The reliability of the electroencephalogram (EEG) to confirm brain death remains a controversial subject. The clinical diagnosis of brain death was established when the following were found: (1) the primary cause of coma was due to an irreversible event; (2) the patient's brain stem was confirmed to be non-functional; (3) the EEG showed continuous electrical silence.

PATIENTS AND METHODS

A total of 56 cases of brain death occurred at Loyola University Medical Center, Maywood, Ill., between January 1984 and May 1986. Every patient with this diagnosis had at least one EEG recording after they fulfilled clinical criteria for brain death.

The medical records were reviewed to confirm that each patient met the clinical criteria for brain death before the EEG recording. All patients were examined by attending neurologists or neurosurgeons.

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of these patients had electrocerebral silence (ECS) on their EEGs and cerebral blood flow studies demonstrated no cerebral perfusion.

All EEG recordings were performed using 17- or 18-channel instruments. Recordings were done in accordance with the standards of the American EEG Society for recording suspected brain death. Silver-silver chloride electrodes were applied with collodion according to the International 10/20 system. Sensitivities of 2 μV/mm, double interelectrode distances, and electrode impedances between 100 and 6000 ohms were used throughout the 30-minute recordings. Electrocardiographic, electromyographic, and electrical activity from a 10,000-ohm "dummy patient" monitor were simultaneously recorded. Filter settings were 0.1 or 0.3 s and 70 Hz. Intravenous pancuronium bromide was given when excessive muscle artifact was present.

The EEG was considered without detectable cortical activity (ECS) when there was no activity above 2 μV for 30 minutes of recording at a sensitivity of 2 μV/mm. Low voltage (4 to 20 μV) activity was interpreted as of cerebral origin when noted to be irregular, aperiodic, present throughout the recording, visible in more than one montage, absent in the extracerebral channels, and not attributable to room noise, body movement, respiratory excursions, and electrocardiographic, ballistocardiographic, or muscle artifact. External interference and internal machine noise were monitored using a "dummy patient" resistor. Movement activity, movement artifact, and room noise were monitored using EMG electrodes placed on the right hand. When any doubt existed as to the origin of observed activity, the reader tended to ascribe the activity to artifact.

In 20 (50.5%) of 66 cases, radionucleotide cerebral perfusion scintigraphic studies (RCPS) were performed, using a portable gamma camera to assess the cerebral blood flow. Twenty-eight (93.3%) of the 20 cases who had an RCPS study showed no cerebral perfusion. Of the two patients who demonstrated some cerebral blood flow after they met the clinical criteria for brain death, one patient was a 7-year-old child with low-voltage EEG activity (patient 2). The other patient was a 39-year-old man who met clinical criteria for brain death 48 hours following a respiratory arrest. The EEG performed at 49 hours, and repeated at eight days, showed ECS. However, an RCPS study performed at two days, then repeated at eight days, showed minimal cerebral perfusion. Following unchanged clinical examination, mechanical ventilation was discontinued on day 8, and an autopsy demonstrated respiratory brain.

Radionucleotide cerebral perfusion scintigraphic studies were usually performed the first day the patient met the criteria for brain death. Technetium-99m pertechnetate (400 μCi per kilogram of body weight) was administered intravenously. The intracranial perfusion was assessed dynamically for 60 s, followed by a delayed static image 20 minutes later. The static image allows for activity to accumulate in the superior sagittal sinus, thereby increasing sensitivity. If both the dynamic and static images failed to demonstrate intracranial activity, the study was considered "positive," indicating a critical deficit of perfusion for at least 20 minutes.

Data were collected regarding the duration of time in hours between onset of brain death, the subsequent EEG recordings, duration of observed EEG activity, and survival.

RESULTS

Eighty EEG recordings were performed on 56 patients after brain death. Forty-eight (85.7%) of 56 patients had ECS recordings, but nine patients required more than one study before an isoelectric trace was obtained. Electroencephalographic silence was observed on the first recording in 39 patients, on the second in seven, and on the third in two. Electroencephalographic silence recordings were never obtained in eight patients, but five died before the test could be repeated despite maximum ventilatory assistance. Artifact produced equivocal recordings in seven (12.5%) of 56 patients.

All 56 patients died without any recovery of neurologic function. Mechanical ventilatory support systems were actively discontinued in 43 (76.3%) patients between two and 246 hours (mean, 38.9 hours) following brain death. Spontaneous terminal cardiac asystole developed despite mechanical ventilation in 18 (26.2%) patients between eight and 340 hours (mean, 60.3 hours) following brain death.

