Received: 10 January 2023 | Revised: 7 May 2023 | Accepted: 9 May 2023

DOI: 10.1111/iwj.14240

IWJ WILEY

ORIGINAL ARTICLE

The effect of prolonged 60° head of bed elevation on sacral subepidermal oedema in healthy adults: A quantitative prospective exploratory study

Sharon L. Latimer[1] | **Madeline Bone**[2] | **Rachel M. Walker**[3,4] | **Lukman Thalib**[5] | **Brigid M. Gillespie**[1,6]

[1]School of Nursing and Midwifery, Menzies Health Institute Queensland, and NHMRC Centre for Research Excellence Wiser Wounds Care, Griffith University, Southport, Queensland, Australia

[2]School of Nursing and Midwifery, Griffith University, Southport, Queensland, Australia

[3]School of Nursing and Midwifery, Menzies Health Institute Queensland, and NHMRC Centre for Research Excellence Wiser Wounds Care, Griffith University, Nathan, Queensland, Australia

[4]Metro South Health, Brisbane, Queensland, Australia

[5]Department of Biostatistics, Faculty of Medicine, Istanbul Aydin University, Istanbul, Turkey

[6]Gold Coast Hospital and Health Service, Southport, Queensland, Australia

Correspondence
Sharon L. Latimer, G01 2.05B NMHRC Wiser Wounds Care, MHIQ, School of Nursing and Midwifery, Griffith University, Southport, QLD 4125, Australia.
Email: s.latimer@griffith.edu.au

Abstract

Head of bed elevation is used to manage some medical and surgical conditions however this may increase a patient's risk of sacral pressure injuries. Novel point-of-care technologies that measure subepidermal moisture can identify changes in localised subepidermal oedema and potential pressure injury risk. This prospective exploratory study investigated variations in sacral subepidermal oedema in healthy adults during 120-min of 60° head of bed elevation. Sacral subepidermal oedema was measured at 20-min intervals using the Provisio® subepidermal moisture scanner. Descriptive analysis, one-way repeated measures analysis of variance and an independent *t*-test were conducted. Slightly more male volunteers (n = 11; 55%) were recruited and the sample mean age was 39.3 years (*SD* 14.7) with an average body mass index of 25.8 (*SD* 4.3). Little variation in the mean sacral subepidermal moisture of healthy adults was observed. There was a statistically significant difference in the mean sacral subepidermal moisture measurements between males and females (Mean difference 0.18; *95%* confidence intervals: 0.02 to 0.35; $P = .03$). Healthy adults can tolerate prolonged 60° head of bed elevation without developing increased subepidermal sacral oedema. This warrants further investigation in other populations, in various positions and over different time periods.

KEYWORDS

pressure injuries, pressure injury risk, pressure ulcers, sub-epidermal Oedema, wounds

Key Messages

- prolonged head of bed elevation is used in the management of medical and surgical conditions, but it increases the risk of sacral pressure injuries due to raised tissue loading
- the effect of prolonged head of bed elevation on sacral subepidermal oedema, and subsequent pressure injury risk in any population is unknown

- overall healthy adults showed little variability in the sacral subepidermal moisture measurements however, females were observed to higher average sacral subepidermal moisture measurements compared with males following 120-min of prolonged 60° head of bed elevation

1 | INTRODUCTION

Pressure injuries (PI) are localised skin and tissue injuries that often develop over bony prominences following shear, friction or 1–6 h of unrelieved pressure when patients are laying down or sitting.[1,2] This injury initiates a microscopic inflammatory response causing increased microvascular permeability resulting in localised subepidermal oedema.[3,4] Subsequently, cell death ensues producing tissue pH deviations which can lead to further subepidermal oedema and additional cell death.[3,4] If prolonged, this microscopic damage produces the first signs of a PI including visible skin discolouration (erythema) and localised palpable heat.[5]

Hospitalised patients are at increased risk of developing PI,[1] with a 2020 meta-analysis of >680 000 patients reporting a 12.8% (95% Confidence Intervals: 11.8–13.9) prevalence and a pooled incidence rate of 5.4/10000 patient days.[6] The body locations where hospital-acquired PI frequently develop include the sacrum (37.3%), heels (29.5%) and hips (7.8%).[6] PI can cause patients considerable pain,[7] increase their hospital length of stay and result in greater clinical workloads for clinicians.[8,9] Finally, PI are expensive, with an estimated annual cost to healthcare systems of £1.4–2.1 billion in the United Kingdom,[10] USD26.8 billion in the United States[8] and AUD$9.11 billion in Australia.[11]

Several factors increase a patient's PI risk including advancing age, reduced mobility, poor nutritional status, and prolonged head of bed elevation (HOBE).[1,12,13] For hospitalised medical and surgical patients, being nursed in various degrees of HOBE between 30° and 90° is necessary to prevent complications such as pneumonia.[14] However, prolonged HOBE raises external tissue pressure or loading on the sacrum and heels, increasing the risk of PI.[1,13,15,16] While international clinical practice guidelines for PI prevention recommend a ≤30° HOBE to reduce the patient's PI risk,[1] for some patients this is not compatible with their clinical needs.[14,16,17] Interface pressure studies have been conducted on the effect of seating position[18] or repositioning[19] and PI risk in hospital patients, and HOBE in healthy volunteers.[20,21] However, data on the angle of HOBE or the length of time prolonged pressure can be tolerated is less well known.[2]

