Intensive and Critical Care Nursing (2017) 38, 1—9

Contents lists available at ScienceDirect

ScienceDirect

journal homepage: www.elsevier.com/iccn

ORIGINAL ARTICLE

Tissue interface pressure and skin integrity in critically ill, mechanically ventilated patients☆

Mary Jo Grap[a,*], Cindy L. Munro[b], Paul A. Wetzel[c], Christine M. Schubert[d], Anathea Pepperl[c], Ruth S. Burk[a], Valentina Lucas[e]

[a] *Adult Health and Nursing Systems Department of the School of Nursing, Virginia Commonwealth University, Richmond, VA, United States*
[b] *College of Nursing, University of South Florida, Tampa, FL, United States*
[c] *Biomedical Engineering Department, School of Engineering, Virginia Commonwealth University, Richmond, VA, United States*
[d] *Department of Mathematics and Statistics, Air Force Institute of Technology, Wright-Patterson Air Force Base, Dayton, OH, United States*
[e] *Department of Surgery, Virginia Commonwealth University Heath System, Richmond, VA, United States*

Accepted 27 July 2016

KEYWORDS
Critical care;
Mechanical ventilation;
Pressure ulcers;
Tissue interface pressure

Summary
Objective: To describe tissue interface pressure, time spent above critical pressure levels and the effect on skin integrity at seven anatomical locations.
Design, setting, patients: Descriptive, longitudinal study in critically ill mechanically ventilated adults, from Surgical Trauma ICU-STICU; Medical Respiratory ICU-MRICU; Neuroscience ICU-NSICU in a Mid-Atlantic urban university medical centre. Subjects were enroled in the study within 24 hours of intubation.
Measurements: Tissue interface pressure was measured continuously using the XSENSOR pressure mapping system (XSENSOR Technology Corporation, Calgary, Canada). Skin integrity was observed at all sites, twice daily, using the National Pressure Ulcer Advisory Panel staging system, for the first seven ICU days and at day 10 and 14.
Results: Of the 132 subjects, 90.9% had no observed changes in skin integrity. Maximum interface pressure was above 32 mmHg virtually 100% of the time for the sacrum, left and right trochanter. At the 45 mmHg level, the left and right trochanter had the greatest amount of time above this level (greater than 95% of the time), followed by the sacrum, left and right scapula, and the

☆ Supported by funding from NIH, R01 NR010381 (Grap PI).
* Corresponding author at: 1000 Lady Jean Ct, Midlothian, VA 23114, United States. Fax: +1 804 794 8376.
E-mail address: mjgrap@vcu.edu (M.J. Grap).

http://dx.doi.org/10.1016/j.iccn.2016.07.004

left and right heels. Similarly, at levels above 60 mmHg, the same site order applied. For those six subjects with sacral skin integrity changes, maximum pressures were greater than 32 mmHg 100% of the time. Four of the six sacral changes were associated with greater amounts of time above both 45 mmHg and 60 mmHg than the entire sample.
Conclusions: Maximum tissue interface pressure was above critical levels for the majority of the documented periods, especially in the sacrum, although few changes in skin integrity were documented. Time spent above critical levels for mean pressures were considerably less compared to maximum pressures. Maximum pressures may have reflected pressure spikes, but the large amount of time above the critical pressure levels remains substantial.

Implications for clinical practice

- Pressure ulcers develop as result of a complex, multi-factorial process.
- Although high tissue interface pressure over extended periods of time are contributory to this process, other factors also place patients are risk.
- Time spent above critical pressure levels in this sample was considerable.
- For those with changes in skin integrity, trends toward greater pressures during the period preceding changes in skin integrity, especially for the sacral and trochanter sites, were identified.

Introduction

In mechanically ventilated, critically ill patients, pressure ulcer risk is high and may result in negative patient outcomes and increased health care costs (Alderden et al., 2011; Anon, 2016; Shahin et al., 2009a). Pressure ulcers, any lesion caused by unrelieved pressure resulting in damage to the underlying tissue, are a serious complication of impaired mobility (Anon, 2016; Cox and Cwocn, 2011). Repositioning is one strategy to mitigate the effects of immobility in pressure ulcer development. Recommendations to reduce pressure ulcer (PrUl) risk place patients in backrest positions of less than 30° to reduce pressure on bony prominences that are most at risk for the development of pressure ulcers (Burk and Grap, 2012; Shahin et al., 2009b).