The patients ranged in age from 15 months to 82 years, with a mean age of 45.3 years. The primary diagnoses of the 56 patients are summarized in Table 1. Electroencephalographic activity following brain death was observed in 11 (19.6%) patients. The clinicopathologic, radiographic, and electroencephalographic findings of these 11 patients are summarized in Table 2. Three recognizable patterns of EEG activity were observed: low voltage activity, sleep-like activity, and alpha-like activity.

Low-Voltage Activity

Nine (16.1%) of the 56 brain-dead patients had monotonous and unreactive low-voltage (4 to 20 μV) theta or beta activity recorded as long as 72 hours after the clinical diagnosis (Fig 1). These patients (patients 1 through 8 and 11) are summarized in Table 2.

The patients ranged in age from 2 to 71 years, with a mean age of 45.6 years. The cause of brain death was head injury in four patients, hypoxic-ischemic encephalopathy in three, and spontaneous intracerebral hemorrhage in one. Head injury was complicated by intracranial hemorrhage in two, cerebral edema in one, and both in one.

Survival time ranged from 13 to 168 hours (mean, 61.9 hours). Four patients (patients 1, 2, 6, and 7) had ECS recorded on their second EEG, which was performed 17 to 28 hours after the first recording. Four patients (patients 3, 5, 8, and 11) died before an ECS recording was obtained. However, two of these patients (patients 3 and 11) had two recordings each. One patient (patient 4) required a third EEG, because the second was equivocal due to excessive artifact.

Radionucleotide cerebral perfusion scintigraphic studies were performed on six of the nine patients. No cerebral perfusion was found in five of six patients consistent with the clinical diagnosis. Neuropathologic studies performed on one patient (patient 3) showed total ischemic neuronal changes (pallor, clumped hypochromatic nuclei, eosinophilic-stained cytoplasm) involving pyramidal neurons throughout all layers of the cerebral cortex, basal ganglia, brain stem, and cerebellum. Pathologic findings were consistent with global hypoxic encephalopathy involving the entire cortex, basal ganglia, brain stem, and cerebellum.

Sleep-Like Activity

Two (3.6%) patients, both of whom had ischemic infarctions of the entire brain stem, demonstrated EEG activity resembling physiologic sleep (Fig 2) for as long as 168 hours after clinical
Table 2.—Summary of Clinicopathologic Findings of 11 Patients With EEG Activity After Brain Death*

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age</th>
<th>Primary Cause</th>
<th>EEG Findings</th>
<th>EEG Activity Observed at No. Hours After Brain Death</th>
<th>ECS Developed After Brain Death</th>
<th>Survival After Brain Death</th>
<th>Nuclear CBF Scan</th>
<th>Neuropathologic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
<td>HI</td>
<td>Low-voltage 4 to 6 μV, 6-8-Hz activity</td>
<td>72</td>
<td>96</td>
<td>168</td>
<td>No flow</td>
<td>ND</td>
</tr>
<tr>
<td>2</td>
<td>7</td>
<td>CRA</td>
<td>Low-voltage 6 to 10 μV, 6-8-Hz activity</td>
<td>66</td>
<td>75</td>
<td>76</td>
<td>Good</td>
<td>ND</td>
</tr>
<tr>
<td>3</td>
<td>47</td>
<td>CRA</td>
<td>Low-voltage 4 to 10 μV beta activity</td>
<td>2 and 41</td>
<td>Died before</td>
<td>75</td>
<td>ND</td>
<td>Diffuse hypoxic changes of entire brain (cerebral cortex, brain stem, and cerebellum)</td>
</tr>
<tr>
<td>4</td>
<td>35</td>
<td>ICH</td>
<td>Low-voltage 4 to 10 μV theta and beta mixed, 10 to 20 μV delta activity</td>
<td>32</td>
<td>77</td>
<td>78</td>
<td>No flow</td>
<td>ND</td>
</tr>
<tr>
<td>5</td>
<td>61</td>
<td>CRA</td>
<td>Low-voltage 5 to 10 μV beta activity</td>
<td>6</td>
<td>Died before</td>
<td>13</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>6</td>
<td>64</td>
<td>HI</td>
<td>Low-voltage 4 to 10 μV, 5 to 6 Hz theta activity</td>
<td>7</td>
<td>31</td>
<td>33</td>
<td>No flow</td>
<td>ND</td>
</tr>
<tr>
<td>7</td>
<td>57</td>
<td>HI</td>
<td>Low-voltage 4 to 8 μV beta activity</td>
<td>2</td>
<td>Died before</td>
<td>23</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>8</td>
<td>61</td>
<td>ICH</td>
<td>Sleep-like activity of synchronous 30 to 40 μV theta and delta slowing with 60 to 80 μV, 8 to 10 Hz spindle-like activity</td>
<td>7</td>
<td>29</td>
<td>55</td>
<td>ND</td>
<td>Ischemic necrosis of entire brain stem and cerebellum; relative sparing of cerebral hemispheres</td>
</tr>
<tr>
<td>9</td>
<td>71</td>
<td>BSI</td>
<td>Sleep-like activity of 25 to 50 μV theta and delta mixed with 60 to 80 μV, 10 to 12 Hz spindle-like activity</td>
<td>8, 56, and 168</td>
<td>Died before</td>
<td>340</td>
<td>ND</td>
<td>Ischemic necrosis of entire brain stem and cerebellum with preservation of the cerebral hemispheres</td>
</tr>
<tr>
<td>10</td>
<td>83</td>
<td>BSI</td>
<td>Sleep-like activity of 25 to 50 μV theta and delta mixed with 60 to 80 μV, 10 to 12 Hz spindle-like activity</td>
<td>3 and 22</td>
<td>Died before</td>
<td>59</td>
<td>No flow</td>
<td>ND</td>
</tr>
</tbody>
</table>

