Failure to Reposition After Sliding Down in Bed Increases Pressure at the Sacrum and Heels

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Abstract: The head of the bed (HOB) for a patient is often elevated since it improves comfort and facilitates respiratory functions. However, elevating the HOB essentially causes the patient support surface to turn into a ramp, forcing the patient's body to slide down. As the patient slides down, weight pressing on the pelvis, the coccyx, and the ischial tuberosities increases, resulting in associated increases in interface pressures. *Methods.* In an institutional review board-approved study, pressure distribution was measured on volunteers at 4 discrete body positions sliding down in bed on 3 commonly used support surfaces. *Results.* This study showed the total contact area of the patients decreased as they slid down, resulting in increased pressure, particularly on the sacral area and the heels. The study also confirmed that these pressure increases are persistent and occurred on all of the support surfaces tested. *Conclusion.* These increases in pressure likely contribute to the development of pressure ulcers.

Key words: support surface, pressure ulcers, sacral ulcer, heel ulcer

Pressure ulcers are a major burden to health care institutions, families and, especially, individual patients. They increase the length of hospital stays[1] and the overall cost of patient care. For example, in the United States the average hospital treatment cost associated with stage IV pressure ulcers and related complications was found to be $129,248 for hospital-acquired ulcers during 1 admission and $124,327 for community-acquired ulcers over an average of 4 admissions.[2] Besides cost, pressure ulcers reduce quality of life[3] and negatively influence morbidity and mortality, with 2.5 million patients impacted annually and approximately 60,000 deaths per year, respectively.[4]

The reported number of patients with pressure ulcers varies widely, depending on factors such as the type of population surveyed and the country where the survey took place. In a survey of acute care patients in the United States, 12.0%-19.7% were reported to suffer from 1 or more pressure ulcers.[5] Alternatively, in a community setting in the United Kingdom, the prevalence rate among adults with pressure ulcers stage I or higher was reported to be between 0.40 and 0.77 per thousand adults.[6] In Swit-

