Publication 1
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 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...
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 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 (Krousok, 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).

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Factors associated with pressure ulcer risk</th>
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<tr>
<td><strong>Intrinsic factor</strong></td>
<td><strong>Effect</strong></td>
</tr>
<tr>
<td>Health status and comorbidities</td>
<td>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</td>
</tr>
<tr>
<td>Age</td>
<td>Increasing age = increased risk of pressure ulcer formation, especially beyond the age of 70 from cardiac and neurological issues, lowered skin elasticity and resilience</td>
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<tr>
<td>Drug history</td>
<td>Steroids, chemotherapy, anticoagulants interfere with skin integrity and wound healing</td>
</tr>
<tr>
<td>Mobility/immobility</td>
<td>Reduced ability to self-reposition due to trauma, surgery, post anaesthesia. Spinal injury can prolong unrelieved pressure exposure times on vulnerable tissues</td>
</tr>
<tr>
<td>Nutritional status</td>
<td>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</td>
</tr>
<tr>
<td>History of previous PUs</td>
<td>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</td>
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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 McClemont ‘cone of pressure’ (McClemont, 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 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).

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 (Verluysen, 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 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.
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 unsustainably 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 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 takes 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-foam’ (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 (Guttman, 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 (Ohara 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 tissue 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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Publication 2
Can the use of dressing materials actually prevent pressure ulcers: presenting the evidence

In part one of this two-part series, the authors discussed the aetiology of pressure damage and how it develops. It was noted that despite widespread research into the field, pressure damage is still commonplace and the incidence and costs incurred can place a significant burden on healthcare resources. The authors questioned if a different approach should be investigated. In part two they examine the scientific evidence that supports the use of dressing materials in the prevention of pressure damage and conclude that there may already be tools available to facilitate this shift in preventative healthcare strategies.

Martyn Butcher, Geoffrey Thompson

**KEY WORDS**

Pressure
Shear
Research
Dressings
Evidence-based practice

Many different approaches to wound care have been adopted to prevent the development of pressure ulcers, yet these wounds remain one of the most significant issues in healthcare today. One approach that has been largely overlooked is the potential benefit of wound dressings not to treat pressure damage, but to help prevent it in the first place.

In this article, the second in a two-part series investigating the aetiology, incidence and treatment of pressure ulcers, the authors undertook a literature search to identify if there are any published articles that refer to pressure ulcer prevention using wound dressings, and whether it can be used as an effective preventative treatment.

**Methodology**

Electronic searches of bibliographic databases and internet sites (Table 1) were supplemented with manual searches of conference proceedings and journals relevant to wound management. This was not intended to be a systematic review — its role was to provide an overview of the evidence.

Pressure ulcers pose a serious risk to patients and represent a significant burden on the NHS. As such, they need to be considered in patient treatment regimens. Pressure ulcer risk assessment and prevention programmes need to be introduced as a priority, as there is evidence to show that these can reduce the institutional incidence of pressure ulcers by as much as 60% (Bergstrom et al, 1995).

A significant amount of research has been undertaken into pressure ulcer risk assessment and prevention, however, it is not the purpose of this article to review or critique this work. There has, though, recently been a focus on using advanced wound dressings to prevent the formation of pressure ulcers. A number of studies have looked at new and existing dressings in both the laboratory and clinical environment.

**The evidence**

In exploring the available evidence for dressing use in pressure ulcer prevention, it is important to differentiate between evidence that emanates from laboratory studies (in vitro — literally meaning ‘in glass’) and that which is derived from studies on human or animal subjects (in vivo, or ‘in a living organism’).

In vitro studies have the advantage of enabling the researcher to control the study environment and conditions, something difficult to achieve in the clinical setting. For example, dressing materials can be objectively tested for their mechanical properties without risk to study subjects, however, it is argued that such studies do not reflect the complex environment and conditions that occur within the clinical settings.
setting. In vitro studies should, therefore, be seen as precursors to subsequent in vivo research.

