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Exploring the effects of lateral pressure to the soft tissue of the buttocks during seating to preserve tissue perfusion

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1. Introduction

Pressure ulcers (PUs) are painful and debilitating wounds caused by the breakdown of tissue due to prolonged pressure or pressure in combination with shear [\[1\]](#page-6-0). They represent a major burden to patients, carers, and healthcare systems worldwide. PUs commonly occur in patients restricted to a bed or wheelchair, at bony prominences such as the sacrum and ischial tuberosities [[2](#page-6-0)]. It is estimated that every month between 1700 and 2000 patients develop at least one pressure ulcer in England, costing the NHS an estimated £3.8 million per day [[3](#page-6-0)]. Despite increased awareness and interventions to improve the efficiency of preventative strategies to reduce the burden of PUs, PU incidence in both the acute and community settings has remained unacceptably high [[4](#page-6-0)].

A primary strategy to prevent PUs includes the provision of pressure-

redistributing support surfaces (cushions and mattresses). Indeed, the NICE Guidelines state that adults in secondary care or at elevated risk of PUs in primary or community care should be considered for pressure redistributing devices [[5](#page-6-0)]. The efficacy of mattress systems has been the focus of several systematic reviews [[6,7\]](#page-6-0) and recent randomized control trials (RCTs) [\[8\]](#page-6-0). By contrast, studies exploring seating technologies have been very limited to date, with the few RCTs conducted demonstrating little difference between cushion designs [[9](#page-6-0)]. This disparity is observed also in clinical practice, for example in the management of high-risk patients, fewer than 50 % patients receive specialist chair equipment and a very small proportion of them receive care plans to prevent PUs whilst seated [\[10](#page-6-0)]. Advances in specialist support surfaces include the use of immersive and envelopment materials such as per-sonalised contoured foam, and dish-shaped cushions [[11,12\]](#page-6-0). Despite these advances, there is insufficient evidence to demonstrate the efficacy of high-tech cushions in reducing PU risk [\[7\]](#page-6-0). This stems from a focus on

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reducing peak pressures at the individual support surface interface and the challenging in assessing the complex nature in which individuals are at risk of developing PUs, which includes both intrinsic (age, comorbidities, history of PU) and extrinsic factors [\[13\]](#page-6-0). Indeed, it has been widely acknowledged that peak pressures poorly correlate to PU risk [[14\]](#page-6-0) current support surfaces may decrease peak pressures, it is less known whether this has a direct causal effect of reducing the risk of developing a PU [[15\]](#page-6-0). Further, a balance is required to provide an immersive cushion material, whilst supporting posture and mobility as they influence tissue health and recovery.

Several methods have been proposed to monitor the health of local tissues during periods of loading [\[16](#page-6-0)]. For example, transcutaneous gas tension of oxygen (TcPO₂) and carbon dioxide (TcPCO₂) have been hypothesized to be markers of tissue viability [[17\]](#page-6-0) and have been shown to provide ischemic thresholds during incremental tissue loading [\[18](#page-6-0)]. A decrease in oxygen and an increase in carbon dioxide is indicative of local tissue ischemia, with anaerobic cellular respiration changing tissue pH, leading to tissue damage and the initiation of PUs [\[19,20](#page-6-0)]. It has been shown that $TcPO₂$ decreases and $TcPCO₂$ increases when seated compared to baseline levels obtained when standing [\[21](#page-6-0)–23]. These observations imply that oximetry may be used as a surrogate marker or increased likelihood of an ischaemic response in local skin and sub-dermal tissues due to oxygen depletion and carbon dioxide accumulation.

Computational studies of the seated patient have demonstrated that tissue distress occurs at the bone-muscle interface, at the ischial tuberosities (ITs), rather than at the more superficial body regions of fat and skin [\[24](#page-6-0)–26]. They have also demonstrated that the introduction of cushions of variable structural behaviours although able to reduce pressures at the cushion-skin interface, they offer little by way of reducing the peak stresses near the ITs in the seated patient. However, many of these studies show the bony prominence in direct contact with a layer of muscle, which some studies show may not be anatomically correct for seated postures [[27\]](#page-6-0). We proposed recently an alternative approach to reducing tissue distress close to the ITs, namely by applying pressure laterally to the seated patient. We showed computationally that this approach decreases tissue lateral bulge and therefore tissue distortion (shape alteration of the buttocks) and, in turn, peak predicted stress values near the ITs [\[26\]](#page-6-0). Here, we introduce a rudimentary device that can apply lateral pressure to the seated patient and use it to conduct a study with healthy participants aiming to determine the effect of lateral pressure application on tissue perfusion in-vivo. The scientific proof of principle is quantified using oximetry data over the ITs as a surrogate marker of local tissue ischemia, which is one of the primary aetiological processes in the development of PUs [[28\]](#page-6-0). A secondary aim was to quantify the potential of an adverse tissue response at the greater trochanter (GT) due to the application of lateral pressure.

