

University of Huddersfield Repository

Fletcher, Jacqui, Bader, Dan, Downie, Fiona, Dowsett, Caroline, Milne, Jeanette, Ousey, Karen and Schoonhoven, Lisette

Recognising, Managing and Preventing Deep Tissue Injury (DTI)

Original Citation

Fletcher, Jacqui, Bader, Dan, Downie, Fiona, Dowsett, Caroline, Milne, Jeanette, Ousey, Karen and Schoonhoven, Lisette (2017) Recognising, Managing and Preventing Deep Tissue Injury (DTI). Documentation. Wounds UK.

This version is available at http://eprints.hud.ac.uk/id/eprint/33948/

The University Repository is a digital collection of the research output of the University, available on Open Access. Copyright and Moral Rights for the items on this site are retained by the individual author and/or other copyright owners. Users may access full items free of charge; copies of full text items generally can be reproduced, displayed or performed and given to third parties in any format or medium for personal research or study, educational or not-for-profit purposes without prior permission or charge, provided:

- The authors, title and full bibliographic details is credited in any copy;
- A hyperlink and/or URL is included for the original metadata page; and
- The content is not changed in any way.

For more information, including our policy and submission procedure, please contact the Repository Team at: E.mailbox@hud.ac.uk.

http://eprints.hud.ac.uk/

CONSENSUS DOCUMENT 2017

Recognising, managing and preventing deep tissue injury (DTI)



PUBLISHED BY:

Wounds UK 1.01 Cargo Works 1-2 Hatfields London SE1 9PG, UK Tel: + 44 (0)20 3735 8244 Fax: +44 (0)800 242 5031 www.wounds-uk.com

Wounds uk

© Wounds UK 2017

This document has been developed by Wounds UK and supported by Mölnlycke Health Care



The views expressed are those of the authors and do not necessarily reflect those of Mölnlycke Health Care

Photographs in Figures 1, 8e, 8f, 8g and 11, and Box 3 are courtesy of Jacqui Fletcher Photograph in Figure 8a is copyright of Mid Yorks Hospital NHS Trust. Permission for publication granted Photographs in Figures 8b, 8c and 8d are courtesy of Fiona Downie and Philippa Clark Photograph in Figure 8h is courtesy of Anne-Marie Perrin

How to cite this document:

Wounds UK Consensus Document. *Recognising, managing and preventing deep tissue injury (DTI)*. London: Wounds UK, 2017. Available to download from: www.wounds-uk.com

EXPERT WORKING GROUP

Chair: Jacqui Fletcher, Independent Consultant, Bedfordshire Dan Bader, Professor of Bio-engineering and Tissue Health, Faculty of Health Sciences, University of Southampton Fiona Downie, Nurse Consultant Tissue Viability, Papworth Hospital NHS

Foundation Trust, Cambridgeshire Caroline Dowsett, Nurse Consultant Tissue Viability, East London

Foundation Trust

Jeanette Milne, Lead Nurse Tissue Viability, Northumbria Healthcare NHS Foundation Trust

Karen Ousey, Professor of Skin Integrity, University of Huddersfield; Adjunct Associate Professor, School of Nursing, Faculty of Health, Queensland University of Technology, Australia

Lisette Schoonhoven, Professor of Nursing, Faculty of Health Sciences, University of Southampton

GUIDE TO USING THIS DOCUMENT

This document was developed by the Expert Working Group with the objectives of:

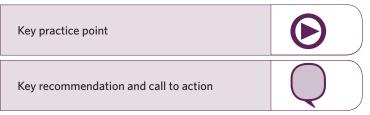
- Raising awareness and understanding of the diagnosis, management and prevention of deep tissue injuries (DTIs)
- Encouraging communication across all healthcare settings, including the community, where there are patients at increased risk of DTI.

In doing so, however, the Group recognises that much remains to be learnt about DTI. Development of the document began with a day-long meeting and was followed by extensive review of the resulting text before production of this final document.

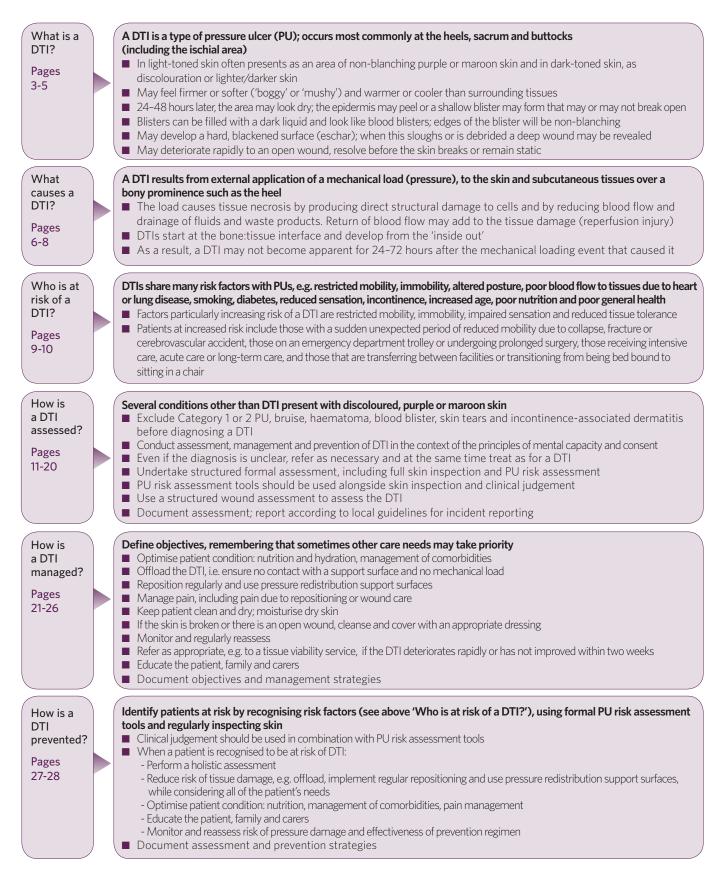
By explaining current understanding of the mechanisms involved in the development of DTI and describing how to recognise, treat and prevent DTI, the document aims to improve diagnosis, management, prevention and reporting of DTIs in the UK.

The chart 'Deep tissue injury (DTI) - an overview' (page 1) summarises the content and guidance provided in this document and links to the appropriate pages.

Key



Deep tissue injury (DTI) – an overview



The challenges of DTI

A deep tissue injury (DTI) is a type of subcutaneous tissue damage that results from an externally applied mechanical load (pressure). Recognition that DTIs have features distinct from pressure ulcers (PUs) led to DTIs being added in 2009 to the international classification of pressure ulcers (PUs) produced by the European Pressure Ulcer Advisory Panel and National Pressure Ulcer Advisory Panel (EPUAP/NPUAP, 2009). A DTI is not always associated with broken skin and does not always become the open wound often defined as a PU. However, DTIs do have the potential to develop into large, deep wounds with significant tissue loss that have serious consequences for patients and healthcare systems (Peart, 2016).

The formalisation of DTIs as a distinct category within PU classification is relatively recent. This, along with recent changes in PU terminology in some parts of the world (Box 1), and variations across the UK's healthcare systems in requirements for reporting and data collection, has caused confusion. It is likely that DTIs are under-reported because they are not always easy to recognise, particularly in the early stages, and may be miscategorised or not reported. In addition, some reporting systems do not include DTI or delay recording of the tissue damage until classification as one of the main PU categories is made.

Box 1. Terminology issues

Suspected deep tissue injury (sDTI) or deep tissue injury (DTI)?

When DTIs were first included in the international classification for PUs, the term DTI was preceded by the word suspected (abbreviated to sDTI) partly because of a lack of understanding of the condition and its relationship to other types of pressure-related damage (EPUAP/NPUAP, 2009). However, the pathophysiology of DTIs is now much better understood, and it is recognised that externally applied mechanical loads, such as pressure and shear, can give rise to DTIs and PUs in different ways (Oomens et al, 2015). For this reason, and because of other concerns over the use of the word, the recently updated PU classification produced by the NPUAP has dropped the 'suspected' preceding DTI (Edsberg et al, 2016).



The Expert Working Group also proposes that 'suspected' is omitted before DTI in the UK to remove any misunderstanding about the existence of DTI as a distinct condition and to encourage appropriate reporting, management and prevention

Pressure ulcer (PU) or pressure injury (PI)?

In 2016, the NPUAP decided to replace the term 'pressure ulcer' (PU) with 'pressure injury' (PI) (Edsberg et al, 2016). This brought NPUAP terminology in line with that used in countries covered by the Pan Pacific Pressure Injury Alliance (PPPIA) (Australia, Singapore, Hong Kong and New Zealand) (AWMA, 2012). Reasons for this change included the desire to emphasise both the preventable nature of many PUs and the fact that not all PUs represent open wounds. However, there are concerns with the use of the term 'injury' because it implies a very acute event leading to the tissue damage, even though it is known that the damage associated with PUs may take some time to develop (Bader & Schoonhoven, 2016; Ousey et al, 2017a). Indeed, the time element underpins the rationale for repositioning in the prevention of PUs. In addition, there are concerns that using the word 'injury' might be seen to imply that healthcare providers have caused the injury; a concern that has also been raised in the US (Mrdjenovich et al, 2016; NPUAP Position Statement, 2017). Finally, replacing the word 'ulcer' removes alignment with other wounds such as diabetic foot ulcers (DFUs) and venous leg ulcers (VLUs). This is particularly unfortunate in the case of DFUs, which often share some of the same aetiological mechanisms as PUs (Vowden & Vowden, 2015).



In common with a recent NHS Improvement consensus meeting that voted to use 'pressure ulcer' in England, the Expert Working Group recommends retaining the term 'pressure ulcer' (PU) in the UK

Defining DTI

Box 2. NPUAP/ EPUAP/PPPIA definition of suspected DTI (NPUAP/EPUAP/ PPPIA, 2014)

"Purple or maroon localized area of discolored 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 to adjacent tissue."

DTI is a type of subcutaneous tissue damage, affecting muscle where present, that originates close to bone and is the result of a mechanical load, such as pressure, applied to the skin. Box 2 contains the definition of DTI from the most recent (2014) NPUAP/EPUAP/PPPIA PU prevention and management guidelines. As noted in Box 1 (page 2), the Expert Working Group recommend using 'deep tissue injury' and omitting 'suspected'.

RECOGNISING DTI

The appearance of a DTI on presentation depends on when it is recognised in the time course of DTI development. As the name suggests, DTI starts deep within tissue and does not usually become apparent until about 24-72 hours after the event that caused the tissue damage (Black et al, 2016). Such events include lying immobile on a hard surface, e.g. on the floor after a fall that has resulted in unconsciousness or that is due to a stroke, or experiencing a period of poor tissue perfusion, e.g. during cardiothoracic surgery. In keeping with pain being a predictor of PUs (Smith et al, 2017), conscious and sensate patients may complain of pain before the appearance of the physical signs of DTI. See pages 9-10 for more detail on risk factors for DTI.

In patients with light-toned skin, DTI presents about 24–72 hours after initiation as a demarcated area of purple or maroon skin, often likened to a bruise. The purple or maroon area may be surrounded by an area of erythema (Figure 1). The discoloured area and erythema are non-blanchable (Box 3, page 4) (Black et al, 2016).

When palpated, the affected area may feel:

- Firmer or hardened (indurated) due to stiffening of dead or dying muscle tissue or
- Softened ('boggy' or 'mushy') as the damaged tissue starts to break down *and/or*
- Warmer or cooler than surrounding areas (Gefen, 2009).

In patients with dark-toned skin, the skin of affected areas may be darker or lighter, and palpation to detect changes in tissue texture or temperature is particularly important (Black et al, 2016). A retrospective review of patients with darkly pigmented skin found that the most commonly described presentation of DTIs was purple discolouration of intact skin (Sullivan, 2014).



Patients who are conscious and have sensation in the affected area may complain of discomfort or pain, and/or find palpation uncomfortable or painful

When assessing the temperature of the skin, it is important to consider whether the area has been in contact with or covered by bedding or clothing or has been exposed (i.e. it is warmer or cooler for extrinsic reasons), the ambient temperature, and the temperature of the assessor's hands.

Figure 1: DTI on side of foot The darkened purple/maroon area is surrounded by an area of non-blanchable erythema.





