# Pressure injury: an exploration of the relationship between risk factors and interface pressure

## Anne Gardner, Ann Marie Dunk, Marlene Eggert, Glenn Gardner & David Wellman

#### **Abstract**

Pressure injuries are a serious risk for patients admitted to hospital and are thought to result from a number of forces operating on skin tissue (pressure, shear and friction). Most research on interface pressure (IP) has taken place using healthy volunteers or mannequins. Little is currently known about the relationship between pressure injury risk and IP for hospital patients.

This relationship was investigated with a sample of 121 adult hospital patients. Pressure injury risk was evaluated using the Waterlow Risk Assessment Tool (WRAT) and IP was measured at the sacrum using a Tekscan ClinSeat™ IP sensor mat. Other factors considered were body mass index (BMI), blood pressure, reason for hospital admission, comorbidities and admission route to hospital. Patients were classified according to WRAT categories ('low risk', 'at risk', 'high risk', 'very high risk') and then remained still on a standard hospital mattress for 10 minutes while IP was measured.

Participants in the 'low risk' group were significantly younger than all other groups (p<0.001) and there were some group differences in BMI. IP readings were compared between the 'low risk' group and all of the participants at greater risk. The 'low risk' group had significantly lower IP at the sacrum on a standard hospital mattress than those at greater risk (p=0.002). Those at greater risk tended to have IP readings at the low end of the compromised IP range.

This study is significant because it describes a new, clinically relevant methodology and presents findings that challenge clinician assumptions about the relationships between pressure injury risk assessment and IP.

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(4):140-149.

#### Dr Anne Gardner\*

RN, PhD

Associate Professor in Nursing,

Cabrini – Deakin Centre of Nursing Research

Ageing and Chronic Illness

Cabrini Hospital, Malvern, VIC

Deakin University, Burwood, VIC

Tel: (03) 9508 1366 Fax: (03) 9508 1908

E-mail: anne.gardner@deakin.edu.au

#### Ann Marie Dunk

RN, BHthSc (Nurs), Wound Care Cert, MRCNA Research Assistant, The Canberra Hospital and University of Canberra Research Centre for Nursing and Midwifery Practice, ACT

### Marlene Eggert

RN, BAppl.Sc (Nsg), MN (Bioethics), Renal Cert, Grad.Cert (Diab.Ed), Grad.Dip (Mental Health Nsg) Shift Coordinator The Canberra Hospital, Garran, ACT

#### Dr Glenn Gardner

RN, PhD

Professor of Clinical Nursing, School of Nursing Queensland University of Technology, QLD

#### David Wellman

BA (Hons), MA Research Fellow, School of Nursing Deakin University, Burwood, VIC

\*Corresponding author

#### Introduction

Development of pressure injuries is a serious risk for patients admitted to hospital. Pressure injuries are usually associated with prolonged bed rest but can also develop during short periods of immobility.

The aetiology of pressure injuries is not fully understood <sup>1</sup>. Injuries result from a number of forces exerted on skin tissue, with the most important being pressure (as well as shear and friction) <sup>2</sup>. Prolonged pressure applied to a localised area of tissue can result in occlusion of capillary blood flow. This occlusion causes tissue ischaemia, cell destruction and tissue death. The resulting pressure injury not only causes great distress to patients but is also very expensive for the healthcare system <sup>3-5</sup>. Part of the expense is due to the widespread occurrence of pressure injuries. Recent reports from Victoria indicate that the point prevalence of pressure injuries amongst public hospital patients was 26.5% in 2003, and fell to 17.6% in 2006 <sup>6,7</sup>. A similar study in the ACT reported rates varying between 18% and 29% across the health sector <sup>8</sup>.

Interface (or contact) pressure (IP) is one component related to pressure injury formation and refers to the pressure applied to a person's body by the supporting surface. IP is usually measured over a small area using a sensor mat and provides scaled readings (milligrams of mercury – mmHg) that represent the level of forces between the body and the support surface <sup>9</sup>. However, the sensor mat does not distinguish the direction in which forces operate and does not measure shear or friction components.

