

Original Research Paper

Pressure Ulcer Prevention: Optimizing a Temporally Redistributing Support Interface

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Abstract: One of the most common complications from long-term wheelchair use or bed rest is pressure ulcers. Pressure ulcers have significant morbidity and are associated with high mortality. Prolonged sitting can cause high pressures in the skin and subcutaneous tissue which can lead to local ischemia and breakdown of skin. Rapid relief of pressure prevents ulcer formation. One prevention strategy is to change the temporal distribution of pressure at the interface between user and surface so that no one area is subjected to high pressures for long periods of time. While there are several dynamic interfaces in use currently, there is no definitive evidence of enhanced pressure ulcer prevention with their use. The purpose of this research was to parametrically evaluate interface array sizes, shapes and patterns for dynamic support surfaces to optimize pressure redistribution to prevent pressure ulcers. Finite element analyses, anatomical phantom deep tissue pressure measurements and interface pressure mapping were used to test various support geometries and sizes and different array spacing and patterns. Results indicate that modulating pressure in an array of supports that are equally spaced is not effective. Only interrupted pattern arrays resulted in sufficient pressure reduction. These data suggest that dynamic surface supports can be optimized based on the geometry and size of the individual supports and the pattern of the array to further reduce the likelihood of pressure ulcer formation.

Keywords: Pressure Ulcer, Pressure Sore, Wheelchair, Seating

Introduction

According to the World Health Organization, there are more than 70 million wheelchair users worldwide. For users with impaired ability to reposition themselves, one of the most common complications from long-term wheelchair use or bed rest is pressure ulcers (Coleman *et al.*, 2013). In 2008, the World Health Organization identified pressure ulcers as one of the top 20 patient safety issues worldwide (WHO, 2008) and in that same year, pressure ulcers were recognized as the most common medical error in the United States (Van Den Bos *et al.*, 2011).

The prevalence of pressure ulcers is more than 3 million in the US and estimated to be more than 7.4 million worldwide (Sen *et al.*, 2009) with higher

prevalence in developing countries (WHO, 2008). Among inpatients, pressure ulcer incidence is as high as 38% depending on nature of injury while in long term care settings, the incidence is reported at 24% (Xakellis, 1992; Cuddigan *et al.*, 2001). Up to 66% of paraplegics will develop pressure ulcers (Richardson and Meyer, 1981).

Pressure ulcers have significant morbidity and are associated with high mortality (Redelings *et al.*, 2005; Khor *et al.*, 2014). In underdeveloped healthcare systems, pressure sores are one of the leading cause of death among individuals with disabilities. Without proper treatment, pressure ulcers can lead to cellulitis, osteomyelitis, sepsis and death (WHO, 2008). Average length of hospital stay for treatment of pressure ulcers is 13 days (Sen *et al.*, 2009) and healing can take up to several years (Stockton and Rithalia, 2007). More than

\$11 billion is spent annually on pressure ulcer treatments in the US (Sen *et al.*, 2009) and the cost of treatment for individual pressure ulcers averages from \$8,730 to \$129,248 depending on how advanced the ulcer is (Van Den Bos *et al.*, 2011; Sen *et al.*, 2009; Palfreyman and Stone, 2015; Lyder, 2003). Prevention is a more cost effective option (Sullivan and Schoelles, 2013).

The physiology of pressure ulcer formation is complex (Reuler and Cooney, 1981). Pressures ulcers are a local breakdown of skin and underlying tissue caused by unrelieved pressure (Brienza *et al.*, 2001; Grey *et al.*, 1974). One of the most significant contributing factors is ischemia from prolonged pressure which occludes vessels in the skin and subcutaneous tissues (Stekelenburg, 2005). Healthy capillary pressure is approximately 32 mmHg. Prolonged sitting can cause high pressures in the skin and subcutaneous tissue in areas where body weight is concentrated. If pressure from sitting obstructs capillary blood flow, ischemia results and pressure ulcers can develop within 2 to 6 hours (Lyder, 2003; Stekelenburg, 2005). If peak pressures exceed 60 mmHg, the incidence of pressure ulcers increases (Conine *et al.*, 1993). Pressure ulcers are most common superficial to the sacrum, coccyx and ischial tuberosities (Cuddigan *et al.*, 2001; Richardson and Meyer, 1981).

