Brainstem Death

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Key Words

Death, Brain, Brainstem, Cerebral
Evoked Potentials
Hemorrhage, Brainstem
Infarction, Brainstem
Tumor, Cerebellopontine Angle

The role of the electroencephalogram in the diagnosis of brain death is still somewhat controversial.[1-6] Although most recommendations have included the prerequisite of an isoelectric EEG, Mohandas and Chou concluded on the basis of 25 autopsied cases that "except in unusual cases EEG need not be a required procedure for the neurosurgeon in the determination of brain death. With certain exceptions the decision of brain death can be made on clinical judgment alone." The University of Minnesota criteria thus allowed for discontinuation of life support systems when there is 1) no spontaneous movement, 2) no spontaneous respiration, tested for a period of 4 minutes at a time, 3) absence of brainstem reflexes defined as: dilated and fixed pupils, absent corneal reflexes, absent cilio-spinal reflexes, absent doll's eye phenomenon, absent gag reflex, absent vestibular response to caloric stimulation, absent tonic neck reflex, 4) persistence of the findings for at least 12 hours, and 5) if the pathological process responsible for states 1-4 is deemed irreparable with presently available means.[7]

The criteria for brain death on a world-wide basis were recently summarized by Pallis, and clinical information suffices in the majority of countries surveyed.[8] The recommendations for the practice of clinical neurophysiology as proposed by the International Federation of Societies for Electroencephalography and Clinical Neurophysiology state: "In the event that there is evidence of drug intoxication or electroencephalography is not available or practicable, the patient may be declared dead on the basis of brain death (1) if all clinical evidences of brain death persist for a period of 3 days or (2) if arrest of brain circulation (absence of cerebral blood flow) is demonstrated for a period of 30 minutes."[9] It is clear, therefore, that clinical criteria may suffice to establish the presence of brain death. While there are no difficulties in this respect when patients are not on artificial life support systems, the situation becomes problematic once such measures have been instituted. They allow the extension of the process of dying over days, weeks or months with various dissociations between clinical and electroencephalographic states. Thus, we can now distinguish between two extremes, namely; neocortical or hemispheric death, also called the apallic syndrome, with isoelectric EEG but persistence of some brainstem reflexes on the one hand, and brainstem death with absent brainstem reflexes but some retention of bioelectric activity on the electroencephalogram, on the other hand.[10-12] The majority of cases with suspected brain death show various degrees of a mixture of irreversible hemispheric and brainstem damage.

A search of the literature in regard to the electroencephalogram in the presence of discrete brainstem death revealed only one detailed case report, but since this patient suffered from meningococcal meningitis there was additional cortical involvement.[12] Inasmuch as there does not appear to be a publication which documents the appearance of the EEG and cerebral evoked potentials in a patient who demonstrated the clinical picture of brain death due to discrete brainstem hemorrhage and infarction, the following case is presented.

Case Report

This 71-year-old woman was admitted to Harper-Grace Hospitals with a diagnosis of a left cerebellopontine angle tumor which had been established by CT scanning 1½ years earlier.

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The patient had at that time refused surgical intervention. A second scan in January of 1984 showed increase in tumor size, but only by May of 1984 had the symptoms of light headedness, dizziness, left sided hearing loss and veering to the left while walking, progressed to a point where she felt she could no longer function independently and therefore agreed to the operation. Upon admission to the neurosurgical service it was found that the past medical history was unremarkable except for a cholecystectomy, hysterectomy, emphysema, arthritis and allergies to thiazide, furosemide, as well as sulfa drugs.

The neurological examination showed normal mentation, horizontal nystagmus on lateral gaze, sensory neural hearing loss on the left, swaying to the left on Romberg testing and while walking. The repeat CT scan (Figure 1) showed further growth of the tumor and a transfemoral anteriogram also showed a tumor stain in the left cerebellopontine angle region. Furthermore, there was inferior displacement of the meatal loop of the inferior cerebellar artery and slight inferior displacement of the medullary portion of the posterior/inferior cerebellar artery, which suggested that the lesion was growing inferiorly causing slight displacement of the cerebellar tonsils on the left. There were no preoperative electrophysiological studies.

