Title Exploring pressures, tissue reperfusion and body positioning: A pilot evaluation

Authors

Fiona Coyer, RN, PGCEA, MSc(Nursing), PhD

Professor of Nursing, Joint appointment - Intensive Care Services, Royal Brisbane and Women's Hospital and School of Nursing, Queensland University of Technology.

Butterfield St, Herston, Queensland, 4006, Australia.

Tel: +61 7 36462140   Email: f.coyer@qut.edu.au

Michele Clark, BOccThy (Hons), BA, PhD

Professor and Director of Post-graduate Studies

School of Clinical Sciences, Faculty of Health, Queensland University of Technology.

Victoria Park Rd, Kelvin Grove, Queensland, 4059, Australia.

Tel: +61 7 3138 3525   Email: mj.clark@qut.edu.au

Peter Slattery, BE(Mech) Hons, MEngSc (Research) CPEng NPER RPEQ

Director, Department of Rehabilitation Engineering

Royal Brisbane and Women's Hospital

Herston Road, Herston, Queensland, 4029, Australia.

Tel: +61 7 3646 7773   Email: Peter_Slattery@health.qld.gov.au

Peter Thomas, BPhty (Hons), PhD

Consultant Physiotherapist, Department of Physiotherapy

Royal Brisbane and Women's Hospital

Butterfield St, Herston, Queensland, 4029, Australia.

Tel: +617 36464319   Email: PeterJ.Thomas@health.qld.gov.au

Greg McNamara, BN, GradCertNsg (IntCare), Grad Dip MH

Clinical Nurse, Department of Intensive Care Medicine

Royal Brisbane & Women's Hospital

Butterfield St, Herston, Queensland, 4006, Australia.

Tel: +61 7 3646 8111   Email: Greg_McNamara@health.qld.gov.au

Chris Edwards, BAppSc (Medical Radiations Tech), MAppSc (Medical Ultrasound)

Senior Lecturer and Course Coordinator (Graduate Diploma) Medical Ultrasound

School of Clinical Sciences, Faculty of Health, Queensland University of Technology.

George St, Brisbane, Queensland, 4000, Australia.

Tel: +61 7 3138 1924 Email: c8.edwards@qut.edu.au

Jessica Ingleman, RN, BN, MAdvPracticeCritCare

Research Assistant, School of Nursing, Faculty of Health, Queensland University of Technology.

Bowen Bridge Road, Herston, Queensland, 4006, Australia.

Tel: +61 7 3138 7453 Email: j121.ingleman@qut.edu.au

John Stephenson, GradStat CMath MIMA PhD

Senior Lecturer, Biomedical Statistics,

School of Human and Health Sciences, University of Huddersfield,

Queensgate, Huddersfield HD1 3DH, United Kingdom.

Tel. +44 (0) 1484 47 1513 Email: J.Stephenson@hud.ac.uk

Karen Ousey, PhD, RGN, FHEA

Professor and Director for the Institute of Skin Integrity and Infection Prevention, University of Huddersfield, Adjunct Associate Professor, School of Nursing, Faculty of Health, Queensland University of Technology

Queensgate, Huddersfield HD1 3DH, United Kingdom

Tel. +44 (0) 1484 473462 Email: k.j.ousey@hud.acuk

Corresponding author

Fiona Coyer, RN, PGCEA, MSc(Nursing), PhD

Professor of Nursing, Joint appointment - Intensive Care Services, Royal Brisbane and Women's Hospital and School of Nursing, Queensland University of Technology.

Butterfield St, Herston, Queensland, 4006, Australia.

Tel: +61 7 36462140 Email: f.coyer@qut.edu.au

ABSTRACT

Objective To assess the relationship in healthy adults and critically ill patients between: patient position, body mass index (BMI), patient body temperature; and interface pressure (IP) and tissue reperfusion (TR). Also to assess the relationship in critically ill patients between: sequential organ failure assessment (SOFA) score, Braden Scale score for predicting pressure injury risk, Acute Physiology and Chronic Health Evaluation II (APACHE II) severity of disease classification score, and IP and TR.

Setting 27-bed intensive care unit (ICU) of an Australia tertiary hospital.

Participants 23 low- and high-acuity ICU patients and 9 healthy adult volunteers.

Methods IP and TR outcomes were measured at the sacrum and greater trochanter. Repeated measures analyses of variance (ANOVAs) and doubly multivariate repeated measures ANOVAs were conducted using peak pressure index (PPI), and peak time (PT), settled time constant (STC) and normalised hyperaemic area (NHA) measures of TR as outcomes. Participant type, body mass index (BMI), Braden and APACHE II scores and patient body temperature were considered as between-groups factors and covariates.

Results Not all IP readings could be obtained from ICU patients. TR readings were collected from all recruited patients, but not all TR measurements were mutually uncorrelated. Controlling for age, PPI readings substantively differed between participant types (p=0.093), with the highest values associated with high-acuity patients and the lowest with healthy adults; the association was not substantive when controlling additionally for age and BMI. The controlling variable of age was also significant (p=0.008), with older participants having higher scores than younger ones. No statistically significant associations between any measured parameter and TR variables were revealed; however, temperature was revealed to be substantively related to TR (p=0.091).

Conclusions While not being powered to detect significant effects, this pilot analysis has nonetheless determined several associations of importance, with substantive differences in outcomes observed between low- and high-acuity ICU patients; and between ICU patients and healthy volunteers.