Prolonged high pressures also result in the accumulation of toxic metabolites which contribute to tissue necrosis even after reperfusion (Stekelenburg, 2005; Krouskop *et al.*, 1978). Temperature, moisture, skin friction and shear forces also contribute to skin breakdown and pressure ulcer formation (Xakellis, 1992; Grey *et al.*, 1974; Stekelenburg, 2005; Reichel, 1958). Pressure ulcers can also initiate in the deep tissues due to sustained mechanical loading of muscle (Stekelenburg, 2005).

There are two parameters that are significant in preventing pressure ulcer formation: Pressure magnitude and duration. Rapid relief of pressure, even at higher pressure magnitude (70 mmHg) prevents ulcer formation (Kosiak, 1959). Tissues exposed to pressures up to 200 mmHg for 1.5 h showed minimal pathologic changes (Dinsdale, 1974). When pressure of short duration is relieved, tissues demonstrate reactive hyperaemia, reflecting increased blood flow to the area (Grey *et al.*, 1974). As shown in Fig. 1, the relationship between pressure magnitude, pressure duration and risk of pressure ulcer formation has been characterized. Individuals on long term bed rest who are repositioned at least every 2 h have a reduced incidence of pressure ulcer formation (Xakellis, 1992). Best practice for long term wheelchair users is to shift weight approximately every 15 min (Xakellis, 1992; Grey *et al.*, 1974).

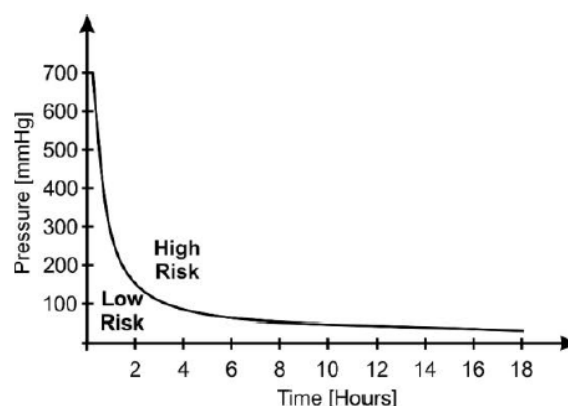


Fig. 1. The relationship between pressure magnitude, pressure duration and risk of pressure ulcer formation indicates that tissues are tolerant of high magnitude pressures if they are relieved quickly. Reproduced from the work of Stekelenburg (2005) and Sacks (1989)

Most current prevention strategies are based on reducing the magnitude of pressure everywhere at the interface between the user and the seat (Xakellis, 1992; Taylor, 2003). This strategy generally involves static cushions some of which are contoured or customized to the user. Static cushions are relatively inexpensive and easy to use and are commonly utilized in developing healthcare systems. However, static cushions do not always reduce pressure sufficiently near bony prominences (Stockton and Rithalia, 2007). They also become ineffective once they “bottom out”, fatigue, or in the case of customized cushions, if the user’s BMI changes (Stockton *et al.*, 2009; Hollington *et al.*, 2014). Gel and air filled cushions mold to the user to increase surface area and reduce contact pressure. However, these cushions can affect user balance, must be constantly maintained for correct function and will leak if punctured (Stockton *et al.*, 2009; Stockton and Rithalia, 2009).

Alternative prevention strategies are based on changing the temporal distribution of pressure so that no one area is subjected to high pressures for long periods any length of time. The interface between the user and the surface is modulated temporally. However, all existing dynamic support surfaces are based on regular patterns of supports with no optimization of support array, size, shape, etc. While there are several dynamic interfaces in use currently, there is no definitive evidence of enhanced pressure ulcer prevention with their use (Reddy *et al.*, 2006; McInnes *et al.*, 2011; Qaseem *et al.*, 2015). Furthermore, the support array sizes, shapes and patterns for dynamic support surfaces has not been optimized. The purpose of this research was to parametrically evaluate various support array sizes, shapes and patterns to determine their effectiveness at optimal pressure redistribution for a dynamic wheelchair interface for prevention of pressure ulcers.

Materials and Methods

Finite Element Analyses (FEA), anatomical phantom deep tissue pressure measurements and interface pressure mapping were used to test for differences between various seating support interfaces to determine which combination of parameters (support geometry and size; array spacing and pattern) would result in maximum relief of pressure during dynamic modulation of the supports. The goal was to identify an interface support array that could be modulated so that at any time, half of the array is supporting the user while the other half allows reperfusion of tissues (pressure less than 32 mmHg). To be clinically viable, when the dynamic support array is modulated, the roles switch so no tissue experiences pressures in excess of 32 mmHg continuously.