It is important to palpate the skin and subcutaneous tissues of any area that may have tissue damage

After a further 24–48 hours, the outer layer of the skin of the affected area may appear dry, start to peel or form a shallow blister (known as epidermal lift or sloughing). The full thickness of the skin may also lift or blister, and breaks in the skin can occur to reveal a purple, maroon, black or white wound bed (Black et al, 2016). In patients with dark-toned skin, blistering and skin breaks may be the first signs noticed of a DTI (Sullivan, 2014). Blisters may become filled with liquefied necrotic tissue and look like blood blisters.

Eventually a hard, blackened surface (eschar) may develop. When this finally sloughs or is debrided it will reveal the extent of the tissue damage and possibly a deep open wound. In some cases, sloughing of the eschar reveals intact or healed skin.



Although the appearance of DTI is often described as a bruise, it should be noted that a DTI does not go through the colour changes typical of a bruise as it resolves

NATURAL HISTORY AND EVOLUTION OF DTI

The potential evolution of a DTI is illustrated in Figure 2, page 5. Although full clarification of the natural history of DTIs and the factors that influence it is still to be established, it is clear that the outcome is variable. Some DTIs can deteriorate very rapidly to become large, deep open wounds despite best practice management. However, not all DTIs evolve or deteriorate. A proportion of DTIs will remain static for quite some time (anecdotally up to several months) or will resolve without forming an open wound (Richbourg et al, 2011).



In the experience of the Expert Working Group, when resolution occurs it is often within 7-10 days of diagnosis of the DTI

Currently, it is not known what proportion of DTIs resolve or whether there are indicators that could be used to predict which DTIs will resolve. A two-year study that followed 128 DTIs for an average of 6 days found that 37.5% of DTIs had resolved at the end of follow-up (Sullivan, 2013).

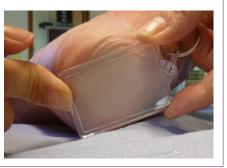


DTIs that resolve or remain static need continued care and monitoring to ensure the area is not exposed to further mechanical loading and to prevent repeated damage or extension of the injury

Box 3. Testing for non-blanchable erythema (NPUAP/EPUAP/PPPIA, 2014)

Gentle pressure is applied to the discoloured or reddened skin for three seconds using either a finger or a clear plastic device.

- a) Blanchable erythema application of pressure will push blood away from the area that has been pressed turning it paler than the surrounding skin (see photograph).
- **b)** Non-blanchable erythema if the blood flow to the area being tested has been damaged, the application of pressure will not make the skin become paler and it will remain discoloured or reddened. Non-blanchable erythema indicates tissue damage.







LOCATION OF DTI

An analysis of US data from a large international pressure ulcer prevalence survey undertaken in 2006–2009 found that DTIs accounted for about 9% of all PUs. The study also found that 41% of the DTIs occurred at the heel, 19% over the sacrum and 13% on the buttocks (VanGilder et al, 2010) (Figure 3).

A more recent analysis of hospital-acquired DTIs recorded over a two-year period (2010–2012) in one US hospital, reported the coccyx, heels and buttocks to be the most commonly affected sites. This study also reported DTIs to have occurred in relation to medical devices, and at the sacrum, intergluteal area, trochanter and ischium (Tescher et al, 2017).

The heels are thought to be particularly at risk of DTI because the underlying bony prominence (the calcaneus) has a small radius of curvature, i.e. is relatively pointed, and the layer of tissue overlying the bone is relatively thin (Salcido et al, 2011). This means that the intensity of a mechanical load applied to the heel can be high.

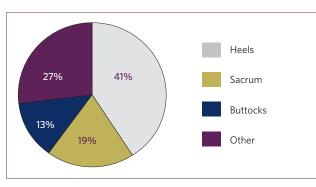
The location of a DTI will depend on the position of the patient. For example, in a patient who is sitting upright in a chair, DTI may occur under the ischial tuberosities, and in a patient who is sitting in bed with the head of bed elevated, DTI may occur over the sacrum. Similarly, a patient who has been lying on the floor after a fall or collapse due to acute illness may have evidence of damage at points that have been in contact with the floor. DTI may also occur under medical devices such as casts, splints, oxygen face masks or elastic bandaging (Tescher et al, 2017).



DTIs are most commonly found at the heels, sacrum and buttocks, but may occur in any location where prolonged pressure is applied, including under medical devices

As DTIs can occur at anatomical sites where PUs may occur, pressure-related damage, including any related to a medical device, should be carefully assessed to determine whether the damage is a DTI or a PU

Figure 3: Sites of DTI occurrence (VanGilder et al, 2010)

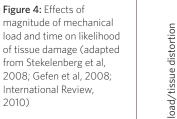


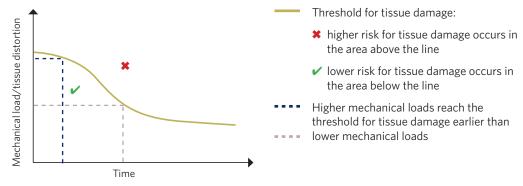
Causes of DTI

PUs and DTIs occur because of tissue damage caused by the application of a mechanical load (Box 4) to the skin, subcutaneous tissues and muscle. In general, higher mechanical loads will result in tissue damage more quickly than lower loads (Figure 4).

Box 4. Mechanical load, pressure and shear in the context of PUs and DTIs (International Review, 2010; Gefen et al, 2013)

- Mechanical load a generic term that covers all forces, including pressure and shear, applied to the skin and subcutaneous tissues, e.g. muscle
- Pressure results from the application of a force perpendicular (i.e. at right angles) to the surface of the skin. The pressure compresses the tissues and can distort or deform the skin, subcutaneous tissues and muscle. Tissue distortion is likely to be greatest when pressure is applied over a bony prominence
 Shear causes layers of body tissues to move relative to each other and may occur:
- a) Superficially e.g. as a result of a force applied parallel (tangentially) to the surface of the skin
 b) More deeply (internally) e.g. as the result of deformation of skin and muscle when pressure is applied over a bony prominence





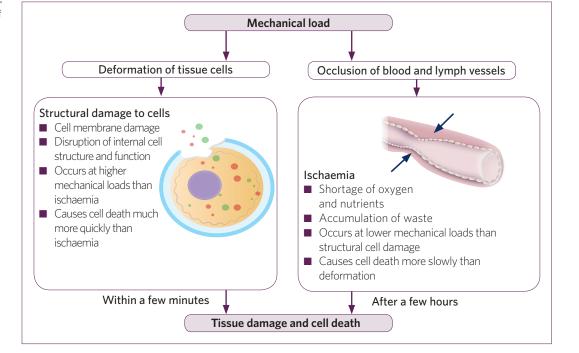
MAIN MECHANISMS

A mechanical load can have several effects that may ultimately damage tissue and cause tissue breakdown and cell necrosis (Figure 5, page 7). The two main mechanisms involved are:

- Structural damage The mechanical load has a direct effect on tissue cells, damaging cell membranes and disrupting internal cell structure to cause cell death. *In vitro* and animal studies have shown that this type of damage can occur within tens of minutes of application of the mechanical load and certainly much more rapidly than the damage caused by ischaemia (Gawlitta et al, 2007; Oomens et al, 2015).
- Ischaemia The mechanical load can compress capillaries causing partial or complete occlusion. This reduces the delivery of oxygen and nutrients to tissues. If the rate of delivery is below the physiological demands of the tissues, ischaemia will occur. If the ischaemia is sustained for long enough and/or at high enough levels, tissue death can occur due to a change in metabolism and the accumulation of waste products. Generally, ischaemia takes a few hours to produce tissue damage, and muscle is more susceptible to ischaemia than skin (Agam & Gefen, 2007; Oomens et al, 2015).

The amount of tissue deformation, i.e. the degree of change in tissue shape in response to the mechanical load, required to produce ischaemia is lower than that required for structural damage (Oomens et al, 2015). However, the threshold at which damage occurs from either mechanism will vary from patient to patient and will depend on numerous factors, e.g. the presence of comorbidities (such as cardiovascular disease and diabetes) that may reduce tissue perfusion and the condition and thickness of the tissue layers between the skin and bony prominence (Oomens et al, 2015). A combination of reduced tissue perfusion and less tissue between the skin and bony prominence may result in DTI particularly quickly.

Figure 5: Main mechanisms of tissue damage and cell death due to mechanical loading in the development of DTIs and PUs (adapted from Oomens et al, 2015)

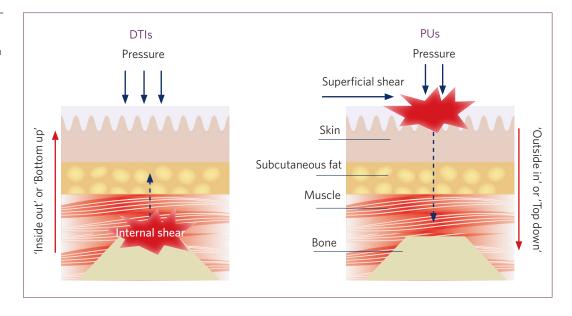


OTHER EFFECTS INVOLVED

Two other mechanisms to consider in the aetiology of DTIs and PUs are:

- Lymphatic occlusion In addition to occluding blood vessels, mechanical loads can occlude lymphatic vessels and may cause the accumulation of metabolic waste products that contribute to tissue damage (Miller & Seale, 1981; Peart, 2016).
- Ischaemia-reperfusion injury Although counterintuitive, restoration of blood flow to an ischaemic tissue can exacerbate ischaemia-induced damage. Reperfusion initiates an inflammatory response that stimulates the formation of oxygen-derived free radicals and other agents that can cause further tissue damage (Collard & Gelman, 2001). An animal study of DTI found that reperfusion reversed tissue damage after a limited time of ischaemia, but that if ischaemia was prolonged, reperfusion exacerbated the existing tissue damage (Loerakker et al, 2011). Patients who experience hypotension, whether as a consequence of disease or as a result of other treatments, may be particularly prone to the effects of ischaemia-reperfusion injury.

Figure 6: Differences in the sites of development of DTIs and PUs (adapted from WUWHS, 2016) This diagram illustrates the different sites of development of DTIs and PUs, and the relative importance of types of mechanical loading in the development of each.



DIFFERENCES IN THE DEVELOPMENT OF DTIs AND PUs

The differences in clinical progression of DTIs and PUs suggest that although the underlying causes are similar, the initial site of damage is different. In DTIs, mechanical loading in the form of externally applied pressure causes tissue damage that occurs initially adjacent to the bony prominence and in the muscle layer if present (Figure 6). The damage then moves towards the surface in a manner sometimes described as 'bottom up' or 'inside out'.

Conversely, in PUs, the mechanical load comprises pressure with superficial shear. These loads cause tissue damage initially at the surface; the damage works downwards, or 'top down' or 'outside in' (Fife, date unknown; WUWHS, 2016).

In another analogy, DTIs have been likened to geological sinkholes. This analogy is also useful because it is in keeping with the frequent finding in DTIs of undermining due to tissue damage that is more extensive than initially apparent from the skin surface. In a similar analogy, PUs have been likened to potholes in a road (WUWHS, 2016).

Although a clear distinction has been made here to aid understanding of the differences in the development of DTIs and PUs, in clinical practice it is possible that the mechanisms sometimes occur together in the development of an area of pressure-induced damage. For example, a patient who has been immobile may start to develop a DTI, but then may regain some movement that introduces superficial shear which also contributes to the tissue damage.



The facts that DTIs can occur very quickly and start below the skin surface (often in muscle) suggest that high mechanical loads may be involved and that structural damage to cells is a major cause of tissue necrosis. These facts reinforce the need for timely risk assessment and frequent skin inspection

Who is at risk of DTI?

An individual's risk of developing a DTI or a PU is dependent on a complicated interplay of numerous factors (Coleman et al, 2013). At a basic level, the risk is related to the intrinsic ability of the patient's tissues to withstand the effects of extrinsic risk factors such as externally applied mechanical loads, i.e. to a level of tissue tolerance. When the effects of the extrinsic risk factors exceed the specific tissue tolerance, the individual is at an increased risk of developing a DTI or PU (WUWHS, 2016). Tolerance to extrinsic risk factors will be reduced by a range of intrinsic risk factors (Box 5, page 10) such as immobility, reduced tissue perfusion and reduced sensation.

