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.\(^1\) 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 these investigations are carried out interictically. In this, the authors (we infer) regard interictal SPECT as giving information analogous to that given by interictal EEG spikes.\(^2\) However, as we pointed 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 combination of abnormalities) that may be crucial to the localisation of areas abnormal in higher proportions of patients\(^3\) do tend to have some patients who localise falsely using SPECT. Hence we feel strongly that localising reliability depends crucially on a conservative reporting of images.

SPECT can of course be performed immediately postictically, 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.

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 assessment of its predict surgical success.

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Drs Septiën and Giroud answer: We thank Drs Septiën and Giroud for their comments. We do, of course, agree entirely with the authors that is, the usefulness of sphenoidal electrodes and other EEG techni ques 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.

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6 Buchner V. Brain death, as death, death. Activity of motor activity obscuring brain death,\(^3\) Damage to the peripheral nerves, nerve roots and the medulla may preclude SPECT recordings. This renders AEP and SPECT 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 in some conditions and close clinical scrutiny brain death may be safely diagnosed without confirmatory tests.\(^7\) 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 al.\(^1\) in proclaiming short latency evoked potentials as the ultimate achievement in diagnosing brain death is unwarranted and their findings are not new.\(^7\) 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 excessive admisions. 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 claim that this is of no importance. 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 of the role of EEG in brain death\(^4\) partly due to technical problems and limited intratracer stability and intertracer agreement,\(^2\) but the same holds true for evoked potential studies.\(^7\)

The key 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) are a diagnostic sign but not 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 information concerning brain stem death.\(^7\) 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.\(^7\) 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).\(^7\)

SSEP may also be contraindicated by muscle activity obscuring brain death.\(^3\) 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 in some conditions and close clinical scrutiny brain death may be safely diagnosed without confirmatory tests.\(^7\) In view of this situation there is little to support the enthusiasm of Dr Facco and his colleagues.


