Mechanical compression-induced pressure sores in rat hindlimb: muscle stiffness, histology, and computational models

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Linder-Ganz, E., and A. Gefen. Mechanical compression-induced pressure sores in rat hindlimb: muscle stiffness, histology, and computational models. J Appl Physiol 96: 2034–2049, 2004.—Pressure sores affecting muscles are severe injuries associated with ischemia, impaired metabolic activity, excessive tissue deformation, and insufficient lymph drainage caused by prolonged and intensive mechanical loads. We hypothesize that mechanical properties of muscle tissue change as a result of exposure to prolonged and intensive loads. Such changes may affect the distribution of stresses in soft tissues under bony prominences and potentially expose additional uninjured regions of muscle tissue to intensified stresses. In this study, we characterized changes in elastic properties of muscles exposed to pressure in vivo (11.5, 35, or 70 kPa for 2, 4, or 6 h) and incorporated the abnormal properties into finite element models of the head, shoulders, pelvis, and heels of a recumbent patient. Using in vitro uniaxial tension testing, we found that tangent elastic moduli of muscles exposed to 35 and 70 kPa were 1.6-fold those of controls (P < 0.05, for strains ≤5%) and strain energy densities were 1.4-fold those of controls (P < 0.05, for strains ≥5%). Histological (phosphotungstic acid hematoxylin) evaluation showed that this stiffening accompanied extensive necrotic damage. Incorporating these effects into the finite element models, we were able to show that the increased muscle stiffness in widening regions results in elevated tissue stresses that exacerbate the potential for tissue necrosis. Interfacial pressures could not predict deep muscle (e.g., longissimus or gluteus) stresses and injurious conditions. We conclude that information on internal muscle stresses is required to establish new criteria for pressure sore prevention.

decubitus ulcers; soft tissue injury; muscle mechanical properties; animal model; finite element analysis

Pressure sores (PS) are soft tissue injuries associated with ischemia, impaired metabolic activity, excessive tissue deformation, and insufficient lymph drainage. Prolonged compression of 250 kPa for >2 h showed loss of muscle fiber cross-striation and infiltration of inflammatory cells, both indicating widespread necrotic cell death (5). Tong and Fung (54) stated that material composition and structure determine the mechanical properties of a tissue, e.g., stiffness. Thus the tissue stiffness is expected to change when composition and structure pathologically change. Specifically, necrosis or partial necrosis in muscular tissue is likely to affect tissue stiffness.

Changes in the mechanical (constitutive) properties of injured muscle tissue may, in turn, affect the distribution of mechanical stresses and strains around the site of injury, thereby potentially exposing additional uninjured regions of muscle tissue to intensified stresses. The study of internal mechanical stresses and strains requires numerical or physical methods such as finite element analysis.
modeling. Focusing on the internal stress state in the buttocks of seated wheelchair users, both finite element (FE) models (41, 53) and instrumented phantom studies (46) concluded that compression and shear stresses in deep soft tissues underlying the bony prominences of the ischial tuberosities were higher than respective contact stresses and could not be predicted by contact pressure measurements. This initial result is important in directing research efforts to the study of deep internal tissue stresses rather than to characterization of interfacial body support stresses, in the context of PS biomechanics. However, these models were limited by assumptions of simple geometry, linear-elastic material behavior of tissues and idealized musculoskeletal loading. Moreover, none of these models could consider the time course of injury, constituted by interdependent effects of local changes in the microstructure and material properties of muscle tissue.

We hypothesize that mechanical properties of striated muscle tissue change in vivo as a result of exposure to prolonged and intensive loads. Such changes may affect the distribution of mechanical stresses in soft tissues under bony prominences and potentially expose additional uninjured regions of muscle tissue to intensified stresses.

The goals of this study were, therefore, 1) to determine changes in mechanical properties of muscles exposed to prolonged intensive compression in vivo as related to histological tissue damage in a rat model and 2) apply the abnormal mechanical properties of injured muscles to computational models of the human body parts vulnerable to PS, to characterize consequent changes in the state of tissue stresses.

METHODS

Experimental protocol. The following protocol was approved by the Institutional Animal Care and Use Committee (IACUC) of Tel Aviv University and was carried out in compliance with institutional guidelines for care and use of animal models (IACUC approval no. M-0241). Mechanical properties of gracilis muscles of adult Sprague-Dawley male rats \( n = 33 \), age 3–4 mo, weight \( 280 \pm 20 \, \text{g} \) were measured in vitro, shortly after exposure of the limb containing the gracilis muscles to compression in vivo.

Before compressing the limb, rats assigned for this purpose were anesthetized with ketamine (90 mg/kg) and xylazine (10 mg/kg) injected intraperitoneally, and one-third of this dose was used for maintenance of the level of anesthesia during experiments. Depth of anesthesia was verified by lack of pinch response. Hair of the compressed limb was carefully shaved and care was taken not to damage the skin during shaving. Anesthetized rats were placed on a specially designed apparatus containing a spring-derived rigid plastic indenter (diameter 20 mm) that applied precalibrated constant pressure on the gracilis of the intact right hindlimb in vivo (Fig. 1A). The gracilis muscle was selected because a relatively large surface of it could be subjected to pressure with this apparatus and because it is located superficially, which made it possible to harvest it by cutting both tendons without damaging muscular tissue at the time of dissection. Groups of three to four animals were exposed to pressure magnitudes of 11.5, 35, and 70 kPa (86, 262, and 525 mmHg) for 2, 4, and 6 h (Table 1). These pressure values were determined to cover the range between the rat (and human) diastolic blood pressure and the near-maximal internal compression stress values in the longissimus muscles of the human pelvis during recumbency as predicted in our FE simulations, reported later on. It should be noted that focal internal compression stresses in human muscles (30–100 kPa) that occur in relatively small regions (5–100 mm\(^2\)) were reproduced over a substantially larger surface in the rat muscle (315 mm\(^2\)) to allow manifestation of their effect on mechanical properties by using standard uniaxial tension testing.

After limbs were compressed, animals were euthanized with an overdose of KCl that was injected intracardially. Control rats were euthanized without prior intervention. The gracilis muscles were harvested from the uninjured (control) and injured limbs (Fig. 1B). All muscles were kept in saline tubes at 3°C until mechanical testing and were tested within no more than 18 ± 2 min from the time of dissection. The testing procedure lasted ~10 min per muscle.