Introduction

Pressure injuries (PI) are a significant cause of morbidity and mortality internationally.[1,2] In the United States it has been identified that individuals who developed hospital-acquired PIs have extended length of stay (4.8 vs 11.2 days), are more likely to die during the hospital stay (3.3% vs 11.2%), more likely to die within 30 days of discharge (4.4% vs15.3%) and more likely to be readmitted within 30 days (17.6% vs 22.6%).[3] In Australia, PIs are estimated to result in a median excess length of stay of 4.31 days, adding $18,964 to the cost of care per critically ill patient.[4,5] Patients in intensive care unit (ICU) settings are known to be at higher risk of PI than other hospital populations[1,6-9] . One recent study showed intensive care patients were 3.8 times more likely (RR 2.7–5.4, 95% CI) than non-intensive care patients to develop a pressure injury whilst in hospital.[10] Thus in-depth understanding of the relationships of PI development in this population is central to prevention.

Pressure injury aetiology is a complex phenomenon. Significant work has been undertaken over previous decades with our understanding of mechanisms involved continuing to evolve. Mechanical load is considered to be the primary cause of PI development due to localised skin, and underlying tissue, damage from pressure, friction, sheer, tissue distortion and skin microclimate changes. Early postulations of PI aetiology focused on pressure as the primary threat to skin integrity, as pressure forces created by the overall mass of the human body push down through the skeletal structure, leading to occlusion of capillary blood flow and resultant ischaemic injury.[11-13] Subsequent work suggested shear plays a role in PI development where shearing forces push in one direction at the top, and the opposite direction at the bottom, causing shearing deformation in the tissues. Further, friction, where the skin surface 'slides' over an opposing force such as bedlinens, contributes to PI development. However, recent work has identified tissue distortion or deformation contributes to PI development.[14,15] In this pathway mechanical loading of the tissues causes cellular damage through cellular deformation. In controlled in-vitro experiments different loading regimens showed the potential the cause cell deformation through intracellular volume changes and cytoskeletal reorganisation which may potentiate early tissue breakdown.[16] The immediate effect of deformation causing cell death is in contrast to models of ischaemia where cell necrosis develops over extended lengths of time.[17] Finally, there is growing evidence that temperature and moisture conditions on, and around (i.e support surfaces, linens), the skin in mechanically loaded areas contribute to PI aetiology.[18] It is thought that these conditions (increased temperature and increased humidity) reduce tissue tolerance and contribute to skin breakdown.

The interplay of multiple models of PI development is linked to individual susceptibility and tissue tolerance where individual patient demographic and clinical characteristics also play an important role. Due to the nature of critical illness the skin integrity of ICU patients is likely to be more compromised than other patient groups by pathophysical (e.g. hypoxia) and biophysical alterations (e.g. haemodynamic instability and reduced/poor tissue perfusion) as well as pharmacological agents (e.g. vasopressors and inotropes causing peripheral vasoconstriction).[19-25]

Whilst multiple models and pathways of PI development are acknowledged, some difficulties lie in current technological capability to measure many of these variables in real time, in clinical settings. Consequently, interface pressure (IP) mapping is a conservative and non-invasive way to measure the internal soft tissue pressures at the surface of the skin when in contact with the supporting surface[26,27] and thus would appear appropriate for ICU settings. However, to date, most of the research conducted on IP mapping has used laboratory mannequins, healthy volunteers or subacute or acute patients[28-30], thus arguably limiting the extent to which the findings can be applied to more vulnerable at-risk ICU populations.[31]

When weight is shifted onto a specific anatomical body surface, the arterial blood flow to the vascular bed can become occluded; causing reduced blood flow and stasis in the areas of high interface pressure. The laser Doppler blood flow meter is a non-invasive way to measure the time it takes for the body to restore the arterial blood flow to these areas.[32,33] When the weight of the body shifts from the load-bearing surface, there is a rapid influx of blood flow to the vascular bed and blood flow steadily returns to the pre-occlusion level. This process is known as reactive hyperaemia. In healthy volunteers, blood flow can be restored to the vascular bed within a few seconds to minutes. There is a direct link between the duration and magnitude of pressure and the length of time of reactive hyperaemia.[33] Sachse et al[32] recorded reactive hyperaemia with a laser Doppler blood flow meter and identified the range of reactive hyperaemia was between 5.7 and 9.7 minutes on healthy volunteers. These results can also be linked to a study which found that standard turning regimes practiced by experienced ICU nurses were unable to reliably unload all areas of high IP, helping to explain why PIs occur despite the implementation of standard preventive measures.[33]

Whilst many studies focus on frequent repositioning to prevent PI, a number of issues remain unresolved.[19,20] Gardner and colleagues[34] identified the relationship between PI risk assessment and IP. Other studies have examined the relationship of body positioning and IP or

IP and blood flow; however only healthy participants were recruited.[27,29,30,33] To date, no research has been conducted to examine the triad relationship between risk assessment, IP and tissue reperfusion.

Research aims and questions

This pilot study aimed to explore the effects of the type of patient position employed and the patient body mass index (BMI) category on IP and tissue reperfusion in the critically ill adult patient population, with analysis also conducted on a parallel group of healthy adults. The research questions were:

1. What is the relationship between patient position, BMI, Sequential Organ Failure Assessment (SOFA) score, IP and tissue reperfusion in the critically ill patient?

   H_1 - ICU patients with high SOFA scores will have high IP and reduced tissue reperfusion compared to ICU patients with low SOFA score and healthy adults.

2. Does the relationship between position, BMI, IP and blood flow differ in healthy adults?

   H_2 - IP mapping alone is a sufficient measure for quantifying the forces involved different positioning.

