

The contribution of pressure gradients to advancing understanding of deep tissue injury to sacral regions

Dunk AM & Gardner A

ABSTRACT

Aims: To explore correlations between peak pressure and pressure gradient at 1.5 cm and 2.5 cm, and selected risk factors for pressure injury including Waterlow risk assessment score and body mass index.

Background: Accurately predicting pressure injury formation remains elusive. Exploring pressure gradients through pressure mapping systems may increase understanding of suspected deep pressure injury development.

Methods: A nested prospective correlational exploratory study recruited 120 medical and surgical patients with convenience sampling. Patients were positioned supine with a 30-degree head elevation, on a computer-linked pressure sensor mapping mat. Mean peak interface pressure and pressure gradients were calculated.

Results: Large correlation coefficients were identified between peak interface pressure and pressure gradients at distances of 1.5 cm and 2.5 cm, indicating that the area at the base of the 'cone-like' pressure damaged area remained essentially constant, rather than increasing with peak interface pressure.

Conclusions: Pressure is experienced in a 'V' shape rather than a 'U' shape. Additionally, the area subjected to the highest pressure gradient is restricted in size and the impact of pressure reduces with distance from the point of peak interface pressure. The results suggest that with increasing peak interface pressure, the surrounding area becomes subject to higher gradients and shearing forces.

Relevance to clinical practice: Increased use of pressure mapping systems in the clinical setting shows educational promise through visualisation of factors affecting deep tissue injury.

Keywords: Pressure injury, pressure gradient, peak interface pressure, pressure mapping.

Ann Marie Dunk

RN, BHthSc(Nurs), Wound Care Cert, MACN,
MNurs(Research)
Clinical Nurse Consultant, Tissue Viability Unit
Canberra Hospital & Health Services, ACT Health
Yamba Drive, Garran, ACT 2605
Tel +61 (2) 6244 2954
Email annmarie.dunk@act.gov.au

Anne Gardner*

RN, Crit Care Cert, BA, MPH, PhD
Professor of Nursing, School of Nursing, Midwifery and
Paramedicine, Australian Catholic University
223 Antill Street, Watson, ACT 2602
Tel +61 (2) 6209 1330
Email anne.gardner@acu.edu.au

* Corresponding author

WHAT THIS PAPER CONTRIBUTES TO THE WIDER GLOBAL CLINICAL COMMUNITY

- Visualising peak interface pressure adds to educational packages on prevention.
- Supports the notion that conical pressure distribution is more likely seen in suspected deep pressure injuries.
- 'Cone-like' pressure damage remains essentially constant rather than increasing as the peak interface pressure increases.

INTRODUCTION

Pressure injuries have been recognised as a patient safety problem and as a major challenge for health care professionals and health care systems¹⁻⁴. They have been identified as a nursing-specific clinical indicator and an indicator for the quality of care provided by health services^{5,6}.

Pressure injuries have been assessed as extending the length of hospital stay for affected patients, impacting on hospital bed availability and reducing overall hospital efficiencies. Data from United States hospitals indicate that pressure injury can increase a patient's hospital stay by up to five times^{2,7} and similar statistics are identified in many other countries⁸.

BACKGROUND

Pressure and shear are important factors in pressure injury development. Successful prevention and treatment of pressure injury requires an understanding of their pathophysiology. The forces of concern for pressure injury formation are pressure, shear and friction, with pressure widely considered to be the most important⁷⁻¹⁰.

A key component is the reduction of mobility associated with unrelieved pressure. Pressure results in compression of the skin and underlying tissue, which then leads to capillary occlusion and ischaemia if prolonged, with the greatest destruction recognised to be at the bony interface¹¹. The probability of pressure injury development is known to increase with duration and magnitude of pressure involved¹², which, in turn, depends on individual tissue tolerance¹³. Dependent on duration, both high and low pressures can lead to pressure injury development¹⁴. Repeated pressure is also important, particularly when repeated within a time period that is inadequate for the tissue to recover^{15,16}.