Length, volume, and weight of each muscle were recorded with use of a digital caliper (resolution 0.1 mm), a measuring tube (resolution 0.5 ml), and a digital scale (resolution 0.01 g), respectively. Muscles were then mounted within an Instron 5544 uniaxial tension system with their tendons firmly fixed between customized jigs that were covered with sandpaper to prevent slipping (Fig. 1, C and D). Tension was applied to the muscles within a transparent aquarium (Fig. 1D) filled with saline at the rat’s body temperature (33°C). A load cell with capacity of 2 kN and resolution of 0.01 N was used for measuring the tensile forces applied to the gracilis muscles. Load-deformation curves were obtained for the injured and uninjured (control) muscles at an extension rate of 1 mm/min. Deformation was visually monitored and recorded with both digital and analog video systems for postexperiment slow motion analysis, in which it was verified that muscles did not slip off the grippers. Plots of Lagrange stress (force divided by the original mean cross-sectional area) vs. true strain (calculated from transient distance between jigs) were derived from the load-deformation curves. The original mean cross-sectional area of muscles was obtained by dividing the volume of the muscle by its unloaded length.

Tangent moduli of elasticity and strain energy densities (SED) were calculated from every stress-strain curve at strain levels of 2.5, 5, and 7.5%, by differentiating polynomial functions of the fourth-order that were fitted to the experimental data \( R^2 = 0.99 \) and by calculating the area under the curves at each strain level, respectively. The range of tissue strains, 2.5–7.5%, conformed our FE simulations of the strain distribution in the longissimus and gluteus muscles of the pelvis during recumbency, as reported in the following sections.

Statistical analysis of measured mechanical properties. Tangent elastic moduli and SED of control (uninjured) and injured gracilis muscles were first evaluated via a two-way ANOVA (Systat v10.2). At each strain level, the ANOVA tested the dependence of tangent elastic moduli and SED (in separate analyses) on the factors of pressure magnitude and duration of exposure. A \( P \) value <0.05 was considered statistically significant. None of the ANOVA tests showed dependence of properties on the exposure time (2, 4, or 6 h), but pressure was a significant factor. Post hoc Tukey-Kramer tests across pressure magnitudes were then used to perform pairwise multiple comparisons between properties obtained from muscles exposed to pressures of 0 (control), 11.5, 35, and 70 kPa. Criteria for exclusion of samples from the statistical analysis included apparent damage caused by surgical tools during dissection, tearing of muscle tissue in the immediate vicinity of the grippers, and slipping of tendons from the grippers that was observed in the video analyses.

Histological evaluation of muscles. Cubic samples (face length ~10 mm) from gracilis muscles of the eight rats assigned for histological evaluation (Table 1) were harvested distally and proximally from the site of compression and fixed in formalin. For noncontrol animals, samples were extracted immediately after delivery of compression. Slicing of the tissue was carried out perpendicularly to the direction of load, at thickness of 5 \( \mu \text{m} \) (diameter of muscle fibers is ~30 \( \mu \text{m} \)), and all slices were saved. Sections were mounted on objective slides and stained with hematoxylin and eosin, toluidine blue, and phosphotungstic acid hematoxylin (PTAH) to explore the viability of cells and integrity of cross-striation immediately after delivering the selected pressure doses.

FE modeling for stress analysis of muscles. Four three-dimensional (3D) FE models of transverse slices through body parts that are most
vulnerable to PS, i.e., the pelvis, shoulders, heels, and head, were developed (Fig. 2). In the simulations, we considered a recumbent patient (weight 60 kg) lying still in a hospital bed on an elastic mattress with thickness of 15 cm, elastic modulus of 150 kPa, Poisson’s ratio of 0.11, and static friction coefficient \(\mu_s = 0.4\) (60). In the neutral position of the bed, the backrest was inclined 45° to the horizon.

For each anatomic site, a corresponding transverse cryosectional image from the “Visible Human” male digital database was trans-

Fig. 1. Experimental procedure: hindlimb of a rat model of pressure sore being compressed (A), the harvested gracilis muscle postcompersion (B), apparatus for uniaxial tension testing (C), and magnification showing the testing aquarium (D).
ferred to a solid modeling software package (SolidWorks 2001). Contours of hard and soft tissues were then detected on each image and segmented to form the cross-sectional geometry (Fig. 2A). Next, each cross section was transformed to a 3D solid model of a slice through the body by projecting it 2 cm along the transverse direction (Fig. 2B). Finally, the solid 3D slices were transferred to a FE solver (NASTRAN 2001) for stress-strain analyses under a musculoskeletal loading system that is typical to recumbency (Fig. 3). For each model, loading included axial skeletal loads \( F_1 \) and \( F_2 \), internal skeletal bending moments \( M_1 \) and \( M_2 \), and friction between the body and the mattress (Figs. 3 and 4, Table 2). The pelvis model included abdominal pressure of 2 kPa (17). The specific weights of all tissues (Table

<table>
<thead>
<tr>
<th>Animal group size (total 6 controls and 35 injured rats)</th>
<th>2 h</th>
<th>4 h</th>
<th>6 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>11.5 kPa</td>
<td>3</td>
<td>3</td>
<td>3(UT) 2(HS)</td>
</tr>
<tr>
<td>35 kPa</td>
<td>3</td>
<td>3(UT) 3(HS)</td>
<td></td>
</tr>
<tr>
<td>70 kPa</td>
<td>3(UT) 2(HS)</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

UT, uniaxial tension; HS, histological evaluation. Size of control group was 6 animals, from which 8 muscles were harvested: 6 control muscles were used for uniaxial tension tests, and 2 control muscles were used for histology.

Fig. 2. Computational modeling of the vulnerable sites of the human body: reconstruction of the geometry from the “Visible Human” male database demonstrated by segmentation of musculoskeletal structures of the shoulders (A) and complete 3-dimensional (3D) solid models (B).
3) were also considered in the stress-strain analyses. The process of calculation of the loading systems for each model is described in detail in the APPENDIX. The models were meshed using hexahedron eight-node elements (Fig. 3).

Skin, muscle, and fat tissues, which underwent larger deformations during recumbency, were assumed to be homogenous, isotropic, and nonlinear-elastic materials, and their properties were fitted to experimental data. For skin, we followed Trelstad and Silver (55) and fitted a second-order polynomial relation to their measurements, where \( \lambda \) is the stretch ratio (deformed to initial specimen length) required to produce a stress \( \sigma \) in the tissue (Eq. 1). Using the same approach, we followed Gefen et al. (25) for fat and fitted a third-order polynomial to their data (Eq. 2), and we used our experimental results from rats for the constitutive equation of striated muscle (Eq. 3).

\[
\begin{align*}
\sigma_{\text{skin}}(\text{kPa}) &= 2,650\lambda^{2} - 4,870\lambda + 2,227 \\
\sigma_{\text{fat}}(\text{kPa}) &= 2,300\lambda^{3} - 7,470\lambda^{2} + 8,160\lambda - 2,990 \\
\sigma_{\text{muscle}}(\text{kPa}) &= 3,707\lambda^{3} - 12,117\lambda^{2} + 13,261\lambda - 4,850
\end{align*}
\]

All other tissues in the models, which undergo smaller deformations, were assumed to behave as linear-elastic materials with elastic moduli and Poisson’s ratios as specified in Table 3. For each model, we considered the friction between the contact surface and the body (\( \mu_s = 0.4 \)). Nodes at the bottom surface of the mattress were fixed for horizontal and vertical translations. The FE models were applied to predict internal stresses and strains in the uninjured body parts and, subsequently, to predict the stress flow in the injured muscle tissues.

