# Sampling the arterial input function in T1 weighted dynamic contrast enhanced perfusion

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**Introduction**

When using dynamic contrast enhanced (DCE) magnetic resonance imaging (MRI) to assess cerebral blood flow (CBF), correct determination of the arterial input function (AIF) is critical to CBF quantification (e.g. (1,2)). One well-known source of error affecting the AIF is the partial volume effect (PVE) (2). Using T1-weighted DCE-MRI, we demonstrate the result of the PVE on CBF quantification in vivo. We further propose a method we denote venous normalization to compensate for PVE.

**Methods**

All MRI experiments were performed on a 3 T system (Philips Achieva, The Netherlands) with an eight-element SENSE head coil. A Gd contrast bolus (Magnevist, 0.1 ml/kg bodyweight to avoid full relaxation) was injected using an automatic contrast injector (Medrad). The bolus passage was imaged using a saturation recovery gradient recalled sequence with a 90 degrees non-selective preparation prepulse (saturation time delay Td = 120 ms). Image parameters were flip angle $\alpha$=30 degrees, TR=3.88 ms, matrix size 96 (reconstructed to 256), scan percentage 80%, centric phase ordering, FOV=240 mm, and 2 slices 8 mm thick, giving a temporal resolution 0.7-0.8 s. 180 dynamic images were obtained. The bolus (5 ml/s, 20 ml saline) was injected after the 10th dynamic. The center of the most caudal slice was placed orthogonal to the internal carotid artery, in order to optimize the arterial input function. The MR signal as function of time s(t) is related to concentration c(t) by

$$[1] \qquad s(t) = M_0 \sin(\alpha)[1-\exp(-Td(R_1+\Delta R_1(t)))] \ , \ \Delta R_1(t)=r_1 c(t)$$

The contrast relaxivity $r_1$ of 4 $s^{-1}mM^{-1}$ was provided by the manufacturer for 3 T, and was assumed equal for tissue and blood vessels. We measured $R_1$ and $M_0$ before contrast injection using the same sequence as for monitoring the bolus passage, using 1 dynamic and a set of 11 Td values from 0.05 to 10 s. The signal equation for a saturation recovery (Eq. [1] with $\Delta R_1 = 0$, since centric phase encoding is used) was fitted to the data points in order to determine $R_1$ and $M_0$ maps. Then c(t) during the bolus passage is found from [1]. Scan time was 10 minutes for $R_1$ measurement and dynamic imaging. Deconvolution with Tikhonov's approach is used to determine the residue impulse response function and the CBF. 1 patient with a minor stroke was imaged.

**Results**

The larger vasculature was easily identified during the bolus passage. In Fig.1A, AIF's for a number of voxels in and in the vicinity of the left internal carotid artery (ICA, insert) are shown. The maximum peak tracer concentration during the bolus passage belongs to the centre voxel (green). All concentrations decrease as the edge of the vessel is approached, reflecting an increasing tissue contribution to the MR signal (although the imaging plane is orthogonal to the ICA, the vessel may not be entirely straight through the 8 mm thick slice). This demonstrates the PVE on the AIF. Note however that the three most central voxels (red, green, dark blue in Fig.1A) show almost the same concentrations, indicating little PVE here. To evaluate perfusion values quantitatively we used the left basal ganglia as a region of interest. Using the AIF's from the voxels in Fig.1, calculated CBF values were found to depend strongly on AIF position (red circles in Fig.1C), except close to the center of the ICA, as expected. When moving the AIF towards the edge of the vessel, the AIF is underestimated and hence CBF is overestimated. We normalize the AIF $c_a(t)$ by a concentration-time curve $c_v(t)$ from a posterior vein, which has a large cross-section and is devoid of PVE in the center. The normalization is defined by requiring that $K\int c_a(t)dt = \int c_v(t)dt$ . Here where the integral is over the measurement time, and K is a fitting parameter. Hence we require that the summed arterial and venous concentrations are equal, which is obvious due to conservation of tracer in case of an intact blood-brain barrier. Correcting the AIF for PVE by a multiplicative factor K can be shown to be correct when the tissue tracer concentration is negligible compared to tracer concentrations in blood. An example of the normalization is shown in Fig.1B. The venous normalization of the AIF's restores the CBF values to within 20% of the value obtained using the unnormalized center AIF, except when using the two AIF's clearly located outside the vessel (Fig. 1C, black circles). The CBF calculated using the AIF at the center of the ICA only changes little upon venous normalization (from 40.4 to 34.5 ml/100g/min). We also placed an AIF in the center of the anterior cerebral artery (ACA, data not shown). The ACA has a smaller diameter than the ICA, hence the PVE is more severe and the CBF is grossly overestimated (Fig.1C, green circle). However, venous normalization restores the CBF value to within the physiologic range (Fig.1C, blue circle).

