Cortical Thickness in Left Temporal Lobe Epilepsy

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Introduction
Hippocampal atrophy is the most commonly reported MRI finding in patients with temporal lobe epilepsy (TLE). Structural damage outside of the hippocampus has been little investigated and the majority of studies have limited their analysis to specific regions of interest. We previously demonstrated parahippocampal atrophy in TLE involving mostly the entorhinal cortex \cite{1,2}. The present study aims to investigate changes in cortical thickness across the entire brain in TLE.

Methods
46 left-TLE patients and 50 normal controls were investigated. Lateralization of seizure focus in TLE was determined by a comprehensive evaluation including video-EEG telemetry. A T1-weighted MRI with 1mm sampling was acquired for each subject. Volumetric MRI showed left hippocampal atrophy in all patients. The MRIs were linearly registered into stereotaxic space formable models \cite{6}. The distance between the corresponding vertices on the white and grey matter surfaces was measured at 40,962 points across the cortex \cite{6}, and the resulting thickness map blurred using a 20 millimetre surface based blurring kernel \cite{7}. Each subjects’ thickness data was centred and scaled to the group mean. Regression against group was performed at every vertex, multiple comparisons corrected for using the False Discovery Rate alpha of 0.01, corresponding to a t-value of -2.48. A further regression against duration of the disorder was performed (t=-4.5) within the patient population (45 df, t = 2.53 at q = 0.01).

Results
As expected, the left parahippocampal gyrus was atrophied in the patients (t=-5.1). This was contralateral to the epileptogenic zone. The ipsilateral insula also showed significant atrophy accompanied by a thickening of the fusiform gyrus bilaterally (ipsilateral fusiform t=5.0, contralateral fusiform t=3.6). There was further atrophy in the frontal lobes (BA 44, 6, 8), stronger corresponding to a t-value of -2.48. A further regression against duration of the disorder was performed within the patient population (45 df, t = 2.53 at q = 0.01).

Discussion and Conclusions
These results both manage to confirm known data (reduction in thickness in the ipsilateral medial temporal lobes) and provide new insights into epilepsy. The latter includes both areas that correlate with the disorder and lead to atrophy as well as thickening, and areas that change as a function of the duration of the disorder.

Several interesting hypotheses could account for the extra MTL results:

- These abnormalities could be the result of secondary eliptogenesis.
- These changes could be secondary effects of the disorder. The increased thickness of the motor areas with duration might, for example, be due to increased motor activity during seizures.
- The findings might be due to developmental disturbances, coexisting with or caused by the epilepsy.

All of these hypotheses will be investigated in further research in order to further understand the impact of epilepsy on the cerebral cortex.

References