To simulate the empirical data, we increased the tangent moduli of muscle tissue exposed to critical stresses of 40 kPa or over by 60%, locally for the muscle elements subjected to these stresses. The tissue injury threshold of 40 kPa was set on the basis of our animal studies and on FE analysis of the animal experiments, as described in the next sections.

Using a modeling approach similar to the one used to reconstruct the human anatomy, we also developed a FE model of the limb of a rat to determine internal compression stresses within the gracilis muscle for the 11.5, 35, and 70 kPa interfacial pressures applied to the skin of the limb during the experiments (Fig. 5). As for the human FE models, specific weights of all tissues were considered in the stress-strain analysis (Table 3). The rat limb model was similarly meshed with hexahedron eight-node elements, and the rigid plate was assumed to be a fixed foundation. Skin, muscle, and bone tissues were assumed to have the same mechanical properties as in the human FE models (Eqs. 1 and 3, Table 3). The elastic modulus and Poisson’s ratio of the indenter tip were taken as those of standard plastic, 13 GPa and 0.3, respectively (51). The material properties for the rigid plate at the base were of stainless steel (elastic modulus 200 GPa, Poisson’s ratio 0.3; Ref. 14).

Model predictions of contact stress between the body and mattress under the head, shoulders, pelvis, and heels were validated using sets of flexible (thickness 0.13 mm) contact pressure sensors (Flexiforce, Tekscan, range 0–4.4 N, accuracy ±5%). The sensors were placed on a specially designed test bench of a hospital bed covered by a mattress with the same mechanical properties as the one simulated in the human FE models. Fourteen healthy subjects volunteered for the measurements of interfacial body-mattress pressures during recum-
bency (7 women and 7 men, age 29 ± 7, weight 71 ± 16 kg, height 174 ± 9 cm). Prior written consent was obtained from all volunteers before the interfacial body-mattress pressure measurements. Using unpaired t-tests, we verified that peak contact pressures predicted by our set of FE models were statistically indistinguishable from peak pressures generated by the group of recumbent subjects (Table 4).

A sensitivity analysis was run to assess the influence of selected important model parameters on the predictions of stresses and strains in deep tissues during recumbency. The parameters that were altered were the body weight, backrest inclination, fat stiffness, skin stiffness, and muscle stiffness. We altered the values of these parameters, one at a time, and examined the effects on the maximal von Mises stress, maximal principal compression stress, and maximal compression strain in muscle tissue of each model. Alteration of the body weight and backrest inclination affected the values of forces and moments applied to the surfaces of each model (APPENDIX, Fig. 4) and thereby also affected the distributions of internal stresses and strains. Alteration of skin, fat, and muscle stiffness, which also influence the pattern of internal stresses and strains, was carried out by changing the stresses required to produce a given strain level of the tissue by ±20% (for skin and fat) or ±25% (for muscle). We used our present standard deviation of tissue stiffness for muscles and adapted variations reported in the literature for skin (47) and fat (25).

The mechanical properties of the supporting mattress may also influence the interfacial and internal stress distributions. To determine whether stiffness of the mattress affects the stress flow within deep muscles, we modified the elastic modulus of the mattress (Em) across a logarithmic scale (i.e., from 0.1Em to 100Em and 1000Em, where Em = 150 kPa) and calculated internal maximal principal stresses, contact pressure, and contact shear for the pelvis model, in each case.
Table 2. Loading systems for finite element modeling of the sites vulnerable to pressure sores in the human body (corresponding to Figures 3, 4 and the APPENDIX)

<table>
<thead>
<tr>
<th></th>
<th>Pelvis</th>
<th>Shoulders</th>
<th>Head</th>
<th>Heels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skeletal force F1, N</td>
<td>144</td>
<td>21</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Skeletal force F2, N</td>
<td>116</td>
<td>48</td>
<td>11</td>
<td>4</td>
</tr>
<tr>
<td>Skeletal moment M1, N·m</td>
<td>0.1</td>
<td>36</td>
<td>33</td>
<td>0.8</td>
</tr>
<tr>
<td>Skeletal moment M2, N·m</td>
<td>74</td>
<td>1.4</td>
<td>0.3</td>
<td>33</td>
</tr>
<tr>
<td>Static friction force f, N</td>
<td>24</td>
<td>15</td>
<td>1.5</td>
<td>3.8</td>
</tr>
</tbody>
</table>

RESULTS

Mechanical properties of muscle tissue. Using our rat model of PS, we found that muscle tissue generally increases its stiffness as a result of injury caused by exposure to a critical dose of prolonged pressure (Fig. 6). A two-way ANOVA for pressure and time revealed that both tangent elastic moduli and strain energy densities depended on the extent of applied pressure. However, tangent elastic moduli and SED did not depend on the time duration of exposure. Specifically, using Tukey-Kramer pairwise comparisons, we found that tangent moduli of muscles exposed to compression of 35 or 70 kPa for 2 h or more were significantly greater than those of normal, uninjured muscles at strains of 2.5 and 5% ($P < 0.05$), but not at a strain of 7.5% (Fig. 6A). Strain energy densities of muscles exposed to compression of 35 kPa for 2 h or more were significantly greater than those of normal, uninjured muscles at strains of 5 and 7.5% ($P < 0.05$) but not at the smallest (2.5%) strains (Fig. 6B). Tukey-Kramer pairwise comparisons revealed that tangent elastic moduli and SED of muscles exposed to pressure of 11.5 kPa were statistically indistinguishable from uninjured muscles (controls) across all strain levels. Similarly, tangent elastic moduli and SED of muscles exposed to 35 kPa were indistinguishable from those of muscles exposed to 70 kPa across all three strains. According to the later result, we pooled property data of animals exposed to 35 and 70 kPa at each strain level (Fig. 6). We then performed an additional one-way ANOVA, for pressure level only, which indicated, consistent with our previous analyses, that muscles exposed to 35–70 kPa for >2 h were significantly stiffer ($P < 0.03$) than noninjured muscles. The stiffness (in terms of the tangent elastic modulus) of the muscles exposed to 35–70 kPa was greater by 60% in average compared with controls, for strains of 2.5 and 5%. Moreover, SED values were greater for muscles exposed to 35–70 kPa by 40% on average, for strains of 5 and 7.5% ($P \leq 0.03$). Because a stiffer material requires more energy to deform to a given strain level compared with a more compliant material, this further indicates the abnormally increased stiffness of the injured muscles.

Fig. 5. Finite element model of the rat limb: transverse cross-sectional anatomy of the limb segment that was compressed (A), the 3D solid computer model (B), and the 3D mesh for finite element analysis (C).

