international
REVIEW

PRESSURE ULCER PREVENTION

pressure, shear, friction and microclimate in context

a consensus document

international
REVIEW

MANAGING EDITOR:
Lisa MacGregor

EDITOR, WOUNDS INTERNATIONAL:
Suzie Calne

PUBLISHER:
Kathy Day

PRODUCTION:
Alison Pugh

PRINTED BY:
Printwells, UK

PUBLISHED BY:
Wounds International
Enterprise House
1–2 Hatfields
London SE1 9PG, UK
Tel: + 44 (0)20 7627 1510
Fax: +44 (0)20 7627 1570
info@woundsinternational.com
www.woundsinternational.com

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EXPERT WORKING GROUP

Mona Baharestani, Wound Care Specialist/Education & Research, James H Quillen Veterans Affairs Medical Center, Johnson City, Tennessee, USA; and Clinical Associate Professor, Quillen College of Medicine, East Tennessee State University, Johnson City, Tennessee, USA

Joyce Black, Associate Professor, University of Nebraska Medical Center, College of Nursing, Omaha, Nebraska, USA

Keryln Carville, Associate Professor Domiciliary Nursing, Silver Chain Nursing Association & Curtin University of Technology, Osborne Park, Western Australia

Michael Clark, Independent Consultant, Cardiff, UK

Janet Cuddigan, Associate Professor, Chair, Adult Health and Illness Department, College of Nursing, University of Nebraska Medical Center, Omaha, Nebraska, USA

Carol Dealey, Senior Research Fellow, University Hospitals Birmingham NHS Foundation Trust and University of Birmingham, Queen Elizabeth Hospital, Birmingham, UK

Tom Defloor, Full Professor, Unit Nursing Science, Department of Public Health, Ghent University, Belgium

Amit Gefen, Associate Professor, Department of Biomedical Engineering, The Iby and Aladar Fleischman Faculty of Engineering, Tel Aviv University, Israel

Keith Harding, Professor of Rehabilitation Medicine (Wound Healing), Head of Department of Dermatology and Wound Healing, Cardiff University, Cardiff, UK

Nils Lahmann, Associate Professor, Department of Nursing Science, Charité Universitätsmedizin Berlin, Berlin, Germany

Maarten Lubbers, Surgeon, Department of Surgery, Academic Medical Center, University of Amsterdam, The Netherlands

Courtney Lyder, Dean and Professor, School of Nursing, Assistant Director for Academic Nursing, Ronald Reagan UCLA Medical Center, University of California, Los Angeles, USA

Takehiko Ohura, Chair, Pressure Ulcer and Wound Healing Research Center (Kojin-kai), Sapporo, Japan

Heather L Orsted, Director - CAWC Institute of Wound Management and Prevention and Clinical and Educational Consultant, Canadian Association of Wound Care, Calgary, Alberta, Canada

Vinoth K Ranganathan, Program Manager, Department of Physical Medicine and Rehabilitation, Cleveland Clinic, Cleveland, Ohio, USA

Steven I Reger, Director Emeritus, Rehabilitation Technology, Department of Physical Medicine and Rehabilitation, Cleveland Clinic, Cleveland, Ohio, USA

Marco Romanelli, Consultant Dermatologist, Wound Research Unit, Department of Dermatology, University of Pisa, Italy

Hiromi Sanada, Wound, Ostomy and Continence Nurse, Department of Gerontological Nursing/ Wound Care Management, Graduate School of Medicine, University of Tokyo, Tokyo, Japan

Makoto Takahashi, Associate Professor, Biomedical Systems Engineering, Bioengineering and Bioinformatics, Graduate School of Information Science and Technology, Hokkaido University, Sapporo, Japan

DEVELOPMENT AND CONSENSUS PROCESS

The development of this document involved a process of text review by the expert working group and revision by the authors. It culminated in consensus as indicated by sign off from each working group member and author.

Pressure, shear, friction and microclimate in context

HL Orsted, T Ohura, K Harding

The overall goal of clinical care is to restore or maintain health. Unfortunately, however, iatrogenic injuries sometimes occur. Although not all pressure ulcers are iatrogenic, most are preventable. Pressure ulcers are one of the most frequently reported iatrogenic injuries in developed countries. Inappropriate care methods, such as leaving vulnerable patients in potentially damaging positions for long periods of time, or massaging reddened areas of skin, often remain in practice long after evidence has shown them to be harmful or ineffective. Education is critical in ensuring that all members of a clinical team act to prevent and treat pressure ulcers according to the best evidence available.

The most recent definition of pressure ulcers, which has been produced by an international collaboration of the National Pressure Ulcer Advisory Panel (NPUAP) and the European Pressure Ulcer Advisory Panel (EPUAP), highlights current understanding of the role of extrinsic factors in the development of pressure ulcers[1,2] (Box 1). Pressure, which is often related to decreased mobility, has long been viewed as the most important extrinsic factor in pressure ulcer development. However, recent and ongoing research is revealing that shear, friction and microclimate also have important roles, and that there are significant and complex relationships between all of the extrinsic factors. For example, pressure and shear are closely linked, friction has a role in the development of shear, and microclimate influences the susceptibility of skin and soft tissues to the effects of pressure, shear and friction.

The concepts involved in understanding pressure, shear, friction and microclimate and their synergistic actions in the formation of pressure ulcers are complex. Consequently, the expert working group involved in producing *Pressure ulcer prevention: prevalence and incidence in context*[3] proposed a new document to aid understanding of these extrinsic factors. The expert working group decided that, even though pressure, shear, friction and microclimate are inextricably inter-related, this new project would tackle each extrinsic factor individually with the aim of building understanding of the physics involved. This understanding should enable clinicians to better comprehend developments in the field and, most importantly, will underpin effective and consistent implementation of pressure ulcer prevention protocols.

The three papers – *Pressure in context*, *Shear and friction in context*, and *Microclimate in context* – follow a similar structure. They start by defining the relevant extrinsic factors and how individually they contribute to the aetiology of pressure ulcers. The relationships between the factors are explained and emphasised, and the evidence for the role of the factors in the development of pressure ulcers is discussed. The latter sections of the three papers describe how patients at risk from each extrinsic factor can be identified. The papers then explain the types of and rationale for the clinical interventions that aim to prevent or ameliorate the adverse effects of each of the extrinsic factors discussed. It should be noted that, although the document covers many major facets of pressure ulcer prevention, discussion of comprehensive prevention protocols is beyond its scope.

Much research remains to be undertaken to further develop our understanding of the intrinsic and extrinsic causes of pressure ulcers. But as this document shows, there are some important underlying principles for preventing pressure ulcers resulting from the extrinsic factors of pressure, shear, friction and microclimate. All clinicians should understand these principles and implement them in their daily practice.

REFERENCES

1. National Pressure Ulcer Advisory Panel and European Pressure Ulcer Advisory Panel. *Prevention and treatment of pressure ulcers: clinical practice guideline.* Washington DC: National Pressure Ulcer Advisory Panel, 2009.
2. European Pressure Ulcer Advisory Panel and National Pressure Ulcer Advisory Panel. *Prevention and treatment of pressure ulcers: quick reference guide.* Washington DC, USA: National Pressure Ulcer Advisory Panel, 2009. Available from: www.npuap.org and www.epuap.org (accessed 23 November 2009).
3. *International guidelines. Pressure ulcer prevention: prevalence and incidence in context. A consensus document.* London: MEP Ltd, 2009.

BOX 1 New NPUAP/EPUAP definition of pressure ulcers[1]

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

Pressure in context

M Takahashi, J Black, C Dealey, A Gefen

INTRODUCTION

Pressure has been recognised as the most important extrinsic factor involved in the development of pressure ulcers for many years. Consequently, it features prominently in definitions of pressure ulcers, including the recent definition produced by the National Pressure Ulcer Advisory Panel (NPUAP) and European Pressure Ulcer Advisory Panel (EPUAP)[1,2].

This paper explains what pressure is, how pressure contributes to pressure ulcer formation and how to identify patients at risk of injury from pressure. It then describes the rationale and mode of action of interventions that reduce the magnitude and duration of pressure and, consequently, the risk of pressure ulcer development.

FACT FILE

- Force is a concept that is used to describe the effect on an object by an external influence. Force has a direction and a magnitude.
- Perpendicular forces cause pressure.
- The pressure at the junction between the skin and a support surface is often called 'interface pressure'.

WHAT IS PRESSURE?

Pressure is defined as the amount of force applied perpendicular to a surface per unit area of application.

A force applied over a small area will produce greater pressure than the same force applied over a larger area (Figure 1). The unit of force is the newton (N). The unit of pressure is newtons per square metre (N/m^2), pascals (Pa) or millimetres of mercury (mmHg).

In addition to the perpendicular force that is involved in pressure, forces may be applied parallel to the skin surface (Figure 2). These are shear forces and contribute to shear stresses, which are also measured in terms of force per unit area (see: *Shear and friction in context*[3], pages 11–18). 'Stress' is a generic name for effects that are defined in terms of force per unit area of application.

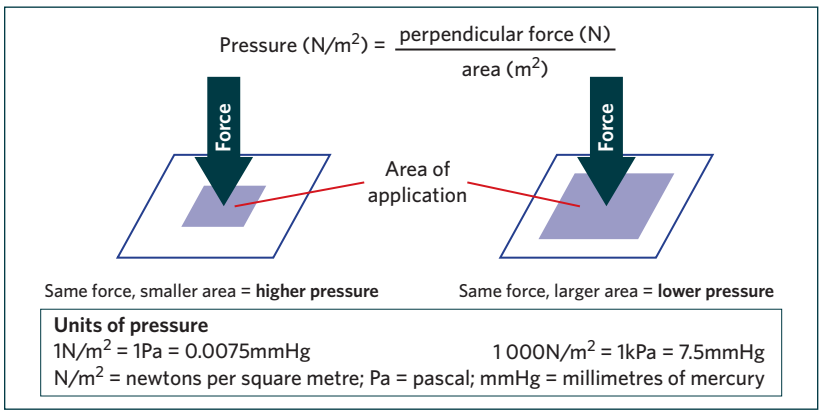


FIGURE 1 Definition of pressure

What types of internal stresses does pressure cause?

When pressure is applied to skin, particularly over a bony prominence, it distorts the skin and underlying soft tissues. In the model in Figure 3, the horizontal lines immediately under the bony prominence become closer together indicating tissue compression. In other places, particularly under the bony prominence, the lines are also elongated, indicating tensile (stretching) and shear (distorting) stresses. **This means that even when only pressure is applied (ie the force applied is only perpendicular), tensile and shear stresses also occur within the tissues near bony prominences[4].**

CLINICAL EFFECTS OF PRESSURE

In alert patients, the effects of continuous pressure usually signal frequent small body movements to relieve the load and restore tissue perfusion[5]. Patients who are unconscious, sedated, anaesthetised or paralysed cannot sense or respond to these signals and do not move spontaneously. As a result, the skin and soft tissues can be subjected to prolonged and unrelieved pressures.

Pathophysiology of pressure damage

Skin that has been subjected to potentially damaging levels of pressure initially appears pale from reduced blood flow and inadequate oxygenation (ischaemia). When the pressure is relieved, the skin quickly becomes red due to a physiological response called reactive hyperaemia. If the ischaemia has been sufficiently short lived, blood flow and skin colour will eventually return to normal.

