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RISK FACTORS FOR DEEP PRESSURE SORES REVEALED THROUGH FINITE ELEMENT SIMULATIONS COUPLED WITH AN INJURY THRESHOLD AND A DAMAGE LAW FOR MUSCLE TISSUE

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INTRODUCTION

Pressure sores are injuries in deep or superficial soft tissues, caused by prolonged mechanical loading of tissues between a bony prominence of a patient and a supporting surface such as a wheelchair seat or a mattress. Traditionally, interface pressure/shear distributions under the ischial tuberosities (IT) and sacrum of wheelchair users are measured through an array of pressure and/or shear sensors, to provide quantitative engineering measures of cushion performances. Other methods were also proposed for analyzing the effectiveness of cushions, based on measurements of tissue shape and deformation, tissue stiffness, seat contour, and thermal properties of the cushions. However, interface pressure measurements remained the gold-standard test for evaluation of cushion performances, and are accepted by both the medical device industry and clinical community. Unfortunately, interface pressure measurements during sitting/lying cannot identify focal stresses in deep tissues adjacent to bony prominences, which may cause deep pressure sore (DPS) [1-3].

Since direct, non-invasive measurements of the distribution of internal strains/stresses in load-bearing soft tissues of paralyzed patients are not feasible, finite element (FE) modeling was used [2-4]. Finite element modeling, however, cannot indicate the biological damage associated with exposure to the predicted level of stresses for different time durations. Prediction of the pattern and severity of biological damage in tissues requires coupling of the FE model with a mathematically-defined tissue injury threshold that is based on animal model studies [5]. Additionally, in simulations that consider development of DPS over time, the altered mechanical behavior of the injured tissue should be considered [2,3]. Using animal models of pressure sores, we previously showed that injured muscle tissue stiffens and that the extent of stiffening depends on the applied pressure and exposure time [2,3]. The objective of this study was, first, to develop an algorithm to couple a FE model of the buttocks during wheelchair sitting with a tissue injury threshold for striated muscle under compression [5], and with the stiffening behavior of injured muscle tissue [2,3], in order to predict the potential for DPS when using different wheelchair cushions. Second, we aimed at employing this algorithm to identify the major risk factors for DPS onset. Importantly, the algorithm developed herein provides a standard engineering tool for evaluation, material selection and shape design of wheelchair cushions.

METHODS

Injury Threshold for Striated Muscle Tissue

An injury threshold for skeletal muscle under compression was determined in a previous study [5], and was coupled with the FE analyses herein. Briefly, we fitted a compressive stress versus time injury tolerance for skeletal muscle tissue of albino rats based on histopathology studies in the literature supplemented by similar, complementary studies conducted in our laboratory. After pressure was delivered, we sacrificed the animals and harvested cubic samples from the compressed muscles for histopathology. We used Phosphotungstic Acid Hematoxylin (PTAH) staining to determine viability of muscle cells and integrity of cross-striation. If cell death or loss of cross-striation could be identified in a PTAH-stained specimen under optical microscopy for a certain stress-time combination, that stress-time combination was classified as injuring. Our histopathology findings were then superimposed with previous histopathology of rat muscles in the literature. It was shown that the following sigmoid relation between the critical compressive stress (σ, kPa) and exposure time (t, minutes) can distinguish all injury cases from non-injuries:

$$\sigma_{[KPa]} = \frac{23}{1+e^{0.15(t-95)}} + 8 \quad (1)$$

Stiffening of Muscle Tissue as a Result of Pressure Injury

Simulations of tissue response to prolonged compression in immobilized individuals need to take the altered mechanical behavior of injured tissue regions into account, as the altered mechanical properties of injured tissues may affect deformations and loads in adjacent, normal tissues [2,3]. We previously reported *in vivo* indentation studies in striated muscle tissue of anesthetized rats before and after subjecting the muscles to compressive stresses of 35 kPa or 70 kPa for durations of 15, 30, 60, or 120 minutes [3]. The long-term shear modulus of muscle tissue was shown to stiffen with injury by a factor of up to 3.3±1.6 for maximal exposures (70 kPa for 120 minutes).

Finite Element Modeling

A plane stress symmetrical model of a seated human buttock was developed based on an MRI section under the IT. Image was obtained from a female (age: 29 years, body weight: 54 kg) in a non-weight-bearing sitting-like posture. Contours of hard and soft tissues were detected and segmented to form the buttock cross-sectional geometry, using a solid modeling software package (SolidWorks 2005). Then, the model was transferred to a FE solver (Nastran 2005) for non-linear strain/stress simulations of interactions with different types of mattresses. The model was subjected to displacement boundary conditions of vertical sagging of the IT, as was shown in an MRI taken during a weight-bearing sitting posture of the same subject. In each simulation, foundations of the mattress were fixed and nodes at the contact surface between the body and cushion were set as "multi-point constrains" (MPC), allowing free translation normal to the supporting surface and frictional slipping horizontally. Nodes along the symmetry axis were constrained for horizontal motion. A bi-linear stress-strain curve, formulating the long-term viscoelastic moduli of muscle under small and large strains, was used ($E^{strain<15\%}$=3.6 kPa and $E^{strain>15\%}$=47 kPa). All other mechanical properties are detailed in reference [2].

