Reliable isolation of the intravascular contribution in Arterial Spin Labelling MRI

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Introduction: Arterial Spin Labelling (ASL) is capable of measuring cerebral perfusion by magnetically labelling water in blood. The signal arising from the labelled water as it arrives in the tissue and undergoes exchange can be measured and cerebral blood flow (CBF) quantified. The labelled water in arterial blood yet to reach the tissue will also give rise to an intravascular contribution, which is commonly minimized by the application of flow-crushing gradients [1]. However, this process has not been fully characterised. Hence practical implementations must make a decision on the applied gradient, leading to variable amount of arterial signal removal. Additionally the arterial signal itself is a further physiological measure available that may provide valuable information in a clinical context, for example, it may be helpful in patients with arteriovenous malformation.

Multiple inversion ASL provides the time course of development of the ASL kinetic curve. It allows parameters such as the arrival delay or trailing edge of the bolus to be estimated. We hypothesized that it should be possible to estimate the intravascular contribution to the signal. Thus this study investigated the reliability of ASL analysis that included this intravascular contribution, to determine whether this is a routine alternative to the use of flow-crushing where it is not desirable or feasible.

Methods: T1 weighted structural images and resting-state PASL data were collected in 38 healthy subjects (age range 20 to 35 years) using a FAIR preparation and a single-shot 3-dimensional GRASE readout [2] at 3T and a series of 11 inversion times (TIs) [400:200:2400 ms], each one repeated 10 times.

The ASL signal in every voxel was modelled using two components: the standard kinetic curve [3] representing the labelled blood once it has arrived in the capillaries and is undergoing rapid exchange with the tissue; and a exponential decay with the T1 of the blood to represent the passage of arterial blood through the voxel destined for elsewhere. The two components are illustrated in Figure 1. The full model contained 6 parameters: CBF, tissue blood arrival time and bolus length, intravascular flow, arrival time and bolus length.

Parameter estimation was performed using a probabilistic inference approach designed for ASL data [4, 5]. This allowed prior information about the parameters to be included to improve the reliability of the estimation. No prior information was set for the CBF value, but temporal parameters (arrival times and bolus lengths) were given priors as in Table 1. In the majority of voxels no intravascular contribution is expected in the signal. The fitting of this extra component (with an extra three parameters) in the model, in tissue-only voxels could result in over fitting. Thus potentially leading to an artifactual intravascular signal arising out of the noise or from fitting to the tissue signal. Hence, the arterial flow value was subject to an Automatic Relevancy Determination (ARD) prior [5, 6]. The ARD prior ensures that the arterial component is only applied in voxels where the data supports the extra component. The ARD prior thus penalises unnecessary model complexity.

The probabilistic inference method produces estimates for the model parameters along with associated confidence measures (variances). The group data were analysed to produce a group mean arterial flow map using an approach that allows both the individual flow and confidence estimates to be included within the analysis [7]. Thus the final group estimate included within-subject as well as across-subject variability.

Results: Figure 2 shows the group average arterial flow results, where the flow has been thresholded at an equivalent level of 10 ml/100 g/min. The pattern of arterial flow in this group appears to match with expected locations of major vessels including the middle cerebral artery and the Circle of Willis. There is some blurring of the flow associated both with the relatively low resolution of the perfusion data and the effect of averaging over 38 subjects each with their own unique vascular structure.

The alignment of the estimated intravascular contribution with the vasculature on an individual level has also been verified. Figure 4 shows the arterial flow map from a single subject along with their time-of-flight angiographic image that has been thresholded to reveal only the vessels. There is good alignment of the intravascular contribution with the vessels despite difficulties in registration. The resolution results in a more spatially blurred looking intravascular flow compared to the TOF angiography, this is compounded by some blurring in the z-direction during acquisition.

Discussion: The results of the current study suggest that it is feasible to extract the intravascular contribution in ASL data collected in the absence of flow-crushing gradients. Here, a probabilistic framework and an ARD prior have been used to avoid over fitting of the more complicated two-component model. This conservative approach potentially means that information from smaller vessels is lost within the tissue component. However, this would be difficult to recover given the resolution of ASL data. This scenario would not be greatly different from the use of flow crushing gradients, since these will typically only remove the contribution from larger vessels. The model used here was relatively simple, since it did not account for the effects of flow dispersion both on the tissue and arterial components. Inclusion of the effects of dispersion may affect the separation of the two components, though the same pattern of results would be expected.

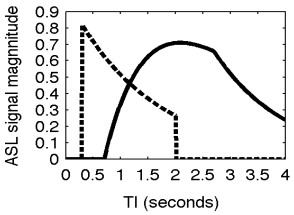

Figure 1: Kinetic curve model (-) and intravascular contribution (--). Bolus length is 1.7 s.

Table 1: Definition of parameter priors.

Parameter prior	Mean	Std. dev
CBF	*Non-informative*	
Tissue arrival time	0.7	0.3
Tissue bolus length	1	0.3
IV flow	*ARD*	
IV arrival time	0.5	0.3
IV bolus length	1	0.3

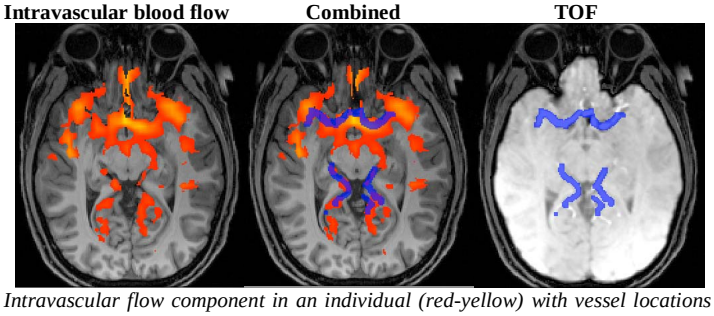

Figure 3: Intravascular flow component in an individual (red-yellow) with vessel locations from TOF angiography (blue).

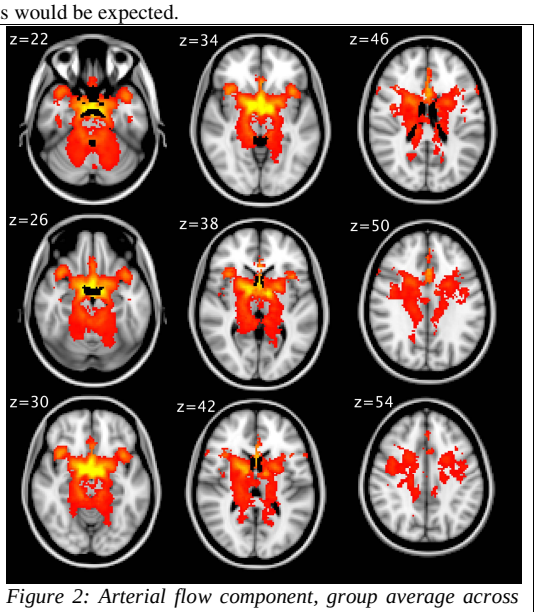

Figure 2: Arterial flow component, group average across 38 subjects.

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