Vascular and Parenchymal Changes in Gradient Echo BOLD Signal during Global Flow Increase as a Function of Magnetic Field Strength

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Introduction

One of the main motivations for performing BOLD-based fMRI experiments at higher field strengths is the reduction of vascular response and improvement of image sensitivity. Activation-induced BOLD signal changes are generally a small fraction of the baseline MRI signal. However these changes are known to increase at higher field strengths, due to the general enhancement of susceptibility-related effects. Although temporal SNR plateaus due to limitations caused by inherent physiological fluctuations in the fMRI data [1], improvements on the effect size of the BOLD contrast are also likely when the images are acquired at higher field strengths. The aim of this study was to investigate the vascular BOLD artifact at higher field strengths and to further explore the echo time dependence of the BOLD response in gray matter and veins. To assess spatial specificity of BOLD methods previous studies have used focal activation. The problem with such an approach is that there is no independent means of identifying the 'true' region of parenchymal response. In this study we avoid this difficulty by using a global challenge and simply measuring response amplitudes in parenchymal and vascular regions of the images. Furthermore, when field-strength comparison studies are employed, it is necessary to ensure that the physiological response was in fact identical on the different imaging systems used. To control for differences in the underlying flow response during experiments at different field strengths, we collected ASL-based flow measurements simultaneously with the BOLD observations and used these to normalize the latter.

Methods

MR Imaging was performed on four healthy human subjects, at two different field strengths on a Siemens Sonata 1.5T and a Siemens Trio 3T system using an 8-channel phased array receive head coil and a whole-body transmit coil for excitation and arterial spin labeling. BOLD measurements were achieved using a multi-echo GE EPI sequence. In order to achieve increases of CBF in cortical gray matter, we used hypercapnia, which is believed to produce little or no change in the rate of metabolic oxygen consumption (CMRO₂). Scanning runs lasted ten minutes, including two intervals of two minute duration each, during which the gas breathed by subjects was switched from atmospheric composition medical air to a mixture of 7% CO2 with balance air (a block design with 2 min off, 2 min on). The imaging parameters were TR=3000ms, ten 3mm thick slices, inter-slice gap=1.5mm, 200 frames, FOV=192x192mm², matrix=64x64, 9 echo times; TE=11, 23, 35, 47, 59, 71, 83, 95, 107 and TE=8, 21, 35, 48, 61, 75, 88, 101, 115 for 1.5T and 3T respectively. To achieve short echo times, the images were acquired and reconstructed using GRAPPA (acceleration factor 2). To measure the relative CBF changes, a pulsed ASL perfusion sequence [2] was used with the same sequence parameters as before, except that the echo time was kept constant at 30msec and 20msec for 1.5T and 3T respectively. Perfusion weighted maps were generated by subtracting the inverted from control images. BOLD sensitivity was determined from the multi-echo data sets, by fitting a General Linear Model (GLM) plus correlated noise after removing motion and linear trends at each echo. The BOLD signal intensity, its effect size and the mean CBF values were then estimated at ROIs in cortical gray matter. For comparison, the BOLD contrast and the signal intensity were also measured on major veins. **Results**

Figures (A) and (B) illustrate the dependence of the BOLD signal changes on echo time at 1.5T and 3T respectively. The t-statistics maps shown generated from the GLM analysis. Measurements showed that the % signal changes increases linearly with echo time, while the absolute signal decreases as an approximately exponential function of the echo time. The absolute signal changes reach the maximum at TE near the T2* of the parenchymal within each field strength (60ms for 1.5T and 35ms for 3T), the veins however attain the peak value at lower TE (40ms for 1.5T and 20ms for 3T). Results in (C) and (D) demonstrate the % activation induced BOLD signal change across echo times normalized to % change of the CBF signal at both 1.5T and 3T, for gray matter and veins respectively. Measurements were estimated from regions of cortical gray matter and veins. In parenchymal percent signal changes at 3T were a linear increase of the % signal change with echo time within each field strength and across them. In the veins percent signal changes at 3T were lower than the 1.5T however they remain higher than the parenchymal tissue on both field strengths.

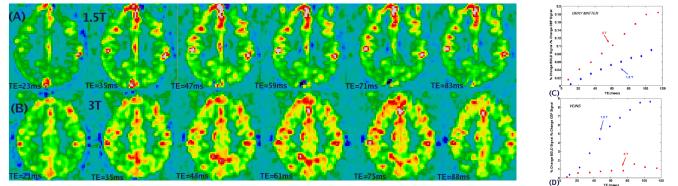


Figure 1. (A), (B) illustrate the contrast fluctuation across echo and across field strengths (top row 1.5T, bottom row 3T). Percentage change of BOLD at 1.5T (diamonds) and 1.5T (squares) normalized to the % of cerebral blood flow for the same ROI of gray matter (C) and veins (D).

Conclusion

In this work we examined venous and parenchymal BOLD responses to a global challenge at field strengths of 1.5 and 3Tesla. We used hypercapnia to modulate BOLD signal changes and a multi echo sequence to compare the signal changes across echo times. Peak parenchymal responses were observed at echo times of \sim 60 and \sim 35 ms at 1.5 and 3T respectively. The maximal venous responses were seen at shorter echo times. Functional experiments should therefore be carried out using gradient echo TE values at or above those at which peak parenchymal response was seen to avoid emphasis of venous response components, which can occur even at high field strength.

References 1) Triantafyllou C., et al, ISMRM,p1701,2004, 2) Wong E.C., et al, MRM,39:702-708,1998.