialised centres. Centres are available in all regions of Germany. A similar system was established in Belgium.

Competencies required may also include: infection control, infection management and microbiology, wound care, total contact casting (TCC), physiotherapy, occupational therapy, nutrition and patient education. Some countries may not have practitioners with a functional title such as podiatrist. However, the functional name is less important than the availability of the skills of a podiatrist. In many European countries, the US and Australia, podiatrists are the main practitioners who manage patients with DFU daily and who refer to the MDT, but this is not always the case. The key competencies of a podiatrist in an MDT include:

- Vascular and neuropathy assessment
- Identifying foot deformities and joint mobility range
- Foot care (removal of calluses, nail care)
- Diagnosis and management of infection through prescription of antibiotics and surgical intervention, especially for osteomyelitis
- Prophylactic and conservative surgery in some countries for the correction of the deformity
- Off-loading
- Prevention of the recurrence or re-ulceration through insoles and therapeutic shoes
- Surgical and sharp debridement.

PUs are most often managed by nursing staff, who refer to the appropriate clinical staff as required, but this is not usually under the auspices of a formal MDT. In the absence of a formal MDT, PUs are managed by an interdisciplinary group of practitioners. This panel recommends considering creating an MDT for managing PUs. An example of the membership is provided by the AHRQ.[18]

Prevention, management and treatment strategies

The key to prevention of both PUs and DFUs is early identification of at-risk patients and prompt implementation of effective targeted prevention strategies. Prevention is targeted at the risk factors and underlying conditions that make ulceration more likely. These strategies are the same for adults and neonates, although some skin sites are more susceptible in neonates, for example, the occipital area. It is important to note, there is no one-size-fits all solution for either PU or DFU prevention; both must be tailored to the individual patient.

Key points

- PU prevention includes: pressure reduction/redistribution; friction and shear reduction; skin care; and nutrition
- DFU prevention includes; pressure redistribution; prescribing appropriate footware; nail care; emollient use
- Managing the underlying cause of the ulcer is key to treatment
- PU or DFU prevention: both must be tailored to the individual patient
- Ulcers should be monitored at least once a week to assess progress

Pressure ulcer prevention

The US AHRQ has published a detailed tool kit that guides health professionals in PU prevention.[18] Where prevention strategies are not already implemented, or existing strategies are under review, it is recommended that the tool kit is consulted. All patients are potentially at risk of developing a PU. The purpose of a risk analysis is to identify those at highest risk and where early skin changes have taken place, and to target preventative interventions to them. The risk analysis should be conducted as soon as possible, and for inpatients no later than six hours after admission.[107] The risk analysis will identify risk of PU formation and any areas of ulceration that already exist.

The starting point is care standards, as laid out in guidelines. The most widely-used are those of the EPUAP, NPUAP and PPPIA.[7] Others include those from the National Institute for Health and Care Excellence (NICE) in the UK.[107] The UK NHS suggests following a five-step process for prevention and treatment of PU, known as the SSKIN Bundle,[108] which follows the main principles of PU prevention and treatment. The acronym refers to: *surface* that the patient is on, *skin inspection* conducted early, *keep the patient moving*, *incontinence* and moisture management to keep the patient clean and dry, and *nutrition* (diet and fluids).

Pressure reduction, redistribution and removal

For individuals at risk of a PU due to activity and mobility problems, there are pressure redistribution options available, namely, continuous low pressure devices, such as high-specification foam, and high-tech surfaces (low air loss, alternating or air fluidised). Selection of the surface should be based on an assessment of the individual's mobility status and general skin condition. If these surfaces are not available, the frequency of repositioning should be considered, as this will need to be increased to protect the individual from the adverse effects of pressure and shear forces.

Mattresses may be augmented by additional pressure-relieving and redistribution foam pads. Pressure-reduction and redistribution may be targeted at a specific at-risk anatomy, for example the heel, byproducts that protect the heel in a pressure redistributing boot. Several such products are available, including the Heelift Suspension Boot (DM Systems, UK; Position Health, US), Devon Boot and Heel Protector (Aria Medical), HeelMedix (Medline Industries), Repose Foot Protector (Frontier Medical), Mölnlycke DAP-600Z Fluidised Heel Protector Boot (Mölnlycke Health Care). Patients who are lying in a position where there may be compression of the common peroneal nerve (i.e. lower leg leaning against rails by the side of the bed, or against a wall or even the hand control panel) are prone to developing nerve

palsy and foot drop. While the protective boot may help keep the limb in a more neutral position, not all facilities/regions have protective boots available. Hence, health professionals should be aware of the possibility of developing foot drop and be on alert, noting the patient's position to prevent the development of nerve palsy.

Pressure between the legs may be managed using products that fit between the legs and keep them separated, for example Devon Utility Pad (Aria Medical). If pillows are used to manage pressure, care must be taken to ensure correct positioning, to avoid undue pressure over any bony prominence. Also note, they increase body temperature and could cause higher levels of moisture on the skin. Furthermore, pillows may increase body temperature.

The tissue at-risk may be targeted with pressure-relieving and redistributing patches that are placed directly on the at-risk site. Examples include Aderma (Smith & Nephew), and KerraPro (Crawford Healthcare). Some dressings specifically designed to manage the risk of PU formation are available, for example Mepilex Border (Mölnlycke Health Care) and foam dressings are often intended to manage the risk of PU formation.

Repositioning the patient is a critical part of removing pressure. Patients at-risk of PU formation should be repositioned every two to four hours, so that they lie or sit with weight supported on a different part of the body. A number of products are available to ensure that the patient remains in the desired position. These include shaped blocks and foams that are placed against the patient to prevent rolling or movement back onto the vulnerable skin site. Examples include Devon Utility Pad (Aria Medical), Mölnlycke Z-Flo Fluidised Positioner Z3 and Z4 (Mölnlycke Health Care) and wedges and foams from a number of companies.

In practice, it is common not to have positioning aids and, in this instance, pillows can be used to help position the patient. Patients who are able to should be advised to reposition themselves no longer than every six hours.[107] In patients who cannot be repositioned because of their medical condition, where available, a high-specification pressure-relieving mattress such as a low air loss or fluidised bed should be used. Where such a mattress is not available, advice should be sought from the MDT; perhaps tilting rather than fully repositioning may be of benefit. However, the risk of PU development due to the inability to reposition should be discussed with the patient/relatives and MDT, where available, and clearly documented in the clinical notes.

Friction and shear reduction

Friction deforms skin and induces internal tissue stress when the patient moves, or is moved, by sliding on a surface such as a bedsheet. Friction is reduced by placing a low-friction interface between the skin and the surface, or by absorbing some of the deformation in the interface. Friction-reducing products should be used where the risk of friction-induced shear stress has been identified. Examples of friction-reducing interfaces include slide sheets, which are distributed by several companies, and low-friction bootees, undergarments and pillow cases (APA Parafricta). Where low friction interfaces are not available, great care should be taken when repositioning and moving of the patient.

Skin care

Barrier creams should be used to protect against moisture-associated skin damage (MASD). Massaging or rubbing the skin should not be performed, to prevent PUs: hand movement used to apply protective creams should be enough only to ensure even spread of the cream. Sprays and dressings are also suggested as they are transparent and/or quick drying on the skin, examples include, Cutimed Protect (BSN medical), Cavilon (3M Ltd) and Opsite (Smith & Nephew).

