In Vivo Performance of Myocardial Background Suppression

Terrence Jao[1], Hung Phi Do[2], and Krishna Nayak[3]

[1]*Biomedical Engineering, University of Southern California, Los Angeles, California, United States,* [2]*Department of Physics, University of Southern California, Los Angeles, California, United States,* [3]*Electrical Engineering, University of Southern California, Los Angeles, California, United States*

Introduction: Myocardial arterial spin labeling (ASL) is a technique for measuring myocardial perfusion and perfusion reserve in humans through the subtraction of labeled and non-labeled images [1,2]. The signal from labeled blood is roughly 1-4% of the signal from background tissue (myocardium), which makes the perfusion estimate particularly sensitive to imperfect subtraction caused by cardiac or respiratory motion. In this work, we examine the potential for background suppression (BGS) through a saturation-double-inversion-recovery preparation to suppress signal from myocardium in an ASL experiment.

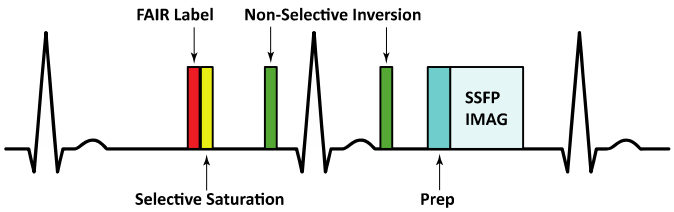

Figure 1: *FAIR-BGS cardiac ASL pulse sequence. yellow and green: saturation-double-inversion-recovery, blue: standard fat-sat and transient reduction prep.*

Methods: *Pulse Sequence:* The proposed pulse sequence, illustrated in Figure 1, is a modification of a current implementation of cardiac ASL composed of flow-sensitive inversion alternating recovery (FAIR) tagging and balanced steady-state free precession imaging [1,2]. The selective-saturation-double-inversion-recovery preparation between tagging and imaging is designed to suppress a broad range of T1s from myocardium (1000 to 1200) [3]. Tailored hard pulse trains was used for slice-selective saturation [4] and adiabatic hyperbolic secant pulses were used for non-selective inversion, both providing B0- and B1-insensitivity. The selective saturation and center of image acquisition were placed at the same cardiac phase (mid-diastole) to ensure the imaging slice was within the saturation slab. *Optimization:* For a given heart rate, the timing of the two inversion pulses was determined using a non-linear optimization scheme that iteratively minimized the squared sum of the longitudinal magnetization across a target range of myocardial T1s [5]. The optimization was performed under the constraints that inversions could not occur during image acquisition or pulses used for transient oscillation reduction. Pulse timings were calculated for RR intervals corresponding to heart rates of 40 to 120 beats-per-min and implemented on the fly during scanning using a look-up table. In vivo suppression levels were measured by dividing myocardial signal with BGS on (S) by myocardial signal with BGS off (S_0).

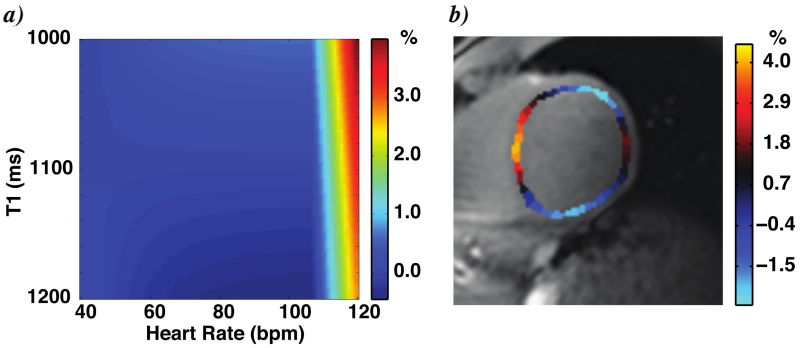

Figure 2: *Myocardial Suppression S/S_0 in (a) simulation and (b) in-vivo for a representative volunteer.*

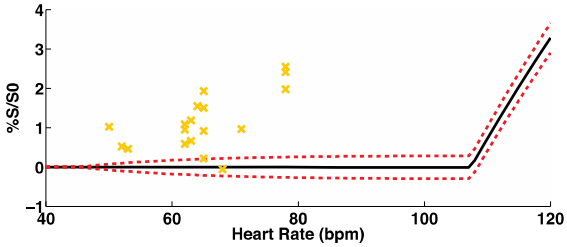

Figure 3: *Comparison of in-vivo myocardial suppression (yellow x's) against simulated values (black line ± one standard deviation red dashed).*

Results: Figure 2 contains simulated suppression levels for T1s from 1000 to 1200 ms and for heart rates from 40 to 120 bpm. Suppression to <1% is predicted at heart rates between 40 and 108 bpm while at higher heart rates, suppression of < 4% is predicted due to the shortened window in which to place the inversions. The average suppression of myocardium *in-vivo* was 1.14% ± 1.24%. In Figure 3, simulated suppression is shown together with in-vivo suppression. Table 1 reports the average suppression in each individual subject.

Subject	1	2	3	4	5	6
Mean Suppression %	0.82± 1.51	0.69 ±1.38	0.67±0.83	2.31±1.10	0.88±0.48	1.45 ±0.98
Average HR	68	64	52	78	62	65

Table 1: In-vivo myocardial suppression and myocardial blood flow

Discussion: This study demonstrates the feasibility of saturation-double-inversion to suppress myocardium over a broad range of heart rates, and in a fashion that is compatible with myocardial ASL. The discrepancy between simulated and in-vivo suppression levels may be due to imperfect inversion efficiency and/or mistiming due to changes in heart rate. The low residual myocardial signal achieved with this study may potentially reduce physiological noise and/or allow for a non-subtractive myocardial ASL approach. The latter would increase throughput, double SNR efficiency, and shorten any required breath-holds.

Acknowledgements: This work is supported by a Wallace H. Coulter Foundation Clinical Translational Research Award.

References:

[1] Zun et. al. MRM. 2009; 62(4):975-83. [2] Zun et al. JACC. 2011; 4(12):1253-61. [3] Noeske et. al. MRM. 2000; 44:978-82
[4] Sung et. al. MRM. 2008; 60(4):997-1002. [5] Maleki et al. MAGMA. 2012; 25(2):127-33.