BOLD response of different muscles to ischemic exercise

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Introduction

Alterations in tissue perfusion are frequent complications in various disorders like cardiovascular diseases or diabetes mellitus. Investigation of their consequences on tissue metabolism is clinically highly relevant. Skeletal muscle metabolism can be monitored by magnetic resonance imaging. Echo planar imaging (EPI) acquisition is sensitive to tissue oxygenation and perfusion and therefore lends itself to the investigation of response to ischemia or exercise.

Methods

We studied five male healthy volunteers (age 25.2±4,4, BMI=22,5±1,2) after a routine physical examination, laboratory tests and ECG, no clinical significant abnormalities were found. MR images were acquired using a Siemens Tim Trio scanner and a flexible coil wrapped around the subjects' calf. An inflatable cuff was wrapped about their thigh. The subjects lay supine in the scanner with their right leg fixated in an ergometer (Fig. 1) to perform plantar flexion (1). Fat-suppressed EPI images (parameters: 90° flip angle, 128x102 acquisition matrix, FOV=18 x 18 cm, GRAPPA 2, T_E=44 ms, T_R=0.5 s, 5 axial slices of 5 mm thickness; s. Fig. 2) were continuously acquired for 45 min. Two minutes after the start of the acquisition, the cuff was inflated to suppress femoral blood flow. 18 min thereafter, the subjects were instructed to perform a planar flexion every 4 s until exhausted. Then the cuff was deflated completely. Postprocessing consisted of resampling and registration of the EPI images (2) and the extraction of manually drawn ROIs, one in gastrocnemius (G), soleus (S) and tibialis anterior (T) muscles respectively. Large vessels were removed based on their characteristic hyperintense signal and their respective absence during ischemia. The time course of the signal in those ROIs was plotted (Fig. 3) and parametrised. All signals are given as % of starting value ± SD, times are expressed in seconds ± SD unless otherwise stated.

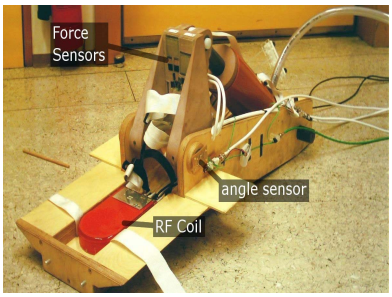

Fig 1: MR compatible ergometer

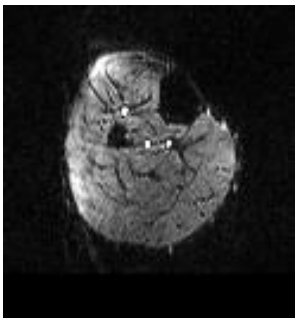

Fig 2: Axial EPI of human calf muscle in one volunteer

Results

In all ROIs we observed an initial signal decrease after cuff inflation (~100s long), followed by a slow decay until the onset of exercise (gastrocnemuius 88±4%, soleus: 79±12%, tibialis anterior: 86±9%). During exercise, lasting 110±50s, we consistently observed strong signal changes, an increase which was attributed to recruitment of fresh unsaturated spins due to motion. Looking at the respective images revealed severe artefacts like strong ghosts and misregistration during exercise, but there always were images in between two flexions not showing those artefacts. Given that these alterations showed a similar time pattern as the instructed flexions, we are confident that these were acquired at time points were the muscle returned to its resting position.

Signal intensities at cuff release were G: 89±13, S: 72±20, p=0.002 vs G, p=0.036 vs T, T: 86±15%. During hyperemia they reached their maximum at G: 187±85, p=0.043 vs S, p=0.010 vs T, S:105±17, T=116±37s of G:106±13, S:118±17, T:125±26 % baseline and returned to a steady state after G: 986±193, p=0.0002 vs. S, S: 506±170, T: 711±127s.

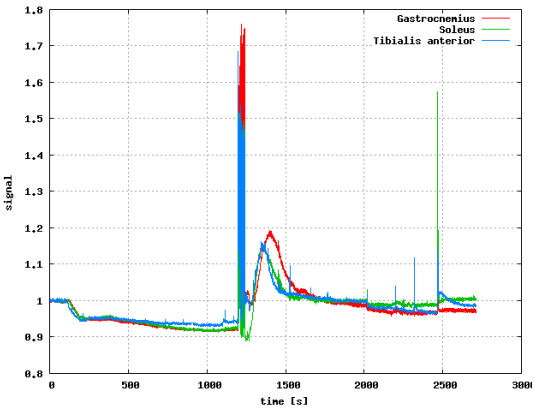

Fig 3: Signal time course of gastrocnemius, soleus and tibialis anterior muscles; At 120 s the cuff is inflated, at around 1200 s the exercise starts, seen by hyperintense signals. At the end, the cuff is released and hyperemic signal increase follows until it returns to baseline.

Conclusion

We investigated the influence of ischemic planar flexion on three different muscles in human calf. Soleus muscle showed a lower signal than the other muscles throughout the ischemic period. Gastrocnemius, which was the most active muscle in this experimental set up, showed a slower and flatter hyperemic response than the other muscles. This is in agreement to data presented in (3). In contrast to their study, which investigated postischemic hyperemia and aging without exercise, in our study offline image registration was essential for the data analysis, as some subjects clearly showed motion artefacts over the relatively long examination. The absence of exercise and the resulting lower stress of the muscle could explain the shorter hyperemic period reported in (3). The conjunction of these two studies suggests that this is an inherent property of the muscle, e.g. the fiber type, and not primarily a result of the experimental design.

References

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2. Walker, P et al, Magn Reson Imaging 1988; http://www.bic.mni.mcgill.ca/software/minc/
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