Histology of muscle tissue under compression. Staining with PTAH was shown to provide the most sensitive visualization of histomorphological changes that were induced by our experimental protocol. From all staining methods that were used in

Table 3. Mechanical properties of tissues for finite element modeling

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Density, g/cm³</th>
<th>Elastic Modulus</th>
<th>Poisson’s Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tissues considered as linear-elastic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortical bone</td>
<td>1.9 [20]</td>
<td>18 GPa [20]</td>
<td>0.3 [28]</td>
</tr>
<tr>
<td>Trabecular bone</td>
<td>0.8 [56]</td>
<td>5 GPa [28]</td>
<td>0.3 [28]</td>
</tr>
<tr>
<td>Colon</td>
<td>1.056 [50]</td>
<td>0.5 MPa [58]</td>
<td>0.5 [53]</td>
</tr>
<tr>
<td>Ileum</td>
<td>1.056 [50]</td>
<td>47 KPa [53]</td>
<td>0.5 [53]</td>
</tr>
<tr>
<td>Blood vessels</td>
<td>1.056 [50]</td>
<td>0.133 MPa [23]</td>
<td>0.4 [23]</td>
</tr>
<tr>
<td>Cartilage</td>
<td>1.1 [44]</td>
<td>10 MPa [42]</td>
<td>0.4 [42]</td>
</tr>
<tr>
<td><strong>Tissues considered as non-linear-elastic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin</td>
<td>1.056 [50]</td>
<td>695 kPa (Eq. 1)</td>
<td>0.4 [24]</td>
</tr>
<tr>
<td>Fat</td>
<td>1.2 [37]</td>
<td>80 kPa (Eq. 2)</td>
<td>0.5 [53]</td>
</tr>
<tr>
<td>Muscle</td>
<td>1.056 [50]</td>
<td>75 kPa (Eq. 3)</td>
<td>0.5 [53]</td>
</tr>
</tbody>
</table>

Numbers in brackets are references.

Table 4. Peak contact pressures: experimental results vs. model predictions

<table>
<thead>
<tr>
<th></th>
<th>Pelvis</th>
<th>Shoulders</th>
<th>Heels</th>
<th>Head</th>
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<tbody>
<tr>
<td>Test-bench measurements</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(r = 14), kPa</td>
<td>12 ± 9</td>
<td>4 ± 2</td>
<td>13 ± 8</td>
<td>5 ± 3</td>
</tr>
<tr>
<td>Model predictions, kPa</td>
<td>10 (NS)</td>
<td>3 (NS)</td>
<td>15 (NS)</td>
<td>5 (NS)</td>
</tr>
</tbody>
</table>

*Subject group included 7 women and 7 men with the following body characteristics: age, 29 ± 7 yr; weight, 71 ± 16 kg; height, 174 ± 9 cm. A t-test was run for each anatomic site to verify that model predictions reflect the normal contact pressure pattern. Significance was set at the 5% level; NS, not significant.
In this study, PTAH was the only one which demonstrated pathological changes for specimens harvested immediately after compression was delivered. The stained slides from animals exposed to interfacial compression of 35 and 70 kPa showed extensive necrotic death of muscle cells that was evident by loss of cross-striation (Fig. 7). Some transitional regions of necrobiosis (partially viable tissue) could also be identified in the group exposed to 70 kPa (Fig. 7). Contrarily to the specimens from muscles exposed to 35–70 kPa, PTAH-stained slides from animals exposed to 11.5 kPa for 6 h were indistinguishable from slides of normal uninjured muscles (controls). Specifically, no evidence of loss of cross-striation or necrosis of muscle tissue was apparent in slides from animals exposed to 11.5 kPa. We conclude that the abnormal 1.6-fold increase in muscle tissue stiffness observed in our uniaxial tension experiments for muscles exposed to pressures of 35 and 70 kPa is accompanied by widespread necrotic damage.

**Computational stress analysis.** According to our rat limb FE model (Fig. 8), the external pressures that were applied on the rat limbs, 11.5, 35, and 70 kPa, induced internal compression stresses of 13, 40, and 80 kPa, respectively. We found abnormal stiffening in muscles that underwent internal compression of 40 kPa or over for 2 h but not in muscles that underwent internal compression of 13 kPa for 6 h. Thus the injury threshold for striated muscle tissue under compression is between 13 kPa delivered for 6 h and 40 kPa delivered for 2 h. Because FE analysis of the injury process in humans requires a definite single value for this threshold, we adopted a conservative assumption (in terms of the rate of diffusion of the injury) and used the greater injury threshold limit, i.e., 40 kPa delivered for 2 h.

In general, peak stresses in deep muscles under the bony prominences of the human pelvis and shoulders were shown to be greater (up to 35-fold for the shoulders model) than respective peak contact stresses (Fig. 9). Maximal von Mises stresses in the normal, uninjured musculature of the pelvis during recumbency were 290 and 150 kPa in the longissimus and gluteus muscles, respectively (Fig. 9A), and were substantially greater than peak contact pressure between the skin and the...

![Fig. 6. Mechanical property data for the normal and injured gracilis of the rat: tangent elastic moduli ($E_t$; A) and strain energy densities (SED; B). Cont, control (uninjured muscles). Data for experimental groups exposed to 35 and 70 kPa were pooled after Tukey-Kramer tests showed that the mechanical properties obtained from these groups were statistically indistinguishable. *Statistically significant differences ($P < 0.05$) between properties from different groups.](http://jap.physiology.org/)

![Fig. 7. Representative phosphotungstic acid hematoxylin (PTAH) staining of control rat muscle tissue (A) and rat muscle tissue injured by exposure to 70 kPa for 2 h (B). Within the frame shown, ~30% of the muscular tissue was identified as necrotic (unstained, e.g., region A) and the other ~70% was viable tissue (stained blue, e.g., regions B). Magnification was set as ×300.](http://jap.physiology.org/)
supporting surface (11 kPa). Both the longissimus and gluteus peak stresses are to exceed the injury threshold obtained for striated muscle in the rat if applied for 2 h or more. Similarly, maximal von Mises stresses in the shoulders during recumbency were 222, 840, and 872 kPa in the infraspinatus, supraspinatus, and subscapularis muscles, respectively (Fig. 9B), exceeding the peak contact pressure (25 kPa) by at least an order of magnitude. The occipitofrontalis muscle in the head model, however, was shown to bear maximal von Mises stress of 21 kPa (Fig. 9D), which is in the same range as the 14 kPa peak contact pressure between the skin of the scalp and the mattress. The only case in which interfacial pressures were greater than internal stresses was for the heel model. Interfacial stresses between the skin around the posterior aspect of the calcaneus and the mattress (50–75 kPa) were three- to fivefold higher with respect to internal stresses in fat tissue around the Achilles tendon (10–25 kPa) (Fig. 9C).