More prolonged ischaemia can cause blood cells to aggregate and block capillaries, perpetuating the ischaemia. Capillary walls can also become damaged, allowing red blood cells and fluid to

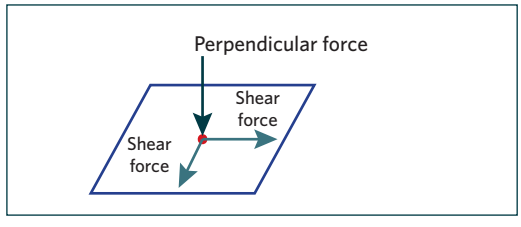


FIGURE 2 Forces applied to a surface

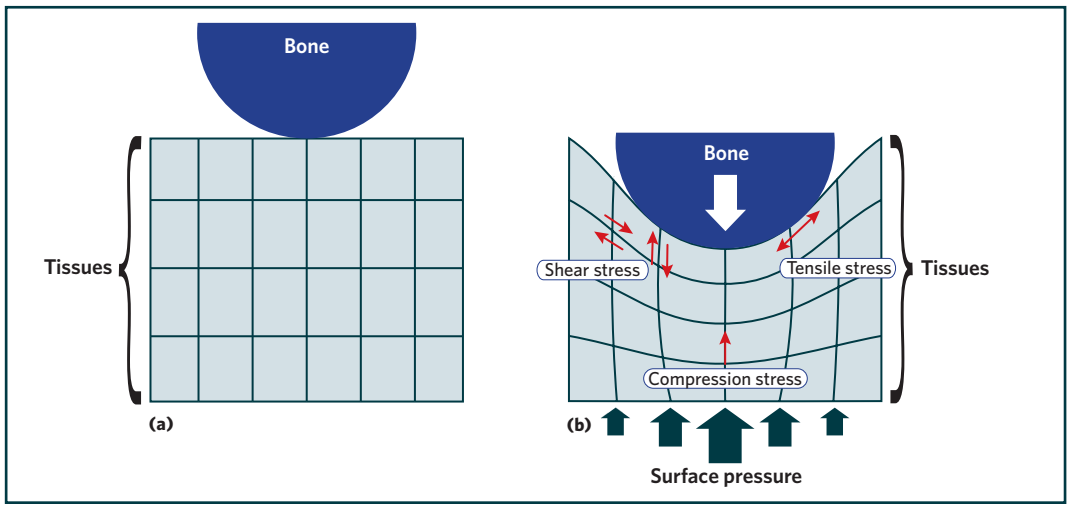
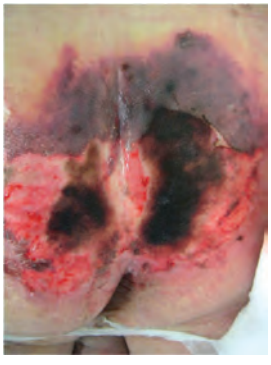


FIGURE 3 Tissue distortion due to pressure (adapted from[6])
Bending of the lines in (b) shows that when external pressure is applied over a bony prominence, compressive, shear (distorting) and tensile (stretching) stresses occur (see bold text on page 2).

FACT FILE

- Localised pressure is thought to contribute to pressure ulcer development by deforming skin and soft tissues, often between a bony structure and an external surface (such as a bed or a chair), thereby distorting cells, reducing blood flow and inducing ischaemia and necrosis.
- Although capillary closing pressure – ie the pressure which halts capillary blood flow – is often quoted to be 32mmHg, it is highly variable.

FIGURE 4 Deep tissue injury (courtesy of J Black)
DTI over the sacral area acquired during a long surgical procedure. It has progressed so that there is now loss of skin and exposure of necrotic subcutaneous tissue.



leak into the interstitial space. This process results in the non-blanchable erythema, skin discolouration and induration that are seen with Category/Stage I pressure ulcers. Continued ischaemia results in necrosis of the skin and underlying tissue, and the superficial and deeper tissue breakdown seen with higher category/stage pressure ulcers.

High pressure is also known to physically damage muscle tissue by deforming and rupturing muscle cells.

Deep tissue injury

The new NPUAP/EPUAP pressure ulcer classification contains an additional category for use in the USA: deep tissue injuries[1]. Clinical experience suggests that these usually present with purple skin around 48 hours following a pressure event, eg being unconscious on the floor, and become necrotic quickly, even when care is provided (Figure 4).

WHAT DO WE KNOW ABOUT PRESSURE AND PRESSURE ULCERS?

Because the primary mechanism of pressure-induced tissue damage is thought to be blood flow reduction, papers that discuss pressure ulcers frequently mention research done in the 1930s by Landis. This work found that the pressure in the arteriolar limb of a capillary in the human finger was on average 32mmHg[7]. This value was then mistakenly generalised to be the pressure required to compress capillaries to prevent blood flow (the capillary closing pressure), and the pressure below which pressure redistributing devices aimed to reduce interface pressure. However, many following studies also demonstrated a wide range of pressure in capillaries at various anatomical locations, with values dependent on age and concomitant disease.

Relationship between duration and intensity of pressure

By the middle of the 20th century, duration of pressure was suspected to be a factor in pressure ulcer development[8,9], but quantitative data were missing until Kosiak started to publish his experiments in 1959. These involved loading tissues with known pressures for specific durations. Histological examination was used to assess tissue viability[10,11].

Kosiak reported a relationship between amount of pressure, duration of application and the development of tissue damage in canine and rat experiments[10,11]. He stated that, "microscopic pathologic changes were noted in tissues subjected to as little as 60mmHg for only one hour"[10].

Pressure–time curve

In the 1970s, Reswick and Rogers published guidelines based on human observations that depicted non-injurious and injurious levels and lengths of exposure to particular interface pressures[12] (Figure 5, page 4). Although consistent with Kosiak's work, the curves at the extremes of the timescale were based on extrapolation rather than data[13,14].

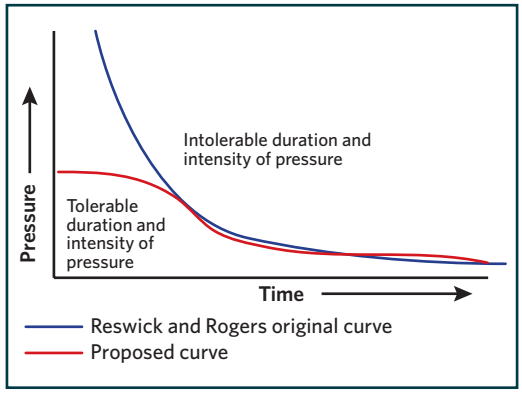


FIGURE 5 Proposed modification to Reswick and Rogers pressure–time curve (adapted from[1,15-17])
The area above the curves represents durations and intensities of pressure that are likely to result in tissue damage; the area below the curves represents durations and intensities of pressure that are unlikely to result in tissue damage.

FACT FILE

- The ability of pressure to cause tissue damage is related to *duration* of application and *intensity* (amount) of pressure applied.
- Pressure on the skin has been shown to produce greater reductions in blood flow in a deep artery than in skin capillaries.
- Interface pressure is relatively easy to measure, but has limitations as a predictor of internal tissue stresses.
- When pressure is applied over a bony prominence, the internal stresses are highest in the soft tissues closest to the bony prominence.
- Patients at highest risk from pressure are those who are unable to move themselves or to ask to be moved.

Recently, it has been proposed that the Reswick and Rogers curve be modified to reflect more recent animal studies and clinical experience that high pressures can cause pressure damage within a relatively short time, but that lower pressures can be applied for long periods without damage occurring[1,15-17] (Figure 5).

Pressure and temperature

The effects of pressure may be modulated by skin temperature. Work by Kokate *et al* and Iaizzo *et al* in pigs concluded that skin and soft tissue damage due to pressure could be reduced by localised skin cooling[18,19] (see: *Microclimate in context*[20], pages 19–25).

Physiological effects

In an experiment to measure the effects of pressure on blood flow in the human forearm, pressure ranging from 0 to 175mmHg was applied to the skin. The results showed that blood flow was affected by pressure on the skin to a greater extent in a deep artery than in a skin capillary[21]. Future investigations to measure **deep** tissue blood flow may contribute to understanding of the ischaemic factors in the mechanism of pressure ulcer formation.

How can internal stresses be measured?

Many studies investigating the role of pressure in the development of pressure ulcers measure pressure at the skin surface (interface pressure). Even so, bioengineering work carried out since the 1980s has indicated that internal tissue stresses cannot be predicted by means of interface pressure measurements[13,14].

Stresses within tissues measured in an animal model demonstrated that pressure is three to five times higher internally near a bony prominence than the pressure applied to the skin over the prominence[22]. Computer modelling has confirmed that the highest stresses are near the bony prominence[23].

IDENTIFYING PATIENTS AT RISK FROM PRESSURE

Patients at highest risk from pressure are those in whom pressure on skin would go unrelieved if the healthcare staff did not move them in a bed or chair. Asking the question, "Can the patient feel pressure **and** move about or ask others to move him?" is an important first step. When the answer to the question is, "No", high risk patients can be identified quickly by all staff.

General patient assessment will indicate other factors, eg reduced tissue perfusion or poor nutrition, which may make a patient more vulnerable to the effects of pressure. Some of these factors increase risk by amplifying the effects of shear and friction, or by reducing skin and tissue tolerance to pressure (see: *Shear and friction in context*[3], pages 11–18 and *Microclimate in context*[20], pages 19–25).

Several tools are available for assessing overall risk of pressure ulcer development; these are based on a number of factors, including pressure[24-26]. Although there are limitations to the use of such tools[1] and alternative approaches have been suggested[27], risk assessment tools are highly valued in clinical practice.

REDUCING RISK FROM PRESSURE

Best practice care of patients at risk of pressure ulceration has numerous facets that aim to ameliorate the effects of intrinsic risk factors (such as poor nutrition, concomitant disease, dry skin) and extrinsic factors (such as shear and friction, or incontinence). (See: *Shear and friction in context*[3], pages 11–18 and *Microclimate in context*[20], pages 19–25.)

With respect to **pressure**, efforts centre on reducing or removing the pressure applied to the skin of vulnerable patients. The principles involved also apply to patients with existing pressure damage. Patients should avoid sitting or lying on areas of non-blanchable erythema or pressure ulcers. If such areas or wounds fail

to improve or deteriorate, practitioners must consider whether continued pressure over the area is contributing to the problem.

Clinical judgement is essential in determining how best to provide care for patients at risk from damage by pressure.

FACT FILE

Interventions intended to reduce a patient's risk from the effects of pressure must:

- be planned in the context of other care and treatment requirements
- centre on encouraging patients to move independently, patient repositioning and the use of support surfaces
- take into account all of the patient's needs, especially when determining the frequency of repositioning and positions used.

PRESSURE REDISTRIBUTION

Pressure redistribution can be achieved by removal of pressure from the affected part of the body or by reducing pressure by spreading weight more widely (Figure 6).

Independent movement

Spontaneous movement is the usual mode of pressure relief for persons with intact neurological systems. An early study found that patients who moved spontaneously fewer than 25 times each night were at significantly higher risk of pressure ulcers than those who moved more frequently[30].

