Sphenoidal electrodes in localising temporal epileptic focus, in association with CT, MRI and SPECT

We read with interest the article by Duncan et al.1

MRI is certainly superior to CT, and SPECT is superior to MRI in detecting lateralising lesions in temporal lobe epilepsy.2

Interictal studies in temporal lobe epilepsy using both PET and SPECT show usually focal temporal hypofusion as the most common abnormality.3 The danger in localising epileptic focus by PET or SPECT lies in the fact that localisation is carried out during the interictal period and not during the ictal period. The major interest of EEG is to record the electroclinical epileptic fit, and to localise exactly the active epileptic focus that may be removed at surgery.

The correlation between laterisation based on single surface EEG and that based on hypoperfusion seen on PET or SPECT, improves with multiple EEG recordings.4 We would like to emphasise the usefulness of sphenoidal electrodes which, even in an extracranial setting, are capable of recording all the spikes coming from the internal temporal lobe,5 and differentiate from spikes coming from the frontal lobe.6

There is not always a strong correlation between the epileptic focus that gives clinical seizures, and the lesion observed by neuroradiology. A physiological testing can be used to observe the true localisation of the epileptic focus during an electroclinical fit.

Sphenoidal electrodes and EEG are therefore necessary to carry out the main lateralising investigation in most cases of temporal lobe epilepsy, and results of CT, MRI, SPECT and PET should be correlated with electrophysiological data, to improve selection of patients who can benefit from temporal lobectomy.

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Drs Septiens and Giroud state that the lack of localising with PET or SPECT lies in the fact that these investigations are carried out interictally. In this, the authors (we infer) regard interictal SPECT as giving information analogous to that given by interictal EEG spikes. However, as we point out, there is no such direct correlation, the correlation between the two is often not good. Our experience suggests that simple unilateral temporal hypoperfusion is indeed a reliable localising finding. We would point out, however, that we find this in only around 30% of our overall series of patients with complex partial seizures. A further 30% have other findings (such as more extensive hypoperfusion, focal hyperperfusion or combinations of the above) which fail to fit the definition of hypoperfusion, the reliability of which we are less sure of. The remaining patients have normal regional cerebral blood flow (rCBF). Series which report localising abnormalities in remaining proportions of patients2 3 4 do tend to have some patients who localise falsely using SPECT. Hence we feel strongly that localising reliability depends crucially on conservative reporting of images.

SPECT can of course be performed immediately postictally, or even during a seizure, as we are increasingly succeeding in doing. Initial data (our own and from elsewhere) suggest that this provides localising information in a higher proportion of patients, and may in particular help the important discrimination between frontal and temporal foci.

We hold very much that the development of SPECT imaging of rCBF (and more recently of benzodiazepine receptor density) will reduce the need for the longterm and invasive EEG monitoring at present necessary in so many patients, rather than simply adding yet another test to an already extensive assessment.

To what extent this turns out to be possible will depend on the results of prolonged assessment of its ability to predict surgical success.

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Sphenoidal electrodes in localising temporal epileptic focus, in association with CT, MRI and SPECT imaging in temporal lobe epilepsy.

We thank Drs Septiens and Giroud for their comments. We do, of course, agree entirely with what they have said. In particular, we have noticed recurring reports of the usefulness of sphenoidal electrodes and other EEG techniques in localising epileptic foci. The main point of our paper was the correlation between different imaging modalities, not the correlation between imaging modalities and electrophysiological localisation. Drs Septiens and Giroud state that the lack of localising with PET or SPECT lies in the fact that these investigations are carried out interictally. In this, the authors (we infer) regard interictal SPECT as giving information analogous to that given by interictal EEG spikes. However, as we point out, there is no such direct correlation, the correlation between the two is often not good. Our experience suggests that simple unilateral temporal hypoperfusion is indeed a reliable localising finding. We would point out, however, that we find this in only around 30% of our overall series of patients with complex partial seizures. A further 30% have other findings (such as more extensive hypoperfusion, focal hyperperfusion or combinations of the above) which fail to fit the definition of hypoperfusion, the reliability of which we are less sure of. The remaining patients have normal regional cerebral blood flow (rCBF). Series which report localising abnormalities in remaining proportions of patients do tend to have some patients who localise falsely using SPECT. Hence we feel strongly that localising reliability depends crucially on conservative reporting of images.

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New criteria for brain death?

