ABC of Brain Stem Death

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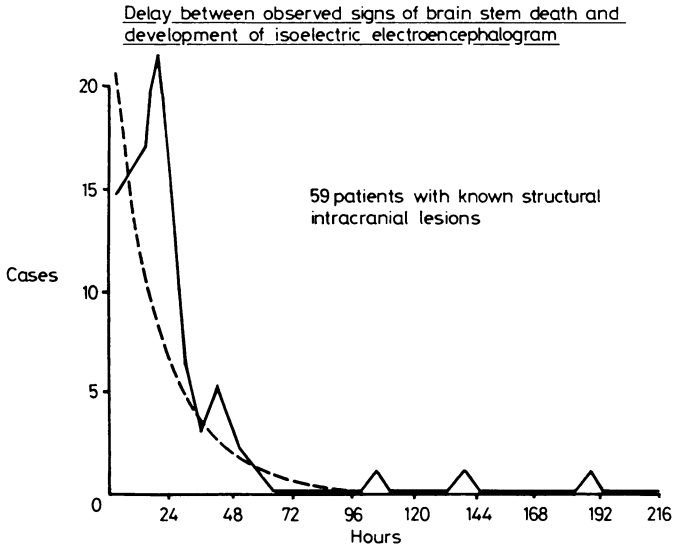
THE ARGUMENTS ABOUT THE EEG

The conceptual argument

The main argument about the electroencephalogram is conceptual, not technical. To what overall concept of death does the electroencephalographic criterion (of electrocerebral silence) relate? Whether they realise it or not the advocates and the detractors of the electroencephalogram are pursuing different objectives, related to different concepts of death. The former are seeking to diagnose the biological "death of the whole brain"—that is, the death of most, if not all, brain cells. With this objective in mind the scalp electroencephalogram may be considered relevant (provided one keeps in mind that it is quite incapable of achieving the desired end). Those who claim that the electroencephalogram is irrelevant are seeking to diagnose death of the brain as a functional unit (death of the "brain as a whole"). They do this by concentrating on what allows the brain to function as a unit: the brain stem. In pursuit of that objective the electroencephalogram is indeed irrelevant. Recording an electroencephalogram from the scalp is not testing a brain stem function.

How important, in practice, is the difference between the two approaches? An important study carried out at the Salpêtrière Hospital in Paris over 10 years ago[1] showed that when apnoeic coma and absent brain stem reflexes occurred in a context of structural brain disease minor residual electroencephalographic activity was common but never persisted. In the vast majority of cases no electroencephalogram could be recorded after 48 hours.



Prognostic significance of brain stem signs[2]
(Structural brain lesions. No drug induced cases)

No of cases	Brain stem areflexia	Apnoea	EEG	Asystole within days
>1000	All	All	"Isoelectric"	All
147	All	All	Some residual activity	All
16	None	None	"Isoelectric"	None

The box seeks to make a different but equally relevant point. It too deals with patients suffering from structural brain disease, contrasting the prognostic implications of clinically dead brain stems (in patients with remnants of electroencephalographic activity) with the prognostic implications of "isoelectric" electroencephalograms (in patients with residual clinical signs of brain stem function).[2] All patients with potentially reversible causes of brain stem dysfunction (such as drugs and metabolic disturbances) were excluded in this survey. Over 1000 cases were identified that combined apnoeic coma, brain stem areflexia, and an "isoelectric" electroencephalogram. All developed asystole within days. A further 147 cases were identified with brain stem areflexia and apnoea in which there was some residual electroencephalographic activity. Again, all developed asystole within a few days. Like the first group, they also had dead brain stems. The conclusion seems to be that, irrespective of what the electroencephalogram may show, a clinically dead brain stem always heralds asystole. A further 16 well-documented case reports were found of patients who had isoelectric electroencephalograms (strictly defined) but some residual brain stem function. None of them developed asystole. Again this is not surprising: parts of their brain stems were still functioning.

The technical argument

The technical argument has centered on the fact that an intensive care unit is about the most hostile environment imaginable for trying to record "electrocerebral silence." Many electroencephalograms in these circumstances show multiple artefacts which may be bizarre and difficult to identify and locate.[3] If the nurse is wearing nylon underwear static electricity may generate false signals. Electromagnetic

disturbances from calls on the Tannoy system may also generate confusing information. But apart from artefacts, some experts have argued that it is intrinsically impossible to record a genuinely isoelectric electroencephalogram at an amplification of 2 μV/mm because this is approaching the noise level of even the most sensitive apparatus.[4] To exclude cerebral activity just over this magnitude in a noisy trace inevitably contaminated by signals several times larger is certainly a major demand not always achievable.

