



# BACKREST ELEVATION AND TISSUE INTERFACE PRESSURE BY ANATOMICAL LOCATION DURING MECHANICAL VENTILATION

By Mary Jo Grap, RN, PhD, Cindy L. Munro, RN, PhD, ANP, Paul A. Wetzel, PhD, Christine M. Schubert, PhD, Anthea Pepperl, PhD, Ruth S. Burk, RN, PhD, and Valentina Lucas, RN, PhD

**Background** Backrest elevations less than 30° are recommended to reduce pressure ulcers, but positions greater than 30° are recommended during mechanical ventilation to reduce risk for ventilator-associated pneumonia. Interface pressure may vary with level of backrest elevation and anatomical location (eg, sacrum, heels).

**Objective** To describe backrest elevation and anatomical location and intensity of skin pressure across the body in patients receiving mechanical ventilation.

**Methods** In a longitudinal study, patients from 3 adult intensive care units in a single institution receiving mechanical ventilation were enrolled within 24 hours of intubation from February 2010 through May 2012. Backrest elevation (by inclinometer) and pressure (by a pressure-mapping system) were measured continuously for 72 hours. Mean tissue interface pressure was determined for 7 anatomical areas: left and right scapula, left and right trochanter, sacrum, and left and right heel.

**Results** Data on 133 patients were analyzed. For each 1° increase in backrest elevation, mean interface pressure decreased 0.09 to 0.42 mm Hg. For each unit increase in body mass index, mean trochanter pressure increased 0.22 to 0.24 mm Hg. Knee angle (lower extremity bent at the knee) and mobility were time-varying covariates in models of the relationship between backrest elevation and tissue interface pressure.

**Conclusions** Individual factors such as patient movement and body mass index may be important elements related to risk for pressure ulcers and ventilator-associated pneumonia, and a more nuanced approach in which positioning decisions are tailored to optimize outcomes for individual patients appears warranted. (*American Journal of Critical Care*. 2016;25:e56-e63)

**T**wo prevalent and costly complications in patients receiving mechanical ventilation are pressure ulcers and ventilator-associated pneumonia (VAP).<sup>1,2</sup> Patients undergoing mechanical ventilation have multiple risk factors for both pressure ulcers and VAP; however, risk reduction strategies for these 2 complications are in conflict. Recommendations to reduce risk for pressure ulcers include positioning patients with the backrest elevated less than 30°, whereas recommendations to reduce VAP include placing patients with the backrest elevated more than 30°. <sup>3,4</sup> Although much research<sup>5,6</sup> has shown that higher backrest elevations reduce the occurrence of VAP, little empirical evidence is available on the effect of these higher backrest positions ( $\geq 30^\circ$ ) on factors that affect formation of pressure ulcers.

Pressure ulcers, any lesions caused by unrelieved pressure that results in damage of the underlying tissue,<sup>4,7</sup> are a serious complication of impaired mobility, and their prevalence in acutely and critically ill patients is high.<sup>8,9</sup> Because the cost of treating pressure ulcers is high, prevention is key. On a per-case basis, VAP is second only to central catheter-associated bloodstream infections as the most costly hospital-acquired infection and adds markedly to both intensive care unit (ICU) and hospital lengths of stay.<sup>2</sup> Backrest elevation and time spent supine are both critical factors in the occurrence of aspiration, which increases the risk for VAP.<sup>10-13</sup> Recommendations of the Centers for Disease Control and Prevention<sup>5</sup> and the National Quality Forum<sup>6</sup> for prevention of VAP include elevating the head of the bed to an angle of 30° to 45°. No data are available that describe tissue interface pressure and shear over time in critically ill patients receiving mechanical ventilation. Because recommendations to reduce VAP include higher backrest elevation than elevations recommended to prevent pressure ulcers, information on the effect of backrest elevation on the occurrence of pressure ulcers is important. Therefore, the aim of this descriptive, longitudinal study in critically ill patients receiving mechanical

ventilation was to describe the effect of backrest elevation on tissue interface pressure.