Nutrition

Where nutritional deficiency has been identified, and where available, a nutritionist should assess the

Table 4. Recommended treatment and follow-up for patients in different risk categories for DFU formation[83]

Risk category	Definition	Treatment/action recommendations	Suggested follow-up
0	No LOPS*, no PAD**, no deformity	Patient education, including advice on appropriate footwear Skin/callus/nail care	Annually (by generalist and/or specialist), or as needed
1	LOPS±deformity	Consider prescriptive or accommodative footwear Daily self-inspection. Routine skin/nail care Consider prophylactic surgery if deformity is not able to be safely accommodated in shoes Continue patient education	Every 3–6 months (by generalist or specialist)
2	PAD±LOPS	Consider prescriptive or accommodative footwear. Daily self-inspection. Routine skin/nail care Consider vascular consultation for combined follow-up Continue patient education	Every 2–3 months (by specialist)
3	History of ulcer or amputation	Same as category 1 Consider vascular consultation for combined follow-up if PAD present.	Every 1–2 months (by specialist)

* LOPS—loss of protective sensation; **PAD—peripheral arterial disease

patient's dietary needs and advise on improvements to minimise the effect of malnutrition.

Diabetic foot ulcer prevention

All patients with diabetes and loss of protective sensation (diabetic peripheral neuropathy—DPN) are at risk of developing a DFU. The purpose of a risk analysis is to identify those at highest risk, to stratify the risk, and to target preventative interventions optimally.

The start point for prevention of DFU is care standards, as laid out in guidelines. The most widely-used worldwide guidelines on preventing DFU are those of the IWGDF.[109] Other guidelines include those prepared by NICE,[28] the Task Force of the Foot Care Interest Group of the American Diabetes Association,[83] the International Diabetes Foundation (IDF),[110] Saskatchewan Ministry of Health (MoH),[111] and the Wound Healing Society.[112]

The key components of DFU prevention that should be followed include:

- Nail care
- Emollient use
- Footwear
- Daily self-examination of the feet
- Not walking in bare feet
- Callus debridement
- Checking footwear and hosiery before wearing
- 'Breaking shoes in' never to be attempted
- No hot water bottles

- Checking bath and shower temperature
- Avoidance of home remedies e.g. corn plasters
- What to do and the appropriate person to contact if foot problems develop.

The Task Force of the Foot Care Interest Group of the American Diabetes Association[83] and Saskatchewan MoH recommend the approach laid out in Table 4 to stratifying and managing the risk of DFU formation.

The health-care services available to all persons living with diabetes should include the following (adapted from Saskatchewan MoH guidelines and others):[111]

- Daily foot inspection/examination and risk assessment
- Nail care
- Callus care
- Skin care
- Foot hygiene
- Podiatric management
- Pressure reduction to foot (offloading).

Appropriate selection of protective footwear includes:

- Commercially available shoes designed for the diabetic foot may be adequate for low-risk patients
- Added-depth shoes should be recommended for high-risk patients who have DPN, vascular insufficiency and/or mild-to-moderate foot deformity (a custom-moulded inlay may be needed)
- Custom-moulded shoes with custom inlays should be recommended for high-risk patients with advanced deformity
- Patients should be advised not to walk at any time without wearing protective footwear.

Further information on footwear for patients with diabetes can be found in updated recommendations from Diabetic Foot Australia.[113]

In addition to the measures that the patient should adopt (Table 4), the temperature of the foot should be assessed and, when higher than normal, the patient should be referred to a health professional. High temperature may indicate tissue breakdown and/or infection. Foot inspection may be assisted by the use of a mirror. However, patients with diabetes may have impaired vision because of retinopathy and should be assisted by a helper who has been educated in how to inspect the foot.

Management

Navigating the patient through the pathway

The first step is to identify a clinician who is the 'wound care navigator' (WCN). The job title of the WCN is less important than the ability to fulfil the requirements of the role. The role of the WCN is to conduct an appropriate assessment and refer quickly where needed. The job function of the WCN will vary from country to country, but the person should be trained and able to do the following:

- Assess the patient to identify those at risk of PU or DFU formation
- Take a patient history
- Identify the basic characteristics of the ulcer (location, size, depth, presence of necrosis, pain, signs of infection)
- Conduct simple tests to identify if an ulcer is most likely to be a PU or a DFU, particularly when the ulcer is on the heel (pulse palpation is crucial)
- Identify the additional tests and assessments required to fill in the gaps in knowledge and competencies
- Identify the appropriate care pathway and clinician to whom the patient should be referred
- Be aware of the urgency of the referral (i.e. a patient with ascending cellulitis, gas gangrene or necrotising fasciitis needs to be referred immediately).

Additional skills required may include: administer a monofilament test and/or vibration perception test; administer ABPI test; perform a Doppler ultrasound; and wound management.

The level of training and competence of the WCN may be at a basic level. Where competence does not include conducting pulse palpation and/or a basic Toe Touch Test, the WCN should know how, and to whom, to refer the patient. At the basic level, no specialist equipment is required to assess the patient. In the case of a possible heel PU or DFU, sensation and neuropathy is assessed by the Toe Touch Test and vascular status is assessed by pulse palpation.

Wound management

Where possible clinically, a PU or DFU should be managed to ensure timely ulcer closure. Standards of care specific to the management of PU and DFU have been published by a number of organisations.[7,53,107,108,111,114,115] However, where available, local guidelines should be followed.

There are also a number of generic guidelines on principles of best practice in wound management, for example, wound bed preparation: TIME and MOIST have also been published.[116–118] These provide information on how the major areas that must be considered when preparing the wound bed to aid healing.

The principles of TIME are used to guide health professionals on what to assess and treat in the wound bed:

- Tissue status: viable, non-viable, deficient
- Infection or Inflammation
- Moisture balance
- Epidermal margin; non-advancing or undermined.

Over the years, these principles have been modified to include other markers, such as TIME-H, which includes a healing score. Another variation on the TIME principle, recently developed by the German wound association, Initiative for Chronic Wounds (ICW) e.V. is MOIST:[118]

- Moisture balance: exudate management, ensure that the wound is neither too moist nor too dry
- Oxygen balance: in the pathophysiology of chronic wounds, hypoxia plays a decisive, central role in nearly all types of wounds
- Infection control: all antimicrobial strategies in wound therapy regimens
- Support: if, despite an apparently adequate therapy, wounds do not heal, specific wound care agents can be used temporarily
- Tissue management: all measures of conditioning the wound bed, for example, neutral wound dressings, biosurgery or physical aids such as negative pressure wound pressure (NPWT), electricity, plasma, or ultrasound.

MOIST covers the general principles of TIME and includes a section on oxygen balance, which if compromised will hinder wound closure and may be of particular importance in ischaemic DFUs.

General principles of wound management

The general principles of effective wound management, embodied in all guidelines in slightly different ways, should be implemented for PU and DFU. The principles common to all guidelines include the following steps:

1. Assessment and diagnosis
2. Development of care plan
3. Management of the underlying condition and causes (including offloading for DFUs and pressure relief for PUs)
4. Management of exudate
5. Management of bioburden (infection and biofilm) and inflammation
6. Debridement
7. Managing hypoxia
8. Nutrition and hydration
9. Monitor progress and adjust care plan
10. Prevent recurrence.

In the next section, the processes (excluding assessment and diagnostics) and procedures recommended for the management of the wound, including its underlying condition and causes, to

maximise the probability of healing, are described. The flow in Fig 4 is a flowchart showsthe basics of these management and treatment pathways.

