33rd ISS International Seating Symposium

Be Empowered

March 2-4, 2017

Department of Rehabilitation Science and Technology
Continuing Education
School of Health and Rehabilitation Sciences
University of Pittsburgh

Course Director: Mark R. Schmeler, PhD, OTR/L, ATP

In Collaboration with:

- Sunny Hill Health Centre for Children
- European Seating Symposium
- Latin American Seating Symposium
- Asia Pacific Region Seating Symposium
- International Society of Wheelchair Professionals

IC29: What Happens When You Sit? Explaining Seated Buttocks Deformation.

Sharon Eve Sonenblum, PhD
Stephen H. Sprigle, PhD, PT

Introduction

Over the past decade, pressure injury research has implicated tissue deformation in pressure injury development. Pressure injuries develop when external loads deform internal tissues, leading to a series of pathophysiological responses and eventually cell death.

To date, models of pressure injury etiology focus on three areas: 1) direct cell damage from prolonged deformation, 2) (deformation-induced) ischaemia of soft tissues, and 3) a (deformation-induced) disruption in the equilibrium in the lymphatic system [1, 2]. When sitting, the external loads often measured with interface pressure mapping are transmitted through the body to internal tissues, which respond by experiencing stress and deformation of their own.

The Role of Tissue Deformation in Pressure Injury Development

Investigations of tissue response to loading with respect to pressure injury development have focused most on muscle tissue. Presumably, this is because of the assumption that muscle is the tissue experiencing greatest loads under the ischial tuberosity, although that assumption has been called into question by our research [3, 4]. In 2003, Breuls et al [5] deformed simulated muscle tissue and found that the dead cells were evenly distributed beneath the indenter. Because damage resulting from oxygen deprivation would be expected to unevenly distributed, with more damage occurring farther from the undeformed tissue, this study provided early evidence of direct deformation damage. Stekelenburg et al expanded on this work in 2006 [6] by pushing on a rat's hind leg for two hours. They found significant changes in the tissue over the hours following, including disorganization of internal structure of muscle fiber, an extensive inflammatory response, and large necrotic regions. Another study in 2007 tested the hypothesis that direct deformation damage exists, and did so by testing strained tissue under hypoxic and normoxic conditions [7]. Two key findings were that tissue damage occurs in presence of normal oxygenation and that direct deformation damage is faster than ischaemic damage.

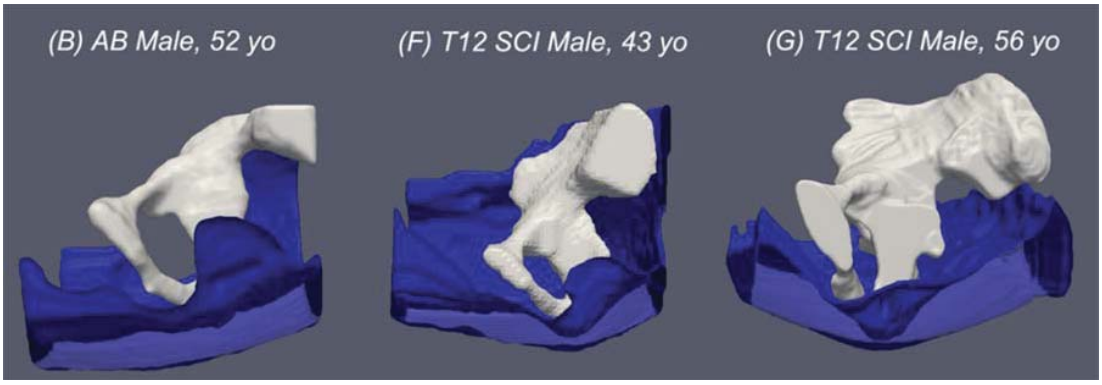
Fewer studies have investigated skin and adipose responses to deformation, although interest in these tissues is growing slowly. In an extensive review of pressure ulcer tissue histology, Dr. Edsberg [8] described skin around a stage II pressure injury and observed disruption of dermal papillae, densely packed collagen, necrosis of skin appendages, and the presence of inflammatory cells. In 2013, Stojadinovic, O., et al. [9] studied young and aged skin and found they responded differently to loading. Aged skin experienced changes to collagen alignment and subepidermal separation that were not present in young skin.