The magnitude and duration of pressure affects PrUl development with increasing tissue interface pressure and time contributing to tissue damage (Bennett et al., 1979; Daniel et al., 1981; Dinsdale, 1974; Kosiak, 1959). The critically ill, with their unstable physiologic status are especially at risk. In healthy individuals, an external pressure of at least 120 mmHg is required for blood flow occlusion, compared with 11—30 mmHg in geriatric hospitalised patients (Ek et al., 1987; Frantz and Xakellis, 1989). Although low levels of external pressure may increase dermal flow, this flow response in critically ill patients is not consistent (Frantz et al., 1993; Herrman et al., 1999; Xakellis et al., 1993), resulting in an impaired and delayed tissue recovery compared with healthy individuals (Aoi et al., 2009; Bader, 1990). Early studies found that a primary cause of PrUls is ischaemia produced by external pressures greater than capillary pressure (12—32 mmHg) and a constant pressure of 70 mmHg applied for two hours produced ischaemic changes (Dinsdale, 1974; Kosiak, 1959). Subsequent studies have supported pressure as a primary culprit in PU development (Bennett et al., 1979; Kottner et al., 2015; Lahmann and Kottner, 2011).

Although use of lower backrest elevation are recommended for pressure ulcer prevention, for critically ill patients who are mechanically ventilated, higher backrest positions are recommended to reduce the risk of ventilator associated pneumonia (VAP) (Guidelines for Prevention of Nosocomial Pneumonia, 1997; Tablan et al., 2004). Since pressure is a primary mechanism in the formation of PrUls, higher backrest elevation positions used for VAP prevention may have deleterious effects on skin integrity (Linder-Ganz et al., 2008). Recently in the parent study for the present, secondary analysis, we found in critically ill, mechanically ventilated patients, that overall, mean tissue interface pressures were less in the scapula and heel than in trochanter and sacral area (Grap et al., 2016). We also found that interface pressure decreased as backrest elevation increased in the scapula, but not in the sacrum, heels or trochanter (Grap et al., 2016). However, there are few data that fully describe tissue interface pressure over time and the effect on skin integrity in critically ill, mechanically ventilated patients (Lippoldt et al., 2014; Sprigle and Sonenblum, 2011; van Nieuwenhoven et al., 2006). Therefore, the purpose of this secondary, descriptive, longitudinal study in critically ill mechanically ventilated adults, was to describe tissue interface pressure, time spent above critical pressure levels and the effect on skin integrity at seven anatomical locations with high risk for development of pressure ulcers.

Methods

Setting and sample

The parent study, from which this analysis is derived, was a descriptive, longitudinal study of skin integrity of 150

intubated and mechanically ventilated adult patients from a medical respiratory ICU (MRICU), surgical trauma ICU (STICU) or neuroscience ICU (NSICU) in an academic medical centre (Grap et al., 2016).

Subjects were recruited from three critical care units: Surgical Trauma ICU-STICU; Medical Respiratory ICU-MRICU; Neuroscience ICU-NSICU in a 933-bed tertiary care, Mid-Atlantic urban university medical centre, were intubated and mechanically ventilated with an expectation of at least 24 hours of mechanical ventilation and enroled in the study within 24 hours of intubation. Subjects with existing pressure ulcers were also recruited since the purpose was to describe the effect on skin integrity and would include any change in already compromised skin.

Power analysis was calculated for the parent study for detectable associations of our proposed sample size of 150 to detect correlation levels between 0.23 and 0.26 with at least 80% power for tissue interface pressure, including considerations for repeated measures.

Key variables and their measurement

Tissue interface pressure

Tissue interface pressure between the subject and support surface was measured using the XSENSOR pressure mapping system (XSENSOR Technology Corporation, Calgary, Canada), one sensor pad containing a matrix of individual capacitance based pressure sensors. The pad is thin (1 mm thick), extremely flexible and was made in full bed size (sensing area: 24'' by 72'') for this project. Pads were placed beneath a hospital sheet reducing the interaction of nursing staff with the pressure sensing system and reduced risk of pad damage. We focused on seven common pressure ulcer sites (left and right scapula, left and right trochanter, sacrum, and left and right heel) using these high pressure areas as recorded from the XSENSOR programme. Based on the patient's position (supine, left or right side lying), pressure measurements were documented when the site was in contact with the XSENSOR pad.

Although there is a clear relationship among tissue interface pressure magnitude, duration and tissue damage, a critical magnitude above which ischemia occurs has not been fully established for all types of patients. Therefore, we investigated several maximum interface pressure levels based on values previously tested by others (Behrendt et al., 2014; Defloor, 1999; Gunningberg and Carli, 2014; Landis, 1930; Lippoldt et al., 2014; Peterson et al., 2008; Sakai et al., 2009). We identified three maximum pressure levels, greater than or equal to 32, 45 and 60 mmHg. Not only pressure magnitude, but pressure duration is important in PrUl risk, therefore, percent time spent above these critical pressure levels was also determined.

Skin integrity

Skin integrity was evaluated with direct observation of the skin by study personnel using the National Pressure Ulcer Advisory Panel staging system. (Cuddigan and Frantz, 1998; Black et al., 2007) Training of all evaluators was conducted by our Wound Care Programme Coordinator (VL). Skin integrity observations were documented for the seven pressure ulcer sites (left and right scapula, left and right trochanter, sacrum, and left and right heel).

