Saturation correction of dynamic contrast enhanced MRI uptake curves for quantitative myocardial blood flow measurements using an assumed T_1 for blood

J. D. Biglands[1], A. Larghat[1], S. Plein[1], D. L. Buckley[1], M. Jerosch-Herold[2], D. Magee[3], R. Boyle[3], and A. Radjenovic[1]
[1]School of Medicine, University of Leeds, Leeds, UK, United Kingdom, [2]Radiology, Brigham and Womens Hospital, Boston, MA, United States, [3]School of Computing, University of Leeds, Leeds, United Kingdom

Introduction: Estimates of myocardial blood flow (MBF) based on analysis of dynamic contrast enhanced magnetic resonance imaging (DCE-MRI) of the myocardium typically rely on the assumption that signal intensity (SI) is proportional to concentration of contrast agent (CA). If the administered concentration of CA is sufficiently low this assumption is reasonable [1], however for the images to be useful for clinical reporting higher doses are preferable, necessitating the conversion of the SI values into concentrations, which requires knowledge of the native tissue T_1. Previously groups have acquired extra data to measure tissue T_1 in order to implement this conversion [2], but this extends the scanning time in an already time critical environment. Given the limited accuracy of such T_1 measurements, we propose that using an assumed value of blood T_1 will not introduce a significantly large error in estimated MBF values.

Method: The SI to concentration conversion is based on the assumption that the change in R_1 ($1/T_1$) due to the CA varies linearly with the concentration [2]. The SI is related to T_1 by the MRI pulse sequence equation i.e. $SI = \Omega f(T_1)$. Ω is a calibration constant relating to signal gain, proton density and other instrumental conditions. This equation is a function of the sequence parameters, which are known, and T_1, which is unknown. The calibration constant is calculated from the pre-contrast SI values and the assumed T_1 of blood. Thereafter SI values are converted to concentration by solving the equation for T_1 using a nonlinear minimization algorithm. By assuming that Ω is the same for the blood and the myocardium the pre-contrast myocardial SI is then used to calculate the T_1 of the myocardium in the same way.

Ten volunteers underwent DCE-MRI of the heart on a 1.5T whole body scanner (Intera Philips Medical Systems, Best, The Netherlands) under adenosine induced stress and rest conditions using 2 x 0.05 mmol/kg of Gd-DTPA (Magnevist, Schering, Berlin, Germany). A saturation recovery turbo FLASH pulse sequence was used to acquire short axis images of the heart. Using dedicated image analysis software (Mass 5.0, Medis, Leiden University, Leiden, The Netherlands) regions of interest depicting the myocardium and left ventricular blood pool were manually drawn, from which SI uptake curves were generated. A weighted average of the native cardiac T_1 blood values taken from [3-6] gave a mean $\pm$ standard deviation (SD) T_1 value of 1393 $\pm$ 126ms, giving a 95% confidence interval of 1141 ms to 1645 ms. The above method was implemented in MATLAB (The Mathworks, Natick, MA), to convert curves using a range of assumed T_1 values encompassing this confidence interval. A Fermi constrained deconvolution method [1], implemented in MATLAB, was used to estimate MBFs from each curve.

Results: The conversion algorithm successfully generated concentration curves for all but one of the volunteers, whose pre-contrast SI values were artefactually low such that there was no T_1 to satisfy the signal equation for the given Ω. The mean MBFs ($\pm$ SD) using the reference T_1 (1393ms) at stress and rest were 2.68 $\pm$ 0.67 ml/g/min and 1.14 $\pm$ 0.37 ml/g/min respectively. The mean myocardial T_1 derived using the reference blood T_1 was 1145 $\pm$ 173 ms. Fig. 1 shows the distribution of MBFs over the patients for each assumed T_1 value at rest and stress. With respect to the reference T_1, the largest mean difference in MBF occurred at T_1=1141ms, which gave an absolute mean difference in MBF at stress and rest of 0.59 ml/g/min and 0.3 ml/g/min giving percentage differences of 25% and 30% respectively.