PI risk assessment is reliant on frequent and comprehensive assessments, which includes risk assessment tools (e.g. Waterlow, Braden, Norton) and visual skin assessment.[1] Although deemed 'gold standard', the reliability of visual skin assessment to detect the first visible signs of a PI such as erythema is questionable due to its subjective nature and high inter-rater variability.[22] The recent availability of objective point-of-care technologies, including ultrasound, thermography and subepidermal moisture (SEM) devices,[23] can aid in determining a patient's PI risk.[24,25] SEM devices, which include the SEM 200® and Provisio®[26] (Figure 1), are handheld, non-invasive, point-of-care scanners that measure changes in localised tissue inflammation and subepidermal oedema and provide quantitative data on potential underlying damage.[5] The device works by measuring the biocapacitance or electrical properties of the subepidermal tissue,[27] with higher values suggesting increased extracellular oedema and possible tissue damage.[28] Recognising their clinical potential, the use of SEM devices is recommended as an adjunct tool to standard PI risk assessment.[1]

There is evidence on the potential benefits of SEM devices in detecting early tissue damage.[4,23,24,29,30] Several studies have examined the relationship between PI development and SEM measurement[24] and found these devices can detect oedema changes up to eight days before erythema is visible or heat palpable.[30-33] However, limited evidence exists on the effect of prolonged HOBE and sacral subepidermal oedema in any population. Hence, the study aim is to explore sacral subepidermal oedema in healthy adults during 120-min of prolonged 60° HOBE. This exploratory research on healthy adults will; provide baseline data, inform future PI research

FIGURE 1 Provisio SEM scanner and sacral measurements. Images used with permission from Bruin Biometrics 2022.

involving hospitalised patients who require HOBE, and potentially inform guide clinical practice in relation to identifying and developing strategies to reduce PI risk.

2 | MATERIALS AND METHODS

2.1 | Design

This quantitative prospective exploratory study was underpinned by two research questions:

1. What are the variations in sacral subepidermal oedema in healthy adults during prolonged 60° HOBE?
2. What are the differences in sacral subepidermal oedema in healthy adults during prolonged 60° HOBE based on participants' sex, age, and body mass index?

The study was conducted in the clinical nursing laboratory located at a university in southeast Queensland, Australia. The airconditioned laboratory contained four electric hospital beds, ensuring a consistent supine positioning with a 60° HOBE over 120-min. Each hospital bed had a pressure relieving static foam mattress (Prema Advanced III) which are frequently used in hospitals for patients at PI risk.[34] Ethical clearance was obtained from the university human research ethics committee (GU: 2021/515) and the study was conducted in accordance with the national ethical standards.[35]

2.2 | Sampling and participants

Using convenience and snowball sampling, our target study population were 20 healthy adult volunteers who worked or studied at Griffith University or were their friends and family members. Due to the exploratory nature of the study, this sample size was deemed sufficient. During September 2021, a range of campus-wide participant recruitment strategies were implemented including the dissemination of posters, newsletter advertisements and staff emails. Healthy adults who expressed an interest in participating were given a study overview, their questions were answered, and they were informed their participation was anonymous and voluntary. Potential participants meeting the following study criteria were eligible for recruitment: aged ≥18 years, had capacity to provide informed consent, and self-reported good health. Potential participants were excluded if they had sacral skin breaks, were unable to lay supine in a 60° HOBE for 120-min, had multiple medical comorbidities (e.g., peripheral vascular disease which increased their PI risk), and pregnancy. In total, 20 healthy adult volunteers were approached, all of whom met the study criteria and agreed to participate in the study.

2.3 | Data collection

Quantitative data collection occurred over five consecutive days during October 2021. A data collection meeting schedule was arranged in advance with each recruited participant to streamline their clinical laboratory attendance. On arrival to the clinical laboratory, participants remained standing during the study consent process. Following written consent, participant self-reported demographic data were first gathered on sex, age, height and weight (used to calculate body mass index), smoking status, diabetes, peripheral vascular disease, heart disease (ischaemic heart disease, hypertension, heart failure). Participants were then immediately positioned supinely on the hospital bed with a 60° HOBE for the duration of data collection, which was a prolonged 120-min period. At 20-min intervals (T0 [baseline], T1 [20-min], T2 [40-min], T3 [60-min], T4 [80-min], T5 [100-min], T6 [120-min]), participants were briefly repositioned laterally to gather sacral SEM measurements (Figure 1), complete a sacral skin inspection and blanching test, and assess their sacral pain score (0–10). Our decision to select 20-min data collection intervals was based on previous observational work which found hospital patients repositioned themselves approximately 3.8 times per hour.[36] Coded data were entered into a study specific, password protected Excel spreadsheet.

