INTEGRATING ANIMAL AND COMPUTER MODELS TO STUDY THE MECHANICS OF PRESSURE SORES

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INTRODUCTION

Pressure sores (PS) caused by extensive and prolonged loading of vascularized soft tissues are considered one of the most severe complications in geriatric and paralyzed patients. Their prevalence in modern western hospitals is about 10%. Recent three-dimensional (3D) biomechanical modeling of the onset of PS in the pelvis musculature indicated that elevated stresses, which could potentially lead to ulceration, are found at deep muscle tissue underlying bony prominences (Linder-Ganz and Gefen, 2002). However, mechanical properties of normal uninjured muscles were used in these models to simulate internal stress distributions. Exposure of muscle tissue to intensive and prolonged compression may affect its microstructure (Bosboom et al., 2001) and thereby, the muscle's constitutive law is also expected to change. In order to study the mechanism of PS injuries, changes in the muscle's constitutive law during its exposure to pressure should also be characterized. The objective of this study is to determine and quantify changes in stiffness of muscle tissue as a result of prolonged mechanical loading, and to apply the modified properties of injured muscles to realistic, anatomically accurate 3D models of body parts vulnerable to PS. This provides comprehensive specification of the mechanical conditions leading to onset of PS.

METHODS

Mechanical properties of injured versus uninjured muscles were analyzed in the rat model. Experiments were approved by the Institutional Animal Care and Use Committee (IACUC) of Tel Aviv University and were carried out in compliance with institutional guidelines for care and use of animal models (IACUC Protocol #11-02-41). 43 male rats (weight 280 ± 20 g) were assigned for this study. Ketamin (10 mg/Kg) and Xylazine (90 mg/Kg) were subcutaneously injected to induce anesthesia and 1/3 of this dose was injected when necessary for maintenance. Depth of anesthesia was verified by lack of pinch response. Limb hair was carefully shaved and rats were positioned on a special apparatus containing a spring-derived rigid indentor (diameter 30 mm) that applied calibrated constant compression to the gracillis muscle of the right hind limb (Fig. 1a).

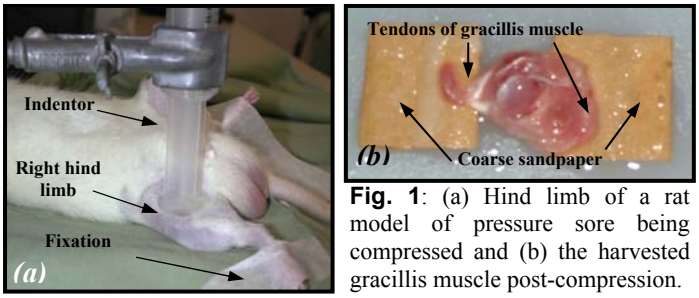

Fig. 1: (a) Hind limb of a rat model of pressure sore being compressed and (b) the harvested gracillis muscle post-compression.

Different pressure doses (product of pressure level and exposure time) were delivered to sub-groups of 4-5 animals: 10, 35 and 70 KPa for 2, 4, 6 hours. Pressure values were determined based on preliminary finite element (FE) simulations of internal stresses in the pelvis muscles during recumbency (Linder-Ganz and Gefen, 2002), which yielded maximum compression of 100 KPa. After pressing the limbs, rats were sacrificed by overdose of Pentobarbital and gracillis muscles were harvested from the uninjured (control) and injured limbs by cutting their tendons at both edges (Fig. 1b). Specimens were kept in saline tubes at 3ºC until mechanical testing (within no more than 30 minutes from dissection). Length, volume and weight of each muscle were recorded. Specimens were then placed in an INSTRON 5544 uniaxial tension system with their tendons compressed between customized jigs covered with sandpaper to prevent slipping (Fig. 2). Tension was applied within a transparent aquarium filled with saline at the rat's body temperature (33ºC). Load-deformation curves were obtained for the injured and uninjured (control) muscles at a rate of 1 mm/min. Deformation was visually monitored and recorded using digital and analog video systems for post-experiment slow-motion analysis to verify that muscles did not slip off the grippers. Plots of Lagrange stress (force divided by original mean cross-sectional area) versus true strain (calculated from transient distance between jigs) were derived from the load-deformation curves. Mean cross-sectional area of muscles was obtained by dividing the muscle's volume by its unloaded length. Tangent modulus of elasticity was calculated for each curve at 5% strain. The lower strain range is of interest to conform with loading/deformation of deep muscles during recumbency.

Statistical analysis included ANOVA and Tukey-Kramer tests to determine effects of pressure and exposure time factors and identify differences between properties of muscles subjected to different pressure doses. Criteria for exclusion of specimens (total ~15%) included apparent muscle damage caused by surgical tools during dissection, tearing of muscle adjacent to a gripper and identified slipping of specimen from the grippers. Experimentally measured mechanical properties of uninjured and injured muscles were fed into 3D FE models of body parts vulnerable to PS. 5mm-thick anatomical slices were reconstructed for the mattress-supported regions of the head, shoulders, pelvis-sacrum and heels using the Visible Human (male) digital database (Fig. 3). Bones (cortical, trabecular), cartilage, muscles, colon, ileum, major blood vessels, fascia, tendons, brain tissue and skin were segmented and their contours transferred to a solid modeling software package (SolidWorks 2003) for organ reconstructions. Anatomical structures were then transferred to a finite element solver (NASTRAN 2001) for stress analysis under musculoskeletal loading during recumbency. Tissues were assumed to be homogenous, isotropic and non-linear elastic materials with their properties adapted from previous studies (e.g. Gefen et al., 2001).