These steps may be achieved with products that range from low-cost and basic to the high-cost and advanced. Care should be delivered using products that have evidence-based data on their effectiveness in the local population. Effectiveness may be measured by clinical efficacy and health economic analysis. Health economics in particular are specific to the patient population and health-care delivery system in which the analysis was conducted. Assessment and diagnosis has been covered in detail in section 3. Tables 5 and 6 show areas to consider when treating a PU or DFU.

Care plans

A care plan describes how the patient will be managed, based on the outcomes of assessment and diagnosis. The plan covers the care that needs to be delivered, the procedures, processes and competencies required to deliver the care, and where treatment will take place. Referral is part of the care plan (section 3), because it requires an assessment to identify what needs to be delivered and the competencies, and, therefore, which health professionals should be involved.

Managing the underlying condition and causes

Delayed wound healing is a consequence of a wound being stuck in the inflammatory stage of the healing cycle. Trapped in this inflammatory phase, there is an excess of inflammatory molecules, including inflammatory cytokines, free radicals, and proteases such as matrix metalloproteinases (MMPs) and tissue

Fig 4. Management and treatment flowchart for diabetic foot ulcer and pressure ulcers

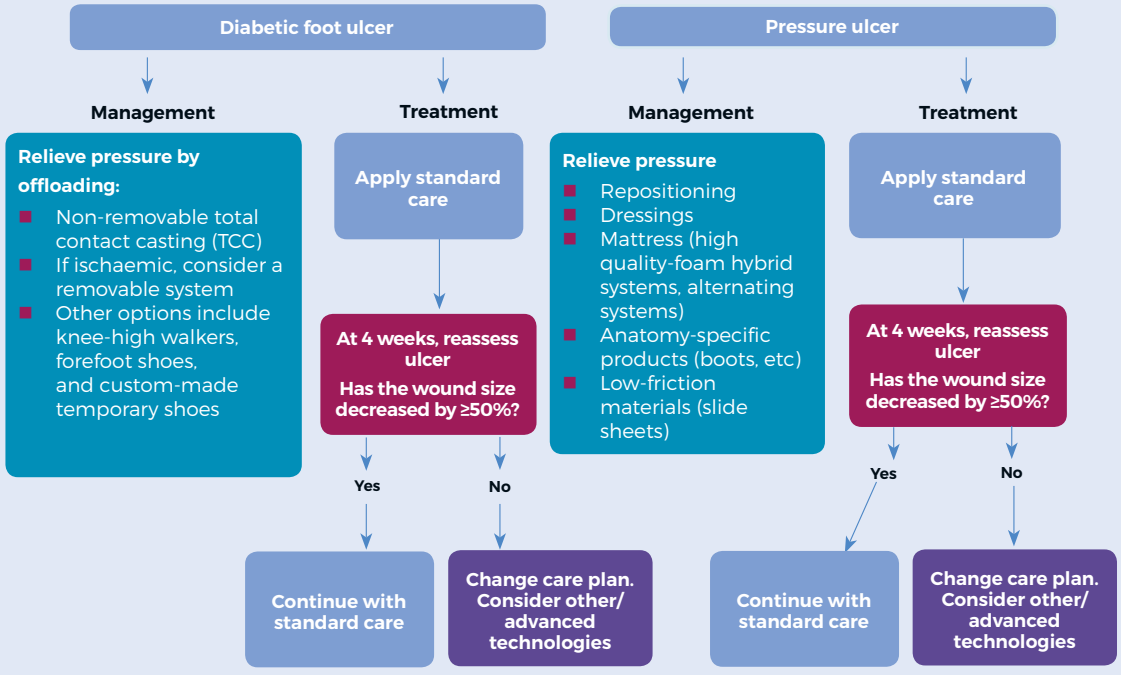


Table 5. Consideration for standard pressure ulcer care.* Will vary depending on the local protocol for standard care. Always refer to local guidelines

Consideration for standard PU care	Examples
Management of pressure/ shear and friction	Repositioning (foam) Dressings, silicone multi-layered foam dressings (Allevyn, Smith & Nephew; Mepilex Border, Mölnlycke; Tielle Foam Dressing, KCI) Mattress (high-quality foam e.g. Trezzo, HS, hybrid systems, alternating systems) Anatomy-specific products (boots, etc) Low-friction materials (Parafricta bootees, slide sheets) Skin protectants (Cavilon, 3M; Cutimed Protect, BSN medical; Opsite, Smith & Nephew; Remedy Olivamine Dimethicone Skin Protectant, Medline Industries; Secura, Smith & Nephew)
Debridement options	Dressings (Cutimed Sorbact Hydroactive, BSN medical; Debrisoft, L&R; Hyrdoclean plus, Hartmann) Hydrosurgery pressurised water (Versajet, Smith & Nephew) Larval/maggot debridement therapy (Biobag, BioMonde; Medical Maggots, Monarch Labs) Pulse lavage (Microaire Stryker) Ultrasonic debridement therapy (Sonoca, Söring; SonicOne, Misonix) Surgical/sharp
Early diagnosis and treatment of Infection	Assess clinical signs of infection: PTB, simple X-ray, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), leukocytosis, Treatment according to infection severity
Prevention and treatment of bioburden (infection and biofilm) and inflammation	Cadexomer/povidone iodine, antimicorbial (Iodosorb, Smith & Nephew, Inadine, KCI) Bacterial-binding dressing (Cutimed Sorbact; BSN medical) Honey, antimicrobial (Activon, Advancis; Surgihoney RO, Matoke Holdings) Polyhexamethylene biguanide (PHMB), antimicrobial (Kerlix AMD Antimicrobial, H&R Healthcare; Tielle PHMB, KCI) Oxidized regenerated cellulose/collagen/silver, MMP and elastase modulator (Promogran Prisma Matrix KCI) Prontosan, antiseptic (B Braun) Octenidine, antiseptic (Octenisept Schülke) Silver, antimicrobial (Acticoat, Smith & Nephew; Aquacel Ag, ConvaTec; Silvercel Non-Adherent, KCI; Sorbasan Silver, Aspen Medical) Superoxidized water, antiseptic (Microcyn, Dermacyn)

*note: the table contains examples of products and technologies and is not an exhaustive list

inhibitor matrix metalloproteinases (TIMPs) , which become harmful to the wound bed and periwound area, disrupting wound healing.[119,120]

The molecular basis of incomplete wound healing and the change from an acute to a chronic wound is a major focus of attention in wound healing research in patients with diabetes. DFUs have a prolonged inflammatory phase with fibroblast dysfunction, impaired neovascularisation, and increased concentrations of MMPs. This excess of MMPs alters the wound healing process through degradation

Table 6. Consideration for standard DFU care.* Will vary depending on the local protocol for standard care. Where available, always refer to local guidelines or IWGDF guidelines[53]