Wherever possible, patients should be encouraged to move themselves. For patients who move spontaneously, sometimes no additional repositioning is needed. Patients who are reluctant to move, due to actual or anticipated pain with movement, or because of the sedative effects of analgesia, need to be reminded to move.

The impact of making small, frequent movements has been studied by testing the idea that nursing staff could move a patient slightly with each contact, eg by lifting a leg or moving an arm, to relieve pressure[31,32]. The studies suggested that interface pressure was reduced under the areas moved[31], and in a small study a reduction in the number of pressure ulcers was observed[32]. However, caution must be applied: unless the heels and pelvis are moved, such body movements do little to reduce pressure intensities and durations at these critical locations.

Repositioning

Repositioning should be considered for all those deemed to be at risk of pressure ulceration[1,33]. More mobile patients will be able to reposition themselves (see above), but others may require assistance.

Repositioning may not be suitable for all patients: some patients in a critical condition may be destabilised by repositioning. However, this is not always the case even in patients in poor haemodynamic condition[34]. Therefore, the decision to reposition a critically ill patient should be individualised.

Frequency of repositioning

A systematic review of pressure ulcer prevention strategies found insufficient evidence to support a specific repositioning regimen[35]. The frequency of repositioning should be based on the patient's tissue tolerance, level of mobility, general medical condition and the support surface in use[1]. The traditional 2-hourly repositioning regimen may provide a useful starting point from which frequency can be adjusted. An effective repositioning regimen will be indicated by the absence of persistent erythema over bony

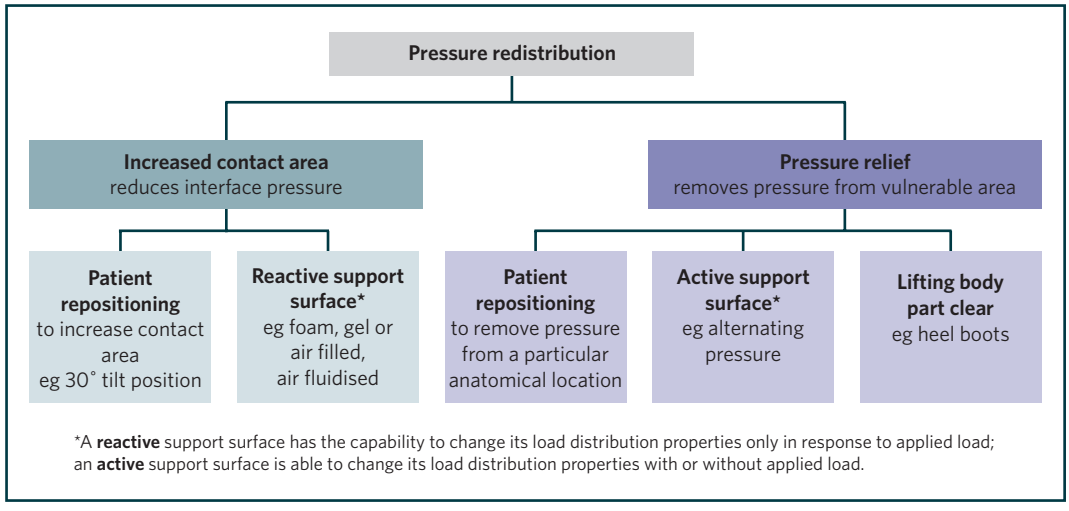


FIGURE 6 Methods of pressure redistribution (adapted from[28,29])

prominences. If persistent erythema occurs, this may indicate that more frequent repositioning is required and that the current support surface is perhaps not optimal for the patient.

The use of a pressure redistributing support surface does not eliminate the need for repositioning. However, it may be possible to reduce the frequency of repositioning. In one study, for example, 4-hourly turning on a viscoelastic foam mattress was associated with lower incidence of Category/Stage II and above pressure ulcers when compared with 2- or 3-hourly turning on a standard mattress[33].

For patients sitting in chairs and wheelchairs, it is advised that repositioning should occur at a minimum every hour[36]. Patients confined to wheelchairs should be taught to reposition every 15 minutes by doing 'push-ups' off the wheelchair or by leaning forwards[37].

Positions

For patients in bed, positions such as 90° side-lying or the semi-recumbent position are best avoided because these increase pressure over the trochanteric or sacral bony prominences respectively[1]. Patients who must have some head of bed elevation, eg because of dyspnoea or to prevent aspiration during tube feeding, should be repositioned more frequently.

The 30° tilted side-lying position is a method of placing a patient so that they are tilted 30° along their vertical axis from the supine position[1]. This position does not suit all patients, but may be a useful alternative for some.

Wheelchair dependent patients may benefit from a tilting wheelchair that helps to offload pressure from the ischial tuberosities.

PRESSURE REDISTRIBUTING SUPPORT SURFACES

Pressure redistributing support surfaces are available in several forms, eg overlays, mattresses and integrated bed systems.

An overlay is a support surface device placed on top of an existing mattress. These devices may elevate the sleeping surface to the level of the side rails and so the risk of the patient falling out of bed must be evaluated. Ideally, the bedrail should be at least 10cm (4 inches) higher than the surface of the mattress.

Pressure redistributing mattresses can often be used to replace standard mattresses, allowing for continued use of the existing bed frame.

An integrated bed system combines a bed frame and a support surface (usually an alternating pressure mattress). They are most often used for extremely high risk patients, for the treatment of pressure ulcers, and for patients who have had surgical reconstruction of pressure ulcers with flaps.

REACTIVE SUPPORT SURFACES

Two important principles of the mode of pressure redistribution of reactive support surfaces are immersion and envelopment.

Immersion refers to the ability of a support surface to allow a patient to sink into it[29] (Figure 7). As the body sinks in, more of the body comes into contact with the support surface, redistributing the patient's weight over a larger area and reducing pressure.

FACT FILE

Reactive support surfaces, eg foams, air or gel filled, and air fluidised, provide pressure redistribution through immersion and envelopment.

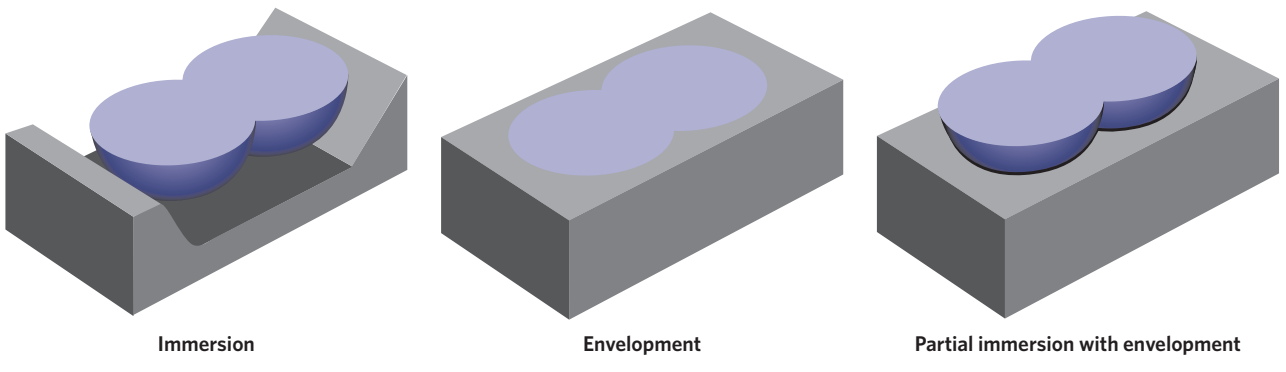


FIGURE 7 Immersion and envelopment

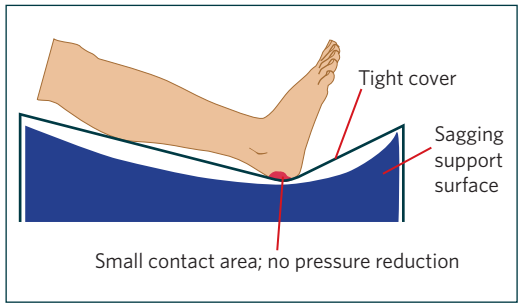


FIGURE 8 Hammock effect
The tight cover prevents immersion and envelopment of the patient, resulting in suspension above the support surface and no pressure reduction.

FACT FILE

- Active support surfaces – also known as alternating pressure systems – redistribute pressure mainly through the inflation and deflation of sections of the support surface.
- The precise indications for and relative efficacy of the different types and models of pressure redistributing support surfaces in reducing the incidence of pressure ulcers remains under investigation.

Immersion is greater on softer surfaces and also has the potential to be higher on thicker surfaces. However, if a material is too soft, the patient may 'bottom out' (ie end up sitting or lying on the underlying structure of the bed or chair because the support surface has become so compressed).

Envelopment refers to how well a support surface moulds to body contours and accommodates irregular areas (such as folds in clothing or bedding)[29] (Figure 7).

Recent research has indicated that the degree of immersion and envelopment of a support surface can be impaired by increased tension at the surface of the support, especially when combined with sagging of the support surface itself[38]. For example, a tight cover over a mattress or seat cushion can create a hammock effect that prevents the support surface moulding to contours and produces high pressures over a small area (Figure 8).

Immersion and envelopment have important implications for patient mobility and independence. For example, it requires relatively little effort to stand from sitting or lying on wood (which has no immersion and envelopment), but the same manoeuvre from water requires more effort because of the high degree of immersion and envelopment.

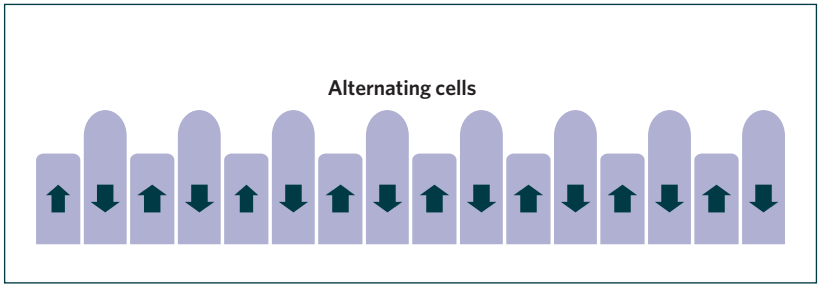


FIGURE 9 Alternating pressure support surface
The air cells cyclically inflate and deflate to periodically remove pressure from soft tissue.

Foam

Basic foam mattresses have become common as the standard mattress for patients in hospitals and long-term care facilities. Higher specification foam mattresses (eg those composed of layers of different densities of foam, or of viscoelastic foam) are recommended to reduce the incidence of pressure ulcers in persons at risk[39].

Foam degrades and loses it stiffness over time, thereby losing its ability to conform. When a foam mattress wears out the patient may 'bottom out'. The life span of any support surface is influenced by number of hours of use and the weight applied; a surface used by thin persons will outlive one used by bariatric patients.

Air or gel filled

Air or gel filled support surfaces comprise air or gel filled columns or compartments. The degree of immersion and envelopment provided depends upon the pressure of the air or gel in the compartments, the depth of the compartments, and the 'give' of the surface.

Air filled support surfaces are sometimes referred to as low air loss surfaces. However, strictly speaking, low air loss relates to a property of some support surfaces that allows air to escape from the cushions to aid management of skin temperature and moisture (see: *Microclimate in context*[20], pages 19-25).