Finite Element Analysis

A previously validated finite element model from Oomens *et al.* (2003) was adapted to predict the effect of support geometries on superficial and deep tissue pressures. An image of an MRI scan of a seated buttocks from a 29 year old female from previously published work (Sopher *et al.*, 2010) was imported into SolidWorks (Dassault Systèmes SolidWorks Corp.; Waltham, MA). The boundaries between bone, fat and muscle tissue on the cross sectional image were traced with splines to obtain a two dimensional model for finite element analysis as shown in Fig. 2.

The model geometry was imported into MARC Mentat (MSC Corp.; Newport Beach, CA) and meshed using quadrilateral elements. The fat and muscle tissues were modeled as materials with non-linear mechanical properties using an Ogden model which describes the mechanical behavior of highly elastic incompressible

“rubberlike” solids (Ogden, 1972) using the strain energy function shown in Equation 1:

$$W = \sum_{n=1}^N \frac{\mu_n}{\alpha_n} (\lambda_1^{\alpha_n} + \lambda_2^{\alpha_n} + \lambda_3^{\alpha_n} - 3) \quad (1)$$

The Ogden model represents material mechanical properties with λ_i denoting principal strains while μ_n and α_n are material parameters determined from empirical testing. The constant μ is analogous to modulus, while n and α are used to describe the power of the strain energy density curve. Previously published finite element models of seated tissue pressures have assumed $N = 1$ and the material parameters shown in Table 1 (Oomens *et al.*, 2003).

In order to reduce computational load, we modeled the ischial tuberosities as rigid bodies due to their significantly higher stiffness than the surrounding tissues. The finite element model was utilized to evaluate the effect on tissue pressure within the buttocks when seated on a flat interface and when seated on a support array with hemispherical supports 15.75 mm in diameter spaced 17.5 mm between arc centers. The supports were modeled as rigid bodies with a coefficient of friction of $\mu_f = 0.4$ with the buttocks. To simulate the stresses and strains due to sitting, a 14.6 mm downward displacement was applied to the ischial tuberosity (Sopher *et al.*, 2010). From the model, we determined the minimum superficial tissue pressures between supports, maximum superficial tissue pressure and deep tissue pressure.

Table 1. Parameters of ogden model materials

Tissue type	Modulus μ (MPa)	Exponent α
Fat	0.010	-5
Muscle	0.003	-30

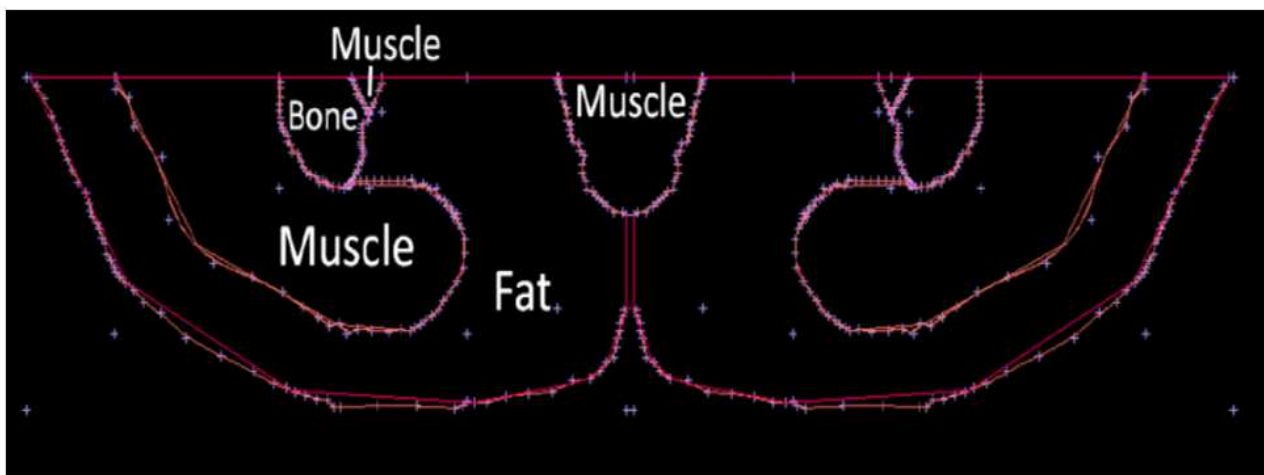


Fig. 2. The cross sectional model of the buttocks is comprised of discrete regions representing anatomical structures of bone, muscle and fat

