Spatiotemporal correlation between alpha modulation and BOLD fluctuation in an eyes-open-eyes-closed task

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Introduction:

Eyes-open and eyes-closed conditions are two widely used brain baselines. Previous studies have found alpha rhythmic modulation in electroencephalogram (EEG) and blood-oxygen-level-dependent (BOLD) fluctuation in functional magnetic resonance imaging (fMRI) when alternating between the two baselines [1, 2]. However, the cross-modal relationship between these modulations is not completely understood. The purpose of the present study is to investigate the spatial and temporal correlations between the neural generators of alpha modulation and BOLD fluctuation using an eyes-open-eyes-closed task. We reconstructed the spatiotemporal cortical activity underlying the task-modulated alpha band scalp EEG, and compared them with the BOLD activation/deactivation contrasting eyes-open versus eyes-closed. We found that the alpha modulation and the BOLD fluctuation were co-localized within the occipital and parietal lobes.

Methods and material:

Subjects were self-paced or audially signaled to alternate between two baseline conditions: (1) eyes-closed and (2) eyes-open in darkness/eyes-open with fixation. Two experiments were performed using simultaneous fMRI (3T, Siemens Trio, Germany) and EEG (64 channels, BrainProducts, Germany) recordings. In addition, two experiments were done using fMRI recordings only and four experiments were done using solely EEG recordings.

In this study, rather than imaging the entire alpha band activity, we aimed to localize the neural origins responsible only for the task-related alpha rhythmic modulation. To achieve this, we (1) employed spatio-temporal separation of independent component analysis [3] to separate the EEG into maximally temporal independent components (ICs) and their corresponding spatial maps; (2) selected ICs in which the alpha-band spectral power modulations were correlated with the experimental paradigm; (3) estimated cortical source distributions from selected ICs' spatial maps using the EEG minimum norm inverse algorithm. The source distributions therefore quantified the locations, extensions and magnitudes of neural generators in which alpha temporal modulations were characterized by the corresponding ICs. (4) Finally, we summed the products of selected ICs' source distributions and time courses, which gave us the spatio-temporal dynamic source distribution underlying the alpha modulation. Fig. 1 illustrates the logic of the analyses.

To localize the brain hemodynamic fluctuation responding to the eyes-open-eyes-closed task, we applied the general linear model (GLM) analysis. Regressors were designed by defining the eyes-open condition as "on" and eyes-closed condition as "off". In order to relate the source locations estimated from EEG and fMRI, fMRI imaging results were projected and interpolated onto the cortical surface.

Results:

Fig. 2 shows the results from a representative subject who went through a self-paced eyes-open-eyes-closed task during fMRI-EEG simultaneous recording. The results, though from one subject, were typical of all the subjects studied. Fig. 2(a) shows the spatial maps and spectrograms of selected ICs. The spectrograms show prominent modulation of alpha band spectral power, and the spatial maps indicate the involvement of occipital and parietal lobes in the rhythmic modulation. fMRI statistical maps in Fig. 2(b) shows an increased BOLD signal in the eyes-open conditions versus the eyes-closed conditions. The occipital and parietal BOLD activation was present in all the four subjects; however, the frontal activation only existed in the subject shown here. There were also deactivations in the occipital parietal sulcus observed in one subject. To visualize the neural source of task-modulated alpha rhythm, we averaged the estimated dynamic source distribution over time, which is plotted in Fig. 2 (c). By comparing the EEG source distribution and fMRI maps (b and d), we found that the neural origins estimated from the two modalities were co-localized with each other in occipital and parietal lobes. Although the EEG estimation has lower spatial resolution, the current density maxima overlapped with the BOLD activation very well. Fig. 2(d) shows the convolution of averaged alpha spectral power with hemodynamic response function, and the BOLD time course from the co-localized regions. The plot indicates that occipital and parietal alpha power modulation and the BOLD activation were negatively correlated.

Discussion and Conclusions:

In the present study, we have investigated the relationship between electrophysiological alpha rhythmic modulation and BOLD signal fluctuation. Our results suggest that during the eyes-open-eyes-closed task, the spontaneous neural activities in the occipital and parietal lobes change correspondingly, resulting in the EEG alpha rhythmic modulation as well as BOLD fMRI fluctuation. Although there was no direct evidence to demonstrate their functional dependence, our data suggest the presence of the spatial and temporal correlations between the alpha modulation and the BOLD fluctuation.

Alpha band activity is prevalent during brain baselines. Its spontaneous fluctuation has been suggested to be temporally correlated with different brain networks [4, 5]. Our data indicates that when alternating between the two baselines, the major brain network exhibiting spatially and temporally correlated alpha modulation and hemodynamic fluctuation resides in the occipital and parietal lobes. This network may be correlated with brain alertness and brain "idling" states [1].

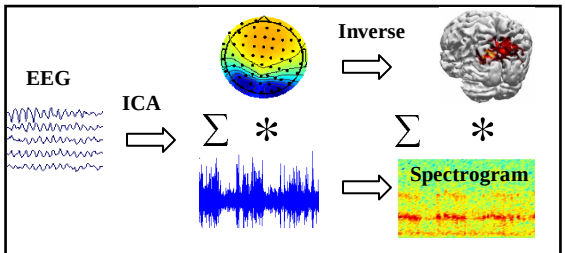

Fig. 1. The schematic diagram of the EEG analyses.

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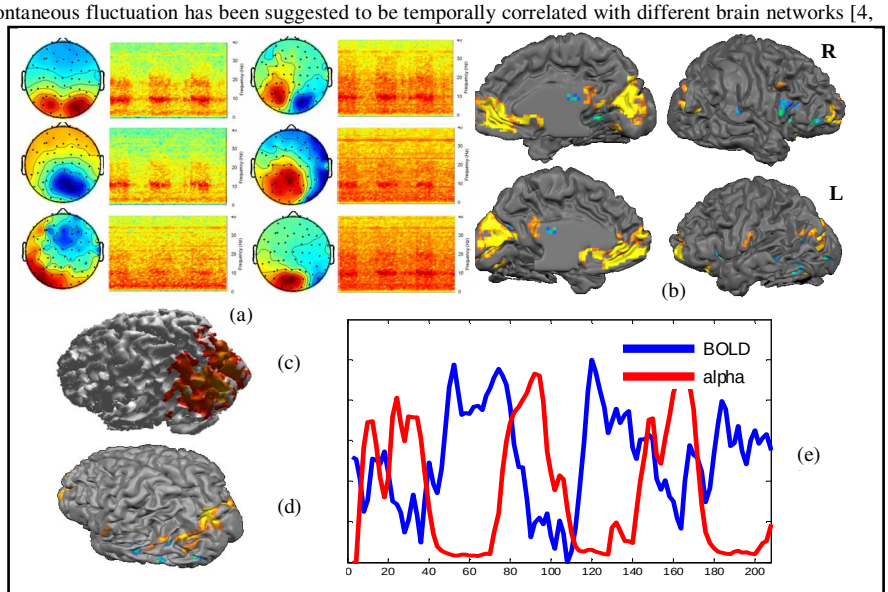

Fig. 2. Results from one representative subject. (a) Selected ICs characterized by alpha modulation; (b) fMRI statistical maps contrasting eyes-open vs. eyes-closed; (c) Temporal averaged cortical source distribution underlying task-modulated alpha rhythm; (d) fMRI map from back view; (e) Spatially averaged alpha spectral power (red) vs. fMRI time course (blue) from occipital and parietal activated regions.