fMRI 'deactivation' of the posterior cingulate during generalized spike and wave

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Synopsis
Using spike triggered fMRI, we demonstrate regional Blood Oxygen Level Dependent (BOLD) signal changes associated with spontaneous generalised spike and wave discharges (S&W). Five adults with Idiopathic Generalised Epilepsy with frequent S&W were studied. EEG was recorded inside a three tesla MRI, allowing acquisition of single, whole brain fMRI images following S&W. Significant S&W related BOLD signal reductions were observed in the posterior cingulate. Some signal increases were seen in the depths of the pre-central sulci, but not in the thalamus. The posterior cingulate may have a special role in the electroclinical phenomenon of S&W and 'absence'.

Spike triggered fMRI - EEG was recorded with the patient in the MRI to allow image acquisition triggered by epileptiform activity (Archer, J., et al., 2001). Eighteen non-metallic scalp electrodes with carbon fibre leads were fixed to the scalp using colloid and conductive gel in the conventional '10-20' EEG format, with two chest electrodes to record ECG signal. Electrodes were twisted in pairs immediately on leaving the scalp (Allen, P. J. et al., 1998), then woven in chains and taken straight out the head end of the scanner bore. A head-box with fibre optic coupling transmitted the EEG signal out of the MR room for display and recording in real time (figure 1). The frequency and morphology of the patients’ epileptiform discharges were characterised during a ten minute recording with this equipment prior to entering the MR room.

MR imaging was performed on a GE Signa 3 tesla MR scanner. Functional sequences used Gradient-Echo Echo-Planar Imaging (EPI) with whole brain coverage (20 axial slices, 4 mm thick, 1 mm gap), 128 x 128 matrix, 24cm x 24cm FOV, 40º flip angle, TE 40ms, TR 3000ms. Manual triggering, with an average delay of 2.5 seconds, was used to acquire a single whole brain EPI MR volume over three seconds immediately following a typical S&W discharge. We acquired single ‘baseline’ images in a similar fashion when the EEG showed no epileptiform activity for at least 15 seconds. Spike and baseline images were acquired interspersed over 60 minutes.

Analysis - Images were analysed using iBrain®, (Brain Research Institute, Melbourne) and SPM99 (Wellcome Department of Cognitive Neurology, London). Processing for group analysis included image realignment, spatial normalisation of all subjects’ volumes to Talairach space, global intensity scaling and Gaussian smoothing of 8 millimetres full-width-at-half-maximum.

Results - A large region of spike related deactivation was observed in the posterior cingulate / retrosplenial area (Puncorrected< 0.001, cluster size 511 voxels); the most significant voxel in this region was almost significant even when correcting for multiple-comparisons across the entire brain (Pcorrected = 0.054). BOLD signal increases were also detected, however they appeared generally less significant and more variable than the BOLD decreases.

Conclusion - In this fMRI study of spontaneous spike and wave discharges in idiopathic generalized epilepsy, we observed significant deactivation of the posterior cingulate / retrosplenial area. This region may have a special role in the electroclinical phenomenon of S&W and 'absence'. This role might be causative, with reduced activity in the posterior cingulate facilitating the onset of S&W. Alternatively, the reduced activity may be the consequence of S&W activity. Decreased posterior cingulate activity may be a marker of altered thalamocortical activity and might be important in the pathogenesis of S&W.

Figure 1. Left: EEG of a subject taken inside the MR scanner. The pattern of polyspike and slow wave is clearly seen at the right hand edge of the trace. EEG is viewed in real time in bipolar montage to reduce ‘cardioballistic’ artefact. Middle & Right: SPM99 group conjunction analysis of spike related ‘deactivation’.

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