EDITORIAL COMMENTARY

Interictal focal activity in temporal lobe epilepsy

When evaluating patients with epilepsy, the integration of data from different modalities is the key to understanding the localisation of onset of seizures, the epilepsy syndrome, and the aetiology. This process is particularly important when patients have refractory partial seizures and neurosurgical treatment is an option; the best results from surgery being obtained when the operative procedure removes the area of brain that gives rise to the seizures but is confined to cerebral tissue, the loss of which does not give rise to significant cognitive or neurological disturbance, with the remaining brain functioning normally. In this issue, the paper by Koutroumanidis *et al* (pp 170) describes 28 patients with temporal lobe epilepsy that was not due to foreign tissue lesions, in whom surgical treatment was being considered. The emphasis of the paper was the EEG finding of interictal regional slow wave activity and the relation of this to data that infer functional abnormality—clinical features, regional cerebral glucose metabolism; and with structural data—MRI and the pathology of the subsequently resected temporal lobes.

The other functional data that may be integrated into the overall picture include the clinical description of seizures, neuropsychological tests, functional brain imaging (functional MRI and single photon emission computed tomography), and functional electrophysiological data (interictal and ictal EEG and magnetoencephalograms (MEG)).¹

In presurgical evaluation of the EEG, it is the distribution of ictal and interictal epileptiform activity that generally receives the most attention and the paper by Koutroumanidis *et al* serves as a timely reminder to pay due attention to abnormalities of the background EEG. A striking finding was the concordance between regional slow activity recorded from anterior, mid-temporal, and posterior temporal electrodes with hypometabolism in the posterior lateral temporal neocortex.

Brain PET with ¹⁸F-deoxyglucose (¹⁸FDG) commonly shows hypometabolism in a region that includes the site of onset of seizures. Regional diaschisis and a functional inhibition are the likely causes for the area of hypometabolism being larger than the pathological abnormality. The use of ¹⁸FDG PET may show abnormalities when MRI is unremarkable.² Comparisons of this type, however, depend crucially on the relative sophistication of the MRI and PET equipment and protocols being used. ¹¹C-flumazenil binds to the central benzodiazepine receptor that is part of most cerebral GABA_A receptors. It has been known for a decade that epileptic foci are commonly associated with an area of reduced flumazenil binding and for 5 years that this area is usually more restricted than the area of hypometabolism.^{3,4}

The mechanisms giving rise to regional slow activity in this setting, in the absence of an underlying neocortical or foreign tissue lesion, are not clearly understood. The parallel with the finding of regional hypometabolism suggests that a reversible diaschisis and inhibitory processes, which result in neuronal dysfunction, may be the underlying causes.

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- 2 Gaillard WD, Bhatia S, Bookheimer SY, *et al*. FDG-PET and volumetric MRI in the evaluation of patients with partial epilepsy. *Neurology* 1995;45:123–6.
- 3 Henry TR, Frey KA, Sackellares JC, *et al*. In vivo cerebral metabolism and central benzodiazepine-receptor binding in temporal lobe epilepsy. *Neurology* 1993;43:1998–2006.
- 4 Savic I, Ingvar M, Stone-Elander S. Comparison of ¹¹C-flumazenil and ¹⁸F-FDG as PET markers of epileptic foci. *J Neurol Neurosurg Psychiatry* 1993;56:615–21.