The differences in aged skin demonstrate how a common risk factor such as age impacts the mechanical structure and properties of the tissue. These changes in mechanical properties modify the tissue's response to loading, providing an explanation for how and why risk factors (such as age in this case) actually increase risk.
The spectrum of risk that exists in high-risk populations such as wheelchair users can be stratified in large part by an individual's Biomechanical Risk, or the intrinsic likelihood of their buttocks tissue to deform in response to loading. Characteristics described above, such as age and BMI, change an individual's Biomechanical Risk, as do things like diagnosis, smoking, and hydration. Unfortunately, our limited understanding of the effects of many clinical factors on Biomechanical Risk restricts our options for identifying the highest risk individuals. As a result, it is difficult to personalize interventions that would respond directly to individuals' Biomechanical Risk.

Differences in Biomechanical Risk

Presented below is an illustration of the adipose tissue (inferior and superior surfaces) of the seated buttocks of 3 similarly aged men with different levels of risk (Figure 1) [4]. Subject G experienced complex adipose deformations, particularly compared with Subject A whose adipose deformed rather uniformly. In the absence of any other information, this clearly illustrates an at-risk buttocks, which is consistent with his history of recurrent pressure injuries on the contralateral side.

Figure 1. Reproduced from [4]. Renderings of the subcutaneous adipose tissue near the IT when seated on foam.

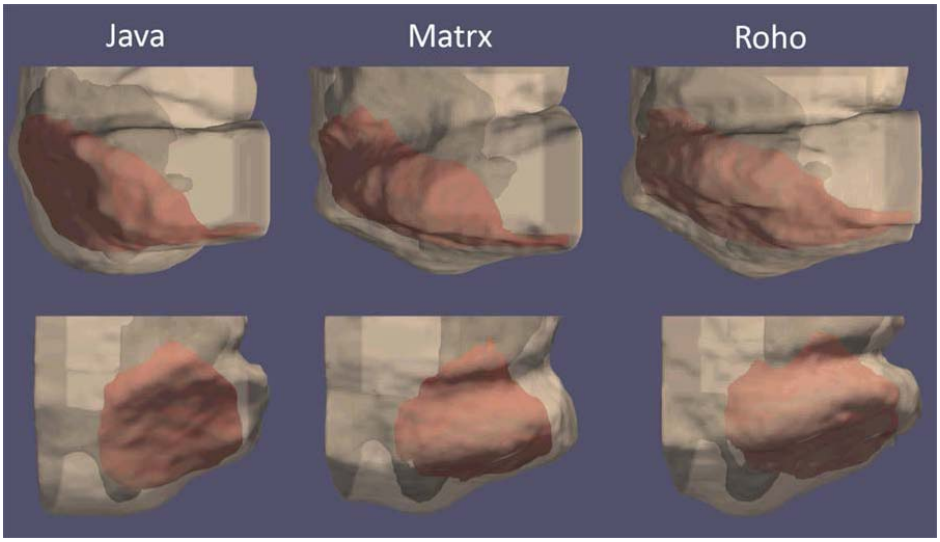


Differences Across Sitting Surfaces

Shape Compliance is defined as the ability of a cushion to support the buttocks with minimal buttocks deformation. It can be considered a metric of cushion performance. Shape Compliance has yet to be measured on human buttocks, but preliminary investigations into the loaded buttocks (specifically highly atrophied buttocks) on commercial wheelchair cushions are currently undergoing analysis. Illustrated below (Figure 2) are coronal and sagittal views of an individual's buttocks seated on 3 different surfaces: an orthotic based offloading cushion (Java, Ride Designs), a pressure redistribution cushion made with contoured foam (Matrx, Invacare), and a pressure redistribution cushion that uses air flotation (Roho, Permobil). The pelvis is visible through the semitransparent skin rendering, as is the gluteus maximus in red. This research participant is a 44 year old man. He is 18 years post injury with a complete T5-6 level SCI. He presents with significant atrophy, very little tissue around the pelvis, and he has a history of pressure injuries, suggesting that he has a high biomechanical risk.

We can make a few observations from these images. First, this participant demonstrates a similar amount of tissue present beneath the peak of the ischial tuberosity for all surfaces. At the same time, the curvature of the buttocks in that region differs suggesting different tissue strain profiles. Second, the contact area and immersion is different on each cushion. Visible by the discontinuity in contour as you travel from the inferior to superior surfaces of the buttocks, the contact area on the Java is considerable in the posterior-lateral location. Immersion on the Roho was greater than that on the Matrx, suggesting that body weight was spread over a greater surface area. Third, we observed that the gluteus maximus does not wrap underneath the ischium in any condition presented below, but it is displaced more significantly in the superior and lateral directions when seated on the Matrx and Roho than on the Java. Given that the gluteus maximus does not wrap underneath the ischium, more attention must be paid to the adipose and connective tissue present under the ischium.

