Journal of Tissue Viability xxx (2017) 1–9

Contents lists available at ScienceDirect

Journal of Tissue Viability

journal homepage: www.elsevier.com/locate/jtv

Beware of the toilet: The risk for a deep tissue injury during toilet sitting

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ARTICLE INFO

Article history:
Received 7 October 2016
Received in revised form
18 April 2017
Accepted 20 April 2017

Keywords:
Sitting-acquired pressure ulcer
Pressure injury
Toilet seat
Computational modeling
Transcutaneous oxygen tension

ABSTRACT

A pressure injury (PrI) compromises quality of life and can be life-threatening. The fundamental cause of PrIs is sustained deformations in weight-bearing soft tissues, e.g., during prolonged sitting on inadequate surfaces such as a toilet seat. In nursing homes and geriatric facilities, patients need assistance using the restroom, and patients being left on the toilet for tens-of-minutes is a real-world scenario, unfortunately. Nevertheless, there are no published studies regarding sustained tissue loads during toilet sitting and their effects on tissue physiology. Here, the biomechanical and microcirculatory responses of the buttock tissues to toilet sitting were investigated using finite element modeling and cutaneous hemodynamic measurements, to explore the potential etiology of PrIs occurring on the toilet. We found that prolonged sitting on toilet seats involves a potential risk for PrI development, the extent of which is affected by the seat design. Additionally, we found that specialized toilet seat cushions are able to reduce this risk, by lowering instantaneous tissue exposures to internal stresses (by up to 88%) and maintaining reduced interface pressures. Furthermore, hemodynamic variables were altered during the toilet sitting; in particular, $tcPO_2$ was decreased by 49% ± 7% (44 ± 2[mmHg] to 22 ± 4[mmHg]) during sitting. The current study confirms that investing in expensive PrI prevention (PIP) products is likely to be ineffective for an immobilized patient who is left to sit on a bare toilet seat for long times. This argument highlights the need for a holistic-care approach, employing PIP devices that span across the entire environment where bodyweight forces apply to tissues.

1. Introduction

A pressure injury (PrI) (also termed Pressure Ulcer), and particularly a deep tissue injury (DTI), compromises the quality of life and can be life threatening [1]. The fundamental cause of PrIs is sustained deformations in weight-bearing soft tissues, especially during sitting [2,3]. The etiology of PrIs has been shown to be multifactorial, with 4 main mechanisms [4]; ischemia [5], cell deformation [6–9], reperfusion injury [10] and impairment of lymphatic vessels [11,12]. Sitting-acquired PrIs usually onset near the interfaces between the ischial-tuberosity (IT) bones and surrounding soft tissues [13,14]. It is a common complication in spinal cord injury (SCI) wheelchair-bound patients, where PrIs develop during wheelchair use [15–17]. Additionally, sitting-acquired PrIs might also be caused by prolonged sitting on other rigid surfaces such as plastic chairs, washroom stools or toilet seats. Products such as pressure-redistribution-cushions (PRCs) are commercially available to relieve mechanical loads within tissues at vulnerable body sites [16], including whilst sitting on toilet seats, yet the importance of continuing protection on these alternate sitting surfaces is often overlooked.

Treatment of PrIs is costly, which makes PrI prevention (PIP) a primary financial goal of health institutes. In the UK alone, the annual expenditure for treating PrIs is estimated between £1.4–2.1 billion, and the cost of treating a single PrI may reach £14,108 [18]. It is estimated that the prevalence of PrIs in SCI patients is 25%–33% of the US and European SCI populations [19,20]. However, a study regarding hospital incident reporting systems found that 86% of injuries go unreported, particularly, 26% of PrI cases go unreported (stage-I, stage-II, or un-staged) [21]. Additionally, there appears to

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http://dx.doi.org/10.1016/j.jtv.2017.04.005
0965-206X/

Abbreviations

ACB	Air-cell-based
CMBC	Concentration of Moving Blood Cells
DTI	Deep Tissue Injury
FE	Finite Element
FC	Foam Cushion
IT-bone	Ischial-Tuberosity Bone
LDF	Laser Doppler Flowmetry
MRI	Magnetic Resonance Imaging
PU	Perfusion Units
PrI	Pressure Injury
PIP	Pressure Injury Prevention
PRC	Pressure-Redistribution-Cushions
RBC	Red Blood Cells
SCI	Spinal Cord Injury
SED	Strain Energy Density
tc-PO_2	Transcutaneous Oxygen Tension

be under-reporting for less severe PrIs [22,23]. Hence, it is reasonable to assume that the incidence of PrIs in general, and of sitting-acquired DTIs in particular, is underreported.