Maximal strain magnitudes in deep muscles of the pelvis, shoulders, head, and heels were between 0.2 and 8% (Fig. 10A). This range of peak strains justifies our selection to experimentally test material properties of the gracilis muscle of the rat at strain levels of 2.5, 5, and 7.5%.

The internal stress distributions in soft tissues are evolving with time during recumbency because of the abnormal stiffening of damaged muscular tissue that was subjected to compression stress of 40 kPa or over for 2 h or more. If certain regions of muscular tissue are damaged (by lack of nutrients and/or excessive cell deformation and/or deficient waste clearance), and, thereby, the local stiffness is increased, the global stress flow in the tissue is also expected to change. Accordingly, the FE simulations predicted that 2 h after a patient was put in a recumbent position, regions in the longissimus (pelvis) that were exposed to compression stresses of 30 kPa or over expand in area by 30% (Fig. 10B). Similarly, regions in the longissimus exposed to lower stresses of 8 and 10 kPa expand in area by 13 and 50%, respectively (Fig. 10B). Thus the PS injury is diffused to the regions more recently exposed to stresses that may exceed the critical injury threshold.

Table 5 details the sensitivity of our predictions of stresses and strains in the human musculature to variations in values of the model parameters. Skin stiffness, fat stiffness, and muscle stiffness all had a minor effect (<6.6%) on peak muscle stresses in the pelvis, shoulders, and head models. Peak von Mises stress in the posterior subcutaneous tissue of the heel model, however, varied by 15% for the ±20% extent of change in fat and skin stiffness. The maximal compressive strains in the longissimus muscle (pelvis) and occipitofrontalis muscle (head) were sensitive to variations in the muscle stiffness and increased by 14 and 35%, respectively, when the overall stiffness of muscle tissue was increased by 25%. Stiffness of the fat tissue also had substantial influence on maximal muscle strains. The maximal strain in the longissimus muscle was amplified by 33% when the fat stiffness increased by 40%. The parameters that had the most dominant effect on stress and strain predictions were the body weight and the backrest inclination, and both affected mostly the results from the pelvis model. Additional 40 kg of body weight increased the maximal von Mises stress, maximal compression stress, and maximal compression strain in the longissimus muscles of the pelvis by 87, 89, and 67%, respectively. A backrest angle (β) of 60° (Fig. 4) produced ~1.6-fold higher peak stresses and ~1.35-fold higher peak strains in the deep musculature of the pelvis compared with a 45° inclination. For example, maximal von Mises stress, maximal compression stress, and maximal compression strain in the longissimus increased by 68, 56.8, and 35.4%, respectively, for β = 60°. Consistently, lower backrest inclination of β = 30° decreased the maximal von Mises stress, maximal compression stress, and maximal compression strain in the longissimus by 117, 43.5, and 29.3%, respectively. We conclude that internal stresses and strains in deep muscles are sensitive to the posture of the body and can be relieved if the posture is adequately changed.

We repeated the stress analyses of the pelvis for a more compliant (0.1Em) and for stiffer (10Em, 100Em) mattresses. Internal maximal principal stresses in the longissimus muscles increased from 117 to 132 kPa when the stiffness of the mattress was reduced from 100Em to 0.1Em. Unlike peak internal stresses, the contact pressure and shear were strongly
affected by the rigidity of the mattress (Fig. 11). For example, the peak contact pressure for the softest \((0.1E_m)\) mattress (predicted to be 8.5 kPa) increased by a factor of 1.6 when the mattress was stiffened to \(100E_m\) (Fig. 11). This indicates that interfacial pressures are a poor, inefficient indicator for deep muscle stresses. Attempts to predict the risk for PS that affect deep muscles on the basis of interfacial pressures are therefore naive.

DISCUSSION

In this study, we employed animal and computer models to demonstrate that deep muscle tissue that undergoes prolonged compression may significantly increase its stiffness during injury. The injured, stiffer tissue bears elevated stresses and projects these stresses to adjacent tissue that was not yet injured, thereby exposing it to the potentially damaging critical pressure dose (the threshold used in this study is internal compression of 40 kPa delivered for 2 h). This may drive a positive-feedback mechanism in muscle PS in which the elevated stresses in widening regions around the bone-muscle interface (Fig. 10) increase the potential for muscle tissue necrosis.

Specifically, our experimental results demonstrated a statistically significant increase in the tissue’s elastic moduli (average rise of 60%) as well as a significant increase in SED (average rise of 40%) as a result of prolonged compression (Fig. 6). We demonstrated, by means of PTAH histology, that these changes in constitutive properties are associated with extensive cell death and tissue necrosis. This finding of abnormally stiff mechanical properties that accompany cell death agrees with previous in vitro studies, which reported of stiffer properties of excised soft tissues postmortem, including cartilage (1.5-fold stiffening, Ref. 21), plantar fascia (1.2-fold stiffening, Ref. 26), and brain (1.2-fold stiffening, Ref. 45). The mechanism causing the stiffening of muscle tissue can be an increase in the swelling pressure of the tissue, which may follow destruction of cell membranes during necrotic cell death due to ischemia (29). Additional damage other than ischemia could have been produced by lack of tissue drainage (39) and excessive tissue deformation (8), but our histology and mechanical testing protocols could not isolate the contribution of each damage cause to cell death and abnormality of mechanical properties. However, we expect the same phenomenon of muscle tissue stiffening in vivo, in humans, during PS onset when muscles become partially necrotic under prolonged bone compression.

Kovanen et al. (35) measured tangent elastic moduli of 47 to 104 kPa for soleus and rectus femoris muscles of rat that were tested under tension in strains lower than 7.5%. Bosboom et al. (6) measured instantaneous shear modulus of 15.6 ± 5.4 kPa for tibialis anterior rat muscles. Utilizing the relation \(G = (E/2)(1 + \nu)\), where \(G\) is the shear modulus, \(E\) is the elastic
bridging of muscle experimental groups requires that the potential effect of cross-SD for 7.5% strain, Fig. 6).

results from previous studies in which mechanical properties of muscles, but our experimental results for controls overlap frame, it should have led to elevated stiffness in control killed. If some mild rigor mortis did occur within this time-

range (0.1

modulus, and \(v = 0.5\) is Poisson’s ratio for an incompressible material, it is shown that their tangent elastic moduli under large strains (>5%) are in the range of 30–63 kPa. Thus both Kovanen et al. and Bosboom et al. reported properties for normal rat limb muscles that overlap our measurements of normal properties of the gracilis muscle: 46–73 kPa (mean ± SD for 7.5% strain, Fig. 6).