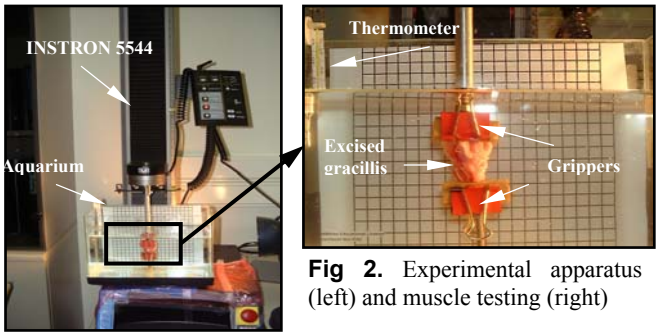

Fig 2. Experimental apparatus (left) and muscle testing (right)

RESULTS

Animal and computer models were developed to provide better understanding the mechanism of PS onset. Tangent moduli E_t (5% strain) of rat muscles injured by exposure of 2-6 hours to compression of 70 KPa (E_t =172 ± 39 KPa) were shown to be 43% higher (p<0.05) compared with uninjured muscles (E_t =120 ± 19 KPa). 3D FE models were built to analyze stress distributions in deep soft tissues subjected to musculoskeletal loads during recumbency (Fig. 3) considering this muscle stiffening effect. The maximal internal stresses at deep muscles generally exceeded the interfacial compression by up to two orders of magnitude (Fig 4.). The internal stress distribution patterns during recumbency are evolving with time due to changes in the injured muscle's constitutive laws. For instance, stresses in muscles surrounding the scapula were predicted to increase and expand within 2 hours post-immobilization due to muscle injury and stiffening, as demonstrated in regions A-C of the shoulders model in Fig. 5. This suggests a mechanism of deterioration in which soft tissues that were not directly affected by the intensified internal stresses could be gradually damaged due to induction of elevated stress by adjacent stiffening injured muscle tissue.

DISCUSSION

Integration of animal and FE models provides a powerful tool for studying PS onset and has the potential of being an aid in design of seats and beds with protective supports. Promising implementation of the integrated animal-computer model data is in development of a risk-monitoring system (Yemal et al., 2002) that provides real-time alerts when PS are suspected to appear based on internal muscle tissue stress calculated using FE and injury thresholds obtained from animal models (patent pending).

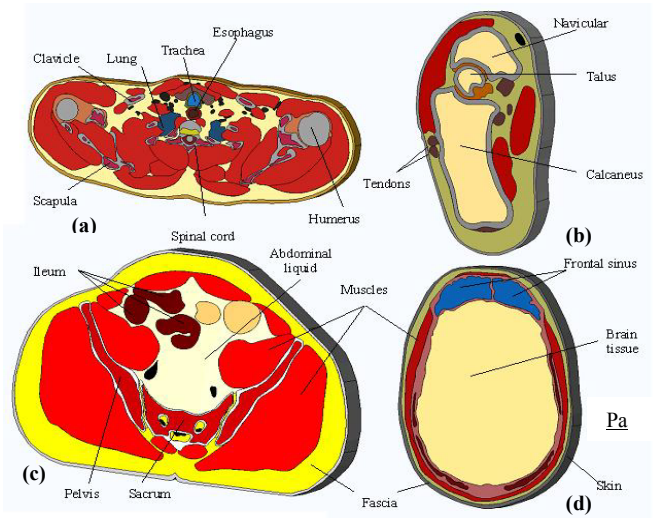

Fig. 3. 3D FE models of the (a) shoulders, (b) heels, (c) pelvis and (d) head for stress analysis.

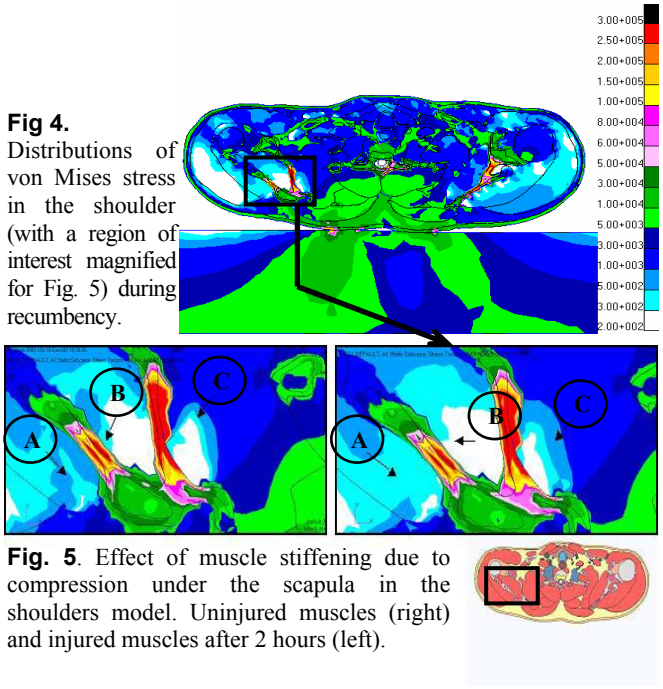

Fig 4. Distributions of von Mises stress in the shoulder (with a region of interest magnified for Fig. 5) during recumbency.

Fig. 5. Effect of muscle stiffening due to compression under the scapula in the shoulders model. Uninjured muscles (right) and injured muscles after 2 hours (left).

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