It is rather intuitive that sitting on a rigid and narrow surface such as standard plastic or wooden toilet seats for a prolonged period inflicts a risk for PrI development. In nursing homes and geriatric facilities, patients need assistance using the toilet or taking a shower. These daily routines can take up to 30-min for each event, and the prolonged sitting, combined with the factors of moisture, heat and skin fragility (particularly in elderly patients), result in an increased PrI risk [16,24,25]. Finite element (FE) computational modeling can be used to analyze internal soft tissue loads due to bodyweight and external forces that are applied during sitting, and eventually, assess the (relative) risk of developing PrIs for different sitting conditions [26]. Several studies employed the FE method to investigate the biomechanical aspects of sitting-acquired PrIs, and the use of PRCs [2,27–35]. However, no such study has been reported in the literature regarding tissue loads during toilet sitting.

Peripheral microcirculatory variables could reflect local and systemic changes, and even predict the development of ischemic stress conditions [36–39]. Laser Doppler Flowmetry (LDF) and transcutaneous oxygen tension (tc-PO_2) are well-established, reliable, noninvasive measures of the cutaneous flux and tissue oxygenation status, respectively, which can be utilized under external mechanical loads [40]. However, to-date, no study concerned the LDF and tc-PO_2 of the buttocks tissues during toilet sitting, and how they may change over time.

In the present work, the immediate biomechanical and microcirculatory responses of the buttocks tissues to toilet sitting were investigated in the context of the risk of sitting-acquired PrIs, especially DTIs. Using FE simulations and cutaneous hemodynamics variables (recorded in healthy subjects), we explored the potential etiology of PrIs occurring on the toilet.

2. Methods

2.1. Finite element model

2.1.1. Geometry

To examine the effects of sitting on a bare toilet seat versus sitting on a toilet seat cushioned with foam, a set of six FE model variants of the buttock were developed (Table 1). Each variant was developed based on a coronal cross-section of the left buttock and included the IT-bone, gluteus-maximus skeletal muscle, colon smooth muscle, fat tissue, skin, and either a foam cushion (FC), or a bare toilet seat (Fig. 1). The model variants hence differed in the sitting configuration and the stiffness property of the FC (Table 1).

A single coronal magnetic-resonance-imaging (MRI) slice was acquired from a male subject with SCI (age 21-years, 90 kg, SCI-level:T6) (Fig. 1a), which is representative of muscle-atrophy. The subject was scanned in our previous published work, details regarding the scan protocol are available in previous publications [2,27,28].

We used the ScanIP® module of Simpleware® [41] to segment the tissue components from the MRI slice and define a 4 mm uniform thickness to the structure. In two of the model variants (#1,#4), the buttock was seated on bare toilet seats: a horizontal, flat type, termed type-A (Fig. 1b), and an inclined type, termed type-B (Fig. 1d). Model variants #2,#3,#5 and #6 were tested with FCs placed above the seats (Fig. 1c and e). Shapes and dimensions of the seats represent real shapes and sizes of products that are currently in the market. Seat type-A is thicker (25 mm) and wider (90 mm) than seat type-B (thickness:10–20 mm, width:60 mm).

2.1.2. Mechanical properties

Constitutive laws and mechanical properties of all tissues were adopted from the literature (Table 2). Specifically, the IT-bone was assumed as linear-elastic isotropic material [13]. The muscle, fat and skin tissues were assumed to be nearly-incompressible, non-linear isotropic materials with their large deformation behavior described using an uncoupled Neo-Hookean material model [46] with a strain energy density (SED) function W (Eq. (1)):

$$W = \frac{G_{ins}}{2}\left(\lambda_1^2 + \lambda_2^2 + \lambda_3^2 - 3\right) + \frac{1}{2}K(\ln(J))^2 \quad (1)$$

where G_{ins} is the instantaneous shear modulus (Table 2), $\lambda_i (i = 1, 2, 3)$ are the principal stretch ratios, K is the bulk modulus and $J = \mathrm{def}(F)$ where F is the deformation gradient tensor.

The FCs were assumed to be isotropic linear-elastic materials with properties based on experimentally reported data from cushion material tests [47,48]. Plastic toilet seats are usually made from Polypropylene and therefore were assumed here to behave isotropically and linear-elastically (Table 2).

2.1.3. Boundary and material transition conditions

Boundary conditions were chosen to simulate the vertical descent of the weight-bearing ITs when sitting on a bare versus cushioned toilet seat in a thin slice model. The front and back planes of the buttock, the toilet seat and the cushion (if present) were fixed in the perpendicular direction to avoid out-of-plane translations. The inferior surface of the toilet seat and the cushion (if included) were fixed for all translations and rotations. Frictional sliding was defined between the skin and the seat or between the skin and the cushion, with the coefficient of friction set as 0.4 in all the simulations. Tied interfaces were defined between all tissue components.

Table 1
The model variants.

Model variant	Toilet-seat Type	Cushion
#1	Type A	–
#2	Type A	Foam, 7 [kPa]
#3	Type A	Foam, 10 [kPa]
#4	Type B	–
#5	Type B	Foam, 7 [kPa]
#6	Type B	Foam, 10 [kPa]

Please cite this article in press as: Lustig M, et al., Beware of the toilet: The risk for a deep tissue injury during toilet sitting, Journal of Tissue Viability (2017), http://dx.doi.org/10.1016/j.jtv.2017.04.005