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The Effect of a Liner on the Dispersion of Sacral Interface Pressures During Spinal Immobilization

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Sacral pressure ulcers are a significant problem following spinal cord injury and are felt to be in part due to the high interface-pressures generated while strapped to the spine board. The objective of this study was to determine sacral interface-pressure and sensing area in healthy volunteers on a spine board and the effects of a gel pressure dispersion liner. Thirty-seven volunteers were placed on a pressure-sensing mat between the subject and the spine board. Measurements were carried out with and without a gel liner. Pressures and sensing area were recorded every minute for 40 minutes. The highest pressure was generated at the sacral prominence of each subject. Mean interface-pressures were higher on the spine board alone than with the gel liner ($p < .0001$). Overall, mean sensing area was lower on the spine board than with the gel liner ($p < .0001$). Standard spinal immobilization causes high sacral interface-pressures. The addition of a gel liner on the spine board decreased overall mean sacral pressures and increased mean sensing area. Generation of sacral pressure ulcers may be related to the initial interface-pressures generated while the patient is strapped to the spine board. The addition of a gel liner may reduce the incidence of sacral pressure ulcers.

Keywords: spinal cord injury, backboard, gel liner, pressure, time, pressure ulcer

Introduction

Recommendations for early management of the trauma victim with a potential spinal cord injury (SCI) by the American College of Surgeons consist of immobilization with a hard spine board, a rigid cervical collar, lateral support devices, and tape or straps to secure the patient (American College of Surgeons Committee on Trauma, 2008). The most significant concern during the initial management of patients with potential SCI is that neurologic function may be impaired due to movement of the unstable or injured vertebrae. It is estimated that 3 to 25% of SCI occur after the initial traumatic insult, either during transit or early in the course of management (Engsberg et al., 2013; Hadley et al., 2002).

While immobilization on a hard spine board is relatively effective in limiting motion, one of the associated complications is skin breakdown (Chan, Goldberg, Tascone, Harmon, & Chan, 1994; Linares, Mawson, Suarez, & Biundo, 1987; Mawson et al., 1988). The incidence of pressure ulcers in newly-admitted

patients with SCI has been reported at 24 to 59% (Chen, Apple, Hudson, & Bode, 1999; Mawson et al., 1988; National Spinal Cord Injury Statistical Center, 2006). The most common location for pressure ulcers was the sacrum in 33 to 57% of these subjects (Chen, Apple, Hudson, & Bode, 1999; Mawson et al., 1988; National Spinal Cord Injury Statistical Center, 2006). Additionally, experimental studies have revealed that the formation of pressure ulcers directly varies with length of time immobilized and amount of pressure generated (Husain, 1953; Linares et al., 1987; Mawson et al., 1988; National Spinal Cord Injury Statistical Center, 2006). Furthermore, a constant pressure of only 60 mmHg for one hour is sufficient to cause irreversible tissue damage in dogs (Kosiak, 1961), and low pressure maintained and localized for a long period of time produces more damage than high pressure for a short period of time (Husain, 1953). It is also noted that the threshold pressure level for damage seems to be reduced after SCI due to a drop in tissue perfusion as a result of neurogenic shock and the use of a spine board (Mawson et al., 1988).

Total time immobilized on the spine board was identified in patients admitted to the Emergency Department at several centers and ranged from 77 to 295 minutes (Cordell et al., 1996; Yeung, Cheung, Graham, & Rainer, 2006; Lerner & Moscati, 2000). Therefore, with the knowledge that individuals with a

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suspected acute SCI may be at increased risk for the formation of a pressure ulcer (Mawson et al., 1988) while strapped to a spine board for 1 to 5 hours (Cordell et al., 1996; Lerner & Moscati, 2000; Yeung et al., 2006), one might conclude that any localized pressures above 60 mmHg may predispose the patient to pressure ulcer formation (Husain, 1953; Kosiak, 1961).

From a financial perspective, the prevalence of pressure ulcers in the United States is estimated to be 1.5 to 3 million adults with an estimated cost of \$500 to \$40,000 to heal each ulcer. (Lyder, Shannon, Empleo-Frazier, McGehee, & White, 2002). In 2006, almost \$11 billion was paid out for hospital stays in which pressure ulcer was either a primary or secondary diagnosis (Healthcare Cost and Utilization Project, 2008). From a practice perspective as of October 1, 2008, the Centers for Medicare and Medicaid Services (CMS) no longer reimburse hospitals for treatment of new stage III to IV pressure ulcers not documented in the medical record by day two of inpatient admission (Federal Registry, 2008; Krapfl & Mackey, 2008). Since the cost reimbursement by CMS is no longer available for newly developed pressure ulcers, there has been a major focus on prevention. Unfortunately, for trauma patients admitted to a hospital strapped to a spine board, the damage may already be done, and there is little one can do except remove the patient from the spine board.