Methods

This pilot study used a prospective, observational split plot design (Figure 1).[35]

Setting

Data was collected from a 27-bed ICU of a tertiary hospital in Australia between April 2015 and October 2015. The ICU case mix included patients with major trauma, burns, neurology diagnoses, neurotrauma and medical and surgical conditions, including cancer. Intensive care patient data was augmented with data from healthy adult volunteers.

Sample

As a pilot evaluation, a formal sample size calculation was not conducted. The study was designed to recruit 27 participants, comprising 9 healthy adult volunteers and a further 18 patients from the ICU. The main inclusion criteria for the healthy adult group was age over 18 years. For ICU patient group, the inclusion criteria were age greater than 18 years, receiving mechanical ventilation and likely to remain ventilated for 24-48 hours post-recruitment to collect data. Exclusion criteria for ICU patients were: burns patients with greater than 40% total body surface area burns and/or with burn injury to the sacrum and hip region; patients

who were unable to be turned in one or more of the body positions; or haemodynamic instability (based on the clinical nurse consultant and bedside registered nurse clinical judgement).

Patient recruitment was designed to include 9 patients with low acuity and 9 patients with high acuity. Acuity was defined by each participant's SOFA score: a validated six-organ failure score measuring daily multiple organ dysfunction/failure. Each organ was graded from 0 (normal) to 4 (most abnormal), providing a score from 0 to 24 points. A SOFA score of 4.9 or less was designated as low acuity; a SOFA score of 5.0 or greater was designated as high acuity. SOFA scores in all members of the healthy adult volunteers group were defined to be zero.

Within each of the three groups, 3 participants were recruited from each of the BMI categories; hence participant type and BMI category were crossed factors in a multi-factor design. Body mass index was categorised into *normal* (BMI less than 25.0 kg/m^2), *overweight* (BMI 25.0-29.9 kg/m^2) and *obese* (BMI 30.0 kg/m^2 or greater).

Measures

Demographic and clinical characteristics were recorded for all participants, including: age, gender, BMI, SOFA score, waist measurement, body and room temperature, and co-morbidities. Length of stay in ICU (days), and data relating to administration of inotropes/vasopressors was also recorded for ICU patients; who were additionally scored according to risk assessment tools of specific interest: the Braden Scale and Acute Physiology and Chronic Health Evaluation (APACHE) II scores; plus patient temperature.

The Braden Scale sore is an internationally accepted and validated pressure injury risk assessment tool containing six categories (sensory perception, moisture, activity, mobility, nutrition, friction and shear).[36,37] Each category is rated on a scale of 1 to 4, excluding the 'friction and shear' category which is rated on a 1-3 scale, combining for a possible total of 23 points, with a higher score indicating a lower risk of developing a pressure ulcer and vice-versa.

The APACHE II score represents an assessment of the intensive care severity of illness on admission for a particular patient. The score is calculated from the patient's age and 12 routine physiological measurements: arterial partial pressure of oxygen, temperature (rectal), mean arterial pressure, arterial pH, heart rate, respiratory rate, serum sodium, potassium, creatinine, haematocrit and white blood cell count and Glasgow Coma Scale.[38]

The SOFA is a scoring system to determine the extent of a person's organ function or failure in ICU.[39] The score is based on six body systems of organ dysfunction including the respiratory, cardiovascular, hepatic, coagulation, renal and neurological systems. Each organ is graded from 0 (normal) to 4 (the most abnormal), providing a daily score of 0 to 24 points. The SOFA provides a mechanism to score severity of illness on a daily basis after ICU admission.[39]

The primary outcome measures were measures of IP and tissue reperfusion. The Xsensor X3 pressure mapping system was used to map full-body pressure at the interface of skin and the support surface at the different anatomical areas for different body positioning. This system is used routinely to measure interface pressure in patients with a range of mobility issues (e.g. severe disabilities; wheelchair users).[40,41] The mapping system used a full body sensor mat (81cm x 203cm) with 1,664 capacitive pressure sensors.[42] For this pilot investigation, PPI was defined as the highest recorded value with a 9-10 cm^2 area – the approximate contact area of a bony prominence.

Tissue reperfusion (TR) measurements were obtained using the Perimed Periflux System 5000 laser Doppler blood perfusion monitoring equipment, which tests the time taken for tissue to re-perfuse after occlusion of blood flow, known as post-occlusive reactive hyperaemia. However, this study used the participant's body weight to provide 'real life' partial blood flow occlusion followed by shifting of body weight off the specific body surface to record reactive hyperaemia. Tissue reperfusion peak time (PT), settled time constant (i.e. time for reperfusion to reach 2% of its maximum reading) (STC) and normalised hyperaemic area (NHA) were assessed in this pilot investigation.

Procedure

Patients were screened, consented and recruited by a research nurse. The pilot procedures were collected first on healthy participants and second on critically ill patients. All participants were placed on a non-powered pressure redistribution mattress or a memory foam mattress. Participants were placed in two positions; the semi-recumbent supine position and the lateral quarter turn position. In the semi-recumbent supine position, the head of the bed was elevated at 30°, and bed knee elevation was 10°; one pillow was used to support the head, a heel wedge was placed under the legs and participant's hands were positioned by their side. In the lateral quarter turn position, the bed head and knee elevation was 0°, and the whole bed was tilted 10° head up; one pillow was used to support the head, one pillow tucked firmly behind the back for support, one pillow was placed between the bent knees to ankles. For both positions the bed control and measured angles were used to ensure correct bed position and body alignment. For

each position, participants were placed on a pressure mapping mat in both the supine and lateral positions and remained in the position for 20 minutes prior to data collection.