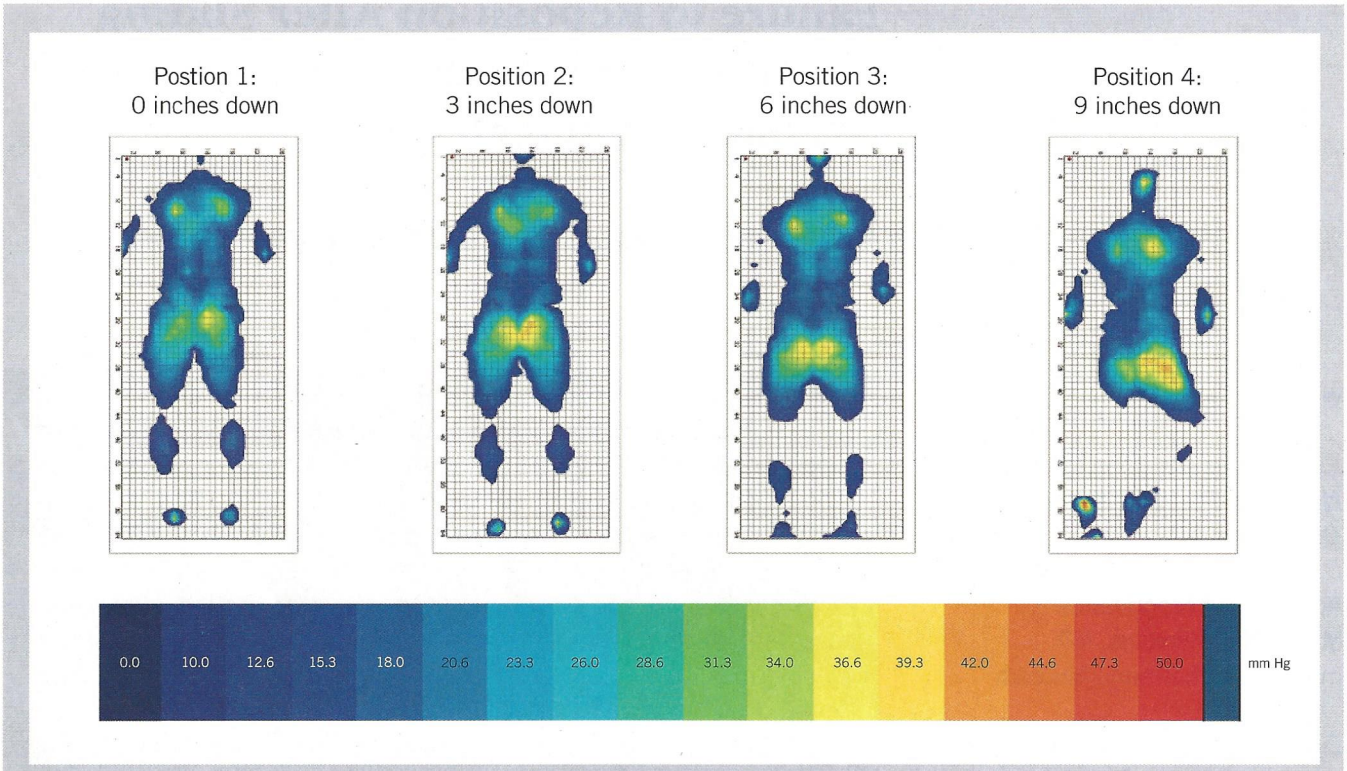

Figure 1. Change in weight distribution and increased pressure with different positions of the subjects in the bed.

zerland, patients with spinal cord injuries had an incidence of developing a pressure ulcer of 2.2 per person per year.[7]

Several internal and external factors may contribute to the development of pressure ulcers. Internal factors, such as certain medical conditions (eg, paraplegia, infection,[8] and malnutrition[9,10]), can negatively contribute to pressure ulcer development. Conversely, healthy tissue in a healthy patient has been shown to be more resistant to the influence of pressure and pressure ulcer development.[11]

Pressure, shear, and friction are the main external factors that lead to the development of pressure ulcers. These variables, through different mechanisms, all contribute to a relative or absolute insufficient level of perfusion,[8,12] resulting in tissue breakdown and the relative failure of circulation.

Pressure is the primary variable that influences pressure ulcer development and is a result of the amount of pressure and the time during which that pressure is exerted.[13,14] If and when pressure remains below a certain threshold for a certain anatomical location, the probability of the development of a pressure ulcer diminishes or may even be nonexistent. This occurs because the local circulation remains sufficient. In 2 studies of patients with spinal injuries, Bogie et al1[5,16] suggested pressure threshold values of transcutaneous partial oxygen pressure ($tcPO_2$) of 30 millimeters of mercury (mm Hg) and a transcutaneous partial carbon dioxide pressure ($tcPCO_2$) of 44 mm Hg. The study concluded that a $tcPO_2$ of < 30 mm Hg and/or a $tcPCO_2$ of > 44 mm Hg correlates with a significantly higher risk of tissue damage. Threshold values depend on several factors, one example being anatomical location. These values change when other forces, such as shear, occur at the same time.[12,17] Ischemia and reperfusion have also been shown to be contributing factors in the development of pressure ulcers.[18,19]

Shear, the deformation of a (solid) body in which a plane in the body is displaced parallel to itself relative to parallel planes in the body, is important since the deformation results in capillary destruction and tissue death.[12] An example of shear would be when a patient slides down in bed or when they are pushed/pulled back up in bed without being lifted off the surface. It should be noted that the influence of shear is not related to time.[17,20]

Many consider friction a force that contributes to

pressure ulcer development by itself; however, if a body or body part is not moving, it is static friction that prevents it from moving. In such a situation, pressure, temperature,[21-23] and/or moisture[24] may be variables that cause pressure ulcer formation. Once movement occurs, it is unlikely the entire moving surface (eg, skin, subcutaneous fat, muscle) will move in exactly the same direction with the same force and with the same level of acceleration. Consequently, the shear forces, rather than friction, are responsible for the development of pressure ulcers[25] relative to body movement.

Guidelines exist that address the prevention of pressure ulcer development and include patient repositioning strategies.[26] Frequent repositioning reduces the time during which a specific part of the anatomy experiences reduced pressure and, thus, reduced perfusion. For example, a study concluded that regularly turning a patient by a dedicated team dramatically decreases the incidence of pressure ulcer development in an intensive care unit setting.[27]

Various types of support surfaces are also recommended for the treatment and prevention of pressure ulcers for different types of patients[26,28-32] based on their risk factors as determined by different algorithms.[28,29]