Identification of factors that prevent the formation of pressure ulcers in the early acute management of SCI would reduce the incidence of pressure ulcers and hospital costs. Since the most common location of pressure ulcer formation in this population is the sacrum, identification of pressures generated over the sacrum would be important to help define the risks for pressure ulcer formation. Others have identified sacral interface pressures and evaluated the effects of spine board overlays. The vacuum mattress-splint was thought to be the answer, with a marked reduction in pain and sacral interface pressures (Chan, Goldberg, Mason, & Chan, 1996; Lovell & Evans, 1994; Sheerin & de Frein, 2007), but when the vacuum is released, the surface is just as hard as that of the standard spine board (Hemmes, Poeze, & Brink, 2010; Keller, Lubbert, Keller, & Leenen, 2005). Soft layered spine boards have also been assessed and compared to the standard spine board and vacuum mattress (Hemmes et al., 2010; Oomens et al., 2013), which showed improved comfort and reduced peak pressure generation, but no data after 15 minutes of immobilization was reported.

The pressure reducing overlays on the spine board in the various studies have all been successful in reducing pressure. Several factors need to be considered when choosing the proper material for spine board overlays. In the field, when IVs are started, blood is drawn, the spine is immobilized, and CPR may be necessary, the material property of the various pressure reducing overlays needs to be considered. The material properties of air, foam, and gel contained overlays are due to their viscous and elastic attributes. When viscous materials (gel and air) are loaded, they tend to flow away from compressive forces and redistribute pressures. When elastic materials (foam) are loaded, they tend to store energy and push back against compressive forces. (Brienza & Geyer, 2000, 2005). Air-filled layers are lightweight and allow for an even pressure distribution but are somewhat unstable and require high maintenance due to the need to monitor proper inflation pressures and the risk of puncture. (Akins, Karg, & Brienza, 2011; Brienza & Geyer, 2000, 2005; Goossens, 2007). Foam surfaces are inexpensive, lightweight, and don't leak. However,

foam tends to degrade over time and lose its stiffness, resulting in higher tissue pressures and risk for bottoming out. Furthermore, the elastic properties of foam give it the highest horizontal stiffness measurement, leading to the potential for shear related tissue injury (Akins et al., 2011, Brienza & Geyer, 2000, 2005; Goossens, 2007). Low-viscosity gel support surfaces on the other hand provide the most comfort with excellent pressure distribution, and have less horizontal stiffness resulting in a reduction of shear stress on tissues (Akins et al., 2011, Brienza & Geyer, 2000, 2005; Goossens, 2007). These characteristics/attributes make the low-viscosity gel material an ideal surface to analyze. The objective of this study was to evaluate sacral interface pressure and sensing area in healthy volunteers during prolonged standard spinal immobilization on a spine board and the effect of a gel pressure dispersion liner (PDL).

Methods

Participants

Thirty-seven volunteers between the ages of 18 and 60 years were recruited for this study. Participants who had acute or chronic back pain or were pregnant were excluded from study participation. All participants gave their informed consent. MetroHealth Medical Center IRB approved the study protocol.

Study Design

Subjects wore street clothes and all items were removed from their pockets. Each subject was placed on a level surface in a supine position on a BaXstrap® Spineboard measuring 16 inches × 72 inches × 2.5 inches with soft head-blocks and straps, as seen in Figure 1a and 1b. A Force Sensing Array (FSA) pressure-sensing mat (Vista Medical, Canada) measuring 18 inches × 18 inches, was placed between the subject and the spine board centered at the belt-line to monitor sacral interface pressures and the distribution of pressure (sensing area). For all volunteers, measurements were carried out on the spine board—on different days—with and without a gel PDL. The low-viscosity gel PDL was an Oasis operating room overlay measuring 70.866 inches × 20.5 inches × .3937 inch. The first surface to be evaluated on each subject (spine board or spine board + gel PDL) was determined by flipping a coin. Pressure and sensing area were recorded every minute (0.0167 Hz) over a 40-minute period.

Study Participant Measures

Age (in years) and Sex (Male/Female) were recorded prior to the start of the study. Participants' height (in inches) and weight (in pounds) and body mass index (BMI) were documented prior to lying on the spine board. For analysis, height was broken down into a short group, <68 inches, and tall group, ≥68 inches. Weight was broken down into a light group, <160 pounds, and a heavy group, ≥160 pounds. BMI was broken down into a low group, <25, and a high group, ≥25.

