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.[2]

Interictal studies in temporal lobe epilepsy using both PET and SPECT show usually focal temporal hypofusion as the most common 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 patients[1-3] 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 elsewhere[4]) 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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New criteria for brain death?

The optimism of Facco *et al*[1] in proclaiming short latency evoked potentials as the ultimate achievement in diagnosing brain death is unwarranted and their findings are not new.[2,3] 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 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 death[4] 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.[7]

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).[9]

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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Facco replies:

Some of the criticisms on which I agree with Dr Lang do not appear relevant. Conversely, some of the remarks appear to misunderstand our conclusions. I shall try to answer concisely all Dr Lang's comments:

a) I did not feel so optimistic as I only suggested adding auditory brain stem responses (ABR) and somatosensory evoked potentials (SEP) to the clinical evaluation of brain stem death. The absence of undue optimism is emphasised by the question mark in the title.

b) I agree that our data are not completely new: nevertheless, in most studies dealing with coma and/or brain death, SEPs are recorded using a frontal reference. This does not allow a clear definition of far field components (namely, P13, 14 and N18) and therefore precludes any evaluation about conduction through the brain stem in patients with absent N20. Our study emphasises the need for the non cephalic reference, when a conduction block at the cervico-medullary junction or in the lower brain stem is to be checked (absence of components following P13 or dissociation N13/P13). In my opinion this is the only right method for SEP recording in brain death. We have already reported elsewhere[1] on the possible reversibility of N20 loss, but so far we have not found a reversible disappearance of the P13–N18 complex.

c) I think that the analysis of consecutive cases may be useful to check the rate of sectors unrelated to brain death which might affect the evoked potentials, rather than check their reliability when properly used: all investigations and clinical signs have their own limits and pitfalls, not only ABR and SEP, and the main concern in clinical practice is to use them properly.

d) The statement that "the EEG is far from being relevant" is not a conclusion based on our results, a detailed account of EEG limits and pitfalls is not necessary, as Pallis in 1983[2] has already published an exhaustive analysis.

e) We recommended the use of evoked potentials, but did not advocate their use instead of the EEG: their use does not prevent the recording of the EEG as well, if thought appropriate.

f) I agree that the relevance of the EEG partly depends upon the very concept of brain death (that is, brain stem death or death of the whole brain), as emphasised by Dr Lang. However, brain stem death must always be fulfilled, whatever the accepted concept of brain death. When the death of the whole brain is to be checked, the EEG and/or cerebral blood flow may be helpful confirmatory tests to be added to diagnostic criteria.

g) I did not mention the EEG in results as this was not strictly relevant to the brain stem. In Italy the EEG is mandatory as quoted in the introduction; as a result our patients must have a flat EEG to be declared brain dead.

h) The ABR was able to confirm brain stem death in more than 11% of cases: that is, in 22·2% who showed a preserved wave I and in four further cases (11%) in whom the disappearance of all waves was checked by serial monitoring. Therefore, a total of 33·2% confirmations was present in this series by ABR.

i) So far we have not found that muscle activity caused problems of interpretation; there is only one case reported by Guerit.[3]

j) Finally, according to the last sentence of Dr Lang and his quotation[4] I 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 check all brainstem explorable 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 enabled 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 medical 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 provoked by certain 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 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 violinist's left forearm, 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 Guillain and Courtellemont[2] in an orchestral conductor. One of the patients described by Woltman and Learmonth[3] played the piano but 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 over 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 a single case of posterior interosseous neuropathy although nerve entrapment has been assiduously looked for both clinically and electrodiagnostically. About 25% of these patients had an entrapment of some sort; most of the remainder had a form of muscle-tendon overuse.[5] Thus I am surprised to learn that Drs Maffulli have been able to find 11 such cases in under four years.

The problem with promoting the diagnosis of this painful but non-paralytic form of posterior interosseous neuropathy is that the temptation to offer surgical correction becomes irresistible, sometimes prematurely. This is reminiscent of the problems often associated with thoracic outlet syndrome, a diagnosis which has received considerably more attention than the radial tunnel syndrome. I believe that both entities exist but we must strive to define both disorders more rigorously so that we can provide the most appropriate therapy.

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