Simultaneous intracranial EEG-fMRI of a seizure
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Introduction: EEG-fMRI has been used to map ictal networks; BOLD patterns consistently include decreases in areas spatially distant from the seizure onset zone[1]. A recent development has been the implementation of simultaneous recording of intracranial-EEG and fMRI (icEEG-fMRI)[2-5] demonstrating significant interictal epileptiform discharges (IED)-related BOLD changes, in all cases studied to date. Here we report on a patient who had a focal electrographic seizure during icEEG-fMRI.

Methods: A 30y.o. male undergoing intracranial-EEG recordings as part of presurgical evaluation for refractory focal temporal lobe epilepsy was scanned as part of series of 14 patients. We used a strict safety protocol [2,3], and obtained informed written consent to acquire icEEG-fMRI data. EEG equipment and electrodes were used ‘off label’. An electrographic seizure from the right amygdala occurred during the icEEG-fMRI recording. The patient subsequently underwent left anterior temporal lobe resection (all clinical seizures recorded by icEEG alone were from the left) and has ILAE Class I postsurgical outcome at 3 years. We constructed two fMRI models of the seizure: 1. A visually-derived model consisting of a 30s pre-ictal phase and ictal onset and late-ictal phases corresponding to transitions observed on the EEG, as in [1]. 2. A quantitative EEG model of spectral dynamics calculated by taking the channel of earliest visual change, transforming into the time-frequency domain, z-transforming relative to baseline and convolving by the canonical haemodynamic response function. Dimensionality of the model is then reduced using the principal components explaining >90% of the variance (see fig. 1). Motion and cardiac confounds were added to both models and estimated using SPM8 ([www.fil.ion.ucl.ac.uk](http://www.fil.ion.ucl.ac.uk)). F-contrasts across each ictal phase and across the PCA components were used to reveal associated BOLD changes at p<0.001 uncorrected.

Results: The ictal onset consisted of fast (beta) activity involving right mesial temporal contacts (duration: 6.4sec). The late ictal phase was marked by fast (gamma) activity in right and left mesial temporal contacts (duration: 14.8s).

Preictal BOLD changes were distributed bilaterally including in the left superior temporal gyrus and right superior temporal gyrus. For the ictal onset phase, BOLD changes were seen in the right fusiform gyrus, middle temporal gyrus, precuneus, posterior temporal, medial temporal pole, orbitofrontal cortex medial superior frontal gyrus, middle frontal gyrus and posterior cingulate. For late-ictal phase, BOLD changes were seen in the precuneus and posterior cingulate, left temporal pole, and right middle frontal gyrus. The PCA model for the right amygdala showed BOLD changes in the right sensori-motor cortex, middle temporal gyrus, posterior temporal and amygdala and medial occipital; on the left changes were found in the superior parietal lobe and posterior hippocampus, (fig. 1). Higher statistical scores were found for the PCA model compared to the visually coded model. The maps were qualitatively similar between the two modeling approaches although the PCA model from the right amygdala additionally included a cluster proximal to the electrode that recorded seizure onset.

Conclusions: We have recorded the first sub-clinical seizure with simultaneous intracranial EEG and fMRI. We used two modeling approaches: using neurophysiologically meaningful features and EEG spectral dynamics quantification. Both models revealed BOLD changes in a network containing nodes around the onset zone and also in remote cortical and deeper structures.


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