*EEG indicates electroencephalographic; ECS, electrocerebral silence; CBF, cerebral blood flow; HI, head injury; CRA, cardiac and/or respiratory arrest; ICH, intracerebral hemorrhage; BSI, brain-stem infarction; and ND, not done. Time is expressed in hours.
†Second EEG artifact at 53 hours.

Examinations showed results consistent with brain death. Cranial computed tomographic scans demonstrated large bilateral hypodense lesions involving the brain stem, cerebellar hemispheres, and in one patient, the left thalamus and portions of the occipital lobes.

Sleep-like activity was recorded at eight, 56, and 168 hours after brain death in one patient. The presence of EEG activity prompted continued mechanical ventilation until a terminal asystole developed 14 days after brain death. The second patient had sleep-like activity recorded seven hours after brain death, but ECS was observed on the second EEG, which was performed at 29 hours, and mechanical ventilatory support was discontinued at 55 hours.

Postmortem neuropathologic studies obtained on both patients showed striking similarities. There was extensive ischemic necrosis of the entire brain stem and cerebellum characterized by areas of marked softening, collapse, and liquefaction with obliteration of the normal cytoarchitecture. Ischemic-cell changes of extensive neuronal and glial cell loss, edematous vacuolization with eosinophilic-stained cytoplasm, hypocromatic nuclei, and elongated pyramidal cells were noted throughout the entire brain stem and cerebellum. A reactive microglial proliferation was seen, suggesting that an antemortem ischemic infarction of the posterior fossa struc-
Fig 1.—Electroencephalogram recorded 32 hours after brain death in a 35-year-old man. Note low-voltage, 4 to 10 μV, 5- to 7-Hz theta activity intermixed with lower-voltage 13- to 14-Hz beta activity and scattered 10 to 20 μV, 2.5- to 3-Hz delta slowing.

Fig 2.—Sleep-like electroencephalographic activity recorded 56 hours after brain death in a 63-year-old man with brain-stem infarction. Note medium voltage 20 to 50 μV, 4- to 5-Hz theta and 2- to 3-Hz delta activity interrupted by frequent paroxysmal bursts of bilaterally synchronous, medium-to-high-voltage 60 to 80 μV spindle-like potentials that resemble stage II sleep. No reactivity was noted.