## Methods

### Setting and Sample

The study sample was obtained from all patients admitted to 3 ICUs (surgical trauma, medical respiratory, and neuroscience) in a 933-bed tertiary care, Mid-Atlantic, urban university medical center. Patients who were intubated and receiving mechanical ventilation that was expected to continue for at least 24 hours were enrolled in the study within 24 hours of intubation from February 2010 through May 2012. Because the beds in the target ICUs include airflow technology designed to reduce moisture, which is a risk factor for pressure ulcers, patients who might derive the greatest benefit from this moisture-reducing feature were excluded from the study. Patients were excluded if results of evaluation with the Braden Scale indicated that their skin was “constantly moist” (kept moist almost constantly by perspiration, urine, and so on; dampness is detected every time the patient is moved). Patients who had pressure ulcers at the time of enrollment in the study were included in the sample. Because backrest elevation and tissue interface pressure were measured continuously, any changes in these due to abnormalities in skin integrity were documented.

Studies of the association between backrest elevation and tissue interface pressure and tissue integrity are novel, and data on observed effect sizes are not available. Therefore, sample size was based on the available population of patients and the proposed study processes and time frame. Initial power analysis was done for detectable associations in the proposed sample size of 150. Such a sample, with

Pressure ulcers and ventilator-associated pneumonia are common in patients receiving mechanical ventilation.

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Pressure ulcer risk may also be affected by illness severity, a patient's weight, and other factors.

the conservative assumption of no efficiencies due to repeated measurements, would have 80% power to detect correlation levels between 0.23 and 0.26 for tissue interface pressure and backrest elevation. Thus, detection of reasonable trends and associations with backrest elevation should be possible.

### Key Variables and Their Measurement

*Backrest Elevation and Knee Angle.* Backrest elevation was continuously measured by using a process based on earlier research<sup>14</sup> and was updated by using microelectromechanical systems–based accelerometers (Analog Devices, Model ADXL203) to measure bed inclination angles. Each accelerometer, used as an inclinometer, was firmly held in place

on each of the 3 steel pivoting sections of the bed (backrest, hip, knee) by small industrial magnets. The knee angle reflected how much the lower extremity was bent at the knee, because beds often adjust this angle automatically as the head of the bed is raised. The filtered

analog signal was sampled at 2 measurements per second (2 Hz), a rate that matched the sample rate of the tissue interface pressure measurement.

*Tissue Interface Pressure.* Tissue interface pressure between the participant and the support surface was measured by using the XSENSOR pressure-mapping system (XSENSOR Technology Corporation), a sensor pad containing a matrix of individual capacitance-based pressure sensors. The pad is thin (1 mm [0.04 in] thick), is extremely flexible, and was made in full-bed size (sensing area, 24 x 72 in [61 x 183 cm]) for this project. Pads were placed beneath a hospital sheet to reduce the interaction of nursing staff with the pressure sensing system and the risk of pad damage. Because pressure ulcers occur most commonly over bony prominences, 7 common sites of pressure ulcer were used for measurements: left and right scapula, left and right trochanter, sacrum, and left and right heel. According to the participant's position (eg, supine, lying on left or right side), pressure measurements were documented when the site was in contact with the XSENSOR pad. Left and right trochanter areas included any recorded pressure as the patient was turned to the right or left side, whether a partial or a full lateral turn.