Subject demographics and other covariates

Support surface

The type of support surfaces may affect the development of PrUls. However, since 2008, the Hill-Rom Total Care Connect bed (TotalCare Duo2 system; Hill-Rom; Batesville, Indiana) is the primary bed type used in the target units. It is a low air loss surface, developed, to manage the microclimate of the skin and has multi-zoned, air-filled bladders in order to redistribute pressure based on patient weight and position.

Subject demographics

PrUl risk may also be affected by illness severity, patient weight and other factors. Severity of illness was documented on study enrolment using the APACHE III (Knaus et al., 1981, 1991). Subject age, gender, ICU type, BMI and presence of sacral pressure ulcer prophylaxis (Mepilex®, Molnlycke Health Care, Norcross, GA) were also collected at study enrolment. BMI was calculated using the morning weight obtained per unit standard and height based on the subject's legally authorised representative's statement of the subject's height (Determann et al., 2007).

A study team member documented the Braden scale, used for daily assessments of PrUl risk, at the time of study enrolment.

Procedures

The university's Institutional Review Board (IRB) approved the study. Patients (or their legally authorised representative) who met study criteria were approached for consent. Subjects were enroled within 24 hours of intubation so that baseline skin assessments were obtained. Descriptive subject data (ICU admission, APACHE III) were collected from the medical records for the 24 hours prior to study enrolment. Tissue interface pressure (XSENSOR) was measured over a continuous 72-hour period and occurred only while the subject was in bed and mechanically ventilated. In an evidence-and consensus-based guideline Stechmiller et al. (2008), found that pressure ulcers develop within the first 72 hours of an ICU admission. Skin observations were documented twice daily (morning and evening) over the seven sites for the first seven ICU days or until ICU discharge. Limited data are available that describe the complete time frame for pressure ulcer manifestation, especially for deep tissue injury (Gefen, 2008). Therefore, to document all changes in skin integrity over time, skin observations were also conducted at day 10 and 14 if subjects remained hospitalised.

Data analysis

Tissue interface pressures were collected over a period of up to 72 hours at a rate of twice per second. Due to patient positioning, equipment concerns and other factors, not every patient had available, complete data across all seven anatomical sites. When such concerns occurred, data

Table 1 Subject demographics.

	N	Mean	SD	Median	Min	Max
Age	132	55.94	17.25	58.00	18.00	90.00
BMI	132	28.08	6.57	26.83	12.05	44.93
Apache III	132	77.36	27.34	75.00	20.00	154.00
Braden score	131	12.72	1.72	13.00	9.00	19.00
ICU length of stay	129	13.34	12.94	8.67	0.00	76.02
Hospital length of stay	109	18.93	16.43	13.54	0.63	78.15
	N	%				
Gender						
Male	76	57.58				
Female	56	42.42				
Race						
Black/AA	65	49.24				
Unknown or not report	2	1.52				
White	65	49.24				
Ethnicity						
Hispanic	2	1.52				
Non-Hispanic	130	98.48				
ICU[a]						
MRICU	55	41.66				
NSICU	34	25.76				
STICU	43	32.58				
BMI group						
bmi < 19	7	5.30				
19 ≤ bmi < 25	41	31.06				
25 ≤ bmi < 30	36	27.26				
bmi > 30	48	36.36				
Sacral Mepilex present[b]	128	96.97				

[a] ICU—MRICU = Medical Respiratory ICU, NSICU = Neuroscience ICU, STICU = Surgical Trauma ICU.
[b] Sacrel Mepilex was present on at last one study day in these patients.

were flagged and computations adjusted so as not to bias the results. Also, due to patient care needs and procedures, not all subjects had available skin integrity data twice daily. Descriptive statistics were used to describe skin interface pressure and the amount of time patients spend at or above specific pressure thresholds of 32, 45 and 60 mmHg. Graphical representations were used to examine each of the seven anatomical locations and to calculate both maximum and mean pressure data at each of the seven major PrUl sites. The percent of time patients' experienced maximum or mean pressure above each of the three thresholds of 32, 45 and 60 mmHg was then determined. Due to the relatively small number of patients with pressure ulcers, data for those with or developing pressure ulcers were examined in a case study approach. The amount of time above the three pressure thresholds and the pressure experienced by those patients developing pressure ulcers were compared to that of the entire population as a whole. Maximum and average pressure experienced by the patient prior to the change in skin integrity was examined. All analyses were conducted using SAS v9.2.

Results

One hundred and fifty subjects were enroled in the study, 132 subjects had evaluations of skin integrity over the study period and segments of usable tissue interface pressure data over at least some portion of the observation period and were included in the analysis. The majority of subjects were non-Hispanic, male ($n = 76$, 58%) overweight (BMI > 25 kg/m^2) and in the MRICU, but were evenly divided between race (Table 1). Median severity of illness score (APACHE III) on admission to the study was 75.0 (range 20—154), indicating high acuity and the median Braden Scale value on admission (1300; range 9—19) indicated high risk for pressure ulcers. The overwhelming majority of patients ($n = 124$, 93.9%) were on the Total Care bed.