2.4 | Instrument and training

The Provisio® SEM scanner (Figure 1) was used to gather sacral data. This pre-calibrated, non-invasive, hand-held device measures subepidermal oedema by assessing variations in the tissue electrical properties or biocapacitance.[26] Following the manufacturer's instructions, at each data collection point, the device sensor was gently pressed against the sacral skin in a pre-determined order.[26] Six individual sacral readings were taken and expressed as unitless SEM values between 1.0 and 4.5 (±0.2).[26] The device then calculated the SEM delta by measuring the difference between the highest and lowest unitless SEM value.[26] A SEM delta of ≥0.6 *may* suggest the tissue has an increased risk for PI development, while a SEM delta of <0.6 *may* suggests a lower PI risk.[26,29] Prior to data collection, a study standard operating procedure was developed to ensure standardisation of the study processes. The research team completed 3-h of Provisio® SEM scanner training with a Bruin Biometrics

Characteristic	Males	Females	Total sample
Sex n (%)	11 (55%)	9 (45%)	20 (100%)
Age (years) Mean (*SD*)	33.5 (*13.4*)	46.3 (*13.5*)	*39.3* (*14.7*)
<40 years	9 (75%)	3 (25%)	12 (60%)
≥40 years	2 (25%)	6 (75%)	8 (40%)
BMI score Mean (*SD*)	26.3 (*2.6*)	25.2 (*5.9*)	25.8 (*4.3*)
Healthy weight (18.5–24.9 kg/m^2)	3 (50%)	3 (50%)	6 (30%)
Under or overweight (<18.5; ≥25 kg/m^2)	8 (57%)	6 (43%)	14 (70%)

TABLE 1 Sample characteristics: healthy adults (n = 20).

Abbreviations: BMI, body mass index; *SD*, Standard deviation.

representative. The content included use and function of the scanner, patient and skin preparation and safety, operator consistency, documenting the measurements, device cleaning and storage, and troubleshooting.[26]

2.5 | Data analysis

Data were entered into the Statistical Package for Social Sciences (version 28.0)[37] then cleaned and checked for accuracy, with no missing data evident. The study dependent variable, sacral SEM delta, was measured as a continuous variable. The independent variables of age and body mass index were continuous measurements, with sex (male/female) categorical. Box and whisker plots were used to present a summary of the sacral SEM delta over 120-min. Descriptive statistics were computed to describe the study sample. Depending on the data distribution, continuous level data were reported as mean (standard deviation) or median (interquartile range). Categorical data were presented as frequencies, as either absolute (numbers) or proportions (%). Prior to inferential analysis, the continuous variables of age and body mass index were recoded (age: younger [<40 years]/older [≥40 years], and body mass index: healthy [18.5–24.9 kg/m^2]/under or overweight [<18.5 and ≥25 kg/m^2]). The assumptions of independence, normality, homogeneity of variance and sphericity were checked.[38] The following inferential analyses were undertaken, with a *P*-value <.05 considered statistically significant.

1. One-way repeated measures analysis of variance (ANOVA) test *within participants* to measure variations in mean sacral SEM delta of healthy adults during 120-min of prolonged 60° HOBE
2. Independent *t*-test *between participants* to compare mean sacral SEM delta of healthy adults at 20-min intervals during prolonged 60° HOBE based on participants' sex, age, and body mass index

3 | RESULTS

The sample of 20 healthy adults comprised of slightly more males (n = 11; 55%) (Table 1). Participant's age ranged from 24 to 67 years (Mean 39.3 years; *SD* 14.7) with the female participants being on average 15 years older than the males. Participants described themselves as non-smokers with no self-reported diabetes, peripheral vascular disease or heart disease. The average body mass index score was 25.8 (*SD* 4.3, *95% CI*: 23.8 to 27.9) with 70% (n = 14) of the sample classified as underweight or overweight. One participant reported mild sacral discomfort (2–3/10 pain score) at the 100-min (T5) and 120-min (T6). No participants developed sacral erythema.

In total, 840 individual unitless sacral SEM values were taken resulting in 140 sacral SEM delta readings. At baseline (T0), the individual unitless sacral SEM vales ranged from 1.6–2.9 with a mean of 2.3 (*SD* = 0.28, *95% CI*: 2.2 to 2.5), while the SEM delta readings ranged between 0.2–0.6 with a mean of 0.4 (*SD* = 0.13, *95% CI*: 0.37 to 0.49). This mean sacral SEM delta reading was below the <0.6 cut-off which may suggest our healthy adults had a lower PI risk prior to commencing the prolonged 60° HOBE.[26,29] Worth noting, 5 (25.0%) participants commenced the study with a baseline (T0) sacral SEM delta of 0.6 which is the minimum threshold value (≥0.6) for possible increased PI risk.[26,29]

Table 2 shows the differences in participant's sacral SEM delta following 120-min of prolonged 60° HOBE. Ten participants, of which 70% were male, experienced a negative change in their sacral SEM delta (−0.1 to −0.3) from baseline (T0) to 120-min (T6), meaning their localised oedema reduced following prolonged 60° HOBE. Two male participants recorded no difference in their sacral SEM delta. The remaining eight participants, of which 75% were female, had a positive change in their sacral SEM delta (0.1 to 0.2), meaning their localised oedema increased following prolonged 60° HOBE. At 120-min (T6) of prolonged 60° HOBE, 3 (17.7%)

TABLE 2 Difference in sacral SEM delta from baseline (T0) to 120-min (T6) (n = 20).