Air fluidised

Air fluidised support surfaces provide the greatest immersion and envelopment of any support surface. Almost two-thirds of the body can be immersed. An air fluidised support surface comprises silicone or glass beads that have pressurised air forced between them. This makes the beads take on characteristics of a fluid.

Several randomised controlled studies have shown that healing outcomes for patients with Category/Stage III and IV pressure ulcers who are managed on air fluidised support surfaces are improved in comparison with standard beds, and foam and other non-fluidised support surfaces[40-43].

ACTIVE SUPPORT SURFACE – ALTERNATING PRESSURE

Alternating pressure support surfaces redistribute pressure by cyclically inflating and deflating zones of the surface (Figure 9). As a result they are less reliant than reactive surfaces on the properties

TABLE 1 Uses of pressure redistributing support surfaces

This table is intended to provide a broad overview of the uses of the different types of pressure redistributing support surfaces. The specifications, quality and usages for individual products may vary. Clinicians should refer to the manufacturer's literature for information about indications, cautions and contraindications for individual products.

Type of pressure redistributing support surface	Patients who may benefit	Notes
Reactive support surfaces		
Higher specification foam	■ Patients who are at low to moderate risk of pressure ulcers due to immobility and inactivity	■ Where possible avoid use of plastic products such as incontinence pads to minimise heat and moisture retention on the skin
Air* or gel filled	■ Patients who are at low to moderate risk of pressure ulcers due to immobility and inactivity ■ Patients who are very heavy or rigid and difficult to reposition ■ Some air filled low constant pressure surfaces can be adjusted for patient weight and weight distribution by adjusting the amount and pressure of air pumped through	■ Accidents have occurred if air cells have suddenly deflated and then reflated, eg following loss of electrical power; ideally should be used when generator back up is available ■ Gel filled support surfaces may increase skin moisture
Air fluidised	■ Patients with existing pressure ulcers who cannot be turned off the ulcer or who have pressure ulcers on two or more turning surfaces (eg sacrum and trochanter) ■ Patients recovering after flap surgery for pressure ulcer repair	■ Patients with large open wounds may become dehydrated because of the large volumes of air moving through the support surface ■ Some patients are not able to tolerate the sensation of floating or the warmth of the surface
Active support surface		
Alternating pressure	■ Patients who cannot be turned side to side or do not move body areas	■ Inflation and deflation can be annoying, especially for certain patient groups, eg those with dementia ■ Patients may feel disturbed by the noise or may feel cold

*Sometimes air filled support surfaces are called low air loss surfaces. Strictly speaking, however, low air loss relates to a property of some support surfaces that allows air to escape from the cushions to aid management of skin temperature and moisture (see: *Microclimate in context*[20], pages 19–25).

FACT FILE

Patients should continue to be repositioned when on a support surface for their comfort and functional ability, as well as for pressure relief, unless medically contraindicated.

of immersion and envelopment to redistribute pressure. The ideal frequency, duration, amplitude and rate of inflation and deflation have not been determined. A draft consensus document has recently proposed a standardised method for evaluating active support surfaces[44].

Iglesias *et al* reported that alternating pressure mattresses were likely to be more cost effective than alternating pressure overlays[45]. In addition, the mean time to develop a pressure ulcer was more than 10 days longer on the alternating pressure mattress than on the alternating pressure overlay[45]. When alternating pressure mattresses were compared with viscoelastic foam mattresses, Vanderwee *et al* found no significant difference in the incidence of pressure ulcers[46]. There was a tendency for more sacral pressure ulcers in patients on alternating pressure mattresses in patients who were identified as being in need of preventive measures based on the Braden scale[46].

A literature review of 15 randomised controlled trials concluded that when taking into account methodological issues, alternating pressure mattresses are likely to be more effective than standard hospital mattresses in the prevention of pressure ulcers[47].

Support surface selection

Selection of a suitable support surface for pressure redistribution (Table 1) should not be based on risk assessment score alone, but should also take into consideration:

- level of mobility within the bed – ie how much the patient can move when in bed and whether they are able or need to be able to get themselves out of bed

FACT FILE

Regular observation is essential in evaluating the efficacy of pressure redistribution strategies: any sign of pressure damage should prompt re-evaluation of the strategies in place.

- patient comfort – some patients find some support surfaces uncomfortable
- need for microclimate management – some support surfaces assist with managing heat and moisture directly below the patient (see: *Microclimate in context*[20], pages 19–25)
- care setting – for example, some integrated bed systems are unsuitable for home settings because of their weight and the need for an alternative power source, eg a generator, in case of loss of electrical power.

Even so, one study has shown that reimbursement guidelines, not patient condition, were most clearly associated with support surface selection[48].

Higher specification foam mattresses (eg viscoelastic foam mattresses) are suitable for many at-risk patients, but those at higher risk will need a powered support surface that is able to change its load distribution properties.

Bariatric patients may be too heavy for some pressure redistributing support surfaces and require versions with extra width or features designed to accommodate high patient weight.

Additional features of integrated bed systems may include lateral rotation or vibration of the support surface to assist patients who have problems with ventilation and perfusion. 'Turn assist' is designed to aid repositioning, examinations and linen changes; it is not intended for patients to use in turning themselves.

OBSERVATION AND RE-EVALUATION

Once pressure redistribution strategies have been set in place, it is important to assess their effectiveness. The most important indicator is the presence or absence of changes in skin status, especially over the bony prominences. If there are indications of pressure damage, the prevention strategies may need to be intensified and/or modified. Changes in the condition of patients and their ongoing risk levels should also be monitored as these may alter the prevention strategies required.

When a specialised support surface is in use, carers should check regularly that the device is working properly and ensure that:

- a foam mattress is still 'springing back' to its original position when pressure is removed
- air filled devices are properly inflated
- gel mattresses have gel throughout them and that there are no areas where gel has been moved away
- an alternating air mattress is inflating and deflating properly
- a powered device is plugged into a power supply.

All support surfaces, hospital beds and integrated bed systems have a finite term of use, but the exact lifespan is currently unknown. Healthcare practitioners need to be mindful of this and when pressure ulcers fail to heal consider whether a 'worn out' support surface may be the cause or play a role.

CONCLUSION

In addition to a direct effect, pressure also acts indirectly through the generation of shear stresses to produce pressure ulcers. The ability of pressure to produce pressure damage in soft tissues is related to the intensity and duration of the applied pressure. Patients who are unable to move or ask to be moved are those most at risk from pressure. Interventions to reduce the effect of pressure and reduce the incidence of pressure ulcers include patient repositioning and the use of specialised support surfaces.

Decisions on which support surface to use can be enhanced through appreciation of how surfaces work and for which patients each device is most suitable. However, despite expert clinical opinion, the choice of support surface is often made on a financial basis. Continued research into the effectiveness of pressure redistributing support systems in reducing the incidence of pressure ulcers will guide educational priorities, aid decision making and help to secure funding for appropriate surfaces, regardless of care setting.

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Shear and friction in context

SI Reger, VK Ranganathan, HL Orsted, T Ohura, A Gefen

INTRODUCTION

Shear and friction are often mentioned alongside pressure in the context of pressure ulcers. For example, the most recent definition of pressure ulcers, produced by an international collaboration of the National Pressure Ulcer Advisory Panel (NPUAP) and European Pressure Ulcer Advisory Panel (EPUAP), emphasises the role of pressure and states that shear can be involved in combination with pressure in the development of pressure ulcers[1]. The same collaboration also cites shear in the context of deep tissue injury, which is defined as "due to damage to underlying soft tissues from pressure and/or shear"[1].

Although disputed as a direct cause of pressure ulcers, friction is considered in this paper because of its close association with shear. Pressure and shear are also intimately linked: pressure on soft tissues, especially when over a bony prominence, will cause some degree of shear through tissue distortion[2,3].

The first part of this paper clearly defines shear and friction and discusses the role of each in pressure ulcer development. The second part of the paper examines how to recognise patients at risk of skin and soft tissue injury due to shear and friction. It then discusses the actions that can be taken to avoid or minimise shear and friction and so complement other measures to reduce the overall risk of pressure ulcer development.

FACT FILE

- Shear stresses arise from forces applied tangentially to a surface and cause deformation of the object involved.
- Shear stresses usually occur in combination with pressure.
- Friction force occurs when two objects rub against each other.
- Friction is not a direct cause of pressure ulcers, but is involved in the development of shear stresses in skin and in deeper tissue layers.

DEFINITIONS

The terminology surrounding shear can be confusing: 'shear' is often used to abbreviate the different terms 'shear stress' and 'shear force'. In addition, shear and friction are often mentioned together in the context of pressure ulcer aetiology, and sometimes, inaccurately, the terms are used interchangeably.

What is shear?

Shear stress results from the application of a force parallel (tangential) to the surface of an object while the base of the object stays stationary. (Note: Pressure is the result of a force that is applied perpendicular (at a right angle) to the surface of an object (see: *Pressure in context*[3], pages 2–10).)

Shear stress causes the object to change shape (deform) (Figure 1). The amount of deformation caused by shear stress is quantified as shear strain.

In common with pressure, shear stress is calculated in terms of the force applied over the area to which it is applied (Box 1) (see: page 14 and Box 2 for more detail). Shear stress is expressed in the same units as pressure: most commonly as pascals (Pa), or sometimes as newtons/square metre (N/m^2).

What is friction?

Friction is defined as the force that resists the relative motion of two objects that are touching, and is measured in newtons (N). However, the term 'friction' is also frequently used to mean the action of one object rubbing against the other (see: page 14 and Box 2 for more detail).

WHAT CAUSES SHEAR STRESSES?

Gravity produces a force that pulls a patient onto the surface they are resting on. The opposing force produced by the surface can be divided into two components:

- a perpendicular component – which results in pressure
- a tangential component – which results in shear stresses (Figure 2, page 12).

BOX 1 Defining shear stress

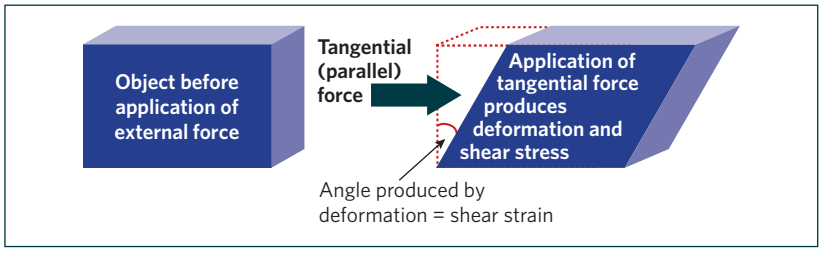
$$\text{Shear stress (pascals or } N/m^2) = \frac{\text{Tangential force applied (N)}}{\text{Area of application of force } (m^2)}$$

$1Pa = 1N/m^2$ $1kPa = 1000N/m^2$

Definitions of shear stress:

- "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 (ie shear strain)."[4]
- "The force per unit area exerted parallel to the plane of interest."[5]

FIGURE 1 Shear stress



Friction contributes to the development of shear stresses by tending to keep the skin in place against the support surface while the rest of the patient's body moves towards the foot of the bed or the edge of the seat. The relative movement of the skin and underlying tissues causes shear stresses to develop in the soft tissues overlying bony prominences such as the sacrum.

