# Arterial Compliance of the Middle Cerebral Artery Measured with Short Inversion Time Pulsed Arterial Spin Labelling

Esther A.H. Warnert[1], Judith E. Hall[2], and Richard G. Wise[1]

[1]*CUBRIC. School of Psychology, Cardiff University, Cardiff, United Kingdom,* [2]*Department of Anaesthetics and Intensive Care Medicine, School of Medicine, Cardiff University, United Kingdom*

**Target audience** Researchers and clinicians interested in non-invasive assessment of arterial compliance of cerebral arteries.

**Introduction** Arterial compliance (AC) is a potential marker of cerebral vascular disease and describes the relationship between a change in arterial blood volume (ΔaBV) arising from a change in blood pressure, such as between systole and diastole [1]. Pulsed arterial spin labelling (PASL) could offer a non-invasive way to measure aBV if used with short inversion times (TI < 1000 ms) such that the labelled blood remains in large arteries [2]. However, differences in dispersion and arrival time of the labelled blood due to the pulsatility of blood flow confound comparison of arterial signal using ASL [2]. Here, we aim to calculate AC of the middle cerebral arteries (MCA) by measuring systolic and diastolic aBV with short TI pulsed ASL while taking the differences in label kinetics between the cardiac phases into account.

**Methods** Multi-inversion time PICORE PASL acquisitions were performed on seven healthy volunteers (age 25.6 ± 1.3 years). Seven inversion times (TIs) were acquired (250 – 850ms, spacing 100 ms). A QUIPSS II [3] cut off of the label was applied at 700 ms for inversion times > 700 ms. Images were acquired with a spiral gradient echo sequence (TE=2.7 ms, TR=1400 ms, 80 tag-control pairs, 14 slices, slice gap 1 mm, voxel size 3.5x3.5x7 $mm^3$). A single TR image was obtained with the same acquisition parameters but without labelling, to obtain the equilibrium magnetisation of cerebrospinal fluid ($M_{0,CSF}$). The cardiac cycle was measured by finger plethysmography. Brachial artery blood pressure (BP) was measured non-invasively with an MRI compatible blood pressure cuff during every ASL scan (seven times in total for each participant).

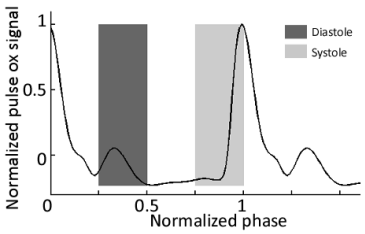


*Figure 1. Average finger plethysmography trace. Images acquired in diastole (dark gray) or systole (light gray) are used for further analysis.*

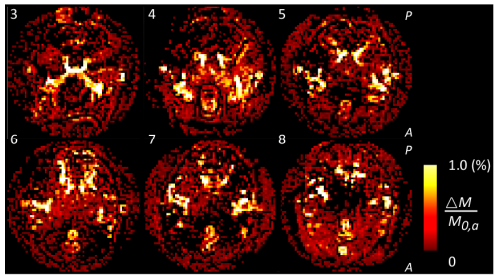


*Figure 2. Example of diastolic arterial signal map, showing 6 axial slices inferior to superior (ΔM/$M_{0,a}$ in %, A = anterior, P = posterior).*

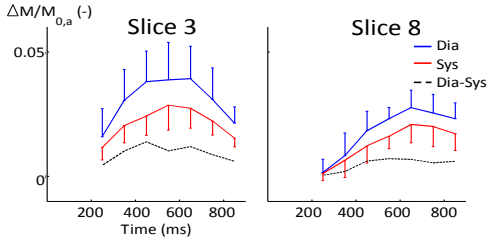


*Figure 3. Example of average (N=7) arterial signal over time in proximal (slice 3) and distal (slice 8) parts of the flow territory of the LMCA. Errorbars are one standard deviation.*