Throughout the paper we refer to "pressure injury"¹⁷ as this classification describes tissue loss rather than ulceration. The term has been taken up by the Australian Wound Management Association (AWMA) and used in the Pan Pacific guidelines¹⁸. The pressure at the skin surface acts perpendicularly on the tissue and can be measured as interface pressure, although the value of interface pressure alone as a predictor of pressure injury development has been questioned^{19,20}.

Prolonged pressure is understood to cause ischaemic changes at and around the point of the pressure attack¹. High pressure gradients have been determined to generate large shear forces and hence contribute to breakdown of the skin. Swain and Bader²⁰ have also highlighted the effect on cells and subsequent cell breakdown are more pronounced at the edges of an area of compression where pressure gradients are greatest.

The pressure distribution from a force applied to the body will be heterogeneous in nature, resulting in different areas suffering differing amounts of interface pressure¹⁹. These differing pressures give rise to shear, a force that acts parallel to tissue. The areas with the highest rate of change of pressure over distance, or the highest pressure gradient, will experience the highest levels of shear strain, and hence be most susceptible to pressure injury development^{10,19,21}.

Research highlights that high pressure gradients are known to generate large shear forces²²⁻²⁴. A number of authors have noted that the effects of pressure are magnified in the presence of shear^{1,13,25-27}. Shear occurs when deep fascia and skeleton move over the skin and upper fascia, resulting in destruction of the vascular supply in the

subcutaneous tissues¹⁴. The concept of a deep tissue injury is that injury originates deep within the tissue rather than at the surface and thereby is largely unseen and difficult to detect until significant damage has taken place.

In 1984, McClellmont described a situation where the opposing forces from the skin and the bone result in a cone-shaped pressure gradient^{2,28}. Within this cone (also called the "McClellmont cone"), the external pressure can increase by a factor of between three and five if the pressure site sits over a bony surface, for example the sacrum. Internal deformation of the tissue will be affected by the different structure and mechanical nature of the bones and tissue layers. These differences result in a heterogeneous distribution of the deformation, areas of differing interface pressure within the tissue and the formation of pressure gradients¹⁹.

Interface pressure mapping systems, typically comprised of multiple "sensors" across a measuring mat, enable the visualisation of the distribution of the pressure at the interface between the skin and the supporting surface. Therefore, these systems provide the means to determine areas of tissue that are under the greatest stress by highlighting rates of change of pressure (the interface pressure gradient) through visual means. Pressure mapping provides numerical and visual real-time data on the interface between the body and support surfaces. The technology can be used as a reliable device in guiding pressure relieving interventions. Visual interpretation of pressure mapping data can be used as a research tool and to enhance educational and teaching programs.

Application of systems has been limited in clinical settings. No controlled investigations have been carried out on pressure and the application time needed to cause pressure injury development²⁹. The relationship between interface pressure and pressure injury prevention, including variables such as acceptable time and pressure limits, requires further research²⁹. It is accepted that pressure monitoring at the interface between the body and the support system is important in the assessment of tissue viability³⁰.

Although interface pressure is the common parameter used to compare support surface performance, the relationship between interface pressure and pressure injury incidence has not been adequately studied³¹. Limitations to application in clinical practice include inconsistencies in the ways that manufacturer devices display pressures, as well as variation in sensory accuracy and drift. Most health clinicians are not trained in how to use this technology in clinical practice.

Under-explored areas include measurement of pressure gradients around peak interface pressure point. Exploration of these areas may lead to enhanced understanding of suspected deep tissue injury (SDTI), as SDTI occurs in tissue that has been subjected to pressures that exceed the tolerance level of muscle tissue³². The aim of the study was to explore correlations between two interface pressure mapping indices (peak pressure and pressure gradient at 1.5 cm and 2.5 cm) and selected risk factors for pressure injury; namely the Waterlow risk assessment score, weight and body mass index (BMI).

