Sphenoïdal 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.

Interictal studies in temporal lobe epilepsy using both PET and SPECT show usually focal temporal hypofusion as the most com-mon abnormality.¹ The danger in localising epileptic focus by PET or SPECT lies in the fact that this examination 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 correlaton between lateralisation based on single surface EEG and that based on hypoperfusion seen on PET or SPECT, improves with multiple EEG recordings.3

We would like to emphasise the usefulness of sphenoïdal electrodes which, even in an extracranial setting, are capable of recording all the spikes coming from the internal temporal lobe,4 and differentiate from spikes coming from the frontal lobe.5

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

Sphenoïdal electrodes and EEG are therefore likely to remain 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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Duncan et al reply:

We thank Drs Septien and Giroud for their comments. We do, of course, agree entirely with their main point that is, the usefulness of sphenoïdal 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 Septien and Giroud state that the danger 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 they themselves point out, 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 hypoperfusion and hyperperfusion), 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 higher proportions of patients1-3 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 elsewhere⁴) suggest that this provides localising information in a higher proportion of patients, and may in particular help make the important discrimination between frontal and temporal foci.5

We hope 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 longterm assessment of its ability to predict surgical success.

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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 even favour an abolishment of the EEG. A statement such as "the EEG ... is far from being relevant", however, is far from being relevant and not at all supported by the facts presented. There is an ongoing discussion about the role of the EEG in determining brain death⁴ partly due to technical problems and limited intrarater stability and interrater agreement,⁵ but the same holds true for evoked potential studies.6

The crucial question is whether one is using whole brain or brain stem death criteria. If brain stem criteria are used, as is done in the UK, short latency auditory evoked potentials (AEP) may in some cases be 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 lesions, as it is strongly recommended by the German guidelines.

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 Facco 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 trauma victims), the recording may be flat due to other reasons. In this case an AEP 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.8 However, loss of cortically generated SSEPs is a bad prognostic sign but no 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).⁴

SSEPs may also be contaminated by muscle activity obscuring brain death.10 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 under certain preconditions and close clinical scrutiny brain death may be safely diagnosed without confirmatory tests.⁴ In view of this situation there is little to support the enthusiasm of Dr Facco and his colleagues.

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New criteria for brain death?

The optimism of Facco $et ql^{+}$ in proclaiming short latency evoked potentials as the ultimate achievement in diagnosing brain death is unwarranted and their findings are not new.23 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

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