*Demographics and Other Covariates.* Immobility is a major risk factor for the development of pressure ulcers, especially in critically ill patients receiving mechanical ventilation, and tools used to evaluate the risk for pressure ulcers include measures of mobility.<sup>15-17</sup> Patient movement was measured by using a wrist and ankle actiwatch (model 198-0101,

Mini Mitter). Wrist and ankle actigraphic data are highly correlated, although movement of the wrist is greater than movement of the ankle in critically ill patients.<sup>18-20</sup> The actigraph contains a single omnidirectional accelerometer that integrates occurrence, degree, and intensity of motion to produce activity counts. Associations between actigraphic measurements, scores on the Richmond Agitation-Sedation Scale, scores on the Comfort Scale, and direct observation have been described.<sup>18</sup> Behavior states based on movement counts per minute measured by using actigraphy have also been described, identifying movement means for calm (arm, 6.79; lower extremity, 3.53), restless (arm, 28.54; lower extremity, 18.70), and agitated (arm, 52.59; lower extremity, 37.65) behavior.<sup>20</sup> For this study, actigraphy movement counts of the lower extremity were collected every second via a wireless data transfer system.

*Support Surface.* The type of support surface may affect the development of pressure ulcers. However, since 2008, the Hill-Rom TotalCare Duo2 system (Hill-Rom) is the primary bed type used in the ICUs in the study. The bed has a low air loss surface, developed to manage the microclimate of the skin, and has multizoned, air-filled bladders to redistribute pressure according to the patient's weight and position.

*Demographics.* The risk for pressure ulcers may also be affected by illness severity, a patient's weight, and other factors. Severity of illness was documented at the time of study enrollment by using the Acute Physiology and Chronic Health Evaluation (APACHE) III.<sup>21,22</sup> Each participant's age, sex, ICU type, and body mass index (BMI; calculated as weight in kilograms divided by height in meters squared) were also collected at the time of enrollment by using the morning weight and height according to the participant's legally authorized representative's statement of the participant's height. Scores on the Braden scale, used for daily assessments of risk for pressure ulcers, were documented by a study team member at the time of enrollment.

### Procedures

The study was approved by the university's institutional review board. Patients (or their legally authorized representative) who met study criteria were approached for consent. Participants were enrolled within 24 hours of intubation so that baseline skin assessments were obtained before consistent and extended use of higher backrest elevations. Descriptive data (ICU admission, APACHE III scores) were collected from the medical records for the 24 hours preceding enrollment. Backrest elevation and tissue interface pressure were evaluated continuously for 72 hours after enrollment. Data were collected solely while the participant was in

bed, because moving the XSENSOR pad to the chair was not consistently feasible.

### Data Reduction and Analysis

Measures of backrest and knee angle as well as tissue interface pressure were collected at a rate of twice per second. Graphical measures were used to examine these trends and determine the patterns by which to reduce data over time. After the stability of measurements over each second and at what point changes could occur were examined, data were condensed to 5-minute intervals for the 72-hour period, yielding upward of 4320 measurement points for the 72-hour observation period. Because of patient positioning, equipment concerns, and other factors, not every patient had available data for each of the 5-minute intervals. When such concerns occurred, data were flagged, and computations were adjusted to avoid biasing the resulting relationships between tissue interface pressure and bed elevation. Data from ankle actigraphy were used in the analysis because the information was more complete than that obtained with wrist actigraphy.

Descriptive statistics and repeated-measures models were used to describe skin interface pressure at various degrees of backrest elevation. All measures were checked for normality and then computed appropriately. Summary measures for backrest elevation were computed for the mean and maximum bed angle elevation (backrest and knee) over each repeated data-reduction period within the 72-hour observation. Backrest elevation was then used in multivariate repeated-measures regression models to determine relationships between elevation levels and tissue interface pressure. Model predictors included interactions between backrest elevation, knee angle, and BMI as well as ankle actigraphy. Any nonsignificant interaction ( $\alpha > 0.1$ ) was removed from the model. These analyses were repeated for each of the 7 major sites of occurrence of pressure ulcers for supine patients (sacrum, right and left scapula, right and left trochanter, and right and left heel). Results are presented according to the 7 major sites of occurrence of pressure ulcers for the mean tissue interface pressure, because the maximum pressure by segment had similar trends and results. All analyses were conducted by using SAS, version 9.2 (SAS Institute Inc), software and the significance level was set at  $\alpha = 0.05$ .