The search revealed relatively few results that showed the use of dressings to prevent pressure damage. While comments on dressing use in pressure ulcer prevention were found (both with positive and negative results), the majority were subjective, unsubstantiated comments. Only those that presented results of research are presented. These invariably had positive outcomes.

**Experimental evidence**

In the laboratory, a variety of dressing materials have been evaluated for the prevention or minimisation of shear force and friction, all of which are major causes of pressure ulceration.

Ohura et al (2005) published a study that looked at the ability of various commonly available adhesive dressing materials to prevent shear forces. The products selected were a hydropolymer (Tielle™, Systagenix), a hydrofoam (Allevyn Adhesive™, Smith & Nephew) and a hydrocolloid (Duoderm CGF™, ConvaTec). A number of standardised tests were carried out on product samples to measure three causes of shear force — static friction, adhesion and shear transmissibility. The study demonstrated that the coefficients of static friction (the drag resistance between patients’ clothing and the outer surface of the dressing) were 1.01, 0.72 and 0.48 for the hydropolymer, hydrofoam and hydrocolloid respectively.

Products were evaluated for adhesion using an industry-standard test in both dry and wet formats. The hydropolymer was withdrawn from this test as it displayed no adhesive qualities. The hydrofoam and hydrocolloid showed identical adhesive qualities when dry, but the hydrocolloid failed when wet (Ohura et al, 2005).

Transmissibility of shear testing was undertaken by measuring the ability of the dressing to deform when under force. The samples were compared with a control (a non-stretch tape). Due to its composition it was impossible to test the hydropolymer dressing in this experiment. When a 2 Newton pulling force was applied to the dressings, the hydrofoam demonstrated a 11 Newton reading compared with 2 Newtons for both the hydrocolloid and the control. This demonstrates that the hydrofoam was able to deform, preventing the transmission of force to the underlying structure (Ohura et al, 2005).

These laboratory results are backed up with clinical observations, although in the paper there are few indications as to how these were achieved. In clinical practice, Ohura et al (2005) identified that the hydrocolloid worked well in dry conditions, its low friction coefficient improving its overall performance. However, when the hydrocolloid was wet, adhesion was lost — this resulted in adhesion to undergarments and bedding, leakage of exudate, and faecal ingress. The hydropolymer did not perform well, with rucking and twisting of its central area. In addition, it had the highest friction coefficient and rapidly lost adhesion when wet. The hydrofoam, however, performed very well, demonstrating adhesion when both wet and dry, as well as good absorbency and low shear transmissibility.

In a more recent experimental study also headed by Ohura et al (2008), the researchers used a porcine skin model to measure the impact of external shear force and pressure on the superficial skin and subcutaneous layers covering an underlying bony prominence. The evaluation also aimed to verify how the influence of these external forces can be reduced after dressings are applied. Within the study, five dressing products in three groups were evaluated against a control (nil product). These were:

- **Group 1**: a hydrocellular foam (Allevyn Adhesive), and a hydropolymer (Tielle)
- **Group 2**: two polyurethane film products (Tegaderm™ [3M Health Care] and Opsite™ [Smith & Nephew])
- **Group 3**: a hydrocolloid (Duoderm CGF).

**Table 1**

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<th>Electronic data sources</th>
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Measurements of pressure in the subcutaneous tissues and shear in the superficial and subcutaneous tissues were taken. The results showed that although all the dressing materials proved to be effective in reducing pressure and shear in the subcutaneous layer compared with the control, film dressings and hydrocolloid dressings were more effective than hydropolymer and hydrocellular dressings. It was also possible to demonstrate how shear is transmitted to the subcutaneous tissues. The authors concluded that reducing shear force and pressure would be clinically important when looking to reduce pressure ulcer formation (Ohura et al, 2008).

An experimental study by Ashford et al (2001) reported on the pressure-relieving properties of four wound dressings. This preliminary study was undertaken to assess the products’ suitability to provide pressure relief in the management of foot ulcers.