Participant	Sex	Age (years)	BMI (score)	SEM delta baseline (T0)	SEM delta 120-min (T6)	SEM delta difference (T6–T0)
1	Female	51	21.6	0.3	0.2	−0.1
2	Female	57	26.7	0.3	0.4	0.1
3	Male	27	23.3	0.3	0.2	−0.1
4	Male	27	26.0	0.4	0.3	−0.1
5	Male	30	26.8	0.4	0.6	0.2
6	Female	58	31.2	0.6	0.5	−0.1
7	Female	52	19.1	0.4	0.5	0.1
8	Female	51	26.3	0.4	0.5	0.1
9	Female	29	20.3	0.5	0.6	0.1
10	Female	25	29.4	0.6	0.7	0.1
11	Male	24	22.0	0.3	0.2	−0.1
12	Male	26	25.6	0.4	0.3	−0.1
13	Male	29	29.0	0.3	0.3	0.0
14	Male	24	28.3	0.5	0.2	−0.3
15	Female	33	17.5	0.6	0.8	0.2
16	Male	50	25.8	0.4	0.3	−0.1
17	Male	27	29.2	0.2	0.2	0.0
18	Male	37	23.2	0.6	0.3	−0.3
19	Female	61	35.0	0.5	0.4	−0.1
20	Male	67	30.0	0.6	0.7	0.1

Abbreviations: BMI, body mass index; SEM, subepidermal moisture.

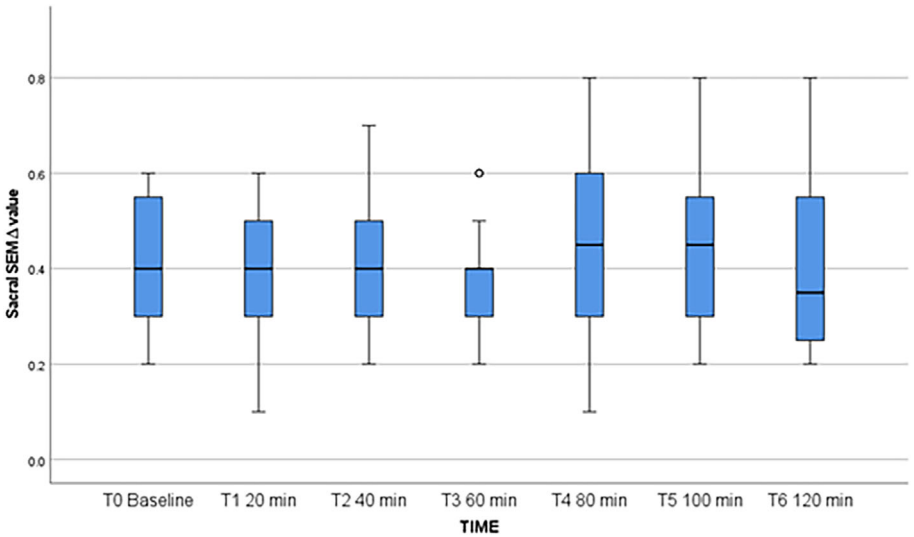

FIGURE 2 Healthy adults sacral SEM delta during 120-min of prolonged 60° HOBE.

participants recorded sacral SEM delta readings ≥0.6 (0.7–0.8) suggesting prolonged sacral loading may have increased their PI risk.[26,29]

The summary data (min, max, median, IQR 25%:75%) of the sacral SEM delta of healthy adults are presented in box and whisker plots (Figure 2). The highest median sacral SEM delta was recorded at 80-min (T4) with the lowest noted at 120-min (T6).

A one-way repeated measures ANOVA comparing the mean sacral SEM delta at baseline (T0), 60-min (T3) and 120-min (T6) was not statistically significant (Wilks' Lambda = .79, F (2, 18) = 2.37, P = .122)

TABLE 3 Independent-samples *t*-test: mean sacral SEM delta during prolonged 60° HOBE for males and females (n = 20).

Timing of SEM measurement	Males (n = 11)	Females (n = 9)	95% CI[a] of difference				
	M (*SD*)	M (*SD*)	*t*	Mean difference	Lower	Upper	*P* value (2-tailed)
Baseline (T0)	0.40 (*0.13*)	0.47 (*0.12*)	1.19	0.07	−0.05	0.18	.25
20-min (T1)	0.35 (*0.13*)	0.46 (*0.16*)	1.57	0.10	−0.03	0.24	.13
40-min (T2)	0.43 (*0.11*)	0.42 (*0.16*)	−.082	−0.01	−0.13	0.12	.94
60-min (T3)	0.36 (*0.07*)	0.40 (*0.14*)	0.71	0.04	−0.07	0.15	.49
80-min (T4)	0.39 (*0.18*)	0.52 (*0.19*)	1.57	0.13	−0.04	0.31	.13
100-min (T5)	0.41 (*0.11*)	0.49 (*0.20*)	1.11	0.08	−0.07	0.23	.28
120-min (T6)	0.33 (*0.17*)	0.51 (*0.18*)	2.38	0.18	0.02	0.35	.03