### Results

Of the 718 patients screened for the study, 470 (65%) were eligible. Of those eligible, 150 gave consent and were enrolled. Reasons for nonenrollment included family unavailable, consent declined, and medical issues. Of the 278 ineligible patients, 24

**Table**  
Demographics of 133 study participants

| Characteristic             | No. | Mean           | SD    | Minimum | Maximum |
|----------------------------|-----|----------------|-------|---------|---------|
| Age, y                     | 133 | 55.86          | 17.21 | 18.00   | 90.00   |
| BMI <sup>a</sup>           | 133 | 27.96          | 6.68  | 12.05   | 44.93   |
| APACHE III score           | 133 | 77.50          | 27.29 | 20.00   | 154.00  |
| Braden score               | 131 | 12.71          | 1.68  | 9.00    | 19.00   |
| Characteristic             | No. | % <sup>b</sup> |       |         |         |
| <b>Sex</b>                 |     |                |       |         |         |
| Male                       | 76  | 57.1           |       |         |         |
| Female                     | 57  | 42.9           |       |         |         |
| <b>Race</b>                |     |                |       |         |         |
| Black/African American     | 66  | 49.6           |       |         |         |
| Unknown or not reported    | 2   | 1.5            |       |         |         |
| White                      | 65  | 48.9           |       |         |         |
| <b>Ethnicity</b>           |     |                |       |         |         |
| Hispanic                   | 2   | 1.5            |       |         |         |
| Non-Hispanic               | 131 | 98.5           |       |         |         |
| <b>Intensive care unit</b> |     |                |       |         |         |
| Medical respiratory        | 61  | 45.9           |       |         |         |
| Neuroscience               | 30  | 22.6           |       |         |         |
| Surgical trauma            | 42  | 31.6           |       |         |         |
| <b>BMI group</b>           |     |                |       |         |         |
| < 19                       | 8   | 6.0            |       |         |         |
| 19 ≤ BMI < 25              | 41  | 30.8           |       |         |         |
| 25 ≤ BMI < 30              | 36  | 27.1           |       |         |         |
| > 30                       | 48  | 36.1           |       |         |         |

Abbreviations: APACHE, Acute Physiology and Chronic Health Evaluation; BMI, body mass index.

<sup>a</sup> Calculated as weight in kilograms divided by height in meters squared.