FACTORS THAT INCREASE THE RISK OF DTI

PUs and DTIs share many risk factors, but there are some risk factors that particularly increase the risk of a DTI. These can be divided into factors that restrict immobility or cause immobility, impair sensation, and reduce tissue tolerance (Figure 7, below; and Table 1, page 10).

In some conditions, several of these factors may be present and contribute to increased risk of DTI. For example, individuals with spinal cord injury can have reduced mobility, impaired sensation, hypotension and muscle atrophy (wasting) and so are at an increased risk of DTI (Gefen, 2014). A recent analysis of factors contributing to DTI in intensive care unit patients reported that the odds of developing a DTI increased by 20% for every hour increase in length of surgery (Kirkland-Kyhn et al, 2017).



Patients in intensive care units (ICUs), undergoing surgery, receiving acute care in hospital or in long-term care are at increased risk of DTI (Fleck, 2007)

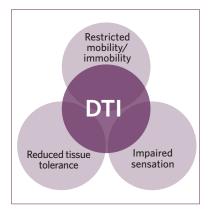


Patients undergoing transitions in care, e.g. when being transferred from one facility to another (Preston et al, 2017), can be at an increased risk of DTI, particularly if not placed on an appropriate support surface during transfer or in the receiving clinical area

Patients undergoing other transitions, e.g. from being bed-bound to sitting in a chair, are also at an increased risk of DTI because the change in position will change the ways and the time period that mechanical loads are applied to the tissues and because the support surface may not be appropriate

Patients that have had a DTI may be at increased risk of the development of further DTIs or PUs because during the healing process the tissues are likely to have been remodelled, e.g. with scar tissue, and to have altered physical characteristics.

Figure 7: Categories of factors that increase risk of DTI



Box 5. Main intrinsic risk factors for PU and DTI (AWMA, 2012; Coleman et al, 2013; Coleman et al, 2014)

Major intrinsic risk factors

- Immobility/reduced activity/altered posture e.g. bed-fast, chair-fast, contractures
 Reduced tissue perfusion e.g. due to peripheral vascular disease, cardiopulmonary disease,
- diabetes, smoking, critical illness
- Previous or existing PU

Other important intrinsic risk factors

- Reduced sensation e.g. spinal cord injury, stroke, local or general anaesthesia
- Increased skin moisture e.g. incontinence, perspiration
- Increased age
- Anaemia
- Poor nutrition, including low blood albumin levels
- Reduced general health status e.g. acute illness, renal impairment, carcinoma

	Limited mobility or immobility	Impaired sensation	Reduced tissue tolerance	
Effect of risk factor	The patient has reduced ability to change position independently and relieve pressure	The patient has reduced ability to detect signals from body tissues indicating that a change in position is required to relieve pressure	The threshold for tissue damage induced by mechanical loading is reduced	
Examples of risk factors	 Neurological disease, e.g. stroke, s Sedation Anaesthesia, e.g. local, epidural, sp Unconsciousness 		 Atrophy, fragility and/ or breakdown of soft tissues, e.g. muscle atrophy through acute 	
	 Prolonged sitting, e.g. in a chair, wheelchair, hard surfaces, such as toilet seats or washroom stools In pain, e.g. from chronic disease, recent surgery Prolonged confinement to a hard surface, e.g. the floor (after a fall or loss of consciousness), an emergency department trolley, radiology department surfaces, spinal board, operating theatre table or during transfer between healthcare facilities Prolonged contact with and usage of medical devices 	Neuropathy, e.g. due to diabetes, alcoholism, autoimmune disease, Guillain- Barré syndrome/ chronic inflammatory demyelinating polyneuropathy, malnutrition	, 0	

Diagnosis of DTI

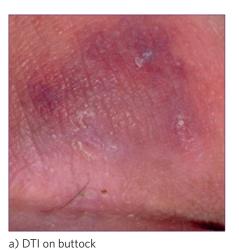
Several conditions other than DTI can present with discoloured, purple or maroon skin, skin loss over a darkened wound bed, or rapidly developing eschar (Black et al, 2016). Healthcare professionals and carers need to be aware of the characteristics of DTI and how to differentiate it from other conditions that may present in a similar way (Table 2 below, and Appendix 1, page 30). Taking a detailed history from the patient and careful assessment will be key to establishing the correct diagnosis.



If there is uncertainty over the diagnosis of a lesion that may be a DTI, the affected area should be treated as if it is a DTI (see pages 21-26) while the concern is escalated or a referral is made to the relevant service. The urgency of the referral should be appropriate to the possible diagnosis, e.g. necrotising fasciitis is life-threatening and requires emergency referral

Table 2. Distinguishing DTI from other conditions that present with discoloured (purple or maroon) skin +/- broken skin
(Nair et al, 2014; NPUAP/EPUAP/PPPIA, 2014; Pieper, 2016; Black et al, 2016; Ousey et al, 2017b)ConditionCharacteristics

Condition	Characteristics	
DTI (Figures 8a and 8b, page 12)	 History of application of pressure over affected area Preceding event may have been 24-72 hours before recognition Presents as a purple or maroon area of non-blanchable discoloured intact skin which may be surrounded by non-blanchable erythema (Box 3, page 4) +/- an overlying area of dry skin, a thin blister over a dark wound bed, a blood-filled blister, an area of broken skin with a purple, maroon, black or white wound bed, or an area of thin dark eschar (see pages 3-4) Affected area may be painful, and firmer/softer, or warmer/cooler than the surrounding areas Usually has a clearly defined edge Most commonly affects the heels, sacrum and buttocks; may occur under a medical device 	
Category 1 PU (Figure 8c, page 12)	 History of application of pressure over affected area Skin of the affected area is intact with non-blanchable reddening (erythema) or discolouration in darkly pigmented skin, and is usually over a bony prominence Affected area may be painful, and firmer/softer, or warmer/cooler than the surrounding areas 	
Category 2 PU (Figure 8d, page 12)	 History of application of pressure over affected area Presents as a shallow shiny or dry open ulcer due to partial thickness loss of the dermis without slough or bruising, and is usually over a bony prominence May also present as an intact or open/ruptured serum-filled blister 	
Bruise (Figure 8e, page 12)	 History of blunt-force trauma is common Due to extravasation of blood into tissues Usually takes about two weeks to heal 	
Haematoma	 History of trauma is common Due to extravasation of blood; may produce a deep-seated palpable nodule Morel-Lavallée lesions are a type of haematoma that are due to trauma that separates the subcutaneous tissues from the underlying fascia and muscle; skin necrosis and infection may occur 	
Blood blister	 History of trauma is common, e.g. pinching of tissue between two hard surfaces Forms a raised blister that is filled with blood; may be red initially and then become purplish or black 	
Skin tear (Figure 8f, page 12)	 History of trauma is common Common on the extremities of older people May cause full- or partial-thickness skin loss Often accompanied by bleeding 	
Incontinence-associated dermatitis (Figure 8g, page 12)	 Usually associated with faecal and/or urinary incontinence Affects perineum, peri-genital area, buttocks, gluteal fold, upper thighs and lower back Affected area often has poorly defined edges Erythema is blanchable and may be blotchy, and may be accompanied by partial thickness skin loss and vesicles 	
Venous engorgement (Figure 8h, page 12)	 Can occur in dependent tissues in patients with poor cardiac output; may be associated with swelling due to oedema The colour of the affected area can change if moved, e.g. during elevation or position change; discolouration is blanchable 	
Arterial insufficiency	 Skin may be pale or mottled red to bluish/purple colour, dry and hairless Often bilateral; patient may keep the limb(s) in a dependent position and complain of numbness, burning sensation and pain Slow capillary refill and pallor on elevation (Box 6, page 13); pulses in an affected limb may not be palpable Embolic events: small blood clots may cause sudden onset of ischaemia with pain and discolouration of the affected tissues, e.g. in the toes 	





b) Sacral DTI



c) Category 1 PU



d) Category 2 PU with blistering



e) Bruise



f) Skin tear



g) Incontinence-associated dermatitis



Figure 8: DTI and other conditions that may have a similar presentation See Table 2, page 11, for more information.

Overview of assessment, management and prevention

The principles underlying the assessment, management and prevention of DTIs are similar to those for the assessment, management and prevention of PUs (Preston et al, 2017).



The assessment, management and prevention of DTIs should be undertaken in the context of mental capacity and consent (Box 7)

FORMAL CARER, INFORMAL CARER OR PATIENT

For patients in community settings, e.g. in their own homes or in nursing homes, signs and symptoms of DTI may first be recognised by him- or herself, or by a formal or informal carer. The patient or carer noticing such signs should contact a registered healthcare professional as soon as possible, and in the meantime should:

- Protect the affected area:
 - Make sure the patient does not sit or lie on the affected area and that nothing presses on it
 - Cover broken skin or blistering with an appropriate low-adherent dressing as per local dressing formulary, if available; if not available, leave the broken or blistered area exposed and avoid touching it
- Prevent any additional areas of pressure-induced damage:
 - Continue any other PU prevention measures
 - Keep the patient clean and dry
- Encourage healthy eating and fluid intake (Figure 9, page 14).

Box 7. Mental capacity and consent (Department of Health, 2009; Mughal, 2014; Nichols, 2014)

- Valid consent is required before touching a patient, whether that is to assist with self-care tasks such as dressing, to examine skin as part of PU prevention, or to undertake invasive procedures
- Valid consent requires that the patient has the mental capacity to give consent, i.e. that the patient can understand and use information to make a decision
- The Mental Capacity Act 2005 covers adults aged over 16 years in England and Wales and sets out criteria for establishing mental capacity. The Act has five main principles:
 - Assume the patient has mental capacity unless it is proven otherwise by undertaking a mental capacity assessment
 - Ensure all practical steps have been taken to support the patient with the assessment before deciding they lack mental capacity
 - An unwise decision does not mean that the patient lacks mental capacity
 - Any decision made on behalf of a patient lacking mental capacity must be made in his/her best interests
 - Always consider whether there is a least restrictive option when making any best-interests decision
- A patient who has mental capacity should be provided with sufficient information in a way that they can understand to inform their decision about consent, such as the options available, the risks and benefits, and the implications of their choices
- Any decision made in relation to a patient who lacks capacity should be made in that person's best interests
- The issues of capacity and consent are covered in Scotland by the Incapacity (Scotland) Act (2000) (Scottish Government, 2000) and in Northern Ireland by the Mental Capacity Act (Northern Ireland) 2016 (TSO, 2016)
- Healthcare professionals and carers should follow the consent policies and procedures of their organisation

Box 6. Clinical examination to test for arterial insufficiency

- Elevation of a limb affected by arterial insufficiency may cause pallor
- In the lower limb, Buerger's test can be used in assessment for lower limb ischaemia (Insall et al, 1989). For example, in the lower limb, elevation of the leg to an angle of 45 degrees to the bed surface for one to two minutes when the patient is lying on his/her back will cause pallor of the skin; then when the patient sits with the leg lowered over the edge of the bed, the skin may become bluish and then reddened

 If arterial insufficiency is suspected, consider determining ABPI (ankle-brachial pressure index) and/ or referral to a vascular service

REGISTERED HEALTHCARE PROFESSIONAL/ALLIED HEALTHCARE PROFESSIONAL

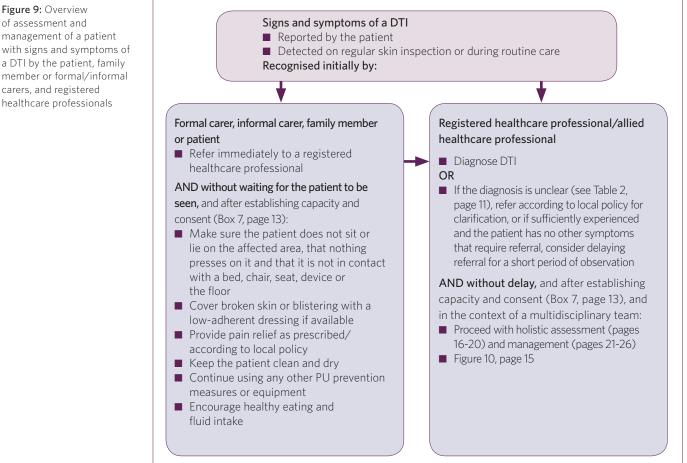
The diagnosis of a DTI may be clear on presentation, but referral as appropriate, e.g. to a tissue viability service, may be needed if it is not (Table 2, page 11). Experienced healthcare professionals aware of the evolution of DTIs may decide to delay referral for a short period (e.g. up to 1-2 weeks) if the area appears stable, and the patient has no other symptoms of illness that warrant onward referral. In such cases, assessment and management should be commenced as if for a DTI alongside frequent monitoring to see whether the diagnosis becomes more evident (Figure 10, page 15).