Consideration for standard DFU care	Examples
Metabolic control and management of the comorbidities	Control glucose levels HbAc1 Renal function (Creatinine, Albumin) Random urine microalbumin, proteinuria
Assess vascular status	Pulse palpation ABPI and waveform Ankle systolic pressure Toe systolic pressure $TCPO_2$, Tissue perfusion
Offloading	Non-removable total contact casting (TCC) (Delta-Cast Conformable, BSN medical) If ischaemic, consider a removable system TCC-EZ (Derma Sciences). Others options include knee-high walkers, forefoot shoes, and custom-made temporary shoes.
Debridement and callus removal	Preferably surgical, except when ischaemia is present; in this case, consider other techniques; autolytic, enzymatic Podiatric drill for callus removal
Early diagnosis Infection	Assess clinical signs of infection: PTB, Simple X-ray, C-reactive protien (CRP), erythrocyte sedimentation rate (ESR), Leuocytosis, ATB treatment according to infection severity (IDSA/IWGDF guideline, guidance)[xxx]
Prevention and treatment of bioburden (infection and biofilm) and inflammation	Cadexomer/povidone iodine, antimicorbial (Iodosorb, Smith & Nephew Inadine, KCl, Povitulle, CD Medical) Bacterial-binding dressing (Cutimed Sorbact; BSN medical) Honey, antimicrobial (Activon, Advancis; Surgihoney RO, Matoke Holdings) Polyhexamethylene biguanide (PHMB), antimicrobial (Kerlix AMD Antimicrobial, H&R Healthcare; Tielle PHMB, KCl) Oxidized regenerated cellulose/collagen/silver, MMP and elastase modulator (Promogran Prisma Matrix KCl) Silver, antimicrobial (Acticoat, Smith & Nephew; Aquacel, ConvaTec; Silvercel Non-Adherent, KCl; Sorbasan Silver, Aspen Medical)
*note: the table contains examples of products and technologies and is not an exhaustive list	

of the extracellular matrix (ECM), affecting both the control of the activities of various effector proteins such as growth factors and the deposition of new ECM.[119,120] DFUs often fail to heal because of persistently high levels of pro-inflammatory cytokines in the wound, which induce high levels of MMPs and subsequently destroy growth factors, receptors and matrix proteins essential for wound healing. MMPs are also responsible for the controlled fragmentation of the basal membrane, induction of inflammation and angiogenesis, as well as for epithelialisation. Modulation of MMPs in the wound area, as well as other regulating factors of wound healing (e.g. PDGF, FGF, EGF, cytocines, etc.), could be a benefit in the treatment of chronic wounds.

Pressure ulcers: pressure and friction/shear should be managed with methods that achieve pressure

reduction and redistribution. These are generally the same as those used for PU prevention and include pressure-relieving and redistributing surfaces, anatomy-specific products such as heel protection boots, dressings, and repositioning the patient.

Moisture management should include barrier creams, and methods to contain and control incontinence. An active PU is itself a source of moisture. Please refer to the section on 'managing exudate' for more detail.

Local treatment guidelines, where available, should be used; however, examples include using the SSKIN bundle with treatment tailored to the condition of the wound.

Diabetic foot ulcers: blood flow, neuropathy and foot deformity leading to pressure and infection should be managed. These are components of VIPS.

As discussed earlier, every patient with a DFU should have a vascular assessment. If a patient has tissue loss and an ABPI of ≤0.90, that will require vascular review. Similarly, tissue loss and a systolic toe pressure of <50mmHg that necessitates vascular review as a TBI of <50mmHg has been associated with impaired healing. Other suggested examinations before referral are: pulse palpation, Doppler isonation, with monophasic/biphasic/triphasic sound. With Doppler isonation, a monophasic pulse would be abnormal and necessitate further assessment/referral, as the presence of a monophasic Doppler is considered indicative of PAD.

Neuropathy leads to inability to sense pain in the foot. Patients with neuropathy may wear shoes that are too tight, because they cannot feel when they do not fit correctly. Furthermore, loss of sensation means that a wound or object in the shoe that could cause injury goes unnoticed. Coupled with repetitive trauma from walking, this will cause ulcer formation. Furthermore, deformity causes high pressure points, which are vulnerable to damage. All patients with neuropathy and a DFU should wear correctly fitted offloading footwear. Amputation leads to abnormally high pressures underneath the foot and requires offloading customised to the foot shape.

The optimal offloading method associated with the highest rate of full DFU closure in the shortest time is non-removable TCC (an example is Delta-Cast Conformable, BSN medical).[121–124] The foot is closely fitted with a cast that distributes pressure over the entire plantar surface of the foot. TCC application is highly skilled and should be done by health professionals fully trained in the technique, to minimise the likelihood of rubbing, causing additional damage and to optimise pressure redistribution. In general, the initial change will take place 2–3 days after the first cast is applied (to ensure that everything is alright). Afterwards, the cast will generally be changed about once a week, or as determined by the health professional, to accommodate any reduction in limb size as oedema reduces and to inspect the skin and foot for damage. A TCC alternative that is easier to apply, but still requires training, is the TCC-EZ (Derma Sciences). This product may be considered instead of a traditional TCC.

Where the competency for traditional non-removable TCC or TCC-EZ is not available, other removable footwear options for offloading should be used when appropriate to the health-care system and the patient's preferences. These include knee-high walkers, forefoot shoes, and custom-made temporary shoes. DFUs heal less well with removable offloading compared with non-removable TCC, because devices are often removed and not used when the patient walks. Non-use reduces the offloading delivered to the foot, impeding effectiveness. It is important therefore to ensure that the patient will wear the offloading device at all times when ambulatory, even in 'safe' environments such as the home. The offloading must be fitted with an interface between the foot and the internal surfaces of the device to ensure optimal pressure redistribution. Where offloading devices are not available, felted foam should be used. An alternative is complete pressure removal with crutches, walkers, wheelchairs or foot elevation. Where possible, all neuropathic and ischaemic DFU should be managed with offloading.

Where offloading is not successful, further options, as required after assessment by the MDT, are required, such as surgical intervention to correct deformities.

Management of exudate

Exudate from a chronic wound not only increases skin moisture when in contact with the surrounding skin, but also contains destructive biological molecules, including protein-degrading enzymes that may harm the wound bed and periwound skin. It is important to minimise the amount of exudate that comes into contact with skin and the duration of contact for both PU and DFU. Exudate is generally managed by dressings or NPWT.

Dressings provide a cover for the wound, to help avoid contamination of the ulcer contamination from exogenous sources and the dispersal of organisms from the wound to the environment. There is widespread agreement that the ulcer should be maintained in a moist, warm environment to encourage healing. Some authorities advise that gauze should not be used, and that the least expensive dressing that fulfils the clinical requirements should be used. Local guidelines should be followed. Dressing selection depends on several factors, including:

- The site and size of the ulcer
- The amount and type of exudate
- The stage of healing of the ulcer and predominant tissue type
- The integrity and condition of the surrounding skin
- The quality of the patient's skin
- The patient's tolerance of adhesives
- Pain
- Comfort and QoL
- The anticipated frequency of dressing change
- The need for topical antimicrobial management of the ulcer
- Compatibility with other elements of the overall care plan
- Cost
- Availability and formularies
- Local guidelines.

Management of bioburden (infection and biofilm) and inflammation

Bioburden, and biofilm in particular, is believed to impede healing.[125] At least 60%, and possibly all, of chronic wounds have mature, established biofilm on the surface and in deeper tissues[126,127] and it is challenging to diagnose clinically.[126] There are no biofilm-specific markers and it cannot be seen by the naked eye. The diagnosis that biofilm is contributing to impeded healing is therefore made by eliminating other factors that may impede healing. When assessment suggests that biofilm contributes to impaired healing, early intervention is recommended. New regimens are being suggested, such as biofilm-based wound care (BBWC),[126,127] which aim to disrupt and suppress biofilm, allowing local antimicrobial agents to kill the bacteria.