Anyway, does an isoelectric tracing from the scalp imply electrocerebral silence, as is often implied? What about signals generated in the depths of the sulci or by the basal cortex? Why are the advocates of the electroencephalogram not requesting traces from pharyngeal or sphenoidal electrodes? And what about attenuation of signals en route to the scalp? Even at normal voltage such attenuation may be considerable. These thoughts evoked a further limerick:

> We sat back and watched with some glee
> All these experts on death disagree
> Are we all being dull
> Or could a thick skull
> Be a cause of a flat EEG?

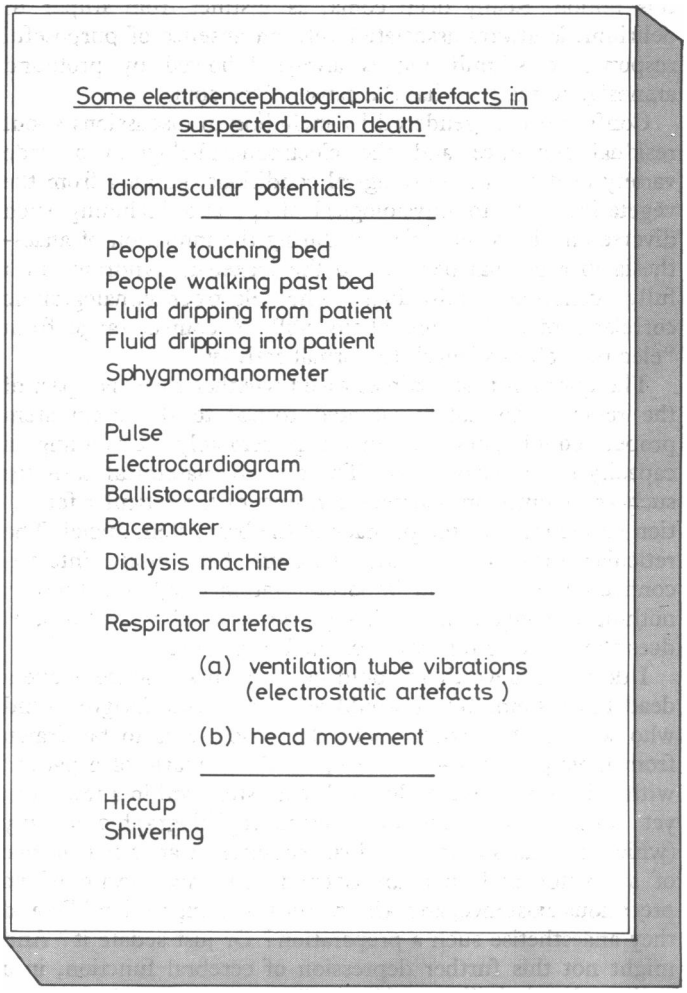
But even if the whole of the cortex could be shown to be electrically silent (which is impossible) would it mean that every cell in the brain was dead? There have been cases where thalamic probing has shown persistent neuronal discharges in the presence of an isoelectric electroencephalogram.[5–7] The electroencephalogram therefore does not test cerebral function with the rigour demanded by the concept of death of the whole brain. If those who accept this concept were logical they would have to drill burrholes and probe with depth electrodes before diagnosing a totally dead brain.

There is a final facet to the technical critique. The electroencephalogram is often said to be "objective." This is not so. In the American Collaborative Study special efforts were made to identify artefacts in the records, yet about 6% of 2256 electroencephalograms were classified as unsatisfactory because of technical difficulties.[8] Discordance between those interpreting the records was put at only 3%,[9] which is exceptionally good. There is no sharp end point, as recordable electroencephalographic activity gradually submerges into noise. These are not the hallmarks of an objective test.