Interface Pressure Measurements

We used the FSA pressure-mapping system to measure contact pressure at the buttock-spine board interface. The system is

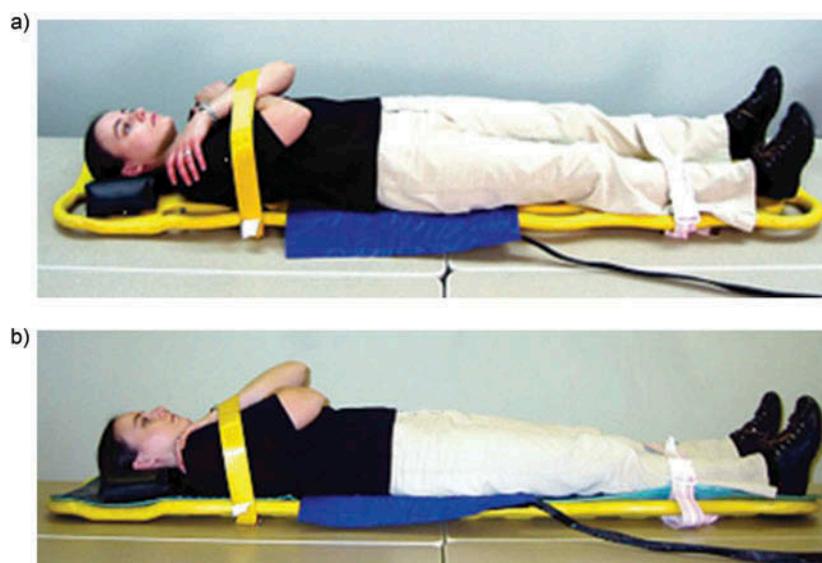


Fig. 1. Experimental set up of a subject on (a) spine board, and (b) spine board with the gel PDL.

comprised of a pressure-sensing mat that contains 256 sensors connected to the FSA computer software via an interface module. Data were recorded as color-coded maps of pressure distribution, three-dimensional grids, and numeric output parameters as illustrated in Figure 2a and 2b. The primary outcome measures were interface pressure, measured as the pressure in mmHg and sensing area measured in inches squared. The peak pressure within the sensing area was recorded on each subject every minute for 40 minutes on the spine board and the board with the gel PDL. From this, we determined the mean of the peak pressure for each subject over 40 minutes and the overall mean peak pressure on all subjects. The sensing area was also recorded on each subject every minute for 40 minutes on the spine board and the board with the gel PDL. From this, we determined the mean of the sensing area for each subject over 40 minutes and the overall mean sensing area on all subjects. The FSA system was calibrated to 500 mmHg prior to the beginning of the study with an autocalibrator specific to the system and according to the instructions in the manufacturer's manual to ensure accuracy.

Analysis

Standard descriptive statistics (frequencies, means, and standard deviations) were calculated to describe the study sample and outcome measures. Paired Student T-test was used to determine whether mean pressure and sensing area on the standard spine board and the board with the gel PDL differed significantly. Student T-tests were conducted to assess significant differences in pressure and sensing area between the height, weight, and BMI groups. IBM's SPSS statistical software Version 12 was used to analyze the data for this manuscript.

Results

We evaluated 37 subjects on both surfaces. We recruited 17 males and 20 females. The mean age of the subject sample was 32.6 years (SD = 10.17), mean weight was 162 pounds (SD =

25.58), mean height was 68.8 inches (SD = 3.64) and mean BMI was 24.7 (SD = 24.73).

Table 1 and Figures 3a and 3b compare the mean peak sacral interface pressures on the spine board and the spine board with the gel PDL. The overall mean peak pressures were higher on the spine board compared to the spine board with the gel PDL ($p = .000$). Table 1 and Figures 4a and 4b compare the mean sensing area on the two board types. The overall mean sensing area was lower on the spine board compared to the spine board with the gel PDL (93.17 in² and 112.75 in², respectively, $p < 0.000$). The overall mean peak pressures on the spine board for all subjects each minute over the 40-minute test period increased from 263.6 mmHg to 271.9 mmHg (3%), and the overall mean peak pressures on the spine board + gel PDL increased from 183.2 mmHg to 194.2 mmHg (6%) over the 40 minute time period; see Figure 5.

When data was analyzed in relation with height of the participant, the overall mean peak pressure on the spine board + gel PDL was significantly higher in the tall group than in the short group (216.00 mmHg vs. 161.22 mmHg; $p = 0.047$); see Table 2. BMI and weight did not show any significant statistical differences in overall mean peak sacral pressure. The high-BMI compared to the low-BMI group had larger sensing areas for the spine board and the spine board + gel PDL ($p = .004$ and $p = .000$, respectively); see Table 2. Heavy participants had a larger mean sensing area on the spine board + gel PDL than the lightweight participants (122.16 vs. 98.94; $p = .005$); see Table 2.

Discussion

Our results reveal that standard immobilization on a spine board in healthy volunteers caused extremely high overall mean peak sacral interface pressures (2 mmHg) and a low mean sensing area (90.82in²). The addition of a gel PDL to a spine board decreased overall mean peak sacral interface pressures (187.87 mmHg) by 27% and increased sensing area (113.34 in²) by 21%. Our study also showed that overall mean peak pressures on the spine

Table 1. Comparisons between pressure and sensing area on the backboard and backboard + gel PDL.