METHOD

Design and materials

The research was a nested exploratory study that utilised a prospective correlational design³³. The larger study was a partnership with a mattress manufacturing company funded through an Australian national research and development grant. Funding was withdrawn before completion of the study because the industry partner became ineligible for the funding (due to international takeover). The larger study sought to compare two interventional mattress types and required a sample size of 140 patients. We used data from patients already enrolled prior to the cessation of the study.

Participants

The study took place at a 450-bed public tertiary referral hospital and a community hospital in the same town with an approximate capacity of 300 beds, treating both public and private patients³³. Medical and surgical clinical areas within both hospitals were used for this study. This mix of clinical areas enabled recruitment of adult patients with a diverse range of conditions, including those who were acutely and chronically ill. All had to be able to tolerate moving from their bed to the study bed and to tolerate lying still for approximately 10 minutes at a time. A convenience sample of 120 medical and surgical patients was used for the study. The main inclusion criteria were patients who were: (1) sixteen years and older; (2) identified as an inpatient and occupied a bed; and (3) who had either no pressure injuries or were identified as having a pressure injury on the sacral region which did not exceed Category 1 as defined by the AWMA 2012 Guidelines¹⁸.

Ethics approvals were gained from the Deakin University Human Research Ethics Committee, the ACT Human Research Ethics Committee, and the Calvary Healthcare Human Research Ethics Committee.

Procedure

Informed consent was obtained from all participants. Patients were then positioned on sacral mapping equipment, supine on a standard hospital mattress and bed with an elevation of 30% at the head. Patients who could not tolerate this arrangement and those who could not lie completely still for the length of time required for data collection were excluded from the study.

Data were collected within the larger Mapping and Intervention for Prevention of Pressure Injury (MIPPI) study conducted between July 2004 and April 2005. This particular investigation further analysed the raw MIPPI data to determine pressure gradients and to conduct correlational analysis between the interface pressure mapping indices and the selected risk factors.

A data collection tool was designed by the investigating team and utilised to record clinical and demographic data taken verbally from the patient and extracted from the clinical notes. The tool was validated in a pilot study as part of the MIPPI study.

Patients were measured for their height status and weighed. Patients with a BMI in the range of 18.5–25 are considered to have a

healthy weight/height ratio. Individuals with a BMI greater than 25 are considered to be overweight, whilst those under 18.5 are considered underweight³⁴. Inter-rater reliability was assessed for all research nurses to ensure consistent data collection practices. This assessment covered the use of the Waterlow risk assessment tool, the calibration of the interface pressure mapping system and set-up of the Tekscan Clinseat™ software. The inter-rater reliability tests were conducted prior to the commencement of data collection.

To assess capability in the use of the Waterlow risk assessment tool, a written multiple-choice test was given to all research nurses prior to commencement of the study, requiring a pass rate of 100%. All data research nurses achieved this requirement. During the data collection process, random testing using the initial test was undertaken on all data collectors on a monthly basis by a member of the investigating team and a 100% pass rate was required and achieved.

The major equipment employed during the study comprised the designated standard hospital mattress (reactive) and hospital bed, and the Tekscan Clinseat™ pressure mapping system. This system comprised Microsoft Windows™-based Clinseat™ software on a dedicated computer laptop, a sensor mat, a parallel interface module and the Tekscan handle³³. A designated study mattress was used to ensure standardisation of the data collection processes and consistency of study equipment as it was impossible to accurately assess the age of mattresses already circulating within the hospital system.

The Tekscan ClinSeat™ system generated data over a user-selected period at one-minute intervals. The results of each measurement were a complete pressure map of the interface pressure across the sensor mat. The Clinseat™ system provided a variety of ways in which to view the results of measured interface pressure data. In this study the 2D (two-dimensional) Contours View, the 3D (three-dimensional) Wireframe View and the Peak Interface Pressure vs. Time Plot were utilised³⁵.

