Epilepsy

Presurgical evaluations of patients with intractable epilepsy

R P Lesser

Neuroimaging is helpful, but not definitive, when deciding whether to perform a surgical resection to treat intractable epilepsy

It seems straightforward: find the seizure focus, take it out, the seizures stop. Advances in neuroimaging and in electroencephalography (EEG), including the use of intracranial electrodes, should make this even easier.

In this issue, Alarcón *et al* (see page 474) compare the results of seizure surgery among patients with normal, or with abnormal, neuroimaging and who did, or did not, undergo recordings with intracranial electrodes.¹ All patients had scalp EEG and magnetic resonance imaging (MRI). Intracranial (subdural or sometimes depth) electrodes were placed when neuroimaging was normal. For all patients, about half became seizure free (Engel class I); about three quarters improved considerably (Engel classes I and II). There were no significant differences based on neuroimaging results, or on the use of intracranial electrodes. The authors correctly point out that these results indicate that surgery can control seizures even when neuroimaging is normal. However, in addition, specialised methods (epilepsy protocol neuroimaging, intracranial electrodes) did not seem to produce better outcomes. why?

One, patients were implanted when seizure focus location was less defined. These patients subsequently did as well as patients who had more straightforward findings on scalp EEG and MRI, and who therefore did not require electrode implants. Two, all abnormal neuroimaging results were grouped, but many epileptologists think that good outcomes are more likely to occur when certain abnormalities are present, such as mesial temporal sclerosis,² a pathological finding which occurs when normal neurons in the hippocampus are replaced by gliosis. In addition, Alarcón *et al* found that mesial temporal sclerosis was not always obvious on neuroimaging.¹ Possibly, higher strength magnets—for example, 3 tesla—would have shown abnormalities.

Three, was enough removed? Since seizures continued in many, apparently not. Some underwent removal primarily of the lesion found on imaging-lesionectomy. Perhaps surgeons remove less tissue when there is a lesion, and more when there is not. This may be true, but it is not proven. Moreover, consider another group of patients, those with

dual pathology—the existence of more than one abnormal region on imaging. In some cases, this means you must remove more than one region to control seizures.³ In others, seizures stop after removing only one.⁴ Less can be enough.

Sophisticated contemporary electrophysiology and neuroimaging allow us to control seizures in many patients that we could not help before. We do not do as well with other patients, and we often don't know why. Technical and conceptual advances should improve our ability to localise and then surgically control seizures in the future. Meanwhile we will need to realise—and tell our patients—that seizures may be controlled, despite a normal test result, or may persist when we think that they should stop. Our brains are smarter than we are.

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Concussion

Neuropsychological dysfunction following repeat concussions in jockeys

T W Teasdale

A second concussion risks permanent cognitive dysfunction

The cognitive effects of a concussion are known to be briefly severe but overwhelmingly transient. The study by Wall *et al*¹ (see page 518) represents an ingenious approach to the question of whether there is nonetheless a compounded cognitive effect of multiple concussions. This is an important issue because of the relatively frequent incidence of concussions generally, and the suggestion that having had a concussion

itself constitutes a risk factor for further concussions, particularly where such concussions are incurred in the pursuit of a riskful sporting activity. The authors are appropriately cautious in their interpretation of results in what, necessarily, is only an observational study from which direct causal inferences cannot be made. Nonetheless, they do provide further evidence consistent with an interpretation that a second concussion can compound

some latent consequences of the first so as to produce manifest cognitive deficit. Furthermore their data would seem to suggest that such an increased vulnerability following a first concussion can remain for long periods of time. If this proves correct, the clear implication is that patients who have suffered a first concussion should be alerted to their increased risk of important and potentially enduring cognitive dysfunction should they suffer a further concussion.

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