Vascular signal contributions in MR-encephalography: direct observation of intra- and extravascular ECG-pulsatility

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MR-Encephalography (MREG) (1) is an extremely fast technique to monitor physiological changes by use of simultaneous readout with multiple small RF-coils. A sampling rate far below breathing and ECG frequencies allows for a very accurate determination of the BOLD response to functional stimulation. Aim of this work was to separate blood flow related contributions of the BOLD signal from those of tissue and to investigate the influence of blood flow on the physiological noise caused by ECG pulsatility.

Materials and methods:

All experiments were performed on a 3T scanner (Trio, Siemens). Measurements were performed with a gradient spoiled FLASH-sequence (TR/TE 50/15ms). The sequence was combined with a dual band, black blood saturation pulse and a bipolar gradient pair (one lobe: 20mT/m*4ms) in read direction. MREG measurements were performed with one dimensional spatial encoding in AP direction. The stimulation paradigm was a checkerboard task frequently used for visual stimulation: A total measurement time of 120s consisted of 3 rest periods (black screen) and 3 activation periods (flickering checkerboard) each 20s long. Areas of activation in the visual cortex were determined by running a conventional EPI-BOLD experiment in combination with online evaluation. Four functional MREG runs were performed on a slice through the activated visual cortex: with inflow saturation, with applied bipolar gradients, with bipolar gradients and inflow saturation and a sequence without modifications.

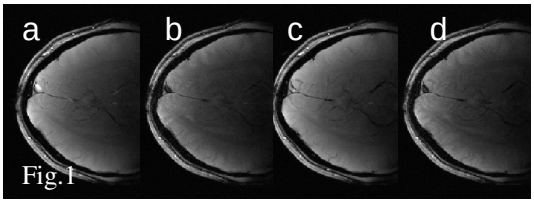

Fig.1

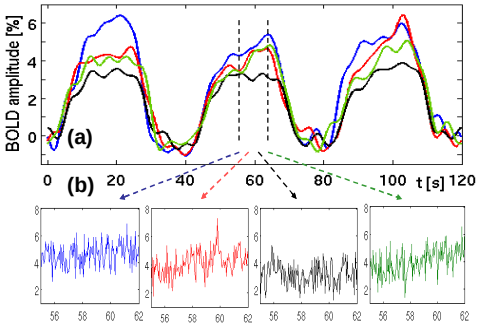

Fig.2 single voxel time course: without flow suppression (—), with inflow saturation(—), with bipolar gradients(—), and with both (—)

Data analysis:

For 1-dim-MREG the spatially encoded signals from the coils were treated as independent measurement channels.

Each voxel time series was then analyzed by applying a general linear model fit using the SPM5 canonical haemodynamic response function, convolved with the boxcar stimulus function, as a predictor(2). The absolute amplitude of the bold response corresponds to the beta return value of the glm-fit. Only active voxels ($p < 0.001$) showing an effect size larger than 0.5% relative to the signal intensity were further processed. The frequency spectrum of each voxel is given by the fourier transform of the time series. The ECG activity was measured by the area under the peak at the individual ECG frequency.

Results:

Fig.1 shows a slice through the visual cortex acquired with the FLASH sequence (a), with inflow saturation (b), with bipolar gradients (c) and with a combination of both (d).

The lowpass filtered single voxel time courses in Fig.2a shows the reduction of the BOLD amplitude if signal from flowing spins is suppressed. Fig.3 displays the distribution of the BOLD amplitude decrease for all activated voxels relative to the unmodified sequence with mean values in Tab.1. Fast signal fluctuations with ECG components up to 1% can be observed (Fig.2b) in the unfiltered data for all time courses. Fig.4a shows the distributions of the ECG activity in functional active voxels. Functional inactive voxels (Fig.4b) have a similar ECG activity distribution.

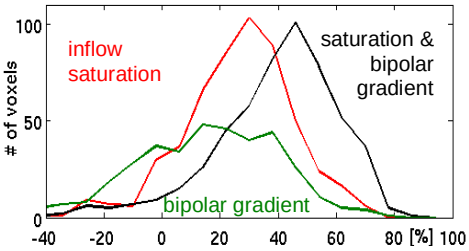

Fig.3 Average decrease in BOLD amplitude

	No flow suppression	Inflow saturation	Bipolar gradients	Inflow sat. & bipol. gradients
Mean BOLD decrease	----	25±19%	10±30%,	38±22%
Mean ECG in active voxels	0.28±0.11	0.31±0.08	0.34±0.19	0.30±0.09
Mean ECG in inactive voxels	0.26±0.08	0.31±0.11	0.31±0.14	0.29±0.09

Discussion:

The extremely high BOLD-sensitivity of MREG is demonstrated. Suppressing the signal from flowing blood by inflow saturation or by flow dephasing gradients leads to a reduced BOLD amplitude because of the reduced intravascular contribution. Combining both techniques results in a further reduction of the BOLD signal. This demonstrates not unexpectedly that inflow suppression and flow dephasing acts on different vascular compartments and that both of which contribute to the BOLD effect. It is surprising, however, that suppression of flow by either mechanism does not lead to reduced ECG pulsatility. Therefore other mechanisms, like ecg-related movement of the static tissue and a change in relative spin density due to the ecg-dependant variation in vascular volume, seem to dominate the ECG induced signal variations.

Conclusion:

It is demonstrated that the suppression of intravascular signal reduces the average BOLD amplitude by approximately 40%. This is in good accordance with simulations based on the theory about the intravascular contribution to the BOLD effect (3). We further conclude that flow dephasing gradients and inflow suppression act independently and that their effects sum up if they are combined. Furthermore our results show that significant ECG pulsatility persists even when intravascular signal contributions are removed.

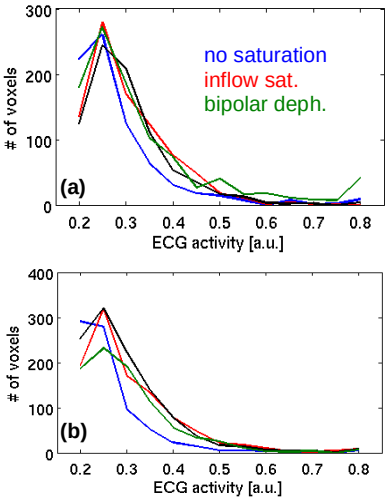

Fig.4 Histogram of ECG pulsatility : (a) active voxel (b) inactive voxel

References: (1) Hennig J, Zhong K, Speck O. Neuroimage 34(1):212-219(2007). (2) available at http://www.fil.ion.ucl.ac.uk/spm/ (3) Kiselev, VG. MagnResonMed 46(6) 1113-1122 (2001)

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