Interpretation of IP readings and the relationship between IP and pressure injuries is complex. IPs of 32mmHg and higher exceed average capillary pressure in healthy adults <sup>10</sup> and can lead to capillary occlusion <sup>11</sup>. Indeed, 32mmHg has been referred to as the critical value for pressure injury development <sup>12</sup>. However, as discussed by Bridel and others <sup>1, 13, 14</sup>, there is evidence that pressures much lower than this may sometimes cause occlusion depending on individual differences such as length of time of pressure application, age, the collagen content of the dermis and amount of shear forces. Whilst IP is not the same as capillary closing pressure, it remains one of the extrinsic factors that can be mitigated in efforts to reduce the likelihood of at risk patients developing a pressure injury <sup>14</sup>.

Nurses use a range of clinical risk assessment tools to identify those patients at particular risk of developing pressure injuries so that appropriate interventions can be implemented. These tools are based on risk factors identified in clinical settings and investigations. Risk factors can include personal characteristics such as age and sex, as well as various health indicators such as continence status and neurological impairment.

The validity of a risk assessment tool is the degree to which the risk is correctly predicted <sup>15, 16</sup>. Establishing the validity of pressure injury risk assessment tools is difficult because there is an onus of responsibility to implement interventions on any patient at risk. Therefore, the probability of developing a pressure injury is altered by the implemented interventions. This is borne out through investigations of existing tools <sup>15, 17-20</sup>.

The major difficulty is in reconciling acceptable sensitivity (how good the test is at predicting who will go on to develop pressure injuries) and specificity (how good the test is at predicting those who will not develop pressure injuries). The most common assessment tools usually have good sensitivity but comparatively low specificity <sup>19</sup>, classifying many people at risk who do not go on to develop pressure injuries. It has recently been argued that sensitivity and specificity are not appropriate to establish the validity of risk assessment tools and that scales should be evaluated inclusive of any implemented interventions <sup>15</sup>.

Pressure reducing surfaces are devised to reduce IP on areas on the body, usually bony prominences, most at risk of pressure injury. Evaluation of the usefulness of these devices is often conducted using equipment to directly measure the amount of pressure exerted at the support surface/ body interface. However, the measurement equipment is currently too complicated, costly, sensitive and labour intensive for routine use in the clinical setting. Therefore, recommendations for use of pressure relieving equipment are based primarily on laboratory research conducted on mannequins, laboratory animals or, at best, healthy volunteers. However, healthy volunteers have patterns of tissue structure and blood flow that are quite different from the tissue structure and blood flow of the frail, ill and elderly patients who require interventions in the clinical setting <sup>13, 21</sup>. It is therefore important to conduct empirical research with the groups towards whom interventions are targeted 22, so that the relationships between IP, level of pressure injury risk and other individual characteristics can be more clearly understood.

# Methodology

As the research partner for an AusIndustry Research and Development Grant, we designed a multi-site study to investigate the relationship between pressure injury risk and the IP with hospital mattresses as part of a larger clinical trial of intervention mattresses. The study population comprised patients admitted to two acute hospitals.

A pilot study with 11 healthy adult volunteers was conducted to develop protocols for the main study and to evaluate the suitability of several mattresses for use as IP measurement surfaces. For the pilot, measurements were taken on both sacrum and trochanter on each of the mattresses. Trochanter measurements were found to be widely variable and measurement at this site was discontinued. The convoluted foam (egg shell) Comfort Plus<sup>TM</sup> mattress was chosen to represent the standard hospital mattress for the study because IP readings were less dispersed and there was greater testretest reliability for IP measured on this mattress when compared to another test mattress (cubed foam mattress). Adding to the external validity of using this mattress was that it was widely used in NSW and ACT hospitals at the time of the study, including the participating hospital sites. The final study protocols are briefly described in the methods section below. Full details of study protocols and training manual are available from the author on request.