The angle of the back support of a bed, or the angle of the backrest of a seat or wheelchair, strongly influences the level of shear stresses in tissues[6,7]. All angles between an erect sitting posture and horizontal lying will cause shear stresses due to the body's tendency to slide downwards along the slope. Lying with the backrest at an angle of 45° will cause a particularly high combination of shear stresses and pressure at the buttocks and sacral area because, in this posture, the weight of the upper body divides equally into perpendicular and tangential forces[6,8].

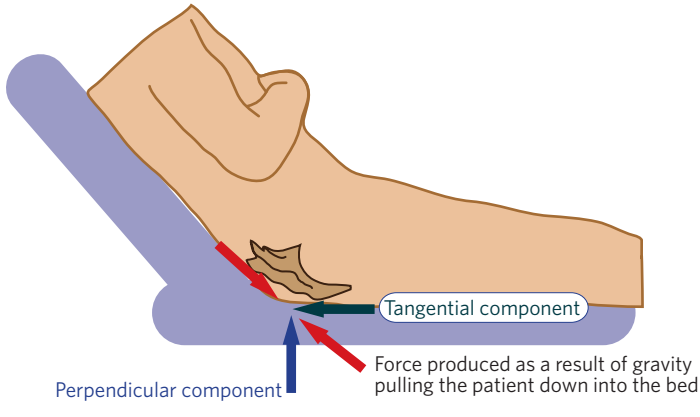
Shear stresses in tissues may also be caused by localised pressure applied to a skin surface. The application of pressure causes compression of the tissues, and by doing so distorts adjacent tissues (Figure 3). This is sometimes known as pinch shear. Steep pressure gradients, ie large changes in pressure across a small surface area, are likely to produce high pinch shear.

FACT FILE

Shear stresses are caused by:

- friction, eg when sliding down a bed
- uneven pressure distribution, eg over a bony prominence.

FIGURE 2 Pressure and shear applied to the sacral area of a partially reclined patient (adapted from[9])



$$\text{Pressure} = \frac{\text{perpendicular component of force}}{\text{contact area}}$$

$$\text{Shear stress} = \frac{\text{tangential component of force}}{\text{contact area}}$$

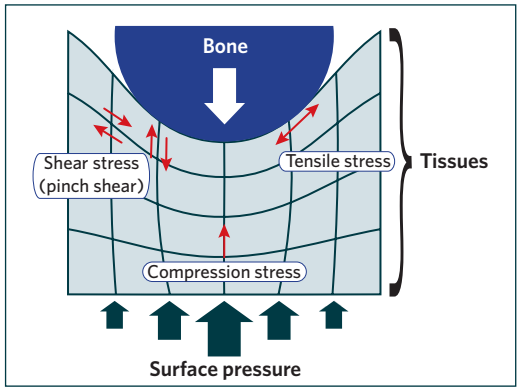


FIGURE 3 Uneven pressure distribution as a cause of shear stresses (adapted from[10])

HOW DO SHEAR STRESSES CONTRIBUTE TO PRESSURE ULCER DEVELOPMENT?

Shear stresses are thought to act in conjunction with pressure to produce the damage and ischaemia of the skin and deeper tissues that results in pressure ulcers. The mechanisms involved include distortion of tissues, pinching and occlusion of capillaries crossing tissue planes, reductions in blood flow, and physical disruption of tissues or blood vessels.

Tissue distortion

In layered objects, eg body tissues, shear stresses can cause one layer to move relative to another (Figure 4). When shear stresses are applied to tissues, the amount of movement between the layers in the tissues – ie the degree of potential for producing blood vessel occlusion and physical disruption of tissues – is affected by the looseness of the connective tissue fibres between the layers[11] and the relative stiffnesses of the tissue layers.

In aged skin, skin elasticity and skin turgor tend to be reduced. As a result, more pronounced skin tissue displacements can take place in skin and subdermal layers when external forces are applied[12].

Differences in the stiffnesses of distinct tissue layers mean that they deform to varying extents when an external force is applied. Stiffer tissues deform to a lesser extent than materials of lower stiffness. Table 1 shows that the greatest difference in stiffness of adjacent tissues, ie the greatest potential for shear stresses to occur, is between the bone and muscle[13-15], but that

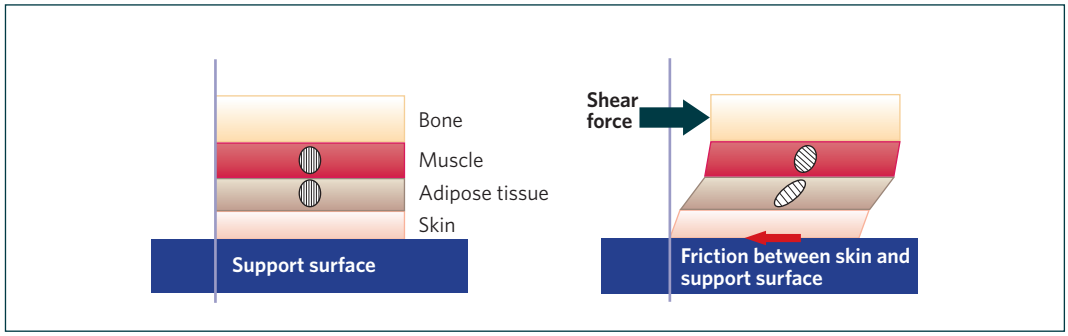


FIGURE 4 Effect of shear stress on body tissue layers (courtesy of T Ohura)
When shear force is applied, friction between the skin and support surface tends to hold the skin in place while deeper tissues are displaced. The amount of displacement, ie shear strain, is greater in the vicinity of the bone than in the superficial tissue layers.

potential for shear stresses also occurs between muscle and adipose tissue, and between adipose tissue and skin.

This helps to explain why pressure ulcers frequently develop over bony prominences, where interface pressures also tend to be highest[16,17]. Patients with prominent bones are particularly prone to shear stresses and pressure, and slender body types tend to have higher shear stresses and pressure at the coccyx and sacrum than do obese body types[18].

FACT FILE

- Shear stresses act in conjunction with, and amplify the effects of, pressure to produce the ischaemia and tissue damage that may result in the development of pressure ulcers.
- Although shear stresses can be measured on the skin surface and computer modelling is helpful, there remains a need for the development of devices that directly measure shear stresses in deeper tissues, eg muscle and adipose tissue.

Effects on blood vessels

Shear stresses can reduce or prevent blood flow through a number of mechanisms:

- direct compression and occlusion of blood vessels (Figure 4)
- stretching and narrowing of dermal capillary beds – when sufficiently high shear stresses are applied, the internal diameter of the capillaries becomes inadequate for blood flow[19,20]
- bending and pinching of blood vessels running perpendicular to the skin surface[21].

The capillaries in adipose tissue are also vulnerable to the effects of shear stresses because adipose tissue lacks significant tensile strength (ie it distorts and tears apart easily)[22].

TABLE 1 Relative stiffness of body tissues (based on animal studies)[13-15]

Body tissue	Stiffness (as indicated by elastic modulus (kPa))
Bone	20,000,000
Muscle	7
Adipose tissue	0.3
Skin	2–5

Deeper and larger blood vessels may also be affected by shear stresses. The blood supply for skin and subcutaneous tissues can be traced back to arteries that arise below the deep fascia and muscle. These arteries – known as perforator vessels – tend to run up perpendicular to the surface and to supply considerable areas. Their perpendicular route makes them particularly prone to shear stresses, and may explain the observation that some larger sacral pressure ulcers tend to follow the supply pattern of specific blood vessels.

Pressure and shear stresses usually work in tandem to reduce blood flow. Biomechanical modelling has demonstrated that shear stresses applied in addition to pressure cause greater obstruction and distortion of capillaries in skeletal muscle around bony prominences than does pressure alone[20]. **At sufficiently high levels of shear stresses, only half as much pressure is required to produce blood vessel occlusion as when little shear stress is present[23].** Conversely, if shear stresses are reduced, tissues can tolerate higher pressures without blood flow occlusion[20].

Measuring shear stresses

Several devices are available for measuring shear stresses at skin surface interfaces[18,24,25]; some devices also simultaneously measure interface pressure[15,26]. Internal shear stresses are difficult to measure directly, but have been estimated using computer modelling[27] and using computer modelling in combination with magnetic resonance imaging (MRI)[16,17].

WHAT AFFECTS FRICTION?

Friction force at the patient-support surface interface is dependent on the perpendicular force and the coefficient of friction of the skin

and the contact surface (Box 2). The higher the perpendicular force, the higher the friction force. Similarly, the higher the coefficient of friction, the higher the friction force and the greater the force required to make the patient move in relation to the support surface.

The **coefficient of friction** of textiles or other materials against skin is mainly influenced by:

- the nature of the textile – eg rougher textiles produce higher coefficients of friction
- skin moisture content and surface wetness – these increase the coefficient of friction and are particularly relevant in the clinical context where skin may be damp from perspiration or because of incontinence (see: *Microclimate in context*[28], pages 19–25)
- ambient humidity – high ambient humidity may increase skin moisture content or induce sweating and therefore increase coefficient of friction (see above)[29].

A study looking at the interaction between skin and a polyester/cotton textile confirmed that as skin moisture increased, the coefficient of friction also increased[29]. The same study found that the coefficient of friction for wet fabric on skin was more than double the value for dry fabric on skin[29].

FACT FILE

- The magnitude of friction force is dependent on the perpendicular force and a characteristic of the interaction of the two objects known as the coefficient of friction.
- Moist skin has a higher coefficient of friction than does dry skin, and is therefore more likely to be exposed to higher levels of friction and shear stresses.
- Much research is required to fully unravel how shear stresses cause tissue damage, the effect of the frequency and/or speed of postural changes on shear stresses, and which patients are at greatest risk of injury from shear stresses[4].
- Many of the interventions aimed at reducing shear stresses and friction revolve around attempts by healthcare professionals, carers or patients themselves to move or reposition patients, as it is during such manoeuvres that there is increased risk of shear stress and friction occurring.

HOW MIGHT FRICTION CONTRIBUTE TO PRESSURE ULCER DEVELOPMENT?

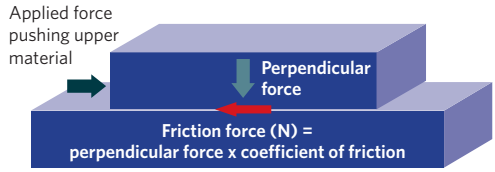
The significance of friction in the context of pressure ulcers lies mainly in its contribution to the production of shear stresses. When the tangential force applied by friction at the skin surface is larger than the perpendicular force (pressure), or when a small amount of pressure with a large tangential force is applied to the skin, abrasions, superficial ulceration or blistering may occur. If the skin is already irritated or inflamed, eg by maceration, incontinence-associated dermatitis or infection, superficial damage due to friction will occur more easily. Friction applied to the skin surface can also cause shear stresses in deeper tissue layers such as muscle.

Measuring friction

Experiments related to the measurement of friction usually determine the coefficient of friction of the materials being examined. A standardised method used commonly calculates the coefficient of friction between a block of metal and a fabric[30]. This standardisation should allow for comparison between textiles to be made easily. However, differences in equipment and methods of measurement used in those studies that have been conducted make comparisons of results difficult[29-33], and the role of textiles in the prevention and formation of pressure ulcers is understudied[34,35].

