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.1 Interstitial studies in temporal lobe epilepsy using both PET and SPECT show usually focal temporal hypofusion as the most common abnormality.2 The danger in localising epileptic focus by PET or SPECT lies in the fact that hypofusion is carried out during the interictal period and not during the ictal period. The major interest of EGG is to record the electroclinical epileptic fit, and to localise exactly the active epileptic focus that may be removed at surgery.

The correlation 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 sphenoidal 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 neuroradiological and physiological testing can be used to observe the true localisation of the epileptic focus during an electroclinical fit. Sphenoidal electrodes and EEG are therefore likely to remain the main localising 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 Drs Septien and Giroud that is, 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 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 we 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 thereof) which may or may not be relevant to the clinical situation.

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 make 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 longer term assessment of its ability to predict surgical success.

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New criteria for brain death?

The optimism of Faccio et al. in proclaiming short latency evoked potentials as the ultimate achievement in diagnosing brain death is unwarranted and their findings are not new. 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 question the reliability of longterm EEG. A statement such as "the EEG... is far from being relevant", however, is far from being relevant and not all supported by the facts presented. There is an ongoing discussion whether the EEG record in brain death partly due to technical problems and limited intrarater stability and interrater agreement, but the same holds true for evoked potential studies.6

A 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 are 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 infraclinically localised lesions. This was 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 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 trauma victims), the recording 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.8 However, loss of cortically generated SSEPs is a bad prognostic sign but not 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 contraindicated 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 their preconditions and close clinical scrutiny brain death may be safely diagnosed without confirmatory tests.11 In view of this situation there is little to support the enthusiasm of Dr Facco and his colleagues.

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References

Matters arising


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 comments. 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 N13), 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 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 ratio of sectors unrelated to brain death which might affect the evoked potentials, rather than check their number. I have properly used: all investigational questions 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 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 also 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 it was not strictly relevant to the brain stem. In Italy the EEG is mandatory as quoted in our 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% 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% 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.

j) Finally, according to the last sentence of Dr Lang and his quotation1 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 the neuroanatomical 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).

FACCO


Transient entrapment neuropathy of the posterior interosseous nerve in violin players

I was most interested to read the article by Drs Maffulli1 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 described 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 "muscular pain", 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 in 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 the condition. I have been able to identify only one case where this seems to have been the reason: it was rare for the individual who plays "left-handed", held in a position of extreme supination, not pronation, while playing.

Posterior interosseous neuropathy is only rarely been identified in musicians. A case of the paralysic form was described by Guerit and Courtellemont2 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 the 500 instrumentals 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 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 Download 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 proposed 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 diagnostic confusion 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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