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Early detection of deep pressure ulcers

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Introduction
Deep pressure ulcers start in deep tissues near bony prominences, as a result of sustained mechanical loading (fig. 1). Especially spinal cord injured (SCI) patients are at risk of developing these ulcers.

Deep pressure ulcers are hard to detect at an early stage because they start in deep tissue layers under intact skin. The aim of this project is to develop a pre-screening method to detect early skeletal muscle damage by measuring the rise of biomarkers present in blood or urine. For this it is necessary to know how tissue damage evolves over time, how this is reflected in a rise in levels of biochemical markers, and to understand the kinetics of this process.

Materials and Methods

The different methods used in this project are (fig. 2):
- An animal model was developed to understand the damage pathways of deep pressure ulcers. In this model the tibialis anterior (TA) muscle of a rat is compressed by an indenter in a MR compatible setup [1].
- The internal deformation of the muscle tissue is modeled with a finite element (FE) model [2].

Results

Deformation of a rat TA muscle leads to a small area of tissue damage (fig. 3a), while the total muscle is ischemic (fig. 3b). MR tagging experiments (fig. 3c) also indicate that there is a relation between maximum shear strain and damage. Therefore, deformation is more damaging than ischemia in these experiments [1].

A dedicated FE model also shows that there is a reasonable correspondence between damage found in MR images (white dots) and maximum shear strain (fig. 4a). Furthermore, the fraction of damaged area increases with the maximum shear strain (fig. 4b) [2].

Future plans

- The existing FE model will be extended to three dimensions. Furthermore, the influence of different parameters and anisotropy due to the fiber structure of muscle, on the model results will be investigated.
- Based on the animal experiments, a theoretical model will be developed that relates the dynamics of markers in blood to the development of tissue damage.

References: