

Positive and negative BOLD-signals from blood vessels in monkey visual cortex

J. Goense¹, and N. Logothetis^{1,2}

¹Max-Planck Institute for Biological Cybernetics, Tuebingen, Germany, ²Imaging science and biomedical engineering, University of Manchester, Manchester, United Kingdom

Introduction

High-resolution fMRI can aid in determining whether and to what extent the BOLD signal arises from capillaries or larger vessels. The SE-BOLD signal is more sensitive to capillaries [1-3] than the GE-BOLD signal and hence better represents actual neural events. In our high-resolution functional activation maps we observe positive and negative BOLD signals associated with vessels; this was seen for both GE- and SE-BOLD, because SE-BOLD is still sensitive to vessels, especially when long readout times are used [4]. We changed the acquisition parameters and gradient direction to examine the origin of these vessel- and capillary BOLD signals.

Methods

Experiments were performed in 6 healthy monkeys (*Macaca mulatta*) using a vertical 4.7T scanner (Bruker BioSpec 4.7T/40v). The experimental setup and anesthesia have been described elsewhere [5,6]. High-resolution SE-fMRI was performed while monkeys were viewing full-field black and white rotating checkerboard stimuli. A 16-segment SE-EPI was used for fMRI, with FOV ranging from 6.4x4.8 to 4.8x4.2 cm², and spatial resolution of 333x333 to 250x175 μm², slices were 2 mm thick and oriented perpendicular to the cortical surface of V1. TE was 46-48 ms and TR 2000 ms. For anatomical reference a 16-segment SE-EPI was used with resolution 140x160 μm², TE = 70 ms, TR = 3000 ms. Data analysis was performed in MatLab (the Mathworks), while SPM was used for image registration to correct for scanner drift.

Results

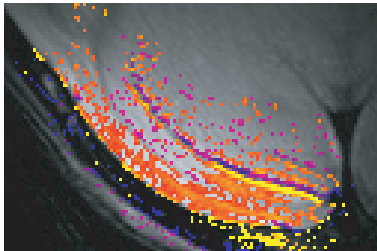


Figure 1

Figure 1 shows a high-resolution (333x333 μm²) SE-BOLD image of striate cortex, showing a strong positive signal flanked by negative signal, from a large vessel in the calcarine sulcus, as well as activation in the gray matter that peaks in layer IV. The location of layer IV is determined based on the Gennari-line in anatomical images. The relatively long acquisition windows (25-30 ms) necessary for high resolution SE-fMRI increases the sensitivity to vessels. Positive and negative signal near vessels are typically present at high resolution. Figure 2b,d shows functional activation maps obtained at a spatial resolution of 250x175 μm², and fig. 2a,c shows the mean profile through the cortex for the functional map and raw image intensity. The profile in fig. 2a shows peak activation in layer IV indicated by the Gennari-line, and vessel signal outside the CSF, suggesting the vessel signal arises from outside the CSF compartment, i.e. the dura, while the negative signal is located to the inside of the CSF signal, at the level of the pia.

However, when the phase-encoding direction was reversed (P-A instead of A-P; Fig. 2c,d), the peak activation in gray matter was still located in layer IV, but the largest vessel signal now arises from the location of the pia. Similarly, when the read and phase-encoding direction were swapped, this did not affect the gray-matter signal in layer IV, but it did change the location of the vessel signals. Negative vessel-related BOLD was observed in both high-resolution GE-EPI and SE-EPI.

Conclusion

In high-resolution fMRI both positive and negative functional signals are observed associated with vessels. While the functional signal in the gray matter is robust to reversal or change of the gradient direction as well as to the choice of acquisition parameters (the maximum always occurred at the same location in layer IV), the vessel signals change location. The origin is possibly due to chemical shift, and such effects will need to be taken into account in the interpretation of high-resolution fMRI results.

Acknowledgements

We wish to thank Mark Augath for technical support, and Kathrin Guhr, Mirko Lindig and Deniz Ipek for preparing and maintaining animal anesthesia. Supported by the Max-Planck Society.

References

- [1] Weisskoff et al. MRM 31:601-610 (1994);
- [2] Boxerman et al. MRM 34:555-566 (1995);
- [3] Kennan et al. MRM 31:9-21 (1994);
- [4] Goense & Logothetis MRI 24:381-392 (2006)
- [5] Logothetis et al. Nat Neurosci 2:555-562 (1999);
- [6] Logothetis et al. Neuron 35:227-242 (2002)

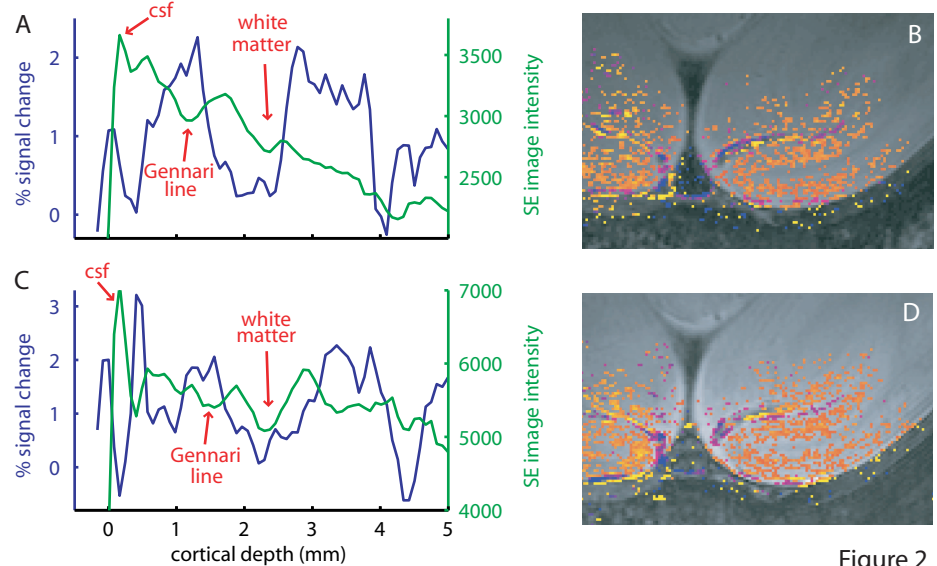


Figure 2