

# Dressings can prevent pressure ulcers: fact or fallacy? The problem of pressure ulcer prevention

In part one of this two-part article, the authors discuss the aetiology of pressure ulcers, the means of identifying those patients at risk, the range of clinical intervention strategies implemented to try and prevent their formation and the problems faced by clinicians in developing cost-effective solutions to pressure ulcer prevention. Part two will set out the scientific evidence to support the use of dressing materials to prevent pressure damage, discuss the clinical realities faced by clinicians and explore if the use of wound dressing materials has any part in a modern pressure ulcer prevention strategy.

Martyn Butcher, Geoffrey Thompson

## KEY WORDS

Pressure  
Shear  
Pressure damage  
Guidelines  
Evidence-based practice

The development of pressure ulcers in vulnerable, at-risk individuals is a significant burden on healthcare resources and it has been stated that their development can be viewed as an indicator of poor quality care (Department of Health, 1993; Olshansky, 2005). Despite position papers indicating some pressure ulcers may be unavoidable (Wound, Ostomy and Continence Nurses Society [WOCNS], 2009), there is still a stigma surrounding their formation and a drive to affect improved preventative strategies. Many different approaches to care have been adopted to prevent their development and yet pressure ulceration remains one of the most significant issues in health care today. One approach which has

Martyn Butcher is an Independent Non-medical Prescriber, Independent Tissue Viability and Wound Care Consultant; Geoffrey Thompson is an Infection Prevention/Control Audit and Research Nurse, Worcestershire Acute Hospitals Trust NHS UK, Independent Tissue Viability Nurse, Trustee member, Wound Care Alliance UK

been largely overlooked is the potential benefit of using wound care materials not to treat damage, but to help prevent it in the first instance.

### Pressure ulcers as an issue

Pressure ulcers are an all too common problem that occur in both hospital and

community environments (Weir, 2007; Stotts and Wu, 2009) and are reported worldwide by numerous authors and agencies (European Pressure Ulcer Advisory Panel [EPUAP], 2003; Clark et al, 2004; National Institute for Health and Clinical Excellence [NICE], 2005). US estimates of pressure ulcer