It is important that an area showing signs and symptoms of a DTI is immediately protected from any further mechanical load even when the diagnosis is not clear; do not wait for a referral to be completed or for confirmation of the diagnosis

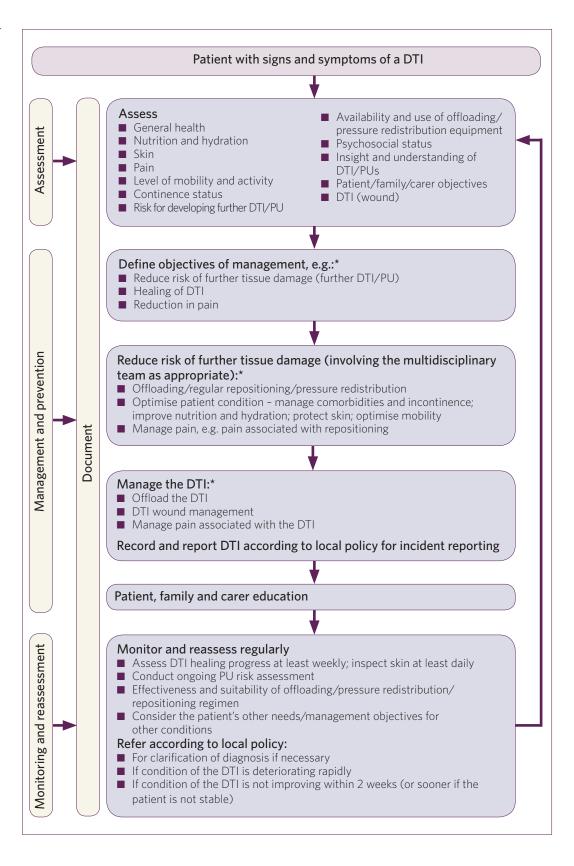
Prevention of DTI should generally adopt the same principles as the management of DTI. A focus of management of DTI is the prevention of further tissue damage (Preston et al, 2017). Information on the:

- **Assessment** of a patient with signs and symptoms of a DTI is on pages 16-20
- **Management** of a patient with signs and symptoms of a DTI is on pages 21-26
- **Prevention** of DTI is on pages 27-28.



of assessment and management of a patient with signs and symptoms of a DTI by the patient, family member or formal/informal carers, and registered healthcare professionals

Figure 10: Principles of assessment, management and prevention of DTI



*Taking into account all of the patient's needs

Assessment of DTI

A patient with a DTI should undergo a structured holistic assessment that includes:

- The patient's overall health, including comorbidities, nutrition and skin condition
- The DTI
- Identification of the risk factors for DTI and PUs (Figure 10, page 15).



A holistic and structured assessment of a patient with a DTI will provide essential information for devising and implementing an appropriate management plan. The results of the holistic assessment must be documented according to local policy (Chamanga & Ward, 2015)

GENERAL HEALTH STATUS

Assessment of general health should include:

- Medical and surgical history may reveal risk factors for the development of pressure-induced damage, e.g. stroke, diabetes
- History of previous PU or DTI may indicate increased risk
- **Current health** active comorbidities; new symptoms that may need investigation
- Medication e.g. medication that causes sedation may increase risk of DTI, or chronic use of steroids may impair wound healing
- Factors that may adversely affect healing (Box 8, page 17).



Ascertainment of general health status will allow proposed management (and prevention) of the DTI to be placed in context. In some situations, e.g. in critical illness or end of life care, the other needs of the patient may take priority over DTI prevention and management. Where this is the case, such decisions should be clearly documented

NUTRITION

Poor nutritional status and inadequate dietary intake are risk factors for PU development and impaired wound healing (NICE, 2014; Posthauer et al, 2015). Patients should be assessed for recent weight loss and problems associated with eating and drinking. The Malnutrition Universal Screening Tool (MUST) is an example of a screening tool cited by the National Institute for Health and Care Excellence (NICE, 2006; Elia, 2003).



Patients suspected of having poor nutrition should undergo nutritional assessment according to local policy, which may include referral to a dietician. Patients with difficulty eating or drinking may also benefit from assessment by an occupational therapist

SKIN ASSESSMENT

Skin assessment should ensure examination of the patient's entire skin for signs of pressure-induced damage, e.g. areas of non-blanchable erythema (Box 3, page 4), including under medical devices, such as face masks, and for general condition. Dry skin, particularly at the heels, is a risk factor for pressure-related damage (Lechner et al, 2017).



Checking for evidence of pressure-induced damage should involve inspecting and palpating the skin, and asking the patient about any areas of discomfort or pain: 'look, feel, ask'

Careful attention should be paid to inspection of areas in which the patient is complaining of pain. If the area cannot be accessed easily for inspection, e.g. because of difficulties with or uncertainty over the removal of a device or bandage, the concern should be escalated as soon as possible. After removal of the device and skin inspection, careful consideration may be required of the suitability of reapplication of the device and whether there is need for an alternative.

Heels are the most common site of DTI, but may be difficult to access. Particular effort may be needed to ensure that any foot coverings (e.g. slippers, shoes, socks, tights, anti-embolic stockings) are removed regularly, e.g. at least daily or more frequently in patients at high risk, to allow examination.

Patients who are incontinent or who had been confined to the floor after falling may have signs of incontinence-associated dermatitis (Table 2, page 11).

All of the patient's skin should be examined: patients may have more than one DTI (Figure 11), also have a PU or a skin condition that increases risk of further pressure-induced damage

lacksquare

Box 8. General factors that may impair wound healing (Guo & DiPietro, 2010; Anderson & Hamm, 2012)

- Extremes of age
- DiabetesPeripheral arterial
- disease
- Severe liver or kidney disease
- Obesity
- Immunosuppression – e.g. radiation therapy, AIDS
- Medication e.g. steroids, immunosuppressants, chemotherapy, non-steroidal anti-inflammatory drugs (NSAIDs)
 Smoking
- Alcoholism
- Alconol
 Stress
- All Malnutrition



Figure 11: Multiple areas of DTI and pressure-induced damage

Skin assessment provides an opportunity to talk to the patient about risk for DTIs and to provide advice on avoidance of pressure-induced damage and the signs and symptoms if it does occur.

PAIN ASSESSMENT

Assessment of pain is important because:

- DTIs can cause pain
- Pain may occur before physical signs of pressure-related damage occur
- In a DTI with an open wound, a sudden onset or an increase in pain may indicate infection
- Pain and psychological stress can adversely affect wound healing
- Pain occurring during movement may reduce mobility, compounding risk for further pressureinduced damage (Gorecki et al, 2009; Woo, 2012; Black et al, 2016).

Repeated use of the same method of assessment will assist in monitoring pain and the effectiveness of pain-relieving interventions. The method of pain assessment should be selected according to the patient's cognitive and linguistic abilities. In patients with reduced cognitive ability, non-verbal cues, body language and the family/carers' knowledge of the patient may aid assessment of pain (RNAO, 2016).

Patients should be assessed for DTI-related pain and non-DTI-related pain. Assessment should include evaluating the location, nature, frequency, timing, and severity of pain (Box 9) and factors that initiate or relieve pain

Box 9. Examples of scales for assessment of pain severity (Solowiej & Upton, 2010)

- Verbal pain rating scale the patient indicates from a list of phrases ranging from 'no pain' to 'severe pain' which phrase describes their pain best
- Numerical pain rating scale the patient is asked to score their pain on a 0 to 10 scale, where 0 = no pain and 10 = the worst pain imaginable
- Visual analogue scale the patient is presented with a line, one end of which represents no pain and the other end the worst pain imaginable, and is asked to draw a mark on the line to represent the level of pain

LEVEL OF MOBILITY AND ACTIVITY

A central principle of the management (and prevention) of DTI is removal of any mechanical load from the affected area and any other areas at risk of pressure-induced damage. A patient who is able to perform purposeful movements, i.e. is able to lift or tilt a body part completely away from the surface they are resting on, can be encouraged to change position regularly as part of a pressure-relieving regimen.

RISK FOR FURTHER PRESSURE-INDUCED DAMAGE (DTI OR PU)

A patient with a DTI is at risk of developing a further DTI or a PU. An understanding of the risk factors for DTI and PUs (Box 5 and Table 1, page 10) will enable healthcare professionals, carers, patients and family members to quickly identify which risk factors are relevant to the patient. This should include understanding that development of an acute illness in a patient already at risk of pressure-related damage will further increase risk. Consideration should also be given to all aspects of care that may be associated with risk, e.g. prolonged sitting without position change in a chair or on a toilet (Lustig et al, 2017).



The patient's level of mobility, along with identification of DTI or PU risk factors and aspects of care that may be associated with risk for pressure-induced damage can be used to inform the management plan

PU risk assessment tools for different patient groups are available and should be used according to local policy (Table 3). Currently, there is no risk assessment tool specific to DTIs.

SUPPORT SURFACES AND PRESSURE REDISTRIBUTION EQUIPMENT

All support surfaces and pressure redistribution equipment in use by the patient, including its physical condition, suitability and whether use is appropriate and safe, should be assessed (RNAO, 2016). Examples of equipment to consider include bed, chair/wheelchair, toilet seat, bathing stool, foot rest, transfer equipment, and interventional medical device.

OTHER

- Psychosocial status including for patients in the community, the formal and informal care systems that may be in place.
- Understanding of DTI and PUs ascertaining the level of insight into DTI and PUs of the patient, family, and, where appropriate, the carer may reveal the need for education to aid understanding of management strategies and involvement in care planning.

Table 3. PU risk assessment tools (Wounds UK, 2013a; Fletcher, 2017)		
Patient population	Appropriate risk assessment tool	
Adults	 Acute care: Waterlow Score (Waterlow, 2005); PURPOSE T (Coleman et al, 2017) Critical care: Jackson/Cubbin Pressure Area Risk Calculator (Jackson, 1999) Community care: Walsall Community Pressure Sore Risk Calculator (Chaloner & Franks, 2000) Orthopaedic: Pressure Sore Prevention Score (Lowthian, 1989) 	
Older people	 Norton Pressure Ulcer Risk-Assessment Scale (Norton, 1989) Braden Scale for Predicting Pressure Sore Risk (Bergstrom & Braden, 1992) 	
Paediatric patients	 Glamorgan Paediatric Pressure Ulcer Risk Assessment Scale (Willock et al, 2009) Braden Q Scale (Curley et al, 2003) 	

DTI ASSESSMENT

Assessment of a DTI will provide a baseline from which to monitor progress or detect deterioration. The assessment should be clearly documented according to local policy. Serial photographs may be helpful and should be obtained after gaining patient consent and stored according to local policy (Farid et al, 2014; Sperring & Baker, 2014).

The assessment should include, as a minimum, the location of the DTI and measurement of the size of the affected area and blistering or open wound if present. Systematic assessment of the DTI will be aided by using a wound assessment framework (Box 10), even when the skin over the DTI remains intact.

If the skin is broken, the wound bed should be assessed for the proportions of granulation tissue, necrotic tissue/eschar, slough and epithelial tissue present (Ousey & Cook, 2012). Exudate levels, though difficult to assess, influence dressing selection. Exudate consistency, colour and smell may also indicate infection (Wounds UK, 2013b). See Box 11 for a summary of signs of possible local wound infection. If bone can be probed in the base of the wound or is exposed, osteomyelitis should be suspected (Rennert et al, 2009). Systemic signs of wound infection include malaise and pyrexia.



