# Sensitivity of the vascular occupancy (VASO) method estimated using a multicompartmental blood-tissue model

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**INTRODUCTION**:

The VASO method, based on inversion recovery principles, was proposed recently[1,2] and is sensitive to the changes in blood volume through complementary signal changes from the remaining tissues. In this paper we investigate the complexity of the signal due to inhomogeneous tissue and blood composition.

**METHODS:**

Simulations of VASO reactivity to $CO_2$ challenges were performed using a model of tissue voxels consisting of CSF, grey (GM), white matter (WM), and blood (CBV) partial volumes, and assuming MRI parameters appropriate to 3 Tesla. The blood compartment is further subdivided into 11 sub-compartments representing an arbitrary division into **A**rteries, **a**rterioles, **c**a**p**illaries, **v**enules and **V**eins, (**S**mall, **M**edium or **L**arge; see labels Fig. 1). The vascular model based on a scaled vascular bed of a dog[3] is presented in a separate abstract. The model is used to analyse the relationship between changes in cerebral blood flow and volume based on direct experimental measurement of vascular reactivity[4,5]. For the purpose of this study the vascular model simulates additionally the $O_2$ blood saturation[6], haematocrit[7] and the resulting compartmental T1 and T2* values[8]. The relaxation parameters for CSF, GM and WM are assumed constant[9] in calculation of the VASO signal[1]and its changes due to $CO_2$-induced effects on CBV and corresponding compression/expansion of the tissue. We simulate the parametric effects of the signal sensitivity to relative CBV change $(dVASO/VASO_0)/(dCBV_0/CBV_0)$ and demonstrate a focally varying map of predicted sensitivity obtained using a pre-segmented brain.

**RESULTS**:

Figures 1a-e show the distribution of haemodynamic parameters and T1, T2* relaxation constants in vascular compartments as a function of $PCO_2$.

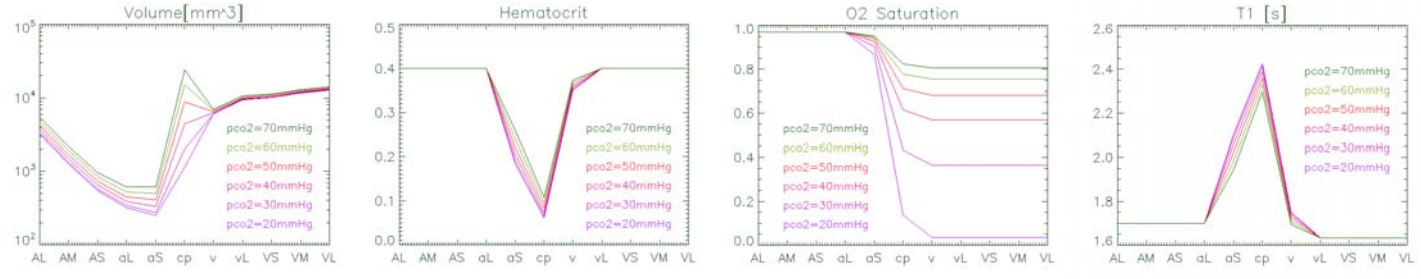


***Fig.** 1a) 1b) 1c) 1d) 1e)*

Fig. 2a below, illustrates the dependence between the relative VASO change $(dVASO/VASO_0)$ and $(dCBV/CBV_0)$. It shows that the relation is not linear in general and that the constant of proportionality depends strongly on voxel composition (defined in the legend), as opposed to the constant theoretical relationship when relaxation effects are ignored (dashed line). Fig. 2b and 2c demonstrate the parametric dependence of VASO sensitivity on the ratio of CSF to parenchyma (Fig. 2b) and the relative content of blood volume (Fig. 2c). A particular difficulty occurs at the border between CSF and parenchyma where the absolute VASO signal is close to zero. Fig. 2d shows the simulated combined effects on a theoretical segmented image (grey matter boundary shown by thin line) characterised by a large regional variability in VASO sensitivity (resolution: 4*4*4mm$^3$, calculated as average of 1mm$^3$ voxels in segmented image).

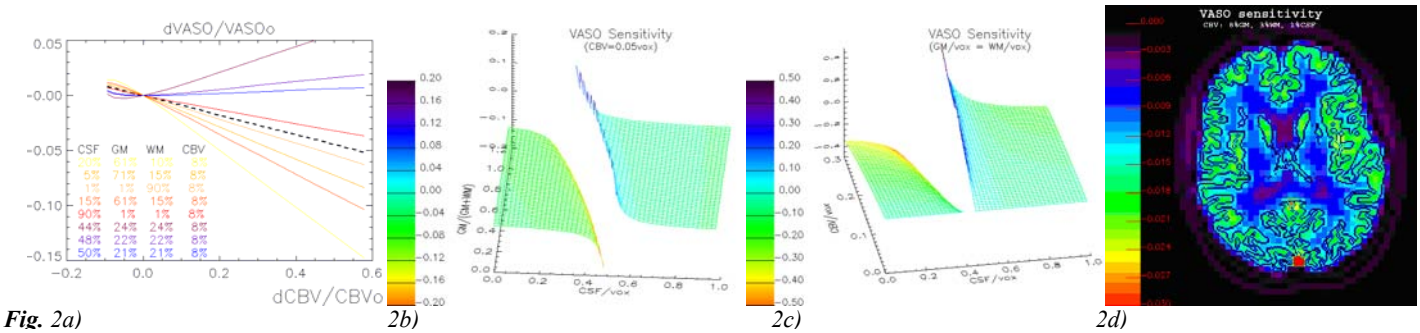


***Fig.** 2a) 2b) 2c) 2d)*

**CONCLUSION:**

VASO reflects changes in CBV that are weighted by the resting CBV and tissue composition. It is also affected by the distribution between macro and micro-vessels, and is particularly noisy at the boundary between CSF and brain tissue. These preliminary data suggest that VASO imaging should not be treated as a direct measure of relative CBV change.

**REFERENCES:** **[1]** Lu H, Golay X,. *Magn Reson Med* 2003, 50:263-274. **[2]** Lu H. *Magn Reson Med* 2004, 51:9-15. **[3]** Milnor WR: *Hemodynamics*. Baltimore: Williams & Wilkins; 1982. **[4]** Tuor UI. *Am J Physiol* 1984, 247:H40-51. **[5]** Wei EP. *Am J Physiol* 1980, 238:697-703. **[6]** Sharan M, Jones. *Ann Biomed Eng* 1989, 17:13-38. **[7]** Goldsmith HL. *Am J Physiol* 1989, 257:H1005-1015. **[8]** Silvennoinen MJ. *Magn Reson Med* 2003, 49:47-60. **[9]** Clare S. *Magn Reson Med* 2001, 45:630-634.