Following this, TR measures were collected by Doppler. To accurately position the laser Doppler probe anatomical landmarks were used to identify the sacrum (a straight line between the two iliac crests, palpating until the spine was felt - the sacrum was identified as 10-12 cm below this point) and greater trochanter (the lateral aspect of the leg was palpated for a raised rounded bony prominence, one hand width inferior to the iliac crest). A baseline tissue perfusion level was recorded for two minutes. The participant was then moved into the position to load the laser Doppler probe with body weight pressure with 5 minutes allowed for in the set position (supine semi-recumbent or lateral). Following this, the participant was rolled to offload pressure and data was recorded for 10 minutes. Both healthy adults and critically ill patients were surrounded by usual sounds in the ICU environment: room temperature was constant, and the bed type, bed sheet type, hospital gown, room thermometer, tympanic thermometer were all consistent in healthy and critically ill groups.

Ethical approval

This study was approved by the respective hospital (RBWH/14/QRBW/37) and university Human Research Ethics Committees (UHREC 1400000285).

Statistical methods

All analyses were conducted using IBM SPSS (Version 22.0) statistical software (SPSS Inc., Chicago, IL). All analyses were conducted using cases with valid data only. Imputation of missing data was considered inappropriate for a pilot study analysis for which assessment of variable significance was not a primary objective.

The sample was summarised descriptively at baseline by the key grouping factor of patient type; and as a complete cohort. Patient characteristics were checked for imbalances across patient types. As a pilot investigation, sample sizes were in general insufficient to justify the analysis of the two types of outcome measures jointly in a multivariate study; hence separate analyses were conducted on the two types of outcome measures independently. For the same reason, the inclusion of multiple predictor variables could not in general be justified; however, due to a large identified disparity between the mean ages of the healthy adults and the ICU patients, age was also included in an analysis of the effect of patient type as a controlling variable.

A series of mixed (i.e. between-groups and within-groups) analyses of variance (ANOVA) were conducted on the PPI measurements taken at the sacrum and greater trochanter, using all patients; including body position as a within-groups variable; and patient type, controlling for age (Model 1), and BMI (Model 2), as predictor variables. A further series of mixed ANOVAs were conducted on PPI data from ICU patients only; also including body position as a within-groups variable; and Braden scores (Model 3), and APACHE II scores (Model 4) as predictor variables.

A series of doubly multivariate ANOVAs were conducted for TR measurements, taken at the sacrum and greater trochanter, using all patients; and considering the PF, STC and NHA measures assessed jointly. Body position was included as a within-groups variable in all models. Patient type, controlling for age (Model 5), and BMI (Model 6) were included as predictor variables. A further series of mixed ANOVAs were conducted on TR data from ICU patients only; also including body position as a within-groups variable; and Braden scores (Model 7), APACHE II scores (Model 8) and temperature (Model 9) as predictor variables. Univariate ANOVAs were planned as follow-up analyses in the event of significant findings. First order interactions between measurement position and categorical factors were included in all models. Statistical significance was assessed at the 5% significance level for all analyses.

Results

Descriptive summary of sample

A total of 42 participants were screened for inclusion in the study. A total of 33 participants were recruited comprising of 9 healthy adult volunteers, 13 low acuity ICU patients and 11 high acuity ICU patients. All demographic and clinical characteristics evaluated for the entire cohort and separately for healthy volunteers, critically ill patients with low acuity and high acuity are summarised in Table 1. Groups were well matched on the majority of key measured demographic and health variables; however, a substantive difference in mean group ages was observed, with healthy adults being about 25 years younger than patients in either of the ICU groups. Consequently age was carried forward for inclusion as a controlling variable in subsequent analyses including patient type as a between-groups factor.

[insert table 1 here]

Summary of outcome measures obtained

Complete PPI and TR readings at both sacrum and greater trochanter were obtained for all 9 healthy adult participants. Pressure mapping was completed in 5 participants in the low acuity ICU group and 1 participant in the high acuity ICU group. TR readings at both sacrum and greater trochanter were completed in 9 patients; at sacrum only in 1 patient and at greater trochanter only in 2 patients in the low acuity ICU group. PPI and TR values (mean, SD) measured at the sacrum and greater trochanter, with participants distinguished by the key factor of participant type, are summarised in Table 2.

[insert table 2 here]

Analysis of PPI and TR data

Due to low numbers of cases with valid data, these analyses were not generally powered to detect significant effects. Despite this, controlling for age, participant type was revealed to be substantively related to the PPI at the sacrum and greater trochanter assessed jointly (p=0.093). The size of the effect was moderate to large (partial-η^2=0.351).

Compared with healthy adult volunteers, PPI values for high acuity patients were 13.1 mmHg higher (95% confidence interval [CI] -17.1 to 43.1 mmHg) when measured at the sacrum and 32.5 mmHg higher (95% CI -5.03 to 70.0 mmHg) when measured at the greater trochanter. Compared with healthy adult volunteers, PPI values for low acuity patients were 2.67 mmHg higher (95% CI -17.5 to 22.9 mmHg) when measured at the sacrum and 2.90 mmHg higher (95% CI -22.3 to 28.1 mmHg) when measured at the greater trochanter.

