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AUDITORY BRAIN-STEM RESPONSES IN BRAIN DEATH

by ARNOLD STARR

(*From the Division of Neurology, University of California, Irvine College of Medicine, Irvine, California*)

THE extension of the legal definition of death to include individuals with irreversible and massive destruction of the brain is relatively recent. The development reflects, in part, the perfection of techniques for maintaining cardiovascular, pulmonary, and renal functions in moribund patients. The techniques are of great value in patients with transient toxic and metabolic abnormalities as they allow the preservation of vital functions until recovery occurs. In contrast, the continued maintenance of vital functions in patients with extensive and irreversible brain damage seems futile since recovery can never occur. A major concern has been the definition of criteria by which a patient can be considered to have suffered irreversible and massive destruction of the brain compatible with brain death. An Ad Hoc Committee of the Harvard Medical School (1968) suggested that the definition should include: (1) 'unresponsivity' manifested by an absence of movements; (2) 'no movements of breathing'; (3) 'no reflexes'; and (4) a 'flat electroencephalogram.' All these criteria were to be present on 2 separate determinations separated by at least twenty-four hours. The criteria have been modified with the recognition that certain spinal reflexes may persist in irreversible brain death (Ivan, 1973) and that the electroencephalogram (EEG) may not be an entirely satisfactory indicator of irreversible brain death (*Lancet*, 1974).

There is need to expand the criteria of brain death with objective measures which are accurate, safe, and of reasonable expense. For instance, the measurement of cerebral blood flow has been used to define the distinction between reversible and irreversible loss of brain function (Bradac and Simon, 1974). However, the blood flow techniques employed require complex apparatus and the arterial injection of contrast material can produce complications. Recently Braunstein, Korein, Kricheff, Corey and Chase (1973) have described a method for measuring cerebral blood flow at the bedside, and Uematsu and Walker (1974) have reported that pulsations from the mid-line reflection in the echoencephalogram are indicative of cerebral blood flow.

Electrophysiological measures can also provide evidence of brain functions which may not be apparent on clinical examination. The evoked cortical responses

to sensory signals as recorded with scalp electrodes and measured at the bedside have been tested along with the EEG and the clinical findings in attempting to diagnose brain death (Trojaborg and Jorgensen, 1973). The correlation was not absolute with instances of patients in coma due to toxic and metabolic abnormalities, in whom an isoelectric EEG and absence of evoked cortical responses were only temporary and the patients subsequently recovered. The EEG and evoked cortical responses presumably measure cortical activity and provide only indirect information as to the functional integrity of the brain-stem. It is the loss of action of the brain-stem in controlling cardiac and respiratory functions that must also be considered crucial in the current definition of brain death.

This report examines a relatively new measure of brain-stem function, that of auditory brain-stem responses, in patients being examined for brain death. Auditory brain-stem responses as recorded with scalp electrodes and computer averaging techniques are the far-field reflection of electrical events generated in the auditory pathway in its course from the cochlea to cortex (Jewett and Williston, 1971). The technique is an objective measure of the functioning of one of the sensory pathways traversing the brain-stem and provides evidence of brain-stem capacities unavailable with current methods.

MATERIALS AND METHODS

Studies were made of 27 patients fulfilling the clinical criteria of brain death due to various causes over a twenty-one month period (September 1973–June 1975, Table 1). Clinical findings always included: (1) absence of spontaneous respiration monitored by visual inspection; (2) absence of motor responses to visual or auditory signals; (3) absence of motor responses of facial or skeletal muscles to cutaneous stimulation; (4) absence of decerebrate or decorticate postures; (5) absence of cranial nerve function to clinical examination including pupillary responses to light and to nociceptive stimulation of the skin of the neck (ciliospinal reflex), eye movements during oculocephalic reflex testing and cold caloric stimulation of the labyrinth, corneal reflex and gag response. Oculocardiac reflexes were not measured. The EEG was isoelectric in all patients.