[a]CI, confidence intervals.

suggesting there was no change in localised subepidermal oedema during prolonged 60° HOBE.[38] The Mauchly's test of sphericity showed this assumption was not violated, ($\chi^2[2] = 4.1$, $P = .129$). The F statistic (2.37) was not statistically significant ($P = .122$) meeting the assumption that sample variance was approximately equal.[38] The multivariate partial eta squared was .20, suggesting a small effect size.[38]

Independent-samples *t*-test at each 20-min interval compared mean sacral SEM delta and participants' sex, age, and body mass index during prolonged 60° HOBE. There were no statistically significant differences in the mean sacral SEM delta measurements based on participants' age and body mass index. Sex was only statistically significant at 120-min (T6) (Mean difference between males and females 0.18; *95% CI*: 0.02 to 0.35; t (18) = 2.38, $P = .03$, two-tailed) (Table 3). The effect size of .17 (*95% CI*: 0.11 to 2.0) between the two group means was small.[39] Using G*Power,[40] a post hoc power analysis was conducted with a p value of 0.05 (two-tailed) and Cohen's d effect size of .17, indicated the sample size was not powered (0.07%) to detect any differences.

4 | DISCUSSION

4.1 | SEM research in healthy adults

This study examined variations in sacral SEM of healthy adults during 120-min of prolonged 60° HOBE. Our Australian findings contribute to the growing body of evidence in healthy adults undertaken in Ireland,[31,41] England[42] and the United States.[43,44] Understanding how healthy human tissue responds to prolonged pressure loading such as HOBE provides important baseline data for research comparison in patients at risk or with a PI.[2,31,42,44] For example, Moda Vitoriano Budri et al.,[31] recently used the mobility data from healthy adults to categorise the movement patterns (high [healthy] and low [unhealthy] movers) of older adults living in long-term care facilities and determine its effect on SEM delta and PI development. While an agreed definition of a healthy adult or volunteer is not provided in the associated literature, two features were evident: individuals lived in the community and had undamaged skin at the study site as determined by the visual and palpation assessment of a clinical skin expert.[31,41-44] These two considerations were applied to our recruited healthy participants increasing the comparability of our findings. While individuals of all ages living in the community might describe themselves as 'healthy adults', it is likely many have one or more chronic conditions[45] and therefore may be at risk of PI in the community or during hospitalisation.[44,46] Hence, undertaking research into healthy adults with various skin types who are placed in other HOBE positions and prolonged timeframes is needed to increase our understanding of the effect of tissue loading in other situations.

4.2 | Baseline sacral SEM measurements

In prospective observational quantitative studies, establishing participant baseline data allows researchers to assess differences across the study participants.[47] In the published literature on healthy adults, baseline SEM data is reported based on the study's data collection and analytic approach.[41,43,44] For example, we collected six SEM values measured at closely positioned skin locations across the sacrum and reported baseline sacral SEM values and the mean SEM delta. In contrast, two recent studies on healthy adults collected seven,[44] and nine[43]

absolute sacral SEM readings at a 4-, and 8-cm circumference away (centre and near-proximate) from the bony sacrum. While, Jayabal et al.,[41] gathered three baseline absolute sacral SEM units (AUs or arbitrary units) and the mean SEM was calculated. There is a burgeoning increase in clinical research into subepidermal oedema and PI risk, so care is required to ensure data equivalence exists (arbitrary SEM units versus SEM delta) when comparing and interpreting findings across multiple studies.

Prior to tissue loading of 60° HOBE, the baseline (T0) mean absolute sacral SEM value in our participants was 2.3 units. Three recent studies on healthy adults reported similar findings in the absence of tissue loading.[41,43,44] Jayabal et al.,[41] found variability at 27 anatomical locations that are known high risk sites for PI, whereas Gershon's[44] study of healthy adults aged >55 years reported a lack of variability in sacral SEM values. Furthermore, the baseline (T0) mean sacral SEM delta in our sample was 0.4 which suggests lower subepidermal oedema and potentially, lower PI risk.[26,29] This confirms the results of Gershon's[44] study where authors concluded the sacral tissue of healthy adult participants was not inflamed. Our findings are important because they suggest healthy adults may have a lower PI risk however, hospitalisation is known to potentially increase risk for PI for all patients regardless of health status.[30,48] For example, a 4-year study of 64 917 patients admitted to 63 Swedish acute hospitals found a 2.8% and 1.1% PI prevalence rate respectively among acute and elective hospital admissions respectively.[49] While the researchers do not report on the health and living status of the sample, they conclude PI prevention must be a priority.[49] Further robust research is needed to examine the influence of extrinsic and intrinsic factors on sacral subepidermal oedema; such information may help identify the predictors of PI risk.[41]

