Detecting Myocardial Oxygen Deficits due to Coronary Stenosis in a Canine Model at 3.0 T with Steady-State Free Precession Imaging

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 Myocardial oxygen deficits can be detected with blood-oxygen-level-dependent (BOLD) MRI at 1.5T [1]. In a number of organ systems [2, 3], BOLD contrast has been shown to be strongly dependent on field strength [3]. However, this has not yet been demonstrated in the heart. Recently, Steady-State Free Precession (SSFP) based myocardial BOLD imaging has been demonstrated at 1.5T [4]. This work examines the benefits and the feasibility of extending the SSFP-based myocardial BOLD imaging methods at 1.5T to 3T. First, using simulations, this work examines the dependence of SSFP-based myocardial BOLD contrast on field strength at 1.5T and 3T. This is followed by an evaluation of the theoretical prediction using a canine model.

Theoretical Methods The changes in microcirculatory oxygenation of the myocardium was simulated with a two-pool model [3,5]. Modified Bloch equations, accounting for spin residence and relaxation times, blood volume, field strength and intravascular oxygenation changes; were solved to compute the SSFP signal magnitudes. The model assumed that under the influence of vasodilator (adenosine), normal myocardial oxygenation is 85% and is reduced to 30% under severe coronary stenosis [6]. Blood volume and hematocrit were assumed to be 0.10 and 0.4, respectively. Changes in intravascular T_2 and frequency shifts between the intra- and extra-vascular pools at 1.5T and 3T were modelled as previously described [3]. The simulations also assumed an excitation flip angle of 60^o and T_R of 5.2 ms. From the vector sum of the transverse components of magnetization, signal magnitudes were computed during stenosis-free (S_{aden}) and severe-stenosis (S_{sten}) states using *BOLD Contrast* = $[S_{aden} - S_{sten}] / S_{aden}$ at 1.5T and 3T. From the *BOLD Contrast* values obtained at 1.5T and 3T, the field dependent effects were assessed with *BOLD Contrast Ratio* = BOLD Contrast (3T) / BOLD Contrast (1.5T).

Experimental Methods In 4 Mongrel dogs, a portion of the left circumflex artery (LCX) was isolated and an occluder was secured around the LCX. To estimate the extent of the LCX stenosis during the MR studies, a Doppler flow probe was secured distal to the LCX occluder. All 1.5T and 3T studies were performed on Sonata and Tim Trio systems (Siemens, Germany), respectively, using a phased-array surface coil for signal reception and body coil for excitation. Each study consisted of three sets of cardiac-gated and breath-held scans (13-19 s) employing balanced-SSFP: (A) baseline adenosine scan with constant adenosine infusion into the RA catheter; (B) at least two levels of LCX stenosis with adenosine; and (C) a first-pass perfusion at the severe stenosis. True flow deficits, due to LCX stenoses, were measured with microsphere flow analysis. Based on the scout images, slice positions were matched between 1.5T and 3T studies and microsphere analysis. Manual shimming, centre frequency scouts, and when the magnetic inhomogeneities were severe, maximum intensity projections constructed from phase-cycled and non phase-cycled acquisitions [7] were used to improve image quality at 3T. The scan parameters were: voxel size = 1.8 x 1.4 x 5.0 mm^3, 10-12 phases/heart beat, T_E/T_R = 2.6/5.2ms, flip angle = 60^o, and averages = 2. From the MR and microsphere flow signals measured at the LCX and left anterior descending artery (LAD) territories, *BOLD Contrast* was computed from signal magnitudes from the LCX territory during stenosis-free and severe stenosis states at systole. MR *BOLD Contrast* was normalized by the microsphere–based perfusion contrast (computed analogous to *BOLD Contrast* using fluorescent signals from microsphere). *BOLD Contrast Ratio* was computed as defined earlier.