Changes in skin integrity

Of the 132 subjects, most (72.9%) had daily skin integrity observations up to day seven of the ICU stay, 63.6% also had observations on day 10 and 53.5% on day 14, with a total of

Table 2 Percent of time above critical levels for maximum and mean tissue interface pressure by region.

	Subjects	Average hours per subject	32 mmHg Level		45 mmHg Level		60 mmHg Level	
			≥32 mmHg	<32 mmHg	≥45 mmHg	<45 mmHg	≥60 mmHg	<60 mmHg
Maximum			Percent of time spent above and below each pressure level					
Sacrum	132	49.50	99.90	0.10	83.93	16.07	59.47	40.53
Left heel	121	10.22	67.50	32.50	31.30	68.70	16.08	83.92
Right heel	121	10.65	68.73	31.27	34.05	65.95	16.46	83.54
Left trochanter	120	0.58	99.94	0.06	95.35	4.65	78.98	21.02
Right trochanter	121	0.57	99.97	0.03	97.09	2.91	74.10	25.90
Left scapula	132	42.87	89.62	10.38	37.93	62.07	18.67	81.33
Right scapula	132	42.65	88.56	11.44	35.68	64.32	16.80	83.20
Mean			Percent of time spent above and below each pressure level					
Sacrum	132	49.50	8.00	92.00	0.04	99.96	0.00	100.00
Left heel	121	10.22	3.63	96.37	0.14	99.86	0.00	100.00
Right heel	121	10.65	4.04	95.96	0.35	99.65	0.00	100.00
Left trochanter	120	0.58	19.30	80.70	1.23	98.77	0.00	100.00
Right trochanter	121	0.57	30.20	69.80	0.50	99.50	0.00	100.00
Left scapula	132	42.87	1.45	98.55	0.04	99.96	0.00	100.00
Right scapula	132	42.65	0.90	99.10	0.15	99.85	0.00	100.00

925 patient days of observation and an average of 7.2 days of skin observations per subject. The majority (74.7%) had both morning and evening observations, while 18.4% had only morning observations and 6.9% had only evening observations. One hundred and twenty subjects (90.9%) had no observed changes in skin integrity on study admission or over the study period. Of the 12 subjects (5.3%) with changes in skin integrity, five had skin changes at the time of study enrolment (four had sacral changes, three had heel changes, one had trochanter changes) but no additional skin integrity changes were observed in these subjects during the study period. The remaining seven subjects showed changes in skin integrity in at least one of the seven anatomical locations over the study period and collectively, the changes in skin integrity across these seven subjects spanned all seven anatomical locations.

Tissue interface pressure above critical levels

Based on the tissue interface pressure critical levels described above, the majority maximum pressures were above 32 mmHg, that is, 99.9% of all observations across all subjects for the sacrum, left and right trochanter, and slightly less (89% of all observations across all subjects) for the left and right scapula, and 68% of all observations across all subjects for the left and right heels (Table 2, Fig. 1). However, a larger proportion of observations were above the 45 mmHg level for the left and right trochanter (95%), followed by the sacrum (84%), left and right scapula (36% and 38%), and the left and right heels (31% and 34%). A similar trend was observed for levels above 60 mmHg, with the left and right trochanter having the largest percent of time above 60 mmHg (74% and 79% of observations across all subjects) and the left and right heels having the lowest amount of time above 60 mmHg (17% and 19% of observations across all subjects).

Despite the large amount of time that the maximum pressure was above the thresholds examined, the mean pressure was not, with amount of time spent above mean critical levels, considerably less compared to maximum pressures. The left and right trochanter had the greatest amount of time above 32 mmHg (19% and 30% of all observations for all subjects), followed by the sacrum (8%), left and right heel (4%), and the left and right scapula (<2%). Similar to maximum pressures, these percentages decreased at the 45 mmHg level (all less than 2%) and none of the seven sites had mean pressure values above 60 mmHg.

Skin integrity changes and tissue interface pressure

Because there were only seven subjects with changes in skin integrity during the study period, we describe each change by anatomical location, study day, skin change type and time of resolution if present (Table 3). We also evaluated percent of time over the critical pressure levels for the time prior to documentation of the change in skin integrity (Table 3). Based on comparisons to the entire sample, trends of greater amounts of time above critical pressure levels were recognised. However, the amount of data time per subject varied due to varying time in the study, including one subject (#144) with a large amount of data time for sacral pressure data and a very small amount of time for left heel pressure data. Compared to data for the entire sample (Table 2) all changes in this subset for sacral tissue were associated with 100% of the time at maximum tissue interface pressure levels greater than 32 mmHg, although just minimally above the entire sample at 99.9% of the time. Four of the six sacral changes were associated with greater amounts of time above

Table 3 Skin integrity changes ($N = 14$ changes in 7 subjects) and maximum pressure by subject and observation period.