		Backboard	Backboard with PDL	95% CI		t	P
				Lower	Upper		
Pressure (mmHg)	Mean	258.19	187.87	-101.77	-38.87	-4.535	<.0001
	SD	143.46	84.43				
Sensing Area (in ²)	Mean	93.17	112.75	12.63	26.53	5.712	<.0001
	SD	22.44	25.72				

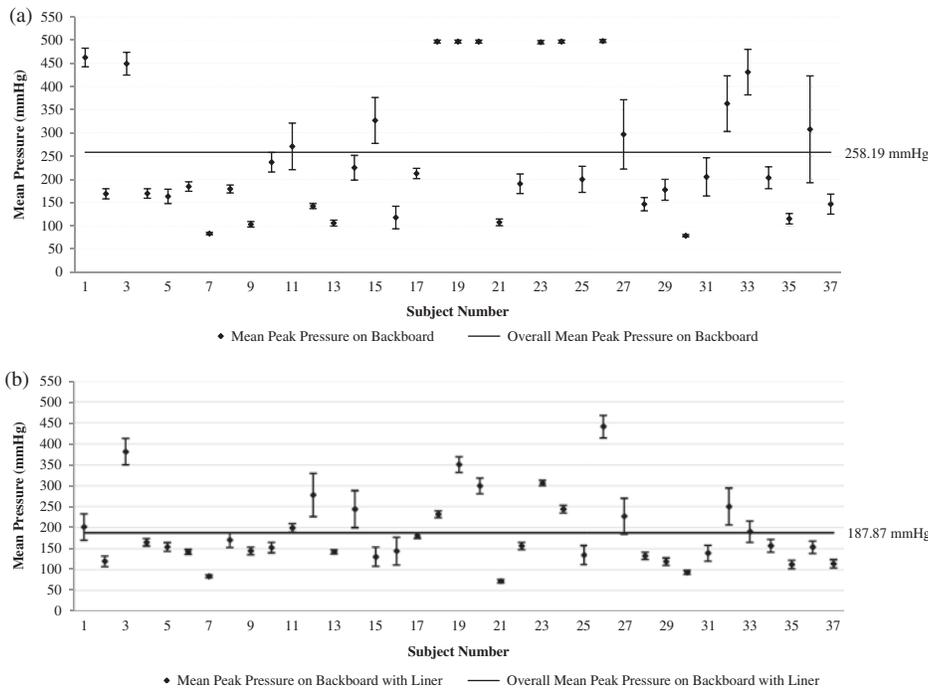


Fig. 3. Mean peak sacral interface pressure for each subject and overall mean peak sacral interface pressure on the spine board and on the spine board + gel PDL.

and may necessitate a further design change in the spine board and or the gel PDL. Furthermore, changes in BMI and weight did not show any significant statistical differences in mean peak sacral pressure in individuals while strapped to the spine board.

Many of these findings are consistent with previous research that has demonstrated high spine board interface pressures and the reduction of this pressure by the addition of air, vacuum, foam, or soft-layered overlays. Lovell and Evans assessed the sacral interface pressures in 30 healthy volunteers on the spine board (147.3 mmHg), foam padded spine board (115.5 mmHg), and vacuum stretcher (36.7 mmHg; Lovell & Evans, 1994). Their findings revealed a significant decline of sacral interface pressures in the foam padded spine board and vacuum stretcher. Cordell assessed the generation of pressure in healthy volunteers that were similarly strapped to a spine board with or without an air mattress (Cordell, Hollingsworth, Olinger, Stroman, & Nelson, 1995). In 20 volunteers, sacral interface pressure was significantly different using the spine board alone (145.5 mmHg) compared to the spine board with an air mattress (48.5 mmHg). Keller et al. assessed sacral interface pressures in 20 healthy

volunteers on a spine board, a vacuum mattress, and an overlay foam mattress (Keller et al., 2005). The peak sacral interface pressures on each subject were reported on each surface, including 174.9 mmHg on the spine board, 165.6 mmHg on the vacuum mattress, and 118.0 mmHg on the overlay foam mattress. Their study concluded that the three support surfaces all produced high ischemic pressures. In a pilot study, Sheerin and de Frein assessed sacral interface pressures in two healthy volunteers on a spine board, a spine board with a flotation device, and a vacuum mattress (Sheerin & de Frein, 2007). The peak sacral interface pressures were reported on each surface, including 154.33 mmHg on the spine board, 76 mmHg on the spine board with flotation device, and 70.67 mmHg on the vacuum mattress. Hemmes et al. assessed sacral interface pressures in 30 healthy volunteers on three support systems, including a spine board, vacuum mattress, and soft-layered long spine board (Hemmes et al., 2010). The mean peak pressure was reported to be the similar in the spine board and vacuum device; it was much lower on the soft-layered spine board. Their study concluded that compared to the soft-layered long spine board, the standard spine board and vacuum mattress produced much higher interface pressures and provided