At operation on 5/14/84 it was noted that the tumor was quite large. It filled the entire cerebellopontine angle on the left, touching the ninth and tenth cranial nerves. The capsule was richly vascularized. Some bleeding from the petrosal vein was encountered and there was also a large vascular channel along the inferior surface of the tumor. Hemostasis was accomplished satisfactorily and the patient left the operating room in good condition. She was responsive in the ICU, but had a left facial palsy.

Later that evening the patient lapsed into unresponsiveness and respirations became shallow. An emergency CT scan showed, apart from the craniectomy, moderate hydrocephalus and bleeding in the left cerebellopontine angle area obliterating the fourth ventricle (Figure 2). It was felt that the patient could have imminent

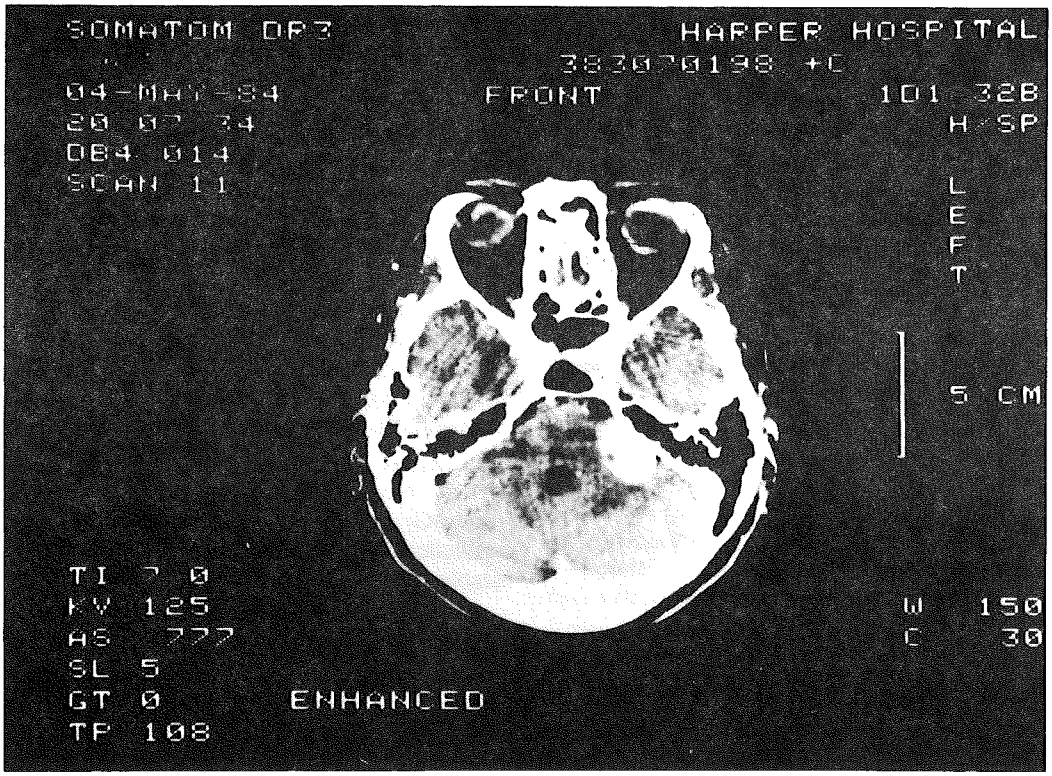

Figure 1. Preoperative CT scan showing lesion in left cerebellopontine angle region.

herniation especially of the left cerebellar tonsil. A ventriculostomy was performed immediately thereafter, intracranial pressure reduced and the patient placed on dopamine. The patient's state continued to deteriorate during the night and the clinical diagnosis of cerebral death was made in the morning. At that time she had no spontaneous respirations, pupils were fixed in mid position, there was no doll's eye phenomenon, no tonic neck reflex, no corneal or ciliospinal reflex and there was no response to caloric stimulation. Muscle tone was flaccid, there were no deep tendon reflexes, no pathological reflexes, and no spinal reflexes. The patient was totally unresponsive to painful stimuli.