4.3 | Prolonged 60° HOBE and sacral SEM

Prolonged HOBE is frequently implemented for medical and surgical patients[14,50] with the associated shearing and loading forces increasing PI risk.[13,20] While our study found limited variability in healthy adults' mean sacral SEM delta following prolonged 60° HOBE, it is important to note 3 (17.7%) participants recorded SEM delta readings ≥0.6 which could increase their PI risk.[26,29] This highlights the importance of individualising care based on a person's assessment. No previous research could be located on the effect of prolonged loading and HOBE on sacral SEM measurements. However, a recent study examined the effect of sacral skin barrier cream on SEM variations in 22 healthy adults following 60-min of supine positioning.[42] The researchers applied the sacral cream post-offloading and collected SEM readings at 5-min interval for 55-min, with an elevated SEM delta recorded only at the 5-min time period in participants with partial cream coverage ($P = .04$) compared with those with full skin cream.[42] The mechanism of SEM variations is not well understood[42] with individual factors such as tissue tolerance considered a potential protective factor.[3,4] This confirms further research into the effect of other prolonged bed positions on sacral subepidermal oedema requires careful investigation to improve our understanding.

4.4 | Prolonged 60° HOBE and sex, age, and body mass index

Gefen's[2] work with invitro models, animal studies and patients undergoing a surgical procedure found PI are known to develop after 1–6 h of unrelieved tissue loading. Increasing age, malnutrition, obesity and prolonged HOBE are also recognised patient risk factors for PI.[13] Although we found a difference in the mean sacral SEM delta measurements between males and females after 120 min of prolonged 60° HOBE, this exploratory study was not powered to detect such a difference. Hence, it is difficult to know if these small differences have any clinical implications or importance. While we were unable to locate comparable research findings, however HOBE and sacral interface pressure in healthy volunteers has been examined.[20,21] Peterson et al.,[21] found compared with supine positioning, a HOBE of 45°, 60° and 75° showed statistically significant increases in sacral interface pressure in healthy adults ($P < .0001$). Another study examined the effect of sacral skin barrier cream in 22 healthy adults and reported no differences between sacral SEM delta values and participant sex, age and body mass index however, their focus was on the reliability of the measurements and not the PI prevention action of the cream.[42] SEM technology has been found to detect increased underlying skin damage that may lead to PI on average 8.2 days before they are visible on the skin.[23,31] SEM technology shows promise in the early detection of PI,[4] so a stronger evidence base is warranted. Rigorous studies are needed to describe the influence of individual factors such as sex, age and body mass index on sacral subepidermal oedema and PI risk.[24,41]

4.5 | Limitations and strengths

We acknowledge the following limitations. First, our small sample size limits the generalisability of our findings to other contexts and populations. This is an accepted limitation considering the primary aim of this exploratory study was to investigate future research possibilities in this

burgeoning research area[24] rather than detecting an effect.[38] Second, we gathered self-reported data on age, height, weight, and co-morbidities, which were not independently verified. This could be a potential source of response bias,[51] and thus needs to be taken into account when interpreting these findings. Finally, while this study focussed on healthy adults without a PI, our study contributes to recommendations for further evaluation on the variability of SEM delta in a range of human populations with different intrinsic and extrinsic risk factors such as prolonged tissue loading.[41]

5 | CONCLUSION

This study provides an initial understanding on the effect of prolonged 60° HOBE on sacral subepidermal oedema in healthy adults. We found no evidence of sacral tissue inflammation following 120 min of continual tissue loading in healthy adults. While these results suggest a difference in mean sacral SEM measurements between males and females after 120 min 60° HOBE, no differences were found based on participants' age or body mass index. HOBE is frequently used in clinical practice and is known to increase PI risk, so the early identification of sacral subepidermal inflammation may prompt clinicians to implement appropriate prevention strategies.

ACKNOWLEDGEMENTS

We wish to acknowledge the valuable contribution of our volunteers. Open access publishing facilitated by Griffith University, as part of the Wiley - Griffith University agreement via the Council of Australian University Librarians.

FUNDING INFORMATION

No external funding.

CONFLICT OF INTEREST STATEMENT

None to declare.

DATA AVAILABILITY STATEMENT

Due to the sensitive nature of the study questions, participants were assured raw data would remain confidential and would not be shared.

ORCID

Sharon L. Latimer https://orcid.org/0000-0003-2704-150X
Madeline Bone https://orcid.org/0000-0002-0683-9005
Rachel M. Walker https://orcid.org/0000-0002-6089-8225
Lukman Thalib https://orcid.org/0000-0002-1211-6495
Brigid M. Gillespie https://orcid.org/0000-0003-3186-5691