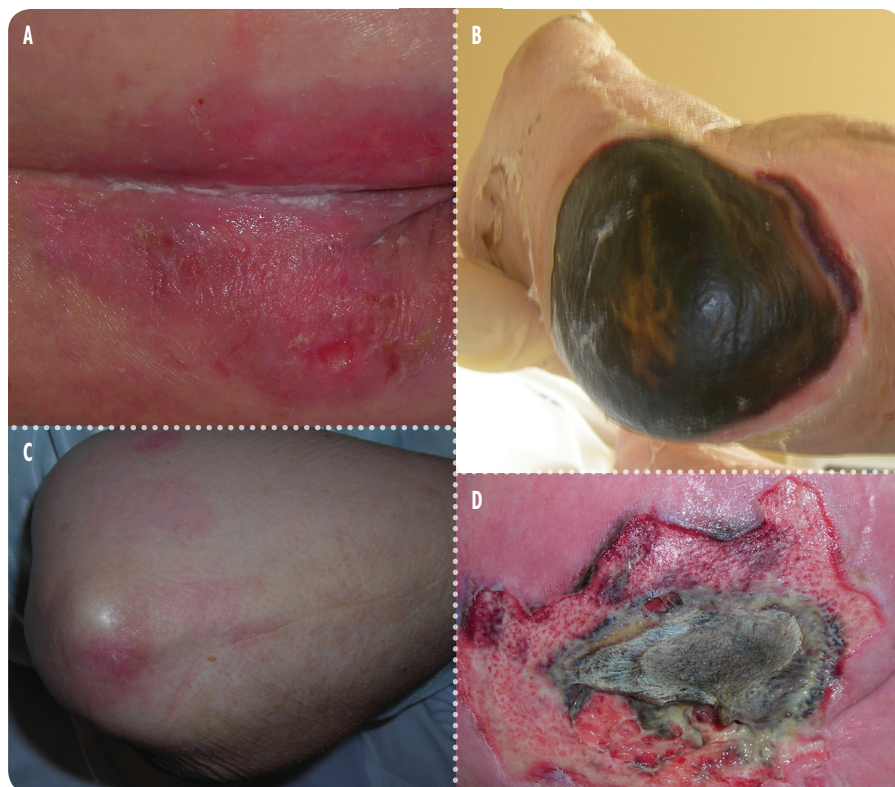


Figure 1. Pressure ulcers: A. Moisture damage to the sacrum; B. Presumed full-thickness damage under eschar; C. Blanching hyperaemia to elbows; D. Mixed levels of tissue damage, grades 1, 2, 3 and possibly 4.

incidence vary. In 1994 Bergstrom et al reported that at least one million people developed pressure ulcers. Subsequently, the Institute for Health Improvement estimated that 2.5 million users of US healthcare institutions develop pressure ulcer each year (Bales and Padwojski, 2009). Ultimately, if not treated appropriately, they can develop into severe and complex wounds with potentially devastating consequences for the patient that may require surgical intervention to bring about healing (Brown et al, 2007).

### Aetiology

Pressure ulcers are caused by prolonged and/or repeated ischaemic insults without adequate time for total tissue recovery, resulting in tissue necrosis (Hagisawa et al, 2004). These are manifested as localised areas of tissue breakdown involving the skin and/or deeper tissues (EPUAP, 2003), and generally occur as a result of unrelieved pressure to any part of the body, especially portions over bony or cartilaginous areas (Weir, 2007), such as the sacrum, elbows, knees, heels and ankles (Figure 1).

When looking at the aetiology of pressure ulcers, Braden et al (2000) developed a conceptual frame to help understand the various risk factors leading to ulcer formation, dividing the causes into two groups; 'extrinsic' and 'intrinsic'.

Extrinsic factors are physical mechanisms, events or circumstances that are external to the patient who develops pressure ulcers. Intrinsic patient-specific factors are unique to the individual, such as:

- ▶▶ Age
- ▶▶ Nutrition
- ▶▶ General health status
- ▶▶ Innate level of activity and mobility
- ▶▶ Morbidities such as diabetes.

While Bergstrom (2005) refers to more than 100 factors associated with pressure ulcer risk, such as previous medical history, comorbidities, fractured hip, spinal cord injury, cardiovascular disease, space in this paper does not permit a detailed listing and discussion

