Correlation of fluctuations in simultaneously recorded VEP and BOLD fMRI

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Introduction

Studies comparing visual evoked potentials (VEPs) and BOLD signals have found a positive correlation between the VEP amplitude and the magnitude and extent of the BOLD signal \cite{1, 2}, but some VEP components are better related to the BOLD signal than others. The strongest spatial correlation have been found between the source location of the N1 peak* and the location of the BOLD signal \cite{3}. While previous studies with simultaneously recorded VEP-fMRI have only compared the correlation between the grand mean VEP and the averaged BOLD signal, there is growing interest in correlating simultaneously acquired VEP and BOLD fMRI data. In this case, however, the N1 peak becomes difficult to identify due to excessive noise amplification of the EEG inside the MR scanner. Therefore, we examined alternative ways to measure neuronal activation in simultaneously recorded VEP-fMRI.

Methods

20 healthy subjects (13 female, 7 male, mean age 28, range 20-54) with normal vision participated in the study. The study was approved by the local ethics committee.

We recorded simultaneous EEG and fMRI during visual stimulation.

Visual stimulation

Whole field (30° horizontal, 23° vertical), pattern reversal checkerboards reversing at 2 hertz with a central fixation point (active blocks) and a black screen with a central fixation point (rest blocks), 3 different contrasts in the checkerboards were used for different runs. Active blocks lasted 10.5 seconds and consisted of 21 stimuli. Rest blocks lasted 24.5 seconds. 1 run comprised 11 rest blocks and 10 active blocks.

EEG recording and analysis

32 channel EEG was recorded using Brainproducts BrainAmp MR with a 5000 Hz sampling rate. The amplifier was synchronized with the scanner clock frequency. During the functional MRI the EEG and the respiratory rate were recorded with the scanner vector cardiogram recorder and pneumatic thoracic belt, respectively. 3 trials with different contrasts were recorded simultaneously with the fMRI data. The 3 trials were repeated outside the scanner as a control setup.

Gradient and pulse artifacts were corrected in Brain Vision Analyzer using the average template subtraction techniques described by Allen et al. \cite{4, 5}. Data was then filtered with a low pass filter at 60 Hz and down sampled to 500 Hz. Pulse artifacts were corrected using R-markers from the vector cardiogram. The data were then exported to Matlab and data were then segmented in trials of 500ms. In active blocks the segmentation was based on stimulus onset. In rest blocks the segmentation was in consecutive intervals of 500ms. A grand mean VEP was calculated as an average of all active trials. ICA was performed with selection of components based on correlation with the occipital channels and the component’s corresponding grand mean average. The occipital channel exhibiting the highest average correlation with its corresponding grand mean VEP was selected for further processing. The P1 and N2 peaks on the grand mean VEP were identified. For each trial 3 different measures of neuronal activation were identified: 1. The difference in signal amplitude between P1 and N2. 2. The standard deviation of the VEP signal in each trial from 50-250ms. 3. The correlation of each trial with the grand mean VEP from 50-250ms. Because we had 7 VEP trials for each fMRI volume, every 7 trials were averaged to make a data point in the VEP time course. The VEP time course was then convolved with a hemodynamic response function and saved for further analysis.

MRI recording and analysis

Functional and structural MRI was performed on a Philips Achieva 3.0T whole body MRI scanner with an 8 channel sense headcoil.

Rest blocks lasted 24.5 seconds. 1 run comprised 11 rest blocks and 10 active blocks.

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For each subject the 3 VEP-derived neuronal activation time courses were correlated with the BOLD time course for the corresponding run. This was done for all 3 contrasts and for data obtained both inside and outside the scanner giving rise to 18 correlation coefficients for each subject. A boxcar design matrix convolved with a hemodynamic response function was also correlated with each BOLD time course.

Results and discussion

Using the P1-N2 amplitude or the standard deviation in the VEP signal as a measure of neuronal activation yielded averaged correlation coefficients from 0.27 to 0.40. This increased to 0.61-0.83 when using the correlation of each trial with the grand mean VEP. The best correlation was seen when using a standard boxcar (0.80-0.92) (Figure 1).

VEP data recorded outside the scanner gave higher correlation coefficients compared to VEP data recorded inside the scanner (Figure 1). This was probably due to residual noise in the VEP data. Ideally one would expect a better correlation when using simultaneously recorded data taken up inside the scanner.

Correlation also increased with increasing contrast, probably due to increasing signal to noise ratio in the VEP data.

Conclusion

The correlation of fluctuations in VEP and BOLD signals depend on the method of measuring neuronal activation in the VEP signals. We observed a doubling of the correlation coefficients when going from a more conventional measure based on peak detection to a measure based on correlation with the grand mean VEP. However the correlation is still better when using a standard boxcar and when using VEP data recorded outside the scanner which indicates that the method can be improved further.

References


*Note: The following terminology is used for components of the VEP signal. N1: A negative peak around 75ms also called N75. P1: Positive peak around 100ms also called P100. N2: Negative peak around 145ms also called N145.