Our observation of stiffening of muscle tissue in some of the experimental groups requires that the potential effect of cross-

bony prominences of the pelvis and shoulders bear substantially higher internal stresses compared with body-
support interfacial pressures (Fig. 9). We found that peak stresses (principal compression) in the longissimus muscles (Fig. 9A) increased by 13% (from 117 to 132 kPa) when the stiffness of the mattress was reduced over three orders of magnitude. It is important to emphasize that even though the stiffness of the mattress was decreased across such a broad range (0.1\(E_m\)=100\(E_m\), principal compression stresses in the longissimus increased and remained highly above the 40 kPa stress threshold for PS. Contact pressures and contact shear of 8 (A), 10 (B), and 30 kPa (C) expand in area by 13, 50, and 30%, respectively.

A

Fig. 10. A: principal compression strains in the normal longissimus muscles. Strain levels in the longissimus of a recumbent subject are predicted to range between 0.5 and 6%. B: principal compressive stresses in the uninjured and injured longissimus muscles. Internal stresses in the longissimus evolve owing to abnormal stiffening of injured muscle tissue so that regions subjected to compression of 8 (A), 10 (B), and 30 kPa (C) expand in area by 13, 50, and 30%, respectively.

B

internal pressures using a needle connected to an interstitial fluid pressure (IFP) transducer. Muscles subjected to pressure of 4.6 kPa for up to 4 h and those subjected to 25.3 kPa for up to 1 h appeared normal. Contrarily, muscles exposed to pressure of 10 kPa for 2 h were damaged. Puncture of the tissue with the IFP needle could, however, cause some of the observed damage or increase the tissue’s sensitivity to pressure. Salcido et al. (49) reported macroscopic lesions in the panniculus carnosus muscle of rats that were associated with cutaneous ulceration after exposure to external pressure of 20 kPa for 6 h. In the most recent study, Bosboom et al. (5) used hematoxylin and eosin staining and IFP measurements to evaluate the pressure tolerance of the tibialis anterior tissue in a rat model of PS. They found that application of either 10 or 70 kPa for 2 or 6 h induced loss of cross-striation when combined with IFP measurements. When IFP measurements were excluded from their protocol, histological damage did not appear. Damage did appear when the pressure level was increased to 250 kPa and was more pronounced when IFP measurements were again incorporated for this pressure level. They suspected that insertion of the IFP needle affected the occurrence of damage and that it reduced the injury thresholds of muscle in their study, as well as in the study of Kosiak. Our results for the injury threshold agree with those of Salcido et al., who avoided an IFP needle. The use of a FE model of the animal experiment provided the level of internal tissue stresses in the present study (Fig. 8) and allowed avoidance of invasive IFP measurements.

The human FE models demonstrated that deep muscles under bony prominences of the pelvis and shoulders bear substantially higher internal stresses compared with body-support interfacial pressures (Fig. 9). We found that peak stresses (principal compression) in the longissimus muscles (Fig. 9A) increased by 13% (from 117 to 132 kPa) when the stiffness of the mattress was reduced over three orders of magnitude. It is important to emphasize that even though the stiffness of the mattress was decreased across such a broad range (0.1\(E_m\)=100\(E_m\), principal compression stresses in the longissimus increased and remained highly above the 40 kPa stress threshold for PS. Contact pressures and contact shear between the pelvis and mattress, however, substantially decreased, from peaks of 13.8 and 2.4 kPa to 8.2 and 1.4 kPa, respectively. This indicates that no simple relations exist between the internal and interfacial stresses for the body-support
contact problem. Previous computational models of the diabetic foot (27) and buttocks (11) also showed that internal stresses are higher than interfacial pressures and that no simple relation between contact pressure and tissue stress can be established (53). The above findings are important in view of the efforts put in predicting risks for PS on the basis of interfacial pressures. For example, interfacial pressures are used to decide about timing for changing postures and for selection of materials for hospital mattresses (10, 15, 22). On the basis of the present simulations and published literature (11, 27, 53), we conclude that contact pressure measurements are insufficient for design of patient management procedures and characterization of mattresses for preventing PS. Alternatively, an insight into the internal stress state under the bony prominences is necessary.

With respect to internal stresses in recumbent humans, we showed that when the backrest angle is decreased, from 45 to 30°, the effect on reducing internal muscle stresses under the pelvis is much more profound and consistent than that caused by decreasing the mattress stiffness (Table 5). When the backrest slope was decreased by 15°, maximal internal stresses in the longissimus, von Mises (377 kPa) and principal compression (125 kPa), decreased by 117% (to 192 kPa) and 43% (to 71 kPa), respectively (Table 5). We conclude that some relief of internal compression in the deep musculature of the pelvis can be achieved through a decrease of the backrest inclination, but internal compression would still be higher than the 40 kPa injury threshold for PS.

Limitations of the present study include measurement of mechanical properties of a complete muscle whereas damage may be localized at certain regions within the muscle, and the assumption that soft tissues in the FE models are homogenous and nonlinear elastic rather than viscoelastic. The mechanical testing of gracilis muscles in this study involved application of tension to the complete muscles including tendon insertions and perimysium. The perimysium, which is primarily connective tissue (collagen, with an elastic modulus of 110 GPa), has a great resistance to tension and thus it is likely that it contributed substantially to the tangent moduli of elasticity. Despite this, our measurements of mechanical properties were sensitive to abnormalities caused by prolonged compression and revealed statistically significant stiffening of muscular tissue postcomprssion with a sufficient statistical power for experimental groups of four to five animals. Muscle tissue was considered in the FE models as a composite comprised of the perimysium, endomysium, and muscle fibers, and stress-strain predictions therefore describe an apparent behavior that cannot be used to isolate deformation of individual muscle fibers, although overstretching of individual fibers may be important in the onset of PS injury (8). The assumption that tissues are elastic and not viscoelastic may have caused some overestima-

![Fig. 11. Influence of mattress stiffness on contact pressure (A) and contact shear distributions (B) between the pelvis and the mattress during recumbency. The elastic modulus of the mattress (E_m) equals 150 kPa.](http://j NagtDy66dp.org)
tion of stresses, because stress relaxation in the long term was not considered. However, the assumption of nonlinear elasticity of soft tissues allowed assignment of considerable computational efforts for obtaining accurate anatomical representations of the pelvis, shoulders, heels, and head, because the computational complexity of the material models was compromised.

In closure, integration of animal and FE models provides a powerful tool for studying PS onset and progression. It also has the potential of being an aid in design of seats and beds with protective supporting surfaces as well as in designing new patient management procedures.