An EEG was ordered to confirm the presence of brain death. The record as shown in Figure 3 revealed medium voltage theta and delta activity, some alpha frequencies and a delta focus in the left posterior head regions corresponding to the operative site. Intermittently there was also some spindle activity. The patient was therefore maintained on full life support systems but without change in the clinical condition. A repeat EEG on 5/16/84 was unchanged.

Brainstem auditory evoked potentials revealed only a low voltage Wave 1 from stimulation of right ear with a latency of 1.9 milliseconds but no further components. There was no detectable response from left ear stimulation (Figure 4). The somatosensory evoked potential from median nerve stimulation showed normal Erb's point responses bilaterally and N 11 as well as N13 were also present from the cervical spine recordings, but cortical potentials were absent (Figure 5).

On 5/18/84 another right transfemoral arteriogram was performed and although there was significant delay in carotid circulation time the capillary and venous phases were adequately documented precluding a diagnosis of brain death. The patient's clinical state persisted unchanged and the electroencephalogram actually showed some improvement (Figure 6). On 5/24/84 a visually evoked potential recording resulting from flash stimuli to either eye was

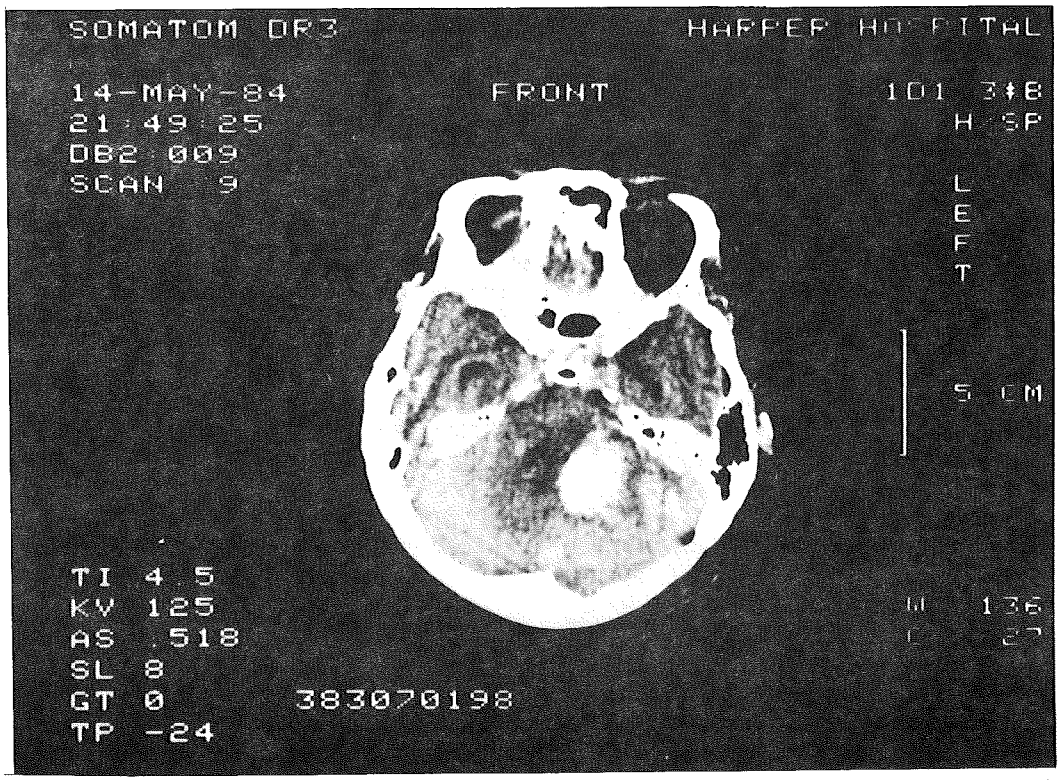

Figure 2. Postoperative CT scan showing hemorrhagic infarct in the pons with obliteration of fourth ventricle.

