Probing Arterial Spin Labeling MR Signal in Human Brain with T1ρ Technique

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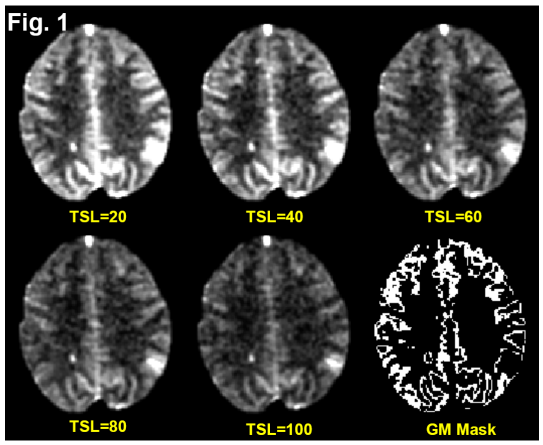
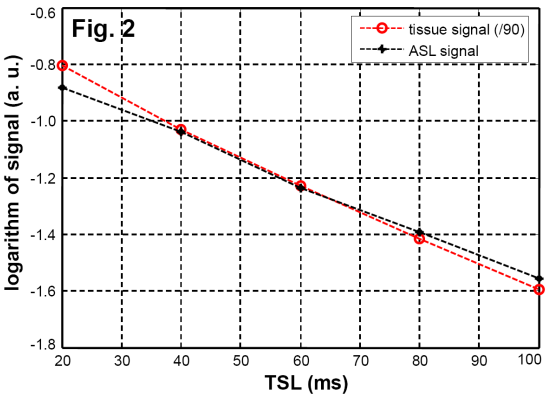
Introduction: Tracking arterial spin labeled (ASL) water in the human brain provides key information on the dynamics of water exchange and brain perfusion measured by PET and MR imaging (1). Recently, some MR based methods (diffusion, T2* and T2), have been proposed for this purpose (2-4), however the published findings were controversial. A study using T2* based pulsed ASL (PASL) experiment reported that ASL water remains mainly in the interstitial space (2). Since the T2* of the interstitial space is significantly longer than that of the tissue, this implies a slow water transport through the neuronal membrane (on the order of tens of seconds). Conversely, a T2-based pseudo-continuous arterial spin labeling (pCASL) study (3) suggested that the ASL signal is significantly influenced by an arteriole contribution for a short post labeling delay (TD, up to 1525 msec), while the T2 of the pCASL signal approaches that of the tissue with a longer TD (2 sec). To resolve this controversy, we investigated the T1ρ relaxation property of the ASL signal. The rationale for our approach is based on the fact that T1ρ of blood is insensitive to the blood oxygenation level, thereby allowing a simplified MR signal modeling method.

Methods: All experiments were performed on 3T Siemens Trio scanner using a body transmit RF coil and 12 channel head receive coil. Five healthy volunteer subjects were scanned. T1ρ preparation technique, designed to be insensitive to both B0 and B1 field inhomogeneity, was applied (5). Time of spin locking (TSL) varied from 20 to 100 msec. The B1 field for spin locking was chosen at 8 μT (350 Hz). FAIR preparation pulses (6) were used to modulate flow sensitivity in control and labeling image. QUIPSS II technique (7) was incorporated to modify the labeling bolus. The labeling area (200 mm thickness) was saturated with periodic RF pulse trains at 1800 ms (TD) prior to the True-FISP data acquisition. The total labeling time (TI) was 2400 msec to ensure the arriving of labeling bolus at the capillary bed. MR parameters for True-FISP were: FOV of 256x192 mm^2, voxel size of 1.3x1.3x8 mm^3, TR of 5000 ms, TE of 1.43 ms, centric ordering with single shot acquisition. Acquisition time was approximately 2.5 mins with 16 repetitions.



Results: Figure 1 shows images with T1ρ weighted perfusion signal at different TSL from a healthy subject. The perfusion signal contrast between GM and WM is clearly demonstrated. Both perfusion and reference signal will be averaged within GM ROI, as illustrated in the last image. The T1ρ decay of the venous blood signal (from the sagittal sinus) was approximately 100 ms; comparable to the T1ρ of the cortical GM. Figure 2 presents T1ρ decay profiles (over TSL) of the ASL signal at two TD values as well as T1ρ decay of the tissue signal with a GM ROI (reduced by 90 times) as a comparison. The T1ρ decay rate of the perfusion signal was always slower than that of the brain tissue signal at both labeling conditions. A decrease in TD from 1800 to 850 ms (data not shown) did not significantly change the T1 ρ decay rate of ASL signal, suggesting an insignificant contribution from the labeled water inside artery or arterioles in the PASL study. These findings are consistent with the results from previous T2* based ASL signal studies.



Discussion: In this study, we used the T1ρ characteristic of ASL signal to investigate the labeled water compartment structure. Our result was in agreement with previously published PASL T2* studies. It was noted that for PASL experiments with total labeling time greater than 2 seconds, the saturation of the labeling area (QUIPSS II) at 850 ms or more prior to imaging would eliminate any apparent contamination from the labeled water in arteries or arterioles. This finding is consistent with the results from other PASL studies (8).

T1ρ of the labeled water measured in this study is much lower than the T2 of 140 ms measured in pCASL study at TD=850 ms, but still is higher than the T2 measured at TD=2000 ms (the same as the GM tissue). We postulate that the discrepancy between the T1ρ PASL and T2 pCASL may be due to the difference between their techniques. When the TD is closer to the arterial transit time (about 600 to 800 ms) in pCASL, the fast-moving, freshly labeled blood stays at the artery and arteriole side during imaging. As a result, T2 values become close to that of slightly deoxygenated arterial blood, as shown in PASL studies (8). Alternatively, when a longer TD value (2000 ms) was used, time lapses from labeling to imaging was 3650 ms, much longer than the cerebral vascular transit time of 2.8 to 3.0 second (9). Consequently, a substantial amount of labeled water may enter venules or veins and result in the apparent T2 of pCASL signal close to that of the tissue. The total labeling time of 2 to 2.4 seconds in our PASL technique was too short for the labeled water to enter venules or veins. A tight labeling bolus (20 mm thickness in this study) also ensures all labeled water to reach the capillary bed before the data acquisition.

References: **1.** Zhou, *et al.,* JCBFM 2001;21:440; **2**. He, *et al.,* Proc. Brain & Brain PET 2009; **3.** Liu, *et al.,* MRM 2010;in press; **4.** Wang, *et al.,* JCBFM 2007;27:839; **5.** McCommis, *et al.,* MRM 2010;63:1442; **6.** Kim, *et al.,* MRM 1995;34:293; **7.** Wong, *et al.,* MRM 1998;39:702; **8.** Donahue, *et al.,* NMR Biomed 2006;19:1043; **9**. Ibaraki, *et al.,* JCBFM 2007;27:404.