Ten separate mapping measurements were taken for the point of peak interface pressure from which the mean peak interface pressure (PIP) and pressure gradients were calculated. Two-dimensional contours views and three-dimensional Wireframe Views were used in data collection. Pressure gradient is the difference between pressure at the peak point and a point a specified distance away (1.5 or 2.5 cm for this study). The steepest gradients are not necessarily in the same direction but are where the greatest gradient is around the point or PIP.

Measurements to determine gradients were taken for each of the 10 recordings for each patient and averaged to determine a single pressure gradient for each patient at 1.5 cm and 2.5 cm using the following approach:

1. The peak interface pressure and the (x_p, y_p) position of the peak interface pressure point were recorded.
2. The difference in pressure between the point of peak interface

Table 1: Demographic characteristics compared according to pressure injury risk category

Characteristics	Number of patients (% of sample)	Mean (SD)	Median (min, max) ^a	Mode
Risk category				
Not at risk	55 (46)			
At risk	65 (54)			
Not at risk				
Females	27 (49)			
Males	28 (51)			
Age (years)		50.6 (18.97)		
Risk score			6 (2, 9)	8
At risk				
Females	25 (38)			
Males	40 (62)			
Age (years)		68 (12.7)		
Risk score			14 (10, 28)	17

^aMedians provided for Waterlow risk assessment tool score because not normally distributed

pressure (PIP) and that at 1.5 cm and 2.5 cm (ΔP_n) was simply determined from:

$$\Delta P_1 = PIP - P_1 \tag{Equation 1}$$

$$\Delta P_2 = PIP - P_2 \tag{Equation 2}$$

- The distance (D_n) between the point of peak interface pressure and the 1.5 cm and 2.5 cm measurements was obtained from the following equations:

$$D_1 = \text{square root } ((x_1 - x_p)^2 + (y_1 - y_p)^2) \tag{Equation 3}$$

$$D_2 = \text{square root } ((x_2 - x_p)^2 + (y_2 - y_p)^2) \tag{Equation 4}$$

- The pressure gradient (G_n) in mmHg/cm was then obtained by

$$G_1 = \Delta P_1 / D_1 \tag{Equation 5}$$

$$G_2 = \Delta P_2 / D_2 \tag{Equation 6}$$

- The gradient for each of the 10 pressure measurements was individually determined at 1.5 cm and 2.5 cm from the peak interface pressure site, and averaged to arrive at the recorded pressure gradients for each patient.

Analysis

SPSS v16 was used for all analyses. The complete set of variables as previously described was individually explored for normality using the Kolmogorov-Smirnov Test (K-S Test). As initial tests indicated that none of the variables were normally distributed the data were transformed using a logarithmic transformation (base 10) and retested. The logarithmically transformed weight, peak interface pressure, gradient 1.5 cm and 2.5 cm were found to be normally distributed. The transformed BMI and Waterlow risk assessment tool scores were not normally distributed.

A series of correlation tests were conducted on all variables to determine the strength and direction of the linear relationship between pairs of variables. The Pearson product moment correlation coefficient was used for variables that were normally distributed. As BMI and Waterlow risk scores were not normally distributed Spearman’s rank-order correlation was employed to assess correlations involving these variables. The strength of the correlations observed were assessed from the coefficient of correlation (r) as being (1) small for r between 0.1 and 0.29, (2) medium for r between 0.3 and 0.49 and (3) large for r between 0.5 and 1.0³⁶.

RESULTS

One hundred and twenty-six participants consented to participate and 124 completed the trial. Data from four further participants were excluded due to erroneous or missing data. The demographic and clinical profile of the final 120 patients is presented in Table 1.

The mean peak interface pressures and gradients recorded are shown in Table 2. As can be seen from the table, the mean pressure reduces as the distance from the peak interface pressure reduces.