However, a gradually de-escalating regimen, informed by assessment of inflammation and wound healing, is recommended. Starting on days 1 to 4, aggressive debridement, topical antiseptics and systemic antibiotics, management of underlying host factors, and profiling microorganisms using genetic methods are recommended.[126,127] Treatment is de-escalated with regular debridement and cleansing as healing improves. Genetic profiling of microorganisms is highly specialised and available in a few institutions. Where required, standard microbiological evaluations may be conducted, using swab, or preferably biopsy, specimens.

Microbiological analysis is used to direct antibiotic therapy, not to diagnose infection. Many guidelines contraindicate systemic antibiotics where clinical infection is absent. Local guidelines should be followed. Antimicrobial agents do not improve healing in wounds where bioburden is not the cause of impaired healing. Their effect is to help manage bioburden, which in turn impairs healing. Antimicrobial dressings are not generally recommended for preventing secondary infection, but may be recommended for mild clinical infection. Topical antiseptics/antimicrobials should be used

where a microbiological cause of impaired healing has been identified. They should be used for up to two weeks, and the wound regularly re-assessed. If healing has improved, topical antiseptics/antimicrobials should be stopped and non-antimicrobial dressings used. If healing has not improved, the wound should be assessed to decide whether to continue the current antiseptic or to switch to a different antiseptic. Assessment should include factors other than ulcer bioburden that may be impairing healing.

Clinically-diagnosed infection should be managed using systemic antibiotics. Topical antibiotics are not recommended, and are associated with increased risk of development of antibiotic-resistant organisms. Antibiotics should be selected based on ulcer specimens and antibiograms. In severe infection, particularly in the DFU, immediate empiric broad-spectrum parenteral antibiotics should be administered as per local guidelines. Once the sensitivity data are available from the microbiology service, antibiotics should be customised to the patient. Duration of antibiotic therapy should be according to clinical assessment outcomes. Antibiotic stewardship guidelines should be followed. Management of infection includes surgical drainage of abscesses and excision of infected bone.

Microbiological specimen collection may be achieved using one of a number of methods, according to local practice and guidelines. General principles include:

- Specimens should be collected before starting antibiotics
- The ulcer should be debrided and cleaned before specimen collection
- Specimens should be transferred quickly to transport medium to preserve the specimen
- The request should include tests for aerobic and anaerobic organisms and antibiotic sensitivity.

Sampling methods include pus collected from the deepest part of the wound, swabs (a number of swabbing methods are available), aspiration, tissue biopsy, and, for osteomyelitis, bone biopsy. Osteomyelitis should be suspected if a probe or finger touches bone. Antibiotic therapy should be continued for up to six weeks for osteomyelitis. Additional diagnostic procedures for osteomyelitis such as X-Ray, MRI, CT scanning and other advanced methods may be used where available. Where systemic infection is suspected, blood cultures should be done. For further reading, see Harries et al.[117] which explains the different forms, infection prevention and management.

Chronic inflammation should also be managed. Kick-starting a wound stalled in the inflammatory stage into healing by modulating excess inflammatory mediators should be considered. Impaired neovascularisation and excess of MMPs are two major factors impeding the healing of chronic wounds, especially the ones encountered with vascular insufficiency.[128,129] Compounds have been shown to modulate MMP levels[130] and have effects on other cellular molecules, including growth factors and neovascularisation (sucrose octasulfate)[132] and other proteases and cytokines (collagen ORC).[133] Modulation of these signals has been shown to improve wound closure of DFUs,[134–136] demonstrating the potential of these healing enhancers.

Debridement

Debridement is an important component of a good standard of wound management. Debridement removes calluses, unwanted and dead tissue, and slough from the wound. It enables accurate assessment, helps drainage, improves healing, removes biofilm, and a reservoir of potential infection. Debridement may be accomplished by a number of methods, which should be selected according to clinical assessment, the needs of the wound, local practice, and availability of equipment and competencies. Debridement methods include:

- Autolytic: hydration of tissue to allow natural host proteolytic enzymes to remove devitalised tissue. Hydration is obtained in dry tissue using hydrogel or honey
- Enzymatic: exogenous proteolytic enzymes used

to dissolve devitalised tissue. The efficacy of enzyme debridement is considered unproven by some authorities
- Larval: 'biosurgery'. Use of greenbottle fly larvae to remove devitalised tissue selectively. Cannot remove callus. Larvae must be prepared by specialist suppliers
- Surgical sharp: invasive debridement with surgical instruments under anaesthesia for sensate patients; anaesthesia may not be required in neuropathic DFU. Surgical debridement should be conducted only by competent practitioners. A curette may be used to scrape loose material gently off the wound. Pain management may be required
- Hydrosurgery: surgical debridement with pressurised water jet to dislodge and remove devitalised tissue
- Ultrasonic:[137,138] ultrasound and fluid to remove devitalised tissue mechanically. Relatively recent introduction means it may not be widely available.

Health professionals must be able to distinguish between tissues and structures to avoid damaging the local anatomy when debriding and have a high level of clinical decision-making to control the extent of debridement. An old mechanical technique known as 'wet-to-dry', in which a wet gauze is allowed to dry on the wound and then pulled off, is no longer recommended, because it causes pain for the patient and removes tissue indiscriminately, causing trauma.

For PU, debride only when clinically indicated by the presence of devitalised tissue or slough and when there is adequate tissue perfusion to the wound. Any of the debridement methods may be used, taking into account the size and depth of the PU, clinical requirement for speed of debridement, patient tolerance, especially with surgical debridement, comorbidities and the care plan, which may include grafting, for which a clean recipient wound bed is essential. Surgical debridement is appropriate for PU with extensive necrosis, advancing cellulitis, crepitus, fluctuance, and/or sepsis secondary to ulcer infection. Larval debridement may be considered where sharp debridement is contraindicated.

For DFU, debridement has been shown to improve DFU healing.[139] Where available, the widely-accepted standard is sharp debridement using scissors or scalpel and forceps. Vascular status should be confirmed before debridement and compromised tissue should not be surgically debrided. Non-surgical debridement should be used where the required competencies are not available or in patients who cannot tolerate surgery. Larval debridement may be considered, if it is available.

Managing hypoxia

Patients with diabetic foot syndrome often display a functional vascular impairment caused by a thickening of the basal membrane and endothelial capillary swelling. As a consequence of the developed neuropathy, the endothelium-dependent regulation of the vascular lumen is affected by nitric oxide (NO) and the neuronal regulation of the precapillary arterioles is deregulated. Due to such dysfunctions, an adequate reaction in the foot to an injury like an increased blood flow in response to the high demand of oxygen and nutrient cannot be achieved. Although the feet of patients with diabetes seem phenotypically healthy, such underlying structural and molecular changes may prevent a sustained oxygen supply when needed after an injury.[118] An important consideration in physiologic wound healing is oxygen supply and oxygen tension in the wound bed. The oxygen balance in wounded tissue is an important challenge, as it affects all other aspects required for appropriate wound healing.[118]

Nutrition and hydration, glycaemic control

Good nutritional status is required for optimal healing. Patients should be assessed by a nutritionist or other health professional competent to conduct a nutritional assessment and diet, and fluid intake adjusted according to clinical need.

The following tools could be employed to assess status:

- Malnutrition Screening Tool (MST)
- Mini Nutritional Assessment (MNA) – short form and long form

- Malnutrition Universal Screening Tool (MUST)
- Subjective Global Assessment (SGA)

Note: in developing countries, the dietitian and not the nutritionist perform the dietary assessments using their Dietitic Care Notes (DCN).

