Comparison of muscle BOLD and transcutaneous PO2 measurements in a human postischemic

H-G. Heidecker[1], H. P. Ledermann[1], A. C. Schulte[2], M. Aschwanden[3], K. Jaeger[3], D. Bilecen[1]

[1]Kantonsspital Basel Stadt, Universitätsinstitut für Diagnostische Radiologie, Basel, Switzerland, [2]MR- and Biocenter, Biozentrum Basel, Basel, Switzerland, [3]Kantonsspital Basel Stadt, Universitätsinstitut für Angiologie, Basel, Switzerland

Introduction

Transcutaneous PO_2-measurement ($TcPO_2$) is routinely used in clinical practice to determine local oxygen supply of the lower extremities in patients with peripheral arterial occlusive disease (PAOD).[1] The blood oxygenation level-dependent (BOLD) signal routinely used in functional MRI of the brain relies on the different magnetic properties of oxy- and deoxyhemoglobine and is thus sensitive to changes of the local deoxyhemoglobin concentration in areas of neuronal activity.[2] It has been shown recently that a BOLD signal can also be measured in skeletal muscle tissue.[3,4] The aim of our study was to correlate the BOLD- and $TcPO_2$-signal of lower extremity skeletal muscle during ischemia and reactive hyperemia.

Methods

11 healthy volunteers (6 male, 5 female, mean age 34 ± 6.2 y) were examined using a 13 minute postischemic hyperemia paradigm: A) 1 min preocclusive baseline, B) 6 min occlusion and C) 6 min postocclusive recovery. $TcPO_2$- and BOLD measurements of each patient were performed within 7 days. Volunteers were examined supine with a pneumatic compression cuff wrapped around the thigh. The cuff was manually inflated to a pressure of 50 mmHg above systolic blood pressure. $TcPO_2$-measurements were performed using a $TcPO_2$-monitoring system (TCM400 RADIOMETER Copenhagen, Denmark) at 37°C with the $TcPO_2$-electrode attached to the dorsal upper third of the calf adjacent to the gastrocnemius muscle. Measurements were recorded every 10 s totalling 78 measurements. BOLD-measurements were performed on a 1.5 T MR-scanner (Sonata, Siemens Erlangen, Germany) using a multi-echo EPI-sequence with four echoes (TE [ms] 16/38/61/83; FOV [mm] 380 x 238; Slice thickness [mm] 5; TR [ms] 1000).[5] 780 axial EPI-measurements were continuously acquired in the upper calf at maximal calf diameter. Data analysis was performed using the statistical parametric mapping software BrainVoyager (Brain Innovation B.V., R. Goebel, Maastricht, Netherlands). BOLD-signal time courses were obtained from regions-of-interest within the dorsal gastrocnemius muscle. For correlation BOLD data were extrapolated to the acquisition-intervals used for $TcPO_2$-measurements. For better comparison of the data, measurements were normalized to the individual preocclusive baseline (100%). Datasets of the occlusion period and postocclusive reactive hyperemia were analysed separately. $TcPO_2$ and muscle-BOLD time-courses were compared using correlation analysis.

Results

$TcPO_2$- and BOLD-signal response during ischemia and reactive hyperemia is summarized in Fig. 1 a and b. After the beginning of the occlusion, $TcPO_2$-values decreased rapidly and reached a steady minimum at 20% of the baseline value after 3.5 minutes. The BOLD-signal paralleled the $TcPO_2$-curve with a steep decline during early ischemia and persistent minor decrease at the end of compression reaching 92%. The calculated correlation coefficient (cc) was 0.97 (Fig. 1 a). After the cuff compression was released, a steep recovery of the signals with an overshoot above preocclusive baseline was detected with both methods. The $TcPO_2$-signal reached its maximum 120 s after the release of the occlusion at 493% followed by a constant decrease that levelled out at 251% by the end of the experiment. The BOLD signal reached its maximum 40 s after the release of the occlusion at 112.5%, followed by a constant decrease to 98% by the end of the experiment (Fig. 1 b). During the postocclusive period the calculated cc-value was = 0.26.

Discussion

Radiological work up of PAOD and of its associated complications is increasingly performed using MRI. MR angiography allows visualization of the entire arterial supply of the lower extremity within seconds and MR imaging of the feet further allows evaluation of the extent of infection during the same examination. Noninvasive measurement of local tissue oxygenation with functional MRI would add clinically important information to plan further therapy. Few reports so far describe BOLD signal analysis in the skeletal muscle of the lower extremity. In our study peripheral desaturation of the skin and subcutaneous tissue correlated well with the BOLD signal in the gastrocnemius muscle during ischemia. Therefore, the BOLD signal decrease during ischemia may well reflect increasing deoxygenation of hemoglobin as suggested by others.[3,4] During reactive hyperemia the steep ascent of the BOLD signal may reflect reoxygenation of desaturated hemoglobin. The time difference between the peak of the BOLD signal and the oxygen saturation in the skin and subcutaneous tissue may have a physiological or technical cause: there could be persistent vasoconstriction of the skin after ischemia or inertness of the measuring device. $TcPO_2$ has shown to be used for monitoring immediate and long-term success of vascular interventions.[1] Eventually muscle BOLD-imaging holds similar potential for the workup of peripheral vascular disease. Whether this holds true for measurements in patients with vascular pathologies as i.e. PAOD remains to be determined.

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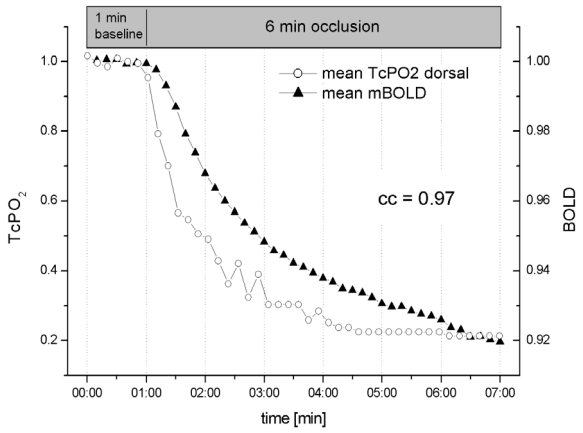

Fig. 1a:
Mean $TcPO_2$- and mean muscle BOLD-response during the first 7 minutes, including preocclusion- and occlusion-period. $TcPO_2$-values correlate to the y-axis on the left, BOLD-values correlate to the y-axis on the right. All values were individually normalized to the mean of preocclusive baseline values. (mBOLD = muscle BOLD)

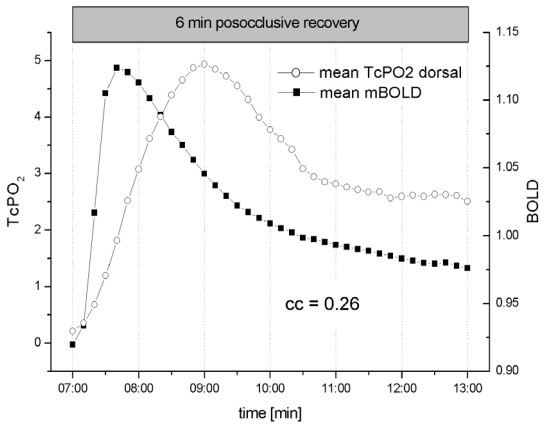

Fig. 1b:
Mean $TcPO_2$- and mean muscle BOLD-response during the recovery period. $TcPO_2$-values correlate to the y-axis on the left, BOLD-values correlate to the y-axis on the right. All values were individually normalized to the mean of preocclusive baseline values. (mBOLD = muscle BOLD)