Saturation correction of dynamic contrast enhanced MRI uptake curves for quantitative myocardial blood flow measurements using an assumed T₁ for blood

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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 T₁. Previously groups have acquired extra data to measure tissue T₁ 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 T₁ measurements, we propose that using an assumed value of blood T₁ 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 R₁ (1/T₁) due to the CA varies linearly with the concentration [2]. The SI is related to T₁ by the MRI pulse sequence equation i.e. $SI = \Omega f(T₁)$. $\Omega$ 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 T₁, which is unknown. The calibration constant is calculated from the pre-contrast SI values and the assumed T₁ of blood. Thereafter SI values are converted to concentration by solving the equation for T₁ using a nonlinear minimization algorithm. By assuming that $\Omega$ is the same for the blood and the myocardium the pre-contrast myocardial SI is then used to calculate the T₁ 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 T₁ blood values taken from [3-6] gave a mean ± standard deviation (SD) T₁ value of 1393 ± 126 ms, 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 T₁ values encompassing this confidence interval. A Fermi constrained deconvolution method [1], implemented in MATLAB, was used to estimate MBF's 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 T₁ to satisfy the signal equation for the given $\Omega$. The mean MBF's (± SD) using the reference T₁ (1393 ms) at stress and rest were $2.68 ± 0.67$ ml/g/min and $1.44 ± 0.37$ ml/g/min respectively. The mean myocardial T₁ derived using the reference blood T₁ was $1145 ± 173$ ms. Fig. 1 shows the distribution of MBF's over the patients for each assumed T₁ value at stress and rest. With respect to the reference T₁, the largest mean difference in MBF occurred at T₁=141 ms, 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.

Discussion: None of the median MBF's in Fig. 1 fall outside of the inter-quartile range of the MBF's estimated assuming the reference T₁, which suggests that the variation in MBF induced by varying T₁ is less pronouced than the experimental variation of MBF's within the reference (T₁ = 1393ms) dataset. It could be postulated that a larger dataset than ours using data not requiring correction, or corrected using measured T₁ 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 $0.85 ± 0.32$ ml/g/min. Our median MBF's for all T₁ values fall within this range suggesting that the variation in our results is not atypical. The mean derived myocardial T₁ in our experimental data was higher than a weighted mean of myocardial T₁ values taken from [3-6], 944 ± 87 ms. This may be due to our assumption that blood and myocardial T₁'s are equivalent or due to artefactually high pre-contrast blood signals.

Conclusion: Using a value of T₁ for the blood taken from the literature allows saturation correction of cardiac DCE-MRI datasets in typical clinical datasets, where native T₁ has not been measured. The subsequent estimates of MBF are consistent with literature values. Investigating a wide range of T₁ values yields modest differences in MBF, which implies that using assumed blood T₁ for correcting DCE-MRI perfusion curves is a realistic alternative to using measured values.