**Table 1**

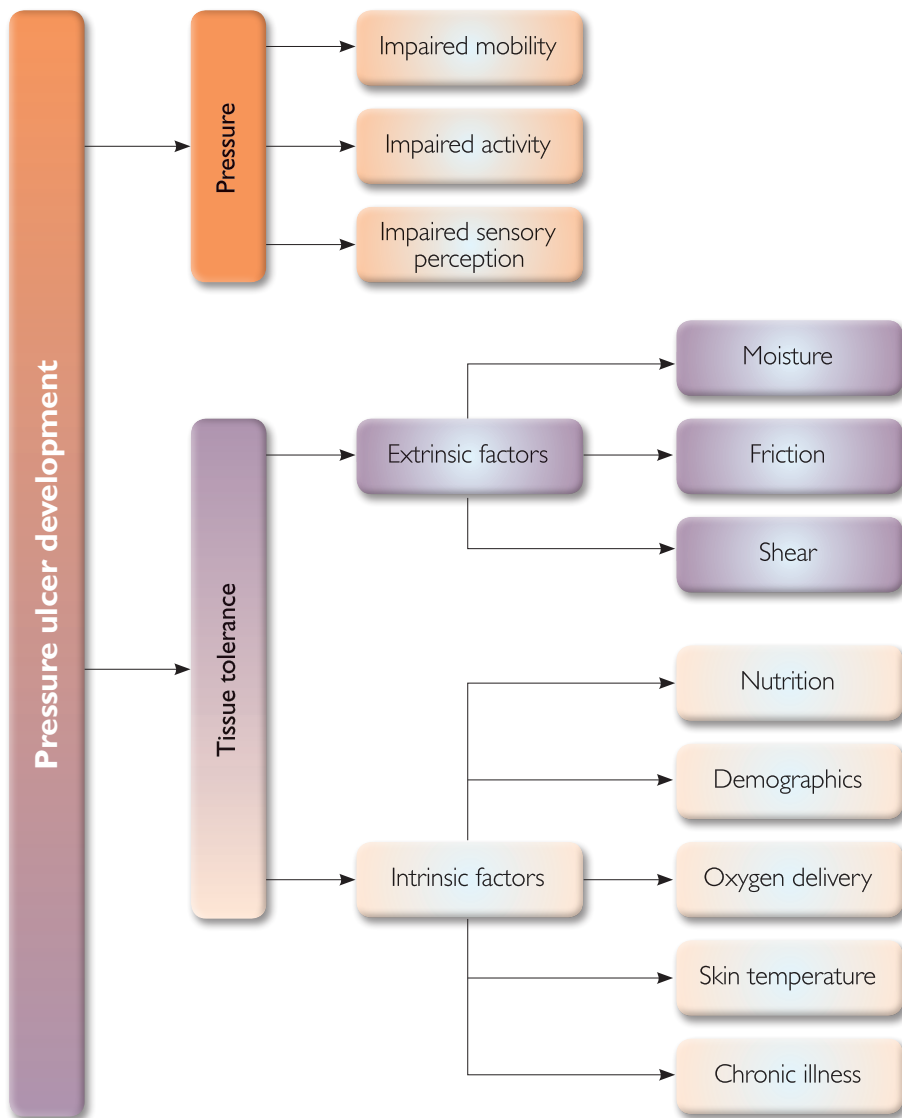
Factors associated with pressure ulcer risk

Intrinsic factor	Effect	References (examples)
Health status and comorbidities	Number of medical conditions: diabetes mellitus, cancer, respiratory disease, peripheral vascular disease (PVD), length of stay all show increased risk, prevalence and incidence of PUs	Makleburst et al, 1994; Papantonio et al, 1994; Allman et al, 1995; Lewicki et al, 1997
Age	Increasing age = increased risk of pressure ulcer formation, especially beyond the age of 70 from cardiac and neurological issues, lowered skin elasticity and resilience	Papantonio et al, 1994; Whittington et al, 2000; Margolis et al, 2002
Drug history	Steroids, chemotherapy, anticoagulants interfere with skin integrity and wound healing	Nixon et al, 2001
Mobility/immobility	Reduced ability to self-reposition due to trauma, surgery, post anaesthesia. Spinal injury can prolong unrelieved pressure exposure times on vulnerable tissues	Munro, 1940; Kosiak et al, 1958; Exton-Smith et al, 1961; Berlowitz and van Wilking, 1989; Allman et al, 1995; Bliss et al, 1999; Schoonhoven et al, 2002
Nutritional status	Poor nutrition can lead to muscle wasting and soft tissue loss + less tissue cushioning and greater bony prominences, as well as reduced collagen and tissue strength	Makleburst et al, 1994; Allman et al, 1995; Collier and Moore, 2006
History of previous PUs	Healed, ulcer sites remain an area of risk of re-breakdown because collagen structure remains mal-organised with scar tissue at between 40–80% of the original tissue tensile strength	NICE, 2005; Ichioka, 2005

on all possible factors. A representative sample can though be seen in Table 1.

Three main extrinsic mechanisms are known to precipitate pressure ulcer damage to the integument: pressure, shear and friction (Collier and Moore, 2006). Other extrinsic factors may also be involved in increasing vulnerability to damage; for example, environmental humidity and temperature can increase the moisture factor (or micro-climate) between the skin and the surface support, alter skin friction co-efficient and therefore increase the risk of shear and friction. This interacts with the unique intrinsic factors relative to each patient, such as the body's moisture level, body temperature, age, continence and medication (EPUAP, 2003; Bouton et al, 2005; Weir, 2007), increasing the chance of pressure ulcer development (Figure 2).