Table 2: Mean peak interface pressures and gradients around peak interface pressure point

Position	Mean pressure (mmHg)	Mean gradient (mmHg/cm)
Peak interface pressure point	54.9	
1.5 cm	29.8	11.4
2.5 cm	24.0	9.3

The results of the analysis, showing the coefficients of correlation for each of the relationships above, are provided in Table 3. Four large correlations, using the Cohen description for size of correlation, were identified³⁶. Weight correlated strongly with BMI as expected, given the direct relationship between these risk factors (correlation coefficient 0.82). Peak interface pressure correlated with gradient 1.5 cm and gradient 2.5 cm with correlation coefficients of 0.77 and 0.78 respectively. In addition, gradient 2.5 cm and gradient 1.5 cm had a correlation coefficient of 0.90. All relationships had a positive correlation, thereby showing that as one value increases the second parameter also increases at some rate.

DISCUSSION

Numerous researchers have highlighted that pressure and shear accompany one another through localised pressure compressing tissue and thereby distorting adjacent tissues^{9,10,13}. Furthermore, it has also been highlighted that high pressure gradients are known to generate large shear forces²²⁻²⁴. Given that peak interface pressure is used in the calculation of gradient, some degree of correlation was anticipated. Given that these gradients were both measured from the point of peak interface pressure again some degree of correlation was expected. The high level of correlation between peak interface pressure and pressure gradient therefore suggests that as peak interface pressure increases, the area surrounding the site of the peak interface pressure becomes increasingly subject to higher gradients and hence to higher shearing forces.

The measured mean pressures and mean gradients provide indications of the nature of the area surrounding the point of peak interface pressure. First, the pressure is experienced to the 1.5 cm distance in a ‘V’ shape rather than as a ‘U’ or bathtub shape. In the event that the latter description was correct the gradient to 1.5 cm would be essentially flat, with a steep drop-off after that point. Second, the area around the peak interface pressure point that is

subject to the highest pressure gradient is restricted in size, and increases slowly with increasing pressure. The correlation between peak interface pressure and gradient at 1.5 cm range indicates that these two indices increase together. In the event that the area subject to high pressure expanded with increasing interface pressure, the gradient to 1.5 cm could be expected to remain static or reduce as either of these scenarios would have a lower correlation coefficient. Third, the impact of pressure reduces with distance from the point of peak interface pressure. The pressure gradient in the region between 1.5 cm and 2.5 cm from the point of peak interface pressure is less than that to 1.5 cm. The distance from 1.5 cm to 2.5 cm can, therefore, be considered as a more gently inclined or flattened ring surrounding this conical region.

In this way this research is consistent with the concept that pressure is transmitted into the tissue layers in a conical formation or V-shaped pressure gradient^{2,28}. It has also been noted that whilst the point of the cone may provide visual indications, the whole of the cone needs to be considered in prevention management²⁸.

The high level of correlation between peak interface pressure and the pressure gradients at both 1.5 cm and 2.5 cm show that the area at the base of the ‘cone-like’ pressure damaged area remains essentially constant rather than increasing as the peak interface pressure increases. Currently there is no empirical measurement of the dimensions of this underlying tissue damage. It may be that staggered measurement of pressure gradients can be used to provide demarcation of the area of suspected deep tissue damage. This study has only investigated gradients to 1.5 cm and 2.5 cm, but it would be interesting to calculate a wider range of gradients to see if there were clearly observable boundaries. From these measurements, a mathematical description of the distribution of pressure across the affected area could be developed. In addition, from that description, and with a time dimension included, it would be possible to

Table 3: Correlation between weight, BMI, Waterlow risk assessment score, peak interface pressure, gradient 1.5 cm and 2.5 cm

Variables	Weight	BMI ^b	Waterlow score	Peak interface pressure	Gradient 1.5 cm
BMI	0.82 ^b p<0.01	-			
Waterlow score	0.15 ^b (p = 0.09)	0.23 ^b (p =0.01)	-		
Peak interface pressure	0.23 ^a (p=0.01)	0.16 ^b (p=0.07)	0.19 ^b (p=0.04)	-	
Gradient 1.5 cm	0.22 ^a (p=0.01)	0.17 ^b (p=0.06)	0.09 ^b (p=0.31)	0.77 ^a (p<0.01)	-
Gradient 2.5 cm	0.18 ^a (p=0.06)	0.09 ^b (p=0.34)	0.10 ^b (p=0.26)	0.78 ^a (p<0.01)	0.91 ^a (p<0.01)

^aPearsons r test applied

^bSpearman’s rho test applied

determine the total amount of pressure contained within that pressure intensity distribution and to more closely examine the nature of the inverse pressure-time relationship³⁷.

