ELSEVIER

Journal of Biomechanics 40 (2007) 1443–1454

JOURNAL OF BIOMECHANICS

www.elsevier.com/locate/jbiomech
www.JBiomech.com

Assessment of mechanical conditions in sub-dermal tissues during sitting: A combined experimental-MRI and finite element approach

Eran Linder-Ganz[a], Noga Shabshin[b], Yacov Itzchak[b], Amit Gefen[a,*]

[a]*Department of Biomedical Engineering, Faculty of Engineering, Tel Aviv University, Tel Aviv 69978, Israel*
[b]*Department of Diagnostic Imaging, Chaim Sheba Medical Center, Tel Hashomer 52621, Israel*

Accepted 26 June 2006

Abstract

A common but potentially severe malady afflicting permanent wheelchair users is pressure sores caused by elevated soft tissue strains and stresses over a critical prolonged period of time. Presently, there is paucity of information regarding deep soft tissue strains and stresses in the buttocks of humans during sitting. Strain and stress distributions in deep muscle and fat tissues were therefore calculated in six healthy subjects during sitting, in a double-donut Open-MR system, using a "reverse engineering" approach. Specifically, finite element (FE) models of the undeformed buttock were built for each subject using MR images taken at the coronal plane in a non-weight-bearing sitting posture. Using a second MR image taken from each subject during weight-bearing sitting we characterized the ischial tuberosity sagging toward the sitting surface in weight-bearing, and used these data as displacement boundary conditions for the FE models. These subject-specific FE analyses showed that maximal tissue strains and stresses occur in the gluteal muscles, not in fat or at the skin near the body–seat interface. Peak principal compressive strain and stress in the gluteus muscle were $74\pm7\%$ and 32 ± 9 kPa (mean $\pm$ standard deviation), respectively. Peak principal compressive strain and stress in enveloping fat tissue were $46\pm7\%$ and 18 ± 4 kPa, respectively. Models were validated by comparing measured peak interface pressures under the ischial tuberosities (17 ± 4 kPa) with those calculated by means of FE (18 ± 3 kPa), for each subject. This is the first study to quantify sub-dermal tissue strain and stress distributions in sitting humans, in vivo. These data are essential for understanding the aetiology of pressure sores, particularly those that were recently termed "deep tissue injury" at the US National Pressure Ulcer Advisory Panel (NPUAP) 2005 Consensus Conference.

Keywords: Pressure sore modeling; Decubitus ulcer; Rehabilitation of wheelchair users; Deep tissue injury; Mechanical properties

1. Introduction

A common but serious malady afflicting wheelchair-bound and bedridden immobile patients are pressure sores, particularly at their severe form which involve sub-dermal tissue damage. Pressure sores involving sub-dermal tissue damage, where damage originates in muscle tissue, were recently termed "deep tissue injury" (DTI) by the US National Pressure Ulcer Advisory Panel, and are attracting growing attention by the medical community (Black and National Pressure Ulcer Advisory Panel, 2005). Generally, pressure sores affect about 10% of all hospitalized patients (Whittington et al., 2000) and are straining the budget of the US healthcare system by more than 1.2 billion dollars annually (Injury fact book of the Center for Disease Control and Prevention (CDC), 2001–2002). Moreover, DTI can lead to osteomyelitis, sepsis and even death (Tsokos et al., 2000; Margolis et al., 2003).

Pressure sores generally develop in two separate varieties, superficial and deep (Bouten et al., 2003a). A superficial pressure sore first affects the layers of skin near epidermal tissue and is typically associated with

*Corresponding author. Tel.: +972 3 6408093; fax: +972 3 6405845.
E-mail address: gefen@eng.tau.ac.il (A. Gefen).

0021-9290/$ - see front matter © 2006 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jbiomech.2006.06.020

presence of moisture and heat combined with damaging frictional and shear forces (Crenshaw and Vistnes, 1989). Infection may follow, and poor nutrition may exacerbate the injury or delay healing (Vidal and Sarrias, 1991). However, the damage caused by superficial sores is generally reversible with adequate medical treatment, since it is contained within the top layers of a regenerative tissue. Deep pressure sores, or DTI, unfortunately stand in stark contrast to their superficial brethren both in their mechanisms and nature of damage (Bliss, 1992). This is a decidedly more serious medical complication (Bliss, 1992), which had motivated the present study. Many researchers agree that the cause for a DTI is prolonged mechanical loads that interfere with the flow of blood and clearance of metabolic byproducts in vascularized soft tissues (Kosiak, 1959; Knight et al., 2001). However, excessive local deformation of tissues, not necessarily coupled with ischemia, was also indicated to be involved in DTI aetiology (Bouten et al., 2003b; Breuls et al., 2003). The ischemia and peak deformation or mechanical stress in tissues typically occur in vicinity of bony prominences of the pelvis in the sitting position, i.e. the ischial tuberosities, the greater trochanter of the femurs and the sacrum (Kosiak, 1959; Brienza et al., 2001; Bouten et al., 2003b; Linder-Ganz and Gefen, 2004; Gefen et al., 2005). Immobilization was identified as a key risk factor for DTI as it suppresses the ability of patients to timely relieve the mechanical loads on soft vascularized tissues, particularly those in muscles under bony prominences (Allman, 1989).