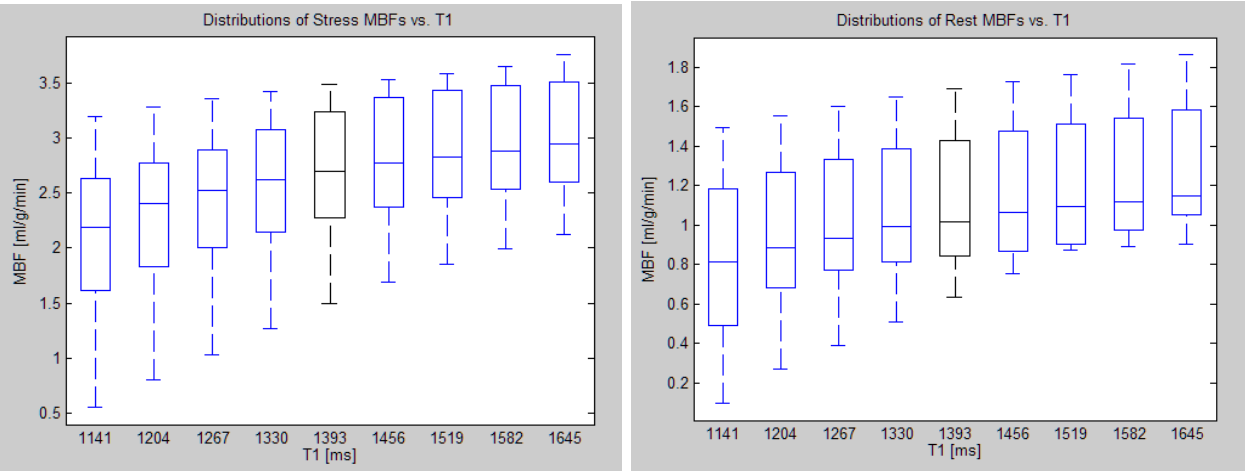

Figure 1. Boxplots showing median, 25th and 75th percentiles of the distribution of MBFs for each assumed T_1 value at stress and rest. The distribution corresponding to the mean T_1 value from the considered literature is shown in black.

Discussion: None of the median MBFs in Fig. 1 fall outside of the inter-quartile range of the MBFs estimated assuming the reference T_1, which suggests that the variation in MBF induced by varying T_1 is less pronounced than the experimental variation of MBFs within the reference (T_1 = 1393ms) dataset. It could be postulated that a larger dataset than ours using data not requiring correction, or corrected using measured T_1 values, might exhibit a narrower variation in MBF. However, the weighted mean of resting MBF measurements taken from studies [7-10], which satisfied these criteria, was 0.85 $\pm$ 0.32 ml/g/min. Our median MBFs for all T_1 values fall within this range suggesting that the variation in our results is not atypical. The mean derived myocardial T_1 in our experimental data was higher than a weighted mean of myocardial T_1 values taken from [3-6], 944 $\pm$ 87ms. This may be due to our assumption that blood and myocardial Ωs are equivalent or due to artefactually high pre-contrast blood signals.

Conclusion: Using a value of T_1 for the blood taken from the literature allows saturation correction of cardiac DCE-MRI datasets in typical clinical datasets, where native T_1 has not been measured. The subsequent estimates of MBF are consistent with literature values. Investigating a wide range of T_1 values yields modest differences in MBF, which implies that using assumed blood T_1 for correcting DCE-MRI perfusion curves is a realistic alternative to using measured values.

References: 1. Jerosch-Herold M., Med. Phys. 1998; 25(1):73:84. 2. Larsson H.B.W., MRM 1996 35:716-726. 3. Flacke S.J., Radiology 2001; 218:703-710. 4. Klein C., JMRI 2004; 20:588-594. 5. Messroghli D.R., MRM 2004; 52:141-14 6. Sharma P. JMRI 2006; 23:323-330. 7. Pack N.A. JCMR 2008; 10:52. 8. Fritz-Hansen T. JMRI 2008; 27:818-824. 9. Icihara T., MRM 2009; 62:373-383. 10. Vallee J.P., JMRI 1999; 9:197-203.