TABLE 1. AUDITORY BRAIN-STEM RESPONSES AS A FUNCTION OF THE ÆTIOLOGY OF BRAIN DEATH. N = 27

Ætiology	*Responses absent*	*Only Wave I detected*
Anoxia	8	6
Trauma	4	4
Hepatic failure	2	1
Brain hæmorrhage	2	0
	16	11

Auditory brain-stem responses were recorded at the bedside with portable signal generating and computer averaging equipment (Starr and Achor, 1975). Click signals were presented monaurally and binaurally through cushion type earphones (Sharpe HA 600) at intensities up to 75 dB HL (hearing level referred to normals). The clicks were generated by passing 0·2 ms square wave pulses of alternating

polarity through an amplifier and an attenuator before activating the earphones. Alternating the polarity of the click signals is an effective means of cancelling the cochlear microphonic potentials in the averaging process. Brain-stem responses were recorded from conventional EEG disc electrodes (vertex-ear lobe, i.e. Cz to A_1 or A_2), amplified 1×10^5 at a band-pass of 0·1–3·0 kHz, and averaged over a 10·24 ms time base (40 μs sampling time, 256 samples) with a small digital computer. A total of 2048 clicks were presented at 10/s for each averaged response. A duplicate average was made at each signal intensity to define reproducibility of results.

Conventional cortical evoked responses to visual, somatosensory and auditory signals were also recorded in many of the patients. For visual and auditory responses records were made between vertex (Cz) and right ear lobe (A_2). For somatosensory responses additional electrodes were placed over the parietal areas bilaterally (P_3, P_4) and records made between the parietal and contralateral ear lobes: P_4–A_1 when the left side was stimulated and P_3–A_2 when the right side was stimulated. The amplifier gain was reduced to 1×10^4 and the band-pass set to 0·1 to 100 Hz. Auditory signals were 65 dB HL binaural clicks. Visual signals were light flashes presented by a photic stimulator (Grass Co.) placed 20 cm from the subject's closed eyes with the intensity set at maximum. Somatosensory signals were square wave electric currents, 0·1 ms in duration, applied percutaneously to the median nerve at the wrist through surface electrodes. The intensity of the stimulus was adjusted to be just subthreshold for eliciting a visible contraction of the thenar muscle. The stimulation rate was 1/s for all cortical evoked responses and 256 signals comprised each average. The computer time base was set for 512 ms (2 ms sampling time). A duplicate average was made to define reproducibility of results.

Clinical neurological examinations and EEG recordings were performed close to the time at which auditory brain-stem and cortical evoked responses were recorded.

Auditory brain-stem responses obtained from a normal individual (fig. 1) consist of 7 components (designated I to VII) during the initial 10 ms following a click signal. Table 2 is summarized from previously published material (Starr and Achor, 1975) and contains the means and standard deviation of latency and amplitude measures at 3 signal intensities (75, 55, 35 dB HL) from 6 normal subjects.

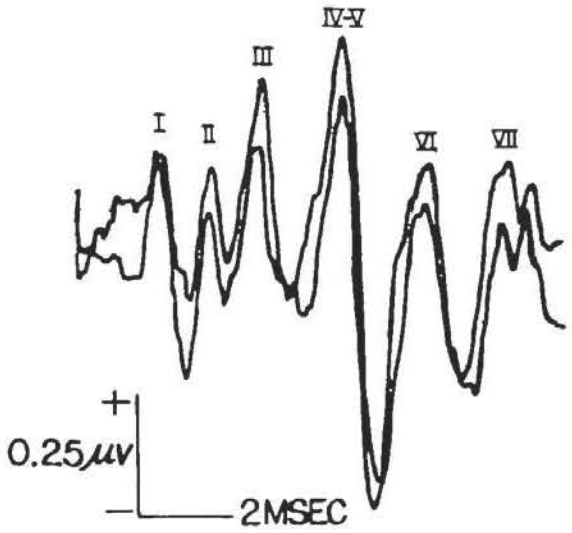

FIG. 1. Auditory brain-stem responses from a normal individual showing 7 components designated by roman numerals. The two sets of averages were made to 2048 monaural clicks at 65 dB HL presented at a rate of 10/s. Recordings were made between vertex and the ear receiving the acoustic signal. Positivity at the vertex is up in this and all subsequent figures.

