Deformation-induced ischaemia in the onset of deep pressure ulcers

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Introduction

Spinal cord injured patients are vulnerable for developing pressure ulcers, the worst wounds starting as deep tissue injury in muscle overlying a bony prominence. They can only be detected after having reached the skin surface, hampering effective treatment, and with variable prognosis. Our research is aimed at understanding the causes of these wounds and developing early detection methods.

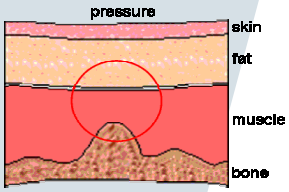

Figure 1 *Initiation of deep pressure ulcer in red circle.*

Most of the literature on pressure ulcers focusses on ischaemia (deficient blood flow), but studies in our group have shown that tissue deformation as such can also be damaging [1, 2], and that ischaemia with deformation leads to more severe damage than ischaemia alone [3].

Research question

Is it possible to understand the individual roles of tissue deformation and ischaemia in the development of muscle damage?

Method

A theoretical finite element model was developed on the microstructural level that describes deformation and oxygen diffusion in a 2D representative volume element.

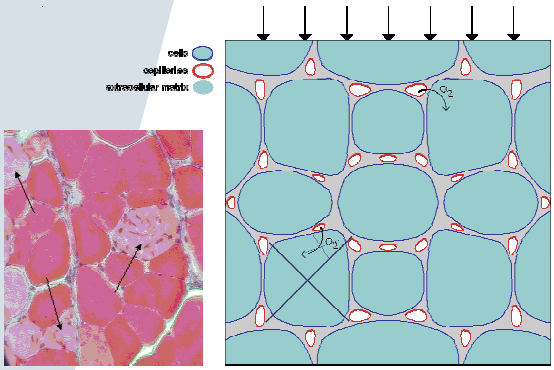

Figure 2 *Histological image of interspersed healthy and dead cells (arrows) [3] (left). Mesh for finite element model with applied deformation, oxygen diffusion from capillaries to cells and cell death if cell is deprived of oxygen (right).*

Cells die if they do not receive oxygen for a long time. The microstructural model introduces cellular interactions and the typical muscle microstructure, needed to explain and couple experiments on cellular and tissue level.

Results

Compression leads to decreased oxygen supply through capillary collapse, leading to diminished oxygen pressures in the cells.

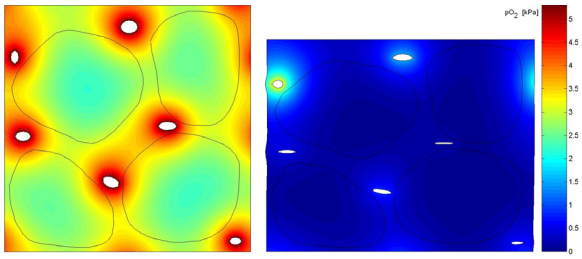

Figure 3 *Oxygen distribution in undeformed situation (left) and at 14% compression (right).*

This oxygen lack can eventually lead to cell death. The model suggests an inverse relationship between tissue compression and time of onset of cell death that is consistent with experimental observations.

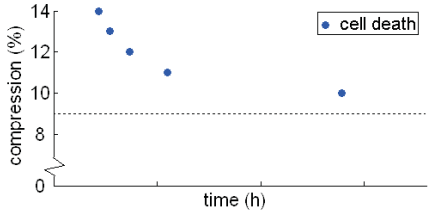

Figure 4 *Model prediction for time of cell death at different compression levels.*

Discussion

The model gives a reasonable description of oxygen transport and utilization, and it provides insight into muscle damage growth due to oxygen shortage. To answer the research question, other damage pathways such as a decrease in pH due to lactate accumulation and damage due to cellular deformation have to be included.

References:

[1] Bouten C.V.C., et al.: *Ann Biomed Eng. 2001 Feb;29(2):153-63*

[2] Breuls R.G.M., et al.: *Ann Biomed Eng. 2003 Dec;31(11):1357-64*

[3] Stekelenburg A.: *thesis: Mechanisms associated with deep tissue injury induced by sustained compressive loading*