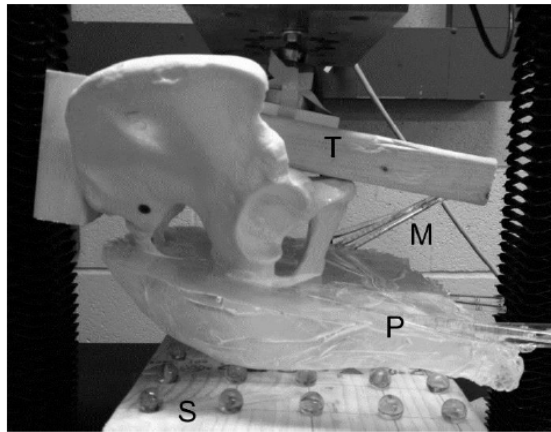


Fig. 3. An anatomical phantom (P) was fabricated to measure the deep pressures adjacent to the ischial tuberosities and the sacrum using manometers (M) for different support array configurations (S) while being loaded in a mechanical testing machines (T)

Table 2. Test interface support array sizes and spacings

Support shape	Support diameter (mm)	Support spacing center to center (mm)
Cylindrical	22.0	7.8
Cylindrical	22.0	25.4
Cylindrical	26.0	7.8
Cylindrical	26.0	25.4
Cylindrical	34.0	7.8
Cylindrical	34.0	25.4
Spherical	16.2	6.4
Spherical	16.2	19.0
Spherical	16.2	25.4
Spherical	16.2	51.0

Anatomical Phantom Testing

An anatomical phantom of a buttocks and pelvis was fabricated to enable experimental measurement of deep tissue pressures (Linder-Ganz *et al.*, 2009). The anatomical phantom was made from Sylgard 184 silicone (Dow Corning; Midland, MI) which was poured into a plaster mold of human buttocks, lower back and legs. A polyurethane foam pelvis replica (Sawbones; Vashon Island, WA) was suspended within the mold in the correct anatomic location with a 3 cm gap between the ischial tuberosities and the surface of the mold as shown in Fig. 3. The silicone was allowed to polymerize for four days. Sylgard 184 was chosen because of its similarities to soft tissue properties with an elastic modulus of 1.8 MPa and Poisson's ratio of 0.45 (Palchesko *et al.*, 2012). The pelvis was instrumented with three pressure manometers (Omega Engineering Inc., Norwalk, CT) on each ischial tuberosity and two on the sacrum to facilitate measurement of deep tissue pressures at these locations.

The phantom model was then placed on ten different interface support arrays as described in Table 2. The

support arrays varied in geometry (cylindrical, spherical), size and spacing. Cylinders were arranged longitudinally across the seat at specified spacing and spheres were arranged in a regular square grid at the designated spacing. The anatomic phantom was placed on each support array and loaded in axial compression up to 490 N with a Mechanical Testing machine (MTS Systems Corporation; Eden Prairie, MN). While loaded, the pressure at each deep tissue location under the ischial tuberosities and sacrum was recorded. Axial load was then removed and the support array was modulated (shifted anteriorly) by a distance of one half of the support spacing so that regions of the phantom surface which were previously unsupported were in contact with a support and regions previously supported were relieved of contact. For each support array, the phantom was loaded and pressures were measured five times ($n = 5$) both before and after modulating the array. Differences in deep tissue pressure as a result of modulating the seat support array were measured using ANOVA and Tukey's *post hoc* test with values deemed statistically significant when $\alpha \leq 0.05$.

Pressure Mapping

Pressure mapping was utilized to measure seat interface pressures as volunteers sat on different interface support arrays. A high resolution pressure mat (Tekscan, South Boston MA) was used to measure interface pressures during sitting on the support arrays described in Table 2 and 3. Prototype support arrays were made by arranging cylinders (longitudinally or axially) or spheres of different diameters in specified patterns. Initially, the axial cylinders were spaced in rows and columns. Within each row, the cylinders were separated by 3.15 mm (between edges) and adjacent rows were spaced as described in Table 3. Pressure data were collected during five replicates ($n = 5$) of sitting for each support array. Array patterns which facilitated pressures of less than 32 mmHg between supports were noted as potentially clinically viable.

Based on the results from the rectangular arrays, two additional "interrupted" array patterns were tested as shown in Fig. 4. These array patterns were designed to support a small circular or square region of tissue at its perimeter leaving it unsupported in the middle. When the array was modulated, the previously unsupported areas became supported and the previously supported areas became unsupported to allow the tissue to reperfuse.