Swain and Bader²⁰ have reported that no link has been discerned between weight and interface pressure, and between BMI and interface pressure. Defloor¹³ has, however, indicated that body build, and by extension weight, is a contributing factor for the intensity of compressive force as a causal factor for pressure injury development. This study has shown no correlation between peak interface pressure and weight, nor between peak interface pressure and BMI, thereby suggesting that further research is required in this area.

In clinical practice, interface pressure mapping can provide valuable visual information that augments assessment of the patient's skin and potential for skin breakdown. It provides visual and real time pressure mapping that allows both clinicians and patients to see peak pressures occurring, and informs targeted preventative interventions.

All too often, currently it is only when skin discolouration occurs that preventative strategies are applied. This is, however, often after the start of underlying tissue and skin damage. The interface pressure mapping system quantifies the peak pressure and potential pressure distribution in a 'V'-shaped gradient in the underlying tissues, therefore signifying that preventative strategies that should focus on a wider distribution of relief.

Preventative strategies such as prophylactic dressings⁸ being used in current practice should include consideration of the pressure and shear distribution wider than the peak pressure or visible injury site. Proper dressing, size, selection and availability in pressure injury prevention play a significant role in the ability to provide a protective impact on the at-risk tissue³⁸. If prophylactic dressings are to play a greater role in skin care, then a greater range of product size needs to be available to clinicians³⁹.

Limitations

There are some limitations to the study. The variation between peak interface pressure and actual interstitial pressure is not known and may be very wide. The highest gradient may not be at a point of peak pressure and it is also difficult to extrapolate the implications of the study findings to suspected deep pressure injury so conclusions must be circumspect.

CONCLUSION

The use of pressure mapping systems, and the associated visualisation of the distribution of pressure across the interface surface, has allowed the identification of areas of high pressure on tissue at the interface and the calculation of associated pressure gradients. The widely held view that high gradients give rise to high shear forces, and that high shear increases the impact of pressure, means that the ability to visualise gradients may provide an almost immediate indication of areas that may be prone to pressure injury development, including the development of suspected deep tissue injury.

The results of this study reinforce the notion of a conical pressure distribution as first postulated by McClemont²⁸. However, a number

of areas for further study are associated with the investigation of pressure gradients. It has been observed earlier in this study that measurement of both the 1.5 cm gradient and the 2.5 cm gradient from the peak interface pressure point resulted in an expected high correlation between these variables and failed to illuminate the true nature of the gradient to the 2.5 cm distance. Further investigation to explore all points on the gradient between the peak interface pressure point and the 2.5 cm distance would provide more information on the structure and extent of the pressure-affected area. In a similar manner, extension of the measured area beyond the 2.5 cm distance, and the determination whether gradients at these extended distances correlated with interface pressure could also be undertaken. This analysis could be useful in determining the typical extent of the pressure-affected region around a pressure point, and whether this area increases with increasing pressure or whether there were clearly observable boundaries.

ACKNOWLEDGEMENTS

The study was funded through an AusIndustry Grant (GRA02857) in partnership with Australian Healthcare Industries (formally Bosshard Medical).

Thanks to staff of the Canberra Hospital and Calvary Health Care ACT for assistance in this research study. Furthermore, thanks to participants of the 15th Annual European Pressure Ulcer Meeting in Cardiff, UK, in 2012 where a summary version of this paper was presented. Feedback and questions have assisted in refining presentation of the ideas.

Thanks also to Associate Professors Diane Phillips and Judy Currey, Deakin University, Co-supervisors for Ms Dunk's MNurs(Research).