Conclusions. Using an integrated approach of animal and computer model studies of the biomechanics of PS, we found the following. 1) Maximal internal principal compression and von Mises stresses at deep muscles around the bony prominences of the pelvis and shoulders exceed the interfacial contact stresses by at least an order of magnitude. Specifically for the pelvis region, we found no correlation between external pressures and internal muscle stresses. Surprisingly, a more compliant mattress was shown to increase deep muscle stresses instead of relieving them. A lower (30°) backrest angle could decrease these deep muscle stresses, but only to a limited extent. 2) Tangent elastic moduli and SED of rat gracilis muscles injured by exposure of 2–6 h to external pressure of 35–70 kPa, which is transformed to internal muscle compression of 40–80 kPa, were 60 and 40% higher in average (P < 0.04), respectively, than those of uninjured muscles. Abnormal properties were accompanied by widespread necrotic cell death. 3) On the basis of these findings, we conclude that mechanical properties of striated muscle can be used as an indicator for a compression injury. 4) Taken together, our histological and mechanical testing results indicate that the injury threshold for onset of PS in skeletal muscles is between internal compression of 13 kPa delivered for 6 h and 40 kPa delivered for 2 h. 5) Incorporating the effect of muscle stiffening into a computational stress analysis of the pelvis during recumbency, we were able to demonstrate a positive-feedback mechanism: the increased muscle stiffness in widening regions results in elevated tissue stresses and thereby exacerbate the potential for tissue necrosis.

APPENDIX

Glossary

<table>
<thead>
<tr>
<th>Model</th>
<th>Symbol</th>
<th>Description</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pelvis</td>
<td>α</td>
<td>Angle of the spinal force vector in the sagittal plane</td>
<td>13°</td>
</tr>
<tr>
<td></td>
<td>β</td>
<td>Backrest inclination</td>
<td>45°</td>
</tr>
<tr>
<td></td>
<td>x₁</td>
<td>Distance between the pelvis slice and the FBD center of mass</td>
<td>0.3 m</td>
</tr>
<tr>
<td></td>
<td>y₁</td>
<td>Distance between the spinal force vector and the mattress</td>
<td>0.07 m</td>
</tr>
<tr>
<td>Shoulders</td>
<td>x₂</td>
<td>Distance between the pelvis slice and the FBD center of mass</td>
<td>0.8 m</td>
</tr>
<tr>
<td></td>
<td>y₂</td>
<td>Distance between the spinal force vector and the mattress</td>
<td>0.06 m</td>
</tr>
<tr>
<td>Head</td>
<td>x₃</td>
<td>Distance between the shoulders slice and spinal force vector</td>
<td>0.6 m</td>
</tr>
<tr>
<td></td>
<td>y₃</td>
<td>Distance between the shoulders slice and the FBD center of mass</td>
<td>0.2 m</td>
</tr>
<tr>
<td></td>
<td>x₄</td>
<td>Distance between the spinal force vector and the backrest</td>
<td>0.06 m</td>
</tr>
<tr>
<td></td>
<td>y₄</td>
<td>Distance between the spinal force vector and the backrest</td>
<td>0.08 m</td>
</tr>
<tr>
<td></td>
<td>x₅</td>
<td>Distance between the backrest and the FBD center of mass</td>
<td>0.05 m</td>
</tr>
<tr>
<td></td>
<td>y₅</td>
<td>Distance between the head slice and the FBD center of mass</td>
<td>0.2 m</td>
</tr>
<tr>
<td></td>
<td>x₆</td>
<td>Distance between the head slice and the FBD center of mass</td>
<td>0.06 m</td>
</tr>
<tr>
<td></td>
<td>y₆</td>
<td>Distance between the head slice and the FBD center of mass</td>
<td>0.04 m</td>
</tr>
<tr>
<td></td>
<td>x₇</td>
<td>Distance between the head slice and the FBD center of mass</td>
<td>0.05 m</td>
</tr>
<tr>
<td></td>
<td>y₇</td>
<td>Distance between the head slice and the FBD center of mass</td>
<td>0.035 m</td>
</tr>
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<td>x₈</td>
<td>Distance between the head slice and the FBD center of mass</td>
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<tr>
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<td>y₈</td>
<td>Distance between the head slice and the FBD center of mass</td>
<td>0.2 m</td>
</tr>
<tr>
<td></td>
<td>x₉</td>
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<td>0.85 m</td>
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<tr>
<td></td>
<td>y₉</td>
<td>Distance between the head slice and the FBD center of mass</td>
<td>0.06 m</td>
</tr>
<tr>
<td></td>
<td>x₁₀</td>
<td>Distance between the head slice and the FBD center of mass</td>
<td>0.025 m</td>
</tr>
</tbody>
</table>

The above data represent a normal adult man and are adopted from Clauser et al. (13) and Morse and Kjeldsen (40). FBD, free body diagram.
Table 7. Weights of body segments used to solve the loading systems for finite element modeling of the sites vulnerable to pressure sores in the human body

<table>
<thead>
<tr>
<th>Model</th>
<th>Symbol</th>
<th>Body Segments</th>
<th>Total Weight, N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pelvis</td>
<td>Wp</td>
<td>Lower pelvis and legs</td>
<td>276</td>
</tr>
<tr>
<td></td>
<td>W2</td>
<td>Upper pelvis, trunk, arms, and head</td>
<td>311</td>
</tr>
<tr>
<td></td>
<td>Wp</td>
<td>Pelvis slice</td>
<td>20</td>
</tr>
<tr>
<td>Shoulders</td>
<td>Ws</td>
<td>Lower shoulders, trunk, and arms</td>
<td>282</td>
</tr>
<tr>
<td></td>
<td>W1</td>
<td>Upper shoulders and head</td>
<td>107</td>
</tr>
<tr>
<td></td>
<td>Ws</td>
<td>Pelvis slice</td>
<td>60</td>
</tr>
<tr>
<td>Head</td>
<td>Whd</td>
<td>Lower head, trunk, and arms</td>
<td>306</td>
</tr>
<tr>
<td></td>
<td>Whd</td>
<td>Upper head</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>Whd</td>
<td>Head slice</td>
<td>6</td>
</tr>
<tr>
<td>Heel</td>
<td>Ws</td>
<td>Feet</td>
<td>16.8</td>
</tr>
<tr>
<td></td>
<td>Ws</td>
<td>Legs, thighs, and pelvis</td>
<td>260</td>
</tr>
<tr>
<td></td>
<td>Ws</td>
<td>Heel slice</td>
<td>2</td>
</tr>
</tbody>
</table>

The above data represent a normal adult male and are adopted from Clauer et al. (13) and Morse and Kjeldsen (40). The positions of the weight vectors are shown in Fig. 4. and the method of calculation is provided in the Appendix.