REFERENCES

1. European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance. Prevention and treatment of pressure ulcers/injuries: clinical practice guidelines. In: Haesler E, ed. *The International Guideline*. 3rd ed. London: European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance; 2019. https://internationalguideline.com/
2. Gefen A. How much time does it take to get a pressure ulcer? Integrated evidence from human, animal, and in vitro studies. *Ostomy Wound Manage*. 2008;54(10):26-28.
3. Gefen A, Soppi E. What is new in our understanding of pressure injuries: the inextricable association between sustained tissue deformations and pain and the role of the support surface. *Wound Prac Res*. 2020;28(2):58-65.
4. Gefen A. The future of pressure ulcer prevention is here: detecting and targeting inflammation early. *Europe Wound Manage J*. 2018;19(2):7-13.
5. Gefen A, Ross G. The subepidermal moisture scanner: the technology explained. *J Wound Care*. 2020;29:S10-S16.
6. Li Z, Lin F, Thalib L, Chaboyer W. Global prevalence and incidence of pressure injuries in hospitalised adult patients: a systematic review and meta-analysis. *Int J Nurs Stud*. 2020;105:103546.
7. Latimer S, Chaboyer W, Gillespie BM. Patient participation in pressure injury prevention: giving patient's a voice. *Scand J Caring Sci*. 2014;28(4):648-656.
8. Padula W, Delarmente B. The national cost of hospital-acquired pressure injuries in the United States. *Int Wound J*. 2019;16(3):634-640.
9. McEvoy N, Avsar P, Patton D, Curley G, Kearney C, Moore Z. The economic impact of pressure ulcers among patients in intensive care units. A systematic review. *J Tissue Viability*. 2020;30(2):168-177.
10. Bennett G, Dealey C, Posnett J. The cost of pressure ulcers in the UK. *Age Ageing*. 2004;33(3):230-235.
11. Nghiem S, Campbell J, Walker RM, Byrnes J, Chaboyer W. Pressure injuries in Australian public hospitals: a cost of illness study. *Int J Nurs Stud*. 2022;130:104191.
12. Gillespie BM, Walker RM, Latimer S, et al. Repositioning for pressure injury prevention in adults (review). *Cochrane Database Syst Rev*. 2020;6(6):CD009958.
13. Lustig M, Wiggermann N, Gefen A. How patient migration in bed affects the sacral soft tissue loading and thereby the risk for a hospital-acquired pressure injury. *Int Wound J*. 2020;17(3):631-640.
14. Güner CK, Kutlutürkan S. Role of head-of-bed elevation in preventing ventilator-associated pneumonia bed elevation and pneumonia. *Nurs Crit Care*. 2022;27(5):635-645.
15. Oomens CW, Bader DL, Loerakker S, Baaijens F. Pressure induced deep tissue injury explained. *Ann Biomed Eng*. 2015;43(2):297-305.
16. Latimer S, Chaboyer W, Gillespie BM. The repositioning of hospitalized patients with reduced mobility: a prospective study. *Nursing Open*. 2015;2(2):85-93.
17. McInnes E, Chaboyer W, Allen T, Murray E, Webber L. Acute care patient mobility patterns and documented pressure injury prevention-an observational study and survey. *Wound Prac Res*. 2013;21(3):116-125.
18. McInnes E, Jammali-Blasi A, Bell-Syer SE, Dumville JC, Middleton V, Cullum N. Support surfaces for pressure ulcer prevention. *Cochrane Database Syst Rev*. 2015;(9):1-122.
19. Peterson M, Gravenstein N, Schwab W, van Oostrom J, Caruso L. Patient repositioning and pressure ulcer risk-