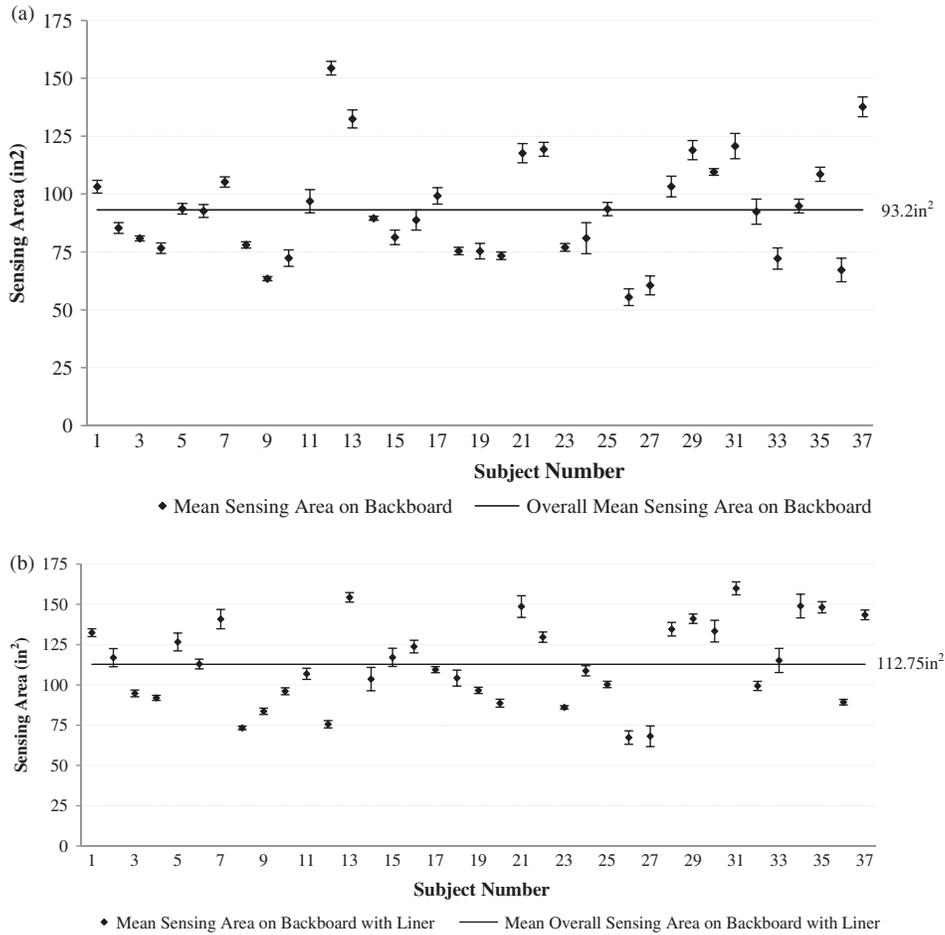


Fig. 4. Mean peak sensing area for each subject and overall mean peak sensing area on the spine board + gel PDL.

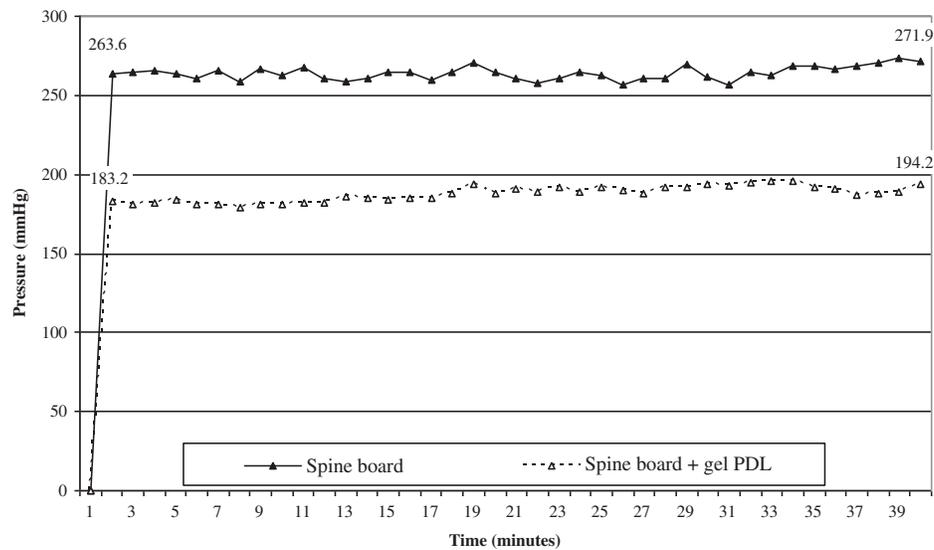


Fig. 5. Overall mean sacral interface pressures for all subjects each minute over the 40-minute test period on the spine board and on the spine board + gel PDL.

poor support for the lumbar spine. Details of each study are outlined in Table 3.

Elevated mean peak sacral interface pressures have been demonstrated by this and other studies; these pressures are felt

to increase the risk of early post-acute sacral pressure ulcers while the patient is strapped to the spine board. However, one must keep in mind that even though the development of pressure ulcers is associated with elevated interface pressures, many other

Table 2. Differences in pressure and sensing area for different categories of height, weight, and BMI.