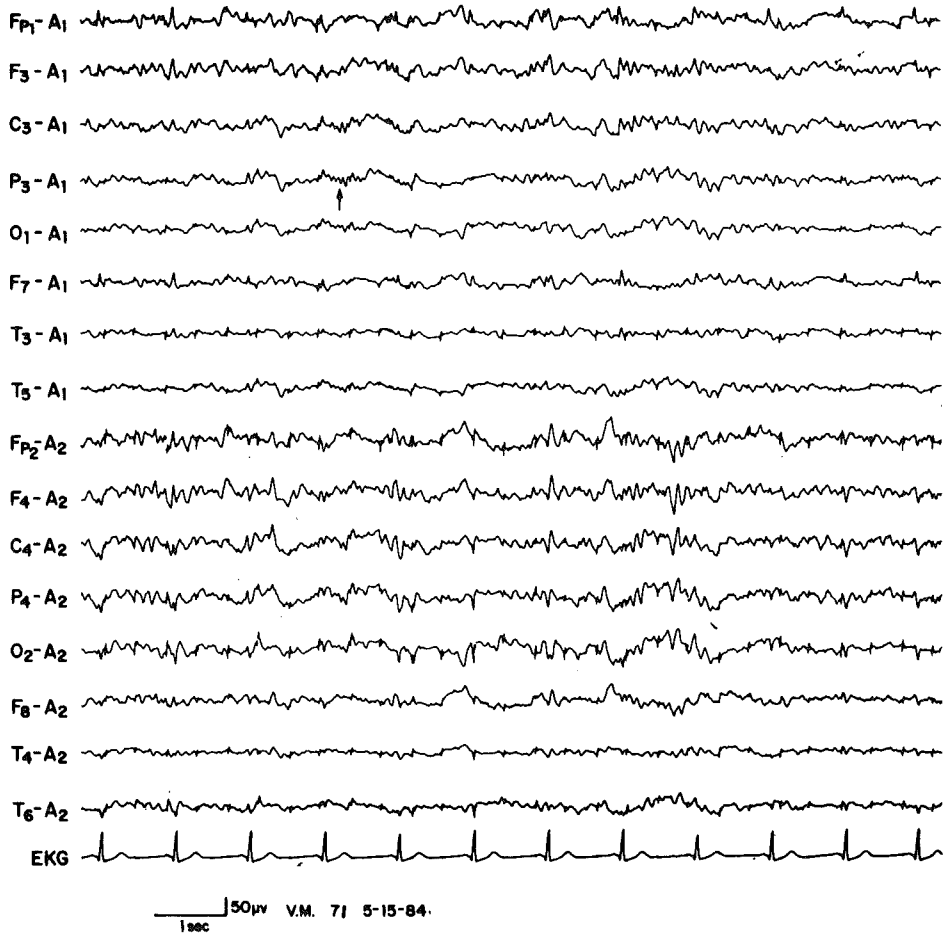

Figure 3. EEG obtained to confirm presence of brain death. Record shows medium voltage and delta and theta activity, higher in amplitude on right than left. Suggestion of sleep spindles at arrow.

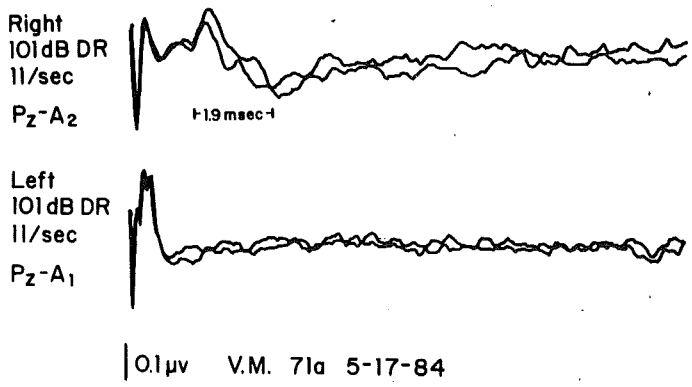

Figure 4. Brainstem auditory evoked potential recording, right-sided stimulation produced delayed Wave 1, but no other components. No evoked potential recordable from left-sided stimulation.