#### Method

#### Design

A prospective multi-site design was used for the study, with a convenience sample of hospital inpatients. Participants were assessed for pressure injury risk and then had IP measured lying on a standard hospital mattress.

#### **Participants**

After gaining approval from relevant Human Research Ethics Committees, participants were recruited from two Australian teaching hospitals. A total of 126 patients consented to participate in the study from a variety of medical, surgical and midwifery wards. Inclusion criteria were that patients were at least 16 years of age and could move or be moved from one bed to another. Only patients thought capable of lying in one position for two 20 minute periods were invited to participate. Patients were excluded if they were clinically too unwell, were being discharged within 3 hours or were expecting surgery within 6 hours.

Two consenting patients were later excluded because they were unable to tolerate the extended period lying motionless required for a component of the larger study. Both patients had a body mass index (BMI) greater than 35 (classified as obese). Three participants had extremely high IP readings

(>1.5 interquariles above the IP mean of participants with similar pressure injury risk), and were subsequently excluded from data analyses; the final sample size was 121. Participants ranged in age from 18-88 years (M=60.10, SD=18.02), and 43% were female (n=52). Eight participants (6.6%) were recruited from obstetric wards, 59 from medical wards (48.8%) and 54 from surgical wards (44.6%).

#### **Survey instrument**

Demographic data, patient characteristics and a pressure injury risk assessment tool were combined into a single survey instrument. Demographic data included sex, age, weight and height. Data collected on patient characteristics included blood pressure, hospital ward (medical/surgical/obstetric), the reason for admission, current comorbidities and admission route to hospital.

Comorbidities were evaluated using the Deyo, Cherkin & Ciol <sup>23</sup> adaptation of the Charlson Comorbidity Index (CCI) <sup>24</sup>, a tool used widely in Australia and internationally. The CCI is designed to measure the level and intensity of comorbid health conditions and contains 19 categories based on the ninth revision of the International Classification of Diseases diagnoses codes (ICD-9-CM) <sup>25</sup>.

For the current study, selected CCI scales were used to form three comorbid condition categories: neoplasm comorbidity, cardiac comorbidity and endocrine comorbidity. The neoplasm comorbid condition category used the CCI scales of any tumour, leukaemia, lymphoma, and metastatic solid tumour. Participants with a myocardial infarction and/or congestive heart failure on the CCI were re-coded as having a cardiac comorbidity. Participants with diabetes and/or diabetes with end organ damage were re-coded as having an endocrine comorbidity.

Pressure injury risk was assessed with the Waterlow Risk Assessment Tool (WRAT) <sup>26, 27</sup>. The WRAT identifies pressure injury risk along 11 separate risk domains. Some domains are measured with mutually exclusive categories (i.e. age group) while others are additive and participants can score along more than one dimension of the domain (i.e. skin type and visual areas). Scores from the 11 domains are added to provide a total pressure injury risk figure which then places a participant into one of four risk groups. These are 'low risk' (<10), 'at risk' (10-14), 'high risk' (15-19) and 'very high risk' (20+). Clinical interventions are usually directed at patients scoring 10 or greater on the WRAT scale. While any person can develop pressure injuries, the low risk group represents participants not targeted for any specific pressure injury

interventions and, for the purpose of this study, this group will be referred to as the 'no risk' group.

## Study equipment

The study mattress [ComfortPlus<sup>TM</sup>] was placed on a nonelectric standard hospital bed with metal base, and bedding was prepared so that the sheet was placed on the mattress without tension and there were no pleats or folds in the area of the pressure sensor mat.

All IP measurements were taken using the Tekscan Advanced Clinical Seating Pressure Assessment System [ClinSeat<sup>TM</sup>], v5.25. A pressure sensor mat (53x49cm) was placed on the mattress and connected to a notebook computer with dedicated software. Following procedures recommended by the supplier, each sensor mat was used for 50 measurements before being exchanged for a new mat to guard against creep. The pressure sensor mat was calibrated daily and prior to the first use of a new mat.