TABLE 2. AUDITORY BRAIN-STEM RESPONSES (6 NORMAL SUBJECTS), dB HL

Latency (ms)	75		55		35	
Wave I	Mean 1·4	SD ±0·2	Mean 1·8	SD ±0·2	Mean 2·7	SD *
II	2·6	±0·2	3·0	±0·3	3·6	±0·4
III	3·7	±0·2	3·9	±0·2	4·7	±0·2
IV	4·6	±0·2	5·0	±0·3	5·8	±0·3
IV–V	5·2	±0·2	5·6	±0·2	6·4	±0·2
V	5·4	±0·2	5·8	±0·2	6·6	±0·2
VI	6·9	±0·3	7·5	±0·2	8·4	±0·2
VII	8·7	±0·2	9·0	±0·3	—	—
Amplitude (μV)						
Wave I	0·20	±0·09	0·09	±0·04	0·08	*
II	0·16	±0·06	0·07	±0·04	0·06	±0·02
III	0·18	±0·05	0·12	±0·03	0·10	±0·02
IV	0·10	±0·01	0·09	±0·03	0·03	±0·00
IV–V	0·34	±0·08	0·25	±0·09	0·18	±0·04
V	0·25	±0·04	0·18	±0·09	0·17	±0·04
VI	0·12	±0·07	0·10	±0·06	0·04	±0·02
VII	0·09	±0·03	0·05	±0·01	—	—

— No response detected.

* Responses in only one or two subjects making the SD measure unreliable.

SD Standard Deviation.

The latencies of the individual components shorten and their amplitudes increase as signal intensity is raised. Components IV and V, which are often fused, can be detected close to hearing threshold and have the largest amplitude from threshold to 55–65 dB HL. At 75 dB HL, Wave I may become the largest component of the response. The amplitude and latency of the brain-stem components are independent of arousal and attention (Picton, Hillyard, Krauz and Galambos, 1974). I have also recorded these responses from patients under general anæsthesia and found them essentially unaltered from responses recorded when the patient was awake. Thus, changes in the state of awareness or arousal as profound as being unconscious from anæsthetics do not significantly alter auditory brain-stem responses.

RESULTS

Auditory brain-stem responses derived from patients fulfilling the clinical criteria of brain death deviated significantly from the normal pattern (Table 2 and fig. 2): responses were either absent (16 out of 27 patients) or consisted of only an initial component, Wave I (11 out of 27 patients). When Wave-I was present it was elicited by stimulation of either ear in 5 of the 11 patients but by stimulation of only one ear in the other 6 subjects. In one patient, Wave I was noted to disappear abruptly, coincident with profound hypotension and bradycardia several minutes before cardiac arrest. When Wave I was present it was always prolonged in latency (1·8–2·1 ms at 75 dB HL) compared to the normal values (1·4±0·2 ms at 75 dB HL).