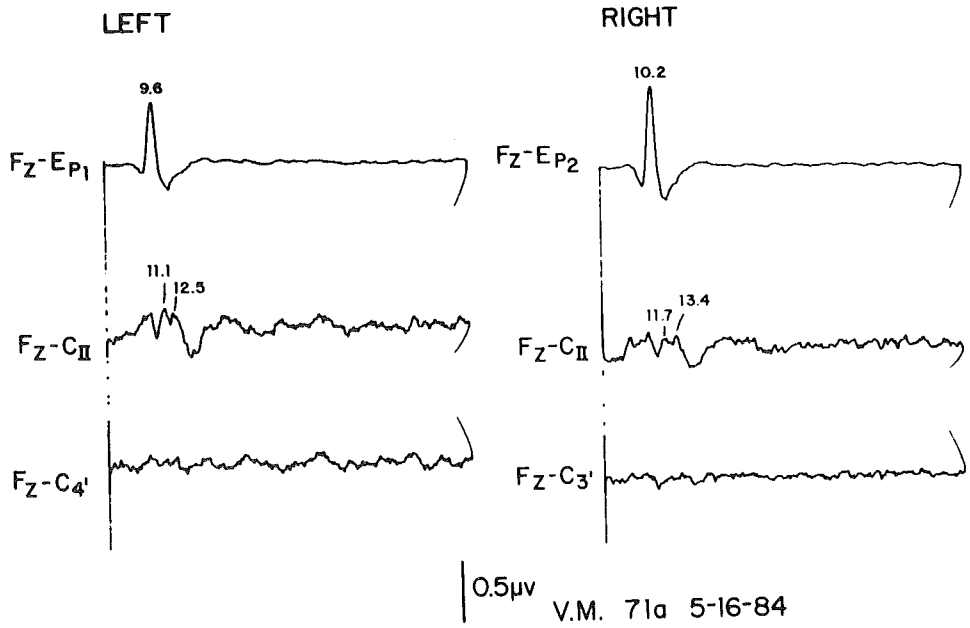

Figure 5. Somatosensory evoked potential showing Erb's point and C2 potentials at appropriate latencies, but absence of cortical response.

obtained which showed a reliable high voltage potential from the occipital regions regardless of which eye was being stimulated (Figure 7). On the same date the CT scan was repeated. It showed that the hematoma had partially resolved, but there was extensive lucency involving much of the posterior fossa more on the left, with additional edema and/or ischemia involving the brainstem up to the level of the midbrain (Figure 8).

Another EEG was not requested until 5/29/84 at which time there was some difficulty maintaining the blood pressure. The record showed only questionable cerebral electrical activity and none was obtained on 5/30/84. Monocular flash stimuli on 5/30/84 failed to produce an identifiable visual evoked potential. Life support systems were then terminated. An autopsy was requested but the family declined.

Discussion

Although the unavailability of an autopsy is regrettable, two major points can be made: 1) brainstem death is not identical with total brain death although it has a fatal prognosis, 2) in presence of life support systems, the clinical examination can only establish a diagnosis of brainstem death but cannot give information about the functional state of the cerebral hemispheres or diencephalon unless spontaneous progressive hypothermia ensues. While these points may appear self evident, they do not seem to be universally accepted. The preface to the United Kingdom code on determination of brain death reads in part: "It is agreed that permanent functional death of the brainstem constitutes brain death and that once this has occurred, further artificial support is fruitless and should be withdrawn."[13]

As for the EEG, Pallis stated recently it "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." He went on to say: "I do not believe there could be residual sentience above a dead brainstem," and he asks those who would disagree ". . . to face up to the scenario of a patient with a dead brainstem 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?"[4]

These views require comment because they assume that only negligible cortical function can remain in a patient whose brainstem is