#### Data collection

Data collection took place in the hospital wards and units, with equipment being transported to each clinical area daily. After gaining participant consent, demographic information, patient characteristics and WRAT data were gathered from a mixture of participant interviews and hospital records. All participants were assisted to transfer onto the study mattress and wore hospital pyjama pants over their underwear for each IP measurement cycle. A paper marker was placed on the sacrum to identify this site by row and column on the IP measurement computer. The marker was carefully removed after site identification and participants were instructed to remain supine with one pillow supporting their head at a measured 30° angle. Participants were requested to remain still and not to cross their legs during the data collection period. IP was measured continuously for 10 minutes as a movie, with a snapshot taken every minute. The colour image snapshots were converted to measures of pressure calculated as mmHg. For each snapshot, the peak or maximum mmHg reading at the sacrum site was entered into the database, making a total of 10 readings for each participant.

WRAT data collection procedures were standardised among the research team to provide consistency of scoring. Standardisation involved education sessions for research staff and multiple scoring of some participants to evaluate consistency of scores amongst research staff. Inter-rater reliability was calculated for the WRAT based on a sample of six participants who were examined independently by the

three nurse research assistants during the course of the study. There was 100% agreement on sub-scale and total WRAT scores.

#### Data analysis

All data were managed using the SPSS v11.5 computer software package. Demographic and patient characteristics were summed and WRAT scores were calculated for each patient and grouped according to WRAT risk level. Blood pressure was examined and classified according to World Health Organization<sup>28</sup> recommendations (low, normal or high). Hospital wards were recoded to represent either medical, surgical or obstetric wards. The reason for admission provided by participants was recoded into eight categories, seven of which represented reasons specific to different anatomical systems (i.e. musculo-skeletal, respiratory, cardiovascular) and the eighth was a generic 'other' category. Comorbidities were examined using the categories of neoplasm comorbidity, cardiac comorbidity and endocrine comorbidity. There were two options available for route of admission to hospital. These were ambulance/emergency admission and scheduled admission from home.

IP scores were examined for consistency. There were some individual IP readings of zero that were not included in further analysis because they represented suspect readings. There were also some IP readings that were not similar to other IP readings for individual participants. The decision was made to delete any IP readings that were greater or less than 5mmHg different from any other IP readings for a participant. As a result, four participants had nine IP measurements available, one participant had eight IP readings and another participant had seven IP readings available. The remaining 115 participants had all 10 IP readings.

IP means were calculated for each participant and for each of the WRAT categories. ANOVAs with planned comparisons were used to compare WRAT categories along scaled demographic variables and IP means. Planned comparisons involved comparing the WRAT no risk group to each of the other WRAT groups. Chi-square was used to compare sex distributions, and descriptive statistics were used for patient characteristics.

#### Results

To investigate whether participants differed along demographic variables according to level of pressure injury risk (WRAT risk group categories), mean age and BMI scores were calculated for each group and ANOVAs conducted,

with planned comparisons calculated between the no risk WRAT group and each of the other groups. Significant group differences were found for age (F [3,117]=12.48, p<0.001) and BMI (F [3,117]=3.73, p=0.01). Sex frequencies were calculated for each risk group. Means, frequencies and the results of the planned comparisons for age and BMI are presented in Table 1.

The results of the planned comparisons indicated that the no risk WRAT group were significantly younger (F=22.55, p<.001) and had a lower BMI (F=6.33, p=0.002) than the very high risk group. Other significant comparisons indicated that the no risk group were younger than both the at risk (F=15.54, p<0.001) and high risk (F=18.22, p<0.001) groups but did not significantly differ from these groups in terms of BMI. WRAT risk groups did not differ significantly in their sex balance. The overall BMI mean was 27.01 (SD=5.67).

# Patient clinical characteristics and WRAT risk categories

To describe the breadth of clinical characteristics of participants, frequencies of clinical characteristics were examined according to WRAT risk group categories. The percentage of participants at no risk of pressure injuries was calculated for each clinical characteristic. The remainder of the participants with that specific characteristic were therefore categorised into one of the WRAT groups at greater risk of developing pressure injuries. Results are presented in Table 2 and describe the distribution across the four WRAT groups. Because of the small group sizes for many of the clinical characteristic groups, no inferential analyses were conducted. Therefore, generalisability of these results is limited.

