

# ASL Perfusion Measurement Using a Rapid, Low Resolution Arterial Transit Time Prescan

W. Dai<sup>1,2</sup>, P. M. Robson<sup>1,2</sup>, A. Shankaranarayanan<sup>3</sup>, and D. C. Alsop<sup>1,2</sup>

<sup>1</sup>Radiology, Beth Israel Deaconess Medical Center, Boston, MA, United States, <sup>2</sup>Radiology, Harvard Medical School, Boston, MA, United States, <sup>3</sup>Applied Science Laboratory, GE Healthcare, Menlo Park, CA, United States

**Introduction:** In general, the arterial spin labeling (ASL) signal reflects a mixture of perfusion and arterial transit time (ATT) effects. Techniques to reduce the ATT contribution have been proposed (1, 2), but they can compromise sensitivity or fail to eliminate ATT effects unless a reasonably good estimate of the ATT is available. Unfortunately, ATT can span a wide range in broad clinical populations and optimization of these scans is problematic (3, 4). Estimation of perfusion and ATT with images acquired at multiple delays has been reported, but these methods decrease the sensitivity of the perfusion measurement. A high resolution map of ATT may not be necessary, however, because nearby areas share similar arterial supply and hence ATT. Here, we propose a rapid, low resolution scan at multiple labeling delays to acquire a map of ATT. This ATT map can be used to either guide the parameter selection for an ATT insensitive perfusion acquisition or to quantify perfusion from an ATT sensitive high resolution scan.

**Methods:** Pulsed-continuous arterial spin labeling (PCASL) (5) was used for all labeling ( $B_{1\text{ave}}=14$  mG,  $G_{\text{ave}}=0.07$  G/cm, and  $G_{\text{max}}/G_{\text{ave}}=10$ ,  $\Delta t=1.5$  ms). Background suppression was achieved with presaturation and 4 inversion pulses applied at optimized times to minimize instabilities from motion and other physiologic or instrumental fluctuations. Images were acquired with a 3D stack of spirals RARE (FSE) sequence. All slice encodes were performed after each ASL preparation. For higher resolution images, interleaved spiral encoding was employed with different spiral interleaves for each preparation. For the low resolution ATT estimate, a single interleave was used to provide a single-shot image. Vessel suppression (VS) was achieved in some image acquisitions by applying an adiabatic vessel saturation sequence (6) immediately prior to image acquisition. Two additional reference images (a. saturation recovery at 2 s; b. saturation at 4.3 s, and inversion pulse at 1.65 s) were appended after the ASL sequence to provide necessary  $T_1$  and  $M_0$  values for quantification.

Three subjects were studied on a GE 3Tesla images with a receive-only 8-channel head array coil. A low resolution transit time acquisition (labeling duration of 2 s and postlabeling delays of 0.7 s, 1.3 s, 1.9 s, 2.5 s, 3.0 s) was performed with one interleave and one average per label-control pair. The total time for acquiring these 5 volumes was 1 min. Vessel suppression was applied for all delays. To provide a sensitive but ATT dependent image, a short post-labeling delay (700 ms), long labeling duration (4.3 s), and vessel suppressed sequence was performed with 8 interleaves and 3 averages (7 Min). Finally, 2 perfusion acquisitions of 8 interleaves and 3 averages were performed using optimal post-labeling delays estimated by an automatic algorithm, as described below, applied to the ATT prescan. To evaluate residual vascular contamination in these images, acquisition with and without vessel suppression was performed.

Quantification of ATT and perfusion was performed offline. The prescan was quantified in two ways. First, a quick algorithm to derive an optimal post-labeling delay was implemented based on subdividing the brain into 8 regions. The optimal post-labeling delay was defined as the maximum of the derived delays. In addition, pixel-by-pixel ATT maps were calculated by comparing signal change with delay to a quantitative model (1, 7). Perfusion was quantified using the ATT map from the prescan and the measured  $T_1$  and  $M_0$ .

**Results & Discussions:** The average calculated postlabeling delay for three volunteers was  $1.67 \pm 0.18$  (from 1.41 s to 2.04 s). The transit times calculated from eight regions shows that posterior regions have longer delay than anterior regions and superior

Table 1. Transit time calculated across three subjects.

transit time	left inferior	left superior	right inferior	right superior
anterior	1.24±0.24	1.32±0.23	1.23±0.23	1.32±0.23
posterior	1.53±0.23	1.63±0.20	1.55±0.21	1.61±0.20

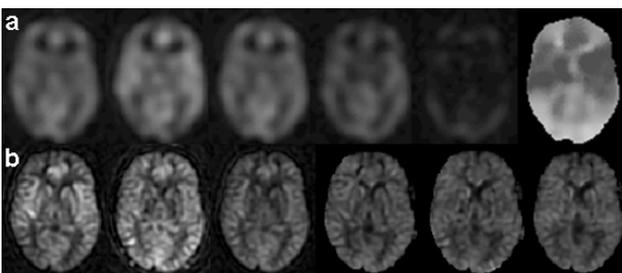


Figure 1. a) The low resolution subtraction images from multiple delays (0.7 s, 1.3 s, 1.9 s, 2.5 s, 3.0 s) and transit time map calculated from delay images; b) the high resolution subtraction images (the first three images) and transit time corrected images (the last three images) from delays (700 ms, optimal delay without and with vessel suppression).

Table 2. Transit times and perfusions from hand-drawn regions

	Basal ganglia	Posterior	Anterior
transit time (s)	0.92 ± 0.20	1.52 ± 0.38	1.06 ± 0.24
perfusion(ml/100g.min) (with delay 0.7 s)	49 ± 3	46 ± 3	62 ± 5
perfusion with no VS (with optimal delay)	58 ± 6	59 ± 4	54 ± 13
Perfusion with VS (with optimal delay)	56 ± 4	49 ± 2	62 ± 10

regions have

longer delay than inferior regions (Table 1). The transit time map shows expected features. The basal ganglia have a shorter transit time than other gray matter regions. Posterior regions have longer transit time than anterior regions (Figure 1a & Table 2). The high resolution image with 700 ms demonstrates the bright signal in the regions with lower transit times (Figure 1b). With transit time map correction, all high resolution scans show consistent perfusion values in gray and white matters. In summary, a series of quick, low resolution ASL prescan is feasible to guide the parameter selection for an ATT insensitive perfusion acquisition and provide accurate perfusion quantification from an ATT sensitive high resolution scan.

**References:** 1. Alsop et al, Journal of Cerebral Blood Flow and Metabolism 1996;16:1236-49. 2. Ye et al, Magn Reson Med 1997;37:226-35. 3. Detre et al, Neurology 1998;50:633-41. 4. Calamante et al, Stroke 2002;33:1146-51. 5. Dai et al, Magn Reson Med 2008; In Press. 6. Wong, Magn Reson Med 2007;58:1086-91. 7. Buxton et al, Magn Reson Med 1998;40:383-396.