As it relates to pressure ulcer development, little attention has been given to the position of the patient in bed, particularly as they slide down in bed when the head of the bed (HOB) is elevated. The HOB is elevated for most every patient to improve comfort,[33] make eating easier, and facilitate respiratory functions. This is especially true for patients with chronic obstructive pulmonary disease, those being tube fed, or those short of breath. In addition, a semi-recumbent position of 30 degrees or higher is often recommended for patients treated with mechanical ventilation to prevent aspiration and pneumonia.[34-36]

However, an elevated HOB turns any support surface into a ramp, gradually causing the patient's body to slide down in the bed as a result of gravity. The mass and body type of the individual, as well as the articulation of the bed frame and the type of support surface being used, play a critical role in the ultimate forces the patient's body is exposed to over time. As the patient slides down in bed, weight pressing on the pelvic region, particularly the sacrum, the coccyx, and the ischial tuberosities, dramatically increases. This can lead to a significant increase in pressure. For example, in a related study, pressure measurements at the pelvis of up to 300 mm Hg were observed.[37]

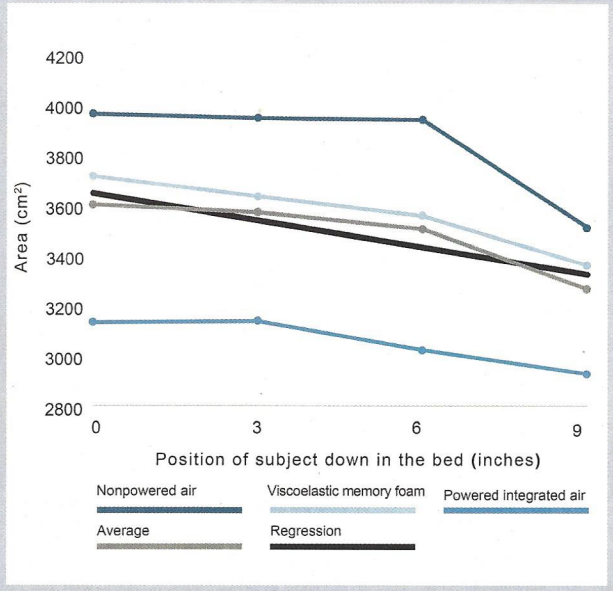

Figure 2. Total contact area.

Most hospital beds are designed to automatically contour the knee section, and sometimes the seat section, of the bed to help prevent the patient from sliding down when the HOB is elevated. However, these features cannot overcome the forces of gravity and all observed patients inevitably slide down toward the foot end of the bed.

In an effort to analyze and better understand the effects of pressure at different positions as the body slides down in bed, an institutional review board (IRB)-approved protocol was used for a descriptive study comparing pressure distribution on different body locations at 4 discrete positions in bed using pressure mapping.

Methods

The study was conducted in the laboratory of Evan Call MS, CSM at Weber State University, Ogden, UT. Participants, 2 male and 2 female, were randomly picked from a pool of volunteers maintained by the laboratory for the purpose of different types of tests. Formal exclusion criteria for participation in this study per the protocol (although they did not apply to the volunteers in any case) were persons who were pregnant, diseased, ill, or otherwise compromised, as well as persons whose medical status would preclude them from lying in a hospital bed for 2-4 hours. Individuals with existing skin tissue breakdown, such as the presence

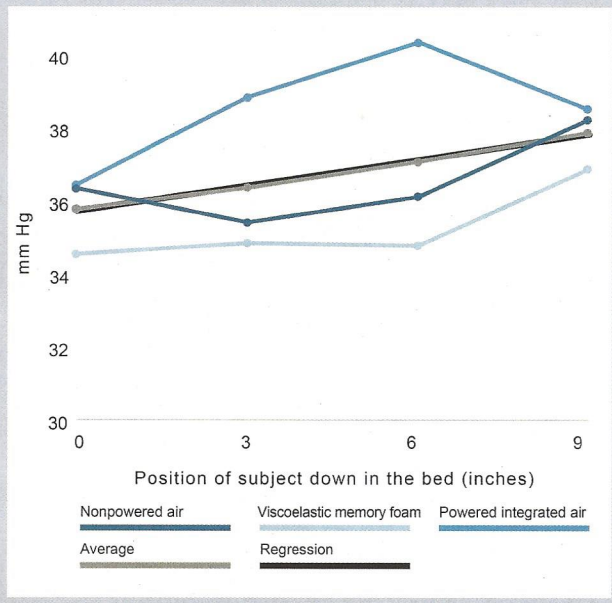

Figure 3. Sacrum peak pressure.