In a laboratory, samples of Allevyn, Biatain™ (Coloplast), Lyofoam™ (Möltycke) and Tielle were subjected to dry and wet compression tests, shearing tests and a cyclical test. In the dry compression test, samples of all four dressings were subjected to a downward force (pressure) of known magnitude. The wet test involved undertaking the same process but with the dressings both saturated...
and dampened with water. During the shear test, samples were exposed to measured lateral force until the dressing materials failed. In the cyclical test, each sample of dressing material was exposed to a maximum force of 500 Newtons, which was repeatedly applied and removed 1,000 times during the course of the test. The thickness of the materials was also compared before and after this ‘stressing’ (Ashford et al, 2001).

The results of the tests showed that all the dressings performed differently under different test conditions. Lyofoam was the dressing that deformed the most during compression testing, Allevyn and Tielle withstood the greatest shear deflection before failure, whereas Biatain withstood the greatest shear force. Overall, Allevyn was the most consistent performer. However, the authors pointed out that further in vitro studies need to be undertaken to substantiate these findings and, most importantly, that real-life clinical environments are very different from those found in a laboratory (Ashford et al, 2001).

Akimoto et al (2007) undertook an experimental study using a two-dimensional finite element mechanical analysis of a human model seated on a thin cushion pad with a range of hardness values (i.e. Young’s modulus). The results showed that in all of the cushion pad models, the peak value of effective stress was less than that of the control model without a cushion pad.

Akimoto et al (2007) were also able to show that as cushion pad softness was increased, the measurements for stress distribution became more diffuse.

These results suggest that the use of a thin cushion pad is an effective way to prevent the development of pressure ulcers. Although not looking at dressings per se, these data could be applied to a similar effect for dressings of the same dimensions/structural components, particularly if combined with the findings of studies on adhesive products.

**Anecdotal clinical evidence**

For several years, the authors have observed nurses in various healthcare settings (e.g., acute, community hospitals, nursing homes, leg ulcer clinics) using proprietary film dressings to reduce friction in vulnerable areas, for example, over patients’ external malleoli, hips or sacrum. Wound product manufacturers, and national or local healthcare institute wound formularies, i.e., British National Formulary (Royal Pharmaceutical Society of Great Britain and the BMJ Group, 2009), also include statements about the use of such dressings in attempting to reduce friction or shear. Nevertheless, such practices or formulary lists do not in themselves constitute a body of scientifically developed research and what little clinical ‘evidence’ exists should always be reviewed.

**Miscellaneous pressure ulcer studies**

Nasal bridge pressure ulcers can occur as a result of the use of nasal intermittent positive pressure ventilation (NIPPV), which can provide symptomatic control and improved quality of life for patients with acute and chronic respiratory failure. In a comparative clinical study undertaken by Callaghan and Trapp (1998), a control group (n=10) receiving NIPPV without protective dressings was compared to a group (n=10) using a hydrocolloid dressing (Granuflex™, ConvaTec), and another group (n=10) using a protective gel pad (Spenco Dermal™, Spenco).

The results demonstrated that 90% of the patients experienced skin deterioration when no protective dressing was used; 70% deteriorated when using the gel pad; but only 30% deterioration occurred with the hydrocolloid dressing. While the mechanisms of pressure damage associated with NIPPV use are more acute, the results do demonstrate the potential beneficial effects of short-term usage of dressings to prevent tissue damage (Callaghan and Trapp, 1998).

**Heel pressure ulcer studies**

Heel pressure ulcers have a high incidence and an increased risk of development when associated with various conditions, such as peripheral vascular disease and diabetes (Fowler et al, 2008).

Preventative measures, such as maintaining the patient in a semi-recumbent posture using a profiling bed with a knee gatch assembly, can reduce pressure on the heels. However, in most cases preventative measures are limited to simple pressure-relieving devices such as the Odstock Wedge (Invacare), which is placed behind the knees infilling the natural hollow in the popliteal space. This supports the underside of the thighs and calves, reducing the pressure under the heels without increasing pressure on the thighs and calves. Furthermore, by maintaining the patient in the semi-recumbent position, the support under the thighs reduces the tendency for the patient to slip down the bed, thereby reducing shearing forces on the sacrum.