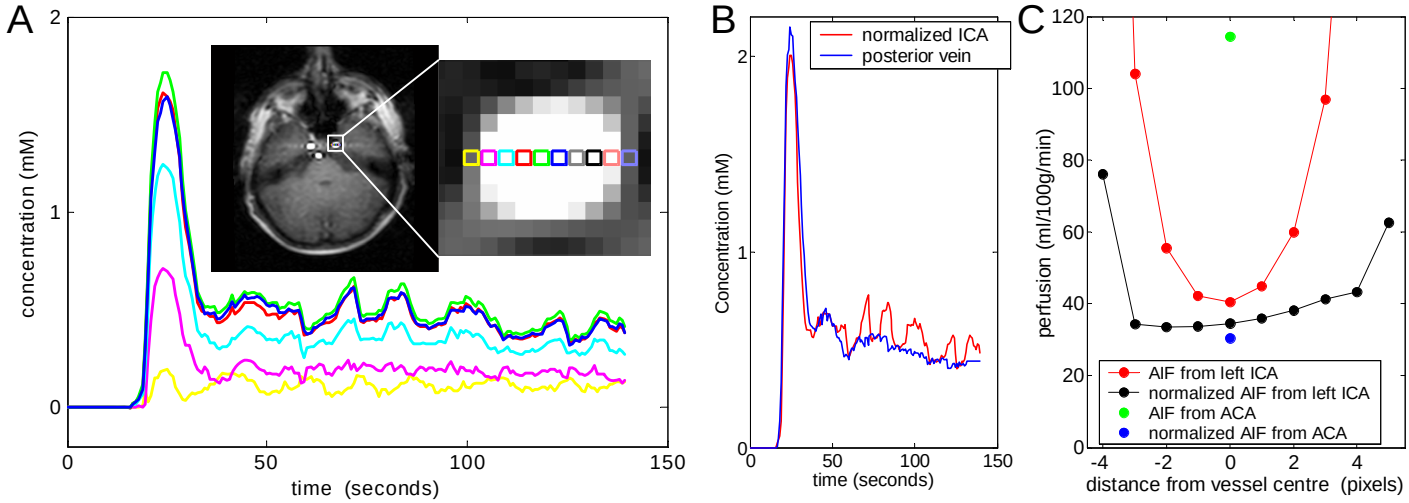


*Figure 1. A. Arterial input functions, line colors correspond to color markings on insert. Insert: Dynamic image, lower slice, at the time of the bolus passage. Zoom on left ICA with AIF positions marked. B. In red, arterial input function from green center pixel in (A), normalized with K=1.17 and time shifted to vein curve (blue). C. CBF values of the left basal ganglia calculated for various AIF's, shown in the legend, as function of displacement from vessel centre.*

**Discussion**

When using T1-weighted DCE-MRI for quantification of brain perfusion, the larger vasculature can easily be identified and a PVE on the AIF can readily be introduced, by displacing the AIF from the vessel centre. We have shown that the PVE can be compensated using venous normalization. This might in particular be utilized to correct local arterial input functions, which will be very sensitive to the PVE. We finally note that for the present data set, the AIF and the CBF quantification is only little affected by PVE if the AIF is sampled in the center of the ICA.

**References**

(1) Chen JJ, et al., JMRI 22:390 (2005); (2) Calamante F, et al., MRM 58:544 (2007)