Again, data were collected during five replicates ($n = 5$) of sitting for each support array. Array patterns which facilitated pressures of less than 32 mmHg between supports were noted as potentially clinically viable.

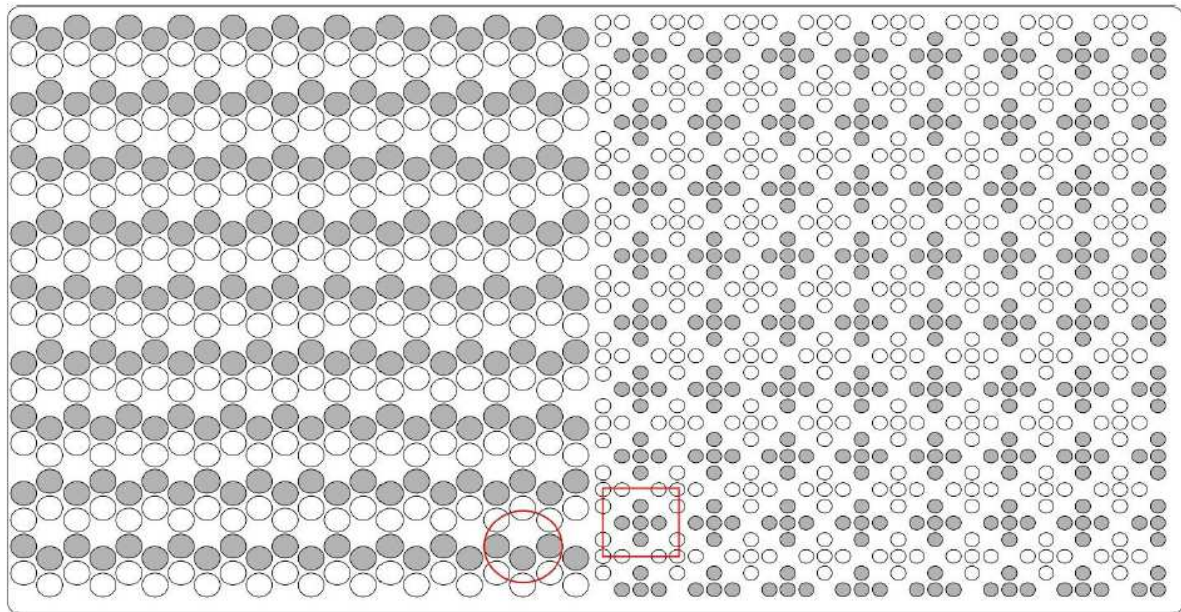


Fig. 4. The test arrays were constructed of dowels 6.35 mm in diameter arranged in an interrupted circle pattern (left) or interrupted box pattern (right). Dark cylinders provide support before modulating array and white cylinders provide support after modulating the array

Table 3. Interface support array sizes and spacings

Support diameter (mm)	Support spacing between rows (mm)
3.15	9.5
3.15	19.0
3.15	28.5
3.15	38.0
3.15	47.5
6.35	12.7
6.35	25.4
6.35	38.1
6.35	50.8
6.35	63.5
12.7	19.0
12.7	38.0
12.7	57.0
12.7	76.0
19	25.4
19	50.8
19	76.2

Results

Finite Element Analysis

The finite element model provided a representation of soft tissue deformation on a flat surface and one support array as shown in Fig. 5. Results indicated that maximum superficial tissue pressures occurred over the individual supports of the support array and maximum deep tissue pressures occurred at the ischial tuberosity as shown in Fig. 6. The mean and peak deep tissue pressure beneath the ischial tuberosity was approximately 7.5 and

22 mmHg, respectively. The mean pressure at the superficial tissue above the individual supports was approximately 12 mmHg with a peak pressure of 15 mmHg. The minimum pressure of superficial tissues located between the individual hemispherical supports was approximately 0.5 mmHg.

Anatomical Phantom Testing

Deep pressures measured during testing of each support array with the anatomical phantom are shown in Table 4. The magnitudes of pressure were consistently greatest at the center of the ischial tuberosities. When the axial load of 490 N was applied, the pressures increased by a mean 141.6 mmHg in the deep tissues. When the phantom was placed on an array of 22 mm diameter longitudinally oriented cylindrical supports, modulating the array resulted in a mean decrease in all pressures of 6.4 and 19.9 mmHg for supports spaced at 7.8 mm and 25.4 mm respectively. Similarly, when the phantom was placed on an array of 26 mm diameter longitudinally oriented cylindrical supports, modulating the array resulted in a mean decrease in all pressures of 13.5 and 24.4 mmHg for supports spaced at 7.8 and 25.4 mm, respectively. When placed on 34 mm diameter longitudinally oriented cylindrical supports, mean pressures decreased 9.5 and 13.8 mmHg for supports spaced at 7.8 and 25.4 mm, respectively. Using spherical supports, modulating the array resulted in a mean change in all pressures of 7.6, 19.5 and 18.4 mmHg when the spacing between supports was 6.35, 25.4 and 51 mm, respectively.