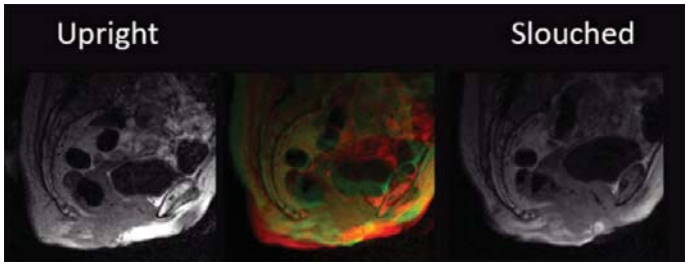
Figure 2. 3D renderings of the right side of the buttocks viewed laterally (top) and from the posterior (bottom). Adipose tissue is presented as semitransparent, with gluteus maximus in red and the pelvis in a dark gray.



Differences Across Postures

In a recent study of new wheelchair users, the majority reported scooting their buttocks forwards and sitting with a posterior pelvic tilt throughout the day (i.e., sacral sitting or slouching). Tissue deformation that occurs in response to slouched sitting is also important to investigate. Below is an example of the sacral area of a participant while seated on a foam contoured cushion (Embrace, Comfort Company). The participant started as upright as we could achieve and the slouched posture included approximately 8 degrees of posterior pelvic tilt from the upright posture. Significant changes to the tissue contour are visible in the slouched posture compared with the upright posture.

Figure 3. Sagittal MRI slices through the midline of the sacrum in the upright (left) and slouched (right) postures. The middle image shows upright (red) and slouched (green) presented overlapped so differences are visible.



Conclusion

Tissue deformation, including displacement of the muscle and compression of the adipose tissue, is present when seated on all surfaces. But different types of wheelchair cushions manage that deformation using different strategies and with differing results. Further investigation of the buttocks tissue during sitting as compared with unloaded upright sitting will allow us to better describe how different wheelchair cushions work and the impact of altering sitting posture on tissue deformation.

References

1. Bouten, C.V., C.W. Oomens, F.P. Baaijens, and D.L. Bader, The etiology of pressure ulcers: skin deep or muscle bound? Arch Phys Med Rehabil, 2003. 84(4): p. 616-9.
2. Oomens, C.W., D.L. Bader, S. Loerakker, and F. Baaijens, Pressure induced deep tissue injury explained. Ann Biomed Eng, 2015. 43(2): p. 297-305.
3. Sonenblum, S.E., S.H. Sprigle, J.M. Cathcart, and R.J. Winder, 3-dimensional buttocks response to sitting: a case report. J Tissue Viability, 2013. 22(1): p. 12-8.
4. Sonenblum, S.E., S.H. Sprigle, J.M. Cathcart, and R.J. Winder, 3D anatomy and deformation of the seated buttocks. J Tissue Viability, 2015. 24(2): p. 51-61.
5. Breuls, R.G., C.V. Bouten, C.W. Oomens, D.L. Bader, and F.P. Baaijens, Compression induced cell damage in engineered muscle tissue: an in vitro model to study pressure ulcer aetiology. Ann Biomed Eng, 2003. 31(11): p. 1357-64.
6. Stekelenburg, A., C.W. Oomens, G.J. Strijkers, K. Nicolay, and D.L. Bader, Compression-induced deep tissue injury examined with magnetic resonance imaging and histology. J Appl Physiol, 2006. 100(6): p. 1946-54.
7. Gawlitta, D., W. Li, C.W. Oomens, F.P. Baaijens, D.L. Bader, and C.V. Bouten, The relative contributions of compression and hypoxia to development of muscle tissue damage: an in vitro study. Ann Biomed Eng, 2007. 35(2): p. 273-84.
8. Edsberg, L.E., Pressure ulcer tissue histology: an appraisal of current knowledge. Ostomy Wound Manage, 2007. 53(10): p. 40-9.
9. Stojadinovic, O., J. Minkiewicz, A. Sawaya, J.W. Bourne, P. Torzilli, J.P. de Rivero Vaccari, W.D. Dietrich, R.W. Keane, and M. Tomic-Canic, Deep tissue injury in development of pressure ulcers: a decrease of inflammasome activation and changes in human skin morphology in response to aging and mechanical load. PLoS One, 2013. 8(8): p. e69223. PMCID: PMC3743891

Acknowledgements

This work was supported by generous donations from Comfort Company, Ride Designs, and Vicair. We also would like to acknowledge the support of Tom Hetzel from Ride Designs who assisted with the logistics of data collection and subject management during data collection. John Greenhalgh, PhD and Eric Stewart also provided instrumental support on MRI protocol development and data acquisition. The contents of this conference paper were also supported by a grant from the National Institute on Disability, Independent Living, and Rehabilitation Research (NIDILRR grant number 90IF0120).