The controlling variable of age was significantly associated with PPI at the sacrum and greater trochanter assessed jointly in a model assessing the effect of patient type on PPI (p=0.008). However, multivariate significance did not correspond to univariate significance: in this model, an increase of 1 year of age was associated with a non-significant increase of 0.412 mmHg of PPI (95% CI -0.078 to 0.893 mmHg) when measured at the sacrum and with a non-significant increase of 0.556 units of PPI (95% CI -0.045 to 1.156 mmHg) when measured at the greater trochanter.

No evidence was revealed for significant associations between PPI and either BMI or patient type in models conducted on healthy adults and ICU patients; or between PPI and either Braden or APACHE scores in models conducted on ICU patients only.

No substantive relationships between TR parameters and either body position, participant type, BMI category, Braden scores or APACHE II scores in any models were observed. A

substantive relationship between body temperature and TR parameters (p=0.091) was observed. This effect was of medium magnitude (partial-η^2=0.296).

Findings of all analyses conducted on the PPI and TR outcomes are summarised in Tables 3 and 4. Marginal mean scores from these models are illustrated in Figures 2 and 3 (PPI outcome) and Figures 4 and 5 (TR outcomes).

[insert tables 3 and 4 here]

[insert figures 2 to 5 here]

Discussion

The descriptive summary of baseline characteristics indicates that participants in the three study groups were well matched on most measured demographic and health-related variables; although a large age-related disparity was recorded, with the healthy adult volunteers being about 25 years younger than ICU patients. Such a disparity on a single attribute may be expected on statistical grounds alone from the number of characteristics considered and the size of the sample; however, the necessity to control for age uses up a valuable degree of freedom, which are limited in a small-scale pilot study, leading to further reduced power. Results from the pilot analysis suggest very strongly that disparities in the mean ages of participants across participant types will have a substantial effect on the outcome of the analysis. The disparity revealed in the pilot analysis suggests the possibility of further disparities across participant types in a subsequent full-scale study, which, without the ability to randomise participants, must be accounted for statistically.

As a pilot, this analysis was not powered to detect significant effects, and the low sample size precluded the use of certain statistical procedures. Furthermore, the sample size imposes limitations on the extent of statistical control that can be applied to the models in a pilot analysis. Nonetheless, certain statistically significant effects, and effects which did not reach statistical significance but indicate a relationship of substantive importance, were observed. For example, the controlling variable age in a model of the effect of participant type on PPI (Model 1); and the covariates of Braden score and temperature, in models of tissue reperfusion data (Models 7 and 9); were all found to have effects of at least moderate magnitude (c > 0.25). The within-subjects variable of body position also has a substantive effect on PPI and TR outcomes, as shown by partial-η^2 scores of 0.25 or above in several models.

Although as a pilot study, the study was not powered to reject or accept the hypotheses set out in the methods section, results suggest that a substantive relationship between acuity and certain outcomes (H$_1$) may exist; for example the relationship between PPI and acuity as illustrated in Figure 2. The picture with relation to the TR outcomes is less clear; while most analyses indicate a monotonic change in TR with acuity, the direction of the change seems to depend on the location of the measurements on the body. However, in both cases, sampling artefacts cannot be ruled out in sample sizes appropriate for pilot analyses.

The analysis suggests that participant type explains rather more variance than BMI category in the PPI outcome measure. This is supported by Bergstrand[43] who also found that age impacted PPI rather than BMI. Further work is required to determine if these initial effects are borne out in studies with larger samples in the intensive care environment.

Some of the models of TR outcome data showed unexpected results, in which values obtained from ICU patients in the overweight category were more extreme than those obtained from ICU patients in either normal weight or obese categories. These may be sampling artefacts. Such artefacts may disappear or be of lower prominence in a larger study in which it is possible to control for a greater number of confounding variables. Also as critically ill patients are unable to respond to changes in pressure, pressure relief with an individualised turning regime may be more appropriate for these patient.[19,20] In every-day clinical practice in the ICU registered nurses routinely conduct PI risk assessment and implement evidence-based, cost-effective strategies for PI prevention. It is essential for registered nurses to be cognisant that while all ICU patients may be considered high risk for PI development,[10] some patients are more vulnerable and should be considered very high risk. A larger, adequately powered follow on study, would assist in determining this issue.

Whilst the collection of TR readings was uniformly successful, a low success rate in the collection of IP measurements from ICU patients suggests that this outcome may not necessarily be appropriate for a full-scale study, unless specified as a secondary outcome. For the pilot analysis, the loss of PPI data from the majority of ICU patients unavoidably leads to a greater degree of uncertainty in parameter estimates and assessment of significance levels with relation to this outcome.

In this pilot investigation, outcome measures were selected on the basis of clinical importance and absence of excessive collinearity. The tissue reperfusion peak time, settled time constant and normalised hyperaemic area measures selected for analysis in the current study are

proposed as suitably broad and mutually uncorrelated measures to be carried forward to a full-scale analysis. Analysis techniques utilised in this study were selected in part due to their applicability to a follow-up full-scale study. The mixed ANOVA, or split plot model, described by Montgomery[35] may be considered the prototype for a multilevel analysis with body positions as the lowest level of the analysis, which may be appropriate for a large scale study. The sample size of the pilot study is probably inadequate to justify the use of this approach in the current investigation. However, the estimates of clinical effect magnitude and data variability of key outcome measures obtained from the pilot study should facilitate an accurate sample size calculation of a subsequent large-scale study.