The optimism of Faccio et al1 2 in proclaiming short latency evoked potentials as the ultimate achievement in diagnosing brain death is unwarranted and their findings are not new.3 4 The data were gathered in a selected sample from which an unspecified number of patients was excluded. A more convincing and scientifically sound method would have been to examine consecutive admissions. The authors advocate the use of evoked potentials instead of EEG for the determination of brain death without having included EEG recordings in their study. They may well be right, but the EEG is a valuable tool in confirming death especially if intoxication is suspected: if a diagnosis of whole brain death is to be made, an additional EEG may even be mandatory, especially with infratentorial pathology.2

The extinction of AEP waves III-V may be indicative of irreversible loss of brain stem function, particularly if their gradual disappearance had been documented. This was the case in only 4/46 (11%) in the report by Faccio et al.1 If all waves including wave I are lacking at the first examination and damage to the eighth cranial nerve cannot be excluded, (as is often the case in brain death), the record- ing may be flat due to other reasons. In this case an EEG cannot be considered confirmatory. Also with somatosensory evoked potentials (SSEP) there may be some pitfalls in certain cases. They are the first to herald a fatal prognosis if cortical potentials disappear bilaterally.6 However, loss of cortically generated SSEPs is a bad prognostic sign but not the proof of brain death, as both brainstem and cortical function must be lost (whole brain criteria), or loss of cortical function is of no relevance (brain stem criteria).6

SSEP's may also be contaminated by muscle activity obscuring brain death.3 4 Damage to the peripheral nerves, nerve roots and the medulla may preclude SSEP recordings. This renders AEP and SSEP in many cases a more valuable tool for excluding brain death than for confirming it.

It should not go unnoticed that there are radiological methods suitable for confirming brain death and that the clinical conditions and close clinical scrutiny brain death may be safely diagnosed without confirmatory tests.3 In view of this situation there is little to support the enthusiasm of Dr Faccio and his colleagues.

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Matters arising


Facco replies:

Dr Lang and his quotation 1 already pointed out the perfect agreement between evoked potentials data and the UK criteria of brain death in our series: that means that a careful clinical diagnosis is reasonably safe even without confirmatory tests. However, the concept of brain stem death implies the diagnosis of the death of the whole brainstem, rather than of a part of it; consequently, I believe that we need to be able to check all these structures, and this is what we routinely do in our patients. There is no reason to avoid the "objective" assessment of easily and non invasively explorable pathways, such as the auditory and somatosensory ones. Our results enable us to recommend ABR and SEP for the sake of coherence with the underlying concept of brain stem death and for the sake of safety (which implies both an "objective" confirmation and the exclusion of false positives).

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Transient entrapment neuropathy of the posterior interosseous nerve in violin players

I was most interested to read the article by Drs Maffulli 1 describing what they consider to be transient entrapment of the posterior interosseous nerve in violin players, and I am grateful to them for quoting several of our publications. However, I have some problems with this report.

The diagnosis of this painful form of posterior interosseous neuropathy, often referred to in the neurophysiology literature as the radial tunnel syndrome, is a difficult one since, as in the cases described here, no neurological deficits are detectable. The occurrence of pain is often associated with repetitive activities of positions, even when it appears to be in the distribution of a single nerve and even when there appears to be tenderness at some point along the course of that nerve, rarely provides convincing evidence of nerve entrapment. The authors do describe transient "muscular deficit" in specific muscles supplied by the radial nerve, although these three patients allegedly had weakness of the extensor carpi radialis brevis, which is supplied by a branch of the radial nerve proximal to the posterior interosseous nerve.

I am particularly puzzled by the repeated description provided by the authors that prolonged pronation of the forearm while playing the violin seemed to predispose to this condition. The problem is that, except for the rare individual who plays "left-handed," is held in a position of extreme supination, not pronation, while playing.

Posterior interosseous neuropathy has only rarely been identified in musicians. A case of the paralytic form was described by Guillon and Courtellemont 2 in an orchestral conductor. One of the patients described by Woltman et al. 3 had to stop at the age of 13 because of right hand weakness. Charness et al. 4 described a flautist with a left posterior interosseous neuropathy.

Of 500 instrumentalists evaluated over the past 12 years, I have seen a flautist and a percussionist with alleged posterior interosseous neuropathy. Both were seen post operatively and had minimal clinical and electromyographic evidence of partial radial neuropathy at that time. I have studied 175 violinists and viola players, including 106 who had exclusively or predominantly left upper extremity symptoms, and I have not been able to identify the single problem that has been associated with thoracic outlet syndrome, a diagnosis which has received considerably more attention than the radial tunnel syndrome. I believe that both exist but we must strive to define both disorders more rigorously so that we can provide the most appropriate therapy.

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New criteria for brain death?

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