BOX 2 Friction

Friction force opposes externally applied forces; movement of one surface against another will only occur when the applied force is greater than the friction force. The friction force produced by two surfaces in contact is dependent on the perpendicular force (related to the weight of the object) and the coefficient of friction. The **coefficient of friction** is a value that is dependent on the properties of the two objects that are in contact.



Definitions from the Support Surface Standards Initiative[5]:

- Friction – "The resistance to motion in a parallel direction relative to the common boundary of two surfaces."
- Coefficient of friction – "A measurement of the amount of friction existing between two surfaces."

MANAGEMENT OF SHEAR STRESSES AND FRICTION

Alongside pressure redistribution, patient repositioning and mobilisation, strategies to reduce shear stresses and friction form an important part of best clinical practice to reduce patients' overall risk of pressure ulcer development.

A number of guidelines for the prevention of pressure ulcers have developed recommendations to assist with decision making about appropriate health care. These include the recent guidelines produced by the NPUAP and EPUAP[1] and those produced by the Registered Nurses' Association of Ontario[36]. Decisions for care will require clinical judgement based on

patient risk, availability of resources, patient comfort and wishes, and other treatment or care needs.

The principles involved in minimising the effects of shear stresses and friction include:

- **decreasing tangential forces –** eg during lying, by minimising head of bed elevation, and during sitting, by avoiding sliding downwards/forwards[7]
- **avoiding actions that induce tissue distortion –** eg avoiding sliding or dragging, by ensuring that patients are positioned in a way that does not allow them to slip easily and by ensuring that body tissues are not dragged upon during repositioning or left distorted following repositioning
- **increasing contact area with support surfaces –** this spreads the perpendicular and tangential loads and friction force over a larger area, reducing the localised pressure and shear stresses[11].

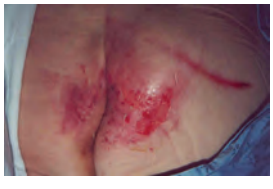
The use of lower coefficient of friction textiles to cover support surfaces will reduce friction force and shear stresses. However, a balance is required: if the coefficient of friction is too low, the patient may slide around on the support surface and be difficult to place in a stable position.

FACT FILE

Patients at particular risk from shear and friction are those:

- at risk of pressure damage
- who require head of bed elevation
- with damp or damaged skin
- who are difficult to reposition.

FIGURE 5 Friction damage (courtesy of H Orsted)
This patient has superficial abrasions related to sheet burn and scratch trauma from a caregiver's ring.



Clinical practice steps

Best clinical practice begins with identification of those at risk and ends with an evaluation of the impact of implementation, ie effect on incidence and prevalence of pressure ulcers. Clinical recommendations from the recent NPUAP and EPUAP guidelines[1] that particularly relate to shear stress and friction are reviewed in the practice steps below. The majority of these recommendations are classified as having 'strength of evidence = C', meaning that they are supported by indirect evidence and/or expert opinion[1].

Step 1: Identify those at risk from shear stresses and friction

- Establish a risk assessment policy in all health care settings[1].
- Consider the potential impact of following factors on an individual's risk of pressure ulcer development: friction and shear, sensory perception, general health status and body temperature[1].

Box 3 lists the types of patients at increased risk of shear stresses and friction.

BOX 3 Patients at risk of shear stresses and friction

Patients that:

- must have head of bed elevation because of difficulty breathing or the use of medical devices such as ventilators or tube feeding equipment
- are difficult to reposition without some sliding across bed sheets or support surface
- slip or slide from a position that they have been placed in when in a bed, chair or wheelchair – eg patients who are unable to or find it difficult to position themselves because they are immobile, have sensory loss or are physiologically unstable
- are too weak or too unstable to be able to reposition themselves effectively without dragging across sheets or support surfaces
- have moist, wet or macerated skin where the skin touches a support surface or another skin surface (skin folds/pannus) – eg due to sweat, incontinence or leaking dressings
- are exposed to high pressures, especially over bony prominences – eg very thin patients
- are obese – risk may be increased because of immobility and difficulties with transfers or repositioning, increased sweating and poor perfusion of adipose tissue[37]
- have decreased skin elasticity and/or turgor – eg due to ageing or dehydration
- have fragile skin – eg due to steroid or anticoagulant use, scar tissue over a healed pressure ulcer, inflammation or oedema
- have signs of existing skin friction damage – eg superficial abrasions or blistering on areas in contact with support surfaces (Figure 5)
- have a current or healed pressure ulcer
- have developed undermining in an existing pressure ulcer – this may signify that shear stresses are being applied; the undermining in such cases will be towards the underlying bony prominence[38]
- have an irregularly shaped pressure ulcer[39]
- tend to rub their heels on the bed due to agitation – eg as a result of pain or dementia
- have dressings that show partial peeling along one edge – the forces involved may be coming from the side of the peeling.

The three most commonly used pressure ulcer risk scales (Braden, Norton and Waterlow) all recognise moisture or incontinence as a risk factor for pressure ulcers[40-42]. However, only the Braden scale specifically evaluates friction and shear; it does so on the basis of the level of assistance required to move, frequency of sliding in a bed or chair, and the presence of spasticity, contractures or agitation that cause friction[40].

Step 2: Assess those at risk from shear stresses and friction

- **Ensure that a complete skin assessment is part of the risk assessment screening policy in place in all health care settings[1].**

Complete skin assessment will enable clinicians to determine the presence of existing pressure ulcers and to look for signs that indicate that the patient is at risk of shear stresses and friction (see Step 1).

Although it is very important to distinguish clinically between pressure ulcers and moisture lesions such as incontinence-associated dermatitis[43], the presence of moisture lesions increases the coefficient of friction of skin, and consequently risk from shear stresses and friction.

If damage due to shear stresses and friction has already occurred, determining how shear and friction were involved may suggest interventions to prevent further damage. For example, if a wheelchair user develops damage, analysis of how transfers are made may reveal that 'drag' is occurring and suggest interventions that reduce drag.

Step 3: Provide care for those at risk from shear stresses and friction

Skin care

- **Do not vigorously rub skin that is at risk for pressure ulceration[1].**

Skin rubbing is an outdated practice that unfortunately persists in some places. When clinicians rub already reddened and inflamed tissues there is a possibility of damage to the underlying blood vessels and/or to the fragile skin[36,44,45].

If emollients are applied to skin, they should be applied gently to avoid unnecessary trauma. Incompletely absorbed emollients that leave a sticky residue on the skin and may increase the coefficient of friction should be avoided. There is anecdotal evidence that application of silicone-based lotions to the skin of patients who have a lot of drag or resistance during repositioning may ease friction.

Management of skin moisture to avoid it becoming damp or macerated is important to avoid increasing the coefficient of friction of the skin (see: *Microclimate in context*[28], pages 19–25).

- **Consider using film dressings to protect body areas at risk for friction injury or risk of injury from tape[1].**

An increasing range of dressing products (including film dressings) that aim to reduce shear stresses and friction over vulnerable areas is under investigation[46]. Transparent dressings, eg films, aid monitoring of the underlying skin. A study using an animal model found that film dressings produced greater reductions in shear and pressure than did other types of dressings[26].

Dressing types that have been studied clinically include a hydrocolloid dressing that has a low coefficient of friction outer surface. This dressing was found to reduce shear force when applied to areas susceptible to shear damage such as the heel[47], and to significantly reduce the incidence of persistent erythema when placed over the greater trochanter[48]. In a more recent study, application of a soft silicone dressing to the sacrum in high risk intensive care patients was associated with a reduction in sacral pressure ulcer incidence to zero[49].

Positioning

- **Select a posture that is acceptable for the individual and minimizes the pressure and shear exerted on the skin and soft tissues[1].**
- **Limit head-of-bed elevation to 30 degrees for an individual on bedrest, unless contraindicated by medical condition. Encourage individuals to sleep in a 30 to 40 degree side lying position or flat in bed if not contraindicated[1].**
- **Use transfer aids to restrict friction and shear. Lift – don't drag – the individual when repositioning[1].**
- **If sitting in bed is necessary, avoid head of bed elevation or a slouched position that places pressure and shear on the sacrum and coccyx[1].**

The input of specialist advisers on seating and support surfaces may be required to ensure that the patient is placed in a comfortable position that minimises shear and friction, and avoids head of bed elevation. Slight knee gatch (elevation of a bend in a support surface at the level of the knees) engagement may help to prevent the patient from sliding down the bed.

The recommendation for limiting head of bed elevation is based on a study performed in healthy volunteers. This found that the 30° semi-Fowler position (which involves 30° head of bed elevation and 30° elevation of the legs) produced lower pressure and shear stresses than did a supine position with 30° head of bed elevation[50]. The same study found that a 30° side-lying position gave lower interface pressure readings than the 90° side-lying position[50].

However, patient positioning needs to consider all of the patient's needs. For example, if the patient is being ventilated, critical care protocols may recommend 30-45° head of bed elevation.

The risk of friction burns can be reduced by careful repositioning of patients to avoid dragging across the support surface cover, and the use of turning sheets or transfer aids[51].

Support surfaces

- **Provide a support surface that is properly matched to the individual's needs for pressure redistribution, shear reduction, and microclimate control[1].**

Support surface selection may require multidisciplinary input. In addition to relief of pressure and shear stress, support surface selection should take into account factors such as ability to manage aspects of microclimate, eg skin moisture and temperature (see: *Microclimate in context*[28], pages 19–25).

Following repositioning, some clinicians advise that the patient is briefly moved away from the support surface to help release shear forces that have built up during the manoeuvre. This also provides an opportunity to check that the support surface has not become wrinkled and that the patient's skin is smooth and has not become distorted.

Shear forces can be reduced during bed operation when a patient is supine by bending the knees, and matching the body's bending points with those of the bed[18].

- **Prevent shear when lateral-rotation features are used. Assess skin frequently for shear injury[1].**

Lateral rotation features of some beds allow the patient to be turned from side to side through mechanical movement of the bed. However, such beds are unable to fully reposition patients and positioning aids will be required to maintain good body alignment and to prevent shift within the bed. Patients should be observed regularly through several rotations to check for sliding movement that could cause shear and friction.

CONCLUSION

Shear stresses – and by association, friction – are important extrinsic factors involved in the development, and sometimes persistence, of pressure ulcers. However, many uncertainties remain about the role and critical levels for shear stress and friction in pressure ulcer development. Even so, a clear understanding of how shear stresses and friction occur will undoubtedly assist clinicians in consistent implementation of aspects of pressure ulcer prevention protocols designed to minimise shear stresses and avoid increasing the coefficient of friction of skin.

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Microclimate in context

M Clark, M Romanelli, SI Reger, VK Ranganathan, J Black, C Dealey

INTRODUCTION

Numerous factors have been implicated in the aetiology and pathophysiology of pressure ulcers[1-3]. Even so, it is clear that there is much still to be learned about the complex interactions between the many intrinsic and extrinsic factors involved. Recently, interest has been building in how modifying the environment at or near the skin surface – the microclimate – may affect risk of pressure ulcer development[4,5].