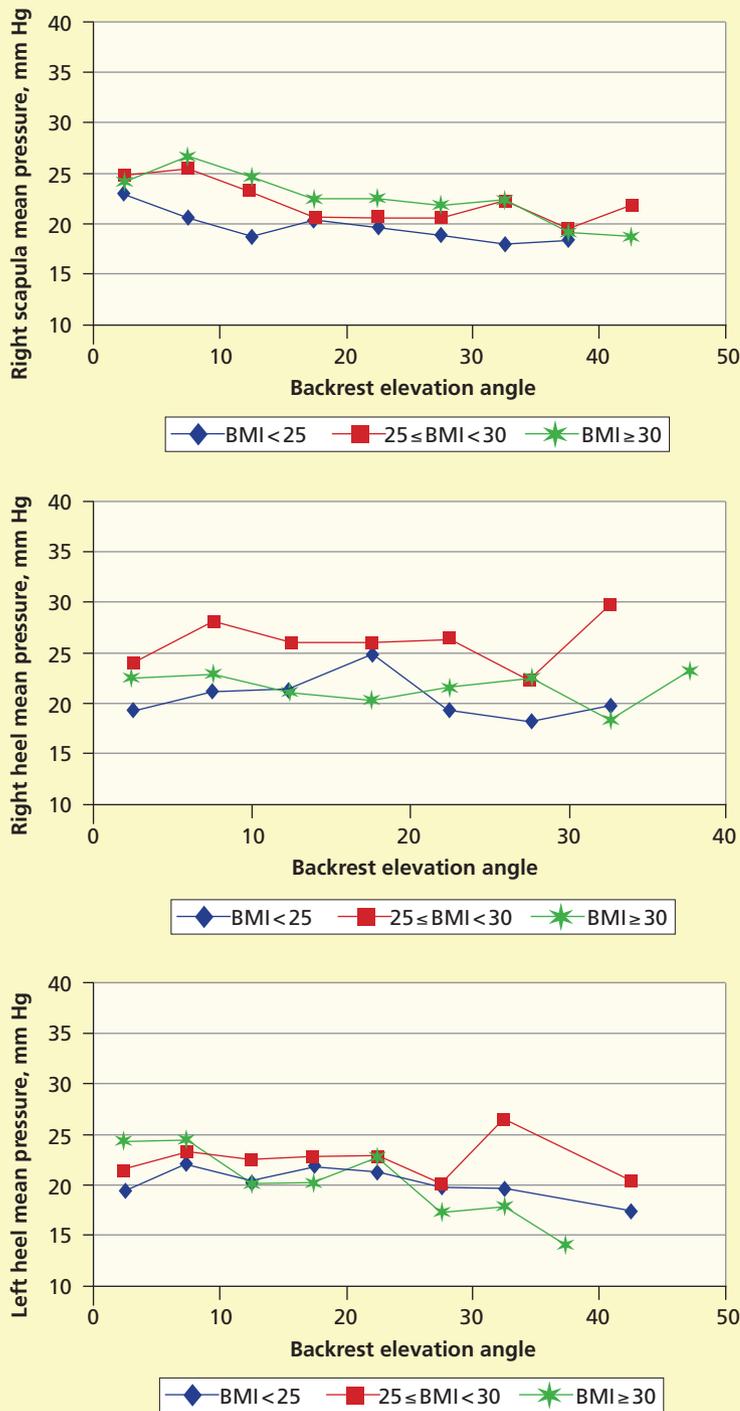
<sup>b</sup> Because of rounding, not all percentages total 100.

(10%) were deemed constantly moist status. Of the 150 patients enrolled in the study, 133 had segments of usable data on tissue interface pressure for at least some part of the observation period, and these data were included in the analysis. The majority of participants were non-Hispanic, male ( $n = 76$ , 57.1%), overweight (BMI > 25), and in the medical respiratory ICU, but were roughly evenly divided

between ethnic backgrounds (see Table). Mean APACHE III score on enrollment in the study was 77.5 (SD, 22.29), indicating high acuity, and the mean Braden Scale value (12.71; range, 9-19) indicated high risk for pressure ulcers. Almost all participants ( $n = 124$ ; 93.2%) were on the TotalCare bed.

Tissue interface pressure did vary by backrest elevation (Figures 1 and 2). Backrest elevation was grouped by every 5° for plotting, and only groupings in which the sample size was large enough (> 5 observations) are shown. Mean interface tissue pressure for the scapula and heel (Figure 1) is shown

**Backrest elevation and tissue interface pressure were evaluated for 72 hours after enrollment.**



**Figure 1** Tissue interface pressure for the scapula and heels by backrest elevation and body mass index (BMI, calculated as weight in kilograms divided by height in meters squared). Left scapula plots were similar to these right scapula plots and are not shown here.

separately from mean interface tissue pressure for the trochanter and sacrum (Figure 2). Mean pressure of the scapula and heel ranged similarly from about 15 mm Hg to 27 mm Hg, with the heel pressure approaching a mean of 30 mm Hg. The mean

interface tissue pressures of the trochanter and sacrum were much higher, however, about 27 to 38 mm Hg for the trochanter and about 20 to 35 mm Hg for the sacrum.

Mean tissue interface pressure in both the left and right scapula decreased significantly ( $P < .001$ ) as backrest elevation increased. In general, for each additional degree increase in backrest elevation, mean interface pressure decreased 0.09 to 0.42 mm Hg (Figure 1). In addition, mean interface pressure increased significantly with increased movement (ie, increased actigraphy counts;  $P < .001$ ); and with increases in pressure for larger BMI values (0.21 to 0.46 mm Hg for each additional BMI unit) and as the knee angle increased from a straight position.