Finally, this pilot study identified certain feasibility issues in the conduct of pressure mapping and tissue reperfusion recording in the ICU setting. Using the XSensor pressure mat in the ICU proved difficult, as the mat was not able to be rolled underneath participants. Logistically this required transfer of the participant using a pat slide from their current bed to the foam mattress bed with the pressure mapping system in place; and transferral of the patient back to original bed after data collection. Extra clinical and support staff were required for this procedure. Data collection itself at times proved challenging, with significant co-ordination and negotiation required between medical assessments, procedures, unexpected changes in the participants condition and clinical staff availability although these issues are not uncommon in the intensive care environment.

Conclusion and recommendations

Peak pressure index and tissue reperfusion are an under-reported phenomena in the critically ill patient population and literature. This pilot analysis has nonetheless determined several associations of importance, with substantive differences in outcomes observed between low- and high-acuity ICU patients; and between ICU patients and healthy volunteers. Further work is recommended on a larger scale in the critically ill patient population using 'real time' periods of load for TR measures to provide indication of optimum repositioning time for these vulnerable patients.

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Table 1 Participant characteristics at baseline by participant type

Selected characteristics	All participants (n=33)	Healthy adults (n=9)	Low acuity critically ill patients (n=13)	High acuity critically ill patients (n=11)
Age in years, mean (Standard deviation (SD))	50.3(18.3)	31.9 (11.6)	57.2 (17.6)	57.1 (12.8)
Male participants, frequency (%)	19 (57.6%)	5 (55.6%)	6 (46.2%)	8 (72.7%)
Race, (White) frequency (%)	30 (90.9%)	9 (100.0%)	12 (92.3%)	9 (81.2%)
Skin tone (white), frequency (%)	27 (81.8%)	9 (100.0%)	10 (76.9%)	8 (72.7%)
Height (cm), mean (SD)	170.6 (8.8)	171.3 (8.7)	169.2 (10.8)	171.8 (6.0)
Weight (kg), mean (SD)	79.3 (19.0)	77.3 (17.2)	77.0 (22.6)	84.4 (15.7)
BMI (kg/m^2), mean (SD)	26.9 (5.2)	25.9 (5.3)	26.4 (5.5)	28.6 (4.9)
Waist$^\beta$ (cm), mean (SD)	98.0 (15.7)	91.2 (14.5)	99.1 (16.0)	103.0 (15.9)
ICU length of stay (days)[†], median(IQR)	14.5 (8.0, 20.5)	-	14.5 (9.0,18.8)	13.0 (7.85,27.8)
Smoker, frequency (%)	11 (33.3%)	0 (0.0%)	7 (53.8%)	4 (36.4%)
Reported co-morbidities$^\alpha$				
None	11 (33.3%)	9 (100.0%)	1 (7.7%)	1 (9.1%)
Hypertension	10 (30.3%)	0 (0.0%)	6 (46.2%)	4(36.4%)
NIDDM	3 (9.1%)	0 (0.0%)	1 (7.7%)	2 (18.2%)
Other	21 (63.6%)	0 (0.0%)	11 (84.6%)	10 (90.1%)
APACHE II risk of death percentage, mean (SD)	43.4 (26.1)	-	44.9 (26.3)	41.4 (27.1)
APACHE III risk of death percentage, mean (SD)	40.0 (29.0)	-	44.0(28.1)	33.7 (30.7)
Stroke associated pneumonia (SAP) risk of death percentage, mean (SD)	40.9 (20.5)	-	40.0 (19.1)	42.1 (23.5)
Braden score on admission, median(IQR)	13.0 (11.0,23.0)	-	11.5 (9.8,13.3)	13.0 (10.0,14.3)
SOFA score, mean (SD)	5.0 (3.2)	-	2.9 (1.8)	8.0 (1.9)
Inotrope administered, frequency (%)	1 (3.0%)	-	0 (0.0%)	1 (9.1%)
Vasodilator administered, frequency (%)	4 (12.1%)	-	4 (30.7%)	0 (0.0%)
Received vasopressors, frequency (%)	0 (0.0%)	-	0 (0.0%)	0 (0.0%)

α - more than one co-morbidity could be reported;

β - waist had one missing case;

Table 2: Summary of PPI and TR measurements at sacrum and greater trochanter; by patient type

| PPI measurements	All participants	Healthy adults	Low acuity critically ill patients	High acuity critically ill patients
PPI at sacrum (mmHg), mean(SD)	57.9 (14.9) (n=15)	52.1 (10.4) (n=9)	65.8 (19.2) (n=5)	70.5 (n=1)
PPI at greater trochanter (mmHg), mean (SD)	65.7 (20.4) (n=15)	57.1 (17.7) (n=9)	74.9 (17.9) (n=5)	96.9 (n=1)
TR PT at sacrum, mean(SD)	2.12 (3.11) (n=25)	1.47 (0.91) (n=9)	1.82 (1.77) (n=10)	3.08 (5.12) (n=6)
TR STC at sacrum, mean(SD)	26.9 (50.2) (n=25)	10.1 (6.15) (n=9)	31.6 (58.6) (n=10)	38.5 (63.8) (n=6)
TR NHA at sacrum, mean(SD)	5.29 (10.6) (n=25)	4.01 (2.46) (n=9)	3.78 (4.09) (n=10)	7.16 (15.7) (n=6)
TR PT at greater trochanter, mean (SD)	1.76 (1.32) (n=24)	2.02 (1.01) (n=8)	1.95 (1.78) (n=11)	1.24 (0.70) (n=5)
TR STC at greater trochanter, mean(SD)	14.3 (13.0) (n=24)	14.5 (6.73) (n=8)	17.1 (18.9) (n=11)	10.2 (7.09) (n=5)
TR NHA at greater trochanter, mean(SD)	3.85 (4.32) (n=24)	5.07 (3.10) (n=8)	5.40 (5.91) (n=11)	1.97 (2.60) (n=5)