	Height	Backboard		t	P	Backboard with PDL		t	P
		<68" (n = 19)	≥68" (n = 18)			<68" (n = 19)	≥68" (n = 18)		
Pressure (mmHg)	Mean	226.86	116.83	-1.382	.176	161.22	216.00	-2.059	.047
	SD	291.26	163.95			49.28	104.37		
Sensing Area (in ²)	Mean	89.74	20.68	-0.953	.347	109.55	96.79	-0.772	.445
	SD	96.79	24.21			23.77	24.21		
Weight		<160lbs (n = 15)	≥60lbs (n = 22)			<160lbs (n = 15)	≥160lbs (n = 22)		
	Mean	262.19	255.45	0.138	.891	188.22	187.63	0.021	.983
Pressure (mmHg)	SD	131.24	154.21			74.66	92.22		
	Mean	85.10	98.67	-1.867	.070	98.94	122.16	-2.976	.005
Sensing Area (in ²)	SD	23.34	20.53			21.05	24.69		
	BMI	<25 (n = 20)	≥25 (n = 17)			<25 (n = 20)	≥25 (n = 17)		
Pressure (mmHg)	Mean	279.55	233.05	0.982	.333	208.21	169.93	1.625	.113
	SD	154.42	129.43			97.00	61.15		
Sensing Area (in ²)	Mean	83.79	104.21	-3.06	.004	99.58	128.24	-4.029	<.0001
	SD	21.24	18.91			21.50	21.64		

Table 3. Previous studies on pressure mapping of backboard interface pressures.

Author, Year	Sensing System	Backboard Interface Pressure (mmHg)	Backboard Interface Pressure With Additional Support Surface Added (mmHg)				
			Air	Vacuum	Foam	Soft-layered	Gel
Lovell et al., 1994	Talley	147.3	—	36.7	115.5	—	—
Cordell et al., 1995	Talley	145.5	48.5	—	—	—	—
Keller et al., 2005	XSENSOR	174.9	—	165.6	118.0	—	—
Sheerin et al., 2007	Electropneumatic	154.33	76.0	70.67	—	—	—
Hemmes et al., 2010	XSENSOR	—	—	—	—	—	—
Nemunaitis et al., 2015 (present study)	FSA	258.19	—	—	—	—	187.87

factors play a role including individual characteristics, injury type, comorbidities, and physiologic factors (Bouten, Knight, Lee, & Bader, 2001; Breuls, Bouten, Oomens, Bader, & Baaijens, 2003; Gawlitta et al., 2007; Loerakker et al., 2010; Reenalda, Jannink, Nederhand, & IJzerman, 2009; Stekelenburg, Oomens, Strijkers, Nicolay, & Bader, 2006; Tsuji, Ichioka, Sekiya, & Nakatsuka, 2005).

Pressure ulcers are defined by the U.S. National Pressure Ulcer Advisory Panel as “a pressure-related injury to subcutaneous tissue under intact skin” (Black et al., 2007). Historically, the capillary-closing pressure of 32 mmHg was felt to be the threshold for tissue damage (Landis, 1930). Husain noted muscle tissue was more susceptible to injury following local pressure than skin tissue (Husain, 1953). Kosiak demonstrated cell death after the application of pressure at 60 mmHg in dogs for one hour (Kosiak, 1959). Notably, superficial ulceration of the skin at the test site did not appear until a mean of 4.5 days after pressure application. Daniel, Priest, and Wheatley demonstrated that capillary blood flow in muscle tissue is more sensitive to external pressures than the skin for both, high pressure/short duration and low pressure/long duration mechanical loads (Daniel, Priest,

& Wheatley, 1981). Mawson et al. evaluated the association of immobilization and the evolution of pressure ulcers in a 30-day post-injury period in patients with spinal cord injury (Mawson et al., 1988). In this study group, 59% developed some type of pressure ulcer, with 57% developing a pressure ulcer in the sacral area and 61% developing a pressure ulcer in the first six days ($p = .01$). Furthermore, the correlation between the increased incidence of pressure ulcers in the 30 days following injury and time on the spine board was no longer significant ($p = .09$). This led the authors to suspect that prolonged immobilization on the spine board is associated with early pressure ulcer formation. This again emphasizes the importance of limiting spine board pressures, as once the damage occurs in the deep tissues, the emergence of the ulcer may take several days.

There were several limitations in our study. First, our sample size of 37 subjects is small, and the data from the study involved normal subjects, which limits one’s conclusion when trying to extrapolate to injured or paralyzed patients. Also the knowledge gained by looking at the variation in individual characteristics on spine board pressures and sensing area are further limited by low

subject numbers. Secondly, the FSA Pressure Mapping System had upper level measurement limits in pressure of 500 mmHg, and several subjects peaked out near the maximum. Finally, comparative studies were not available using similar pressure mapping systems, pressure-relieving spine board overlays, or spine board types.