Pressure is described as the load applied at right angles to the tissue interface (Krouskop, 1983; Bennett and Lee, 1986; Shear Force Initiative [SFI], 2006). External pressure forces evenly applied over the surface of the body, as when a diver is submerged in water; do not appear to be a problem in that pressure ulcers do not form (Sprigle, 2000). However, when the pressures are unevenly applied, with gradient pressure differences between the point of pressure focus and the adjacent tissues, damage can occur with pressures conducted through the skin to the underlying tissues particularly close to the bone (Le et al, 1984). This causes occlusion of the blood vessels which, if unrelieved, leads to cellular anoxia, the build-up of metabolic waste and eventual cell death (Collier and Moore, 2006).



**Figure 2. Flowchart for the prediction and prevention of pressure ulcers. Taken from the Australian Wound Management Association (AWMA) Clinical Practice Guidelines (AWMA, 2001).**

The amount of pressure required to precipitate cell damage is dependent on the intensity of pressure, the duration of exposure (Kosiak, 1961), and to the individual's ability to cope with pressure loading (Daniel et al, 1981).

Controversy reigns over what pressure is required to induce capillary closure (Russell, 1998), but what is widely accepted is that even low pressures may cause tissue damage if exposure is prolonged (Read, 2001). This may be due to the way in which the pressure gradient is transmitted through tissues, a phenomenon known as the McClellmont 'cone of pressure' (McClellmont, 1984). An interface pressure such as 50mmHg between the skin and the support surface is

transmitted through the different underlying tissues; skin, subcutaneous fat, muscle and finally bone, with a cone-shaped increase in pressure of three to five times that at the interface so that pressures as high as 200mmHg might be experienced at the bony prominence (Collier and Moore, 2006).

It is commonly quoted that a safe level of pressure is 32mmHg, with 32mmHg being the arteriolar closing pressure and 12mmHg the venous limb side of the capillary loop (Landis, 1930). However, this early experimental work was undertaken on nail-bed pressures in healthy volunteers and so is now widely regarded as a guide rather than a definitive measure. Many experts believe that there is no direct link between

the internal pressures generated in the tissues under compression and the external pressure at the interface between the support surface and the skin under compression. As the average interface pressure is usually much greater than 32mmHg, it is assumed that the internal pressure will be high, although this cannot be measured in the clinical setting (Bader and Oomens, 2006).

Normal physiological response to pressure stressing includes the development of blanching erythema. This occurs as an adaptive response to short-term ischaemia in which previously stressed blood vessels dilate causing a temporary red 'flush' in the tissues (Dealey, 1994). This flush fades on light finger pressure and normally fades shortly after blood flow is restored.

Non-blanching erythema arises from either prolonged exposure to low-level pressure or short exposure to high pressure (the specific level of pressure varying between individuals), indicating that tissue damage has occurred. In this case the erythema is not due to a temporary flush of blood rushing into the area, but to local capillary disruption and leakage of blood into the surrounding tissues. Normal skin colour is not restored. This is considered to be the beginning of a pressure ulcer or grade 1 damage in some ulcer classification systems (Bethell, 2003) (Figure 3).

In darker pigmented individuals this 'blanching' may not be apparent. Thus, it is important to contrast the differences between the pressure points and the surrounding skin, as early damage, although not visible, may feel hotter; colder; harder or look shinier than the healthy skin (Bethell, 2003). With this in mind, healthcare staff should be familiar with the normal skin colour and tone of their patients.

The degree of vulnerability to pressure varies from person to person due to:

- ▶▶ Tissue tolerance variations between individuals through the combination of extrinsic and intrinsic factors unique to the individual (Bridel, 1993)

- ▶ Pressure duration over the pressure points which can result in damage from high pressure for short intense periods, which can be as damaging as low pressure for prolonged periods (Bell, 2005)
- ▶ Collagen function protecting the microcirculation helps to maintain the pressures inside and outside the cells preventing cell bursting. Collagen levels vary from person to person with lessening protective qualities with aging (Russell, 1998)
- ▶ Autoregulatory processes initiated when external pressure is sensed, leading to increased internal capillary pressure, reduced blood flow and reactive hyperaemia to counteract the pressure loading.

