Investigating pressure induced deep tissue injury uing MRI and 3D finite element analysis

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INVESTIGATING PRESSURE INDUCED DEEP TISSUE INJURY USING MRI AND 3D FINITE ELEMENT ANALYSIS

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INTRODUCTION

Pressure ulcers occur as a result of (prolonged) mechanical loading. They represent a common complication in the management of a range of patients and can be considered as an indicator of quality of healthcare. In many cases, pressure ulcers are avoidable [1]. Currently the proposed mechanisms for pressure ulcer formation are: 1) ischemia, 2) direct deformation, 3) ischemia-reperfusion injury, and 4) lymphatic blockage [2]. To understand how these processes induce pressure ulcer formation controlled experiments can unravel the specific temporal and spatial effects of each of these mechanisms. In the past a combined experimental-numerical approach has been used. A load was applied via an indenter to the tibialis anterior muscle (TA) of the hind leg of Brown-Norway rats. The experiment was monitored with magnetic resonance imaging (MRI), providing data on changes in tissue geometry and physiology. By correlating finite element analysis (FEA) and T_2-weighted MRI a threshold level of applied deformation energy was identified above which damage occurred [3]. However, this study was limited to a local 2D analysis directly under the indenter. The aim of the current study was to determine the global effects of loading muscle tissue by extending the analysis to 3D. The experimental protocol was adapted accordingly and 3D FEA was performed.

METHODS

Experimental: The TA of Sprague- Dawley rats (♀, n=7) was loaded for 2 hours using an indenter with a spherical head (diameter: 3 mm, Fig 1). Before, during and after loading MRI was performed using a 7T Bruker small animal scanner with a 86mm excitation coil and 20mm diameter receiver coil. Anatomy and geometry was determined by T_1 weighted MRI and physiological changes with T_2 mapping MRI (FOV 25x25x20 mm, MTX 256x256x20, T_1: TE/TR 11.50/800.0 ms, T_2: TE 6.95-180.7 ms, 26 echos).

Damage/T_2-analysis: T_2-maps ware obtained by fitting the MR signal (S) voxelwise to equation (1). The TA was manually drawn on the T_2-scans ro represent the region of interest (ROI). Voxels were accepted for analysis if the $R^2 > 0.8$ and SNR > 4. Slices were accepted for further analysis if >90% of the voxels in the ROI fulfilled the previous criteria. The mean and standard deviation of the mean (SD_μ) were determined for the ROI in the T_2-map prior to loading. Voxels were considered elevated (damaged) if they exceeded a threshold (Th) (2). Regions > 3 adjacent voxels were used for analysis. The leg was divided into 3 regions of 4 slices: distal to, underneath and proximal to indentation. The results per region were averaged over the analyzed slices

$$S = S_0 e^{\frac{-TE}{T_2}} \quad (1)$$

$$Th = \mu_{T_2} + 3 \times SD_{\mu_{T_2}} \quad (2)$$

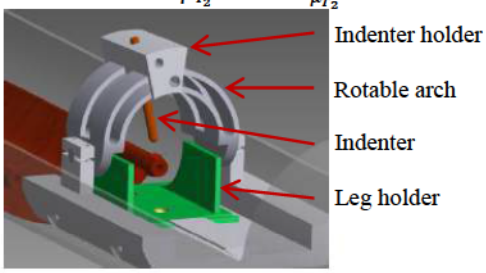

Fig. 1: The experimental set-up. The hind limb of the rat is fixated with a cast in the holder. The optimal indenter position is obtained by rotating the arch and the indenter holder. The indenter is applied manually.

FEA: Animal specific FEA was performed (n=5). Skin and bone contours were determined from the T_1 scans before and during loading using the GIBBON toolbox [4] (Fig 2A-B). The orientation and endpoint of the indenter were obtained from the T_1 scans during loading. The start point of the indenter was obtained by giving an offset to the indenter in its direction to make sure the start point was outside the contours of the model. Essential boundary conditions were the movement of the indenter and the bone. To match the experiment a rigid cast was simulated (Fig 2C). The model was solved in Abaqus 6.14 using the Ogden material law. To determine the elements of the TA, the ROI before indentation was interpolated on the original model using a natural neighbor algorithm.

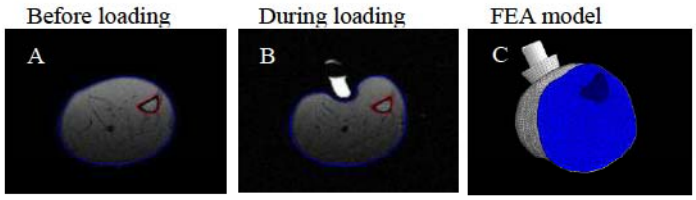

Fig. 2: T_1 images before (A) and during (B) loading. The skin contour is shown in blue and the bone contour in red. C) The front view of a reference state of one finite element model. Soft tissue in blue, the cast and indenter in white.

