Deep tissue injury: how deep is our understanding?

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Introduction

Pressure ulcers are a serious health and financial problem. Prevalence figures are very high: 15% in general hospitals and up to 19% in nursing homes. Pressure ulcers can initiate either at the skin layer (superficial ulcers) or within deeper tissues (deep tissue injury). The underlying mechanisms of deep tissue injury are not well understood. Hypothesis included the role of 1) ischemia, 2) ischemia-reperfusion damage, 3) impaired interstitial fluid flow and 4) deformation of cells.

Objective

To test different hypothesis on the aetiology of deep tissue injury in a rat-model using MRI techniques and finite element modelling.

Method

A rat model was developed in which a muscle in the hindlimb was loaded with an indenter for 2 hours (Figure 1). In the area under the indenter (dashed slice) the following parameters were measured and calculated: deformation, damage, perfusion, diffusion and strain.

Results

The deformation and damage during indentation was measured using T2-weighted MRI (figure 2, top row). Figure 2c clearly indicates a small area with increased T2-values indicating cell-damage (arrow).

The diffusion is measured using Diffusion Weighted MRI (figure 2, bottom row). Only a small decrease in the whole leg was found, associated with a temperature decrease of the leg.

The perfusion in the leg was measured using the contrast-agent Gd-DTPA and T1-weighted MRI. During indentation a large region is ischemic (blue area, 3b). After indentation a large influx is seen (3c).

Internal tissue strains were measured using tagging MRI. The tagging pattern before and during loading is shown in 3d and e. The calculated strain values show a small region with high values (3f).

For a direct correlation between strain and damage a dedicated finite element model (figure 4, bottom row) was developed for each experiment based on the contours obtained from the MR-images.

Conclusion

Comparing the location of damage (2c) to the regions of ischemia (3b), decreased diffusion (2e) and high strains (3f and 4f), we demonstrated that the damage was mainly caused by the large strains (figure 5). It is evident that the understanding of deep tissue injury is significantly ‘deepened’ by our study. Instead of the generally accepted factor ischemia, deformation plays the most important role in initiating deep tissue injury.