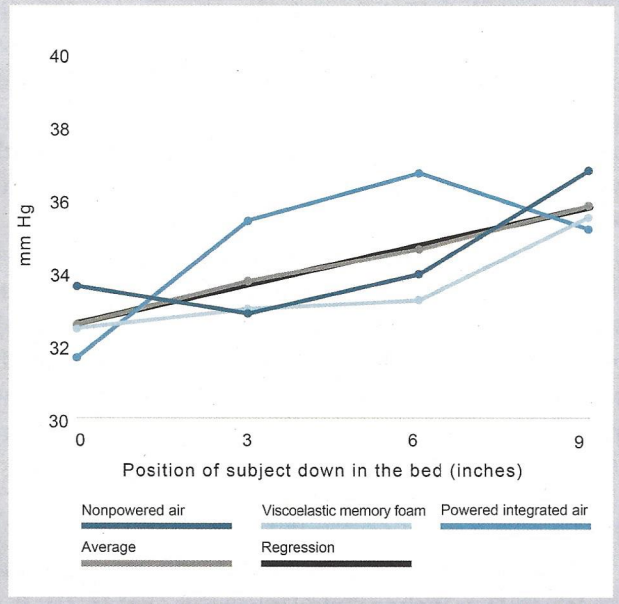

Figure 4. Sacrum peak pressure index.

of a pressure ulcer, abrasions, and excoriations, were also excluded. Sex, weight, length, and body mass index (BMI) of the volunteers were as follows: female, 145 lb, 67 in tall, BMI 22.7; female, 148 lb, 68.75 in tall, BMI 22.0; male, 178 lb, 71.5 in tall, BMI 24.5; and male, 220 lb, 74 in tall, BMI 28.2.

All volunteers wore typical hospital gowns with their own undergarments. The following 3 common types of hospital bed surfaces were used: powered integrated air surface (VersaCare A.I.R. Surface, Hill-Rom, Batesville, IN); nonpowered air surface (Accumax Quantum VPC, Hill-Rom, Batesville, IN); and viscoelastic memory foam surface (Hercules Dream Sleep Surface, The Morel Company, Batesville, IN). All were FDA Class II exempt medical devices as per CFR 21CFR880.5150.

For each volunteer and each surface, 4 different patient positions were used to achieve baseline pressure measurements:

- 30 degree elevation of HOB using the bed's auto profile function, which adjusts the knee angle to approximately 14 degrees when the HOB is at 30 degrees, with the volunteer sliding 0 inches down in the bed;
- 30 degree elevation of HOB using the bed's auto profile function, with the volunteer sliding 3 inches down;
- 30 degree elevation of HOB using the bed's auto profile function, with the volunteers sliding 6 inches down;
- 30 degree elevation of HOB using the bed's auto profile function, with the volunteer sliding 9 inches down.

A pressure map (Xsensor X3 Pro Pressure Map, 36 x 84, XSensor, Calgary, Canada) was placed directly on the support surface. In this map, each sensor covered 1.25 square inches. The map was calibrated as per the manufacturer's Calibration Device User's Guide using a 10 lb and 50 lb calibrated weight and using the manufacturer's software (Xsensor X3 Pro software). Each volunteer was assessed while lying on a clean sheet placed directly on top of the pressure map. The results were measured in units of mm Hg.

Each volunteer was asked to lie down on the bed in a comfortable position with the HOB elevated to 30 degrees. A skin marker was used to identify a location on the volunteer's side between the pelvis and rib cage. A fixed laser level was pointed at the mark. As the volunteer was asked to take different positions in the bed, the distance between the skin marker and the laser light was used to measure the distance from the original position to determine the amount of sliding.

Baseline pressure maps were recorded in the initial position (0 inches down, no sliding) for each volunteer on each surface. Next, each volunteer was measured at the 3 subsequent sliding positions (3 in, 6 in, and 9 inches down). After each of the 4 positions, the pressure mapping process was repeated 5 times and aver-

aged per volunteer, per position, and per surface. Each measurement lasted for 120 seconds.

For this study, the pelvis was defined as the area from the top of the lateral iliac crest to the base of the perineum, as identified in the pressure mapping image. All other anatomic zones were defined for each volunteer by the anatomic features visible in the pressure maps.

Statistical Analysis

Output data were entered into spreadsheet software (Excel, Microsoft, Redmond, WA) for further calculations, which are described here.