This paper defines the main parameters involved in current understanding of microclimate and explores what is known about the relationship between microclimate and pressure ulcer development. It also describes interventions that may beneficially alter microclimate. Discussion includes the effect of different support surfaces on microclimate and how management of microclimate may help to avoid pressure ulcers.

FACT FILE

In the context of pressure ulcers, microclimate usually refers to skin temperature and moisture conditions at the skin–support surface interface.

WHAT IS MICROCLIMATE?

In 1976, Roaf reported that the first UK-based conference on pressure ulcers highlighted known contributory factors to pressure ulcer development: "We know how to avoid bed sores and tissue necrosis – maintain the circulation, avoid long continued pressure, abrasions, extremes of heat and cold, maintain a favourable micro-climate, avoid irritating fluids and infection"[6].

The maintenance of a favourable microclimate was seen in early pressure ulcer publications to be a key modifier of the ability of skin and underlying soft tissue to withstand prolonged stress, ie pressure and shear. The important role of microclimate modification in the prevention of pressure ulcers has largely been overlooked since the 1970s, but is now regaining attention.

Microclimate was suggested by Roaf to include skin temperature, humidity and air movement[6]. However, today, the use of the term microclimate in relation to pressure ulcers usually refers to:

- skin surface or tissue temperature and
- humidity or skin surface moisture at the body–support surface interface[2,7].

As described later, some support surfaces use air movement to influence temperature and humidity/moisture at the interface between skin and the support surface.

Studies examining the effects of elements of microclimate on skin and pressure ulcer development are inconsistent in the definitions used, making study interpretation and comparison difficult. Some of the definitions that have been used are discussed below.

Skin surface temperature

Methods used to measure skin surface temperature include:

- measurement at 'radiative equilibrium' – ie when the temperature of exposed skin has reached steady state following exposure to air
- measurement at the skin–support surface interface with the patient still in contact with the surface or very soon after moving out of contact with the surface.

The first method – measurement at radiative equilibrium – provides an indication of the 'intrinsic' skin (but not core body) temperature of the patient (although still subject to external variables such as ambient temperature). The second method provides an indication of the temperature at the skin–support surface interface.

Temperature may be measured directly, eg with a thermometer, or indirectly by using infrared thermal imaging (thermography)[8].

Humidity

Within the literature relating to microclimate, 'humidity' and 'skin moisture' are sometimes used synonymously. However, strictly speaking humidity relates to the amount of water vapour in the air:

- **absolute humidity** – is expressed as the weight of water in grams per cubic metre of air (g/m^3)
- **relative humidity** (often abbreviated to humidity) – is a ratio expressed as a percentage that relates the amount of water vapour in the air at a specific air temperature to the maximum amount of water vapour that body of air would hold at that temperature (Box 1). The relative humidity of the general surroundings is known as ambient humidity.

BOX 1 Definitions of humidity

Absolute humidity (g/m^3)	=	weight of water vapour / volume of air
Relative humidity at a specific temperature (%)	=	amount of water vapour in the air at a specific temperature / maximum amount of water vapour that can be held in the air at that temperature

FACT FILE

- Definitions of each element of microclimate require further clarification; the terminology around skin moisture and humidity can be particularly confusing.
- Raised skin temperature may be related to pressure ulceration by increasing susceptibility to the ischaemic effects of pressure and shear stresses, and by weakening the stratum corneum.

Absolute humidity is affected by air temperature: warmer air is able to hold more water vapour than cooler air. Therefore, for the same absolute humidity, warmer air will have a lower relative humidity than cooler air.

Relative humidity is measured using a device known as a hygrometer. In relation to skin, relative humidity may be measured at the interface between the skin and a support surface, or just above exposed skin[9,10].

Skin moisture

Skin moisture is difficult to define: it may refer to the presence of fluid on the skin surface ('wetness') from perspiration, incontinence or wound/fistula drainage, or to the moisture content of the outermost layer of the skin itself (the stratum corneum).

Skin moisture can be assessed subjectively, eg using the moisture subscale of the Braden scale for assessing risk of pressure ulceration: this classifies skin as dry or having some degree of moisture as indicated by frequency of linen changes and detection of dampness on the skin surface[11]. Methods to quantitatively assess the moisture content of the stratum corneum include measurement of electrical properties such as skin surface conductance or capacitance[12].

Air movement

Air movement is the least investigated factor of Roaf's original definition of microclimate, but is used by some support surfaces to aid microclimate control by modifying temperature and humidity/skin moisture. The flow of air can be expressed quantitatively as the velocity (speed) of airflow across the skin, eg in metres per second, or by the rate at which air is pumped through a support surface, eg in litres per minute.

HOW IS SKIN SURFACE TEMPERATURE RELATED TO PRESSURE ULCER RISK?

Raised **body** temperature (pyrexia) is a recognised risk factor for pressure ulcers[13,14]. It is well established that increasing body temperature by 1°C raises the metabolic activity (ie the need for oxygen and energy) of body tissues by about 10%[15]. By definition, ischaemia occurs when tissue perfusion is not sufficient to meet the needs of the tissue. Therefore, when metabolic needs are raised, a smaller reduction in tissue perfusion will produce ischaemia than when metabolic needs are steady. This suggests that in a patient with elevated body temperature and compromised tissue perfusion due to exposure to pressure and shear, ischaemia and tissue damage may occur more quickly and at lower levels and/or shorter durations of pressure/shear than if body temperature was normal[16].

This concept has been extended to suggest that increased **skin** temperature may play a role in the development of pressure ulcers.

In addition, temperature affects the strength of the stratum corneum: at 35°C the mechanical strength of the stratum corneum is 25% of that at 30°C[17].

In contrast, low core body temperature during surgery is associated with pressure ulcer development[14]. To examine whether preventing hypothermia during surgery would reduce the incidence of pressure ulcers, Scott *et al* provided forced air warming therapy during surgery to 338 patients. There was an absolute risk reduction in pressure ulcer incidence of 4.8% and a relative risk reduction of 46% between those who received warming and those who received standard care. However, this difference did not achieve statistical significance[18].

What affects skin temperature?

It seems intuitive that raised core body temperature would correlate with increased skin temperature, and that this would perhaps help to explain why pyrexia is a risk factor for pressure ulcers. However, clear evidence of a correlation is lacking and one small study found a negative correlation between these parameters[19].

Other factors that may increase skin temperature include high ambient temperature, high ambient humidity, low exposure to air, and contact with another surface (eg clothing, support surfaces, dressings and incontinence pads).

FACT FILE

- Skin temperature is highly variable and influenced by a very wide range of environmental, physiological and pathological factors, including ambient humidity and temperature and disease processes.
- Further research is required to establish whether skin temperature can be used to determine risk of or predict imminent pressure ulcer development.
- Animal models have suggested that skin cooling may protect tissues against the effects of pressure. However, the association between intraoperative hypothermia and pressure ulcers suggests that there may come a point where cooling-induced vasoconstriction may exacerbate pressure-induced ischaemia.

The skin has a major role in regulating body temperature. The two main mechanisms involved in cooling are:

- dermal vasodilatation – which increases skin blood flow and causes heat loss by convection and conduction
- sweating – perspiration cools the skin through evaporation.

These responses may be triggered by increases in core body temperature, eg during pyrexial illness or exertion, increases in ambient temperature, or covering the skin with clothing or a support surface. Local perspiration increases considerably when the skin is warmed above about 33°C[20]. Perspiration may also be triggered in conditions such as shock, hyperthyroidism, and hypoglycaemia. When ambient humidity is high, evaporation of perspiration may be slowed causing perspiration to accumulate on the skin.

Increased perspiration is particularly relevant to pressure ulcer risk because moisture on the skin surface can reduce the skin's resilience and increase the coefficient of friction of skin, making it more prone to pressure, shear stresses and friction (see: *Pressure in context*[21], pages 2–10 and *Shear and friction in context*[22], pages 11–18).

Conversely, the skin's contribution to conservation of body heat is largely mediated by dermal vasoconstriction.

What is normal skin temperature?

Surprisingly, there is limited information on normal skin temperature over anatomical sites prone to pressure ulcer development. One key study proposed that sites prone to pressure ulcer development were cooler than adjacent body sites[23]. However, there were several weaknesses in this study; for example, ambient room temperature was unreported, as was the duration for which the skin was exposed to room temperature prior to temperature measurement.

Can skin surface temperature predict pressure ulcer development?

Several studies have investigated skin temperature associated with **early (Category/Stage 1) pressure damage**. These found inconsistent changes in skin temperature of pressure damaged areas: it could be increased (possibly as a result of inflammation), the same, or decreased (possibly as a result of ischaemia) in comparison with temperature at undamaged areas[24,25].

The few studies that have examined whether skin temperature can **predict** pressure ulceration have been inconclusive. In a prospective cohort study, Clark measured the sacral skin temperature of 52 elderly hospital patients[9]. Of this cohort, six developed pressure ulcers; however, the skin temperature of the patients who did and did not develop pressure ulcers was similar. This study was confounded by a non-uniform allocation of support surfaces to the recruited subjects[9]. However, one small study of neurologically impaired patients found that sacral skin temperature may increase 24–96 hours before sacral pressure ulcer development by at least 1.2°C[26].

A recent study has examined whether skin temperature regulation may predict pressure ulcer development[27]. A small sample of nursing home patients wore skin temperature monitors taped to the right mid-axillary line continuously for five days. The study found that patients at high risk or who went on to develop pressure ulcers had least variability in skin temperature, suggesting impaired skin temperature regulation[27]. However, it is unknown whether reduced ability to regulate skin temperature is directly related to pressure ulcer development or if it is a general marker of declining physiological condition.

How does changing local skin temperature affect risk of pressure ulcer development?

Some investigators have examined how changing local skin temperature affects the impact of pressure on tissues. In an animal study, a known pressure (100mmHg) was applied for five hours with indentors heated to 25, 35, 40 or 45°C[28]. Moderate muscle damage was seen at 35°C and cutaneous and deep tissue damage were observed at 40 and 45°C (note that there may have been an element of thermal damage at 45°C)[28]. No cutaneous or muscle damage was observed where load was applied at 25°C, suggesting that local cooling may have a protective effect.

More recently, Lachenbruch has argued from prior studies (including that of Kokate *et al*[28]) that a 5°C reduction in skin–support surface interface temperature would confer tissue protective effects similar in magnitude to the interface pressure reductions afforded by the most expensive support surfaces[20]. This hypothesis remains untested.

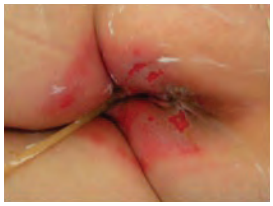


FIGURE 1 Incontinence-associated dermatitis (courtesy of J Black)

Although cooling may provide some protective effects, as mentioned previously hypothermia during surgery may contribute to the development of postoperative pressure ulcers[17].

HOW DO HUMIDITY AND SKIN MOISTURE RELATE TO PRESSURE ULCER RISK?

Increased skin moisture, especially when due to incontinence, has long been recognised as an important risk factor for pressure ulcer development[29-31]. However, there is limited quantitative data on skin moisture or humidity to support this view.

Clark reported that the humidity just above the sacral skin of elderly hospital patients who subsequently developed Category/Stage II pressure ulcers was higher than the humidity of patients who did not[9].