**Analysis** Images acquired during systole and diastole were separated based on the plethysmography trace (Figure 1), taking the delay of the finger pulse compared to the cerebral pulse into account. Average tag and control images were calculated for these two cardiac phases and subtracted to obtain maps containing arterial signal (example in Figure 2). In these maps, regions of interest (ROIs) were determined in the flow territories of the right and left MCA (RMCA and LMCA) in 6 slices (slice = 3-8 inferior to superior, as in Figure 2). Per slice, the 1% of voxels with the highest signal intensity on the right half of the brain were defined as the ROI containing the RMCA flow territory. The same was done for the LMCA flow territory. Regional average time courses of the label were plotted for each slice individually (examples in Figure 3). The fully relaxed magnetisation of arterial blood ($M_{0,a}$) was calculated based on $M_{0,CSF}$, and was used to calculate aBV: $\Delta M_a(t) = 2\alpha M_{0,a} e^{-t/T_{1,a}} aBV$, with α=1 (label efficiency) and $T_{1,a}$ = 1664 ms ($T_1$ arterial blood at 3T)[4]. This equation gives aBV as a volume fraction of the voxel (expressed as $\%_v$) and would represent arterial volume if time point $t$ lies between arrival of the leading and trailing edge of the labelled bolus. AC (in $\%_v$/ mmHg) was then calculated as $\Delta aBV / \Delta BP$, with $\Delta aBV = aBV_{dia} - aBV_{sys}$ and $\Delta BP = BP_{sys} - BP_{dia}$.

**Results and Discussion** The difference between systolic and diastolic arterial signal plateaus at early TI (350ms in proximal slices and 450 ms for distal slices, see Figure 3) and decreases again later on (TI > 650ms). To not be confounded by differences in dispersion and arrival time between systole and diastole, TI=550ms was used to calculate ΔaBV. Table 1 shows the estimated AC of the most proximal portion of the flow territory of the RMCA (0.025±0.017 $\%_v$/mmHg) and LMCA (0.029±0.020 $\%_v$/mmHg), which are not significantly different (paired t-test, p>0.05). These values are smaller than previously reported AC for the common carotids, which is to be expected of arteries further down the vascular tree [1]. In addition, Figure 4 illustrates the decrease in AC with increasing slice number, i.e. further along the (cerebral) vascular tree. Higher spatial resolution and intravascular BP estimation, would offer a more accurate estimate of local AC. However, here we have shown that short TI PASL has the potential to assess local cerebral AC.

***Table 1. Average Blood pressure (BP) and arterial compliance (AC) and their standard deviations.***

| | | |
|---|---|---|
| $BP_{sys}$ (mmHg) | 114.2 ± 5.8 | |
| $BP_{dia}$ (mmHg) | 61.5 ± 4.3 | |
| $BP_{sys}$-$BP_{dia}$ (mmHg) | 53.3 ± 8.1 | |
| | ***RMCA*** | ***LMCA*** |
| ***AC - Slice 3 ($\%_v$/mmHg)*** | *0.025 ± 0.017* | *0.029 ± 0.020* |

*No significant differences between flow territories of RMCA and LMCA (paired t-tests, p>0.05)*

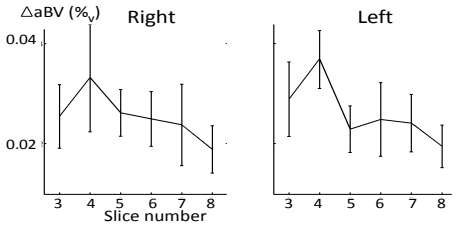


*Figure 4. ΔaBV ($\%_v$) in flow territories of the RMCA (Right) and LMCA (Left), calculated for different slices at TI = 550ms. Errorbars indicate standard error of the mean. (N=7)*

**References** 1. O'Rourke, MF, et al., Am J Hypertens, **5**, p: 426-44 (2002). 2. Wu, WC, et al., IEEE Trans Med Imaging, **1**, p: 84-92 (2007). 3. Wong, EC, et al., Magn Reson Med, **5**, p: 702-8 (1998). 4. Chappell, MA, et al., Magn Reson Med, **5**, p: 1357-65 (2010).