Less than one third of participants in the high blood pressure group were in the no risk WRAT group, compared to approximately one half of the participants with normal blood pressure and three quarters of those with low blood pressure. Less than 30% of participants admitted to hospital for respiratory, cardiovascular and skin reasons were in the no risk WRAT group. This compares to 100% of the gynaecological participants and almost 70% of the genitourinary participants graded in the no risk WRAT group.

Only 14% of participants with cardiac comorbidities (n=4) and 15% of those with endocrine comorbidities (n=3) were classified in the no risk WRAT category. For participants with a neoplasm comorbidity, this figure was almost 40% (n=11). Additionally, 13 participants had two comorbid conditions. Four of these had both a neoplasm and a cardiac comorbidity, eight had a cardiac and an endocrine comorbidity, and one had a neoplasm and an endocrine comorbidity. It is of note that all nine participants in the very high risk WRAT category presented with dual comorbidities.

## IP and WRAT risk categories

IP means were calculated according to WRAT risk categories. Means are presented in Table 3.

Participants in the no risk group had an IP mean that was approximately 4.2mmHg lower than the at risk group and more than 7mmHg lower than the other two groups. Because group sample sizes were so variable, participants in the at risk, high risk, and very high risk WRAT categories were combined into a single group (risk). The IP mean for the risk group (n=66) was 32.44mmHg (SD=11.96). A two-tailed t-test was then conducted comparing the means for the risk and no risk groups. Levene's test for equality of variance suggested that the two IP means did not have equal variance and the t-test was therefore conducted without assuming equal variance. The result indicated that the risk group had a significantly higher IP mean than the no risk group (t=3.11, df=116.05, p=0.002).

Table 1. Demographic means and frequencies by WRAT risk group categories with planned comparisons.

No risk (n=55)		At risk (n=35)		High (n=22)		Very high (n=9)	
M	SD	M	SD	M	SD	M	SD
50.61	18.97	*66.15	12.78	△68.83	12.62	† <b>73.16</b>	12.09
25.67	4.78	27.43	5.20	27.64	6.40	<sup>f</sup> 32.00	7.97
27/	28	12/	/23	9/	13	4.	/5
	M 50.61 25.67	M SD 50.61 18.97	M         SD         M           50.61         18.97         *66.15           25.67         4.78         27.43	M         SD         M         SD           50.61         18.97         *66.15         12.78           25.67         4.78         27.43         5.20	M SD M SD M 50.61 18.97 *66.15 12.78 ^68.83 25.67 4.78 27.43 5.20 27.64	M SD M SD M SD  50.61 18.97 *66.15 12.78 ^68.83 12.62 25.67 4.78 27.43 5.20 27.64 6.40	M         SD         M         SD         M           50.61         18.97         *66.15         12.78         ^68.83         12.62         †73.16           25.67         4.78         27.43         5.20         27.64         6.40         /32.00

BMI calculated as weight divided by height

Significant planned comparisons with the 'no risk' WRAT group:

† F=22.55, p<0.001

f F=6.33, p=-.002

F=15.54, p<0.001 Δ F=18.22, p<0.001

#### Discussion

This study presented data about the relationship between pressure injury risk, as measured by the WRAT, and IP measured at the sacrum for hospital patients lying on a standard hospital mattress. Whereas previous studies have investigated pressure injury risk and IP separately <sup>19, 29</sup>, the relationship between the two has not previously been evaluated. Previous research has measured IP on various support surfaces using healthy volunteers or mannequins <sup>29</sup>. This study is therefore unique because participants represented a broad cross-section

of adult inpatients across a wide range of ages and health concerns.

Participants who were not considered to be at risk of developing pressure injuries were found to have IP readings within the acceptable IP range. In contrast, those at risk of developing pressure injuries recorded average IP readings generally at the low end of the compromised IP range based on the average capillary pressure found by Landis <sup>10</sup> (IP<32mmHg).