Subj ID[a]	Pressure data time in hours	Site	Study day, time change observed	Skin integrity change[b]	Resolution of Skin Integrity Change[c]	Percent of time above 32 mmHg prior to skin integrity change	Percent of time above 45 mmHg prior to skin integrity change	Percent of time above 60 mmHg prior to skin integrity change
144	102.54	Sacrum	Day 3 (am)	Stage II	Did not resolve[c]	100	99.9	95.3
148	61.45	Sacrum	Day 2 (am)	Suspected DTI	Did not resolve[c]	100	90.9	85.6
187	49.49	Sacrum	Day 2 (pm)	Suspected DTI	Worsened	100	94.4	66.2
187	49.49	Sacrum	Day 7 (am)	Stage 2	Improved	100	94.4	66.2
209	35.59	Sacrum	Day 2 (am)	Stage 2[d]	Worsened	100	75.3	56.2
209	35.59	Sacrum	Day 4 (am)	Stage 3	Improved	100	75.3	56.2
157	5.59	Left scapula	Day 1	Stage 1	Improved	100	99.87	83.53
157	81.16	Left scapula	Day 4	Suspected DTI	Did not resolve	99.97	72.21	49.97
152	41.42	Right scapula	Day 2 (pm)	Stage 1	Worsened	96.4	25.6	3.2
152	41.42	Right scapula	Day 7 (am)	Stage 2	Did not resolve[c]	96.4	25.6	3.2
157	74.44	Right scapula	Day 6 (am)	Suspected DTI	Did not resolve[c]	100	99.9	89.9
157	0.97	Right trochanter	Day 6 (am)	Suspected DTI	Resolved	99.9	66.4	41.8
231	4.35	Right trochanter	Day 4 (am)	Stage 2	Resolved	99.9	99.5	98.8
144	0.01	Right heel	Day 3 (pm)	Suspected DTI	Resolved	100	79.4	8.8

[a] Subject ID numbers used in the study ranged from 101—250.
[b] A skin integrity change was any deterioration in the observed skin integrity.
[c] Did not resolve indicates the injury continued through the study period. Suspected DTI—suspected deep tissue injury.
[d] Subject admitted with State 1 ulcer, progressed to State 2.

