Physiological component in background flow velocity in MR phase contrast measurements

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Background: MR phase contrast (PC) mapping is commonly used to measure flow velocity and the bulk flow of fluids in the body. Inconsistencies in the gradient waveforms and higher order Maxwell terms, however, can produce offsets in measured velocity values [1]. Objective calculation of these terms and validation is difficult and a pragmatic approach is usually taken whereby velocities from an area of tissue apparently devoid of larger vessels are averaged and used as a correction term. This approach requires that in such a large region, the velocity components arising from unresolved small arteries and veins will cancel each other - an assumption that we tested in this study.

Simulation: We used a recently published model in which the cerebral vascular tree was divided into 19 vessel categories by calibre and type [2] to provide estimated volumes and velocities in vessels with diameters smaller than 0.3 mm in normocapnia and during moderate hypercapnia (PaCO$_2$=40 and 50mmHg, respectively). The size threshold corresponds roughly to the vessel sizes needed to perfuse about 1 ml of tissue (i.e. 2cm^2 reference region with 5mm a slice thickness) and involved 6 arterial and 5 venous compartments from the original model. These vessels are below the typical pixel size of MR phase contrast, ensuring that they would not be seen individually. We modelled the resultant velocity estimates by MR phase contrast assuming, for simplicity, that the arterial vessels were directed through the slice. The venous outflow is directed at a variable angle α that was changed from 0° (through flow) to 180° (anti-parallel flow, see inserts in Fig. 1A) to scale its impact by cos(α). The capillary compartment consisting of vessels less than 10μm was assumed to be randomly orientated and was excluded from calculations. The effective magnetisation of blood is the total of 11 vectors with amplitudes equal to partial volumes of each compartment and phases representing their mean velocity relative to the velocity encoding (venc) combined with a large partial volume of static tissue (PVtissue=95%). The effective velocity measured across a background voxel can be expressed as the phase of this total scaled by venc:

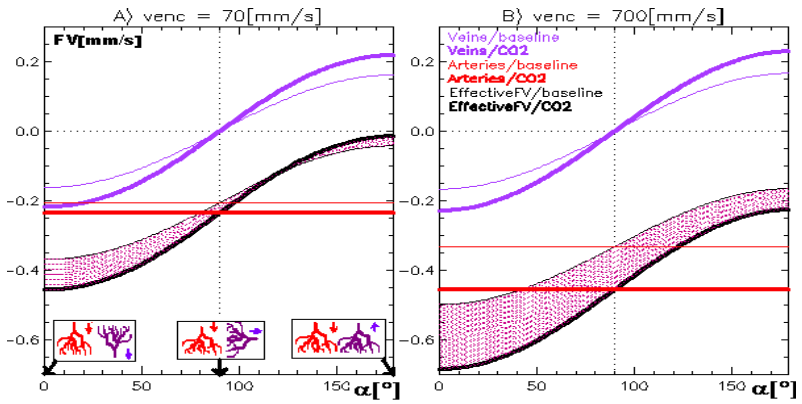

Fig. 1. Simulated effective background flow velocities (black lines, Eq. 1) for velocity encodings A) venc=70, B) venc=700mm/s. Red and mauve lines represent separated contributions of small arteries and veins in the presence of static tissue. X axis corresponds to the angle between the direction of arterial and venous flow with selected conditions shown as inserts in A). Shaded areas indicate the effect of moderate hypercapnia on the effective bFV.

$$bFV = \frac{Venc}{\pi} arctg\left(PV_{tissue} + \sum_{i\in\{arteries\}} PV_i e^{\frac{j\pi FV_i}{Venc}} + \sum_{i\in\{veins\}} PV_i e^{\frac{j\pi FV_i \cos\alpha}{Venc}} \right) \quad \text{Eq. 1}$$

Experimental: 12 normal control subjects (10 male, 2 female; aged, 32±10 years) underwent MR velocity mapping to measure 1) CSF flow through the cerebral aqueduct (venc=60-110mm/s); 2) Foramen Magnum (FM, venc=70mm/s); and 3) blood flow through the 6 large cranial vessels at the level of 1st vertebra (CBF, venc=700mm/s) in a 1.5T Scanner (Signa Excite, GE Medical Systems, WI, USA). The acquisitions were retrospectively triggered using a finger pulse oximeter, and had repetition and echo times in the ranges TR=13.3–16.4ms, and 6.3–7.4ms, respectively (specific value depending on venc) and a flip angle α=15°. Velocity maps were reconstructed for 32 time points in a single heart cycle at the resolution of 0.55x0.55x6mm. Measurements were repeated 3 times under normocapnia (baseline) and once under administration of a 5% CO$_2$ mixture in air (hypercapnia equivalent to simulation). The PC signal magnitude images were segmented to exclude low signal regions such as bone and air cavities and phase maps were then phase unwrapped [3]. The background mean flow velocity (bFV) was determined for each image from a large manually placed ROI located in the midbrain (in the case of the Aqueduct) or in the spinal cord (FM&CBF) that contained no visible vessels or CSF spaces.

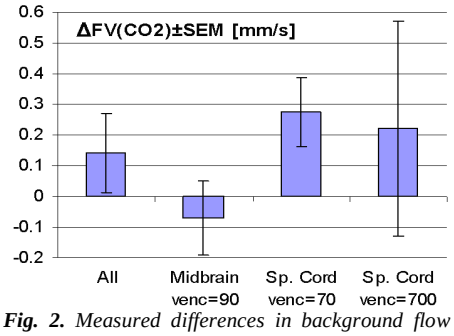

Fig. 2. Measured differences in background flow velocity between normocapnia and hypercapnia condition across all data sets and separated for specific structures and velocity encoding.

Results: 135 data sets were analysed with the remaining 9 lost due to timing or reconstruction errors. On average the bFV was 3.37±0.12[mm/s] at the baseline and 3.50±0.12[mm/s] in hypercapnia. While the difference was not significant on average (P=0.27), in regions drawn across the spinal cord bFV showed a CO$_2$ response of a similar order of magnitude as predicted by the simulation (Fig. 1) in the flow-through case. This difference reached statistical significance for venc=70mm/s (P=0.03, Fig. 2).

Discussion&Conclusions: The simulations shown in Fig. 1 demonstrate that perfect velocity cancellation of the microvascular compartments should be treated as an exceptional coincidence of factors. The worst case can be seen to arise when arterial and venous flows are in the same direction, even though the large partial volume of the static tissue serves to minimize their impact on the effective bFV. Phase cancellation, first of relatively fast flow in small arteries, and then of successively smaller vessels is a consideration at low vencs. The precise value of the background flow velocity, however, will also depend on numerous parameters and mechanisms that are not included in our simple model, such as inflow effects or operator bias in ROI positioning that may skew arterio-venous proportion due to vessel visibility. Although physiological factors seem to be an order of magnitude smaller than the average value of bFV observed experimentally, they may further contaminate corrections of measurements in slow flowing compartments, such as CSF.

References: **[1]** Bernstein. Magn Reson Med 1994. 32:330-4. **[2]** Piechnik. Neuroimage 2007. DOI 10.1016/j.neuroimage.2007.08.022: **[3]** Jenkinson. Magn Reson Med 2003. 49:193-7.