Conclusion

Significant mean peak interface pressures are generated when an individual is strapped to a spine board in the supine position. The mean peak interface pressures generated when strapped to a spine board can be reduced with the addition of a gel PDL. The mean peak sacral interface pressure generated in all subjects was significantly higher than minimum pressures noted by others to cause irreversible tissue damage. Adding a gel PDL to the spine board decreased overall mean peak sacral pressures by 27% and increased mean sensing area by 21%. Variation in individual characteristics may have an impact on sacral pressures and sensing area while strapped to the backboard. Nonetheless, pressures remained extremely high and well above the capillary closing pressure of 32 mmHg. As a preventive measure, we recommend that the spine board be redesigned to include a gel PDL along with shortening the time spent on the spine board, so as to reduce pressures that frequently lead to the formation of sacral pressure ulcers. This additional measure can potentially curb medical cost and improve the overall quality of life of trauma patients.

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References

- Akins, J. S., Karg, P. E., & Brienza, D. M. (2011). Interface shear and pressure characteristics of wheelchair seat cushions. *Journal of Rehabilitation Research & Development*, 48, 225–234.
- American College of Surgeons Committee on Trauma. (2008). *Advanced trauma life support for doctors, Student course manual* (8th ed.). Chicago, IL: American College of Surgeons.
- Black, J., Baharestani, M., Cuddigan, J., Dörner, B., Edsberg, L., Langemo, D., Posthauer, M.E., Ratliff, C., Taler, G., National Pressure Ulcer Advisory Panel. (2007). National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system. *Urologic Nursing*, 27, 144–50.
- Bouten, C. V., Knight, M. M., Lee, D. A., & Bader, D. L. (2001). Compressive deformation and damage of muscle cell subpopulations in a model system. *Annals of Biomedical Engineering*, 29, 153–163.
- Breuls, R. G., Bouten, C. V., Oomens, C. W., Bader, D. L., & Baaijens, F. P. (2003). Compression induced cell damage in engineered muscle tissue: An in vitro model to study pressure ulcer aetiology. *Annals of Biomedical Engineering*, 31, 1357–1364.
- Brienza, D. M., & Geyer, M. J. (2000). Understanding support surface technologies. *Advances in Skin & Wound Care*, 13, 237–244.
- Brienza, D. M., & Geyer, M. J. (2005). Using support surfaces to manage tissue integrity. *Advances in Skin & Wound Care*, 18, 151–157.
- Chan, D., Goldberg, R., Tascone, A., Harmon, S., & Chan, L. (1994). The effect of spinal immobilization on healthy volunteers. *Annals of Emergency Medicine*, 23, 48–51.
- Chan, D., Goldberg, R. M., Mason, J., & Chan, L. (1996). Backboard versus mattress splint immobilization: A comparison of symptoms generated. *Journal of Emergency Medicine*, 14, 293–298.
- Chen, D., Apple, D., Hudson, L., & Bode, R. (1999). Medical complications during acute rehabilitation following spinal cord injury-current experience of the Model Systems. *Archives of Physical Medicine & Rehabilitation*, 80, 1397–401.
- Chen, D., Apple, D., Hudson, L., & Bode, R. (1999). Medical complications during acute rehabilitation following spinal cord injury-current experience of the Model Systems. *Archives of Physical Medicine & Rehabilitation*, 80, 1397–1401.
- Cordell, W. H., Ayers, J. A., Olinger, M. L., Nelson, D. R., Hollingsworth, J. C., & Barney, R. N. (1996). Duration of immobilization and pain experienced on rigid spine boards. *Annals of Emergency Medicine*, 27, 147–148 (abstract).
- Cordell, W. H., Hollingsworth, J. C., Olinger, M. L., Stroman, S. J., & Nelson, D. R. (1995). Pain and tissue-interface pressures during spine-board immobilization. *Annals of Emergency Medicine*, 26, 31–36.
- Daniel, R., Priest, D., & Wheatley, D. (1981). Etiologic factors in pressure sores: An experimental model. *Archives of Physical Medicine & Rehabilitation*, 62, 492–498.
- Engsberg, J. R., Standeven, J. W., Shurtleff, T. L., Eggars, J. L., Shafer, J. S., & Naunheim, R. S. (2013). Cervical spine motion during extrication. *The Journal of Emergency Medicine*, 44, 122–127.
- Federal Registry. (2008). *Proposed changes to the hospital inpatient prospective payment systems and fiscal year 2009*. Retrieved January 6, 2014, from <http://www.gpo.gov/fdsys/pkg/FR-2008-04-30/pdf/08-1135.pdf>
- Ferguson-Pell, M., & Cardi, M. D. (1993). Prototype development and comparative evaluation of wheelchair pressure mapping system. *Assistive Technology*, 5, 78–91.
- Gawlitta, D., Li, W., Oomens, C. W., Baaijens, F. P., Bader, D. L., & Bouten, C. V. (2007). The relative contributions of compression and hypoxia to development of muscle tissue damage: An in vitro study. *Annals of Biomedical Engineering*, 35, 273–284.
- Goossens, R. H. M. (2007). A short history of progression of research into seating and postural support. *Disability and Rehabilitation: Assistive Technology*, 2, 249–254.
- Hadley, M. N., Walters, B. C., Grabb, P. A., Oyesiku, N. M., Przybylski, G. J., Resnick, D. K., . . . Mielke, D. H. (2002). Guidelines for the management of acute cervical spine and spinal cord injuries. *Clinical Neurosurgery*, 49, 407–498.
- Healthcare Cost and Utilization Project. (2008). *Statistical brief #64, Hospitalizations related to pressure ulcers among adults 18 years and older, 2006*. Retrieved January 6, 2014, from <http://www.hcup-us.ahrq.gov/reports/statbriefs/sb64.jsp>
- Hemmes, B., Poeze, M., & Brink, P. R. (2010). Reduced tissue-interface pressure and increased comfort on a newly developed soft-layered long spineboard. *The Journal of Trauma*, 68, 593–598.
- Husain, T. (1953). An experimental study of some pressure effects on tissues, with reference to the bed-sore problem. *The Journal of Pathology and Bacteriology*, 66, 347–358.
- Keller, B. P., Lubbert, P. H., Keller, E., & Leenen, L. P. (2005). Tissue-interface pressures on three different support-surfaces for trauma patients. *Injury*, 36, 946–948.
- Kosiak, M. (1959). Etiology and pathology of ischemic ulcers. *Archives of Physical Medicine & Rehabilitation*, 40, 62–69.
- Kosiak, M. (1961). Etiology of decubitus ulcers. *Archives of Physical Medicine & Rehabilitation*, 39, 19–29.
- Krapfl, L., & Mackey, D. (2008). Medicare changes to the hospital inpatient prospective payment systems: Commentary on the implications for