Table 2. Participant clinical characteristics by WRAT risk group categories.

WRAT risk group	No risk (n=55)	At risk (n=35)	High (n=22)	Very high (n=9)	Total (n= 121)	
Characteristics	n (%)*	n	n	n	'n	
Blood pressure group						
Low	7 (78%)	1	1	0	9	
Normal	38 (49%)	21	14	7	80	
High	10 (31%)	13	7	2	32	
Admission reason						
Musculo-skeletal	7 (32%)	7	6	2	22	
Gastrointestinal	12 (57%)	6	3	0	21	
Genito-urinary	10 (67%)	4	1	0	15	
Cardiovascular	5 (28%)	8	4	1	18	
Gynaecological	11 (100%)	0	0	0	11	
Respiratory	2 (22%)	3	3	1	9	
Skin	2 (29%)	2	1	2	7	
Other	6 (33%)	5	4	3	18	
Comorbidities <sup>A</sup>						
Neoplasm	11 (38%)	11	7	0	29	
Cardiac	4 (14%)	14	5	5	28	
Endocrine	3 (15%)	7	6	4	20	
Admission route						
Ambulance/emergency	28 (39%)	23	15	6	72	
Home	27 (55%)	12	7	3	49	

<sup>\*</sup> Percentages represent the percent of participants in each patient characteristic category in the 'no risk' group.

Table 3. Interface pressure means by WRAT risk categories.

Risk category	n	М	SD	Range	95% CI
No risk	55	26.64	8.45	9.0 – 47.2	24.36 – 28.93
At risk	35	30.80	11.17	14.8 – 60.8	26.96 – 34.64
High	22	34.42	13.74	16.3 – 62.8	28.33 – 40.51
Very high	9	33.97	10.54	24.1 – 55.4	25.86 – 42.07

Δ Thirteen participants had more than one comorbidity.

From a clinical perspective, this finding adds weight to the claim that the WRAT, like many other risk assessment tools, over-estimates the risk of pressure injury <sup>19</sup>. Over-estimation of risk is likely to lead to complacency in clinical practice. As Gebhardt <sup>30</sup> argues, clinicians realise that few patients identified as being 'at risk' actually go on to develop a pressure injury. This results in complacency when there are many competing claims on clinicians' time and intervention can be delayed, possibly resulting in greater prevalence. More work is therefore needed to reduce the over-estimation of risk.

In this study, although there were significant differences in IP readings depending on WRAT risk category, the statistical comparisons between each WRAT category were not possible given the variety of WRAT group sizes. Future research could further investigate whether there are any other significant differences in IP across the WRAT risk categories. It is also unclear how IP varies across WRAT risk categories for people with specific health concerns and across specific ages. For example, do people in their 60s with heart conditions have different IP readings if they are in different WRAT categories based on other demographic or clinical characteristics.

There were patterns of clinical characteristics that need to be tested in future studies. For example, a greater percentage of participants with high blood pressure were in one of the groups at risk of developing a pressure injury compared to those with low or normal blood pressure. Participants in medical wards were more likely to be classified as at risk of pressure injury than participants in surgical or obstetric wards. Not surprisingly, all participants admitted for obstetric reasons were classified in the no risk WRAT category. This contrasts with the four admission reasons with the smallest number of participants: respiratory, cardiovascular, skin and other. Participants admitted to hospital for these reasons were less likely to be classified in the no risk WRAT group than one of the three at risk groups. However, sample size in these groups restricts generaliseability to other populations.

Admission route to hospital results suggest that patients admitted from home were more likely to be in the no risk WRAT group than those admitted as accident and emergency patients. There is some indication in the literature that those transferred from other hospitals are more likely to be at risk of pressure injury <sup>22</sup>. One possible explanation for this increased risk is that inter-hospital transfer may be a surrogate for increased severity of illness as patients are transferred from smaller hospitals to larger tertiary centres. Increased pressure injury risk may also apply for patients requiring emergency admission to hospital.