The change in magnitude of deep pressures for each location measured is shown in Table 4. While none of the support arrays tested with the anatomical phantom statistically significantly reduced pressure at all deep tissue locations, the trend was that pressures everywhere were

reduced when the support array was modulated. The support arrays which most effectively reduced pressure when they were modulated were the 26 mm cylinders spaced at 25.4 mm, the 16.2 mm diameter spheres spaced at 25.4 mm and the same spheres spaced at 51 mm.

Table 4. Deep pressure change measured in the anatomical phantom

Sensor location	22 mm cylinder 7.8 mm spacing	22 mm cylinder 25.4 mm spacing	26 mm cylinder 7.8 mm spacing	26 mm cylinder 25.4 mm spacing	34 mm cylinder 7.8 mm spacing	34 mm cylinder 25.4 mm spacing	16.2 mm sphere 6.2 mm spacing	16.2 mm sphere 25.4 mm spacing	16.2 mm sphere 51 mm spacing
Posterior left IT ^b	13.4* ^a	-25.8*	-2.1	-31.2*	7.2	11.3	-6.2	-48.6*	-48.6*
Central left IT	-4.1	27.9*	-21.7*	39.8*	19.6*	51.7*	-14.4*	31.0*	16.5*
Anterior left IT	0.0	-7.2	1.0	21.5*	-5.1	-8.3	15.5*	-48.6*	-7.2
Anterior right IT	-4.1	0.42	0.0	-44.3*	-15.5*	6.2	0.0	-2.1	-4.1
Central right IT	-21.7*	6.2	-41.3*	-8.9*	-20.7*	-1.0	3.1	-15.5*	32.1*
Posterior right IT	-5.17	-47.5*	34.1*	-23.1	4.1	-15.5*	2.1	34.1*	-17.5*
Left coccyx	4.1	35.1	7.3*	-12.4	-2.6	7.2	-9.3	-1.0	-2.1
Right coccyx	3.1	-1.0	-1.0	-14.0*	-2.7	-9.3	-10.3	15.5*	18.6*

a. *Denotes reductions in pressure which are statistically significant ($p < 0.05$)

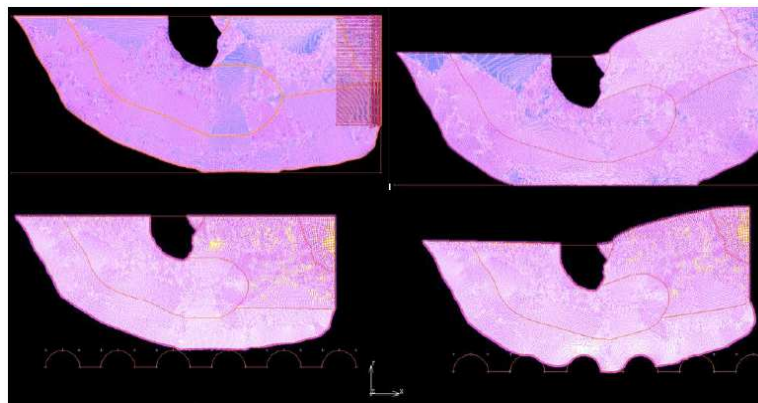


Fig. 5. The finite element model before (left) and after (right) ischial tuberosity displacement indicates how the soft tissues deform on a flat surface (top) and spherical support array (bottom)