Increased skin moisture as measured by electrical capacitance has been found in a study in Indonesia and in a small pilot study to correlate with the development of pressure ulcers[32,33]. However, further studies are required to determine whether measuring skin moisture in this way may prove useful in identifying patients who would benefit from additional intervention to prevent pressure ulceration.

Effects of excessive skin moisture

Excessive moisture on the skin surface, eg as a result of perspiration, urinary or faecal incontinence, wound/fistula drainage or vomit, is thought to contribute to increased risk for the development of pressure ulceration by weakening skin. Moisture can weaken the crosslinks between the collagen in the dermis and soften the stratum corneum[34]. This can cause maceration (or incontinence-associated dermatitis if the liquid involved is urine – Figure 1) and also increases the exposure of underlying blood vessels to the effects of pressure and shear stresses.

In addition, excessive moisture can significantly increase the skin's coefficient of friction[35], leading to increased likelihood of skin damage from friction and shear stresses (see: *Shear and friction in context*[22], pages 11–18).

Relative humidity also affects the strength of the stratum corneum: at a relative humidity of 100% the stratum corneum is 25 times weaker than at 50% relative humidity[16].

Effects of excessive skin dryness

Ageing skin is less resilient and more vulnerable to damage than is younger skin because it is generally thinner, structurally weaker and drier[36]. Dry skin has reduced lipid levels, water content, tensile strength, flexibility and junctional integrity between the dermis and the epidermis. Low ambient humidity reduces water content in the stratum corneum[12]. In the USA, the Agency for Health Care Policy and Research guidelines on the prevention of pressure ulcers, recommends avoiding ambient relative humidity below 40% to reduce the likelihood of dry skin[37].

HOW DOES AIR MOVEMENT RELATE TO PRESSURE ULCER DEVELOPMENT?

There appears to have been no research to look specifically at the possible role of air movement in the aetiology of pressure ulcers. The relevance of air movement may relate to its potential to affect skin temperature and moisture content through convection and evaporation.

WHAT DON'T WE KNOW ABOUT MICROCLIMATE AS A CAUSE OF PRESSURE ULCERS?

The body of literature linking pressure ulcers and microclimate is relatively small, with little characterisation of interactions between skin and fabric (eg support surface covers). Furthermore, there is evidence of wide intra- and inter-individual differences in skin microclimate parameters, but the effect of these on pressure ulcer development is unclear. Interpretation of available data is therefore challenging.

The *in vivo* experimental studies on interactions between skin and fabrics that have been performed have rarely led to any significant results or definitive conclusions[38]. This may be partly explained by the considerable differences that may exist in skin condition (eg hydration, surface roughness, adhesion between skin layers) between individuals and between different anatomical locations in the same individual[38].

CLINICAL MANAGEMENT OF MICROCLIMATE

The needs of the patient should be carefully assessed before modifications in temperature and humidity/skin moisture are undertaken.

FACT FILE

- Excessive skin moisture and high relative humidity weaken skin and increase the coefficient of friction of skin, increasing the likelihood of damage from pressure, shear and friction.
- Dry skin is weakened and more vulnerable to damage, eg by pressure, shear stresses and friction.
- Investigations into the effects of air movement need to take place to confirm or refute whether this aspect of microclimate is significant in pressure ulcer aetiology.
- Until therapeutic ranges for temperature and humidity/skin moisture at the patient–support surface interface are identified, clinical judgement should be exercised to avoid extremes (high or low) in these factors.

A key strategy in the management of microclimate is control of the underlying cause of the extreme of temperature or skin moisture, eg pyrexia or incontinence. Patients who are hot may be cooled using simple measures such as reduction in the number of covers, avoidance of plastic mattress coverings, frequent repositioning, use of a fan and wearing breathable cotton clothing. Where available in climates with high ambient humidity, air conditioning may be helpful in cooling and reducing humidity.

Repositioning

The importance of the role of repositioning in controlling microclimate should not be underestimated. Some mattress covers are made of material that prevents dissipation of heat. Repositioning patients allows skin that has been in contact with the mattress cover to be exposed to air and to become cooler. It also provides an opportunity for perspiration to evaporate.

Skin care

Barrier creams and sprays can be useful in protecting moist skin from further damage, especially from urine[2,39]. However, it is best to manage incontinence where possible so that the skin does not come into contact with urine in the first place.

For patients who are incontinent and require the use of absorbent underpads, use of breathable pads which allow transmission of moisture vapour is preferable. Care is required to ensure that the use of underpads does not interfere with the pressure redistribution or microclimate management properties of any support surface in use.

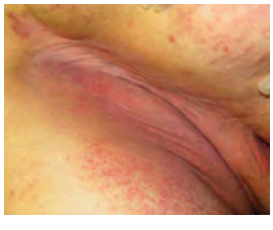
Bariatric patients who are prone to excessive perspiration may benefit from frequent washing and changing of clothes and bed linen to control skin moisture. Accumulation of moisture in the skin folds of bariatric patients may be a particular problem, and result in intertriginous dermatitis with bacterial and candidal infections (Figure 2).

The use of emollients can assist in ameliorating dry skin and may reduce the risk of skin damage[2]. They should be applied liberally and frequently (eg up to three to four times daily) when the skin is very dry, and following washing or bathing when they will help to trap water onto the skin.

FACT FILE

Management of microclimate is one component of pressure ulcer prevention and needs to take place within the context of a comprehensive pressure ulcer prevention protocol that takes into account patient comfort.

FIGURE 2 Candidal infection in skin fold of a bariatric patient (courtesy of J Black)



Role of support surfaces in managing microclimate

Any surface that comes into contact with skin has the potential to alter microclimate by changing the rate of evaporation of moisture and the rate at which heat dissipates from the skin. The overall effect on microclimate is dependent on numerous factors, including the nature of the support surface itself (ie what is made from, how the material is conformed, what sort of cover it has)[15,40].

For example, foam surfaces tend to increase skin temperature because they have poor heat transfer properties[15]. The effect of foam products on moisture depends on the porosity of the cover[15]. Gel filled products may initially have a cooling effect that wears off after more than two hours of patient contact and tend to increase humidity at the skin surface[15]. Fluid filled products that utilise high heat capacity liquids have the potential to reduce skin temperature[15]. Alternating pressure air mattresses may limit skin temperature increases[41].

Some specialised support surfaces for beds have features that aid active management of microclimate by allowing air to flow through their surfaces, for example low air loss features or air fluidised beds. The air flow cools skin through convection and the evaporation of moisture from the skin surface[42].

Low air loss surfaces

Low air loss surfaces pump air into a series of cushions and then allow the air to escape through small holes (porosities) in the cushions' covers. The air flows along the inside of a

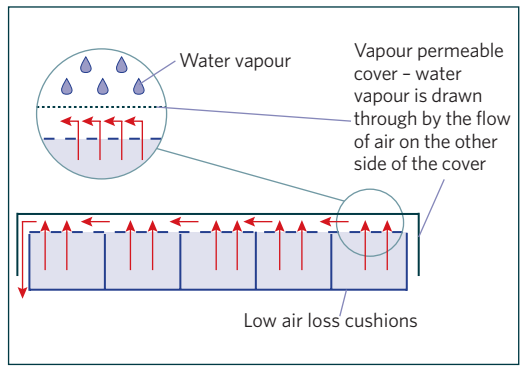


FIGURE 3 Mode of action of low air loss surfaces

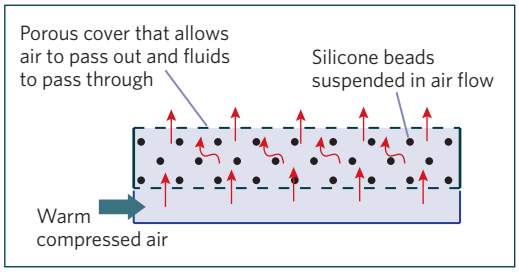


FIGURE 4 Mode of action of air fluidised support surfaces

vapour permeable patient contact layer drawing moisture and heat through the contact layer and away from the skin[16] (Figure 3). Low air loss surfaces have been shown to lower skin temperature and to produce lower moisture gain than standard hospital mattresses[43].

Air fluidised support surfaces

Air fluidised support surfaces comprise solid silicone particles the size of sand through which air is forced. The air flow causes the particles to take on properties of a liquid. The cover over the particles is porous allowing air to escape and body fluids (eg sweat, urine) to flow through (Figure 4). Air fluidised beds are considered to be the most drying support surface; the fluid loss increases linearly with increased air flow temperature[44].

The air flowing through low air loss and air fluidised devices is generally warmed in the range of approximately 28–35°C and can be adjusted. This facility is undeniably useful, but has to be used with considerable care to prevent inappropriate cooling or heating.

Choosing a support surface to manage microclimate

Despite the availability of several forms of low air loss and air fluidised support systems, there is little information to guide decisions about which surface to use for which patient. Choice of support surface will be guided by clinical judgement and take into account numerous factors including the patient's need for pressure redistribution, the patient's size, ability to turn or move independently and body temperature, the presence of moisture on the skin or concomitant conditions such as incontinence. (See: *Pressure in context*[21], pages 2–10, for further information on pressure redistributing support surfaces.)

Whilst air loss systems may be helpful in keeping immobile patients cool and dry, it is important to recognise that patients must still be turned and repositioned on these devices. According to patient needs, the frequency of positioning may be reduced in comparison with standard mattresses (see: *Pressure in context*[21], pages 2–10).

Recent research has highlighted the importance of minimising layers of bedding between patients and low air loss support surfaces to prevent skin temperature rises[45].

Impact of microclimate management on pressure ulcer prevention

Support surfaces designed to aid microclimate management also provide pressure redistribution. This complicates assessment of the impact of microclimate management on incidence of pressure ulcers, and evidence demonstrating that management of microclimate directly prevents pressure ulcers is currently lacking.

Even so, clinical studies have shown that some advanced support surfaces that affect skin temperature and moisture, eg air fluidised and low air loss surfaces, are more effective than standard foam mattresses for the **treatment** of pressure ulcers[46]. In addition, there is some evidence that low air loss beds reduce the incidence of pressure ulcers in intensive care[47].

CONCLUSION

The concept of microclimate in relation to pressure ulcers has existed for some time. However, microclimate and its elements remain to be fully defined, and its relationship to pressure ulcer development clearly characterised. Evidence to date suggests that extremes of skin temperature and/or humidity/skin moisture appear to increase the sensitivity of skin to the damaging effects of pressure, shear stresses and friction.

This suggests, therefore, that the overall aim of microclimate management should be to avoid extremes of temperature or skin moisture and to enhance patient comfort. However, further investigation is needed to establish the effects of 'traditional' preventive interventions (such as repositioning) and those of support surfaces on elements of skin microclimate and pressure ulcer incidence. Low air loss support surfaces and air fluidised beds are designed to assist with microclimate management, but in the absence of evidence defining optimal skin temperature and moisture levels, clinical judgement is required for effective and safe use.

FACT FILE

- Low air loss and air fluidised support systems provide mechanisms for active microclimate control.
- In the absence of clinical evidence, choice of support surface for the management of microclimate remains reliant on clinical judgement.
- Further research is required to evaluate the effectiveness of microclimate control features in preventing pressure ulcers.

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