These mechanisms can fail when the external pressure exceeds the person's diastolic pressure rather than the 32mmHg often quoted (Nixon, 2001).

The response of tissue to external forces varies greatly, being dependent on a large number of factors. It is therefore not possible to establish a 'safe level of pressure'. In addition, tolerance to pressure can vary greatly from individual to individual due to the interplay of external factors listed in *Table 1*. Given the highly variable nature of pressure transmission, capillary closure and the individual's normal and adaptive responses to pressure stress, the production of time/pressure curves (mathematical models for predicting the time likely to cause tissue damage when tissue is exposed to specific levels of pressure [Reswick and Rogers, 1976]) may be of little practical benefit to the clinician in everyday practice (Sharp and McLaws, 2005; Grefen, 2009).

Shear is a mechanical stress applied parallel to the skin. The SFI describes it as: 'An action or stress resulting from applied forces which causes or tends to cause two contiguous internal parts of the body to deform in the transverse plane (i.e. "shear strain")' (SFI, 2006). This sliding or twisting force occurs continuously within soft tissues even when perpendicular pressure is applied, but increases greatly when combined with lateral movement, as seen when

the body is adapting to the inclination of the bed or when sitting in a chair. If the skin adheres to the surface support (which is more likely if the skin is moist or wet from environmental factors or intrinsically from incontinence or sweating) (Weir, 2007; Beldon, 2008), the tissues attached to the gradually moving skeletal frame become distorted which, in turn, distort the blood vessels leading to their collapse or rupture.

Shear forces are generated as a result of the interplay of friction and pressure (Collier and Moore, 2006). When applied, shear increases the effects of pressure resulting in vascular occlusion at only half the pressure of non-stressed tissues (Bennett and Lee, 1986). Shear forces may also have a significant role in the development of deep tissue damage, although this is difficult to measure in the clinical setting (Russell, 1998). Potentially, shearing is the most serious extrinsic risk factor due to the rapidity with which it can result in tissue damage (Sharp and McLaws, 2005). This is more likely to occur in the elderly as a result of loose, fragile skin and the ease with which the different tissue types can be sheared off their respective attachments (Allman et al, 1995).

The edges of ulcers caused by shear forces appear to be ragged with more uneven wound margins, often with surrounding epidermal scuffing. Bruising may also be a feature (*Figure 4*).

The mechanisms of shear damage have important consequences for the planning and delivery of preventative care interventions, even though there are few clinical methods to estimate shearing forces or their resultant effects on tissues (Verluyzen, 1985). It is hoped that the work of the SFI will add to this body of knowledge.

Friction is a complex phenomenon which depends on complex physical science and engineering concepts. In simplistic terms, within the context of friction-induced tissue damage, we are referring to kinetic friction. Kinetic (or dynamic) friction occurs when two objects are moving relative to each



**Figure 3. Sacral area showing clinical features of blanching hyperaemia.**



**Figure 4. Shear pressure damage occurring in a young woman during childbirth.**

other and rub together: Bergman-Evans et al (1994) define it as the resistance to lateral movement. Kinetic friction is dependent on mass, force applied and the friction co-efficients of the surfaces involved. Clinically, the effect of friction between the skin and a support surface has important dynamics that can initiate pressure ulcer formation:

- ▶ It can cause excessive wear to the cornified layers of the skin with resultant exposure of the underlying structures (Read, 2001)
- ▶ It can cause the formation of blisters as separation occurs between the layers of the epidermis leading to

exposure of the underlying dermis (Butcher, 1999)

- ▶▶ The deformation of skin can lead to further deformation in deeper tissues (shear damage).

The amount of damage caused depends on tissue resistance and the interplay of friction and pressure. Pressure and friction together cause more damage than friction alone and will induce greater shear forces (Figure 5).