Conflict of interest

We appreciate the help of Australian Healthcare Industries, who provided financial support as part of the AusIndustry grant agreement through provision of some equipment but did not influence the design or analysis of the study in any way.

REFERENCES

1. Collier M & Moore Z. Etiology and risk factors. In: Romanelli M, Clark M, Cherry G, Colin D & Defloor T (eds.). *Science and Practice of Pressure Ulcer Management*. London: Springer, 2006, pp. 177–81.
2. Maklebust J & Sieggreen MY. *Pressure ulcers guidelines for prevention and management*. 3rd edn. Pennsylvania: Springhouse Corporation, 2001.
3. Ousey K. *Pressure area care*. Oxford: Blackwell Publishing, 2005.
4. Torra i Bou JE, Garcia-Fernandez FB, Pancorbo-Hidalgo PL & Furtado K. Risk assessment scales for predicting the risk of developing pressure ulcers. In: Romanelli M, Clark M, Cherry G, Colin D & Defloor T (eds.). *Science and Practice of Pressure Ulcer management*. London: Springer, 2006.
5. Baharestani MM, Black JM, Carville K *et al.* Dilemmas in measuring and using pressure ulcer prevalence and incidence: an international consensus. *Int Wound J* 2009; 6:97–104.
6. Harrison MB, Logan J, Joseph L & Graham ID. Quality improvement, research, and evidence-based practice: 5 years experience with pressure ulcers. *Evid Based Nurs* 1998; 1:108–10.

