

Quantification of the Effect of Induced Graded Ischemia in the Popliteal Artery and Vein by MR Oximetry

M. C. Langham¹, T. Floyd², J. Magland¹, M. A. Fernandez-Seara³, E. Mohler, III⁴, and F. W. Wehrli¹

¹LSNI, Department of Radiology, University of Pennsylvania, Philadelphia, Pennsylvania, United States, ²Anesthesia, University of Pennsylvania, Philadelphia, Pennsylvania, United States, ³Neuroscience Department, University of Navarra, Pamplona, Navarra, Spain, ⁴Department of Medicine, Cardiovascular Division, University of Pennsylvania, Philadelphia, Pennsylvania, United States

Introduction

The non-invasive measurement of hemoglobin saturation is of great interest in medicine and abnormal venous or arterial oxygen saturation occurs in a variety of vascular disorders. Quantification of reactive hyperemia is a strong candidate for evaluating vascular disorders. The current non-invasive modalities for quantifying oxygen saturation include near-infrared reflectance spectroscopy (NIRS)[1] and ultrasound [2]. NIRS is limited to surface tissue or the smallest vessels. Measurement of flow-mediated dilatation using ultrasound is very sensitive to small differences in the measurements and no standard method to assess reproducibility exists. MR susceptometry requires no calibration as in the T2 based method of [3] and its feasibility has already been demonstrated [4]. We extend the method of [4] to the study of oxygenation changes in cuff-induced leg ischemia with temporal resolution of 20 seconds.

Methods

Images were acquired with a multi-echo GRE sequence that was coded with Sequence Tree Version 3.1; the sequence includes standard fat suppression using a spectrally selective sinc pulse and flow compensated slice selection gradient. The key parameters were: voxel size = 1 x 1 x 5 mm³, FOV = 128x128 mm², resolution = 1 x 1 x 5 mm³, BW = 488 Hz/pixel, TR = 156 μs, flip angle = 40°, total scan time/image = 20s. Written informed consent was obtained from all volunteers prior to the investigation. The automatic cuff (Aspen Labs A.T.S 1500 Tourniquet System) was wrapped around the middle thigh and inflated to a pressure of 200 mmHg. Baseline measurement of the hemoglobin oxygenation was made for the first two minutes. Occlusion periods of 1, 3 and 5 minutes were applied followed by 5 minutes of recovery time. To correct for vessel obliquity the vessel tilt angle was measured by acquiring a separate 10-slice axial image. The raw data was saved and reconstructed with MATLAB. A phase difference map was constructed by taking the difference of the phase images of the two different echo times. The same Hanning filtering technique as in [4] was used except that the filter was applied to the IFFT of the phase difference map rather than the raw data itself. Oxygen saturation was computed from the phase difference in a ROI encompassing the vein and in the adjacent muscle. The phase value of the reference tissue was subtracted from the intravascular phase. The phase difference is expressed as $\Delta\phi = \gamma\Delta B\Delta TE$, where ΔB is the field difference between the vessel and the nearby tissue and $\Delta TE = TE_2 - TE_1$ is the elapsed time between the two subsequent echoes. If the vessels are taken to be an infinitely long paramagnetic cylinder tilted at an angle of θ with respect to the static field B_0 , then the field difference is given by [5], $\Delta B = \frac{1}{3}\Delta\chi_{do}^{SI}(3\cos^2\theta - 1)\text{Hct}(1 - \text{HbO}_2)B_0$, where Hct is the hematocrit and $\Delta\chi_{do}^{SI}$ is the susceptibility difference between fully deoxygenated and fully oxygenated erythrocytes expressed in terms of ppm in SI units.