The general consensus of opinion is that total heel off-loading, for example using pillows and specialist off-loading devices, is the only effective method of heel ulcer prevention (Cadue et al, 2008; Fowler et al, 2008).

However, the literature does indicate that dressings have been used to prevent heel pressure ulcers with a good deal of success. For example, Nakagami et al (2006) performed a clinical study on 30 elderly patients, which evaluated whether an ulcer-preventive dressing and a thin film dressing could reduce shear force on the heel. The results indicated that a dressing with a low-friction external surface could significantly reduce shear force (P<0.01, Wilcoxon signed-rank test) (2.2 +/- 1.4 Newtons in the preventive dressing and 11.7 +/- 5.8 Newtons in the film dressing). However, results also suggested that these external dressings do not significantly reduce interface pressures and cannot be used as a substitute for heel elevation in an immobile patient (Nakagami et al, 2006).

More recently, the same investigators undertook an evaluation...
of a semi-occlusive dressing containing ceramide 2 (one of the nine naturally occurring lipids found in the subcutaneous layer of the skin and marketed as the Remois Pad, Alcare Corp, Japan). The study featured 37 elderly patients at risk of pressure ulcer development. The findings demonstrated that no pressure ulcers occurred in either the intervention or control area (in the study, each bedridden patient had the product applied to one trochanteric region while the contralateral hip was used as the control). However, there was a significantly lower incidence of persistent erythema within the intervention area than the control area (P=0.007, RR 0.18 [95% CI: 0.05–0.73] and NNT 4.11 [2.50–11.63]). The authors concluded that this dressing was effective in preventing pressure ulcers in patients with highly prominent bones and dry skin (Nakagami et al, 2007).

In 2004, and following several years of investigations, Bots and Apothecker undertook a trial evaluating whether a self-adhesive hydropolymer dressing (Tielle, Systagenix) could reduce the incidence of heel damage. The study evaluated Tielle’s effectiveness in preventing heel pressure ulcers in a wider surgical patient population (n=140). This demonstrated a reduction in heel pressure ulcer prevalence from 36.5–8.5% (a total reduction of 76.7%).

However, some technical problems were found with the dressing in this study (distortion and detachment), which necessitated the use of secondary cotton tubing. The authors considered it necessary to continue monitoring the effectiveness of the intervention (Bots and Apothecker, 2004).

A study carried out in an emergency department in eastern Australia (Sansom and Flynn, 2007) followed the progress of a group of patients (n=100) considered at risk of pressure ulcer development. Members of the group had a heel-shaped foam dressing (Allevyn Heel™, Smith & Nephew) applied prophylactically. A random selection of these patients (n=20) were followed-up two weeks later and none had gone on to develop pressure damage. Although uncontrolled, this study did indicate that the dressing has potential in pressure ulcer prevention and that clinicians are considering dressing products as a preventative intervention.

Another heel-shaped dressing study featuring Allevyn Heel was conducted in Spain (Torra i Bou et al, 2002). In this randomised controlled trial, 130 patients were assigned either to a standard care or preventative dressing group. Altogether, 111 patients completed the study, which demonstrated that 44% of patients in the control group exhibited pressure damage compared to 3.3% of patients in the intervention arm. This was considered highly significant (P=<0.001) and was said to prove that the use of this product was effective in reducing the incidence of pressure ulcers when compared with traditional prevention methods.

An early clinical study looked at the use of a film dressing (OpSite™) to prevent pressure ulcer formation in elderly orthopaedic patients (Hall, 1983). At first the results looked promising — in the test group, which was comprised of 18 patients on one ward, the film dressing was applied to all pressure points and resulted in a pressure ulcer incidence of 5.5%. In the control group, which comprised 16 patients on another ward, a higher number of patients (43.7%) developed pressure ulcers. However, closer evaluation of the data indicated that there were discrepancies between treatment regimens and quality of care between the two groups, which in all probability made significant contributions to the variations in pressure ulcer incidence (Hall, 1983).