Monitor progress and adjust care plan

The ulcer should be inspected and assessed at least weekly to monitor progress. Where clinical improvement is not seen, regular assessment will indicate an alternative care plan, which should be documented and implemented. An accepted time point is 4 weeks following the start of DFU treatment when the ulcer should be assessed using the methods previously described. Healing progress measured as area reduction and wound bed improvement at this time point is generally regarded as an indicator of the likelihood of complete ulcer healing.[140-143] In cases where the ulcer size has reduced by <50% at 4 weeks, an alternative care plan should be considered. The new care plan may need referral for tests and evaluations or other more advanced interventions where local guidelines recommend them. These may include advanced therapies or surgical procedures for debridement, grafting or vascular reconstruction. In the US, the Centers for Medicare and Medicaid Services (CMS) uses the 4-week statistic as a trigger for reimbursement of advanced therapies. Other jurisdictions advise not using some advanced therapies, because the health economic advantage has not been adequately proven.

Where the expected clinical progress is met, treatment should continue according to the care plan.

Prevent recurrence

Prevention has already been covered in detail in this section; however, it is worth mentioning certain treatments should be performed until complete wound closure, to avoid recurrence of infection/slough/exudate/pain or to stall wound healing.[144] The main causes for recurrences in DFUs are: location of the ulcer (plantars surface and specially beneath first metatarsal head), use of non-appropriate shoes, presence of foot deformities and previous amputation.

Technologies and therapies to consider

Treatment of PUs and DFUs is not effective in every patient; some wounds do not heal in a timeframe consistent with expectations and clinical experience. When this happens, alternative approaches are required, including new or advanced therapies. This modification of the care plan must be founded on objective information. A stepwise approach based on detailed patient assessment should be adopted.

This section looks at some of the options and alternatives available, recognising that these options are not available in all countries and that, where they are available, national guidelines and payment systems may not cover their use. Table 7 shows a number of options. It should be noted that the therapies suggested here and in the standard care options described above reflect variations in preferences worldwide. For a full review of new advanced therapies, see the EWMA document, *Advanced therapies on wound management.*[146]

What to do if not healing with standard care

If standard care has failed to lead to a reduction in the wound size ≥50% over four weeks,[140-143] the first step is a thorough and detailed reassessment of the ulcer and the patient. The accuracy of the original diagnosis should be validated and the treatment choices reviewed. Has the underlying condition changed? New tests may be required. For example, where a basic test such as pulse palpation was carried out, would better information be provided by a more advanced test such as a full vascular work up, if available? Perhaps a basic IpTT or vibration perception threshold test gave inaccurate information. Would a more detailed analysis of nerve conduction provide better diagnostic fidelity? Where a diagnosis of uninfected was made, would a white blood cell count or C-reactive protein (CRP) test give more helpful information? Is it inflammation? Is there something about the wound, such as carcinoma, that was previously not detected?

Key points

- If the wound has not healed by ≥50% over four weeks, reassessment is required
- Debridement may need to be more aggressive if healing is stalled
- Where offloading is not successful, non-removable or complete offloading may be appropriate
- Diagnostic tools can aid the choice of new treatment
- Use therapies that are evidence-based.

Once the assessment has provided up-to-date information and it is confirmed that the previous standard of care was correct, it may be appropriate to consider other therapies and more aggressive treatment regimens. A benchmark for making a decision on switching to therapies is the healing response after four weeks' care with best practice.[140-143] If the wound has not decreased by ≥50% from the start of treatment, then a switch may be indicated.

The advancement may be escalation of the intensity of treatment or a change to a different way of managing the condition of the ulcer. Some examples are detailed below; this is not an extensive list, but aims to provide examples for consideration.

Diagnostic methods

There are more advanced methods to assess PAD, such as magnetic resource angiograms or computer tomography angiograms, which may be performed by a vascular specialist, if required.

Early detection of sub-epidermal moisture (SEM) changes can be measured using the electrical properties of skin (bioimpedance).[101–102] A recent literature review concluded that the SEM scanner (Bruin Biometrics) is an objective and reliable method of local bioimpedance, and therefore, enables assessment of tissue damage before there are visible signs of it present.[102]

Table 7. Potential therapies to consider if not part of local standard care.* Note these will vary for region to region as with protocols for standard care. Always refer to local guidelines

Debridement	Dressings (Cutimed Sorbact Hydroactive, BSN medical; Debrisoft, L&R; Hyrdoclean plus, Hartmann) Hydrosurgery (Versajet, Smith & Nephew) Larval/maggot debridement therapy (Biobag, BioMonde; Medical Maggots, Monarch Labs) Ultrasonic debridement therapy (SonicOne, Misonix; Sonoca, Söring)
Prevention and treatment of bioburden (infection and biofilm) and inflammation	Bacterial-binding dressing (Cutimed Sorbact, BSN medical) Honey, antimicrobial (Activon, Advancis; Surgihoney RO, Matoke Holdings) Gas plasma, antimicrobial antibiofilm (SteriPlasma, AdTech)
Exudate management	Superabsorbant dressing, moderate-to-high absorption (Cutimed Sorbion Sachet S, BSN medical) Gelling fibre (BIOSORB, KCI) NPWT (PICO, Smith & Nephew; RENASYS touch, Smith & Nephew; SNAP, KCI, VAC Therapy, KCI) Foam dressings (Cutimed Siltec, BSN medical; Tielle Foam Dressings, KCI)
Topical agents/ healing enhancers	Oxygen delivery (Granulox, SastoMed; Natrox, Inotec; epiflo, Ogenix) Oxidized regenerated cellulose/collagen/silver , MMP and elastase modulator (Promogran Prisma Matrix KCI) Sucrose octasulfate (Nano Oligosaccharide Factor), MMP inhibitor, aids neovascularisation (UrgoStart, Laboratoires Urgo Medical) Biologics/skin substitutes (Apligraf, Organogenesis; Dermagaft, Organogenesis; EpiFix, MiMedix, Omnigraft, Integra)
Diagnostic methods	Clinical information on bioburden (Moleculight i:X, Smith & Nephew; Woundchek Bacterial Status, Woundchek Laboratories Sub-epidermal moisture (SEM) measurement (Bruin biometrics) Hyperspectral imaging of oxygen levels (TI-CAM, Diaspective Vision)
*note: the table contains examples of products and technologies and is not an exhaustive list	

Advances in detection of bacteria in wounds have been made and a handheld fluorescence imaging device (Moleculight i:X; distributed by Smith & Nephew) has been made available. Moleculight uses safe 405nm light to visualise bacteria by detecting porphyrins that fluoresce red[146] and pyoverdine/siderophores produced by *Pseudomonas aeruginosa* that fluoresce cyan. Moleculight helps the practitioner visualise where bacteria are located in the wound to target and monitor debridement effectively,[147] providing information as to a clinically relevant level of biobuden (>10^4 colony forming units/g).

Point of care swab tests may also aid the assessment of whether wounds are non-healing due to elevated host protease activity or bacterial pathogenesis. Woundchek Bacterial Status detects bacterial protease activity, a common virulence factor that is indicative of pathogenic behaviour of bacteria in the wound before clinically observable infection, at a point in the infection continuum where antimicrobial treatment is typically required. Woundchek Protease Status detects elevated host protease activity (MMPs and neutrophil elastase), a marker of chronic wound inflammation.