monitoring interface pressures of at-risk patients. *J Rehabil Res Dev*. 2013;50(4):477-488.
20. Lippoldt J, Pernicka E, Staudinger T. Interface pressure at different degrees of backrest elevation with various types of pressure-redistribution surfaces. *Am J Crit Care*. 2014;23(2):119-126.
21. Peterson M, Schwab W, McCutcheon K, van Oostrom JH, Gravenstein N, Caruso L. Effects of elevating the head of bed on interface pressure in volunteers. *Crit Care Med*. 2008;36(11):3038-3042.
22. Edsberg LE, Black JM, Goldberg M, McNichol L, Moore L, Sieggreen M. Revised national pressure ulcer advisory panel pressure injury staging system: revised pressure injury staging system. *J Wound Ostomy Continence Nurs*. 2016;43(6):585-597.
23. Scafide KN, Narayan MC, Arundel L. Bedside technologies to enhance the early detection of pressure injuries: a systematic review. *J Wound Ostomy Cont Nurs*. 2020;47(2):128-136.
24. Chaboyer W, Coyer F, Harbeck E, et al. Oedema as a predictor of the incidence of new pressure injuries in adults in any care setting: a systematic review and meta-analysis. *Int J Nurs Stud*. 2022;128:104189.
25. Moore Z, McEvoy N, Avsar P, et al. Measuring subepidermal moisture to detect early pressure ulcer development: a systematic review. *J Wound Care*. 2022;31(8):634-647.
26. Bruin Biometrics LLC. Provisio SEM scanner user guide. n.d. https://sem-scanner.com/wp-content/uploads/2021/09/OTH-SEM-IFU-OUS-0359-SEM-Scanner-250-IFU-Manual-English-OUS-Rev-H.pdf Accessed March 9, 2022.
27. Peko Cohen L, Gefen A. Phantom testing of the sensitivity and precision of a sub-epidermal moisture scanner. *Int Wound J*. 2019;16(4):979-988.
28. Martins de Oliveira AL, Moore Z, O'Connor T, Patton D. Accuracy of ultrasound, thermography and subepidermal moisture in predicting pressure ulcers: a systematic review. *J Wound Care*. 2017;26(5):199-215.
29. Bryant R, Moore Z, Iyer V. Clinical profile of the SEM scanner—modernizing pressure injury care pathways using sub-epidermal moisture (SEM) scanning. *Expert Rev Med Devices*. 2021;18(9):833-847.
30. Martins de Oliveira AL, O'Connor T, Patton D, Strapp H, Moore Z. Sub-epidermal moisture versus traditional and visual skin assessments to assess pressure ulcer risk in surgery patients. *J Wound Care*. 2022;31(3):254-264.
31. Moda Vitoriano Budri A, Moore Z, Patton D, et al. Impaired mobility and pressure ulcer development in older adults: excess movement and too little movement-two sides of the one coin? *J Clin Nurs*. 2020;29(15–16):2927-2944.
32. *Modernising PI/PU care pathways with sub-epidermal mositure assessment* [Webinar]: BBI. 2020.
33. Gefen A, Gershon S. An observational, prospective cohort pilot study to compare the use of subepidermal moisture measurements versus ultrasound and visual skin assessments for early detection of pressure injury. *Management*. 2018;64(9):12-27.
34. Shi C, Dumville JC, Cullum N, et al. Beds, overlays and mattresses for preventing and treating pressure ulcers: an overview of Cochrane reviews and network meta-analysis. *Cochrane Database Syst Rev*. 2021;8(8):Cd013761.
35. National Health and Medical Research Council, Australian Research Council, Australian Vice-Chancellors' Committee. National statement on ethical conduct in human research Canberra, Australia2007-updated 2018:1-101.
36. Chaboyer W, Mills P, Roberts S, Latimer S. Physical activity levels and torso orientations of hospitalized patients at risk of developing a pressure injury: an observational study. *Int J Nurs Pract*. 2013;21(1):11-17.
37. IBM SPSS Statistics for Windows [computer program]. Version 28.0. Armonk, NY2021.
38. Pallant J. *SPSS Survival Manual: A Step by Step Guide to Data Analysis Using SPSS*. 4th ed. Maidenhead: Allen & Unwin; 2010.
39. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. New York: Routledge; 2013.
40. Faul F, Erdfelder E, Lang A-G, Buchner A. G*power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods*. 2007;39:175-191.
41. Jayabal H, Bates-Jensen B, Abiakam NS, Worsley P, Bader D. Anatomical variability of sub-epidermal moisture and its clinical implications. *J Tissue Viability*. 2021;30(3):434-438.
42. Evans P, Cole A, Voegeli D. The impact of skin barrier cream on variation in sub-epidermal moisture readings. *Wounds UK*. 2020;16(2):29-35.
43. Gershon S, Okonkwo H. Evaluating the sensitivity, specificity and clinical utility of algorithms of spatial variation in sub-epidermal moisture (SEM) for the diagnosis of deep and early-stage pressure-induced tissue damage. *J Wound Care*. 2021;30(1):41-53.
44. Gershon S. Using subepidermal moisture level as an indicator of early pressure damage to local skin and tissue. *Adv Skin Wound Care*. 2020;33(9):469-475.
45. Australian Institute of Health and Welfare. Chronic Conditions and Multimorbidity. 2022 https://www.aihw.gov.au/reports/australias-health/chronic-conditions-and-multimorbidity Accessed 12/07/2022.
46. Maeda T, Tamai N, Minematsu T, et al. The relationship between pressure injuries and dynamic buttock pressure distributions during performance in wheelchair basketball athletes. *Wound Prac Res*. 2021;29(4):211-218.
47. Cheng AC, Kessler D, Mackinnon R, et al. Reporting guidelines for health care simulation research: extensions to the CONSORT and STROBE statements. *Adv Simul*. 2016;1(1):1-13.
48. D'Amour D, Dubois C, Tchouaket E, Clarke S, Blais R. The occurrence of adverse events potentially attributable to nursing care in medical units: cross sectional record review. *Int J Nurs Stud*. 2014;51(6):882-889.
49. Gunningberg L, Sving E, Hommel A, Ålenius C, Wiger P, Bååth C. Tracking pressure injuries as adverse events: national use of the global trigger tool over a 4-year period. *J Eval Clin Pract*. 2019;25(1):21-27.
50. Metheny N, Frantz R. Head-of-bed elevation in critically ill patients: a review. *Crit Care Nurse*. 2013;33(3):53-67.
51. Polit D, Beck C. *Essentials of Nursing Research: Appraising Evidence for Nursing Practice*. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2010.

How to cite this article: Latimer SL, Bone M, Walker RM, Thalib L, Gillespie BM. The effect of prolonged 60° head of bed elevation on sacral subepidermal oedema in healthy adults: A quantitative prospective exploratory study. *Int Wound J*. 2023;20(9):3619-3627. doi:10.1111/iwj.14240