Sacral pressure ulcer studies

In a recent clinical case study series, the use of an absorbent soft silicone self-adherent bordered foam dressing was evaluated for its ability to decrease sacral pressure ulcers in a surgical trauma intensive care unit (ICU) (Brindie et al, 2009). The baseline incidence of pressure ulcers in the ICU was stated as between 8 and 12%.
5–24%. In one three-month period, 93 patients were admitted to the ICU and of these 41 were identified as ‘high risk’ using a customised tool. The patients were then treated with the soft silicone prophylactic dressing (Mepilex® Border Sacrum [Mölnlycke Health Care]). The ultimate outcome of the study was a zero incidence of sacral pressure ulcers in those using this dressing (Figure 1).

Brindle et al (2009) stated that the dressing had qualities that were beneficial in the prevention of sacral pressure ulcers, for example:
- Excellent absorption capabilities
- Atraumatic adhesion technology
- An occlusive outer covering
- Shape that covers the sacrum and separates the gluteal folds.

In addition, it was postulated that the dressing may reduce friction, shear and moisture by (Brindle et al, 2009):
- Preventing friction between the gluteal skin folds
- Absorbing moisture collection on the intact skin
- Providing a barrier between the bed surface and the skin for patient positioning
- Allowing for routine skin assessments and removal without skin trauma because of the silicone technology
- Resisting minor faecal incontinence due to the occlusive outer layer.

Discussion
Both in the laboratory and in the clinical environment, there appears to be growing evidence that topical dressings can help to prevent the development of pressure ulceration. This could have serious implications for treatment.

Heel ulceration
Pressure ulcers on and around the heel are the second most common pressure ulcers seen in healthcare (Clark et al, 2004), and their impact can be enormous. The absence of deeper soft tissue and the proximity of bone to the skin’s surface mean that when ulceration occurs it is likely to be severe. This may lead to osteomyelitis and even limb amputation (Black, 2004).

Pressure damage can also have a catastrophic effect on patients’ mobilisation and rehabilitation due to the difficulty in wearing footwear; pain and the need to prevent deterioration by off-loading. However, the frequent occurrence of pressure ulcers on the heel implies that successful clinical prevention strategies are not being widely employed. Even where research projects are implemented, the lack of standardised heel-based protocols and management support can lead to failure (McElhinny and Hooper, 2008).

Sustained pressure to the heel occurs as a result of limb immobility, either through a lack of motor activity or pressure and pain sensation (e.g. following neurological injury or anaesthesia). While the plantar surface of the foot is anatomically adapted to cope with high levels of pressure (Cichowitz et al, 2009), the heel does not have the same structure and is more vulnerable (Donnelly, 2001). Friction and shear are particularly prevalent in the heel and Jay (1995) argued that these account for the occurrence of tissue damage, even when support surfaces deliver interface pressures below capillary closing pressure.

Friction and shear forces impact on the heel as a result of patients moving themselves, movement of the patient by the nurse or movement by the position or action of the bed (Read, 2001). This may explain the phenomenon seen by the authors where patients undergoing lower limb orthopaedic procedures can develop heel damage on the contralateral limb.

While the provision of pressure-relieving equipment for patients who have to remain in bed is commonplace, and there are a wide number of seating products available, preventative interventions for chair-bound patients are rare (Gebhardt and Bliss, 1994).

A cost-effective conforming dressing with a low friction coefficient (thereby reducing shear force), which could also contour around the surface anatomy to minimise high pressure over the bony prominences of the calcaneum and malleolus, would be a successful adjunct to current care practices.

Sacral/natal cleft damage
Pressure damage most commonly occurs in the sacral and pelvic area (Vanderwee et al, 2007; Barrois et al, 2008). The sacral, and especially the natal cleft area, are particularly difficult areas to treat once pressure...
damage occurs (Beldon, 2008). Dressing fixation and retention is problematic, off-loading is difficult while maintaining normal activities, and the close proximity of the anus means faecal contamination is likely, particularly when incontinence is an issue. This makes secondary infection a significant risk. Anatomically, this area of the body is prone to high levels of shear, especially when sitting or lying in a semi-recumbent position and when transferring between bed and chair (Russell, 1998). The mobility of the buttocks, particularly when muscle tension is lost, creates intense stretching forces, which can severely compromise tissue perfusion.