7. European Pressure Ulcer Advisory Panel & National Pressure Ulcer Advisory Panel. Prevention and treatment of pressure ulcers: Clinical Practice Guideline. Washington DC: National Pressure Ulcer Advisory Panel, 2009.
8. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel & Pan Pacific Pressure Injury Alliance. Prevention and treatment of pressure ulcers: Clinical practice guideline. Perth: Cambridge Media, 2014.
9. Reger SI, Ranganathan VK, Orsted HL, Ohura T & Gefen A. Shear and friction in context. In: Calne S (ed.). International review. Pressure ulcer prevention: pressure, shear, friction and microclimate in context. A consensus document. London: Wounds International, 2010, pp. 11–8.
10. Takahashi M, Black JM, Dealey C & Gefen A. Pressure in context. In: Calne S (ed.). International review. Pressure ulcer prevention: pressure, shear, friction and microclimate in context. A consensus document. London: Wounds International, 2010, pp. 2–10.
11. Rycroft-Malone J & McInnes E. Pressure ulcer risk assessment and prevention. Improving Practice: improving care. London: Royal College of Nursing, 2001.
12. Defloor T & Grypdonck MFH. Do pressure relief cushions really relieve pressure? *Western J Nurs Res* 2000; 22:335–50.
13. Defloor T. The risk of pressure sores: A conceptual scheme. *J Clin Nurs* 1999; 8:206–16.
14. McNally P. Commitment to prevention policies should be a priority. Irish Nurses and Midwives Organisation.
15. Bader DL. The recovery characteristics of soft tissues following repeated loading. *J Rehab Res Dev* 1990; 27:141–50.
16. Hagsisawa S, Shimada T, Arao H & Asada Y. Morphological architecture and distribution of blood capillaries and elastic fibres in human skin. In: Clark M (ed.). Pressure Ulcers: Recent advances in tissue viability. Salisbury: Mark Allen Healthcare Ltd, 2004, pp. 39–55.
17. Dunk AM & Arbon P. Is it time for a new descriptor 'pressure injury': a bibliometric analysis. *Wound Practice & Research* 2009; 17:201–7.
18. Australian Wound Management Association. Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury. Western Australia: Cambridge Media Osborne Park, 2012.
19. Oomens CWJ, Loerakker S & Bader DL. The importance of internal strain as opposed to interface pressure in the prevention of pressure related deep tissue injury. *J Tissue Viability* 2010; 19:35–42.
20. Swain I & Bader D. The measurement of interface pressure and its role in tissue breakdown. In: Clark M (ed.). Pressure Ulcers: Recent advances in tissue viability. Salisbury: Mark Allen Healthcare Ltd, 2004, pp. 39–55.
21. McLane KM, Krouskop TA, McCord S & Fraley J. Comparison of Interface Pressures in the Pediatric Population Among Various Support Surfaces. *J Wound Ostomy Continence Nurs* 2002; 29:242–51.
22. Mueller MJ, Zou D & Lott DJ. 'Pressure gradient' as an indicator of plantar skin injury. *Diabetes Care*. 2005; 28:2908–12.
23. Rithalia S. Evaluation of alternating-pressure air mattresses: How to do it. In: Clark M (ed.). Pressure Ulcers: Recent advances in tissue viability Salisbury: Mark Allen Healthcare Ltd, 2004, pp. 68–79.
24. Rithalia S. Assessment of patient support surfaces: principle, practice and limitations. *J Biomed Eng* 2005; 29:163–9.
25. Ayello EA, Baranoski S, Lyder CH & Cuddigan JE (eds). Pressure ulcers. In: Baranoski S & Ayello EA (eds.). Wound Care Essentials and Practice Principles. 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2008, pp. 254–86.
26. Chow WW, Juvinal RC & Cockrell JL. Effects and characteristics of cushion-covering membranes. In: Kenedi RM, Cowden JM & Scales JT (eds.). *Bedsore Biomechanics*. London: Macmillan Press, 1976.
27. Exton-Smith AN. Prevention of pressure sores: Monitoring mobility and assessment of clinical condition. In: Kenedi RM, Cowden JM & Scales JT (eds.). *Bedsore Biomechanics*. London: Macmillan Press, 1976, pp. 133–9.
28. McClellent EJW. No pressure — no sore. *Nursing (Lond)*. 1984; 2:S1–3.
29. Stinson MD, Porter-Armstrong A & Eakin P. Seat-interface pressure: a pilot study of the relationship to gender, body mass index, and seating position. *Arch Phys Med Rehabil* 2003; 84:405–9.
30. Bader DL & Hawken MB. Pressure distribution under the ischium of normal subjects. *J Biomed Eng* 1986; 8:353–7.
31. Brienza DM, Karg PE, Geyer MJ, Kelsey S & Trefler E. The relationship between pressure ulcer incidence and buttock-seat cushion interface pressure in at-risk elderly wheelchair users. *Arch Phys Med Rehabil* 2001; 82:529–33.
32. Black JM, Brindle CT & Honaker JS. Differential diagnosis of suspected deep tissue injury. *Int Wound J* 2015.
33. Gardner A, Dunk AM, Eggert M, Gardner G & Wellman D. Pressure injury: an exploration of the relationship between risk factors and interface pressure. *Primary Intention* 2006; 14:140–9.
34. World Health Organisation. BMI Classification. 2010.
35. Tekscan. Clinical setting pressure assessment system: User manual. South Boston: Massachusetts, 2001.
36. Cohen JW. Statistical power analysis for the behavioral sciences. 2nd edn. Hillsdale, New Jersey: Lawrence Erlbaum Associates, 1988.
37. Stekelenburg A, Gawlitta D, Bader DL & Oomens CW. Deep tissue injury: How deep is our understanding? *Arch Phys Med Rehabil* 2008; 89:1410–3.
38. Call E. Characterization of wound dressings — physical properties and their potential impact on prevention of ulceration. 13th European Pressure Ulcer Advisory Panel. Birmingham, England, 2010.
39. Dunk AM, Gardner A & Waddington G. Anatomical location of injury in Stage 1 and Stage 2 heel pressure injuries — a pilot study. *Wound Practice & Research* 2012; 20:130–4, 6–41.



BrightSky
AUSTRALIA

specialist healthcare products at your door

**The trusted provider to Epidermolysis
Bullosa patients nationwide**

**We also provide traditional and advanced
wound care for everyone**

To find out more: ☎ 1300 88 66 01 @ info@brightsky.com.au 🌐 www.brightsky.com.au