The clinical argument

Those who argue that the electroencephalogram is irrelevant to establishing the presence of a dead brain stem are often misunderstood. They are not saying the electroencephalogram is irrelevant to the diagnosis of the condition causing the coma (it may be most useful, for instance, in establishing a diagnosis of hepatic encephalopathy or of herpes simplex encephalitis). Nor are they denying the prognostic value of the electroencephalogram after head injury or acute cerebral anoxia,[10] although even here there is accumulating evidence that judiciously directed clinical assessments may provide very reliable prognostic data.[11 12] There is even evidence that in patients rendered comatose after a cardiac arrest the clinical signs elicited within the first hour may indicate whether the electroencephalogram will remain isoelectric.[11]

There seems to be a difference of opinion among the advocates of the electroencephalogram about its exact purpose. Some believe it to be necessary for ascertaining that unspecified preconditions have been met. Others consider it part of the final testing, an isoelectric trace being deemed the ultimate proof that the brain is dead. Neither attitude is warranted. In patients in deep coma the electroencephalogram may generate misleading data.[13 14] To those unaware of the pitfalls it may suggest death in patients who may survive. There is a report of a patient being declared dead on the basis of a single electroencephalogram.[15] Conversely, persistent electroencephalographic activity often generates false hope in relation to "beating heart cadavers," doomed to develop asystole because their brain stems are already dead. It has been claimed that to do an electroencephalogram in the clinical context of brain stem death is "reassuring" to the relatives. If the electroencephalogram is recorded in the knowledge (which the relatives do not share) that it is non-contributory this is manipulative behaviour.



In summary, the electroencephalogram relates (inaccurately) to an unformulated (but unacceptable) concept of death. It provides answers of variable reliability to what is widely felt to be the wrong question. This should make its use questionable for anyone with any concern for intellectual clarity in this field. To the more pragmatically minded the ability of the electroencephalogram to lead to wrong practical decisions should suggest caution in its use.

The argument about residual sentience

Is there more than atavistic mysticism in the essentially untestable supposition of residual sentience in the isolated forebrain, or in cell aggregates elsewhere in the cortex or deeper structures? Clinical experience offers no support for

this notion. Really deep coma, as distinct from stupor or delirium, is always associated with an absence of purposeful response to stimuli and is always followed by profound amnesia, no matter what the cause of the coma.

Confusion is engendered by including in discussions about residual sentience and the electroencephalogram a wide variety of different neurological conditions, ranging from the vegetative state to physiological sleep, and including such diverse entities as experiences during the induction of anaesthesia in a normal person and the locked-in syndrome in a fully conscious individual. The electroencephalographic correlates of such a miscellany will, of course, range from "electrocerebral silence" to normal activity.

The question is sometimes asked whether the small part of the reticular formation situated rostral to the brain stem proper could generate anything remotely resembling a capacity for consciousness? There is no anatomical basis for such an assumption. Current concepts of the reticular formation still emphasise the primacy of the brain stem nuclei. The reticular formation of the thalamus has purely internal connections.[16] It has an important gating role,[17] but there is nothing to suggest that it has global cerebral projections, as does the reticular formation of the brain stem.

I do not believe there could be residual sentience above a dead brain stem. But I would ask those who disagree—and who want to be logical about the conclusions to be drawn from their premises—to face up to the scenario of a patient with a dead brain stem, doomed to asystole within a few days, yet showing remnants of electroencephalographic activity (which they equate with residual sentience). Can they conceive of a greater hell than an isolated sentience, aware of its precarious existence, and with no means of expression? Would they anaesthetise such a preparation? Or just sedate it? And might not this further depression of cerebral function, in a patient already in "coma dépassé," prove to be the last straw?

The problem has, of course, fascinated physiologists and philosophers for generations. With appropriate corrections of time scale it is the problem of what happens, for a few seconds, in a decapitated head. The following limerick, which could have been written by one of the *tricoteuses* sitting at the foot of the guillotine in Paris in 1793, puts the forbidden question:

We knit on, too blasées to ask it:
"Could the tetraparesis just mask it?
When the brain stem is dead
Can the cortex be said
to tick on, in the head, in the basket?"

The cultural argument

Electroencephalograms are nevertheless still widely resorted to in the USA in the diagnosis of brain death. Few people are prepared to discuss the cultural (rather than neurological) dimensions of this addiction. Our American colleagues practise in a litigious atmosphere in which "a climate of general public unease about brain death exists, partly engendered by sensational fiction."[18] For good or ill, instrumental medicine has taken giant steps forward—often evicting good clinical practice in its wake. Many American jurors have a touchingly naive faith in the supremacy of machines such as the electroencephalograph, do not realise that there is at least a 3% variance in the reading of such records,[9] and are blissfully unaware of the problems of obtaining artefact-free traces at high amplification.

Leading neurologists in the USA readily endorse these doubts about the scientific relevance of the electroencephalogram and emphasise that in the "less legally demanding" conditions of the UK "it is doubtful that the experienced physician needs the electroencephalogram to tell him that the brain is dead."[19] But, as an American colleague wrote to me, they "have to protect the young people who are educated with them against the malevolent ravages of opportunistic lawyers." They lived "in a climate where physicians have been brought to court as potential murderers for having killed an already dead patient." Physicians resorted to electroencephalograms "to save a great deal of later polemical accusation."