In patients with more than one DTI, or a DTI and another wound or area of skin damage such as incontinence-associated dermatitis, each area of damage or wound should be individually assessed and categorised to facilitate appropriate care planning and management

Box 10. Frameworks to aid systematic wound assessment

- TIME(S) (Schultz et al, 2004; Wounds UK, 2016)
 - **T**issue
 - Infection/inflammation
 - Moisture imbalance
 - Edge of the wound
 - (**S**urrounding skin)
- Triangle of Wound Assessment (Dowsett et al, 2015)
 - Wound bed
 - Wound edge
 Periwound skin
 - Periwound skin

Box 11. Signs and symptoms of possible wound infection in a DTI with broken skin or an open wound (WUWHS, 2008)

- New, increased, or altered pain
- Malodour or change in odour
- Increased or altered/purulent exudate
- Delayed healing
- Periwound oedema
- Bleeding or easily damaged granulation tissue
- Altered wound bed colour
- Induration of periwound skin
- Pocketing and bridging

The Expert Working Group recommends that a DTI remains recorded as a DTI, and is not recategorised at any point (e.g. to a Category 3 or 4 PU), even if it evolves

The Expert Working Group recommends that the notes of a patient with a DTI are flagged to alert all healthcare professionals because of the likely increase in risk for further pressure-induced damage

DIAGNOSTIC TESTS FOR DTI

The difficulties of identifying the early stages of DTI have generated interest in the potential of tests that may indicate the presence of tissue damage. Tests under investigation for the detection of pressure-induced damage include assessing biomarkers in plasma, sweat and urine related to muscle damage and changes in concentrations of inflammatory mediators (Ferguson-Pell & Hagisawa, 1988; Knight et al, 2001; Loerakker et al, 2012; de Wert et al, 2015; Krishnan et al, 2016; Worsley et al, 2016).

Ultrasound scanning (USS) can detect subcutaneous tissue changes in DTI prior to changes becoming visible on the skin surface (Scheiner et al, 2017). Therefore, USS has potential as a screening tool in patients considered to be at risk. However, the clinical utility of USS for this indication is likely to be limited because of the resource, training, time and cost implications of widening access to USS.

In areas of pressure-induced damage, the amount of interstitial fluid increases in response to the inflammatory process. As a result, hand-held devices to measure subepidermal moisture (SEM) have been developed to aid early identification of PUs (Moore et al, 2016). Clinical studies have reported that SEM values increase with increasing tissue damage (Oliveira et al, 2017).

Thermography has been investigated to assess its ability to predict progression of discoloured intact skin to necrosis (Cox et al, 2016). The study found that intact discoloured areas with cooler skin temperatures in the centre in comparison with surrounding skin were significantly more likely to develop necrosis by day 7 than if the skin was warmer than the surrounding areas. However, nurses participating in the study were unsure of the feasibility of using thermography in clinical practice and more investigation is required.



Currently, there is no widely accessible and reliable test to aid diagnosis of DTI

A DTI may be subject to reporting and investigation according to local policy

Management of DTI

Management of DTI:

 Treatment of the local area of tissue damage

PLUS

Prevention of DTI: ■ Measures to

prevent further pressure-induced damage

Figure 12: Management of DTI includes prevention



The management and prevention of a DTI share many similarities. In fact, prevention strategies are used in DTI management to aid healing and prevent deterioration of the DTI and to prevent any further pressure-induced damage (Figure 12). Management of DTI therefore comprises local treatment of the DTI plus DTI prevention. Prevention of DTI is discussed on pages 27-28.

The assessment of the patient with a DTI will indicate appropriate objectives that will form the basis of the management plan. The plan needs to take into consideration all the patient's needs. In some cases, the DTI aspects of the plan may need to be adjusted or take lower priority.

The objectives and plan should be discussed and agreed where possible with the patient, family and carers. Management of DTI is likely to require a multidisciplinary team approach, which may also include as appropriate nurses, tissue viability nurses, healthcare assistants, physiotherapists, occupational therapists, podiatrists, dieticians, medical specialists, surgeons, bioengineers and social workers (RNAO, 2016).

Patients with a DTI often have other conditions. In some situations, e.g. in patients who are haemodynamically unstable, the management of these conditions needs to take priority over, or may complicate, the management (and prevention) of DTIs or PUs. In such cases, this may mean that it is necessary to recognise that the DTI is likely to evolve and deteriorate

OPTIMISE PATIENT CONDITION AND NUTRITION

Optimising the patient's condition aims to remove or ameliorate any modifiable risk factors for pressure-induced damage, e.g. comorbidities or incontinence, and any factors that may impede healing identified during assessment.

Patients should be encouraged to eat healthily and to maintain good levels of hydration. If a patient is unable to achieve an adequate nutritional intake with meals, fortified foods and/or high protein oral nutritional supplements or enteral/parenteral nutritional support as appropriate should be considered (NPUAP/EPUAP/PPPIA, 2014).

OFFLOADING, PRESSURE REDISTRIBUTION AND REPOSITIONING

As mechanical loading, mainly in the form of pressure, is the main cause of DTI, it is logical to remove pressure from the affected area and, as far as possible, reduce the intensity and duration of pressure over other areas that may be at risk of pressure-induced damage.



Wherever possible, it is important to offload a DTI and avoid exposing it to any form of mechanical load

In an at-risk patient who reports pain in an area in which a DTI may occur, but who has no other signs of DTI, the affected area should be offloaded if possible and monitored very frequently

A reduction in mechanical load can be:

- **Partial** i.e. produce a reduction in mechanical load, known as pressure redistribution; this may be intermittent or continuous
- **Complete** i.e. totally remove mechanical load, known as offloading; this may be intermittent or continuous.

In practice, regular repositioning, which is used to offload areas, and equipment, which may redistribute pressure or offload, are often used in combination. Table 4, page 22, provides information on offloading according to the most common locations of DTI.

Location of DTI	Tips on repositioning/offloading/pressure redistribution
Heel	 Reposition the patient to offload pressure from the heels when sitting or lying by ensuring they are not in contact with the bed, floor, foot rest etc, i.e. the heels are 'suspended' or 'floating' Devices that can be used to aid heel offloading: Boot - air, gel, foam, fibre Heel zone mattress Pillow or cushion (Figures 13 and 14, pages 24 and 25) Silicone cup Trough Wedge Rigid framed devices may be needed for ambulant patients On intact skin, consider use of a dressing designed to reduce exposure of the DTI to mechanical loads* (see page 26 for the management of DTI with broken skin or an open wound)
Sacrum/ ischial tuberosity	 Reposition the patient to offload the sacrum/ischial tuberosities when lying or ask the patient to reposition themselves if possible and safe to do so In bed, use the 30-degree tilt side-lying position (Figure 14, page 25) and avoid the patient lying on their back (supine) Minimise time seated; avoid sitting patients with an ischial DTI in a fully erect posture; patients should be encouraged to offload intermittently while sitting, e.g. by lifting themselves completely off the seat, sideways leans, forward tilts and leans, and standing On intact skin, consider use of a dressing designed to reduce exposure of the DTI to mechanical loads* (see page 26 for the management of DTI with broken skin or an open wound)
Under a medical device	 Where possible, change the location or shape of the medical device, e.g. for oxygen masks, use a different mask shape/size and/or alternate mask use with nasal cannulae Regularly lift and reposition the device (at least as frequently as general repositioning), ensuring that the tension of any holding straps or fixation is sufficient for function of the device but is not higher than necessary The use, positioning and repositioning of medical devices in patients who are oedematous requires careful consideration On intact skin, consider use of a dressing (see page 26 for the management of DTI with broken skin or an open wound); care should be taken not to select a dressing that does not interfere with the function of the device and that does not increase pressure, e.g. is not too thick

*Dressings with low friction outer surfaces may reduce superficial mechanical loads. Dressings with multiple layers have been found in computer models of the heel to dissipate mechanical loads at the bone:tissue interface more effectively than single-layer dressings; this effect may be due in part at least to the horizontal displacement of the different dressing layers relative to each other and to a cushioning effect.



Even when equipment for offloading and pressure redistribution is in use, the patient should be repositioned regularly, unless contraindicated, and should undergo regular reassessments and skin inspections (NPUAP/EPUAP/PPPIA, 2014)



Patient preference, comfort and concordance should be taken into account when offloading and repositioning

Repositioning

Frequent repositioning is an important method of intermittently offloading the tissues. Positioning can also be used to avoid applying pressure to an existing DTI. The exact nature of the repositioning regimen will depend on the:

- Patient's level of mobility and whether, and to what extent, they can reposition themselves
- Patient's ability to tolerate the new position
- Location of existing pressure-induced damage e.g. while in bed a patient with a sacral DTI might be positioned on their left and right sides only and not on the back
- Location of the patient e.g. in bed or a chair
- Support surface in use (NPUAP/EPUAP/PPPIA, 2014).

A repositioning regimen should describe the positions to be used along with the frequency and duration of the position change, and should be reviewed regularly (Box 12) (NPUAP/EPUAP/PPPIA, 2014). The frequency of repositioning will depend on patient risk. Those who are at higher risk and are more susceptible to pressure-induced damage need to be repositioned more frequently.

As a guide, patients should be repositioned every 2–4 hours when lying on a pressure redistributing mattress and should shift their weight every 15 minutes if sitting (NPUAP/EPUAP/ PPPIA, 2014; RNAO, 2016). Any aids used to assist with repositioning (e.g. hoists, glide sheets) should prevent further tissue trauma and use should be documented. After repositioning, sheets, garments and incontinence products should be wrinkle-free. In general, the number of layers between a patient's skin and a therapy surface should be kept to a minimum.

Devices including pillows, wedges, gel or fluidised mouldable positioners can be used to support a patient in a new position (Preston et al, 2017; NPUAP/EPUAP/PPPIA, 2014). Beds that allow the angle of the head and knee/foot sections to be adjusted independently can aid patient positioning (Preston et al, 2017).



Offloading and repositioning do not necessarily require specialist equipment; pillows can be used to good effect to support a patient in a position or can be used to provide offloading, e.g. of the heels if placed under the lower legs, or of bony prominences if placed either side

After repositioning, always check that the area(s) of concern are properly offloaded

Independent movement by a patient can move a body part away from a preventative device, e.g. if the legs are moved from pillows used for heel offloading, the heels may come into contact with the support surface. Consider the use of an appropriate alternative, e.g. a patient-worn removable or non-removable device or a mattress with inbuilt heel zone, if independent movement is problematic

Box 12. Repositioning a patient with a DTI (NPUAP/EPUAP/PPPIA, 2014; Moore & van Etten, 2014; Preston et al, 2017; Stephens & Bartley, 2017)

- Avoid whenever possible positioning a patient directly on to a DTI (or a PU)
- Where safe and possible encourage patients to reposition themselves by lifting vulnerable tissue areas clear of the support surface
- When assisting or moving a patient to a new position:
 Follow local manual handling procedures and policies
 - Avoid 'dragging' the patient along the support surface as this creates external shear forces
- Ensure manual handling equipment and medical devices are
- not left under the patient, unless designed to be left in place Repositioning in a bed:
 - Use the 30-degree tilted side-lying position (Figure 14, page 25) and alternate moving from right side to back to left side as appropriate; if this cannot be tolerated use the prone position if not contraindicated
 - Avoid 90-degree side-lying or semi-recumbent postures
 - Limit head of bed elevation to 30 degrees or less, unless medically contraindicated or if higher elevation is required for feeding, digestive or respiratory considerations

- Pillows, wedges, and gel or fluidised positioners can aid positioning
- If necessary, a knee break (gatch) can be used to reduce the risk of the patient sliding down the bed
- For taller patients, consider use of bed extensions and foam squabs to avoid feet being wedged against the foot plate of the bed
- Reposition every 2–4 hours or more frequently if there are signs of deterioration
- Repositioning while seated (in a chair or wheelchair)
 - Ensure the chair is suitable for the size and weight of the patient and fits correctly (Box 13, page 24)
 - Select a seating position that:
 - Is stable
 - Is acceptable to the patient and allows them to carry out their full range of functions
 - Ensures support for the feet (on the floor or appropriate footrest) and arms
- Reassess the effectiveness of the repositioning regimen regularly with respect to enabling the DTI to heal and prevention of other areas of pressure-induced damage



Figure 13: Heel DTI offloading using a pillow for a patient lying on their back in bed The pillow should be positioned under the full length of the calf with the knee slightly flexed. The pillow should not be in contact with the area of the Achilles tendon. Check the heel is not touching the bed by passing a hand underneath.