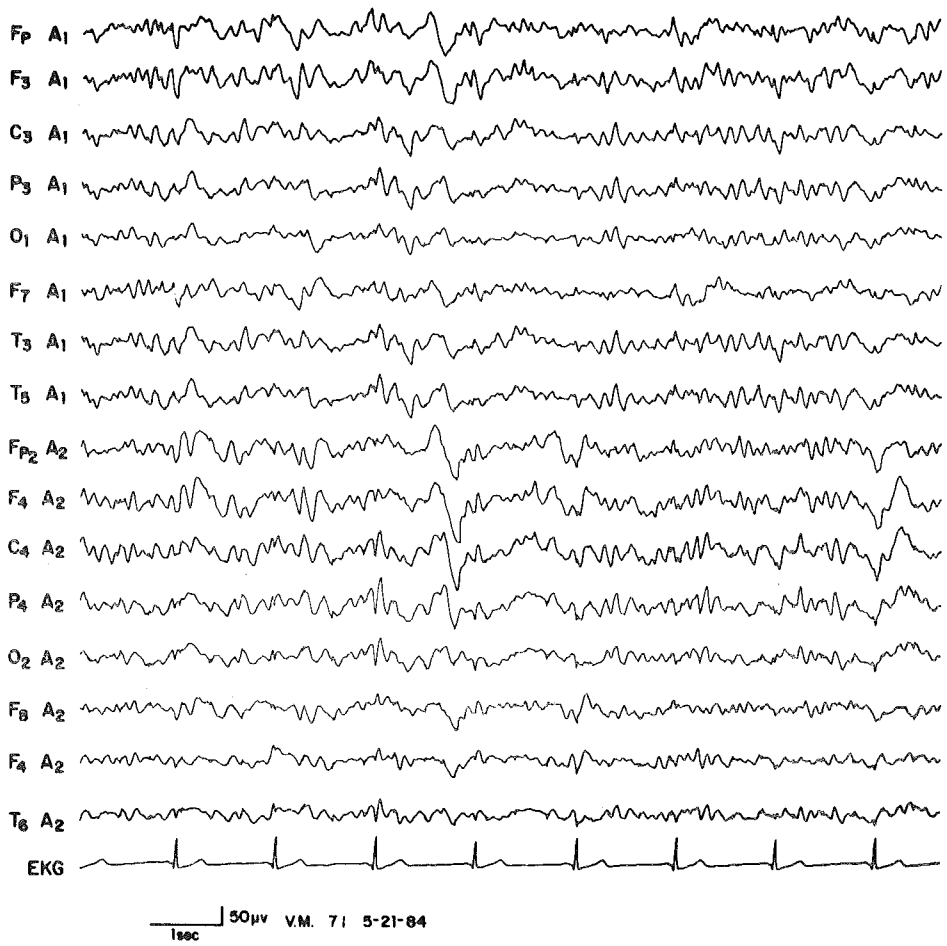

Figure 6. EEG 7 days after clinical death shows higher amplitude activity than previously with less delta activity. The record appears to reflect a state of sleep.

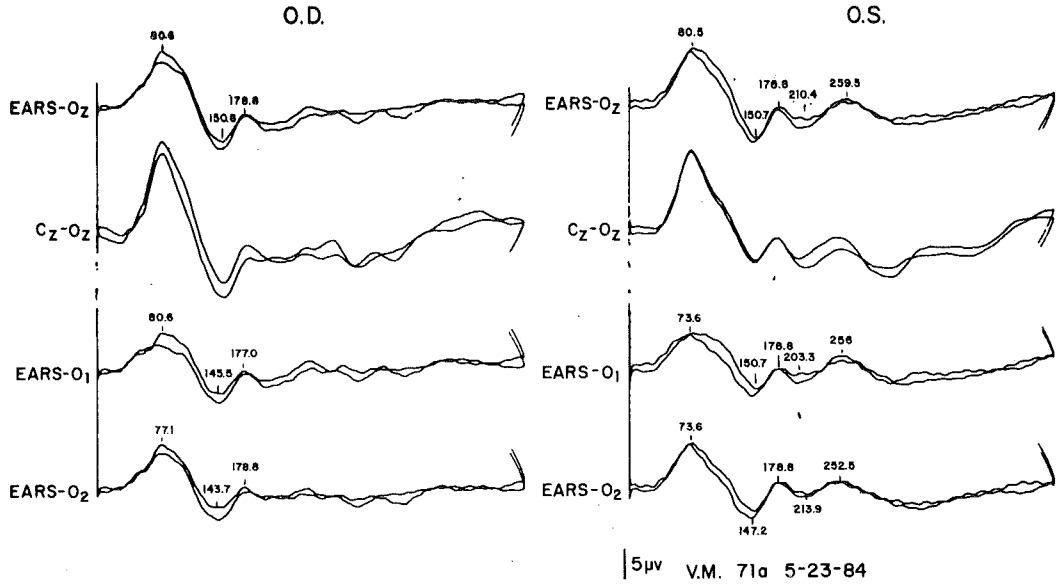

Figure 7. Flash evoked visual potentials with normal latencies but unusually high amplitudes 8 days after clinical death.