#### Moisture

Although not directly indicated as a mechanism of pressure damage, the role of moisture is pivotal in the development of friction damage and so is a secondary factor in shear forces (Beldon, 2008) (Figure 6). Moisture levels within the cells of the epidermis have a direct bearing on the friction co-efficient of this tissue. Even at relatively low levels, moisture causes a rise in friction co-efficient making skin 'stick' to surfaces (Nacht et al, 1981). In addition, when exposed to moisture for prolonged periods, the keratinised cells of the epidermis swell and become waterlogged. This reduces their ability to withstand friction and can result in epidermal stripping.

These features have new relevance since the re-classification of moisture lesions (Bethell, 2003; Butcher, 2005; Beldon, 2008). There is a close association between incontinence

dermatitis, moisture-induced damage and superficial pressure ulceration. The EPUAP have suggested that moisture-induced damage should be categorised separately from pressure ulcers. In practice, this differentiation can be difficult to interpret clinically. Defloor and Schoonhoven (2004) and Defloor et al (2005) identified that reliability of the EPUAP tool was low when used to differentiate moisture lesions and superficial pressure ulcers from photographic evidence. Indeed, writers such as Houwing et al (2007) argue that such a distinction should not be made as it distracts clinicians from the need to implement appropriate pressure ulcer prevention strategies, and, as McDonagh (2008) points out, these two phenomena can co-exist within a client at a given point in time.

#### Aetiological pathways

Controversy exists as to the aetiological route by which pressure ulcers form and progress. It is acknowledged that pressure ulcers are primarily caused by sustained mechanical loading, however, prevention of ulcer formation by reducing the degree of loading alone remains difficult to achieve. This is mainly due to poor understanding of the underlying pathways whereby mechanical loading leads to tissue breakdown (Bouten et al, 2005).

Three theories have been postulated to explain this process:

1. Pressure ulcers form via the top-to-bottom model
2. Pressure ulcers form via the bottom-to-top model
3. Pressure ulcers form via a middle approach model.

#### Theory 1

Pressure and shear induce local ischaemia, and impaired drainage impairs the transport of oxygen and nutrients to and metabolic waste products away from the cells within the affected tissues. Eventually this leads to cell necrosis and the formation of an ulcer. There are sound arguments for damage to muscle tissue as it is metabolically more active than skin.

#### Theory 2

This model states that when pressure is relieved from the compressed tissues by patient repositioning or the use of an alternating pressure-alleviating mattress (APAM), it is the restoration of blood flow after the load-removal rather than impaired blood flow during pressure loading that is the mechanism of tissue necrosis. It is claimed that it is an over-abundant release of oxygen-free radicals during pressure off-loading that causes the damage.

#### Theory 3

In the third model, tissue damage may start anywhere between the skin and the underlying bone, but can include the skin surface and bone interface, concurrently or haphazardly, to produce a pressure ulcer.

#### Prevalence and incidence of pressure ulcers

Unless correctly identified and treated, pressure ulcers can have a significant effect upon the patient's quality of life



Figure 5. Shear and friction damage to stump from badly fitting prosthesis.



Figure 6. Sacral region showing clinical features of moisture damage combined with shear and pressure.

and may, under certain circumstances, prove fatal. The deaths of thousands of patients are attributed to pressure ulcers and their complications every year (Agam and Gefen, 2007). Data relating to incidence (a statistical measurement of the number of individuals developing a condition) and pressure ulcers varies considerably. A recent literature review investigated pressure ulcer prevalence and incidence in intensive care patients. The analysis of data from published papers highlighted these variations with pressure ulcer prevalence (the number of individuals with pressure ulcers as a percentage of the total defined population at one point in time) in intensive care settings, ranging from 4% in Denmark to 49% in Germany, while incidence ranged from 38% to 124% (Shahin et al, 2008). In a Canadian study in 2004 the national prevalence figure across all care settings was estimated at 26% (Woodbury and Houghton, 2004). More specifically, a recent study has shown that the prevalence of pressure ulceration within the population receiving health care in Bradford, UK was 0.74 people with a pressure ulcer per 1000 population (95% CI 0.6–0.8) (Vowden and Vowden, 2009).