Box 13. Checking chair fit (Stockton et al, 2009; Stephens & Bartley, 2017)

- Seat height: The patient should be able to put their feet on the floor comfortably or, if in a wheelchair, on the footrests, with the ankle in a normal position
 - If the seat is too high, the patient may slide forward and slump back to put their feet on the floor or footrest and increase loading over the sacrum
 - If the seat is too low, additional pressure may be put over the ischial tuberosities
- Seat depth: There should be a space equivalent to the width of two fingers or 2.5cm between the front of the seat and the back of the patient's legs
 - If the seat is too deep the patient may need to slide forward to be able to put their feet on the floor or footrest
 - If the seat is too shallow, the front edge of the seat may dig in to the back of the patient's legs and cause pressure-induced damage
- Seat width: There should be a space of 2.5cm between the patient's hips and the side of the chair/ wheelchair on each side
 - If the seat is too wide, the patient may not able to maintain a good posture
 - If the seat is too narrow, movement and transfers may be hampered and pressure-induced damage may occur on the hips and buttocks
- Armrest height: Should support the elbows comfortably when seated
 - Armrests that are too low may cause postural problems that result in pressure-induced damage in other body parts
 - Armrests that are too high may cause elbow joint discomfort

For more information on seating, see Stephens & Bartley (2017).

Pressure redistribution

Patients with a DTI, or at increased risk of a DTI or other pressure-induced damage, should be placed on a pressure redistribution support surface selected according to local policy. Pressure redistribution support surfaces distribute the mechanical load more uniformly and so reduce the load or pressure over bony prominences (International Review, 2010). Pressure redistribution support surfaces are available in a wide range of formats including mattresses, integrated bed systems, mattress overlays, seat cushions and toilet seats.



Support surface selection should consider the patient's needs and should be appropriate for use in the care setting (NPUAP/EPUAP/PPPIA, 2014)

Pressure redistribution surfaces should be considered for the bed and seating (including chairs, wheelchairs, toilet seats and bathing/showering stools) (Table 5, page 25). Air-fluidised and alternating pressure surfaces can be useful for patients who are acutely ill and/or who cannot be repositioned (Ovens, 2012).



Support surfaces should not replace good care and repositioning, and their use should be reassessed regularly

As the patient improves, the support surfaces in use should continue to be reassessed carefully to ensure suitability and that the surface does not hamper independent movement.

PAIN MANAGEMENT

In addition to management of any background pain, pain management may need to be considered for repositioning and during dressing changes or debridement.

SKIN INSPECTION AND MANAGEMENT

The patient's skin should be inspected regularly (at least daily) for signs of pressure-induced damage including non-blanchable erythema and DTI (NPUAP/EPUAP/PPPIA, 2014). The skin under medical devices should be inspected at least twice daily for signs of pressure-induced damage (NPUAP/EPUAP/PPPIA, 2014).



Regular skin inspection is an essential part of DTI management and of pressure-induced damage prevention

If the patient is incontinent, reversible causes such as urinary tract infection or constipation

associated dermatitis (Beeckman et al, 2015; Fletcher, 2015).

The patient's skin should be kept clean and dry. Cleansing should use a pH balanced skin cleanser. Dry skin can be moisturised with a non-sensitising, fragrance and alcohol-free moisturiser (Norton et al, 2017).

should be treated. The skin should be cleansed at least once daily or after each episode of faecal incontinence. A skin protectant/barrier should be applied to areas with or at risk of incontinence-

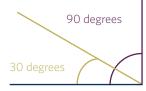








Figure 14: The 30-degree tilted side-lying position (Wilson, 2008)

After positioning the patient so that they are at a 30-degree angle to the surface of the bed, check that the sacrum and heels are not in contact with the bed; patients with obesity may need to be turned to a higher angle (45 degrees) to offload the sacrum (Moore & van Etten, 2014).

Table 5. Types of pressure redistributing support surfaces (Ovens, 2012; Fletcher et al, 2015)

Type of support surface	Description	
Reactive support surfaces		
Foam	Comprise a single type of foam or layers of foam of different densities	
Air- or gel-filled	Comprise air- or gel-filled compartments or columns	
Low air loss	 Air is permitted to escape through small holes in an air-filled support surface; may aid control of skin temperature and moisture 	
Air-fluidised	Contains small particles, e.g. silica beads. When air is forced through the particles they take on the properties of a liquid (i.e. become fluidised). Air escapes through the cover	
Active support surface	s	
Alternating pressure	 Sections of the support surface inflate and deflate cyclically to intermittently remove pressure 	
Hybrid support surfaces		
Non-powered	Comprise air cells with a layer of foam or air cells that have a foam core	
Powered	Comprise alternating pressure air cells with a layer of foam or alternating air cells that have a foam core	

LOCAL DTI MANAGEMENT

The approach to the local management of a DTI depends on whether the skin is intact.

DTI with unbroken skin

A DTI with unbroken skin can be left uncovered if acceptable to the patient, family and carers. If a dressing is applied, it should have a low friction outer surface. The skin under the dressing should be inspected at least once daily and the dressing changed in accordance with the manufacturer's instructions (WUWHS, 2016). In general, blisters should be left intact. On occasion, a blister may be deroofed under aseptic conditions, e.g. if a heel blister is preventing mobilisation.

DTI with broken skin or an open wound

A DTI with broken skin or an open wound should be cleansed and covered with a dressing that is appropriate for exudate level, wound bed tissue type and condition of the periwound skin with the aim of promoting moist wound healing (NPUAP/EPUAP/PPPIA, 2014). The cleansing agent and dressing should be selected according to local policy. If exudate levels are high, negative pressure wound therapy may be appropriate (Wounds UK, 2013b).

In general, debridement of a DTI should be approached conservatively. Surgical debridement is reserved for severe cases. An antimicrobial dressing can be considered for an 'open' DTI that is at high risk of infection or that is showing clinical signs and symptoms of infection (Box 11, page 19) (NPUAP/EPUAP/PPPIA, 2014).



The choice of dressing applied to a DTI with broken skin or an open wound should be based on the needs of the wound as determined by structured wound assessment and should follow local dressing formulary guidance

Healing progress should be monitored at least weekly and documented. For a DTI with an open wound, a tool such as the Bates-Jensen Wound Assessment Tool (BJWAT), the Pressure Ulcer Scale for Healing (PUSH) or the Pressure Sore Status Tool (PSST) may aid monitoring of healing progress (NPUAP/EPUAP/PPPIA, 2014).

PATIENT, CARER AND FAMILY EDUCATION

Education should include what a DTI is, how it is caused, how to recognise it, what the treatment is, how it can be prevented and where to get help or further information. Management of expectations including time frames and likely outcomes, where possible, should also be included.

The content and mode of delivery should be tailored to the needs of the individual. A combination of educational methods may be helpful. A discussion allows the information presented to be tailored to the understanding of the individual and the opportunity to ask questions.



Combining discussions with other methods of providing information, e.g. printed leaflets and website links, will give further opportunities for learning and reflection (Colledge et al, 2008)

MONITORING AND REASSESSMENT

The patient should receive ongoing risk assessment and should be reassessed if there is a change in his/her condition. The effectiveness and suitability of the offloading, pressure redistribution and repositioning regimen should be reviewed.

Referral to a tissue viability service may be needed if the condition of the DTI:

- Deteriorates rapidly or
- Does not improve within two weeks or within a timeline defined in local policies.

DOCUMENTATION

All aspects of the patient's management plan should be documented, along with rationale for each element and timing for review. The patient's notes should be flagged to show that they have had a DTI because of the likely increase of increased risk for future pressure-induced damage.



The presence of a DTI, along with current management, should be clearly communicated to the receiving organisation or department if a patient is transferred or admitted elsewhere

Prevention of DTI

Guidance from NICE does not specifically address assessment of risk for DTI, but states that an assessment of PU risk should be carried out and documented for adults:

- "... being admitted to secondary care or care homes in which NHS care is provided"
- "... receiving NHS care in other settings (such as primary and community care and emergency departments) if they have a risk factor" (NICE, 2014).

The 2005 NICE guidelines state that the initial assessment should be conducted within six hours of the first episode of care in a setting (NICE, 2005).

Healthcare Improvement Scotland's standards for prevention and management of PUs states that PU risk should be assessed and documented:

- "Within 8 hours of admission to hospital or care home
- Within 24 hours of admission to any other care setting
- On the first visit from community services or teams, for example, community nurse, hospital at home, social care or care at home" (HIS, 2016).

The NPUAP/EPUAP/PPPIA guidelines recommend that a structured risk assessment is carried out "as soon as possible (but within a maximum of eight hours after admission)" (NPUAP/EPUAP/PPPIA, 2014).

ASSESSMENT OF RISK FOR DTI

Patients should be reassessed regularly and according to local policy for risk for pressureinduced damage. Reassessment of risk should also be carried out if the patient's condition changes and/or they develop an additional risk factor (Box 5 and Table 1, page 10) (NPUAP/ EPUAP/PPPIA, 2014).



Patients, families and carers should be alert to any indicators or changes that may indicate increased risk for DTI or pressure-induced damage and contact a healthcare professional for consideration of a formal assessment

Underpinning DTI prevention are:

- Formal assessment of risk for pressure-induced damage
- Regular skin inspections to detect early signs of pressure-induced damage, e.g. nonblanchable erythema.

PU risk assessment tools

PU risk assessment tools for different patient groups are listed in Table 3 (page 18) and should be used according to local policy. Currently, there is no risk assessment tool specific to risk for DTI.

PU risk assessment tools provide a useful indication of level of risk for DTI and PUs. However, clinical judgement that considers all risk factors also has a very important and complementary role in guiding risk management (NPUAP/EPUAP/PPPIA, 2014).



Regular 'head-to-toe' inspection of the patient's skin is an essential element of DTI prevention and should not be replaced by a PU risk assessment tool

ASSESSMENT AND MANAGEMENT OF A PATIENT AT RISK OF DTI

The assessment and management of a patient considered at increased risk of DTI needs to take account of mental capacity and consent, patient preferences and the patient's other needs. Figure 10, page 15, outlines the assessment and management of a patient considered at increased risk of DTI:

- Holistic assessment (see pages 16-20)
- Take action to reduce risk of tissue damage (see pages 21-26), including:
 - Offload, where possible, the anatomical locations at risk of DTI
 - Regular repositioning and pressure redistribution
 - Optimise patient condition, including:
 - Nutrition
 - Pain management
 - Management of comorbidities
- Patient, family and carer education (see page 26)
- Monitoring and reassessment, including (see page 26):
 - Regular skin inspections
 - Repeated PU risk assessments
 - Evaluation of the effectiveness of the offloading/pressure redistribution/ repositioning regimen.



Patients being transferred from one facility to another may be at increased risk of DTI. The level of risk for a patient should be communicated clearly to the receiving facility. Appropriate pressure-related damage prevention measures and support surfaces should be implemented during transfer



If signs of pressure-induced damage are detected, e.g. non-blanchable erythema, the patient and the DTI/PU prevention strategies in place should be reviewed and revised as appropriate

Research needs and health economics

Further research is needed to establish many aspects of DTI, including incidence and the costs of management and prevention (Box 14).

Little is known about the health economics of DTI in the UK. However, from what is known of the incidence and costs of managing PUs, the socioeconomic impact of DTIs is likely to be considerable (Box 15).

Box 14. Research needs

- Large-scale studies of the epidemiology* of DTI in the UK to answer questions such as:
 - What is the incidence of DTI, including:
 - At different anatomical locations?
 - In different healthcare settings?
 - What factors are associated with increased risk of DTIs and how do they differ from those for PUs?
 - Are some risk factors for DTI more important than others? - What proportion of DTIs:
 - Resolve before the skin breaks?
 - Evolve to become open wounds?
- Are there predictors of resolution and evolution of DTIs? Assessment of the quality of life impact of DTI
- Health economic assessment of the prevention and management of DTIs

*The most efficient way to collect large-scale data is by using data in existing registries provided that reporting is comprehensive, and includes, for example, pressure-induced damage related to medical devices. In the UK, PUs and DTIs are recorded in incident reporting systems. However, currently, there is variation in when and how DTI is recorded. In some places, DTI is not reported in monitoring systems, while in others it is observed and only recorded when it can be assigned a pressure ulcer category (1 to 4) (Coleman et al, 2016). In other places, a DTI that evolves is re-categorised as a Category 3 or 4 PU.



