

In Vitro and In Vivo Measurement of Pseudo Continuous Arterial Spin Labeling Efficiency

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Introduction Pseudo continuous arterial spin labeling (PCASL) uses a train of discrete RF pulses and slice selective gradients to adiabatically invert blood flowing through a labeling plane. Several groups have previously shown theoretically and in vivo how blood velocity, off- resonance and gradient imperfections can contribute to discordance between expected and achieved labeling efficiency (LE) [1], defined by $\text{abs}(\text{control-label})/(2*\text{control})$. However, previous in vivo measurements of LE measure relative degrees of labeling efficiency or are susceptible to all other quantitation issues of cerebral arterial spin labeling (ASL). In order to quantitatively assess LE we used a novel technique developed by [2] that labels blood in the feeding vessel and measures the perfusion signal a short distance further downstream in the blood pool. We measured LE in a flow phantom and in vivo, then compared these results to Bloch simulations of PCASL efficiency based on velocity profiles measured using phase contrast.

Methods All studies were conducted under an approved IRB protocol using a 3T Philips MRI scanner. The unbalanced gradient PCASL train consisted of 500 μ s Hanning shaped RF pulses with maximum gradient amplitude of 10 G/cm and mean gradient of 1 G/cm over the inter-pulse interval of 1000 μ s. Phantom LE was measured with B_1 strengths of .2 μ T, .6 μ T, 1.0 μ T and 1.4 μ T. The total labeling train was 500ms with a label delay of 30ms. A Fast Field Echo

acquisition with a short TE was used to limit flow thru artifacts. Ten pairs of control and label images were acquired for each LE measurement totaling a 5 minute scan. A phase contrast sequence with a velocity encoding gradient of 150 cm/s, 3mm slice thickness and 0.7 mm in plane resolution was acquired for velocity profile estimates. LE with respect to flow velocity and B_1 was simulated using a Bloch model. A gravity feed, constant flow phantom was constructed and velocity was modulated by varying the circuit resistance. The flow channel consisted of polyvinyl chloride tubes filled with 3% agar surrounded by a water reservoir. Lumen size ranged from 2mm to 8mm in order to control velocity profile. In vivo measurements were made in 5 healthy controls in the vertebral and carotid arteries.

Results and Conclusions Figure 1 shows theoretical LE estimates with respect to velocity and B_1 amplitude. Figure 2 shows a representative velocity profile histogram across the cardiac cycle of a healthy control measured via gated phase contrast. Phantom LE measurements show high agreement with theoretical Bloch model predictions of LE with respect to velocity (Figure 3), except for a +4% bias which is likely

explained by the use of water instead of blood as the labeling medium (longer T1 and T2). In vivo LE could often be explained by differences in arterial velocity.

However, in several of the in vivo measurements, LE variability was significantly lower than predicted by velocity alone. Since B_0 or B_1 inhomogeneity may lead to incomplete inversion at labeling plane, these data suggest that direct measurement of LE or use of a technique that is robust to B_0 inhomogeneity (e.g. multiphase ASL) may be necessary for accurate ASL quantification.

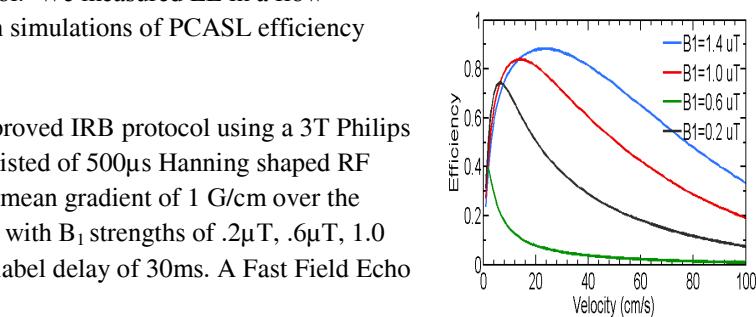


Figure 1. Theoretical Bloch LE estimates with respect to velocity and B_1 .

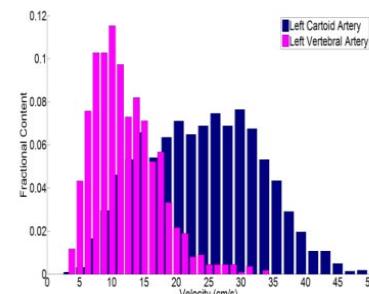


Figure 2. Representative velocity histogram across the cardiac cycle in the left carotid and left vertebral artery of a single healthy control.

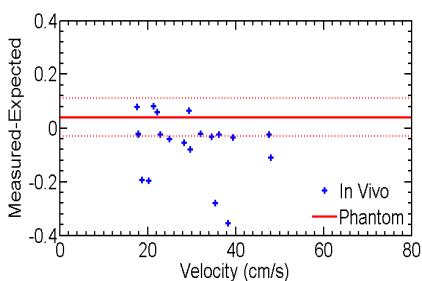


Figure 4. Bland Altman plot of measured - predicted LE vs velocity. Red bars represent mean and standard deviation of phantom LE measurements.

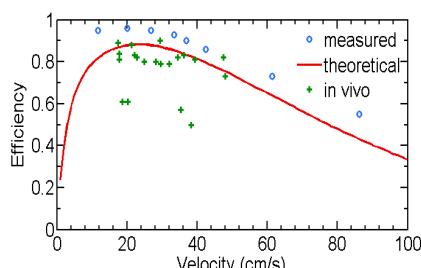


Figure 3. In vitro, in vivo and Bloch simulated LE with respect to velocity at B_1 strength of 1.4 μ T.

References:

1. Jung et al. *MRM* 2010
2. Schmithorst et al. *JMRI*, 2013