Vascular space occupancy weighted imaging with control of inflow effect and higher signal-to-noise ratio

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Introduction

It has been recently proposed that local cerebral blood volume change (ΔCBV) during brain activation can be measured by a series of images whose contrast is dependent on vascular space occupancy (VASO) (1). VASO utilizes the inversion recovery (IR) sequence to acquire images when the longitudinal magnetization (M_z) of blood is relaxing through zero. While blood is eliminated, M_z of other tissues is also small, which makes low SNR an inherent disadvantage of VASO. Here we propose an alternative VASO-weighted imaging without blood suppression. By optimizing the M_z difference between blood and non-blood tissues, higher SNR can be achieved and ΔCBV is calculated with the T_1 of parenchyma measured or assumed. In addition, the signal is less sensitive to inversion efficiency (α). In the following, the original and presented methods are referred to as method 1 and 2, respectively.

Materials and Methods

1. Computer Simulation. The voxel is divided into blood and non-blood compartments, labeled as $V_{b,off}$, $V_{nb,off}$ and $V_{b,on}$, $V_{nb,on}$ for "activation-off" and "activation-on" states, respectively. $V_{nb,off} + V_{b,off} = 1$, $V_{nb,on} + V_{b,on} = 1$. When CBV increases with neuronal activation, the composition of non-blood tissue is assumed unchanged. Since the VASO signal change (ΔVASO) is negative with positive ΔCBV, for the convenience of comparison, ΔVASO is expressed as: $1 - (V_{nb,on} \cdot Q_{nb} + V_{b,on} \cdot Q_b)/(V_{nb,off} \cdot Q_{nb} + V_{b,off} \cdot Q_b)$…[1], where $Q_{nb} = M_p(TI) \cdot \xi$, $Q_b = M_b(TI) \cdot \xi$, $M_i(TI)$ is the normalized M_z at image acquisition, ξ is the water density normalized by CSF ($i = b, p$ for blood and parenchyma, respectively). The formulation assumes that TE is very short as compared with the T_2^* of blood and non-blood components. Parameters employed in the simulation are as follows, $V_{b,off} = 4\%$-8%, ΔCBV = 25%-50%, $\xi = 0.75$, $\xi = 0.82$, $T_{1b} = 1627$ms (2), $T_{1p} = 1300$ms (3), $\alpha = 1$.

2. MR Experiments. Experiments were performed on healthy volunteers (n = 7, 24-37 years). An 8-Hz black-white radial checkerboard was used for visual stimulation (30s baseline, 4 cycles of 24s on and 36s off). Extra 10s dummy scan preceded each experiment to allow the signal to reach a steady state. Imaging parameters included: TR = 2000ms, flip angle = 90^0, FOV = 22cm, in-plane matrix size = 64x64, single 5mm slice (oblique, encompassing the calcarine fissure), single-shot gradient echo with a spiral readout (3 Tesla GE EXCITE system). *Exp1: If blood signal is completely suppressed by IR sequence, the superposition of diffusion weighting should yield no difference to ΔVASO.* In diffusion-weighted VASO scans, diffusion gradient was applied along anterior-posterior direction with a b-value of 2.02 s/mm^2. Identical TE (9.3ms) was used for both diffusion-weighted and non-diffusion-weighted scans. Three TI values were selected from {700,705,710,715} ms and applied in a random order. *Exp2: Comparison of method 1 and 2.* A series of IR scans were performed to measure T_1 and α distribution. TI = {30,80,150,300,600,1000,1500, 2000,3000,5000}ms, TR = TI + 10s, TE = 2.7ms. For functional studies, identical slice prescription was used. Five VASO scans were performed: TE = 2.7 ms, TI = 1469ms with a global saturation after each imaging acquisition (PostSat) = {on, off}, TI = 1449ms with PostSat and TI = {690, 710}ms without PostSat. The TI value of 1469 was calculated by maximizing M_z difference between blood (T_1 = 1627ms) and gray matter (T_1 = 1300ms). In method 1, TI = 710ms and 690ms were used to suppress blood for $\alpha = 1$ and 0.95, respectively. In method 2, M_z relaxed to the expected value calculated by ideal α 10ms earlier when α was 0.95. For comparison, a 20ms shorter TI (1469-1449=20, 710-690=20) was chosen. Correlation analysis was performed after baseline correction (c.c. = 0.25, $p < 0.05$). ΔCBV was then calculated by Eq. [1].

Results and Discussion

Exp1: Smaller ΔVASO is consistently observed with the application of diffusion weighting (ΔVASO - ΔVASO$_{dif}$ ~ 0.7%, Fig1). Diffusion-related signal attenuation in CSF and parenchyma was calculated and found to be ten-fold smaller than the experimental data, and with opposite sign. These findings indicate the existence of intravascular signal. In method 1, blood suppression can be imperfect due to T1 dispersion and variations in inversion efficiency Exp2: Compatible activation maps are obtained with both methods (TI = 690ms vs. TI = 1469ms, PostSat; Fig2). With the voxel size of 3.4×3.4×5.0 mm^3, ΔVASO is 2.2% in method 1 and 0.6% in method 2 (Fig3), which approximately corresponds to ΔCBV of 36% and 44%, respectively(assuming that $V_{b,off} = 6\%$). α is homogeneous throughout the slice and is 0.95±0.08 at visual cortex. In method 1, a 20ms shorter TI accounts for 0.05 variation in α which affects ΔVASO by 0.5%/2.2%. In method 2, a 20ms shorter TI corresponds to larger variation in α (~0.1) but ΔVASO changes no more than 0.04%/0.6%. The absolute ΔVASO is approximate1y 1.7-fold higher in method 2. These findings are in agreement with computer simulation. Because method 2 retains blood signal for imaging, inflow effect can be up to 0.2%/0.6% and PostSat is necessary for the purpose of resetting blood signal.

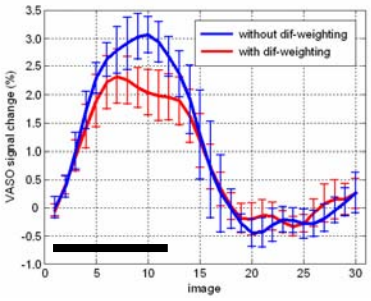

Fig 1

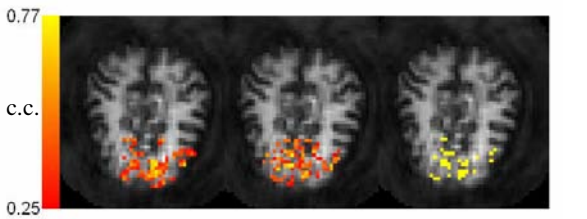

Fig 2. Activation maps obtained by method 1 (left) and 2 (middle). The rightmost image shows the pixels detected by both methods.

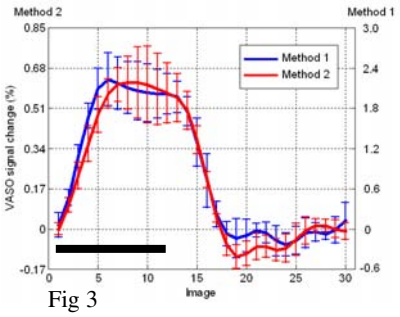

Fig 3

References

1. Lu et al, Magn Reson Med 2003;50:263.
2. Lu et al, Magn Reson Med 2004;52:679.
3. Wansapura et al, J Magn Reson Imaging 1999;9:531.