To aid consistency and data analysis, the Expert Working Group proposes that a DTI is recorded when first observed, and that it remains categorised as a DTI even if it resolves or evolves further with skin breakdown

Box 15. Incidence and costs of PUs

- Incidence of PUs has been estimated to be 153.000 per year in the UK (Guest et al. 2015)
- Cost of healing a PU increases significantly with severity (from £1214 for a Category 1 PU to £14,108 for a Category 4 PU) (Dealey et al, 2012)
- Using 2013/2014 prices, the cost of managing PUs in the UK was estimated to be £507-£531 billion per year (Guest et al, 2017)

Recommended reading

- Black JM, Brindle CT, Honaker JS (2016) Differential diagnosis of suspected deep tissue injury. Int Wound J 13: 531-39
- Moore Z, Van Etten M (2014) Ten top tips: repositioning a patient to prevent pressure ulcers. Wounds Int 5(3): 6-9*
- National Institute for Health and Care Excellence (NICE) (2014) Pressure ulcers: prevention and management. Clinical guideline CG179. Available at: www.nice.org
- National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP) and Pan Pacific Pressure Injury Alliance (PPPIA) (2014) Prevention and treatment of pressure ulcers: quick reference guide. Cambridge Media: Osborne Park, Melbourne. Available at: www.epuap.org
- Stephens M, Bartley C (2017) Understanding the association between pressure ulcers and sitting in adults what does it mean for me and my

carers? Seating guidelines for people, carers and health & social care professionals. J Tiss Viabil doi: 10.1016/j.jtv.2017.09.004

- World Union of Wound Healing Societies (WUWHS) Consensus Document (2016) Role of dressings in pressure ulcer prevention. Wounds International*
- Worsley PR, Prudden G, Gower G, Bader DL (2016) Investigating the effects of strap tension during non-invasive ventilation mask application: a combined biomechanical and biomarker approach. Med Devices (Auckl) 9: 409-17
- Wounds UK (2013) Best Practice Statement. Eliminating pressure ulcers. London: Wounds UK**
 - *Available at: www.woundsinternational.com **Available at: www.wounds-uk.com

Appendix 1

DISTINGUISHING DTI FROM RARE CONDITIONS THAT PRESENT WITH DISCOLOURED

(PURPLE) SKIN +/- BROKEN SKIN (Mustafa et al, 2009; Harr & French, 2010; Misiakos et al, 2014; Gameiro et al, 2015; Vaiman et al, 2015; Black et al, 2016)

Condition	Characteristics	
Calciphylaxis	 May be excruciatingly tender and produce skin nodules or plaques; in later stages areas of painful necrosis produce non-healing ulcers with deep, black eschar that are often followed by infection and gangrene Associated with chronic renal failure; rare in the general population Due to vascular calcification and skin necrosis Most commonly seen in the lower extremities and at adipose tissue sites rather than over bony prominences 	
Perirectal abscess	 Presents with a dull aching or throbbing pain in the perineal area; pain worsens with sitting and on defaecation but eases after defaecation May produce a tender, fluctuant mass at the anal margin 	
Necrotising fasciitis	 Rare, severe, potentially lethal infection of the skin, soft tissues and muscles that may involve the abdominal wall, extremities or perineum/genitalia (Fournier's gangrene) Patients report severe pain and sometimes a history of trauma, and rapidly become unwell with sepsis Skin may look pale initially, then red or bronze, and become warm and swollen. In later stages, the skin turns violet and develops large blisters and areas of gangrene Requires rapid treatment with antibiotics and surgical debridement 	
Warfarin necrosis	 Usually occurs within a few days of starting warfarin treatment Patients are often premenopausal women being treated for deep vein thrombosis or pulmonary embolism Affects the buttocks and thighs with paraesthesia and flushing of the skin followed by a <i>peau d'orange</i> appearance; haemorrhagic blisters develop within 24 hours 	
Ecthyma gangrenosum	 Uncommon skin infection that may be caused by bacteria such as <i>Pseudomonas aeruginosa</i> or by fungi; reported in immunocompromised and healthy individuals; patients may develop sepsis Starts with an erythematous area that develops a nodule or haemorrhagic vesicles and progresses to a necrotic ulcer, often containing eschar 	
Gluteal compartment syndrome	 Rare; due to necrosis of the gluteal muscles following disruption of the artery (hypogastric) supplying blood to the muscles due to prolonged immobilisation, drug abuse, alcohol intoxication or surgery Causes severe buttock pain at rest and on movement of the hip, bruising, altered sensation and swelling of the buttock Usually requires surgical treatment 	
Pyoderma gangrenosum	 Rare; occurs in patients with autoimmune or neoplastic diseases and most often in middle-aged adults Lesions form tender papules, vesicles or pustules that evolve into painful ulcers that enlarge rapidly over a few days and that have raised violet-coloured (violaceous) and undermined borders 	
Toxic epidermal necrolysis/ Stevens- Johnson Syndrome	 Rare; potentially fatal Causes haemorrhagic erosions, erythema, blisters and areas of denuded skin Usually due to an adverse reaction to medication, but may be due to infection with Mycoplasma pneumoniae or Herpes simplex 	

References

- Agam L, Gefen A (2007) Pressure ulcers and deep tissue injury: a bioengineering perspective. J Wound Care 16(8): 336–42
- Anderson K, Hamm RL (2012) Factors that impair wound healing. J Am Coll Clin Wound Spec 4(4): 84–91

Australian Wound Management Association (AWMA) (2012) Pan pacific clinical practice guideline for the prevention and management of pressure injury. WA, Cambridge Media Osborne Park

- Bader D, Schoonhoven L (2017) What's in a name? *J Tiss Viabil* 25: 191–92
- Beeckman D, Campbell J, Campbell K, et al (2015) Proceedings of the Global IAD Expert Panel. Incontinence-associated dermatitis: moving prevention forward. Wounds International. Available at: www. woundsinternational.com (accessed 17 July 2017)
- Bergstrom N, Braden B (1992) A prospective study of pressure sore risk among institutionalized elderly. J Am Geriatr Soc 40(8): 747-58
- Berlowitz DR, Brienza DM (2007) Are all pressure ulcers the result of deep tissue injury? A review of the literature. Ostomy Wound Manage 53(10): 34–38
- Black JM, Brindle CT, Honaker JS (2016) Differential diagnosis of suspected deep tissue injury. *Int Wound J* 13: 531–39
- Chaloner DM, Franks PJ (2000) Validity of the Walsall Community Pressure Sore Risk Calculator. *Br J Comm Nurs* 5(6): 266–76
- Chamanga E, Ward R (2015) Documentation and record-keeping in pressure ulcer management. *Nurs Standard* 29(36): 56–63
- Coleman S, Gorecki C, Nelson EA, et al (2013) Patient risk factors for pressure ulcer development: systematic review. *Int J Nurs Studies* 50: 974–1003
- Coleman S, Nixon J, Keen J, et al (2014) A new pressure ulcer conceptual framework. J Adv Nurs 70(10): 2222-34
- Coleman S, Smith IL, Nixon J, et al (2016) Pressure ulcer and wounds reporting in NHS hospitals in England part 2: survey of monitoring systems. *J Tiss Viabil* 25: 16–25
- Coleman S, Smith IL, McGinnis E, et al (2017) Clinical evaluation of a new pressure ulcer risk assessment instrument, the pressure ulcer risk primary or secondary evaluation tool (PURPOSE T). J Adv Nurs doi: 10.1111/jan/13444
- Collard CD, Gelman S (2001) Pathophysiology, clinical manifestations and prevention of ischaemia-reperfusion injury. *Anesthesiology* 94: 1133–38
- Colledge A, Car J, Donnelly A, Majeed A (2008) Health information for patients: time to look beyond patient information leaflets. *J R Soc Med* 101: 447–53
- Cox J, Kaes L, Martinez M, Moles D (2016) A prospective, observational study to assess the use of thermography to predict progression of discoloured intact skin to necrosis among patients in skilled nursing facilities. *Ostomy Wound Manage* 62(10): 14–33
- Curley MA, Razmus IS, Roberts KE, Wypij D (2003) Predicting pressure ulcer risk in pediatric patients: the Braden Q Scale. *Nurs Res* 52(1): 22–33
- De Wert LA, Bader DL, Oomens CW, et al (2015) A new method to evaluate the effects of shear on the skin. *Wound Repair Regen* 23(6): 885-90
- Dealey C, Posnett J, Walker A (2012) The cost of pressure ulcers in the United Kingdom. J Wound Care 21(6): 261–66

Department of Health (2009) Reference guide to consent for examination or treatment (2nd ed). The Stationery Officer, London

- Dowsett C, Protz K, Drouard M, Harding KG (2015) *Triangle of wound assessment Made Easy.* Wounds International. Available at: www. woundsinternational.com
- Edsberg LE, Black JM, Goldberg M, et al (2016) Revised National Pressure Ulcer Advisory Panel Pressure Injury Staging System. *J Wound Ostomy Continence Nurs* 43(6): 585–97
- Elia M (2003) Screening for malnutrition: A multidisciplinary responsibility. Development and Use of the Malnutrition Universal Screening Tool ('MUST') for Adults. Redditch: BAPEN. Available at: www.bapen.org.uk (accessed 17 July 2017)
- European Pressure Ulcer Advisory Panel and National Pressure Ulcer Advisory Panel (EPUAP/NPUAP) (2009) *Prevention and treatment of pressure ulcers: quick reference guide.* Washington DC: National Pressure Ulcer Advisory Panel
- Farid KJ, Gefen A, Harder Y, et al (2014) Highlights from the International Forum on Deep Tissue injury evolution: a researchbased scientific collaboration. *Ostomy Wound Manage* 60(2): 18–28
- Ferguson-Pell M, Hagisawa S (1988) Biochemical changes in sweat following prolonged ischemia. J Rehabil Res Dev 25(3): 57–62
- Fife C (undated) A tale of two tail bones. Available at: www.medlineuniversity.com (accessed 17 July 2017)
- Fleck C (2007) Suspected deep tissue injury. *Emerg Med Critical Care Rev* 42-44
- Fletcher J (2015) Appropriate selection and use of barrier creams and films. *Wound Essentials* 10(2): 64–68
- Fletcher J (2017) An overview of pressure ulcer risk assessment tools. *Wounds UK* 13(1): 18–26
- Fletcher J, Gefen A, Jones L, et al (2015) *Hybrid support surfaces Made Easy.* Wounds International. Available at: www.woundsinternational.com
- Gameiro A, Pereira N, Cardoso J-C, Goncalo M (2015) Pyoderma gangrenosum: challenges and solutions. *Clin Cosmet Investig Dermatol* 8: 285–93
- Gawlitta D, Oomens CJ, Bader DL, et al (2007) Temporal differences in the influence of ischemic factors and deformation on the metabolism of engineered skeletal muscle. *J Appl Physiol* 103: 464–73
- Gefen A (2008) How much time does it take to get a pressure ulcer? Integrated evidence from human, animal, and in vitro studies. *Ostomy Wound Manage* 54(10): 26–35
- Gefen A (2009) Deep tissue injury from a bioengineering point of view. Ostomy Wound Manage 55(4): 26–36
- Gefen A (2014) Tissue changes in patients following spinal cord injury and implications for wheelchair cushions and tissue loading: a literature review. *Ostomy Wound Manage* 60(2): 34–45
- Gefen A, Farid KJ, Shaqwitz I (2013) A review of deep tissue injury development, detection and prevention: shear savvy. *Ostomy Wound Manage* 59(2): 26–35
- Gorecki C, Brown JM, Nelson EA, et al (2009) Impact of pressure ulcers on quality of life in older patients: a systematic review. *J Am Geriatr Soc* 57(7): 1175–83