It is of note that the BMI mean values for the no risk, at risk and high risk groups were all in the clinically overweight category <sup>31</sup>, and the very high risk group mean was in the obese category. The relationships between BMI, IP and pressure injury risk are not straightforward. For instance, the WRAT scores more risk to people who are underweight than people who are obese. In contrast, a recent study of hospitalised patients found that being 95kg or heavier was more strongly associated with the development of pressure ulcers than being 54kg or lighter <sup>32</sup>.

There are some limitations to the study. We have used IP as a surrogate measure for changes in skin viability at the sacrum. It is one of the three most important extrinsic parameters to examine when evaluating intervention mattresses used to prevent pressure injury (the other two being bloodflow and microclimate) <sup>33</sup>. While it cannot be assumed that IP and bloodflow correspond exactly, IP is much more easily measured non-invasively. There is an established clinical literature in the use of IP for development of devices for individual orthopaedic and paraplegic patients, so the strengths and limitations of the technology are well understood <sup>33-35</sup>.

## Conclusion

This research has made important contributions to the field on two levels. First, the findings are meaningful for clinicians in bringing new knowledge to our assumptions about risk factors, risk assessment and pressure injury. Second, this study has established a methodological breakthrough in moving this approach to pressure injury research out of the laboratory into the clinical environment and therefore producing more clinically relevant results. As such, we have instituted a new approach to researching pressure injury risk assessment strategies.

# Acknowledgements

The study was funded through an AusIndustry Grant (GRA 02857) in partnership with Australian Healthcare Industries (formerly Bosshard Medical). Thanks to staff of the Canberra Hospital and Calvary Health Care ACT for assistance in this research project. Thanks to Ms Donna Mowbray for her valuable input in the early stages of the project. We would also like to acknowledge the dedication of the research nurses who collected the data – Mel Symons, Cecelia Wakeling, Stephen Roderick, Vicki Brown and Sam Wright.

#### Disclaimer

Australian Healthcare Industries, whilst providing support to the project, has not influenced the design or analysis of the study in any way.