2. Materials and methods

This was an exploratory observational repeated measures design, using a cohort of healthy volunteers. Ethical approval was obtained from the Imperial College Research Ethics Committee (approval no: 6440832) and informed consent gained before testing. Ten healthy participants were recruited for the study (Table 1), corresponding to similar

Anthropometrics characteristics of participants in this study.

mechanistic study designs [\[20](#page-6-0)]. Exclusion criteria included history of pressure ulcers, or any period of being restricted to a bed or wheelchair in the past 12 months.

A seating lateral support prototype device was built consisting of three inflatable chambers; one underbody chamber on which the patient would sit, and two lateral chambers that lie between the sides of the pelvis and the arms of the chair ([Fig. 1\)](#page-2-0). The purpose of the underbody chamber was to serve as a surrogate metric of body weight, based on which the amount of pressure applied by the lateral chamber was determined. Each chamber was connected to a reservoir through an array of valves and internal pressure sensors (make and model). The reservoir was in turn connected to a diaphragm pump. A control unit was built to regulate the inflation of the lateral cushions to a desired pressure relating to the pressure reading in the underbody cushion (creating a ratio of lateral support to the underbody load).

Prior to testing, the underbody chamber was inflated to 1 kPa and placed under the seat cushion, and the lateral chambers were placed against the arms of the chair. Preliminary tests demonstrated that, if inflated above 1 kPa, the change in pressure of the underbody cushion was proportional to the weight applied to it.

All testing was performed in a laboratory setting where ambient temperature was maintained at 20 ± 2 °C. Participants were required to attend the laboratory setting in a pair of shorts. Measurement of weight, BMI, percentage fat, and percentage muscle were obtained using smart scales (RENPHO, UK). Measurements of height were taken using a calibrated tape measure.

The transcutaneous oximeter (TCM5 Flex, Radiometer, Denmark) was calibrated and the participant was asked to place an oximetry electrode with a fixation ring at their right ischial tuberosity, identified by feeling the bony prominence in the tissue. The electrode was attached to a monitor recording at a frequency of 1 Hz. Physiological measures of transcutaneous oxygen and carbon dioxide tensions (TcPO₂, TcPCO₂) were monitored at the ischial tuberosity throughout the test period, acquired in mmHg.

The testing protocol is shown in [Fig. 2.](#page-2-0) Participants were asked to stand for 20 min while the skin was heated to 43 ◦C to allow maximum vasodilation (as per the oximeter operating manual), before being asked to sit on the chair for 10 min with no lateral pressure applied. This provided control readings of gas tensions for unloaded tissues in standing (baseline) and whilst in normal seated loading (seated control). The participant then returned to standing for 10 min to allow the tissue to return to baseline gas levels. For a further 10 min, the participant sat on the device, with the lateral pressure chambers inflated to 30 % of the underbody chamber pressure, followed by 10 min of recovery in standing. This was then repeated for 50 % and 70 % of lateral to underbody pressure ratios.

The time period of 10 min for each phase of the protocol was arrived at based on preliminary studies; it was observed that transcutaneous gas tensions altered very quickly (within 20 s) after a change in posture and

Fig. 1. The lateral pressure device, including lateral cushions, an underbody cushion, control box, remote control, and an air reservoir.

Fig. 2. Pictorial summary of participant testing protocol. LPR: Lateral to underbody pressure ratio.

plateaued within 5 min. Ten minutes therefore was considered adequate to understand the extent of the change in perfusion while keeping test time for participants to a minimum.

At the end of this sequence, the sensor was removed from the IT and the oximeter calibrated. The participant was then asked to place the sensor at their right GT and the protocol repeated.

The mean change in gas tensions between minutes 3 and 7 during each standing period was calculated to establish the standing control baseline, to which data from each corresponding seated condition were normalised (calculation of percentage change between standing and sitting). Table 2 shows the mean and standard deviation values used for each participant's baseline.

Additionally, the mean change in gas tensions was calculated over minutes 5–10 during all sitting periods. These specific timeframes were chosen based on preliminary findings suggesting that most individuals reached a plateau in their gas tensions within 5 min. Changes in gas tensions were categorised according to the established characteristic responses defined by Chai and Bader [[29\]](#page-6-0), namely.