Table 3: Summary of ANOVAs conducted on PPI data

Model	Variable	df	F	p	η^2
1 (n=14)	Body position	1	0.126	0.729	0.011
	Patient type	2	2.97	0.093	0.351
	Body position $\times$ patient type	2	0.338	0.721	0.058
	Age	1	10.4	0.008*	0.486
	Error (position)	11	-	-	-
2 (n=14)	Body position	1	1.66	0.221	0.122
	BMI category	2	0.404	0.676	0.063
	Body position $\times$ BMI category	2	0.573	0.579	0.087
	Error (position)	12	-	-	-
3** (n=7)	Body position	1	0.107	0.760	0.026
	Braden score	1	0.327	0.598	0.076
	Body position $\times$ Braden score	1	0.336	0.593	0.077
	Error (position)	4	-	-	-
4** (n=7)	Body position	1	3.62	0.130	0.475
	APACHE II score	1	0.964	0.382	0.194
	Body position $\times$ APACHE II score	1	2.04	0.226	0.338
	Error (position)	4	-	-	-

*significant effect at 5% significance level
** analysis conducted on ICU patients only

Table 4: Summary of doubly multivariate ANOVAs conducted on TR data

| Model	Variable	Wilk's Λ	df	F	p	η^2
5 (n=22)	Body position	0.928	3,22	0.565	0.644	0.072
	Patient type	0.821	6,44	0.760	0.605	0.094
	Body position × patient type	0.756	6,44	1.01	0.378	0.130
	Age	0.926	3,22	0.590	0.628	0.074
6 (n=22)	Body position	0.898	3,23	0.871	0.470	0.102
	BMI category	0.790	6,46	0.961	0.462	0.111
	Body position × BMI category	0.812	6,46	0.843	0.544	0.099
7 (n=14)	Body position	0.701	3,10	1.42	0.294	0.299