For low knee angles, mean tissue interface pressure in both the right and left heels decreased slightly as backrest elevation increased: about 0.04 to 0.10 mm Hg per  $1^\circ$  increase in backrest elevation (Figure 1). For higher knee angles, mean pressure increased slightly, from 0.1 to 0.6 mm Hg per  $1^\circ$  increase in backrest elevation. However, these changes were significant solely for the left heel ( $P = .002$ ). Mean tissue interface pressure increased significantly in both the left and right heels with increasing movement, that is, actigraphy counts (both  $P < .001$ ).

Mean tissue interface pressure in the left and right trochanters did not change significantly with changes in backrest elevation ( $P = .15$  and  $P = .13$ , respectively), with knee angle ( $P = .64$  and  $P = .95$ , respectively) or with actigraphy ( $P = .70$  and  $P = .21$ , respectively). However, as BMI increased, the mean tissue interface pressure in the right and left trochanters increased ( $P < .001$  and  $P = .001$ , respectively). For each unit increase in BMI, mean trochanter pressure increased 0.22 to 0.24 mm Hg (Figure 2).

Finally, despite a small change in the mean tissue interface pressure of the sacrum as backrest elevation angle increased (Figure 2), this difference was not significant ( $P = .43$ ). Mean tissue interface pressure of the sacrum increased significantly with increased BMI ( $P < .001$ ), actigraphy ( $P < .001$ ), and increased knee angle ( $P < .001$ ), although the increases were slight (an increase of  $1^\circ$  in knee angle resulted in an increased mean pressure of 0.06 mm Hg).

Analysis revealed that knee angle and movement were time-varying covariates in the models for tissue interface pressure. Despite a range of knee angles from  $0^\circ$  to  $38.6^\circ$ , the distribution of observed knee angles was highly skewed, with a median of  $2.5^\circ$  (25th and 75th percentiles,  $0.27^\circ$  and  $7.04^\circ$ , respectively) and a mean of  $3.8^\circ$ . About 64% of observations were for knee angles between  $0^\circ$  and  $60^\circ$ ; 35.8% of observations were collected with the knees at  $100^\circ$ , and 17% had no knee angle ( $0^\circ$ ). Similarly, leg actigraphy counts per 5 minutes were also skewed.