Fig 3.—Alpha-like electroencephalographic activity recorded three hours after brain death in a 32-year-old woman. Monotonous, unreactive, anterolateral predominant, medium-voltage 9- to 12-Hz alpha-like activity was seen throughout the recording.
<table>
<thead>
<tr>
<th>Source, y</th>
<th>Patient No./ Age, y</th>
<th>EEG Activity</th>
<th>EEG Activity Observed at</th>
<th>Survival Time After Brain Death, d</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levy-Alcover and Babinet, 1969</td>
<td>1/26</td>
<td>Very-low-voltage theta</td>
<td>7 h</td>
<td>44 h</td>
<td>Intracerebral hemorrhage</td>
</tr>
<tr>
<td>2/4</td>
<td>Low-voltage theta</td>
<td>9 h</td>
<td>15 h</td>
<td>Poisoning</td>
<td></td>
</tr>
<tr>
<td>3/45</td>
<td>Low-voltage theta</td>
<td>10 h</td>
<td>15 h</td>
<td>Intracerebral hemorrhage</td>
<td></td>
</tr>
<tr>
<td>4/44</td>
<td>Very-low-voltage theta</td>
<td>14 h</td>
<td>27 h</td>
<td>Head injury</td>
<td></td>
</tr>
<tr>
<td>5/46</td>
<td>7 to 10 Hz theta</td>
<td>28 h</td>
<td>53 h</td>
<td>Poisoning</td>
<td></td>
</tr>
<tr>
<td>6/35</td>
<td>Bursts of theta</td>
<td>48 h</td>
<td>5</td>
<td>Cardiopulmonary arrest</td>
<td></td>
</tr>
<tr>
<td>7/27</td>
<td>5 to 6-Hz theta</td>
<td>60 h</td>
<td>57 h</td>
<td>Intracerebral hemorrhage</td>
<td></td>
</tr>
<tr>
<td>8/12</td>
<td>Low-voltage 7-Hz theta</td>
<td>6 d</td>
<td>6</td>
<td>Cardiopulmonary arrest</td>
<td></td>
</tr>
<tr>
<td>Mohandas and Chou, 1971</td>
<td>9/7</td>
<td>Very-low-voltage fast</td>
<td>2 d</td>
<td>4</td>
<td>Head injury</td>
</tr>
<tr>
<td>10/37</td>
<td>Very-low-voltage fast</td>
<td>2 d</td>
<td>3</td>
<td>Brain tumor</td>
<td></td>
</tr>
<tr>
<td>11/3</td>
<td>Very-low-voltage fast in occipital areas</td>
<td>?</td>
<td>3</td>
<td>Meningitis</td>
<td></td>
</tr>
<tr>
<td>12/21</td>
<td>Low-voltage fast over the right hemisphere only</td>
<td>?</td>
<td>1</td>
<td>Head injury</td>
<td></td>
</tr>
<tr>
<td>13/17</td>
<td>Very-low-voltage</td>
<td>?</td>
<td>1</td>
<td>Head injury</td>
<td></td>
</tr>
<tr>
<td>14/12</td>
<td>Very-low-voltage</td>
<td>5 d</td>
<td>6</td>
<td>Head injury</td>
<td></td>
</tr>
<tr>
<td>Deliannis et al., 1975</td>
<td>15/21</td>
<td>Low-voltage 1 to 2-Hz delta mixed with theta and alpha</td>
<td>13 d</td>
<td>18</td>
<td>Meningitis</td>
</tr>
<tr>
<td>Ashwal et al., 1977</td>
<td>16/neonate (32 wk)</td>
<td>Low-voltage beta over right centrotemporal area</td>
<td>5 d</td>
<td>?</td>
<td>Asphyxia</td>
</tr>
<tr>
<td>Reppaport et al., 1978</td>
<td>17/42</td>
<td>Sporadic frontotemporal activity</td>
<td>?</td>
<td>1</td>
<td>Head injury</td>
</tr>
<tr>
<td>Ashwal et al., 1977</td>
<td>18/neonate</td>
<td>Low-voltage delta</td>
<td>12 d</td>
<td>12</td>
<td>Meningitis</td>
</tr>
<tr>
<td>19/2 mo</td>
<td>Irregular low-voltage delta and theta</td>
<td>9 d</td>
<td>32</td>
<td>Meningitis</td>
<td></td>
</tr>
<tr>
<td>20/4 mo</td>
<td>Low-voltage delta</td>
<td>9 d</td>
<td>13</td>
<td>Subarachnoid hemorrhage</td>
<td></td>
</tr>
<tr>
<td>21/neonate</td>
<td>Low-voltage delta and theta</td>
<td>10 d</td>
<td>15</td>
<td>Asphyxia</td>
<td></td>
</tr>
<tr>
<td>22/30 mo</td>
<td>Low-voltage delta</td>
<td>2 d</td>
<td>2</td>
<td>Near drowning</td>
<td></td>
</tr>
<tr>
<td>Rodin et al., 1985</td>
<td>23/71</td>
<td>Sleep-like potentials medium-voltage theta and delta, some alpha delta slowing in posterior regions</td>
<td>14 d</td>
<td>16</td>
<td>Brain-stem hemorrhagic infarction</td>
</tr>
<tr>
<td>Forbert et al., 1966</td>
<td>24/48</td>
<td>40 to 60 µV slow alpha intermixed with delta</td>
<td>30 h</td>
<td>?</td>
<td>Brain-stem hemorrhage</td>
</tr>
</tbody>
</table>

*Question mark indicates data not available or uncertain.*

...tures had occurred prior to the hypoxic changes.