Contact Area (CA, cm²): the area with pressure readings greater than or equal to 10 mm Hg. The contact area was calculated from CA = (A x N10) / N_{Total} where A = area of the pressure map containing sensors (cm^2), N10 = number of sensors with pressure readings greater than or equal to 10 mm Hg, and N_{Total} = total number of sensors in map (or within a specified zone).

Peak Pressure (PP, mm Hg): the highest recorded reading on the pressure map or within a specified zone.

Peak Pressure Index (PPI, mm Hg): the highest recorded average in a given area, measured using 4 sensors (2 x 2 array) including the PP.

Results

Figure 1 demonstrates the change in weight distribution and increased pressure that patients experience when they slide down in bed. This creates a reduction in total contact area and results in an increase in pressure as the patient slides down. Overall, the total contact area drops from an average of 3,602 cm^2 to 3,266 cm^2, representing a 9.3% reduction. This decrease in total contact area resulted in an increase in pressure, peak pressure, and peak pressure indices in both the sacral area and heels.

Basic physics supports this finding; a given amount of force applied over a small area will produce greater pressure than the same amount of force applied over a larger area.

The sacral area PP and PPI measurements both revealed significant increases when the 0-inch position was compared to the 9-inch position (Figures 3 and 4). The average PP increased from 35.8 mm Hg to 37.9 mm Hg, representing a 5.9% increase. The average PPI increased from 32.6 mm Hg to 35.8 mm Hg, representing a 9.8% increase. Additionally, as Figure 1 demonstrates, when the volunteers' feet contacted the footboard, the sacral anatomical areas bearing weight shifted more towards the trochanters as the patients knees bend and the torso progressively slides down in the bed.

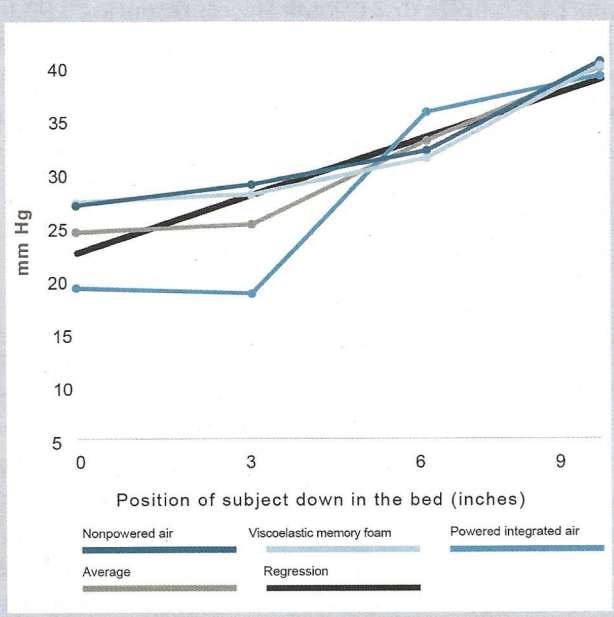

Figure 5. Heel peak pressure.

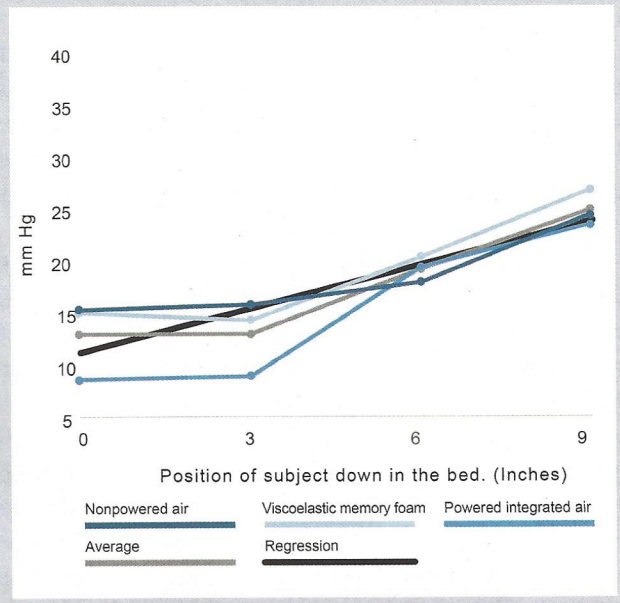

Figure 6. Heel peak pressure index.

