Understanding the underlying mechanisms leading to deep pressure ulcers

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Introduction

Pressure ulcers are a serious health and financial problem. Prevalence figures are very high: 20% in general hospitals up to 29% in nursing homes. A lack of knowledge on the aetiology makes prevention difficult. Pressure ulcers can initiate either at the skin layer or within deeper tissues. The latter are termed deep pressure ulcers and often initiate in the muscle layer near bony prominences.

Objectives

To study the influence of deformation, ischaemia (no blood supply) and reperfusion on the onset of muscle damage after sustained compressive loading using MRI techniques and a dedicated finite element model.

Methods

A novel experimental set-up was designed and built to mechanically load the tibialis anterior (TA) of anesthetized Brown Norway rats while the animal resides inside a MR scanner with a 6.3 Tesla magnet. The procedure was approved by the animal care committee of the University of Maastricht.

Results

A series of T2-weighted MR images collected before, during and after indentation is shown in figure 1 (T2 is a measure for tissue damage). It is evident that after a loading period of two hours, higher signal intensity (arrow) is visible in the loaded region of the TA compared with images taken prior to loading.

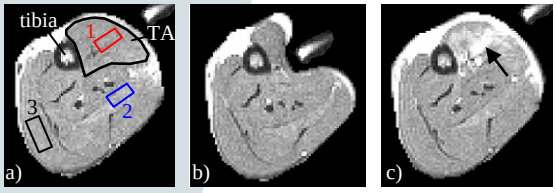

Figure 1 *T2-weighted MR images a) before, b) during, and c) after loading. TA is indicated by black line in figure a)*

In figure 2 the perfusion index (PI) maps are shown, measured with contrast-enhanced MRI. A large ischaemic region (dark region, figure 2b) is visible during loading.

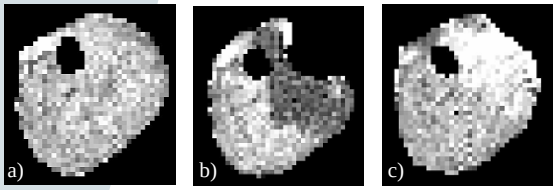

Figure 2 *Perfusion index (PI) maps taken a) before, b) during and c) after loading.*

To separate the effects of ischaemia and deformation, T2-values were evaluated in three ROIs (indicated in figure 1a). As a control, experiments were performed using an inflatable tourniquet, which was positioned above the knee, to induce pure ischaemic loading. T2-values are indicated in figure 3, showing that only compression led to irreversible damage.

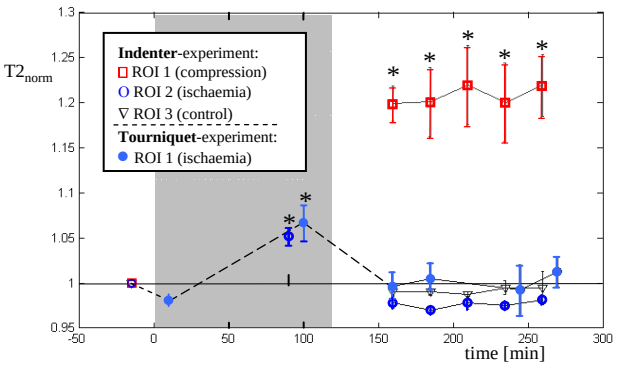

Figure 3 *Time course of normalized T2 for ischaemic and compressive loading. Shaded rectangle indicates loading period.*

The differences in response to ischaemic and compressive loading were confirmed by histological examination (figure 4).

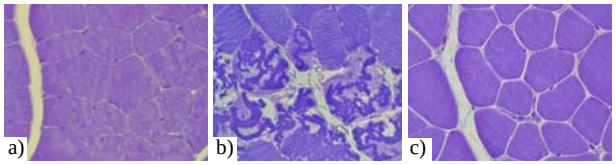

Figure 4 *Histological slices a) control, b) after 2 hours compression, showing necrotic fibres and c) after ischaemia.*

To further examine the influence of deformation on tissue damage, tagging MRI and a dedicated finite element (FE) model were used to couple local strain fields to damage location (figure 5).

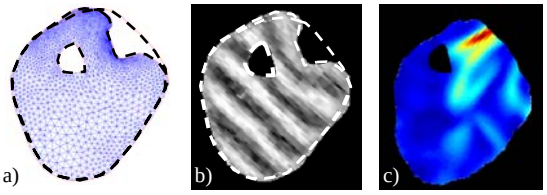

Figure 5 a) *FE model and b) tagging MRI to determine c) local strain fields during loading.*

Conclusion

By combining different MR techniques the importance of deformation in the onset of deep pressure ulcers could be demonstrated by the difference in response to ischaemic versus compressive loading (figure 3), and by the correlation between location of damage (figure 1c) and max shear strain (figure 5c). In addition, the MR techniques used in the present study can be applied in clinical practice for early detection of deep pressure ulcers, which is extremely important. A pre-screening method is however necessary, since MRI is not available on a daily basis. Therefore, identification of early damage markers in blood samples will be the next step in this research project.