Temporal lobe epilepsy

Surgical treatment of temporal lobe epilepsy

S F Berkovic

It is important that presurgical evaluation of patients with temporal lobe epilepsy is carried out by multidisciplinary teams

The paper published in this issue by Jutila *et al* (this issue, pp 486–94) adds to the literature regarding surgical treatment of temporal lobe epilepsy.[1] This paper presents results from a national centre for adults in Finland. The strength of this report is that it has long term follow up from a relatively defined population. The benefit of temporal lobectomy in treatment of refractory temporal lobe epilepsy has been accepted for many years and a recent creative controlled trial from a Canadian group clearly established its efficacy over medical therapy.[2] The efficacy of treatment appears to be maintained over the long term, although there is a significant attrition of cases who are initially seizure free for the first 12 months. However, according to the Finnish experience reported here, such late relapses generally do not represent a return to severe intractable temporal lobe epilepsy.

Can selection of cases be improved and why do not all patients respond?[3] These issues have been central to over 100 studies of temporal lobectomy in the last decade (for review see Hennessy *et al*). Relatively few markers have emerged as definitively helping in choice of patients. The finding of a localised lesion on magnetic resonance imaging and a predominance of focal seizures were positively associated with good outcome by Jutila *et al*, findings that are supported by earlier reports.[4] The current study also found earlier age of onset as predictive of good outcome, but this has not emerged as a robust factor in previous studies.[4] Methodological problems in such post hoc analyses can be critical.[4] Improvement in our ability to select cases for operation, and perhaps more importantly reject inappropriate cases, will await more detailed studies. However, Jutila *et al* found that some of their "palliative" cases, by which they meant patients in whom there were less convincing evidence for a unilateral confined epileptogenic zone, did have a significant seizure free rate. The study emphasises the importance of a multidisciplinary team in the complex presurgical evaluation of candidates. Seizure surgery should be performed in centres with multidisciplinary expertise and experience to maximise outcomes.

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Alzheimer's disease

Hippocampal atrophy and neocortical dysfunction in early Alzheimer's disease

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Is there a relationship between medial temporal lobe atrophy and brain dysfunction in Alzheimer's disease?

Although it would appear straightforward that neuronal loss in and by itself explains the cognitive deficits in Alzheimer's disease (AD), things are considerably more complicated as shown by Garrido *et al* (this issue pp 508–16).[1] In this elegant study of nine patients with very mild AD, the authors assessed the relationships between hippocampal volume (assessed with structural magnetic resonance imaging (MRI) and voxel based morphometry), and the changes in cerebral perfusion (mapped with single photon emission computed tomography (SPECT)) during a verbal recognition memory task. Increasing hippocampal atrophy was associated with reduced activation of the extensive left sided network normally engaged by this episodic task, but also with *increased* activation in several frontal regions. They interpret these findings as suggesting that medial temporal lobe (MTL) pathology negatively modulates structures belonging to this network and reciprocally connected with the hippocampus, while also positively modulating activity in non-specific executive control centres—also indirectly connected to the hippocampal complex—probably reflecting an attempt by the AD brain to maintain performance by increased attentional/strategic load.

Although this study has some acknowledged limitations, the results are convincingly robust. Furthermore, they are entirely consistent with other work. Using positron emission tomography, Meguro *et al* reported a significant correlation between the degree of hippocampal atrophy and the severity of resting state glucose hypometabolism in the ipsilateral parietotemporal association cortex (PACx).[2] This correlation remained significant even after controlling for degree of dementia, thus ruling out a simple effect of disease severity. This suggested that the striking PACx hypometabolism present from the very early stages of AD may not simply reflect the degree of local pathology but also in part a remote effect