It was suggested in the *Panorama* TV programme on brain death (13 October 1980), and is still believed in the USA,[20] that our reluctance in the UK to use the electroencephalogram for diagnosing brain death is due to the paucity of such machines in our hospitals. Economics, it was claimed, was a consideration in formulating our code. The paucity of machines is admitted but the implication is unwarranted. As the question of economics has been raised, let me say that I believe it to be relevant to the continued advocacy of instrumental diagnosis in the USA. Vested interests should be openly declared. They rarely are, in either verbal or written discussions on the use of electroencephalography in the diagnosis of death.

Conclusions

Modern technology, in its desperate attempts to save human life, has produced an entity widely known as brain death. It has also generated a conceptual crisis: that of knowing—at the simplest, bedside level—whether a patient is alive or dead.

I have argued that the conceptual challenge can, and should, be met. We must evolve a concept of death that is in keeping with the cultural context of our age and which would in practice enable us to steer a course between "treating the putrefying body as if it were alive, and treating patients who are mentally retarded as if they were dead."[21] The recognition of a dead brain stem is the first step along such a course. In these articles I have sought to show how such a state can be identified clinically and how it relates to an overall concept of death.

The lay public, however, is not on the whole interested in physiological argument about the reticular formation, or in philosophical controversies about the nature of death. People are concerned that their kidneys should not be removed while they are comatose from treatable conditions. The UK code[22] can give the public absolute reassurance in this respect. It is scientifically sound and clinically foolproof (provided the necessary attention is given to preconditions and exclusions and provided the doctors carrying out the tests are reasonably competent and know what they are doing and why). The whole ethos subtending the code is humane. In practice it will be of help to relatives, nursing staff, and doctors who "may unintentionally find themselves caring for a biological preparation with no other human attributes than physical form."[23]

As if anticipating later developments, Shakespeare had Macbeth proclaim (act III, scene IV) that there was once a time "that when the brains were out, the man would die." The challenge today is a double one: to replace the words "would die" by the words "is dead"—and to be more specific about "the brains being out." The death of the brain stem would surely be enough.

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The graph was reproduced, by permission, from Gaches J, Calsican A, Findji F, *et al. Semaine des Hôpitaux de Paris* 1970;**46**: 1487-97, and the table, showing the prognostic significance of brain stem signs, from Pallis C. *Lancet* 1981;i:379.

A book of the ABC of Brain Stem Death, including some additional material on the neurological controversies, will be published on 24 January.

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Clinical Topics

Confirming the diagnosis of mild hypertension

R M HARTLEY, R VELEZ, R W MORRIS, M F D'SOUZA, R F HELLER

Abstract

Patients with newly found raised blood pressure are known to have lower pressures at subsequent measurements even when not treated. A study was undertaken to determine the extent to which (a) the number of follow-up measurements and (b) the duration of the intervals between them contributed to this fall in pressure. In 42 general practices 110 patients were identified as having for the first time a diastolic pressure (phase V) greater than 90 and less than 110 mm Hg. Both diastolic and systolic pressures were appreciably lower when measured at return visits when compared with the first measurement. The systolic pressure dropped appreciably in the intervals between the first and the second visits and again between the second and third visits. The diastolic pressure fell appreciably only between the first and second visits. The duration of the interval between visits was not associated with a fall in either systolic or diastolic pressure, but the number of measurements was. This pattern of fall in pressure was not affected by the patient's age or sex. From these results we conclude that patients with newly identified blood pressures that are mildly raised should be seen at two further visits before a decision about treatment is made. The timing of these follow-up visits is not crucial.

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Introduction

The results of several studies have shown that even without treatment patients who have a raised blood pressure at one visit usually have a lower pressure at follow-up visits.[1-3] This has been attributed to several factors, in particular regression to the mean and habituation to the method of measurement. In trials of treatment the placebo effect may also operate in a control group of patients who take an inert substance. Evidence from the Medical Research Council's pilot trial, however, has shown that in a control group of patients taking nothing the reduction in blood pressure equals that of a group taking a placebo.[4]

The practical importance of this fall in blood pressure without treatment is that it is necessary to measure the blood pressure more than once before identifying a patient as hypertensive and requiring treatment. It is not clear, however, how best to follow up these patients. There has been little investigation into whether the number of measurements or the time period over which they