- Category 1: minimal changes in both $TePO₂$ and $TePCO₂$ values;
- Category 2: >25 % decrease in TcPO₂ with minimal change in TcPCO₂.

Table 2

Table of baseline values of transcutaneous carbon dioxide and oxygen, calculated for each participant's gas tensions over minutes 3–7 of each standing period.

ID	Carbon Dioxide (mmHg), standard deviation	Oxygen (mmHg), standard deviation
1	30.0, 0.96	77.2, 1.83
$\overline{2}$	37.9, 0.93	71.3, 3.30
3	35.3, 0.46	62.7, 3.67
4	32.1, 0.80	71.8, 1.84
5	29.8, 0.39	61.5, 2.55
6	32.4, 0.52	51.7, 1.68
7	28.0, 1.79	65.2, 2.07
8	33.7, 0.68	78.2, 1.39
9	38.9, 0.65	60.5, 1.85
10	31.3, 0.59	85.0, 2.44
Mean	32.9, 0.78	68.5, 2.26

• Category 3: $>$ 25 % decrease in TcPO₂ associated with a $>$ 25 increase in TcPCO₂.

Category one is the safest and therefore most desirable tissue state, whereas category three is associated with risk of tissue damage and

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should be avoided [[29\]](#page-6-0).

Statistical analysis was performed in GraphPad Prism (v8.4.0 for Windows, GraphPad Software, USA) using a paired, one-tailed Student's t-test.

3. Results

An example tissue gas tension trace for one participant's IT is shown in Fig. 3. The figure depicts transient events whereby $TcPO₂$ decreases and $TePCO₂$ increases during the initial seated positions (LPR 0 % and LPR 30 %). By contrast, in this participant there are more modest changes in TePO_2 and TePO_2 during the LPR 50 % and 70 % conditions. Across the cohort of ten volunteers the relative change in transcutaneous gases at the right IT during the 4 seated conditions (0, 30, 50 and 70 % lateral to underbody pressure ratio) are shown in [Fig. 4.](#page-4-0) All seated conditions whereby lateral pressure was applied showed an improvement in the gas tensions relative to the control seated condition, where no lateral pressure was applied.

Initially, in the seated control condition the mean change in $TePO₂$ among participants was $-57.6 \% \pm 31.4 \%$ when compared to the standing unloaded control. With lateral to underbody pressure at 30 %, the relative change in oxygen from standing was $-39.5\% \pm 32.4\%$, which was smaller when compared to the seated control ($p = 0.03$). Similarly, for lateral to underbody pressures at 50 % and at 70 % the oxygen changed by $-36.7 % ± 31.6 %$ and $-33.1 % ± 28.3 %$, respectively, and both were smaller compared to the seated control ($p =$ 0.04 and $p = 0.01$, respectively).

The corresponding TcPCO₂ values under no lateral pressure (seated control) revealed that the relative change from standing was an increase of 28.0 % \pm 26.5 %. The introduction of lateral-to-underbody pressure of 30, 50 and 70 % resulted in a reduction of carbon dioxide change to $+9.5$ % \pm 9.5 %, 13.5 % \pm 24.7 %, and 11.3 % \pm 23.7 %, respectively, all of which were reduced when compared to the seated control condition ($p = 0.04$, $p = 0.05$, $p = 0.03$).

[Table 3](#page-4-0) shows the tissue response categories [[29\]](#page-6-0) for each partici-pant during each seating condition. [Fig. 5](#page-4-0) shows the proportion of participants in each category in each seating condition.

[Fig. 6](#page-5-0) depicts alterations in TcPCO2 (right) and TcPO2 (left) levels at

the right greater trochanter. In the control condition, with no lateral pressure applied, the mean change in carbon dioxide was $+6.3\% \pm 7.2$ %. When 30 %, 50 %, and 70 % lateral to underbody pressure was applied, the mean changes in carbon dioxide were $+6.9 \% \pm 4.4 \%$, +6.2 % \pm 5.7 %, and +14.7 % \pm 24.9 %, respectively. Notably, there was little change in the carbon dioxide among any of the conditions. In contrast, the mean change in oxygen was $+3.3 \% \pm 10.4 \%$ with no lateral pressure applied, decreasing to $-7.8\% \pm 8.1\%$, $-13.2\% \pm 16.4$ %, and −25.9 % ± 30.4 % when 30 %, 50 %, and 70 % lateral to underbody pressure was applied, respectively ($p = 0.005$, $p = 0.006$, $p =$ 0.010). However, it is of note that fewer participants reached the 25 % thresholds stated by Chai and Bader, than the IT site.