Results and Discussion

Figure 1 shows sample *in vivo* magnitude and phase difference images. Typical time course of the hemoglobin oxygenation is shown in Figure 4. During the three and five minute-long occlusion significant drop (in the arterial oxygenation occurs after approximately two minutes, which explains why arterial desaturation is not seen during one minute occlusion period. The delay occurs because the artery is not completely occluded (Figure 2). In general, one minute occlusion does not seem to produce any significant effects compared to the 3 and 5 minute occlusions; we will only refer to the longer occlusion periods. Venous oxygenation consistently increased during the cuff inflation because complete arterial occlusion was not achieved. In all cases, rapid venous desaturation followed the cuff deflation. Figure 4 demonstrates MR susceptometry can distinguish different degrees of cuff-induced ischemia; the magnitude of arterial and venous desaturation differ by 31% and 11%, respectively. Furthermore, the venous runoff appears to be more rapid in the longer occlusion and overshoots the baseline value by 8% more. Figure 3 shows a plot of the phase difference in the muscle tissue of two subsequent echoes for the entire scan time (baseline, occlusion and recovery); its constancy validates the use of muscle tissue as a reference. Scale of the graph is based on the average baseline value (0.5 rad) in the vein. The time course of muscle T2* is shown in Figure 5. The average increase in T2* (caused by high inflow of oxyhemoglobin [6]) during the postocclusive reactive hyperemia was 9.6%, which agrees within the standard deviation of [6] for non-PAD patients.

Conclusion

MR susceptometry is a viable method for monitoring hemoglobin saturation as a function of time, however, the temporal resolution must be improved. The temporal resolution is most critical after the cuff deflation because the blood flow rate during the hyperemic response can increase by as much as six-fold. MR oximetry also allows simultaneous measurement of T2*, which can be used to identify PAD[6].

References:

[1] Boushel et al. Scand Med Sci Sports 2001; 11: 213–222. [2] Corretti et al. J Am Coll Cardiol 2002;39: 257–65). [3] Wright et al. J Magn Reson Imaging 1991;1:275–283. [4] Fernandez-Seara et al. Magnetic Resonance in Medicine 55:967–973 (2006) [5] Haacke et al. *Magn Resonance Imaging*, New York: John Wiley & Sons; 1999. [6] Ledermann, et al. Circulation 113 (25): 2929 (2006).

Acknowledgement: NIH Grant T32EB000814

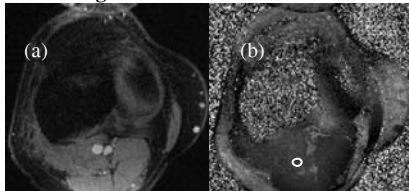


Figure 1: The popliteal artery is on the left. In the phase difference image the vein is more visible due to larger difference in the susceptibility. The white circle marks the location of the reference tissue.

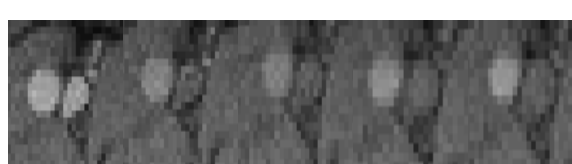


Figure 2: Magnified images of the popliteal vessels. Subsequent images following the first are acquired while the cuff is inflated. The artery is not completely occluded; some inflow effect is still present. On the other hand, the vein is occluded causing swelling.

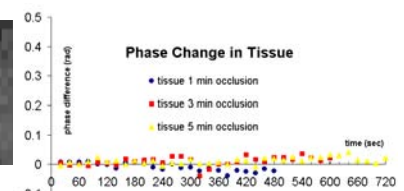


Figure 3: The negligible variation in the phase difference in the muscle justifies the use of muscle tissue as a reference.

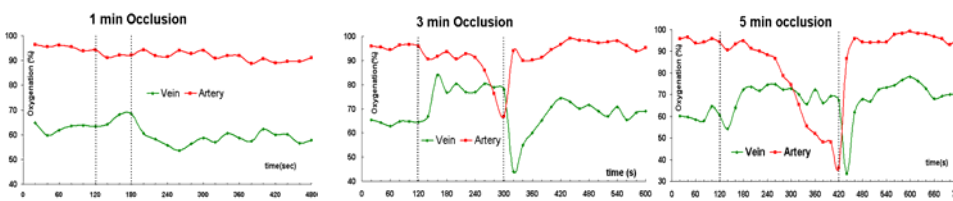


Figure 4: Vertical lines indicate the beginning and end of the cuff inflation.

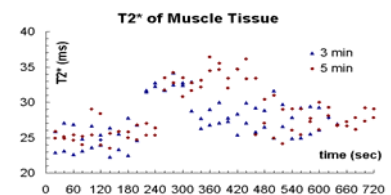


Figure 5: Time course of muscle T2* near the popliteal vessels.