A Comparison of Myocardial Signal Intensity Correction Methods in First-Pass Perfusion MRI

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Target Audience: Researchers and clinicians performing quantitative first-pass myocardial perfusion MRI.

Purpose: Quantitative myocardial perfusion MRI is able to provide *in vivo* estimates of absolute myocardial blood flow (MBF). Do to the non-linear relationship between signal intensity (SI) and gadolinium contrast agent (CA), the MR signal must be corrected prior to model fitting to avoid underestimation of MBF due to signal saturation in the myocardium. Several methods have been developed for avoiding saturation in the left ventricular blood pool; however, many studies assume saturation in the myocardial tissue is negligible. In stress perfusion studies with high flow, and thus high CA concentration in the myocardial tissue, this assumption may not be accurate. The purpose of this study is to compare three methods for correcting for saturation in the myocardial tissue in canine models of coronary stenosis measured during pharmacologically induced stress. Flow results from each method are compared to each other and to uncorrected signals.

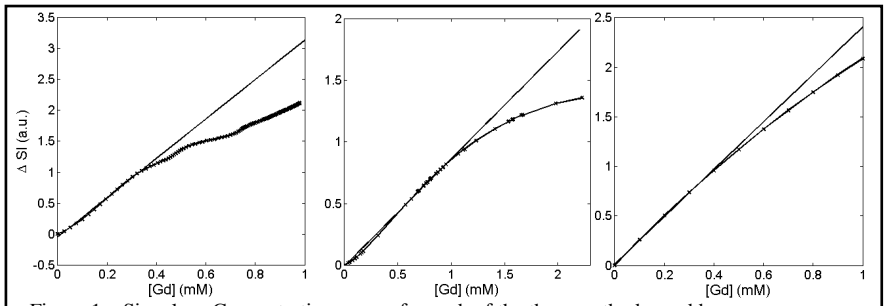

Figure 1 – Signal vs. Concentration curves for each of the three methods used here.

Methods: Ten perfusion studies were acquired on four canines, each of which had been chronically instrumented enabling the creation of variable degrees of stenosis to the left anterior descending and/or left circumflex coronary arteries during global coronary vasodilation with intravenous adenosine. A saturation recovery, turboFLASH sequence was used with TD/TE = 166/1.39 ms, flip angle of 12°, slice thickness of 8 mm, and isotropic in-plane resolution of 1.79 mm. A dual bolus protocol was used to obtain the arterial input function for MBF quantification with a 0.005mmol/kg dose Gd-DTPA followed by a second dose of 0.05 mmol/kg. Immediately following the acquisition, fluorescent microspheres were administered into the left atrium and aortic blood was sampled to enable calculation of myocardial blood flow. For each study a mid-ventricular slice was selected and the myocardium was manually segmented and divided into six equiangular regions. The SI in each region was averaged and the signal saturation was corrected using three methods: 1) an empirically determined curve calculated from previous animal imaging experiments; 2) a single-point T1 calibration (1); and 3) a simulation-based correction based on numerical solution of the Bloch equations (2). MBF values for each of the six regions were calculated from each study using the uncorrected and corrected myocardial time courses using the Tofts-Kety compartmental model. The results were then compared to the microsphere values with a one way analysis of variance (ANOVA) test. Student's t-test was used to determine significant differences between the groups and the Tukey-Kramer test was used to correct for multiple comparisons.

Results: Figure 1 displays SI versus CA curves for each of the three signal saturation correction methods. In each case, the relationship between signal and CA remained linear up to a concentration of approximately 0.5 mM, beyond which the relationship becomes increasingly nonlinear. Representative time courses from a region of high and low flow are shown in figure 2. In curves with moderate flow (right), the signal saturation is negligible. However, in curves with high flow (left), the saturation at the peak of the myocardial time course is 6.4%, 20.3%, and 3.2% for the three methods. The impact of saturation on MBF estimates can be seen in figure 3, which shows MBF estimates from uncorrected curves plotted with the corrected MR-based and microsphere flow values. The uncorrected MBF values were significantly lower than those from method 2 and the microsphere results ($p<0.001$). MBF values calculated with curves corrected with method 3 were also significantly lower than those from method 2 ($p=0.007$) and the microsphere results ($p<0.001$). MBF values calculated from curves corrected with methods 1 and 2 were not significantly different from the microsphere results.

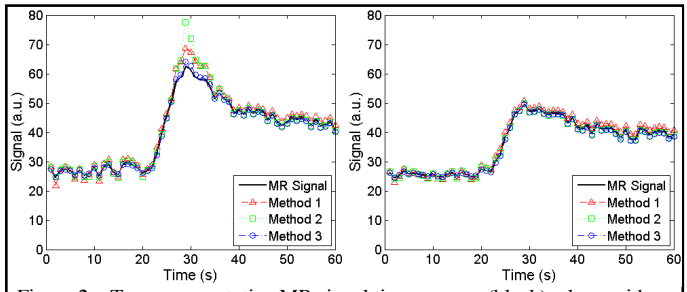

Figure 2 – Two representative MR signal time courses (black), along with corrected curves.

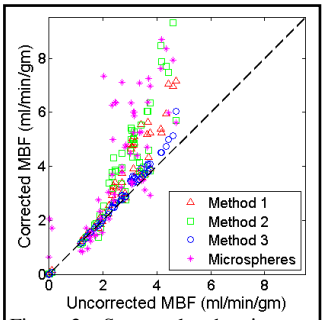

Figure 3 – Scatter plot showing uncorrected flow (x-axis) plotted against corrected flow values from the three methods tested and the microsphere results.

Discussion: The results shown in figure 1 suggest that signal saturation will affect myocardial tissue curves with CA concentration greater than or equal to 0.5 mM. The concentration in the myocardial tissue will depend on both the injected dose and the MBF. For a dose of 0.05 mmol/kg, as used here, saturation greater than 10% was seen on approximately 55% of myocardial segments. It should be noted that in this study, adenosine doses were selected to maximize MBF during imaging, and thus, this amount of saturation is likely greater than would be seen clinically. We also note than methods 2 and 3 are theory-based, and cannot account for variations in the flip angle due to field inhomogeneity or other sequence imperfections. In contrast, the correction for method 1 is determined empirically using T1 mapping at multiple concentration injections *in vivo*. Any changes to the imaging protocol would necessitate the calculation of a new correction curve.

Conclusion: Myocardial signal response during stress myocardial perfusion imaging displays a nonlinear response to CA concentration. Failure to correct for this response will result in underestimation of MBF. Future work should focus on testing and validating myocardial signal correction methods.

References: 1. Cernicanu A, Axel L. Theory-based Signal Calibration. Acad. Radiol. 2006;13:686-693. 2. Hsu LY et al. Nonlinear Myocardial Signal Intensity Correction. JMRI. 2008; 27:793:801.