4. Discussion

This study investigated how the application of lateral pressure to a seated individual affected soft tissue perfusion [[26\]](#page-6-0). Oximetry has been used as a surrogate marker for tissue damage in numerous other studies looking at seated individuals [21–[23,30\]](#page-6-0). Previous research has found that an increase in $TePCO₂$ could be indicative of a metabolic change in the local tissues due to ischemia, offering an indication of early tissue damage during mechanical loading [\[31](#page-6-0)]. This supports that a category 3 response should be avoided [\[19](#page-6-0),[31\]](#page-6-0). The results from this exploratory study indicate that lateral pressure application improves tissue perfusion, with lesser changes from an unloaded baseline in $TePO₂$ and TcPCO2 when compared to a seated control with no lateral pressure applied.

The improvement in local tissue perfusion over the ischial tuberosity when compared to the control seated condition was achieved here even by applying lateral to under body pressure as low as 30 %. This finding supports the computational work of Boyle et al. [\[26](#page-6-0)] where a reduction in local tissue stress was observed during low levels of lateral pressure. However, there was a considerable degree of inter-subject variability of this effect, evidenced in the transcutaneous tissue gas changes. [Fig. 5](#page-4-0) shows the number of participants in each of the ischemic categories. Although the number of participants in category 2 remained mostly constant throughout all testing conditions, two out of the 3 participants in category 3, transitioned to category 1 with the application of 50 % of

Fig. 3. Line graph showing the percentage change in transcutaneous carbon dioxide (TcPCO₂) and oxygen (TcPO₂) for a single participant over time. The green areas are the periods of standing. LPR: Lateral to underbody pressure ratio. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

Fig. 4. Percentage change compared to baseline in transcutaneous (A) carbon dioxide and (B) oxygen of all ten participants at their right ischial tuberosity for lateral to underbody pressure ratios of 0, 30, 50, and 70 %. The y-axis shows percentage change of transcutaneous gas tension relative to the participants' standing control gas tensions. The * denotes 0.001 *<* p *<* 0.05.

Table 3

Percentage change in transcutaneous gas tensions of oxygen and carbon dioxide relative to their standing baseline for each participant during each seated stage of the study, colour coded by categories described in Chai and Bader [[29](#page-6-0)]: white is category 1, light grey is category 2 and dark grey is category 3.

ID		Lateral to underbody pressure ratio								
	0%		30 %		50 %		70 %			
	CO ₂	O ₂	CO ₂	O ₂	CO ₂	O ₂	CO ₂	O ₂		
1	26.0	-61.2	6.0	-10.7	1.5	-8.3	2.0	-9.7		
$\overline{2}$	45.0	4.7	11.6	9.8	49.7	6.2	47.5	9.3		
3	43.7	-57.9	10.8	-47.4	7.0	-15.2	4.2	-12.7		
$\overline{4}$	9.7	-57.4	4.3	-36.0	12.2	-82.7	1.5	-37.9		
5	23.8	-77.5	13.6	-41.5	8.0	-35.4	7.2	-43.7		
6	14.4	-84.4	20.9	-81.9	9.3	-37.7	8.7	-44.6		
7	18.8	-26.5	6.8	-40.6	5.8	-27.9	6.8	-17.9		
8	1.7	-9.0	2.8	-2.5	2.6	-6.7	3.8	-14.7		
9	5.2	-46.8	3.4	-19.7	0.8	-36.1	-0.8	-27.5		
10	6.6	-14.9	3.8	-3.6	1.4	-9.4	1.2	-10.7		

lateral to underbody pressure (#P1 and #P3). By contrast, Participant 2 exhibited a category 3 response when sat and application of lateral pressure did not result in a meaningful change in their tissue perfusion. It is of note that this participant had the lowest body fat of all

when seated was minimal and so lateral pressure application could not be concentrated appropriately to make a meaningful difference. Further research is needed to establish how tissue morphology may influence the efficacy of lateral pressure application.

participants. It could be hypothesized that their tissue's lateral bulge

[Fig. 7](#page-5-0) shows the relationship between percentage increase in carbon dioxide when a participant sits with no lateral pressure, against their BMI. Albeit the sample size is small, these results could be indicative of a relationship between PU risk and BMI; individuals with BMI *<*23 kg/m3 , or BMI $>$ 28 kg/m³ experienced a larger increase in TcPCO₂ than those with BMI between 23 and 28 kg/m³. This is consistent with a retrospective study where PU incidence in populations of patients with BMIs of underweight, healthy weight, obese, and extremely obese were found to be 8.6 %, 5.5 %, 2.8 %, and 9.9 %, respectively [[32\]](#page-6-0). The study also showed that patients who were considered healthy weight, were twice as likely to develop a PU than those who were obese. Similarly, Worsley et al. found that participants with relatively low BMIs (20.3–25.0 kg/m³) exhibited a category 3 response [[33\]](#page-6-0). Future work should consider measuring $CO₂$ of participants across BMIs and to investigate whether lateral pressure has a positive effect, particularly to those BMIs considered most at risk.