Damage Accumulation Simulations

To evaluate the risk for DPS onset in muscles we coupled the FE stress analyses with the muscle injury threshold (Eq. 1), and considered stiffening of injured muscle tissue [2,3]. We simulated different normal and pathological anatomical conditions (reduction in the IT radius of curvature and atrophy of muscles) for different cushion types (rigid, semi-rigid and soft). Long-term modulus of each muscle element that was under critical stress exposure conditions (Eq. 1) was increased according to experimental data [3]. Strain and stress fields were calculated iteratively to simulate the progress of DPS promoted by muscle tissue stiffening [2,3], until a steady state of the strain and stress distributions was reached (Fig.).

RESULTS

We found that the range of peak principal compressive stresses in non-injured gluteus muscles was 22-32 kPa for the different cases. Muscle atrophy with loss of 50-70% of muscle mass caused local muscle stresses of 33 kPa, which exceeds muscle tolerance (Eq. 1), and, after 30 minutes, triggers the injury-stiffening spiral [2,3] (Fig.). For example, in muscles that underwent atrophy and loss of 50% of the muscle mass, 210 minutes of continuous immobilized sitting on a semi-rigid cushion (100 kPa) caused a wide-spread (simulated) DPS. Within the time-course of progression of such DPS, the accumulative area of muscle elements exposed to an injuring compressive stress of 33 kPa or over expanded by 650% (Fig.). This demonstrates that loss of muscle mass and thickness due to atrophy, which is characteristic to immobilized patients, is a major risk factor for DPS, as mechanical stress concentrations in the thinned muscle are more easily formed.

DISCUSSION

Among spinal cord injury patients at the chronic stage, muscle cross-sectional area and muscle weight are significantly less than in normals. We showed that this is a major risk factor for DPS that involve gluteus muscles, as the thinned muscles are more susceptible to stress concentrations and elevated deformations in vicinity of the small radii of curvature of the IT. The present computer algorithm now allows to evaluate if manipulating a cushion geometry or material properties can resolve this problem and reduce the risk for DPS.

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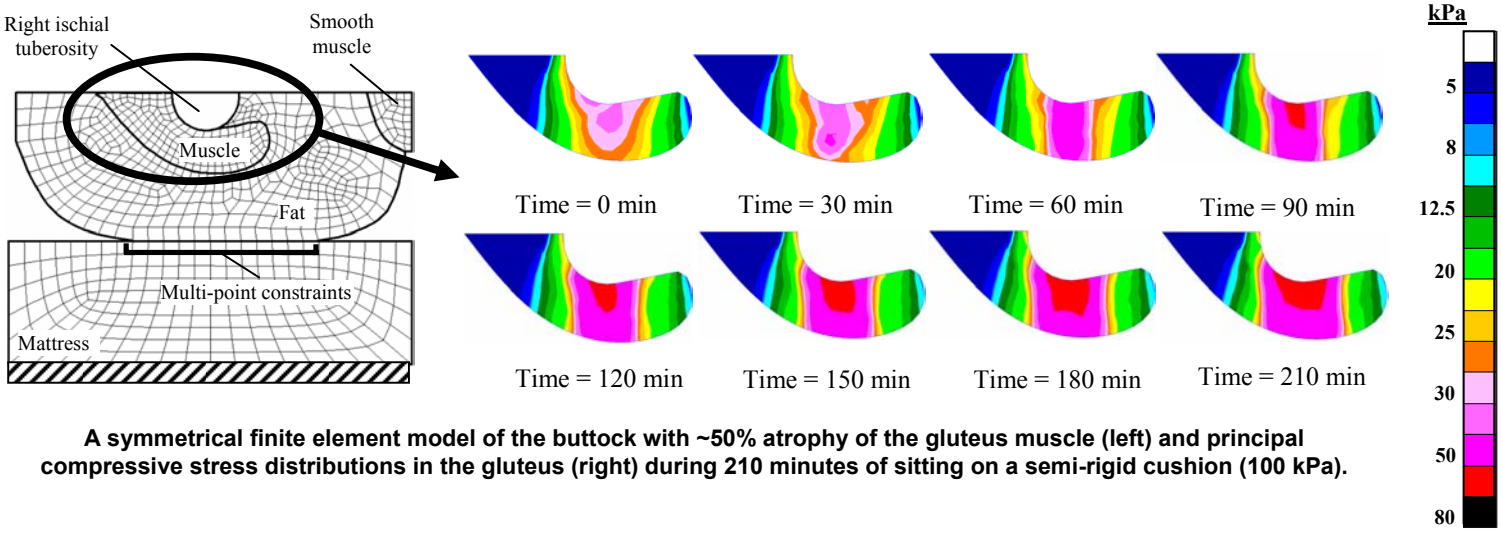

A symmetrical finite element model of the buttock with ~50% atrophy of the gluteus muscle (left) and principal compressive stress distributions in the gluteus (right) during 210 minutes of sitting on a semi-rigid cushion (100 kPa).