In contrast, the cerebral cortex and white matter were normal, except for small patches of neuronal dropout. The occipital and thalamic regions in one patient had a minimal number of ischemic neurons and early antemortem reactive microglial proliferation. In summary, the pathologic findings were consistent with massive ischemic infarction and necrosis of the entire brain stem and cerebellum, with relative sparing of the cerebrum and basal ganglia.

**Alpha-Like Activity**

After cardiorespiratory arrest, one patient demonstrated diffuse and unreactive 25 to 40 µV, 9 to 12 Hz alpha-like EEG activity three hours after brain death (Fig 3). A second EEG performed at 22 hours showed low-voltage activity. An RCP study done the first day showed no cerebral perfusion. Mechanical ventilation was discontinued 59 hours after brain death. Permission for autopsy was denied.

**COMMENT**

This study of 56 consecutive cases of brain death reports residual electrocerebral activity in 11 (19.6%) patients as long as 168 hours after the onset of the clinical diagnosis. All 11 patients with EEG activity had fulfilled stringent clinical criteria for brain death (ie, deep coma, absent cephalic reflexes, and apnea). Five of the patients had cerebral perfusion studies performed the same day, which showed no cerebral perfusion. The presence of EEG activity, despite clinical examinations entirely consistent with irreversible brain death, resulted in continuation of supportive care and delayed the final declaration of death for hours, days, and in one case, weeks, without altering the final fatal outcome. The recording of EEG activity in brain-dead patients has been previously observed, but only as scattered case reports. The Cerebral Survival Collaborative Study, while presenting important data regarding irreversible coma, did...
not analyze the outcome of patients with coma, apnea, and absent cephale reflexes who had EEG activity. Table 3 summarizes 24 previously reported cases, where sufficient clinical and electroencephalographic details were described. Our study is a systematic effort to examine how often EEG activity can be observed when electroencephalography is used as a routine confirmatory test of brain death.

Electroencephalographic activity observed following brain death, especially when only a minimal low voltage, could be discounted as artifactual in origin. Although we cannot entirely rule out this possibility, we believed that the observed electrical activity was cerebral in origin because it was irregular, demonstrated variability in amplitude, frequency, and distribution during and between serial recordings, and was not present in the electromyogram, "dummy patient," or other extracerebral recording channels.

The presence of residual EEG activity when radionuclide cerebral blood-flow studies demonstrated "no cerebral perfusion" created a striking dissociation. The RCPS studies were performed on the same day EEG activity was observed in six of the 11 patients, and five showed no cerebral perfusion. Such a dissociation was previously reported by Ashwal and Schneider in five children in whom low-voltage EEG activity was observed, despite clinical examinations, RCPS, and cerebral angiographic studies consistent with brain death. A negative radionuclide total studies only implies cerebral perfusion is less than 24% of the normal predicted blood flow. Thus, some minimal cerebral blood flow may persist, which while insufficient for visualization, could adequately perfuse the areas of cortex responsible for the observed EEG activity. Leptomeningeal collaterals from the external carotid circulation could be another possible source of limited perfusion.

Three patterns of electrocerebral activity were observed in patients after brain death. The most frequently observed pattern, both in our series and in other reported cases, was low-voltage theta or beta activity. When voltage activity was recorded in nine (16.1%) of our patients as long as 72 hours after the clinical diagnosis. Several authors noted residual EEG activity was more common when the EEG was performed within 24 hours of brain death. However, only seven of our 11 patients had their first EEG showing activity performed less than 24 hours after brain death. Five of these patients had RCPS studies performed the same day, which showed no perfusion. The presence of conflicting results of the confirmatory investigations persuaded most clinicians to delay the discontinuation of mechanical ventilatory support until an isoelectric tracing was obtained. Gachas et al noted that residual EEG activity, especially when of low voltage, rarely persists beyond 48 hours. However, ECS does not always develop. Thirteen cases, including three of ours, have been reported where EEG activity persisted between two and 14 days after brain death.