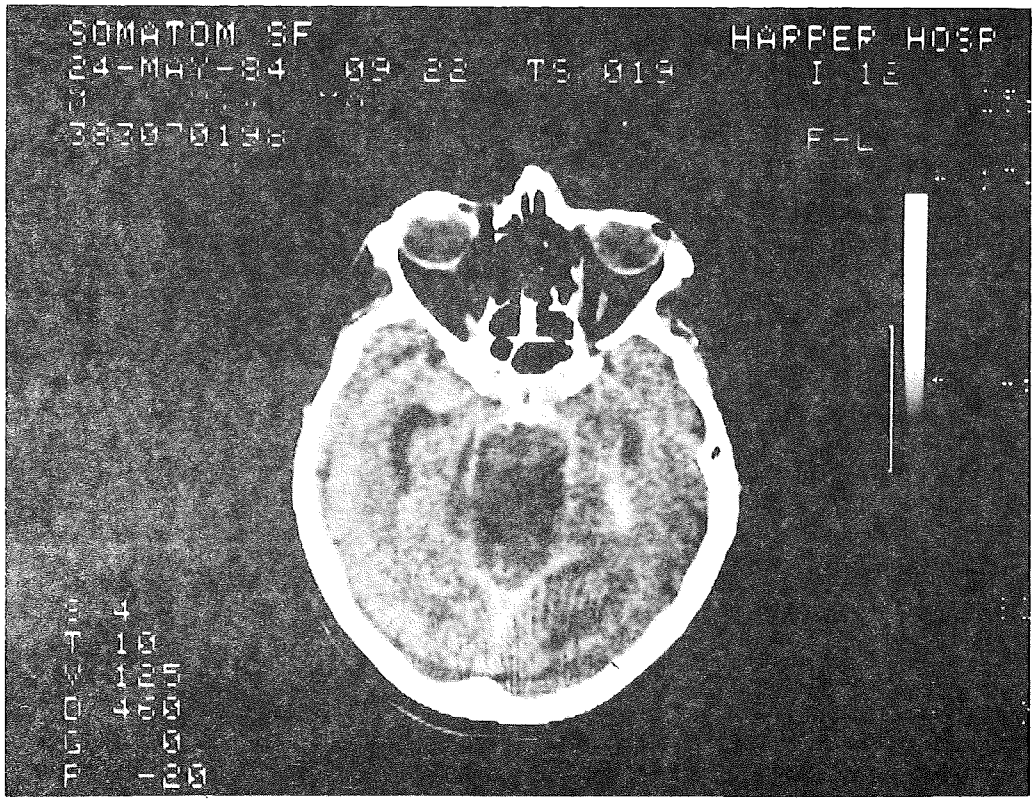

Figure 8. CT scan obtained on same day as VEP recording showing extensive lucency involving brainstem up to midbrain level.

infarcted to an extent that the person meets all the clinical criteria of brain death. As our case shows, this is not necessarily the case. We were not dealing with a remnant of electroencephalographic activity, but an electroencephalogram which when read on a "blind basis" would certainly be compatible with survival. Except for unresponsiveness to external stimuli, it was of a type one finds in stuporous or semicomatose patients and even showed suggestions of sleep spindles. It thus represented the human analogue to the cerveau isolé preparation as described originally by Bremer[14] and subsequently confirmed by Batsel.[15]

In our patient the hemispheres were deafferented from auditory and somatosensory as well as deep pain impulses all of which have to traverse the brainstem. The fact that the brainstem was nonfunctioning was demonstrated by somatosensory evoked potential recordings. These showed a brachial plexus potential and the early negativities which are regarded as originating from the cervical medullary junction, as well as the absence of an auditory brainstem potential in presence of a Wave 1, indicating activity in the auditory nerve on the right. These are the typical findings in brain dead patients.[16,17] The absence of a left ear response cannot be used in this instance because of the patient's original pathology involving the cerebellopontine angle. In addition, our patient did show, however, excellent and unusually high responsiveness of the cortex to visual stimuli. This indicated that not only retina, optic nerve and chiasm but also thalamic structures e.g., lateral geniculate, and cortex were intact. The high amplitude could be due to the absence of the desynchronizing influence of the brainstem reticular formation and/or the total absence of other stimuli arriving in the hemispheres, thus making an unusually large neuronal pool available for responding.