- Guest JF, Ayoub N, McIlwraith T, et al (2015) Health economic burden that wounds impose on the National Health Service in the UK. *BMJ Open* 5: e009283
- Guest JF, Ayoub N, McIlwraith T, et al (2017) Health economic burden that different wound types impose on the UK's National Health Service. *Int Wound J* 14: 322–30
- Guo S, DiPietro LA (2010) Factors affecting wound healing. *J Dent Res* 89(3): 219–229
- Harr T, French LE. Toxic epidermal necrolysis and Stevens-Johnson syndrome. Orphanet J Rare Dis 2010; 5: 39
- Healthcare Improvement Scotland (HIS) (2016) *Prevention and management of pressure ulcers*. Standards. Available at: www. healthcareimprovementscotland.org (accessed 17 July 2017)
- Insall RL, Davies RJ, Prout WG (1989) Significance of Buerger's test in the assessment of lower limb ischaemia. J R Soc Med 82: 729–31
- International Review (2010) Pressure ulcer prevention: pressure, shear, friction and microclimate in context. A consensus document. London: Wounds International. Available at: www.woundsinternational.com (accessed 17 July 2017)
- Jackson C (1999) The revised Jackson/Cubbin Pressure Area Risk Calculator. Intensive Crit Care Nurs 15(3): 169–75
- Kirkland-Kyhn H, Teleten O, Wilson M (2017) A retrospective, descriptive, comparative study to identify patient variables that contribute to the development of deep tissue injury among patients in intensive care units. *Ostomy Wound Manage* 63(2): 42–47
- Knight SL, Taylor RP, Polliack AA, Bader DL (2001) Establishing predictive indicators for the status of loaded soft tissues. J Appl Physiol 90(6): 2231–37
- Krishnan S, Karg PE, Boninger ML, et al (2016) Early detection of pressure ulcer development following traumatic spinal cord injury using inflammatory mediators. *Arch Phys Med Rehabil* 97(20): 1656–62
- Lechner A, Lahmann N, Neumann K, et al (2017) Dry skin and pressure ulcer risk: a multi-center cross-sectional study in German hospitals and nursing homes. *Int J Nurs Stud* 18(73): 63–69
- Levy A, Frank MO, Gefen A. The biomechanical efficacy of dressings in preventing heel ulcers. *J Tissue Viabil* 2015; 24: 1-11
- Loerakker S, Manders E, Strijkers GJ, et al (2011) The effects of deformation, ischemia, and reperfusion on the development of muscle damage during prolonged loading. *J Appl Physiol* 111: 1168–77
- Loerraker S, Huisman ES, Henk AMS, et al (2012) Plasma variations of biomarkers for muscle damage in male nondisabled and spinal cord injured subjects. *J Rehabil Res Dev* 49(3): 361–72
- Lowthian P (1989) Identifying and protecting patients who may get pressure sores. *Nurs Standard* 4(4): 26–29
- Lustig M, Levy A, Kopplin K, et al (2017) Beware of the toilet: the risk for a deep tissue injury during toilet sitting. *J Tiss Viabil* http://dx.doi. org/10.1016/j.jtv.2017.04.005 (accessed 17 July 2017)
- Miller GE, Seale JL (1981) Lymphatic clearance during compressive loading. *Lymphology* 14(4): 161–66
- Misiakos EP, Bagias G, Patapis P, et al (2014) Current concepts in the management of necrotizing fasciitis. *Front Surg* 1(36): 1–10
- Moore Z, van Etten M (2014) Ten top tips: repositioning a patient to

prevent pressure ulcers. Wounds Int 5(3): 6-9

- Moore Z, Gershon S, Fletcher J (2016) SEM Scanner Made Easy. Wounds International. Available at: www.woundsinternational.com
- Mrdjenovich D, Simman R, Fleck C, Luttrell T (2016) The American College of Clinical Wound Specialists (ACCWS) Rebuttal to the Recent NPUAP Pressure Ulcer Definition (July 2016). J Am Coll Clin Wound Spec 7: 53
- Mughal AF (2014) Understanding and using the Mental Capacity Act. *Nursing Times* 110(21): 16–18
- Mustafa NM, Hyun A, Kuma JS, Yekkirala L (2009) Gluteal compartment syndrome: a case report. *Cases J* 2: 190
- Nair AV, Nazar PK, Sekhar R, et al (2014) Morel-Lavallée lesion: a closed degloving injury that requires real attention. *Indian J Radiol Imaging* 24(3): 288–90
- National Institute for Health and Care Excellence (NICE) (2006) Nutrition support for adults: oral nutrition support, enteral tube feeding and parenteral nutrition. Available at: www.nice.org (accessed 17 July 2017)
- National Institute for Health and Care Excellence (NICE) (2014) Pressure ulcers: prevention and management. Clinical guideline CG179. Available at: www.nice.org (accessed 17 July 2017)
- National Institute for Health and Clinical Excellence (NICE) (2005) The prevention and treatment of pressure ulcers. Quick reference guide (CG29). Available at: www.nice.org (accessed 29 September 2017)
- National Pressure Ulcer Advisory Panel (NPUAP) (2017) NPUAP Position Statement on Staging – 2017 Clarifications. Available at: http://www.npuap.org/resources/position-statements/
- National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP and Pan Pacific Pressure Injury Alliance (PPPIA) (2014) *Prevention and treatment of pressure ulcers: quick reference guide.* Cambridge Media: Osborne Park, Melbourne
- Nichols E (2014) Mental Capacity Act and its relevance to wound care. *Wound Essentials* 9(2): 57–61
- Norton D (1989) Calculating the risk: reflections on the Norton Scale. Decubitus 2(3): 24–32. Erratum in: Decubitus 1989 2(4): 10
- Norton L, Parslow N, Afalavi A, et al (2017) *Best practice* recommendations for the management of pressure injuries. Canadian Association of Wound Care. Available at: www.woundscanada.ca (accessed 17 July 2017)
- Oliveira AL, Moore Z, O'Connor, Patton D (2017) Accuracy of ultrasound, thermography and subepidermal moisture in predicting pressure ulcers: a systematic review. J Wound Care 26(5): 199–215
- Oomens CWJ, Bader DL, Loerakker S, Baaijens F (2015) Pressure induced deep tissue injury explained. Ann Biomed Eng 43(2): 297-305
- Ousey K, Cook L (2012) *Wound assessment Made Easy.* Wounds UK 8(2). Available at: www.wounds-uk.com (accessed 17 July 2017)
- Ousey K, Schoonhoven L, Moore Z, et al (2017a) Should the EPUAP adopt the NPUAP's new pressure ulcer terminology and definitions? *Wounds UK* 13(3): 10–16. Available at: www.woundsuk.com (accessed 17 July 2017)
- Ousey K, O'Connor L, Doughty D, et al (2017b). *Incontinenceassociated dermatitis Made Easy*. Wounds International 8(2). Available at: www.woundsinternational.com (accessed 17 July 2017)

- Ovens L (2012) Selecting a support surface how to guide. *Wound Essentials* 7(2). Available from: www.wounds-uk.com (accessed 17 July 2017)
- Peart J (2016) The aetiology of deep pressure injury: a literature review. *Br J Nurs* 25(15): 840–43

Pieper B (2016) Pressure ulcers: impact, etiology, and classification. In: Bryant RA, Nix DP. Acute and chronic wounds. *Current management concepts*. Elsevier; 124–39

- Posthauer ME, Banks M, Dorner B, Schols JMGA (2015) The role of nutrition for pressure ulcer management: National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel, and Pan Pacific Pressure Injury Alliance white paper. *Adv Skin Wound Care* 28(4): 175–88
- Preston A, Rao A, Strauss R, et al (2017) Deep tissue pressure injury: a clinical review. *Am J Nurs* 117(5): 50–57
- Registered Nurses' Association of Ontario (2016) Assessment and management of pressure injuries for the interprofessional team, third edition. Toronto, ON: Registered Nurses' Association of Ontario. Available at: mao.ca/bpg/guidelines/pressure-injuries (accessed 17 July 2017)
- Rennert R, Golinko M, Yan A, et al (2009) Developing and evaluating outcomes of an evidence-based protocol for the treatment of osteomyelitis in stage IV pressure ulcers. *Ostomy Wound Manage* 55(3): 42–53
- Richbourg L, Smith J, Dunzweiler S (2011) Suspected deep tissue injury evaluated by North Carolina WOC Nurses: a descriptive study. J Wound Ostomy Continence Nurs 38(6): 655–60
- Salcido R, Lee A, Ahn C (2011) Heel pressure ulcers: purple heel and deep tissue injury. *Adv Skin Wound Care* 24(8): 374–80
- Scheiner J, Farid K, Raden M, Demisse S (2017) Ultrasound to detect pressure-related deep tissue injuries in adults admitted via the Emergency Department: a prospective, descriptive, pilot study. *Ostomy Wound Manage* 63(3): 36–46
- Schultz Brillo DJ, Mozingo DW, et al (2004) Wound bed preparation and a brief history of TIME. *Int Wound J* 1(1): 19–32
- Scottish Government (2000) Adults with Incapacity (Scotland) Act 2000: a short guide to the act. Available at: http://www.gov.scot/Publications/2008/03/25120154/3 (accessed 17 July 2017)
- Smith IL, Brown S, McGinnis E, et al (2017) Exploring the role of pain as an early predictor of category 2 pressure ulcers: a prospective cohort study. *BMJ Open* 7: e013623.
- Solowiej K, Upton D (2010) The assessment and management of pain and stress in wound care. Br J Comm Nurs 15(sup 4): S26–S33
- Sperring B, Baker R (2014) Ten top tips for taking high-quality images of wounds. *Wound Essentials* 9(2): 62–64
- Stekelenberg A, Gawlitta D, Bader DL, Oomens CW (2008) Deep tissue injury: how deep is our understanding? Arch Phys Med Rehabil 89: 1410–13
- Stephens M, Bartley C (2017) Understanding the association between pressure ulcers and sitting in adults what does it mean for me and my carers? Seating guidelines for people, carers and health & social care professionals. *J Tiss Viabil* doi: 10.1016/j.jtv.2017.09.004
- Stockton L, Krytstof S, Gebhardt S, Clark M (2009) Seating and pressure ulcers: clinical practice guideline. *J Tiss Viabil* 18: 98–108

- Sullivan R (2013) A two-year retrospective review of suspected deep tissue injury evolution in adult acute care patients. *Ostomy Wound Manage* 59(9): 30–39
- Sullivan R (2014) A 5-year retrospective study of descriptors associated with identification of Stage I and suspected deep tissue pressure ulcers in persons with darkly pigmented skin. *Wounds* 26(12): 351–59
- Tescher AN, Thompson SL, McCormack HE, et al (2017) Descriptive analysis of deep tissue pressure injuries (DTPI): predisposing factors, presentation and evolution. Poster presented at: *Wound Care: From Innovations to Clinical Trials (WCICT2017)*, Manchester, UK
- The Stationery Office (TSO) (2016) *Mental Capacity Act (Northern Ireland) 2016*. Available at: http://www.legislation.gov.uk/ nia/2016/18/contents (accessed 17 July 2017)
- Vaiman M, Lasarovitch T, Heller L, Lotan G (2015) Ecthyma gangrenosum versus ecthyma-like lesions: should we separate these conditions? *Acta Dermatovenerol* 24: 69–72
- VanGilder C, MacFarlane GD, Harrison P, et al (2010) The demographics of suspected deep tissue injury in the United States: an analysis of the International Pressure Ulcer Prevalence Survey 2006–2009. Adv Skin Wound Care 23(6): 254–61
- Vowden P, Vowden K (2015) Diabetic foot ulcer or pressure ulcer? That is the question. *The Diabetic Foot Journal* 18: 62–66
- Waterlow J (2005) *Waterlow Score Care*. Available at: www.judywaterlow.co.uk (accessed 17 July 2017)
- Willock J, Baharestani MM, Anthony D (2009) The development of the Glamorgan paediatric pressure ulcer risk assessment scale. *J Wound Care* 18(1): 17–21
- Wilson M (2008) Repositioning patients to prevent pressure ulcer formation: the 30° tilt. *Wound Essentials* 3:100-1
- Woo KY (2012) Exploring the effects of pain and stress on wound healing. *Skin & Wound Care* 25(1): 38–44
- World Union of Wound Healing Societies (WUWHS) (2008) Principles of best practice: wound infection in clinical practice. An international consensus. London: MEP Ltd. Available at: www.woundsinternational.com (accessed 17 July 2017)
- World Union of Wound Healing Societies (WUWHS) Consensus Document (2016) *Role of dressings in pressure ulcer prevention.* Wounds International. Available at: www.woundsinternational.com (accessed 17 July 2017)
- Worsley PR, Prudden G, Gower G, Bader DL (2016) Investigating the effects of strap tension during non-invasive ventilation mask application: a combined biomechanical and biomarker approach. *Med Devices (Auckl)* 9: 409–17
- Wounds UK (2013a) *Best Practice Statement. Eliminating pressure ulcers.* London: Wounds UK. Available at: www.wounds-uk.com (accessed 17 July 2017)
- Wounds UK (2013b) Best Practice Statement: Effective exudate management. London: Wounds UK. Available at: www.wounds-uk.com (accessed 17 July 2017)
- Wounds UK (2016) Best Practice Statement: Holistic management of venous leg ulcers. London: Wounds UK. Available at www.wounds-uk.com (accessed 17 July 2017)