A more uncommon pattern of EEG activity resembling stage II physiological sleep was observed in two patients for as long as 168 hours after the clinical diagnosis. Both patients had extensive brain-stem ischemic infarctions with preservation of the cerebral hemispheres. Two single case reports of sleep-like EEG activity after brain death were found. Roizen et al described intermittent, spindle-like activity with medium voltage theta, delta, and alpha activity present for as long as four days after brain death due to pontine hemorrhage with secondary brain-stem infarction. Breger noted a case following pontine hemorrhage, where the EEG showed diffuse slowing and 7 to 8 Hz spindle-like potentials, despite a clinical examination consistent with brain death.

Sleep-like EEG activity has been noted in deep coma, but not clinically brain dead, patients following brain-stem lesions or head injury. Chatran et al characterized the activity as 12 to 14-Hz spindle-like potentials and vertex sharp waves, with intermittent periods of diffuse high-voltage delta and low-voltage 5- to 8-Hz activity. He thought such activity may have represented a temporary derangement of the midbrain reticular formation.

The observed diffuse and synchronous bursts of slow waves and spindle-like potentials occasionally alternating with periods of low-voltage desynchronized activity resembled EEG patterns seen in the classic cerebrum isolated animal models of Bremer and Bates and after experimental transection of the brain stem at the level of the mesencephalon. The presence of sleep-like activity and the neuropathologic findings of ischemic necrosis of the entire brain stem suggest these two cases represented "brain-stem death." Brain-stem death can be regarded as a naturally occurring model of cerebrum isolated. When EEG activity resembling sleep is seen in patients meeting clinical criteria for brain death, brain-stem death should be suspected.

The most unusual EEG activity was alpha-like activity observed in one patient three hours after the clinical criteria for brain death were met and 48 hours following a cardiorespiratory arrest. An RCPS study performed on the same day showed no cerebral perfusion. A second EEG performed on the next day showed low-voltage activity. The diffusely distributed, anteriorly predominating, unreactive, sinusoidal 8- to 12-Hz alpha-like activity resembled the "alpha-coma pattern," described in comatose patients after cardiorespiratory arrest, acute vascular, or traumatic brain-stem lesions, accidental electrocution, and drug overdose.

Gurvitch et al studied synchronized alpha activity in dogs after global cerebral ischemia that resembles the human alpha-coma pattern. Using a series of destructive lesions, they showed that the amygdaloid nuclei appeared to be the primary generator, and subcortical structures, especially the thalamus, functioned as secondary sources for the generation and generalization of the activity. Experimental removal of the overlying cortex did not obliterate the subcortically generated synchronized alpha activity.

Neuropathologic findings, when reported in cases of alpha-coma due to postanoxic coma, demonstrated either diffuse hypoxic changes involving the entire brain or selective lesions of the cerebral cortex with sparing of the subcortical and brain-stem structures.

Can patients who fulfill all clinical criteria of brain death and yet have EEG activity be considered brain dead? The answer depends on "accepted medical standards" of what constitutes brain death. The United Kingdom Code states that brain death is present when a "permanent functional death of the entire brain stem" has occurred. The presence or absence of electrocerebral activity is not considered relevant to the question of viability of the brain stem. The United States Uniform Determination of Death Act defines brain death as an irreversible "cessation of all functions of the entire brain, including the brain stem." However, clearly stated in the United States guidelines is that the determination
of the cessation of "all functions of the brain" is restricted to only those functions "that are clinically ascertainable." Moreover, they reaffirm the contention of others that the determination of brain death can be based solely on clinical grounds, and the EEG is not required but may prove "useful" when "objective confirmation is desirable." Only when an EEG is performed and shows activity, is the question of the presence of this activity an issue. Conversely, Brierley et al. described the occurrence of isoelectric EEGs in two patients in whom there was massive bilateral cerebral hemispheric destruction, but with relatively preserved brain-stem function.

Even if one requires cessation of all functions of the entire brain to fulfill a diagnosis of brain death, this does not imply, nor require, the death of each and every neuron. Brain death is present when a critical number of neurons have been irreversibly damaged, such that all the integrative neuronal capacities of the brain are lost. The presence of EEG activity after clinically determined brain death demonstrates that the clinical criteria of brain death may be fulfilled before the death of every cell within the brain has occurred. However, residual bioelectric activity, possibly derived from patchy islands of electrophysiologically active cortical or subcortical brain tissue, need not be regarded as reflecting integrated neuronal function.

The relatively frequent occurrence of EEG activity after brain death would suggest reliance on the EEG to confirm brain death may be unwarranted. The presence of EEG activity in patients who are clinically brain dead does not change the final mortal outcome. The advocacy of the EEG as a confirmatory test of brain death may be of questionable value.

References