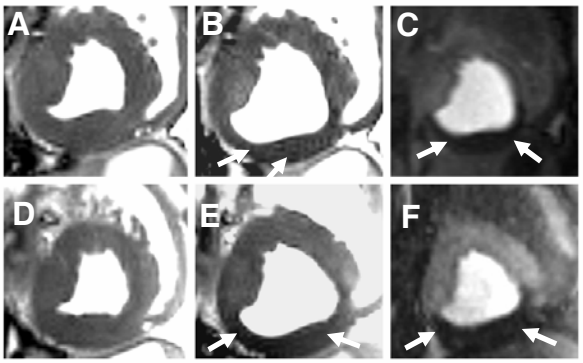

Fig. 1 shows a set of short axis images at 1.5T (top row) and at 3T (bottom row). Images A and D are SSFP images at systole under adenosine infusion (with no occlusion), images B and E are SSFP images at systole under LCX stenosis of similar extent, and images C and F are the corresponding first pass images acquired under the same stenosis levels as in B and E, respectively. Note the perfusion deficits and its close correspondence to BOLD images in the LCX territories. Also note the overall improvement in image quality at 3T compared to 1.5T, allowing for a more accurate visualization of oxygen deficit (B and E) in the LCX territory.

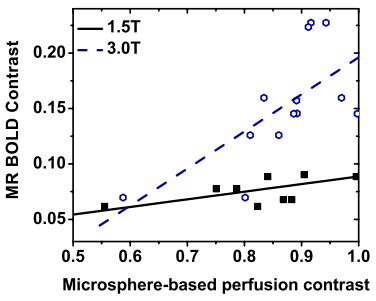

Fig. 2 shows the correlation between MR BOLD contrast and microsphere-based perfusion contrast at 1.5T (■) and 3T (○). Regression curves: y = 0.07x + 0.05 (r = 0.72, p < 0.01, at 1.5T) and y = 0.33x+0.03 (r = 0.67, p < 0.01 , at 3.0T). Note the increase in slope between the regression curves at 1.5T and 3.T, indicating the improved BOLD contrast at 3T.

Results *Theory:* The simulation results showed that the expected *BOLD Contrast Ratio* to be 3.8. *Experiments:* Fig. 1 shows typical short axis SSFP-based cardiac BOLD images obtained at adenosine baseline (A/D) and stress-stenosis (B/E) conditions and a first pass perfusion image (C/F) obtained during the severe stress-stenosis at 1.5T and 3T, respectively. Regional BOLD contrast was observed at the LCX territory at both field strengths and correlated well with the perfusion deficits observed from the first pass images. The experimentally measured *BOLD Contrast Ratio* was 2.5 and was deemed significantly different from 1.0 (t-test, $p<0.01$). Fig. 2 shows that microsphere-based perfusion contrast and MR *BOLD Contrast* were strongly correlated at both field strengths ($r = 0.7$, $p <0.01$).

Discussion and Conclusion Using theoretical simulations and an experiment canine model**,** this work investigated whether (1) the myocardial BOLD contrast is significantly augmented at 3T relative to 1.5T and whether (2) SSFP-based BOLD imaging can be used to acquire high quality, artifact-reduced images that show myocardial oxygen deficits due to acute coronary stenosis at 3T. The theoretical and experimentally derived results show that the BOLD contrast in the heart is significantly increased at 3T compared to 1.5T. However, there is a discrepancy between the theoretically derived and experimentally measured *BOLD Contrast Ratio* (2.5 vs. 3.8). This discrepancy may be due to physiological changes (hematocrit and blood volume) or system imperfections such as reduced RF penetration and/or B_1-inhomogeneities at 3T [8]. In spite of this discrepancy, based on experimental results alone, it is anticipated that the sensitivity of SSFP-based cardiac BOLD imaging may be increased significantly at 3T. However further improvement in image quality is necessary to apply cardiac BOLD imaging at 3.0T clinically.

References [1] Wright KB et al. Magn Reson Med 2001 Sep;46(3):573-8;**[2]** Ogawa et al. Magn Reson Med 1990 Oct;16(1):9-18;**[3]** Dharmakumar R et al. Magn Reson Med 2006;55(6):1372-80; **[4]** Dharmakumar R et al. Proc 14th ISMRM. P. 3571; **[5]** Dharmakumar R et al. Magn Reson Med 2005; 53(3):574-83; **[6]** Li D et al. Cardiovascular Magnetic Resonance 2001; 447-454; **[7]** Bangerter NK Magn Reson Med 2004;51(5):1038-1047 **[8]** Bottomley P et al. Phys Med Biol 1978;23:630-643