The Influence of Imaging Parameters on SSFP-based Microcirculatory Blood-Oxygen-Level Dependent (BOLD) Contrast: Theory and Experiment

J. Mangalathu Arumana[1], D. Li[1,2], and R. Dharmakumar[1]

[1]Department of Radiology, Northwestern University, Chicago, IL, United States, [2]Department of Biomedical Engineering, Northwestern University, Chicago, IL, United States

Introduction The capability to rapidly assess blood oxygenation changes in microcirculation could provide valuable insight for understanding normal and pathological states of the body. Recently, steady-state free precession (SSFP) has been proposed as a means for detecting oxygen saturation changes in microcirculation [1]. Although previous studies in whole blood have shown that oxygen-sensitive contrast with SSFP is strongly dependent on repetition time T_R and flip angle α [2], it is unclear whether these findings are also applicable to microcirculation. In order to address this, this work investigates whether the SSFP-based oxygen contrast in microcirculation is dependent on T_R and α using theoretical and experimental models of ischemia/reactive hyperaemia response in skeletal muscle [3] at 1.5T and 3.0T.

Theoretical Methods A two-pool model [1,2] was used to simulate the SSFP signal behaviour during an ischemia/reactive hyperaemia response in skeletal muscle. The MR signal was computed by numerically solving the modified Bloch equations describing the microcirculation of the skeletal muscle [1]. The relative blood volume and mean oxygen saturation was set to 2% and 63% during ischemia and altered to 4% and 68% during reactive hyperaemia, respectively [4,5]. To simulate the influence of blood oxygen saturation, intravascular T_2 and the frequency shift between the intra- and extravascular pools were modified, as described previously in literature [1,2]. From the signal magnitudes computed from the simulations, normalized oxygen contrast (OC) was computed as: $OC = (S_{hyperaemia} - S_{ischemia}) / S_{hyperaemia} \times 100\%$, where $S_{ischemia}$ and $S_{hyperaemia}$ are the SSFP signal magnitudes during ischemia and reactive hyperaemia, respectively.

Experimental Methods Six healthy volunteers (25–35 years) were imaged on a Sonata 1.5T and a Tim Trio 3.0T scanner (Siemens, Erlangen, Germany). SSFP was prescribed with following scan parameters: voxel size = $1.3 \times 1.3 \times 5.0$ mm^3, total scan time = 7 min; α = 30°- 90° (increments of 30°), T_R = 3.5 ms / 6.3 ms / 9.9 ms at 1.5 T, T_R = 4.5 ms / 6.3 ms / 9.9 ms at 3.0 T, $T_E = T_R / 2$. In order to induce temporary ischemia in the calf, a blood pressure cuff was placed on the thigh proximal to the knee and connected to the Hokanson E20 rapid cuff inflator (D.E.Hokanson Inc., Bellevue, WA) and the cuff was inflated to a pressure of 200-205 mmHg. Axial images of the mid calf were acquired during a 1 min baseline period, followed by 3 min of imaging with the cuff inflated (ischemia), and then another 3 min of imaging with the cuff deflated (reactive hyperaemia). Each volunteer was scanned at 3 different T_R's (α of 90°) and at 3 different α's (30°, 60°, 90° with a T_R of 9.9 ms) with a 5 minute break in-between each scan. From three carefully chosen ROI's within the muscle area devoid of flow artefacts, signal intensities were measured and averaged. From the signal magnitudes at ischemia and reactive hyperemia, OC was computed for each volunteer and were averaged over all volunteers. Tukey post-hoc test was used to determine if significant differences ($p<0.05$) in mean OC existed between different imaging parameters at 1.5T and 3.0T.

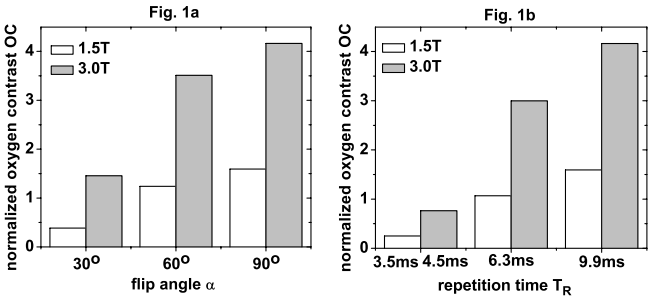

Fig. 1 Theoretical Results. Fig. 1a (left) and **Fig. 1b** (right) show the dependence of α (for T_R = 9.9 ms) and T_R (for $\alpha = 90°$) on OC, respectively. Results show that as T_R or α increases, so does OC.

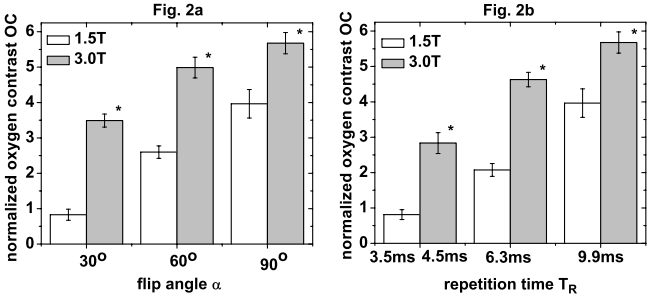

Fig. 2 Experimental Results. Fig 2a (left) and **Fig. 2b** (right) show the experimentally observed dependence of SSFP-based OC on α and T_R at 1.5T and 3.0T, respectively. The dependence of SSFP-based OC on T_R and α were studied by fixing $\alpha = 90°$ and T_R = 9.9 ms, respectively. Note that the OC is statistically significant different from 3.0T to 1.5T for all imaging parameters (denoted by asterisk sign *).

Results Simulated and experimentally observed OC as a function of T_R and α at 1.5T and 3.0T are shown in Fig. 1 and Fig. 2, respectively. Both simulations and experiments showed that increasing the field strength, T_R and/or α, leads to statistically significant increases in SSFP-based OC. Statistical analysis also showed that, for all imaging parameters studied, the OC at 3.0T is significantly different from 1.5T. In particular, doubling the field strength increased the experimentally observed OC by a factor of 2.3 ± 0.04 (represents averaged OC over all subjects, T_R's, and flip angles).

Discussion and Conclusion Our work investigated the dependence of SSFP-based OC on imaging parameters (T_R, α) using theoretical and experimental models of ischemia/reactive hyperaemia in skeletal muscle at 1.5T and 3.0T. The results showed that OC with SSFP is strongly dependent on T_R and α at both field strengths. Specifically, we found that increasing T_R or α can increase SSFP-based OC at 1.5T and 3.0T. Although we anticipate that long T_R SSFP BOLD imaging may provide amplification in oxygen contrast, an optimal choice in T_R will depend on the tissue of interest, shimming limits of the scanner, the extent of bulk magnetic inhomogeneities, and blood flow. In this regard, techniques that can reduce the SSFP banding and flow artefacts [6] may be very useful for long T_R SSFP BOLD imaging. Although we expect the results to hold generally, similar studies in other microcirculations are warranted.

References [1] Dharmakumar R et al. Magn Reson Med 2006;55(6):1372-80; **[2]** Dharmakumar R et al. Magn Reson Med 2005; 53(3):574-83; **[3]** Toussaint JF et al. Magn Reson Med 1996;35(1):62-9; **[4]** Litter J et al. J Clin Invest 1954;33(5):798-806; **[5]** Yu G et al. J Biomed Opt 2005; 10(2) **[6]** Bangerter NK et al. Magn Reson Med 2004;51(5):1038-1047