- the hospital-based wound care. *Journal of Wound Ostomy Continence Nursing*, 35, 61–62.
- Landis, E. M. (1930). Micro-injection studies of capillary blood pressure in human skin. *Heart*, 15, 209–228.
- Lerner, E. B., & Moscatti, R. (2000). Duration of patient immobilization in the ED. *The American Journal of Emergency Medicine*, 18, 28–30.
- Linares, H. A., Mawson, A. R., Suarez, E., & Biundo, J. J. (1987). Association between pressure sores and immobilization in the immediate post-injury period. *Orthopedics*, 10, 571–573.
- Loerakker S., Stekelenburg A., Strijkers G. J., Rijpkema J. J., Baaijens F. P., Bader D. L., . . . Oomens C. W. (2010). Temporal effects of mechanical loading on deformation-induced damage in skeletal muscle tissue. *Annals of Biomedical Engineering*, 38(8):2577–2587.
- Lovell, M. E., & Evans, J. H. (1994). A comparison of the spinal board and the vacuum stretcher, spinal stability and interface pressure. *Injury*, 25, 179–180.
- Lyder, C. H., Shannon, R., Empleo-Frazier, O., McGehee, D., & White, C. (2002). A comprehensive program to prevent pressure ulcers in long-term care: exploring costs and outcomes. *Ostomy/Wound Management*, 48, 52–62.
- Mawson, A. R., Biundo, J. J., Jr., Neville, P., Linares, H. A., Winchester, Y., & Lopez, A. (1988). Risk factors for early occurring pressure ulcers following spinal cord injury. *American Journal of Physical Medicine & Rehabilitation*, 67, 123–127.
- National Spinal Cord Injury Statistical Center, University of Alabama at Birmingham. (2006). *Annual statistical report: 2006*. Birmingham, AL: National Spinal Cord Injury Statistical Center.
- Oomens, C. W., Zenhorst, W., Broek, M., Hemmes, B., Poeze, M., Brink, P. R., & Bader, D. L. (2013). A numerical study to analyse the risk for pressure ulcer development on a spine board. *Clinical Biomechanics (Bristol, Avon)*, 28, 736–742.
- Reenalda, J., Jannink, M., Nederhand, M., & IJzerman, M. (2009). Clinical use of interface pressure to predict pressure ulcer development: A systematic review. *Assistive Technology*, 21, 76–85.
- Sheerin, F., & de Frein, R. (2007). The occipital and sacral pressures experienced by healthy volunteers under spinal immobilization: A trial of three surfaces. *Journal of Emergency Nursing*, 33, 447–450.
- Stekelenburg, A., Oomens, C. W., Strijkers, G. J., Nicolay, K., & Bader, D. L. (2006). Compression-induced deep tissue injury examined with magnetic resonance imaging and histology. *Journal of Applied Physiology*, 100, 1946–1954.
- Tsuji, S., Ichioka, S., Sekiya, N., & Nakatsuka, T. (2005). Analysis of ischemia-reperfusion injury in a microcirculatory model of pressure ulcers. *Wound Repair and Regeneration*, 13, 209–215.
- Yeung, J. H., Cheung, N. K., Graham, C. A., & Rainer, T. H. (2006). Reduced time on the spinal board-effects of guidelines and education for emergency department staff. *Injury*, 37, 53–56.