

Is cardiac gating necessary in ASL? A computational and experimental study of flow dispersion and cardiac pulsations

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Introduction

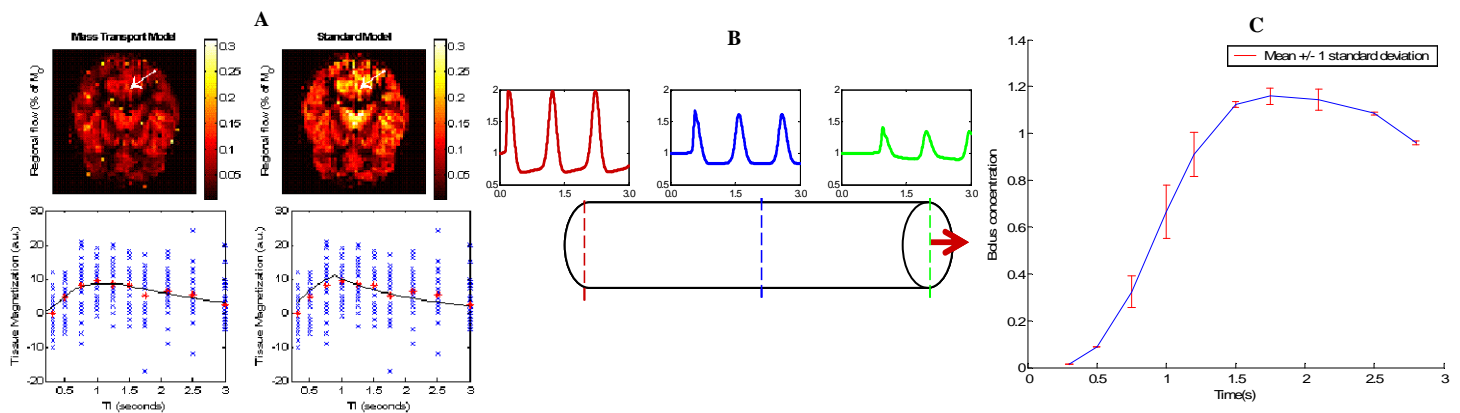
Interpretation and quantification of ASL measurements are confounded by the presence of significant levels of physiological variability in the signal. A greater understanding of the causes of this variability would aid in improving the predictive power of ASL in clinical practice. One of these causes is thought to be the presence of pulsatility in the blood flow entering the brain, due to the cardiac cycle. Experimental data^{*} has previously shown that such pulsatility has a significant effect on the ASL signal.

Methods

Incorporating pulsatile flow into existing models of the ASL signal is not possible in their current form. We have thus developed a full mass-transport model to simulate the transport of the ASL signal from the tagging to the imaging band, using fluid dynamics equations to describe flow in an elastic vessel. For simplicity we assume a single arterial vessel supplying a tissue compartment. Cardiac pulsatility can then be incorporated using pulsatile inlet flow field. The model was verified against ASL data that were acquired from a healthy subject at 3T with a spatial resolution of 4 mm by 4 mm by 6 mm. Data were collected using a pulsed ASL sequence of Q2TIPS with PICORE at 10 different inversion times (TI) (0.3, 0.5, 0.75, 1.0, 1.25, 1.5, 1.75, 2.1, 2.5, 3.0 seconds), Q2TIPS saturation at 0.7 seconds, each measurement was repeated 30 times.

Results

We validated the model performance by comparison with results obtained via the standard model[†]. Figure A shows the estimated CBF in a slice from a healthy subject (top) and a typical voxel (bottom). There is good overall agreement between the model predictions, although our mass transport model provides a better fit to the TI-averaged experimental data (giving a lower RMS error), as the fitted tissue magnetization curve is smoother reflecting dispersion of the tag during transit. There is significant variability in the ASL signal that cannot be accounted for, however, with assumed steady state flow conditions. We thus considered the response of the model with a pulsatile inlet flow to quantify the level of variability in the ASL signal that can be attributed to this effect. The flow field at three positions along the arterial vessel is shown in Figure B, illustrating the variability in the flow field with both distance and time. To simulate a real experiment, we tag the blood at random points in the cardiac cycle for each sample and simulate the tissue magnetization curve accordingly. The resulting variability in this curve is shown in Figure C, enabling us to quantify the likely variability in the ASL signal due to cardiac pulsatility.



Panel A: Regional flow maps as a percentage of equilibrium magnetization; bolus tissue concentration for the voxel indicated by the white arrow (^{*} experimental data; ^{*} averaged experimental data; solid line fitted model). Panel B: Simulated blood flow rate at different locations in arterial vessel. Panel C: variation, due to cardiac pulsations, in model predictions of bolus tissue concentration.

Discussion

Our simulations indicate that cardiac pulsatility contributes up to around 20 % of the ASL signal variability. This has implications in the choice of single or multiple inversion times for quantifying CBF using ASL. Whether cardiac gating is needed to obtain more accurate measures of CBF using ASL remains, however, an open question, which we will be able to quantify using the modelling approach outlined here. Our model allows us to base the ASL on a proper physiological basis and hence investigate physiological effects on the signal.

^{*} W. C. Wu, Y. Mazaheri, and E. C. Wong, "The effects of flow dispersion and cardiac pulsation in arterial spin labeling," IEEE Trans Med Imaging

[†] Buxton R. B. (2005) "Quantifying CBF with arterial spin labeling," J Magn Reson Imaging