### Cost of pressure ulcers to health care

Patients with pressure ulcers place a burden on health care as they require a significant amount of medical resources to treat. A recent survey evaluated the impact of wound care in Bradford and Airedale NHS Primary Care Trust in the UK (Vowden et al, 2009), and showed that the prevalence of patients with a wound was 3.55 per 1000 population. The estimated cost to the US hospital sector is \$11 billion per annum (Bales and Padwojski, 2009). This has been considered unsustainable and unacceptable. In an effort to control costs and raise quality standards, the Centers for Medicare and Medicaid Services (CMS) has determined it will no longer reimburse hospitals for treating a range of hospital-acquired conditions including pressure ulcers (Bergquist-Beringer et al, 2009). This is having a serious impact on US healthcare management and service provision and has lessons for the UK

healthcare sector. The majority of wounds were surgical/trauma (48%), leg/foot (28%) and pressure ulcers (21%). Prevalence of wounds among hospital inpatients was 30.7%. Of these, 11.6% were pressure ulcers, of which 66% were hospital-acquired. Further cases have received attention; over \$3 million was awarded by a Florida court in 2008 (Legal Eagle, 2008), while the Supreme Court of Mississippi approved a \$1 million award against a nursing home (Legal Eagle, 2007).

In a study undertaken on patients developing a pressure ulcer to estimate the annual cost of treating pressure

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ulcers in the UK, the actual costs were derived from a bottom-up methodology, based on the daily resources required to deliver protocols of care reflecting good clinical practice. The results showed that at this time the cost of treating a pressure ulcer varied from £1,064 (grade/stage 1) to £10,551 (grade/stage 4). Costs increase with ulcer grade/stage because the time to heal is longer and because the incidence of complications is higher in more severe cases. At the time of writing, the total cost in the UK was estimated at £1.4–£2.1 billion annually (4% of total NHS expenditure). The study also showed that most of the associated cost was related to nurse time (Bennet et al, 2004). Vowden et al (2009) also concluded that the most important components are the costs of wound-related hospitalisation and the opportunity cost of nurse time (the indirect cost incurred to the healthcare provider by the nurse undertaking care for this individual which would otherwise be utilised caring for other patients). In total, 32% of patients treated in hospital accounted for 63% of total costs, of which the development of

hospital-acquired pressure ulcers were a significant component and focus for potential cost reductions.

### Legal issues

The direct costs of patient treatment are not the only area of expense. Increasingly, the spectre of the threat of legal action is taking a greater place in pressure ulcer management. In a US study, hospital stays for the treatment of pressure ulcers have been estimated to be in the region of \$37,800 (Weir, 2007). It has been shown that these patients require 50% more nursing time, remain hospitalised for significantly longer periods, and incur higher hospital charges (Bradon and Endowed, 2008). Pressure ulcers are the leading iatrogenic causes of death reported in developed countries, second only to adverse drug reactions (Barczak et al, 1997).

In November 2000 the State of Hawaii convicted an individual of manslaughter in the death of a patient at a nursing home for permitting the progression of decubitus ulcers without seeking medical help, and for not bringing the patient back to a doctor for treatment of the ulcers (Di Maio and Di Maio, 2002). A number of authors have highlighted the increase in litigation associated with malpractice related to pressure ulcers not only in the US (Bennet et al, 2000; Levine et al, 2008; Meehan and Hill, 2009), but also in Europe (Cherry, 2006). It therefore makes clinical and economic sense to take measures to minimise pressure ulcer risk by taking preventative actions (Meehan and Hill, 2009).

### Standard preventative interventions

Possibly due to the emphasis of scientific research on the role of pressure within pressure ulcer aetiology, most effort appears to have gone into strategies to reduce or attempt to eliminate pressure in the clinical setting. Over the past thirty years many manufacturers have developed a wide variety of support surfaces, principally mattresses, aimed at this particular endpoint. With such a wide range of products there can be confusion over product selection for a given pressure

ulcer risk (Rithalia, 1996), and there is a need for an understanding of the difference between the mattress and cushion classes (Finucane, 2006). Standard interventions to prevent pressure ulcer formation have included the use of specific redistributive surfaces as either pressure-reducing appliances or pressure-relieving mattresses or cushions.