Preliminary data have suggested that an hyperspectral imaging system (TI-CAM, Diaspective Vision) may be useful as a diagnostic tool to help aid health professionals decide on treatment options by providing rapid tissue perfusion measurements, including superficial oxygenation (StO_2 [%]), Tissue Haemoglobin Index, NIR Perfusion Index and Tissue Water Index in the wound.[148] By combining the various pieces of information, it maybe possible to get a holistic picture of the condition of a wound.

Offloading

In most cases, offloading can be achieved with low technology and relatively low-cost products. Where offloading is not successful, perhaps because the patient removes it or a previous amputation that has made it difficult to effectively offload the foot, the use of non-removable offloading or complete offloading using crutches or a wheelchair may be appropriate.

Debridement

Debridement may need to be more aggressive to remove devitalised tissue. Where access to the operating theatre is possible, this is often achieved by surgical debridement. Other less aggressive debridement methods may be effective at removing biofilm without causing discomfort to the sensate patient. This in itself may enable better debridement, because the patient is able to tolerate the procedure better. An example is monofilament debridement pads.[149] An alternative to sharp surgical debridement is hydrosurgery—using a pressurised water jet to remove devitalised tissue.[150] This method may cause less pain and discomfort to the patient and be more suitable for patients who cannot tolerate surgery. Ultrasonic debridement should be considered, especially in DFU with grade of ischaemia when surgical debridement is contraindicated. Another example for wound cleansing is VAC Veraflo cleanse choice dressing (KCI), used with VAC Veraflo therapy, to initiate immediate wound cleansing and it may be considered when surgical debridement is not appropriate.

Managing infection

The cornerstones of managing infection remain wound cleansing, debridement and antimicrobial agents. More aggressive debridement may be required to ensure that all reservoirs of infection and sites of potential re-infection have been removed. Once achieved, a change to a different topical antiseptic may be appropriate, in combination with systemic antibiotic therapy. Prophylactic antibiotics are not appropriate for all patients, but where the risk justifies it, then prophylaxis may be required.

Bioburden and biofilm

Options for management of bioburden and biofilm include products containing cadexomer/provodine iodine (Iodosorb, Inadine), topical antimicrobials that also assists in desloughing. An alternative to the microbicidal activity of iodine is physical removal of organisms. An example is bacterial-binding dressings (Cutimed Sorbact, BSN medical) that facilitate the passive hydrophobic binding of organisms, which become trapped in the dressing and are removed at dressing change.[151] Advantages of this sort of therapy include lack of bacterial resistance, its ability to attract antibiotic-resistant bacteria, non-allergenicity and no cytotoxicity. In addition, as organisms are removed rather than killed, endotoxins are not released into the wound.

Exudate management

Where exudation from the wound is high, options include highly absorbent dressings and NPWT. Highly absorbent dressings are able to absorb and retain large amounts of exudate, removing it from the wound and keeping it isolated from the skin. Examples include Cutimed Sorbion (BSN medical), Tielle Liqualock (KCI). Where the amount of exudate is too great for dressings, NPWT is a long-established alternative. Examples include Renasys Touch (Smith & Nephew) and VAC Therapy (KCI).

Healing enhances

Outside of the normal therapies that are known to aid wound healing, there are adjunct therapies which do not fit in to the areas described.

In recent years, topically applicable adjunctive therapeutic options have been developed in this area of wound care and a number of consensus documents support the use of oxygen as in chronic non-healing wounds. All approaches aim to deliver oxygen locally to increase the oxygen concentration in a specific area where it is most needed at a particular time. It is known that wound healing has a high oxygen demand. In many cases, DFUs and PUs are hypoxic, which requires additional oxygen supply. One such product is Granulox (Sastomed), which enhances the oxygen diffusion by using purified haemoglobin. Based on the available clinical evidence (Grade 1B),[152] it has successfully been implemented in treatment regimens of PU and DFU.

Other local oxygen therapies deliver an oxygen-rich atmosphere to the wound area, either by topical continuous delivery of non-pressurised (normobaric) oxygen (CDO) through small cannulas or thin tubes (Natrox/Epiflo) to wound dressings, or by small chamber-based constant pressure devices (TWO_2/TO_2).[118,152]

There are also a number of healing enhancers which affect the mediators of inflammation, such as MMPs and elastase (collagen/ORC (Promogran, KCI). A systematic review of collagen wound dressings used in treatment of DFUs demonstrated that they can be effective in aiding healing.[135]

Another healing enhancer, NOSF (sucrose octasulfate, Urgo Start), which inhibits MMPs as well as effecting neovascularisation, reported positive outcomes in a recent double-blind RCT.[134] The results showed an increased healing rate and significant decrease of time to closure.

Growth factors (GFs) and tissue equivalent (TE) products are available in some countries. GFs include becaplermin (e.g. Regranex; Smith & Nephew). TE products for which a range of cellular or acellular extracellular matrix-based sheets are commercialised (e.g. amniotic membrane allografts, foreskin-derived bioengineered grafts, split-thickness skin grafts), including Omnigraft (Integra), which decreased the time to complete wound closure and increased the rate of wound closure in a recent clinical trial.[153] Many authorities do not recommend growth factors and TE. These products are reimbursed by CMS in the US once the 4-week clinical response threshold has been reached.

Advanced treatment modalities that are cost-effective and time-sensitive are often indicated for chronic non-healing wounds to facilitate wound closure. Recent advances in wound care technologies, especially the advent of bioengineered alternative tissue, have provided numerous options to help with wound closure.

Summary

Not every country has access to all these products. Where products are available, the diagnosis may govern which are covered by reimbursement or insurance. An example is the US, where a diagnosis of diabetes leads to access to advanced products and more highly-skilled practitioners. Furthermore, treatment modalities vary across the world, depending on local guidelines and professional groups that manage the wound. This document hopes to create some equity on how patients are managed, to provide information that enables adoption of best practices, and, where needed, to stimulate development of standards of care and education.

Education

Wound management grows ever more sophisticated, as our understanding of the fundamentals of wound formation, management and prevention increases. Standards of care advance; products, technologies and processes that improve care and outcomes are developed and launched. Education, for health professionals and patients, is vital. Education is the first step in ulcer prevention and management.

Education is a long-term commitment for the learner and the educator, requiring messages to be repeated consistently over time in different formats. People learn in different ways, which the educator must acknowledge, and they must offer content that fits different learning styles. Furthermore, they must understand that the ability of the learner to see/hear the message, assimilate and reduce it to practice, varies by individual. Educational materials must account for these differences, understanding that many patients and family members may have basic educational attainment and poor language skills. Many patients, and indeed practitioners,[154] already have long-standing and firmly-held beliefs about the condition, gleaned from sources which do not accord with medical opinion. For many, perhaps most patients, medical language is impenetrable.

This consensus focuses on three areas for education:

- Empower patients, families and carers
- The health system
- Social welfare.

A useful mnemonic for education is EDUCATION defined as follows:

E: Empower
D: Develop/deserve what they need
U: Understand problems/risks
C: Care
A: Advocate
T: Teach
I: Inquire

Key points

- Education is the first step in ulcer prevention
- Health professionals should recognise the problem, know what to do, or who to refer the patient to
- Health professional education must be informed and supported by evidence-based guidelines on best practice

O: Observe
N: Nurture.