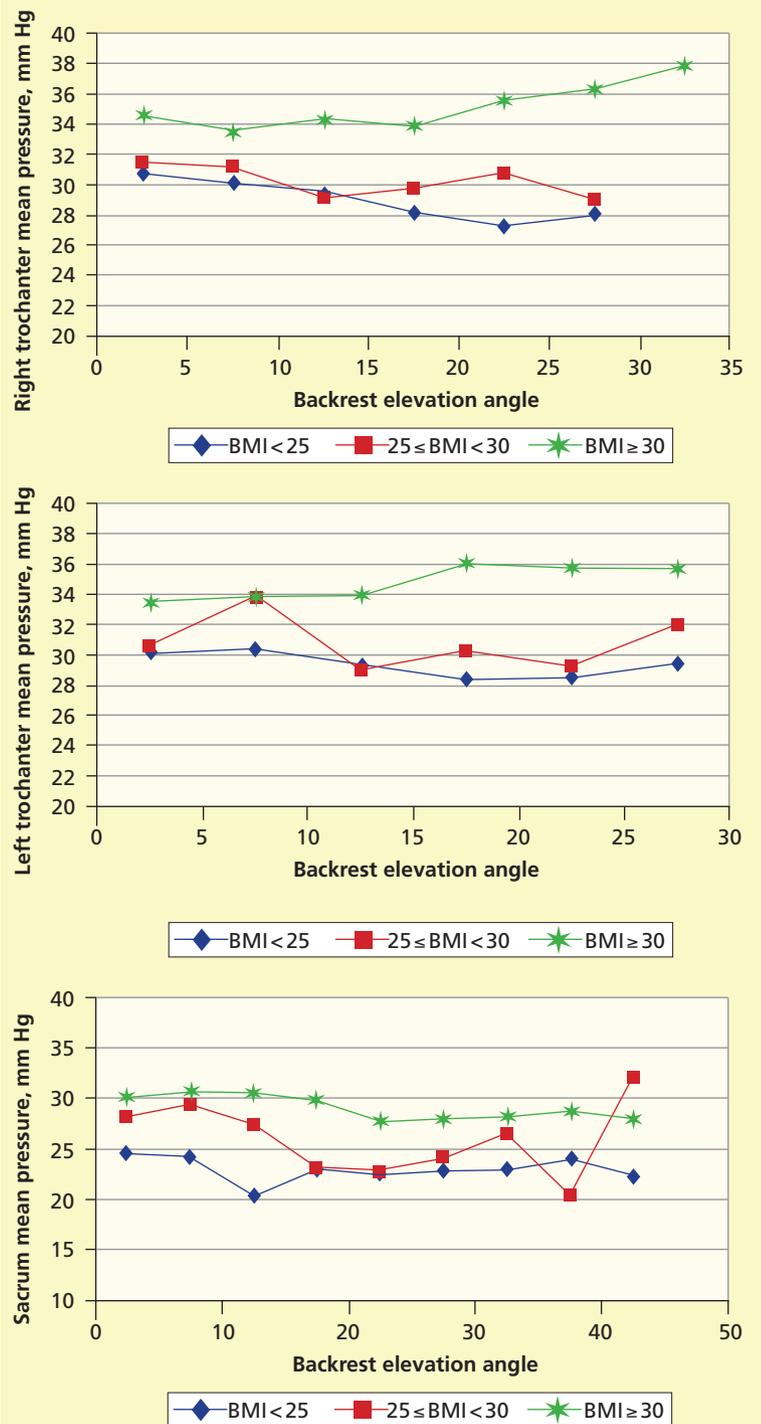
The counts ranged from 0 to 105; the median was 0.20, and the mean was 2.09 movements per 5 minutes, indicating calm behavior.<sup>20</sup>

## Discussion

The purpose of this descriptive, longitudinal study in critically ill adults receiving mechanical ventilation was to describe the effect of backrest elevation on tissue interface pressure. The acuity of all participants was high, and both critically ill medical and surgical patients receiving mechanical ventilation were well represented in the sample. Although backrest elevation did affect tissue interface pressure in some areas, the relationship was also affected by knee angle, BMI, and patient mobility.

Overall tissue interface pressures were less in the scapulas and heels than in the trochanter and sacral areas. Tissue interface pressure decreased as backrest elevation increased, specifically in the scapula, but not in the sacrum, heels, or trochanter. A more upright position (ie, higher backrest elevation) should relieve scapular pressure, although pressure did not increase in the sacrum as might be expected. Despite limited movement by the participants in this sample (the behavior of most participants was rated as calm), tissue interface pressure increased with movement, specifically in the heels, a finding that may reflect participants' use of the heels to move or lift the rest of the body. Although increased mobility (eg, out of bed) certainly reduces occurrence of pressure ulcers, the effect of a patient's movement in the bed, as we tracked by using actigraphy, is not well understood, and so far as we know, no researchers have used actigraphy to assess the risk for or incidence of pressure ulcers related to patient mobility. Although we did not measure shear forces directly, possibly increased restlessness added to skin shearing forces increases rather than reduces the risk for pressure ulcers.