The PP and the PPI in the heel area showed significant upward trends (Figures 5 and 6) as the volunteers slid down in bed. The average heel PP increased from 24.4 mm Hg to 39.8 mm Hg, representing a 63.1% increase from 0 in to 9 in. The average heel PPI increased

from 12.9 mm Hg to 24.9 mm Hg, representing a 93.0% increase. This correlates well with the pressure shift demonstrated in Figure 1. The increase in heel pressure occurred as the volunteers' feet contacted the footboard, causing their knees to bend and shifting additional weight forces onto their heels. There was no significant difference in either the sacral or heel PP and PPI indices between the 3 surfaces tested.

Discussion

Several reasons exist to elevate the HOB, such as providing comfort to the patient and reducing the risk of aspiration.[33-36] However, when the HOB is raised, gravity forces the patient to slide down in bed. This has a number of potentially negative consequences with regard to weight distribution. The total contact area of the patient decreases as they slide down, resulting in increases in pressure (ie, PP and PPI), particularly with regard to the sacral area and the heels. This, essentially, is basic physics as a given amount of force applied over a small area will produce greater pressure than the same amount of force applied over a larger area.

In addition, the following observations were made during the study: as the patients' feet contacted the footboard their knees bent, resulting in significant pressure increase on the heels; and as the patients' knees bent, their bodies rotated onto their sides, increasing pressure in the trochanter area.

It is highly likely that the above experiences will contribute to a higher incidence of pressure ulcer development for patients. Indeed, the study results presented here are consistent with recent literature findings. Okuwa[38] observed that in some patients in the supine position and with the HOB elevated to 30 degrees, wound margins at the sacrum and coccyx regions were thickened. This study also showed that pressure, measured at this thickened skin was higher than pressure in normal skin in the same patient while the thicker-skinned margins healed more slowly, leading to the conclusion that a 30-degree HOB elevation negatively influences healing by increasing pressure.

In another study where $TcPO_2$ and $TcPCO_2$ levels were measured, $TcPO_2$ was shown to decrease and $TcPCO_2$ to increase in the sacral area if the HOB angle was 45 degrees or higher, indicating compromised tissue viability,[39] while peak interface pressure significantly increased in the same anatomical area when the HOB was raised to 30 degrees, both in volunteers[40] and in at-risk patients.[41]

The current study confirms that regularly and frequently repositioning the patient to reduce the amount of time a patient spends in a compromised position in bed is crucial in the efforts to prevent the formation of pressure ulcers as has been suggested by other authors.[42,43] However, in spite of this recognition, Peterson and coleagues[41] note the practice of repositioning needs improvement. Several reasons have been identified why repositioning does not take place frequently enough, including the patient being asleep, refusal by the patient or the family, the patient suffering from pain and, for the nursing staff, the lack of time and/or help for the repositioning effort.[44]

Limitations

Since this was a study with healthy volunteers, there is a theoretical chance the values measured in this study are not applicable to patients who are seriously ill. In addition, because of the limited number of volunteers and their physical attributes, the study results may not be applicable to patients with significantly different properties (eg, patients who are morbidly obese) or proportions (eg, very tall or very short subjects).

A future study might use a larger variety of body morphologies with a larger number of volunteers to reach a category B level of evidence.[45] A study of patients, as opposed to volunteers, could compare the results of repositioning vs those of not repositioning the patient. However, given the known consequences of not repositioning the patient, the latter type of study would be ethically questionable and be unlikely to receive IRB approval. In addition, it is unlikely that diseases with a systemic impact (eg, lower oxygen saturation, diaphoresis) would have an influence on the type of measurements presented here given the relative pressure differences within the body would not change.

Conclusion

The position of a patient in bed strongly correlates to pressure distribution and the angle of the HOB also plays a major role. When the HOB is elevated, gravity forces the patient to slide down, causing significant increases in pressure to appear, particularly in the sacral area and the heels. This study confirmed this observation and also showed that this pressure increase is persistent, independent of the type of support surface.

Frequent repositioning of a patient is essential to mitigate these increases in sacral and heel pressure. Unfortunately, repositioning puts a major burden on

the medical staff with regard to the physical demand required to reposition. In addition, the lack of nursing personnel in many regions of the world[46] and many different health care settings[47-49] leads to problems with regard to time and staff availability for repositioning. However, frequent and timely repositioning of patients must be an integral part of any effective pressure ulcer management program.

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