The seriousness level of pressure sores is clinically classified into 4 different categories, from stage 1 which is mildest to stage 4 that is most severe, and this classification attempts to describe the extent and depth of tissue damage (Shea, 1975). In terms of the time course of injury, DTI are thought to initiate adjacent to bony prominences, and to erode the local soft tissue surrounding the bony prominence—typically muscle tissue first (Nola and Vistnes, 1980; Daniel et al., 1981; Salcido et al., 1995; Linder-Ganz and Gefen, 2004; Gefen et al., 2005). Computational simulations indicated that as this deep muscle tissue necroses, it also stiffens and subsequently, projects mechanical stresses to the more superficial tissues, which then bear the mechanical burden (Linder-Ganz and Gefen, 2004; Gefen et al., 2005). Subsequently, the more superficial layer necroses, thus creating a positive-feedback detrimental cycle, which may continue until the lesion finally breaks through the epidermal layer (Linder-Ganz and Gefen, 2004; Gefen et al., 2005). This process may initiate in less than 30 min of immobilization, and widespread DTI may develop within ~4 h of lying or wheelchair sitting (Linder-Ganz and Gefen, 2004; Gefen et al., 2005; Linder-Ganz et al., 2006). According to current clinical practice, a developing DTI may be misdiagnosed as a mild (stages 1–2) sore since the true extent of tissue damage is not visible until breakdown of the skin (Wound, Ostomy and Continence Nurses Society (WOCN), 2005; Black et al., 2005). Hence, the frontier in biomechanical research of pressure sores focuses on understanding the aetiology of DTI (Bouten et al., 2003a). A basic, first step for such investigations is to characterize the mechanical conditions in muscle and fat tissues during supported postures such as sitting. To this end, not only that data from sitting patients on the deformations, strains and stresses in deep soft tissues are missing in the literature, but there is also paucity in information on normative mechanical conditions in muscle and fat tissues during sitting.

Some indications on the intramuscular mechanical conditions during sitting are provided by means of computer simulations and physical models. Specifically, Brosh and Arcan (2000) developed a two-dimensional (2D) finite element (FE) model of the buttock during sitting in a chair. Their model included the pelvic bone and a homogeneous surrounding soft tissue. They found that the internal principal compressive stresses in soft tissues under the ischial tuberosities exceeded the peak interface pressure between the buttock and seat. An axisymmetric 3D FE model of the buttock was developed by Ragan et al. (2002), to evaluate the efficiency of different cushions during sitting. Similarly, maximal intramuscular compressive stress of 37 kPa was found under the ischial tuberosities corresponding to peak interface pressure of 10 kPa. Oomens et al. (2003) presented a more detailed 3D axisymmetrical non-linear FE model of the buttock, which distinguished between muscle, fat and skin tissues. Again, peak compressive stress in muscles under the ischial tuberosities was 2.4-fold (~120 kPa) the interface pressure (~50 kPa). Gefen et al. (2005) recently studied muscle stresses around the sacrum using a 3D FE model of a slice through the buttock during wheelchair sitting. Consistently with all previous literature, they found that peak principal compressive stress in deep muscle tissue under the sacrum is 266-fold greater (4 MPa) than the interface pressures at that site (15 kPa). The most recently published 3D FE model of the buttock during sitting was developed by Sun et al. (2005) based on (non-sitting) MRI images. They found that the maximal compressive stress is located in deep muscles under the ischial tuberosities of the pelvis (~76 kPa), corresponding to an interface pressure of ~21 kPa. They also found maximal tissue deformations to occur in muscle and fat. The physical models (phantoms) of the buttock by Reddy et al. (1982) and later by Candadai and Reddy (1992) agree very well with computational findings, that interface pressures are substantially lower than peak stresses in deep muscles. In these phantoms, internal deformations were measured in a polyvinyl chloride gel (PVC) layer under a wooden core representing the