Empower patients, families and carers

The patient's socioeconomic status should be acknowledged, with the aim of maximising the role of the patient in reaching outcomes. Key areas are: understanding the condition; the implications and how they may affect the patient; risk categories; understanding glycaemia and managing it well in diabetes; how to prevent a wound forming; the role of foot protection by offloading and the importance of adherence; and daily foot care inspection and monitoring; what to do if problems arise with the foot and how to contact the right practitioner; what to do once the wound has formed (how to dress it, how to bathe with it); increase the level of knowledge about therapies, treatments, prevention of complications and prognosis; understand the patient's, family's and carer's role in managing the wound and adhering to the care plan; how to help and encourage the patient; and how to explain to others about their condition. The patient should also be able to help the practitioner during consultations. Preliminary evidence from Malaysia suggests patient education is successful in facilitating prevention and healing.[155]

Delivery

Delivery can take a number of forms: clinic leaflets, posters, group sessions, face-to-face by the practitioner,

pharmacists (when patients collect prescriptions), and websites. Facebook and social media can be a problem, with fake items and 'crystal waving'—trying to heal people by the use of precious stones.[100]

The health system

Consistent delivery of care across the health-care system depends first and foremost on educating the health professionals in best practices, which are underpinned by effective products, technologies and organisational support. However, consistent understanding of standards of care does not always exist.[156] The current baseline understanding of managing PU and DFU should be established. Health professionals should recognise the problem, know what to do and know how to operate within the health service. They must understand how best to educate their patients. Where gaps are identified, education should be provided and regularly updated. Health professional education must be informed and supported by guidelines on best practice developed in many countries and through a number of specialist national and international professional organisations. Where these guidelines are not already adopted, or require updating, health professionals should introduce relevant guidelines and education. The focus in many health systems is management of existing ulcers; a shift to prevention, supported by education of both patients and health professionals, would benefit patients, health professionals, and health-care systems and, importantly, be cost-effective.[157]

The UK National Minimal Skills Framework[158] covers health professional competencies required to manage foot diseases associated with diabetes. This is a good starting point for the basic competencies for DFU, including identifying risk status, basic foot care and advice, and managing a newly-presenting ulcer. It further details the higher level skill sets required for assessment of PAD, neuropathy, specialist education, advice on footwear, arranging surveillance based on risk status, tissue imaging management of Charcot foot and other skills.

NICE in the UK has also proposed a list of practitioner education topics for PU prevention.[107] Education includes: identifying patients at risk, recognising pressure damage, prevention, referral, conducting a risk assessment, repositioning, pressure redistribution devices, how to discuss prevention with patients and carers, and sources of help and advice

A comprehensive set of topics for patient education that the practitioner should be able to communicate to a patient with diabetes at risk of foot disease, and which the patient should expect to be told, is proposed by the Saskatchewan Ministry of Health in Canada.[111] The list, a good template for patient-focused education on self-management, suggests:

- Self-care and monitoring of diabetes
- The potential impact of diabetes on the feet
- Daily examination of feet and knowing when to seek advice from a health professional. Indicators include: any colour change; swelling; breaks in the skin; pain or numbness; alerting professionals, if self-care and monitoring is not possible or difficult
- Implications of loss of protective sensation
- Possible consequences of neglecting the feet
- Methods to help self-examination/monitoring, for example, the use of mirrors, if mobility is limited
- Hygiene (daily washing and careful drying)
- Skin care (moisturiser use)
- Nail care
- Dangers associated with inappropriate mechanical and chemical skin removal
- Footwear (the importance of well-fitting shoes and hosiery)
- Injury prevention and the importance of not walking barefoot when reduced sensation is present
- Annual foot examination by trained professional, to assess for neuropathy and vascular disease
- Prompt detection and management of any problems are important, and seeking help as soon as possible.

Societal

The general public tends to have at best incomplete

knowledge of medical matters, and at worst 'knowledge' gleaned from unreliable sources. The current media attention focused on multiple drug-resistant organisms is an example of how this can be addressed, although compelling evidence showing wider understanding and behaviour change is yet to be seen. October is Breast Cancer Awareness month and this is widely known—for a disease that is known by all and feared in equal measure and for which people are motivated and mobilised to raise funds through charity events. However, few people know that September is PAD Awareness month (It is also worth noting and publicising international days such as Stop the Pressure—15 November 2018 and World Diabetes Day—14 November 2018). These messages show how societal education perhaps should be tackled. The messages should raise awareness, understanding and societal support for patients suffering from non-healing wounds. The public should be made aware not only of the causes of PUs and DFUs and how their choices affect the causes, but also the impact non-healing wounds have on QoL, and life itself. A DFU, for example, is associated with increased risk of mortality.[159] Vehicles for achieving greater awareness are NGOs, religious organisations, the media. The impact that chronic wounds have in the UK was discussed in Parliament in 2017. The awareness that triggered the debate was raised by publications demonstrating the financial and organisational impact of non-healing wounds. Perhaps this is a model for the future.

Future research

Development of consensus guidelines for chronic wounds is based on available information on the underlying disease, the pathophysiology of ulceration and tissue breakdown, the influence of pathophysiology on recalcitrance, conversion of a chronic non-healing ulcer into a healing wound that will close and remain closed, the causes of recurrence, the influence of individual treatments, the efficacy of standards of care, and nutrition. Consensus opinions consider the quality of available data by examining the methodological strength of published studies, as well as consideration of real-world expert opinion based on experience. In so doing, gaps are identified in the evidence base, which are filled initially by expert opinion, but should be strengthened by methodologically sound studies. A number of gaps were identified by the expert panel convened to develop this document.

A considerable body of evidence has been amassed to show how chronic wounds form and this is largely understood at the tissue level for PU and DFU.[130,160–166] The role of PAD is also largely understood. The changes in skin due to diabetes before and after ulceration are described,[167] the impact of advanced glycation end products on inflammation has been described[168] and a possible role for *Staphylococcus aureus* has been identified.[169] Some genetic associations are becoming clearer.[170] A clearer understanding of the physiology of PU and DFU at the cellular level may help develop products targeted more effectively at the pathophysiology of these ulcers.

Nutrition in DFU: nutrition is a key component in standards of care for chronic wounds, but little is understood for DFU. The diabetic patient has metabolic challenges with glycaemic control and the full impact of the changes that happen in tissues of patients with diabetes that affect healing may not yet be fully elucidated.

Key points

- A number of gaps were identified by the expert panel convened to develop this document
- Nutrition is a key component in standards of care for chronic wounds, but little is understood for DFU
- This document should be regarded as a working document aimed to help health professionals make sense of a very challenging area.

Evidence to support advanced modalities for PU. Many wound-management technologies have not been subjected to rigorous high-quality randomised clinical trials (RCT). Where trials and evaluations have been conducted, often they are methodologically poor. When data from these studies are analysed using health technology assessment methodology, they are often found wanting, leaving interpretation of the clinical efficacy equivocal and supported by expert opinion. Patient care would be well-served by advanced technologies, with claims for effectiveness supported by high-quality, methodologically and statistically rigorous evidence.

This is a consensus document developed by an expert panel. The panel reached a consensus on differentiating between PU and DFU on the heel in particular. In so doing, it arrived at a series of recommendations that would ideally be implemented. However, the panel recognised that the recommendations in their entirety may not fit every health-care system, for a variety of reasons discussed in the document. These recommendations should therefore be used in line with local/national guidelines that are relevant to the reader's own country/area. This should be regarded as a working document aimed to help health professionals make sense of a very challenging area.

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