	Braden score	0.718	3,10	1.731	0.325	0.282
	Body position × Braden score	0.735	3,10	1.20	0.359	0.265
8 (n=22)	Body position	0.713	3,18	2.42	0.100	0.287
	APACHE II score	0.921	3,18	0.513	0.679	0.079
	Body position × APACHE II score	0.785	3,18	1.64	0.215	0.215
9 (n=22)	Body position	0.789	3,18	1.61	0.222	0.211
	Temperature	0.704	3,18	2.52	0.091	0.296
	Body position × temperature	0.785	3,18	1.64	0.215	0.215
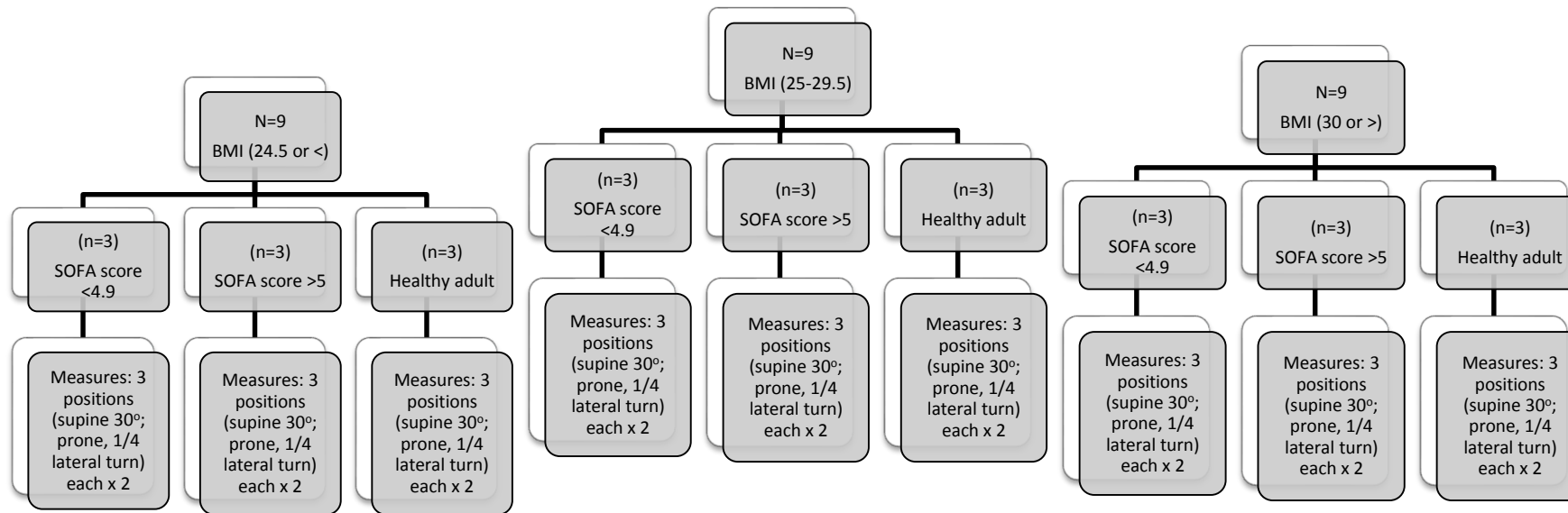
Figure 1 Study design
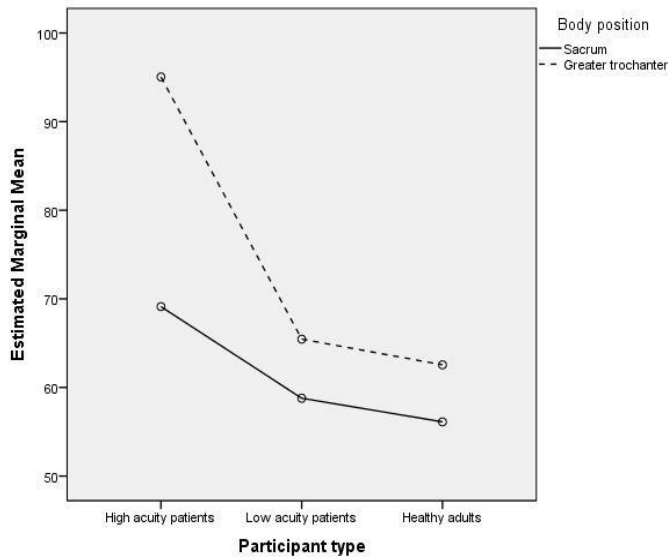
Covariates appearing in the model are evaluated at the following values: Age = 41.67
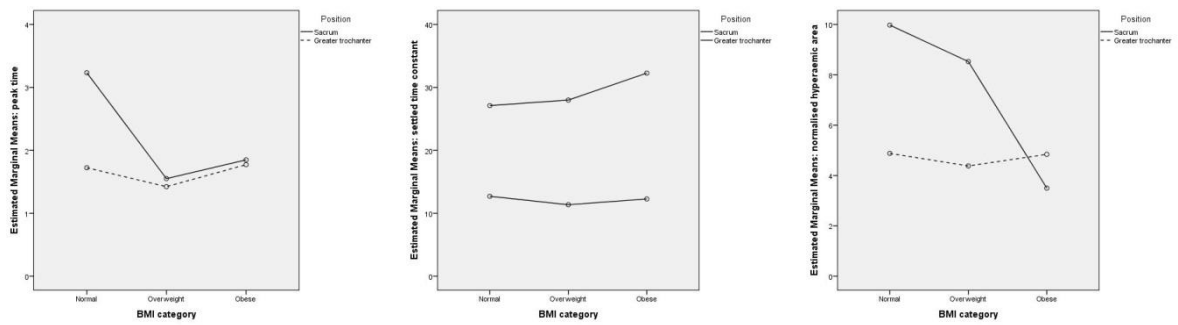
Figure 2 (Model 1): Estimated marginal PPI values at sacrum and greater trochanter for different patient types: controlling for age
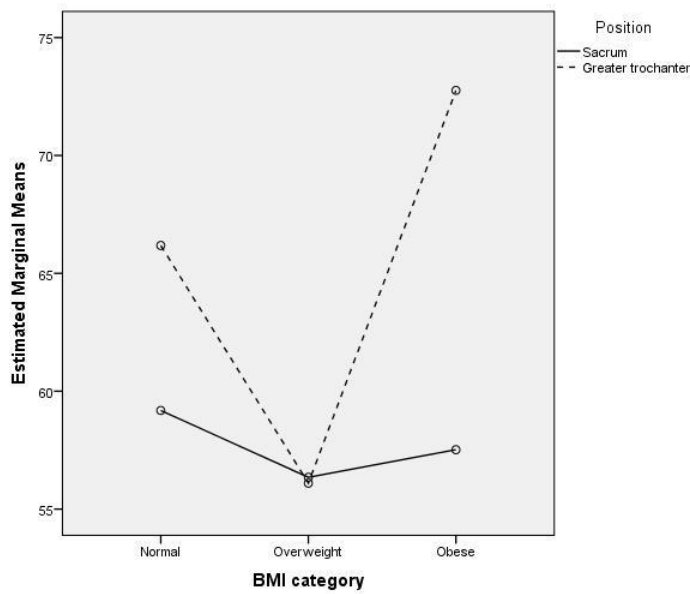
Figure 3 (Model 2): Estimated marginal PPI at sacrum and greater trochanter for different BMI categories
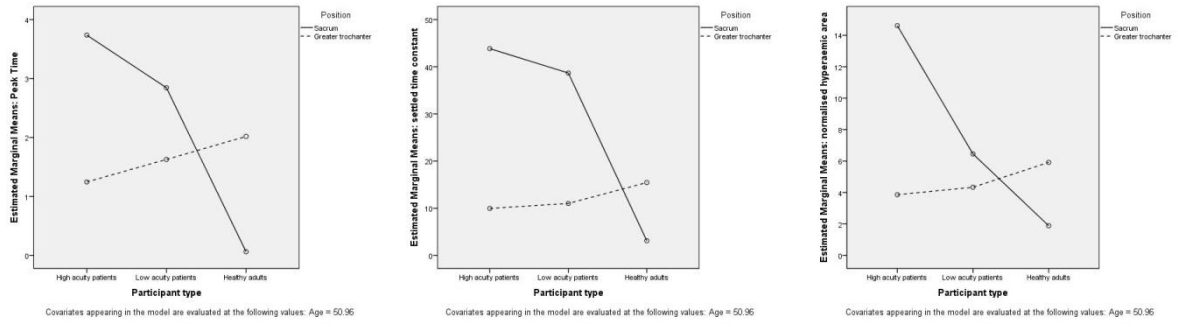
Figure 4 (Model 5): Estimated marginal TR PT, STC and NHA values at sacrum (in supine position) and greater trochanter (in ¼ lateral position) for different participant types (controlling for age)

Figure 5 (Model 6): Estimated marginal TR PT, STC and NHA values at sacrum (in supine position) and greater trochanter (in ¼ lateral position) for different BMI categories