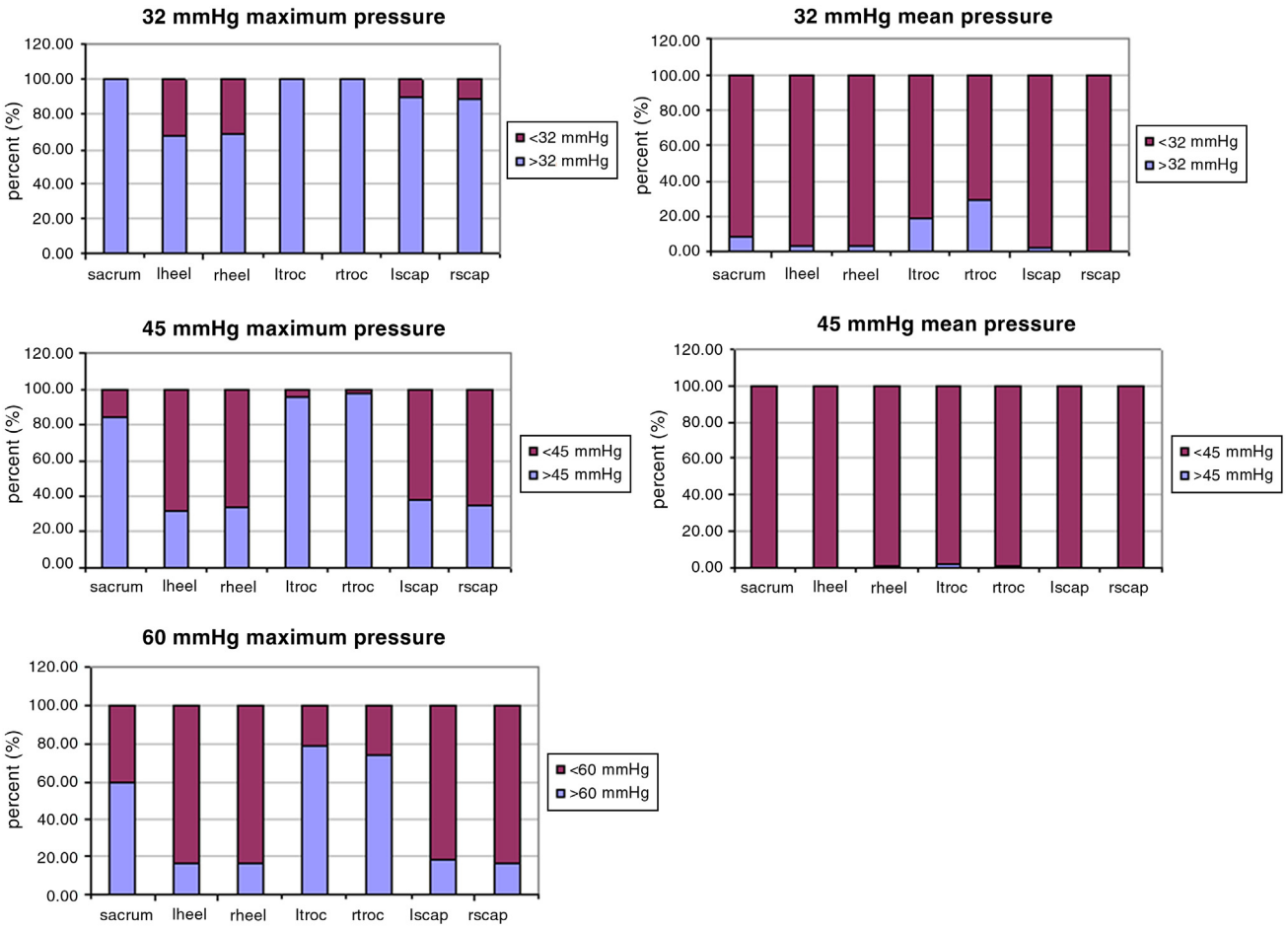

Figure 1 Percent of time above and below maximum and mean pressures of 32 mmHg, 45 mmHg and 60 mmHg.

both 45 mmHg and 60 mmHg than the entire sample. For those with left and right scapular skin changes, all time above 32 mmHg was greater than the entire sample, three of five changes were associated with greater time above both 45 mmHg and 60 mmHg. Similarly, one of the two subjects who experienced skin changes in the right trochanter had equal or greater time spent above 32 mmHg, 45 mmHg and 60 mmHg than the entire sample.

Discussion

To our knowledge, this is the first longitudinal study in critically ill, mechanically ventilated adults, to describe maximum tissue interface pressure, time spent above critical pressure levels and the effect on skin integrity. Although the incidence of pressure ulcers in the critically ill population has been recently reported to range from be 7% to 28% (Kaitani et al., 2010; Terekeci et al., 2009; Ülker Efteli and Yapucu Günes, 2013), we found relatively few changes, i.e. 5.3% of the subjects. In our setting just prior to study initiation pressure ulcer prevention strategies changed to include a skin barrier (Mepilex®, Molnlycke Health Care, Norcross, GA). The barrier was applied prophylactically to the sacral site for 97% of subjects in this study, for at least one day during the study period. However, of the 14 individual changes identified during the study period, almost half ($n = 6$) were still found in the sacral region, although it is unknown how this prophylaxis may have affected the incidence of sacral skin changes overall.

For the entire sample, the time spent above critical pressure levels was considerable with all time spent above 32 mmHg for maximum pressure for all anatomical locations. For sacral and trochanter sites, time spent at the 45 mmHg and 60 mmHg pressure was also extensive, ranging from 59% to 97% of the time. Although maximum pressures may have reflected short periods (i.e. a spike of pressure) above these maximal pressure levels, the large amount of time above the critical pressure levels remains substantial. Critical tissue interface pressure levels associated with changes in skin integrity are not standardised and a critical magnitude above which ischaemia occurs has not been established. While some authors have used the 32 mmHg (Defloor, 1999; Lippoldt et al., 2014; Peterson et al., 2008) others have used higher levels (Behrendt et al., 2014; Gunningberg and Carli, 2014; Sakai et al., 2009). As these data suggest, in addition to tissue interface pressure, other factors are contributory to the development pressure, including diastolic and systolic blood pressure, sheer forces, patient age, hydration and metabolism (Cox and Cwocn, 2011; Cox, 2013; Lachenbruch

et al., 2013). This sample including both medical and surgical critically ill patients had both high acuity (APACHE III score) and high risk for pressure ulcers (Braden Scale) and therefore may include many of the additional factors associated with pressure ulcer formation, although not specifically measured in this study.

Although time spent at maximum pressures above critical levels was considerable, when mean pressures were reviewed, time spent above critical pressure levels was minimal except for left and right trochanter sites above 32 mmHg (19.3% and 30.2% respectively) indicating that higher pressure were not ongoing.

Subjects that did experience changes in skin integrity also experiences tissue interface pressure greater than the mean pressures experienced by the entire sample and although this relationship was not statistically analysed due to small sample sizes, trends toward greater pressures during the period preceding changes in skin integrity, especially for the sacral and trochanter sites, were identified.

These results may be limited by the use of a heterogeneous sample, which included critically ill subjects from medical and surgical units, varying degrees of pressure ulcer risk, BMI and age. Although this may limit generalisation of the findings, our goal was to describe generally, tissue interface pressure magnitude and duration, which may serve as a stepping stone to identify critical issues in changes in skin integrity.