As to the capacity for residual sentience of the patient's hemispheres one can obviously only

speculate, but Pallis' visions of Hell need not be correct. The electroencephalographic state would suggest that if there was cognition at all it would probably have been in the realm of dream type rather than waking reality. The contents of the patient's "dreams", if there were any, might just as well have been of Heaven or ordinary terrestrial life rather than Hell, and in this context it is very useful to keep the difference between objective and subjective reality clearly in mind. This was discussed in a previous publication in regard to what has been called the near death experience.[18]

Of more practical importance is the fact that we should remain intellectually honest and not confuse present state with future outcome as the equation between brainstem death and total brain death implies. We should therefore adhere to accurate terminology as suggested by Korein[19] and keep the three definitions of cerebral death or apallic syndrome, brainstem death and brain death separate. In the first, the EEG is usually isoelectric, while it is retained in the second and always absent in the third. The survival time as measured by ultimate cessation of cardiac function may be months or years in the apallic syndrome,[11] up to 14 days in brainstem death as reported here, and up to 63 hours in brain death when strict criteria are employed.[20] These time courses refer to adults and may be significantly longer in children.

The question may well be asked whether or not the use of the electroencephalogram in patients with documented brainstem death leads to improved outcome or do life support measures merely prolong the dying process. It is virtually certain that the latter is the rule, but this appears to be an inevitable byproduct of the technology which is available in tertiary care hospitals and has to be accepted as such. In absence of ventriculostomy, brainstem compression would have persisted as a result of increased intracranial pressure, the cerebellar tonsils would have herniated and the patient suffered not just brain death but also systemic death by the morning of May 15. Decompression of the brain, stabilization of blood pressure and artificial ventilation allowed the emergence of pure brainstem death which can therefore be regarded as an iatrogenic event. All the steps leading to this state had to be taken as a result of existing standards of medical practice. To terminate life support systems in the presence of demonstrable cerebral functions would have been morally unjustified in spite of the fatal prognosis.

In considering these extremely complex problems which involve not just medical but also ethical, legal and religious considerations, it is advisable that we do not confuse quality of life with presence of life. It is clearly undesirable to merely prolong the dying process, but the physician has to adhere in these instances to institutional guidelines of the hospital and the wishes of the family.

In order to reduce the possibility of inconclusive EEG recordings, the recommendation for the practice of Clinical Neurophysiology should be followed. They state quite clearly that the EEG is to be recorded no sooner than 12 hours after the patient has been declared clincally brain dead. The proper role of the EEG is therefore as a last resort rather than first measure.

To aid the family in understanding the difference between clinical death, brain death and systemic death, it appears advisable to inform the relatives that dying is a process which can extend over varying periods of time depending on the severity of the original illness or injury and the intensity of resuscitative efforts. Instead of telling the family that the patient has entered coma or coma dépassé, it may be useful to describe the functional losses and their implications for survival as well as potential future quality of life. Once the physician is convinced that the patient is indeed no longer just comatose but dying, this information should be transmitted as such to the family who can then decide on the intensity of measures they want to have taken, and this should be reflected in the patient's chart.

Summary

A patient with the clinical picture of brain death resulting from brainstem hemorrhage and subsequent infarction is presented. The EEG showed activity similar to what has been described in the cerveau isolé animal preparations. Cortical evoked potentials were unobtainable from auditory or somatosensory stimulation, but of unusually high amplitude to flash stimuli. The point is made that a diagnosis of brain death cannot be made on clinical grounds alone when a patient is on life support systems, and the differences between cerebral death, brainstem death and brain death are discussed.

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