RESULTS

A structured increase in T_2-value was found after indentation (Fig 3). The region of indentation consistently has the highest volume of elevated T_2-values (Fig 4). High strain energies were found in the regions with the most T_2-elevation (Fig. 3 K,N). However, in regions with low strain energy values, namely the proximal and distal regions, T_2-elevation was also evident (Fig 3,J,L,M,O).

DISCUSSION

This study shows that the effects of mechanical loading extends outside the region of indentation. The loading threshold for deformation damage found previously [3] was clearly exceeded in all experiments in the current study. However, these experiments show that the linear relationship previously found does not extend to regions outside indentation. This may be explained as follows. An increase in T_2-value represent an increase in free water, which occurs due to oedema formation and/or intracellular fluid leakage [5], both considered early signs of tissue damage by the authors. In the destruction phase of muscle injury, transection of muscle fibers creates a release of intracellular fluid [6]. The buildup of fluid within the muscle can potentially diffuse between the muscle fibers, explaining the propagation of T_2-elevation. A previous study has shown that 24 hours after loading T_2-elevation highly correlated with a loss of cross striation and infiltration of inflammatory cells [7]. This indicates the applicability of T_2-values as an early sign of muscle damage. To further map the physiological changes occurring in regions outside of indentation histology needs to be performed.

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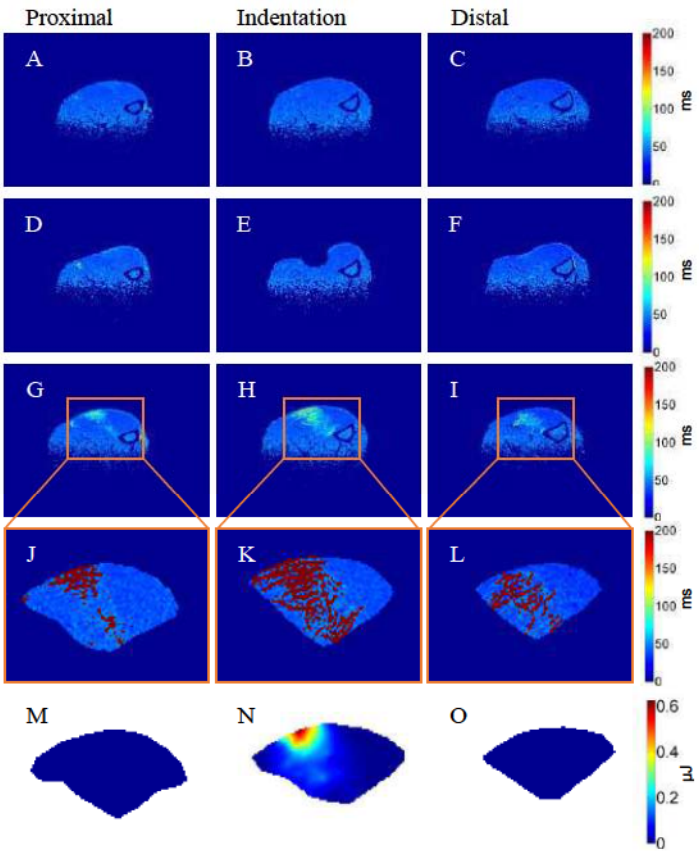

Fig. 3: T_2 an FEA results of the proximal (column 1), indentation (column 2) and distal regions (column 3). T_2 –map (A-L) with voxels accepted for analysis: (A-C) before (D-I) during and (G-I) 90 minutes after indentation. The regions of elevated T_2-values are depicted with red in the ROI (J-L). The corresponding strain energy in the ROI of the TA is shown (M-O).

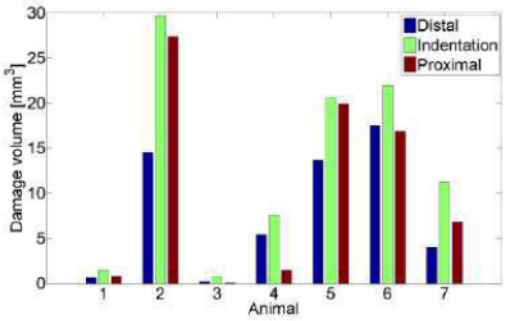

Fig. 4: The averaged elevated T_2 volume per region per animal (n=7).

REFERENCES

[1] Black, J et al., *Ostomy Wound Manag*, 56:28-44, 2010
[2] Mak, A et al., *Annu. Rev. Biomed. Eng*, 12:29-53, 2010
[3] Loerakker, S et al., *Ann Biomed Eng*, 38:2577-2587, 2010
[4] Moerman, K et al., *Proc. 11th Int Symp. CMBBE,* 2013
[5] Berquist, T et al., *MRI of the Musculoskeletal System*, 6th ed., 2012
[6] Jarvinen, T et al., *Am J Sports Med*, 33:745-764, 2005
[7] Bosboom, M et al., *J Appl Physiol*, 95:2235-2240, 2003