Conclusions

Maximum tissue interface pressure was above critical levels for the majority of the documented periods, especially in the sacrum, although few changes in skin integrity were documented. Time spent above critical levels for mean pressures were considerably less compared to maximum pressures. Maximum pressures may have reflected pressure spikes, but the large amount of time above the critical pressure levels remains substantial.

Pressure ulcers develop as result of a complex, multifactorial process and although high tissue interface pressure over extended periods of time are contributory to this process, other factors also place patients are risk. Subjects included in this analysis had high acuity and a high risk for pressure ulcers based on their APACHE III score and the Braden Scale results suggesting that a variety of pressure ulcer risk factors may have been present.

References

Alderden J, Whitney JD, Taylor SM, Zaratkiewicz S. Risk profile characteristics associated with outcomes of hospital-acquired pressure ulcers: a retrospective review. Crit Care Nurse 2011;31(4):30—43, http://dx.doi.org/10.4037/ccn2011806.

Anon. Pressure ulcers in America: prevalence, incidence, and implications for the future. An executive summary of the National Pressure Ulcer Advisory Panel monograph. Adv Skin Wound Care 2016;14(4):208—15.

Aoi N, Yoshimura K, Kadono T, et al. Ultrasound assessment of deep tissue injury in pressure ulcers: possible prediction of pressure ulcer progression. Plast Reconstr Surg 2009;124(2):540—50, http://dx.doi.org/10.1097/PRS.0b013e3181addb33.

Bader DL. The recovery characteristics of soft tissues following repeated loading. J Rehabil Res Dev 1990;27(2):141—50.

Behrendt R, Ghaznavi AM, Mahan M, Craft S, Siddiqui A. Continuous bedside pressure mapping and rates of hospital-associated pressure ulcers in a medical intensive care unit. Am J Crit Care 2014;23(2):127—33, http://dx.doi.org/10.4037/ajcc2014192.

Bennett L, Kavner D, Lee BK, Trainor FA. Shear vs pressure as causative factors in skin blood flow occlusion. Arch Phys Med Rehabil 1979;60(7):309—14.

Black J, Baharestani M, Cuddigan J, et al. National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system. Dermatol Nurs 2007;19(4):343—9, http://dx.doi.org/10.1097/01.ASW.0000269314.23015.e9, quiz 350.

Burk RS, Grap MJ. Backrest position in prevention of pressure ulcers and ventilator-associated pneumonia: conflicting recommendations. Heart Lung 2012;41(6):536—45, http://dx.doi.org/10.1016/j.hrtlng.2012.05.008.

Cox J, Cwocn A. Predictors of pressure ulcers in adult critical care patients. Am J Crit Care 2011;20(5):364—75, http://dx.doi.org/10.4037/ajcc2011934.

Cox J. Pressure ulcer development and vasopressor agents in adult critical care patients: a literature review. Ostomy Wound Manage 2013;59(4):50—4, 56—60.

Cuddigan J, Frantz RA. Pressure ulcer research: pressure ulcer treatment. A monograph from the National Pressure Ulcer Advisory Panel. Adv Wound Care 1998;11(6):294—300, quiz 302.

Daniel RK, Priest DL, Wheatley DC. Etiologic factors in pressure sores: an experimental model. Arch Phys Med Rehabil 1981;62(10):492—8.

Defloor T. The risk of pressure sores: a conceptual scheme. J Clin Nurs 1999;8(2):206—16.

Determann RM, Wolthuis EK, Spronk PE, et al. Reliability of height and weight estimates in patients acutely admitted to intensive care units. Crit Care Nurse 2007;27(5):48—55, quiz 56.

Dinsdale SM. Decubitus ulcers: role of pressure and friction in causation. Arch Phys Med Rehabil 1974;55(4):147—52.

Ek AC, Gustavsson G, Lewis DH. Skin blood flow in relation to external pressure and temperature in the supine position on a standard hospital mattress. Scand J Rehabil Med 1987;19(3):121—6.

Frantz RA, Xakellis GC. Characteristics of skin blood flow over the trochanter under constant, prolonged pressure. Am J Phys Med Rehabil 1989;68(6):272—6.

Frantz R, Xakellis GC, Arteaga M. The effects of prolonged pressure on skin blood flow in elderly patients at risk for pressure ulcers. Decubitus 1993;6(6):16—20.

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—8, 30—35.

Grap MJ, Munro CL, Wetzel PA, Schubert CM, Pepperl A, Burk RS, Lucas V. Backrest elevation and tissue interface pressure by anatomical location during mechanical ventilation. Am J Crit Care 2016;25(3):e56—63, http://dx.doi.org/10.4037/ajcc2016317.

Guidelines for Prevention of Nosocomial Pneumonia. Centers for disease control and prevention. MMWR Recomm Rep 1997;46(RR-1):1—79.