#### References

- Thompson D. A critical review of the literature on pressure ulcer aetiology. J Wound Care 2005; 14(2):87-90.
- Australian Wound Management Association. Clinical Practice Guidelines for the Prediction and Prevention of Pressure Ulcers (1st ed). West Leederville: Cambridge Publishing; 2001.
- Graves N, Birrell FA & Whitby M. Modeling the economic losses from pressure ulcers among hospitalized patients in Australia. Wound Repair Regen 2005; 13(5):462-7.
- Voss AC, Bender SA, Ferguson ML, Sauer AC, Bennett RG & Hahn PW. Longterm care liability for pressure ulcers. J Am Ger Soc 2005; 53(9):1587-92.
- Franks PJ, Winterberg H & Moffatt CJ. Health-related quality of life and pressure ulceration assessment in patients treated in the community. Wound Repair Regen 2002; 10(3):133-40.
- Victorian Department of Human Services. PUPPS 3: Pressure Ulcer Point Prevalence Survey: Statewide Report 2006. Melbourne: Quality and Safety Branch, Victorian Government, Department of Human Services, 2006.
- Victorian Department of Human Services. VQC Statewide: PUPPS
   Report 2004: Pressure Ulcer Point Prevalence Survey. Melbourne: Metropolitan Health and Aged Care Services Division, Victorian Government, Department of Human Services, 2005.
- Gardner A & Dunk AM. Establishment of Sustainable Processes for Pressure Injury Management in the Australian Capital Territory: Project Report August 2004. Canberra: ACT Health, 2004.
- Rithalia S & Kenney L. The art and science of evaluating patient support surfaces. Worldwide Wounds 2001.
- Landis EM. Micro-injection studies of capillary blood pressure in human skin. Heart 1930; 15:209-28.
- 11. Armstrong D & Bortz P. An integrative review of pressure relief in surgical patients. AORN 2001; **73(3)**:645, 7-8, 50-53.
- Defloor T. The effect of position and mattress on interface pressure. Appl Nurs Res 2000; 13(1):2-11.
- 13. Bridel J. The aetiology of pressure sores. J Wound Care 1993; 2(4):230-8.
- Bell J. The role of pressure-redistributing equipment in the prevention and management of pressure ulcers. J Wound Care 2005; 14(4):185-8.
- Defloor T & Grypdonck MFH. Validation of pressure ulcer risk assessment scales: a critique. J Adv Nurs 2004; 48(6):613-21.
- Polit DF & Beck CT. Nursing Research: Principles and Methods (7th ed). Philadelphia: Lippincott Williams and Wilkins, 2004.
- 17. Anthony D, Reynolds T & Russell L. A regression analysis of the Waterlow score in pressure ulcer risk assessment. Clin Rehab 2003; 17:216-23.
- Murray LD, Magazinovic N & Stacey MC. Clinical practice guidelines for the prediction and prevention of pressure ulcers. Primary Intention 2001; 9:88-97.
- Jalali R & Rezaie M. Predicting pressure ulcer risk: comparing the predictive validity of 4 scales. Adv Skin Wound Care 2005; 18(2):92-7.
- Pancorbo-Hidalgo PL, Garcia-Fernandez FP, Lopez-Medina IM & Alvarez-Nieto C. Risk assessment scales for pressure ulcer prevention: a systematic review. J Adv Nurs 2006; 54(1):94-110.
- Li Li, Mac-Mary S, Sainthillier J-M, Nouveau S, de Lacharriere O & Humbert P. Age-related changes of the cutaneous microcirculation in vivo. Gerontology 2006; 52(3):142-53.
- Papanikolaou P, Lyne PA & Lycett EJ. Pressure ulcer risk assessment: application of logistic analysis. J Adv Nurs 2003; 44(2):128-36.
- Deyo RA, Cherkin DC & Ciol MA. Adapting a clinical comorbidity index for use with ICD-9-CM administrative data. J Clin Epid 1992; 45:613-9.
- Charlson ME, Pompei P, Ales KL & MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. J Chronic Dis 1987; 40:373-83.

- World Health Organization. Manual of International Statistical Classification of Diseases, Injuries and Causes of Death. Geneva: World Health Organization, 1977.
- Waterlow J. Pressure sores: a risk assessment card. Nurs Times 1985; 81(48):49-55.
- Waterlow J. The history and use of the Waterlow Card. Nurs Times 1998; 94(7):63-7.
- World Health Organization. 1999 World Health Organization: International Society of Hypertension – Guidelines for the management of hypertension. Guidelines Subcommittee. J Hypertension 1999; 17(2):151-83.
- Rithalia S. Assessment of patient support surfaces: principle, practice and limitations. J Med Eng Technol 2005; 29:163-9.
- Gebhardt KS. Pressure ulcer research: where do we go from here? Br J Nurs 2004; 13(19):S14-S8.
- 31. World Health Organization. Global strategy of diet, physical activity and health: obesity and overweight. In: World Health Organization: http://www.who.int/dietphysicalactivity/publications/facts/obesity/en/; 2006.
- Schoonhoven L, Grobbee DE, Donders ART, Algra A, Grypdonck MH, Bousema MT et al. Prediction of pressure ulcer development in hospitalized patients: a tool for risk assessment. Qual Safety Health Care 2006; 15:65-70
- Jonsson A, Lindén M, Lindgren M, Malmqvist L-Å & Bäcklund Y. Evaluation of antidecubitus mattresses. Med Biolog Eng Comp 2005; 43:541-7.
- Moody P, Gonzales I & Cureton VY. The effect of body position and mattress type on interface pressure in quadriplegic adults: a pilot study. Dermatol Nurs 2004; 16(6):507-12.
- Karwowski W, Rodrick D & Boratyn G. Experimental evaluation of pressure distribution changes when using clinical support surfaces. Occupat Ergonomic 2004; 4:41-50.

