Understanding the underlying mechanisms leading to pressure ulcers

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Introduction

Pressure ulcers are a serious health 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.

Hypotheses associated with the pathogenesis of pressure ulcers involve localised ischemia, reperfusion injury, and sustained deformation of cells. To distinguish between these effects and explain the onset of damage is difficult. In most animal models used to study the aetiology the tissue is examined using histology after a certain period of loading, however, what happens inside the tissue during and after loading remains unclear. Magnetic resonance imaging (MRI) can give detailed spatial and temporal information about deformation, damage, perfusion and metabolism.

Objectives

To study the influence of deformation, ischemia 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. It is evident that after a loading period of two hours, higher signal intensity is visible in the loaded region of the tibialis anterior (TA) compared with images taken prior to loading. This increase is not yet visible in the image taken just before unloading (b).

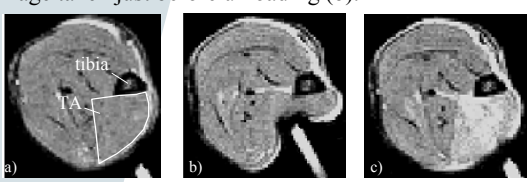

Figure 1 *T2-weighted transversal slices of the hind limb showing a) the indenter applied to the skin, b) indenter applied to the muscle, and c) image taken 1 hour after removal of load showing higher signal intensities in the TA region.*

In figure 2 histological slices taken 24 hours after unloading are shown of the damaged muscle. Perfusion in the TA is measured by contrast enhanced MRI. In figure 3 the increase in signal intensity, which is a measure for the perfusion, is shown after injection with the contrast agent Gd-DPTA.

The perfusion maps were measured in the same experiment as the T2-weighted images (figure 1).

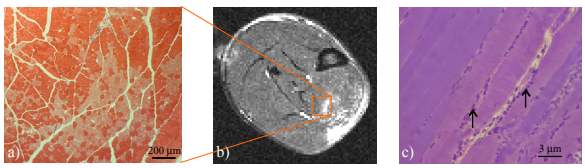

Figure 2 *a) Transversal section of TA (Gomori's trichrome staining) showing alternating healthy and damaged muscle fibers which leads to (b) local signal increase in a T2-weighted MR image. c) Longitudinal section of TA showing the loss of cross-striation of muscle fibers and the infiltration of monocytes (arrow).*

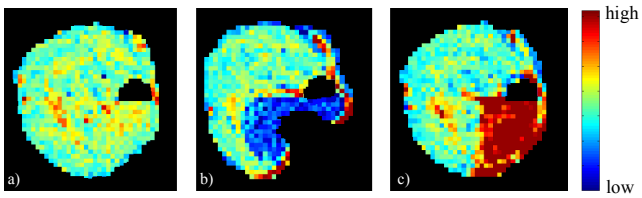

Figure 3 *Perfusion maps taken before, during and 30 minutes after loading showing that b) during loading the TA is ischemic and c) the contrast agent leaks into the damaged region.*

To measure the precise deformation of the muscle MR tagging is used (figure 4a) and a finite element model is used to calculate relevant mechanical quantities (figure 4b and c).

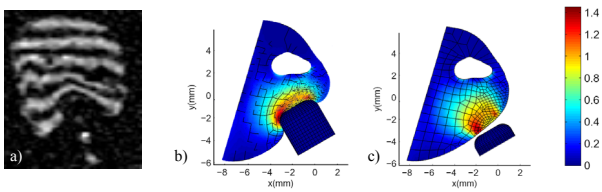

Figure 4 *a) MR tagging lines during indentation. b and c) Maximal shear strain distribution in deformed and undeformed mesh.*

Discussion

The different factors that may play a role in the onset of damage can be measured in this animal model. We have shown that during indentation large deformations and ischemia occur in the muscle. Furthermore, the damage starts after unloading, and not during loading, which proves that large deformations and ischemia for 2 hours alone do not lead to damage. This suggests that reperfusion damage plays an important role. To separate the effects of ischemia and deformation, experiments will be performed with a cuff around the leg (ischemia but no deformation). By separating the effects and combining the results with the FEM we will be able to better understand the mechanism leading to muscle damage associated with pressure ulcers.