Gunningberg L, Carli C. Reduced pressure for fewer pressure ulcers: can real-time feedback of interface pressure optimise repositioning in bed? Int Wound J 2014(September)., http://dx.doi.org/10.1111/iwj.12374.

Herrman EC, Knapp CF, Donofrio JC, Salcido R. Skin perfusion responses to surface pressure-induced ischemia: implication for the developing pressure ulcer. J Rehabil Res Dev 1999;36(2):109—20.

Kaitani T, Tokunaga K, Matsui N, Sanada H. Risk factors related to the development of pressure ulcers in

the critical care setting. J Clin Nurs 2010;19:414—21, http://dx.doi.org/10.1111/j.1365-2702.2009.03047.x.

Knaus WA, Zimmerman JE, Wagner DP, Draper EA, Lawrence DE. APACHE-acute physiology and chronic health evaluation: a physiologically based classification system. Crit Care Med 1981;9(8):591—7.

Knaus WA, Wagner DP, Draper EA, et al. The APACHE III prognostic system: Risk prediction of hospital mortality for critically ill hospitalized adults. Chest 1991;100(6):1619—36.

Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med Rehabil 1959;40(2):62—9.

Kottner J, Dobos G, Andruck A, et al. Skin response to sustained loading: a clinical explorative study. J Tissue Viability 2015;24(3):114—22, http://dx.doi.org/10.1016/j.jtv.2015.04.002.

Lachenbruch C, Tzen Y-T, Brienza DM, Karg PE, Lachenbruch PA. The relative contributions of interface pressure, shear stress, and temperature on tissue ischemia: a cross-sectional pilot study. Ostomy Wound Manage 2013;59(3):25—34.

Lahmann NA, Kottner J. Relation between pressure, friction and pressure ulcer categories: a secondary data analysis of hospital patients using CHAID methods. Int J Nurs Stud 2011;48(12):1487—94, http://dx.doi.org/10.1016/j.ijnurstu.2011.07.004.

Landis E. Micro-injection studies of capillary blood pressure in human skin. Heart 1930;15:209—28.

Linder-Ganz E, Shabshin N, Itzchak Y, Yizhar Z, Siev-Ner I, Gefen A. Strains and stresses in sub-dermal tissues of the buttocks are greater in paraplegics than in healthy during sitting. J Biomech 2008;41(3):567—80, http://dx.doi.org/10.1016/j.jbiomech.2007.10.011.

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—26, http://dx.doi.org/10.4037/ajcc2014670.

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—42, http://dx.doi.org/10.1097/CCM.0b013e31818b8dbd.

Sakai K, Sanada H, Matsui N, et al. Continuous monitoring of interface pressure distribution in intensive care patients for pressure ulcer prevention. J Adv Nurs 2009;65(4):809—17, http://dx.doi.org/10.1111/j.1365-2648.2008.04935.x.

Shahin ESM, Dassen T, Halfens RJG. Incidence, prevention and treatment of pressure ulcers in intensive care patients: a longitudinal study. Int J Nurs Stud 2009a;46(4):413—21, http://dx.doi.org/10.1016/j.ijnurstu.2008.02.011.

Shahin ESM, Dassen T, Halfens RJG. Pressure ulcer prevention in intensive care patients: guidelines and practice. J Eval Clin Pract 2009b;15(2):370—4, http://dx.doi.org/10.1111/j.1365-2753.2008.01018.x.

Sprigle S, Sonenblum S. Assessing evidence supporting redistribution of pressure for pressure ulcer prevention: a review. J Rehabil Res Dev 2011;48(3):203—13.

Stechmiller JK, Cowan L, Whitney JD, Phillips L, Aslam RBA, et al. Guidelines for the prevention of pressure ulcers. Wound Repair Regen 2008;16:151—68.

Tablan OC, Anderson LJ, Besser R, Bridges C, Hajjeh R. Guidelines for preventing health-care—associated pneumonia, 2003: recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee. MMWR Recomm Rep 2004;53(RR-3):1—36.

Terekeci H, Kucukardali Y, Top C, Onem Y, Celik S, Oktenli C. Risk assessment study of the pressure ulcers in intensive care unit patients. Eur J Intern Med 2009;20(4):394—7, http://dx.doi.org/10.1016/j.ejim.2008.11.001.

Ülker Efteli E, Yapucu Günes Ü. A prospective, descriptive study of risk factors related to pressure ulcer development among patients in intensive care units. Ostomy Wound Manage 2013;59(7):22—7.

van Nieuwenhoven CA, Vandenbroucke-Grauls C, van Tiel FH, et al. Feasibility and effects of the semirecumbent position to prevent ventilator-associated pneumonia: a randomized study. Crit Care Med 2006;34(2):396—402.

Xakellis GC, Frantz RA, Arteaga M, Meletiou S. Dermal blood flow response to constant pressure in healthy older and younger subjects. J Gerontol 1993;48(1):M6—9.