Pressure-reducing support surfaces vary from relatively simple foam and slashed foam constructions to gel, fluid, and air-filled systems. There are also more complex dynamic pressure-reducing low airloss systems and dynamic foam (Thompson, 2006; Gray et al, 2008), or forms of 'air-float' (Thompson et al, 2008) in which pressure at the interface between the dependent skin and the support surface is reduced through the use of the conforming support surface, thereby spreading load and reducing pressure per square centimetre.

In addition, the materials that used to cover such devices have become more technically advanced with non-stretch PVC covers giving way to two- and three-way stretch which encourages greater conformity between the body and the mattress/cushion. Improved vapour permeability with PU materials also reduces the risk of moisture build-up at the interface, with the aim of reducing the friction/shear co-efficient (Jay, 1995).

It stands to reason that if one of the major components of pressure ulcer formation is the application of unrelieved pressure, then the reduction of this pressure to sub-morbid levels is a key factor in pressure damage prevention. Pressure redistribution through offloading provides tissues with the time needed for cellular repair and the restoration of normal cellular activity. In its basic form, this is achieved by offloading tissues through either manual repositioning or the use of splints, wedges and other repositioning devices (Guttmann, 1955, 1976).

Cyclical offloading teamed with the use of a conforming interface is one

approach to this problem. This approach is adopted by those using APAMs where load is supported by alternating, conforming air cells. These cells periodically change their pressure profile in a pre-set cycle, thereby altering the area of tissue exposed to compression stresses. However, some clinicians prefer constant low pressure support surfaces, such as those found in air fluidised and low air loss systems. Unfortunately, there is little data to indicate which approach is preferable.

### Reduction of friction and shear

While friction and shear are cited as the other mechanisms of pressure damage, due to technical and ethical issues little research has been undertaken in this area (Ohura et al, 2005). For this reason, the reduction of these components in clinical practice has generally been undertaken based on anecdotal evidence. Due to the risk of increasing shear forces, previous practices such as massage of high risk tissues have been indicated as dangerous (Dyson, 1978; Pritchard and Mallett, 1993; Buss et al, 1997; Shahin et al, 2009), and so have been largely abandoned. Clinicians have been advised to use care in positioning patients to minimise shear force, (Maklebust, 1987; AWMA, 2001) and to use low-friction turning/repositioning aids to minimise skin and soft tissue damage (Butcher, 2005). Some writers have also indicated that the use of dressings and skin sealants may help in reducing friction and therefore reduce the risks of friction damage and shear forces (AWMA, 2001; Black, 2004; Butcher, 2005).

The practice of using simple adhesive dressings to minimise friction is accepted by many authorities as commonplace among healthcare workers and the general public. How many of us have used adhesive tape or wound plasters on our heels to prevent new footwear from rubbing and producing painful blisters? (Is this any different from the concept of using dressings to prevent pressure ulcers?) The effects of 'rubbing' are to produce friction which is, by definition, one of the primary mechanisms of pressure

ulcer formation. However, some wound care practitioners continue to warn that dressings do not prevent pressure damage and, as such, their use is neither scientifically validated nor cost-effective.

This is a contentious issue which demands further inspection. Its relevance cannot be overstated when one considers that while the clinical community is aware of the mechanisms of pressure damage and enormous amounts of money have been invested in pressure redistributive surfaces, particularly the dynamic devices, pressure ulcers remain such a common occurrence (Vangilder et al, 2008).

In the second part of this paper in a subsequent issue of *Wounds UK*, the authors will look at the evidence available to support the use of dressings to prevent pressure ulcer formation, and what properties such products might need to make them an effective tool in clinical use. **WUK**

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## Key points

- ▶▶ Pressure ulcers have a significant impact on health-care budgets.
- ▶▶ The development of pressure ulcers has been cited as being an indicator of poor quality care.
- ▶▶ Pressure, shear and friction are considered to be the main mechanisms of damage.
- ▶▶ Pressure ulcer incidence remains at a worryingly high level.
- ▶▶ Alternative strategies to pressure ulcer prevention need to be considered.

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