Towards a vascular model of layer specific activation

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Introduction

The blood-oxygen-level dependent (BOLD) signal in a region is tightly coupled with the underlying vasculature. Understanding the contribution of the different vascular compartments could be key for comparing results from different regions and obtaining a quantitative basis for the interpretation of functional MRI (fMRI) experiments [1]. Further, as the characteristics of the brain vasculature vary across the cortical depth, understanding these is necessary for examining the cortical depth dependent point spread function (PSF), which will determine the spatial resolution achievable by high resolution fMRI sequences for identifying layer specific functional activation.

In this work we developed a simplified model of the cortical vasculature based on reported experimental data. This model is then used for simulating BOLD activation for Gradient Echo (GE) and Spin Echo (SE) sequences at different cortical depths at 7T.

Methods

In this simplified model of the vascular architecture, the cortex is divided into 5 vascular layers, as many as groups of intracortical veins (ICV) reported in [2]. Each group of ICVs is considered to originate in one vascular layer, but a layer is drained by all the ICVs that pass through or originate in it. The ICV with the largest diameter reach the deepest cortical layer, while the group with the narrowest lumen drains only the most superficial layer. The density and diameter of the ICV increase with decreasing cortical depth [3] and the diameter for each group of ICV at the surface of the cortex is 120, 74.3, 54.3, 34.6 and 18.8 μm, which are close to the surface diameters reported for the visual cortex in [2]. The microvascular densities in each layer are extrapolated from experimental data obtained in the visual cortex [4] and pial veins are not taken into account in this simplified model. The conservation of mass principle is used for characterizing the flow and diameters of the ICV whereas the microvascular vessel diameters and blood flow are taken from [5].
The static spatial response of the contrast mechanisms that contribute to the BOLD signal change are simulated in each layer largely based on [6] for a field strength of 7T. The TR was considered to be sufficiently long so the signal did not contain any T1 weighting and the echo time was set to be the T2 (for SE) or T2* (for GE) time of gray matter at 7T to maximize BOLD signal changes. The model was implemented in Matlab.

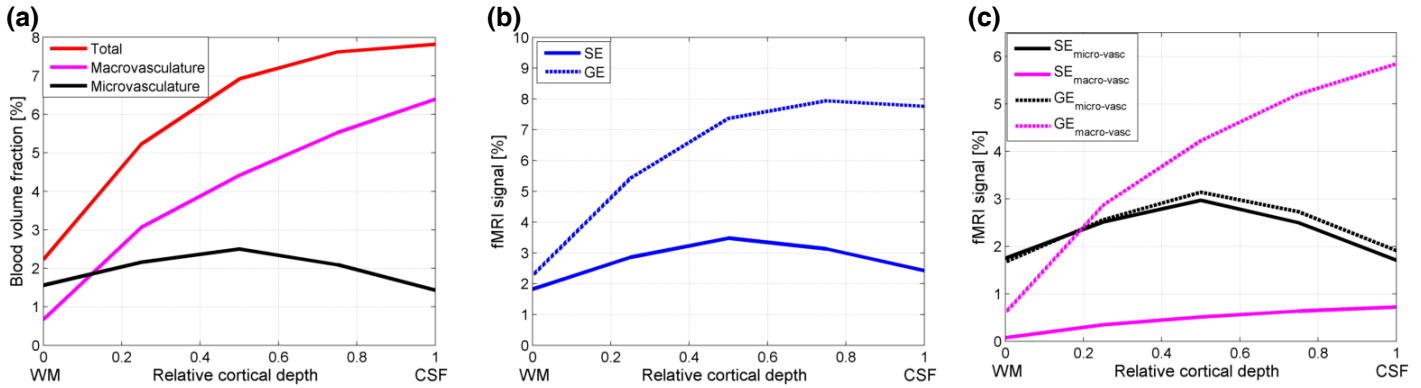

Figure 1: **(a)** Total (red), micro (black)- and macrovascular (pink) volume fractions across the cortical depth obtained in our vascular model of the cortex. **(b)** Spin Echo (line) and Gradient Echo (dotted line) fMRI signal. **(c)** The micro (black) and macrovascular (pink) compartments' contribution to fMRI signal for Spin Echo (line) and Gradient Echo (dotted line).

Results

Figure 1 (a) shows the blood volume fraction across the cortex obtained in our model. The microvascular density peaks in the middle of the cortex, whereas the macrovasctular density increases steadily from lower cortical layers to the surface, as expected. The total fMRI signal across the cortical length for SE and GE is seen in Figure1 (b) whereas Figure1 (c) shows the contribution of the micro- and macrovasculature to the fMRI signal for each type of sequence. For GE, the macrovascular contribution dominates the BOLD signal if the macrovascular blood volume is bigger than the microvascular volume, which in our model is the case for depths above 12% of the relative cortical depth. For SE, the microvascular contribution dominates across the whole cortical thickness. The total SE fMRI signal peaks where the microvascular density is highest.

Discussion

Despite its simplicity, the proposed model reproduces facts observed in experimental measurements. For instance, the BOLD signal is strongly driven by microvasculature and peaks at a depth in which the microvascular density is also highest when SE sequences are used at higher fields, whereas in GE measurements macrovasculature dominates the fMRI signal. Future developments will be aimed at accurately determining the layer specific PSF by taking into account the temporal dynamics of the BOLD signal upon activation and refining simplifications made when determining the vascular architecture.

References

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