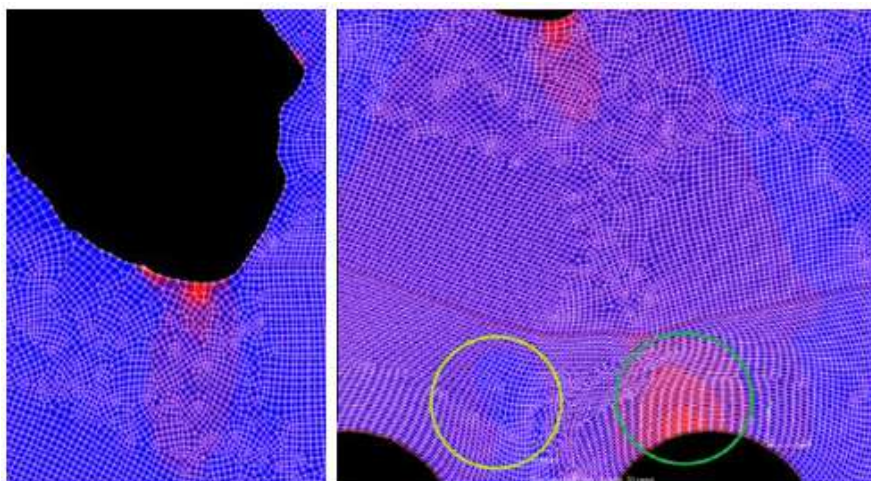


Fig. 6. A close up of the soft tissue adjacent to the ischial tuberosity (left) shows regions of peak pressure (red elements). A close up of the soft tissue over the hemispherical support (right) shows a region of maximum superficial tissue pressure (green circle) and minimum superficial tissue pressure (yellow circle) between supports

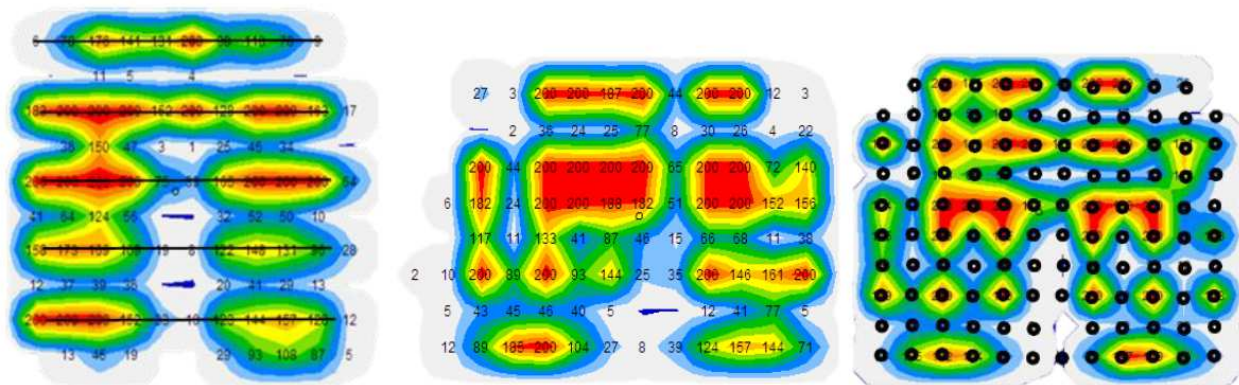


Fig. 7. Pressure maps of sitting on an array of longitudinally oriented cylinders (left), spheres (middle) and a regularly spaced array of axially oriented cylinders (right) indicated that tissue pressure is not localized to the area contacting the supports (indicated with black lines and circles) but also demonstrated hammocking. Colored areas show pressures which exceeded 32 mmHg

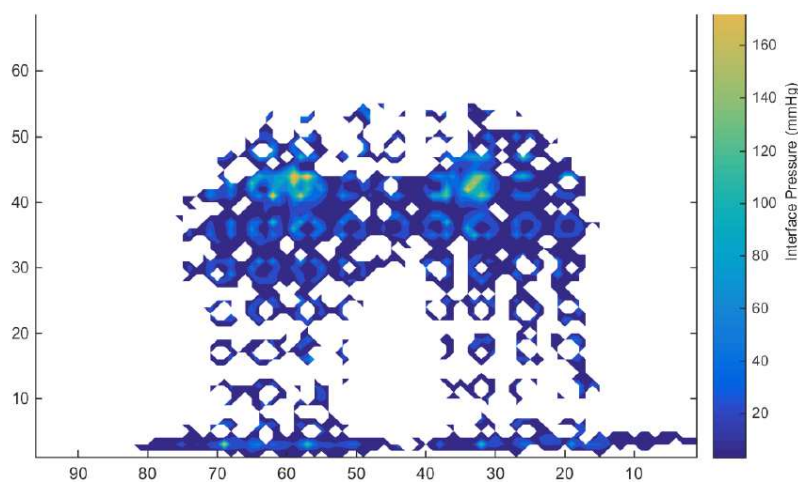


Fig. 8. High resolution pressure maps show that for the interrupted arrays pressure is localized to the perimeter of the support array, but in the unsupported regions the pressures are below 32 mmHg