Preventing this damage is of the greatest importance (Reddy et al, 2006). The fundamental tenants of preventative management need to be employed, including the use of appropriate support devices, patient positioning, moisture reduction with barrier creams, urinary catheters, faecal incontinence collectors, and nutrition (Exton-Smith and Sherwin, 1961; Vanderwee et al, 2005). However, action needs to be taken to reduce friction and minimise shear, as well as relieving prolonged pressure to these vulnerable areas. The use of an appropriately designed low-friction preventative dressing would be a significant benefit.

Elbows
Damage to the tissues around the elbows can occur both while resting in bed and also while leaning on hard surfaces, such as bed-tables and armrests. Like the heel, this area has little soft tissue protection over the bony prominences and once damage occurs bone and joint infection can result. The development of a simple dressing to reduce friction and shear forces, dissipate high points of pressure and protect the structure of the elbow would help prevent in this area.

The future
If there is evidence to support the use of dressing materials in pressure ulcer prevention, then why has this not been widely adopted? One answer of course is that it already has. While the positive effects of modern wound care products are well documented, it is impossible to say how much of this benefit is due to their ability to maintain a positive wound environment and how much is owed to their action on pressure, shear and friction at the wound and peri-wound area.

In addition, many clinicians already use wound care products preventatively by applying foam-based heel dressings or film dressings to vulnerable areas. This is supported by company literature and national and local wound product formularies or pressure ulcer prevention protocols, although how successful this approach is when non-specialised products are employed is difficult to ascertain.

To be successful in clinical practice, any dressing material employed preventatively would need to possess a number of key characteristics, including:

- A low surface friction coefficient: the dressing would need to reduce friction forces on the skin, therefore reducing the risk of desquamation, exposure of the fragile germinative layers and transmission of shear forces to underlying structures
- An adhesive interface that has a high tack but which can be removed atraumatically: the dressing would need to ensure correct placement and product retention even when under stress, to stabilise any fragile epidermis and prevent secondary epidermal stripping on removal
- A high moisture vapour transmission rate and absorbent capability: the dressing would need to take-up excess moisture and prevent epidermal maceration
- A conforming ‘memory core’: this should mould around bony prominences but not flatten-out following repeated exposure to perpendicular stress. It should also be robust enough to prevent material failure under significant shear forces
- Ease of single-handed application: the product would be easy for one carer to apply, e.g. by the patient themselves or their informal carer
- Availability in a range of shapes: the dressing would need to fit a variety of anatomical ‘at-risk’ areas
- Cost-effective: this would make it an affordable option for widespread adoption
- Robust in vivo and in vitro research data: the dressing would need to support clinical use and convince clinicians of its effectiveness, therefore speeding its adoption as an appropriate pressure ulcer prevention strategy.

Conclusion
Over the past 30 years, clinicians have sought hi-tech solutions to the problem of pressure ulcer prevention, and manufacturers have developed a range of equipment designed to reduce the causative mechanisms of pressure damage at the patient/support surface interface. Although this approach has had some success, it comes at a price and there are still a significant number of patients for whom access to equipment is difficult.

The results of the studies above indicate that there is a role for dressing materials in pressure ulcer prevention, although further targeted research is needed to support this. Certainly, the clinical experience of the authors of this two-article series, the damning published data on pressure ulcer occurrence, the
financial costs to healthcare providers, and the amount of patient suffering, indicate that there is a need to consider an alternative approach to pressure ulcer prevention. Ideally, this would involve both existing wound care products that can reduce friction and shear as well as next generation dressings, which have been specifically developed for this purpose.

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References


Hall (1983) Prophylactic use of op-site on pressure areas. Nurs Focus 1: 148


Key points

- Wound dressings have been used to help prevent pressure damage.
- Authors have regularly indicated the use of dressings from anecdotal experience.
- There is a body of evidence to support the use of dressings to prevent pressure damage.
- There are a number of clinical situations where dressing-based pressure ulcer prevention may be appropriate.