Higher tissue interface pressures were associated with higher BMI in the trochanters and sacral area, as might be expected. These data are similar to those of Hyun et al<sup>23</sup> who found that extremely obese patients were about 2 times more likely to experience an ulcer than were normal weight patients even though addition of the BMI did not improve the accuracy of the Braden Scale for predicting pressure ulcers. However, in a secondary data analysis of 10 surveys on the prevalence of pressure ulcers, Kottner et al<sup>24</sup> found that the prevalence of pressure ulcers on the trunk (sacrum, ischial tuberosity, trochanter, and shoulder) was significantly higher in thin patients than in normal weight and obese patients. In addition, irrespective of the degree of mobility and activity, thin patients were at higher risk for pressure ulcers on areas of the trunk than



**Figure 2** Tissue interface pressure for the trochanter and sacrum by backrest elevation and body mass index (BMI, calculated as weight in kilograms divided by height in meters squared).

were normal weight and obese patients, but heel pressure ulcers seemed to be unrelated to BMI. Kottner et al indicated that the etiology and pathogenic mechanisms of the development of pressure ulcers on the trunk and heel may be different. Furthermore, in a comparison of the prevalence of pressure ulcers in patients with a BMI of 40 or more and Braden

Scale scores of 16 or more with the prevalence in patients with lower BMI, Drake et al<sup>25</sup> found that the prevalence among patients with a BMI less than 40 was 12.5% compared to a prevalence of 26% in patients with a BMI greater than 40 ( $P=.01$ ). Patients with BMIs greater than 40 were almost 3 times more likely to have a pressure ulcer than were patients with BMIs of 40 or less, after controlling for scores on Braden Scale indicative of risk ( $P=.01$ ).

Our study is the first evaluation of the relationship between knee angle and tissue interface pressure. Although the data on knee angle were limited, we found that pressure increased as the knee angle increased from a straight position. Most beds used in critical care automatically elevate the knee as the backrest is elevated, thus creating a greater knee angle. Although this change in knee angle did increase sacral tissue interface pressure, the increase was minimal. Patients may find that elevation of the knees provides more comfort when the backrest is elevated, and this knee elevation may not adversely affect tissue interface pressure.

Backrest elevation is recommended to reduce the risk for pneumonia; however, concerns about adverse skin effects due to elevated tissue interface pressure remain. Our data, however, do not support significant elevations of tissue interface pressure on the basis of backrest elevation alone; patients' movements and BMI also affect the pressure.

Our study had several limitations. Measurement of tissue interface pressure via a pressure-sensing device may result in measurement error due to movement of the sensor pad, patients' movements, or issues related to pressure-relieving and alternating air mattresses. Furthermore, pad manufacturers vary, and comparisons across pads may be difficult. We attempted to reduce errors by using a full-bed pressure pad that had extensions on the top and bottom that could be secured under the bed mattress. In addition, we secured the pad with tape to the mattress and bed frame to reduce movement of the pad. Recent recommendations include standardized processes for evaluating support surfaces by using pressure measurements.<sup>26</sup>

However, these recommendations are focused on laboratory processes that may not apply in the actual critical care setting. We did not measure shear forces in this study, although shear may be an important risk factor for the formation of pressure ulcers,<sup>1,8,9</sup> especially with higher backrest elevations, because patients may slide down as the backrest is elevated.

Therefore, any conclusions drawn from our data should also include consideration of shear forces.

In summary, our data indicate that the relationships among backrest elevation, risk for pressure ulcers, and risk for VAP are more complex than what is reflected in the current conflicting recommendations for reducing risks for pressure ulcers and for VAP. Individual factors such as patients' movements and BMI appear to be important elements related to risk, and a more nuanced approach in which positioning decisions are tailored to optimize outcomes for individual patients appears warranted. Additional research is needed to inform individualized interventions.

#### FINANCIAL DISCLOSURES

This study was supported by National Institutes of Health grant R01 NR010381 to Dr Mary Jo Grap.

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For more about backrest elevation, visit the *Critical Care Nurse* Web site, [www.ccnonline.org](http://www.ccnonline.org), and read the article by Metheny and Frantz, "Head-of-Bed Elevation in Critically Ill Patients: A Review" (June 2013).

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Backrest elevation affected tissue interface pressure but the relationship was also affected by knee angle, BMI, and patient mobility.

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