Pressure Mapping

Pressure mapping provided a quantitative two-dimensional representation of pressure magnitude at the interface between the buttocks and support array. For each support array configuration tested, peak pressures were always located at the ischial tuberosities. As expected, pressures were highest at the location where the supports contacted the user and were lowest in the unsupported regions (the gaps between supports). The magnitude of pressure in the unsupported locations was analyzed to determine if pressures were below 32 mmHg - the magnitude at which reperfusion is likely to occur. In all regularly spaced arrays (rows and columns) of longitudinal cylinders, axial cylinders and spheres, there was hammocking, or high pressure *between* supports. In all cases of regular spaced arrays the pressure in the unsupported regions (between the supports) exceeded 32 mmHg in several locations (Fig. 7), indicating that the

support array would not allow reperfusion of the tissues in those regions. This was particularly prominent near the ischial tuberosities. Based on these initial results, we tested additional array configurations which included interrupted support patterns of axially oriented cylinders (Fig. 4). The interrupted support patterns enclose regions of tissue within their perimeter. Results from the interrupted arrays demonstrated that pressure is localized to the perimeter of the support array (the perimeter of the circles or squares), but in the unsupported regions (the area within the circle or square) the pressures are substantially lower and in most cases are below 32 mmHg everywhere, as shown in Fig. 8.

Discussion

In spite of decades of awareness of the pathomechanisms underlying pressure ulcer formation, the incidence continues to rise worldwide. The cost of

treatment has also increased substantially and is a sizeable burden on healthcare systems. New prevention recommendations include educating caregivers, enhancing nutrition and using dynamic support surfaces as interfaces (Qaseem *et al.*, 2015). The use of dynamic support surfaces purports to cause a reduction in pressure ulcer formation in high risk patients (Inma *et al.*, 1993). However, the effectiveness of existing dynamic support surfaces in reducing incidence of pressure ulcers remains controversial (Reddy *et al.*, 2006; McInnes *et al.*, 2011; Qaseem *et al.*, 2015).

There are additional challenges for wheelchair users. Current dynamic support surfaces employ electric pumps which alternate air or water pressures in a pad or fluidized bed. These are generally expensive, large and costly to operate (Lyder, 2003; Reddy *et al.*, 2006; McInnes *et al.*, 2011). Ideally, dynamic support systems modulate at 10-20 min intervals to allow reperfusion of tissues (Stockton and Rithalia, 2007). Existing dynamic support systems are hard to make transportable, difficult to adjust and difficult to maintain.

For wheelchair users who are able, self-repositioning every 15-30 min can be an effective means of preventing ischemia and pressure ulcer formation (Stockton *et al.*, 2009). However, compliance is low and so alternative preventive measures are needed (Van Den Bos *et al.*, 2011; White *et al.*, 1989). One potential shortcoming of existing dynamic support surfaces is that they are based on regular arrays of supports (i.e., rows and columns). Results from this study demonstrate that modulating pressure in an array of supports that are equally spaced is not effective in reducing pressure below 32 mmHg. Hammocking of tissues between supports results in high pressures even in areas that are unsupported.

In our parametric approach to testing various array configurations and support geometries, only the interrupted array of axially oriented cylinders eliminated the effects of hammocking. Whether longitudinally oriented cylinders, axial oriented cylinders, or spherical supports, neither the deep pressures nor the surface pressures were substantially reduced when the array was comprised of a regular pattern of supports.

Based on our pressure mapping, the interrupted arrays of circles, squares and rectangles resulted in pressure reduction below 32 mmHg when the support array was modulated. Tissue supported by the array demonstrated pressures in excess of 32 mmHg while those unsupported had pressures below 32 mmHg. These data suggest that dynamic surface supports can be optimized based on the geometry and size of the individual supports and the pattern of the array to further reduce the likelihood of pressure ulcer formation. The simple pattern of supports is also conducive to mechanical actuation and the small size and weight needed for a wheelchair interface. Further research should target additional optimization of

the array and demonstrate efficacy for reducing deep and superficial pressures.

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Author's Contributions

Joshua Peterson contributed: Experimental designs, prototype fabrication, data collection, analysis and manuscript preparation. Colleen P. Healey was responsible for: experimental design, data collection, analysis. Jacobus Visser contributed: Computational analysis, prototype fabrication, data analysis. Cameron Crombie was responsible for: Computational analysis, data analysis. Eric H. Ledet was the principal investigator and responsible for: Study design, data analysis, manuscript preparation.

Ethics

The authors have nothing to disclose.

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