Regional Homogeneity: Epileptic Patients vs. Healthy Volunteers

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Introduction

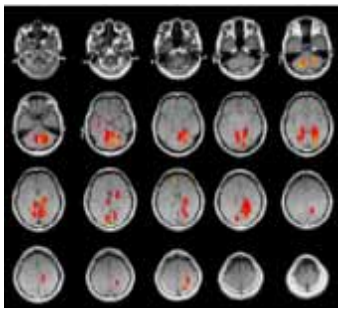
Current fMRI techniques have pitfalls that cause low sensitivity and limit its use in clinical epileptic diagnoses. For example, in a spike-trigger fMRI method, an EEG expert must be on site to identify the interictal epileptiform discharges (IEDs) and to initiate the MRI manually within 3-5 s of the event. Some IEDs may not be identified because the EEG is obscured by the MRI artifact during the scan. In addition, only one measure can be made after each spike, whereas the hemodynamic response of the BOLD effect is thought to last more than 10 s. Although an alternative technique of continuous fMRI acquisition combined with EEG recording appears more sensitive than the spike-trigger method, epileptic activity could be found in only 39% of 31 patients studied [1]. In 1990, a Kendall's Coefficient (KCC) correlation method was proposed to measure a regional homogeneity (ReHo) of the time series of a given voxel with those of its nearest neighbors in a voxel-wise analysis[2]. Based on a hypothesis that ReHo could be modulated by epileptic neuronal activity, KCC analysis may be utilized to measure the similarity of time series of neuron activation on the resting-state fMRI data sets to distinguish the activity patterns between epileptic patients and healthy subjects.

Methods

Five health volunteers (aged 26–36 years; mean 30.7 years; one male) and 10 epileptic patients (aged 20–40 years; mean 30.1 years; five males) participated in this study. The resting-state fMRI datasets were acquired on both 1.5 and 3 T MRI scanners. EPI sequence was used with parameters TR/TE = 2 s/30 ms, matrix 64 x 64, FOV = 24 cm, 4 mm thickness/1 mm gap, and 23 slices. BOLD-based fMRI images were acquired while subject was at rest with eyes closed in a period of 400s scan time, and a total of 200 images for each slice resulted. The KCC values were calculated by combining the neighboring nine-voxel fMRI signal according to the KCC method [3]. A threshold of KCC > 0.6 ($p < 0.05$) was chosen to obtain the functional KCC map.

Results and Discussion

Figure 1 shows a typical KCC functional map from a single subject. The other four healthy subjects' KCC maps show a similar pattern as Figure 1: correlated activation areas are mainly located in occipital lobe, posterior cingulated cortex (PCC), hippocampus and cerebellum. This finding is consistent with the results reported for brain spontaneous low-Frequency BOLD signal fluctuation (default model of brain function) in PCC, part of the occipital lobe and cerebellum [3]. Figure 2 shows results from a lipoma epileptic patient. The correlated activation areas are mainly located in frontal lobe and left temporal lobe, which is near the patient's lipoma symptom in his left temporal lobe. No activation in occipital lobe or cerebellum was found in this patient. The results of the other nine epileptic patients show similar activation patterns; that is, little or no activation found in occipital lobe, PCC, hippocampus and cerebellum area. The activation is located in the areas surrounding the epileptic symptom. The difference observed between patients and healthy subjects in the KCC maps may result because the abnormal neuron discharge evoked epileptic activity enhanced the correlation within the areas associated with the epileptic sources. Our results are consistent with the functional connectivity studies performed on Alzheimer and schizophrenia patients. The default model study showed that activation detected from Alzheimer patients is significantly smaller than that in healthy man in the areas of PCC and Hippocampus [4].



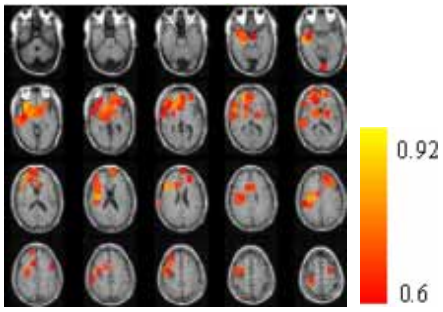


Figure 1 Functional KCC map from a healthy subject. Figure .2 Functional KCC map from an epileptic patient.

Acknowledgment

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Reference

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