To calculate the static friction force between the upper body (Fig. 4B) and mattress $f_1^u$:

$$f_1^u = N_1^u \mu. \quad (2.7)$$

We solve for $N_1^u$, $F_1^u$, $M_1^u$:

$$N_1^u = \frac{F_1^u \cos \alpha}{\mu} \quad (3.4)$$

$$F_1^u = \frac{W_1^u \mu}{\cos \alpha - \mu \sin \alpha} \quad (3.5)$$

$$M_1^u = x_1^u(N_1^u - W_1^u) - N_1^u y_1^u \quad (3.6)$$

and calculate the static friction force between the upper body and the mattress $f_1^u$:

$$f_1^u = N_1^u \mu. \quad (1.7)$$

Similarly, from static equilibrium of forces and moments on the free body diagram of the upper body part located above the slice that was selected for the pelvis model (Fig. 4B):

$$\sum F_y = 0: f_1^u + F_2^u \cos \beta - W_2^u \sin \beta = 0 \quad (2.1)$$

$$\sum F_y = 0: N_1^u - W_1^u \cos \beta - F_2^u \sin \beta = 0 \quad (2.2)$$

$$\sum M_y = 0: -M_2^u - W_2^u x_2^u \cos \beta + f_2^u y_2^u + N_2^u x_2^u = 0 \quad (2.3)$$

We solve for $N_2^u$, $F_2^u$, $M_2^u$:

$$N_2^u = F_2^u \sin \beta + W_2^u \cos \beta \quad (4.4)$$

$$F_2^u = \frac{W_2^u \sin \beta - W_2^u \mu \cos \beta}{\mu \sin \beta + \cos \beta} \quad (4.5)$$

$$M_2^u = (N_2^u - W_2^u \cos \beta)x_2^u + N_2^u y_2^u \quad (4.6)$$

and calculate the static friction force between the head-neck-shoulder part (Fig. 4D) and mattress $f_2^u$:

$$f_2^u = N_2^u \mu. \quad (4.7)$$

We obtain the friction force under the shoulders slice $f^p$ from balance of forces on this slice:

$$f^p = F_1^p - F_2^p + W^p \sin \beta \quad (4.8)$$

**Shoulders model.** The method of derivation is similar to the one used to obtain the loading system for the pelvis model. We start with static equilibrium for the free body diagram of the trunk, considering only the trunk segment below the slice used to model the shoulders (Fig. 4C):

$$\sum F_y = 0: F_1^u \sin \alpha + f_1^u \sin \beta - W_1^u + N_1^u \cos \beta - F_1^u \sin \beta = 0 \quad (3.2)$$

$$\sum M_y = 0: M_1^u + f_1^u y_1^u + W_1^u x_1^u \cos \beta - N_1^u x_1^u - M_1^u \quad (3.3)$$

and calculate the static friction force between the trunk part that is under the shoulders slice (Fig. 4C) and mattress $f_1^u$:

$$f_1^u = N_1^u \mu. \quad (3.7)$$

where $F_1^u$ and $M_1^u$ in Eqs. 3.1–3.6 are calculated from Eqs. 1.5 and 1.6, respectively.

We continue with static equilibrium of the free body diagram of the head, neck, and segment of the shoulders located above the shoulders slice model (Fig. 4D):

$$\sum F_y = 0: F_2^u + f_2^u - W_2^u \sin \beta = 0 \quad (4.1)$$

$$\sum F_y = 0: N_2^u - W_2^u \cos \beta = 0 \quad (4.2)$$

$$\sum M_y = 0: -M_2^u + N_2^u x_2^u - W_2^u x_2^u \sin \beta + f_2^u y_2^u = 0 \quad (4.3)$$

Finally, from static equilibrium of forces and moments on the free body diagram of the upper body part located above the slice that was selected for the pelvis model (Fig. 4B):

$$\sum F_y = 0: f_1^u + F_2^u \cos \beta - W_2^u \sin \beta = 0 \quad (2.1)$$

$$\sum F_y = 0: N_1^u - W_1^u \cos \beta - F_2^u \sin \beta = 0 \quad (2.2)$$

$$\sum M_y = 0: -M_2^u - W_2^u x_2^u \cos \beta + f_2^u y_2^u + N_2^u x_2^u = 0 \quad (2.3)$$

We solve for $N_2^u$, $F_2^u$, $M_2^u$:

$$N_2^u = F_2^u \sin \beta + W_2^u \cos \beta \quad (2.4)$$

$$F_2^u = \frac{W_2^u \sin \beta - W_2^u \mu \cos \beta}{\mu \sin \beta + \cos \beta} \quad (2.5)$$

$$M_2^u = (N_2^u - W_2^u \cos \beta)x_2^u + N_2^u y_2^u \quad (2.6)$$

and calculate the static friction force between the upper body and the mattress $f_2^u$:

$$f_2^u = N_2^u \mu. \quad (2.7)$$

**Shoulders model.** The method of derivation is similar to the one used to obtain the loading system for the pelvis model. We start with static equilibrium for the free body diagram of the trunk, considering only the trunk segment below the slice used to model the shoulders (Fig. 4C):

$$\sum F_y = 0: F_1^u \sin \alpha + f_1^u \sin \beta - W_1^u + N_1^u \cos \beta - F_1^u \sin \beta = 0 \quad (3.2)$$

$$\sum M_y = 0: M_1^u + f_1^u y_1^u + W_1^u x_1^u \cos \beta - N_1^u x_1^u - M_1^u \quad (3.3)$$

We solve for $N_1^u$, $F_1^u$, $M_1^u$:

$$N_1^u = \frac{F_1^u \cos \beta - F_1^u \cos \alpha}{\mu \cos \beta - \sin \beta} \quad (3.4)$$

$$F_1^u = F_1^u(\mu \cos \alpha \sin \beta + \sin \alpha \sin \beta + \cos \alpha \cos \beta \quad (3.5)$$

$$M_1^u = M_1^u + N_1^u x_1^u - W_1^u \cos(90 - \beta + \alpha) \quad (3.6)$$

$$- W_1^u \cos \beta - N_1^u \mu y_1^u \quad (3.7)$$

and calculate the static friction force between the trunk part that is under the shoulders slice (Fig. 4C) and mattress $f_1^u$:

$$f_1^u = N_1^u \mu. \quad (3.7)$$

where $F_1^u$ and $M_1^u$ in Eqs. 3.1–3.6 are calculated from Eqs. 1.5 and 1.6, respectively.
We continue with equilibrium of the free body diagram of the legs, to account for forces and moments acting above the slice of the heel model (Fig. 4H)

\[
\sum F_x = 0: F_1^h + F_2^h - F_1^s \cos \alpha = 0 \\
\sum F_y = 0: N_2^h - W_2^h - F_2^s \sin \alpha = 0 \\
\sum M_0 = 0: -M_2^h + N_2^h x_2 - W_2^h x_2 + f_2 y_2^h = M_2^h + F_2^h \cos \alpha - F_1^h \sin \alpha = 0
\]

We solve for \( N_2^h, F_1^h, M_2^h \)

\[
N_2^h = W_2^h + F_2^h \sin \alpha \\
F_1^h = F_1^h(\cos \alpha - \mu \sin \alpha) - W_2^h \mu \\
M_2^h = N_2^h x_2 - W_2^h x_2 + N_2^h y_2^h + M_1^h + F_2^h \cos \alpha - x_2^h \sin \alpha
\]

and calculate the static friction force between the legs (Fig. 4H) and mattress \( f_2^s \)

\[
f_2^s = F_2^s - F_2^h
\]

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