Evidence of decoupling between perfusion and diffusion in skeletal muscle: an MR study with arterial spin labeling and diffusion-weighted imaging

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Introduction

Previous studies have demonstrated increases in transverse magnetization and water diffusion of skeletal muscle following rigorous exercise and ischemia-reperfusion (1-3), yet the biophysical/physiologic mechanism remains unclear. Several factors have been proposed as plausible mechanisms underlying the observed MRI signal enhancement, including cell swelling, vascular reperfusion, fluid volume increase or the mixture of the above. In the present study, we employed both arterial spin labeling (ASL) perfusion MRI and diffusion MRI as dynamic indices to follow temporal evolutions of perfusion and diffusion in skeletal muscle using an arterial-occlusion paradigm. We demonstrated a temporal decoupling between blood flow and water diffusion, which may provide insights on the biophysical mechanism of hyperemia response following ischemia in skeletal muscle.

Materials and Methods

Studies were performed on four healthy subjects (1 female, 3 males, age = 25 to 46 years) on a 3.0 Tesla Siemens Trio whole-body MR system. Subjects were placed in a supine feet-first position with mid calf stabilized with foam pads and centered at a custom designed transmit/receive knee coil. A nonmagnetic orthopedic tourniquet system was used with cuff wrapped around the thigh. A pressure of 50 mmHg above systolic blood pressure was maintained for 4 min to create a temporary ischemia. Imaging started 1 min before cuff inflation and continued for 3 min after deflation, which gave a paradigm of 8 min (preceded by a 8 s dummy scan). Inflation and deflation completed in 10 s after the buttons were pressed. Three axial slices (1.4 mm gap) were chosen at mid-calf with voxel size of 3.4x3.4x7 mm^3. Perfusion imaging was performed using a continuous ASL (CASL) method with amplitude modulation in control session (TR/TE = 4000/17 ms, post-labebling delay = 1500 ms, tagging duration = 2000 ms) (7). A series of diffusion-weighted images were acquired with the same slice prescription (TR/TE = 4000/75 ms, b = 0 and 500 s/mm^2, superior-inferior diffusion encoding). A gradient-echo echo-planar sequence was used for all acquisitions. ASL signal was generated by surround subtraction (8). At last, the signal time curves of ASL and apparent diffusion coefficient (ADC) were smoothed with two adjacent data points.

Results and Discussion

Figs 1 and 2 show the signal evolution of ADC, CASL and T2*-weighted images and signal increase from baseline to hyperemia of 21%, 620% and 10%, respectively. ADC correlates well with T2* whereas blood flow returns to baseline faster than T2* does. One notices that different TE was used in Figs 1 (75ms) and 2 (17ms) for the purpose of comparison. Fig 3 shows ASL images obtained before, during and after arterial occlusion. Although the signal-to-noise ratio of ASL signal was relatively low at baseline, we were able to track the dynamic changes in blood flow in response to cuff occlusion with a temporal resolution of 4 s. By assuming the baseline flow of 20 ml/100g/min (13-30 ml/100g/min in literature), the peak flow is 144 ml/100g/min. We report the evidence of decoupling between perfusion and diffusion in skeletal muscle, suggesting cuff-occlusion induces a brief vascular hyperemic response followed by more long-lasting effects in diffusion and T2/T2* which may be associated with cell swelling and metabolic changes. One caveat of this study is that to apply ASL to induced hyperemia, non-100% water extraction and T2* change should be both taken into account.

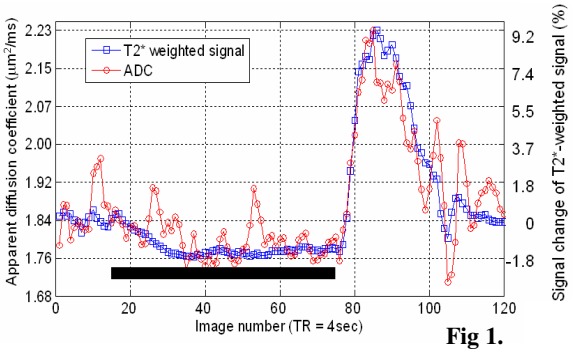

Fig 1.

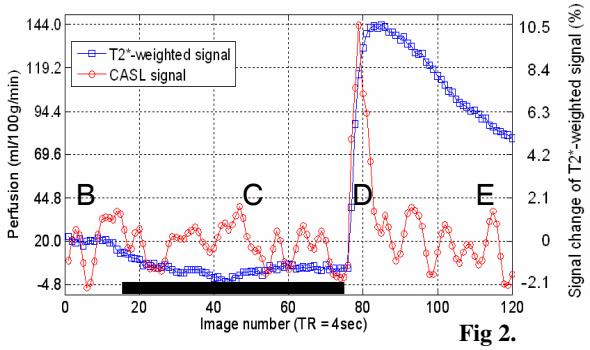

Fig 2.

Figs 1 and 2. Signal evolution of ADC, CASL and T2*-weighted images. Data are extracted from the soleus muscle of a subject. The black horizontal bar indicates the duration of arterial occlusion.

References

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Fig 3. EPI image (A) and ASL maps obtained at the time points marked in Fig 2 (B-E).