By combining the observations at the IT and GT for each participant, the 50 % level of lateral to underbody pressure ratio was shown to be the

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Fig. 6. Percentage change compared to baseline in transcutaneous (A) carbon dioxide and (B) oxygen of all ten participants at their right greater trochanter for lateral to underbody pressure ratios of 0, 30, 50, and 70 %. The y-axis shows percentage change of transcutaneous gas tension relative to the participants' standing control gas tensions. The ** denotes $0.0001 < p < 0.001$.

Fig. 7. Percentage change in carbon dioxide at the ischial tuberosity (IT) when sat with no lateral pressure, against participants' body mass index, including categories defined by Chai and Bader [\[16](#page-6-0)].

most effective at supporting perfusion of the tissue at the IT without negatively impacting the tissue at the GT. Lateral to under body pressure ratios of 30 % and 50 % resulted in a change in oxygen of less than − 25 %, but a ratio of 70 % resulted in a change that was similar to -25 %. There was little change in carbon dioxide at the GT during the different seating regimes; this is in line with previous studies looking at the GTs [[30\]](#page-6-0).

The results from this study are clinically relevant as they introduce an evidence-based approach for the support of ischial perfusion and consolidate the method to further investigate the efficacy of pressurerelieving support surfaces. The potential of lateral pressure application in improving tissue viability was demonstrated in this limited samplesized exploratory study despite limitations in the device design itself and the placement of the oximeter sensor. Indeed, the rudimentary design of the lateral chambers means that they are not optimized currently to apply the pressure consistently over a specific area or to cater for the variety of body shapes. The participants were asked to fit the sensor over their right IT themselves and so the location of the sensor may have deviated in some participants; due to the length of time needed to perform the study with each participant, repeat readings were not taken to minimize the potential effect of poor sensor placement on the results. Further limitations in this study include the use of a single type of chair and cushion, and the posture in which participants were placed. Future work should consider exploring these parameters and quantify the resulting variability in effectiveness of lateral pressure application.

in this study. This is significantly shorter than the maximum sitting time of 4–6 h before being moved recommended in the NICE Guideline [[5](#page-6-0)]. The 10 min were chosen as preliminary tests with one participant who sat for 4 h did not show a change in the tissue-response category assumed within a few minutes of seating and is consistent with previous protocols in the area [[33\]](#page-6-0). Comfort-related metrics when lateral pressure was applied were not considered in this preliminary study, but no complaints were made by any of the participants of any discomfort at any stage.

This study should be deemed as preliminary not only due to the rudimentary design of the chambers, but also due to the small sample size of participants. In fact, all participants were healthy volunteers of a relatively young age and the variation of BMIs between them was small. Body composition and the buttocks shape of the cohort is likely to not be very representative of those at risk of PUs. However, category 3 responses were still observed in 30 % of the participants during normal seating, and in most cases improved with the introduction of lateral pressure. This is consistent with previous studies looking at transcutaneous gas tension of participants with conditions that put them at risk of PUs; those who had previously had PUs required lower loads to significantly change their gas tensions, compared to a participant with no history of PUs [[17\]](#page-6-0). In relation to body habitus, for participants with wider hips, or a larger bi-trochanteric distance, the contact area between the lateral cushions and the body may be larger than someone narrower. This was not controlled or monitored in this study; future work should consider monitoring this or controlling for the distance between the arms of the chair and the hips of the participant. Finally, the heating of the tissue by the oximeter can cause a physiological response, however, as all participants underwent testing at the same temperature and for the same amount of time, the effect is likely to be small.

5. Conclusion

The findings of this study demonstrate that controlled application of lateral pressure to a young healthy seated individual can improve local tissue perfusion at the IT without adversely affecting the tissues at the GT. These observations were obtained using a rudimentary, nonoptimized prototype device on a small-sized cohort. This suggests that with further device-design efforts and patient evaluations, the lateral pressure system has the potential to provide a preventative solution for seated patients at risk of PUs.

The test period for each seating condition was decided to be 10 min

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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