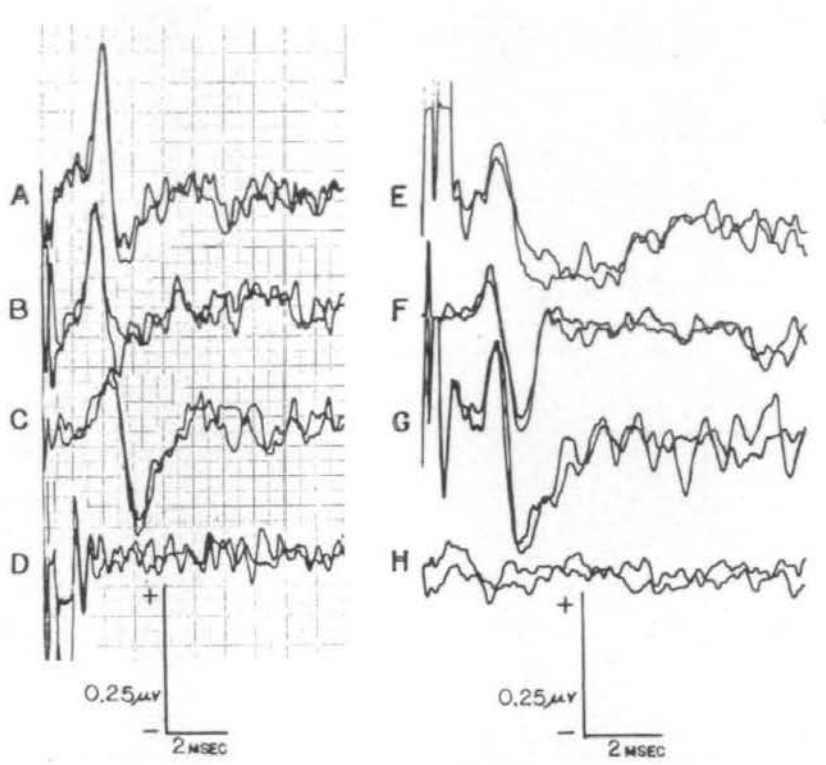

FIG. 2. Two types of auditory brain-stem responses recorded from patients fulfilling the criteria of brain death. The top three tracings of each column (A, B, C, E, F, G) are the first type and show the presence of only an initial component (Wave I) at approximately 2·0 ms. In the second type there are no components evident (D, H). The click intensity used in this figure were all 75 dB HL. The deflections occurring at the onset of the response represent a recording artifact of the voltage applied to earphone.

In 3 patients the amplitude of Wave I varied considerably between repeated measures. In the patient shown in fig. 3, tested sequentially over a three-hour period, Wave I was initially 350 nV (fig. 3A), fifteen minutes later the response was 560 nV (fig. 3B) and three hours later the response had increased further to 750 nV (fig. 3C) without any latency shift. There is a certain amount of variation encountered when recording low level (nanovolt) signals due to amplifier noise, patient-generated electrical events such as cardiac or skeletal muscle potentials and the inadvertent recording of electrical signals from other equipment. The contribution of amplifier noise in our system tested with a 0·5 μv calibration signal was ± 50 nV. Skeletal muscle potentials were not a factor in the patients in this study who were comatose without muscle tone in the neck or face. The 3 patients who were tested several times during a single day showed fluctuations in amplitude between 300 and 500 nV. We have tested several normal subjects repeatedly within a single day and noted fluctuations in the amplitude of Wave I amounting to a maximum of 150 nV.

In all the patients fulfilling the criteria of brain death, cortical evoked responses to somatosensory and auditory signals were difficult to define. Visual signals evoked only low amplitude, short latency components of the electroretinogram

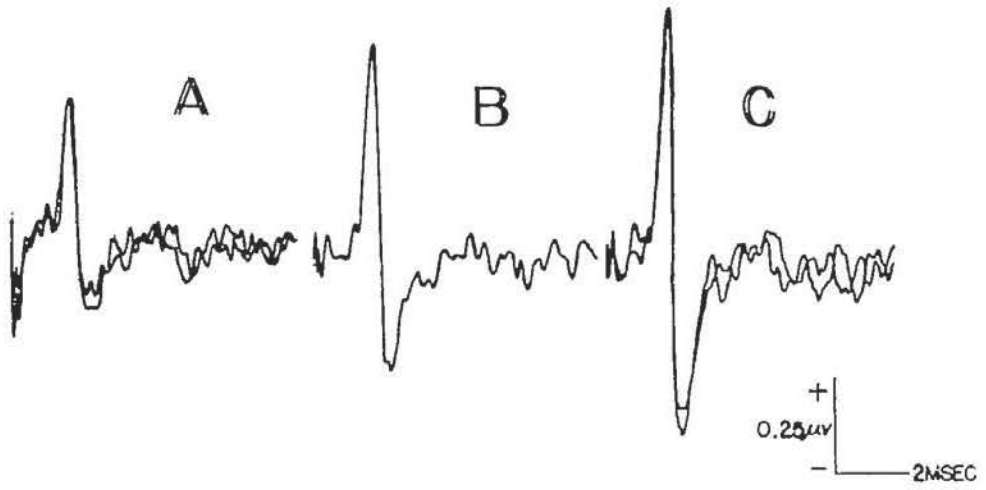

FIG. 3. Auditory brain-stem responses to monaural clicks at 75 dB HL (*re* normal) from a patient over a three-hour period showing a twofold increase of the amplitude of Wave I without an appreciable latency shift. A, control response, B, fifteen minutes later, C, three hours after A.

(ERG) during the initial 50–75 ms following the flash, as defined by Trojaborg and Jorgensen (1973) without any of the longer latency components of central nervous system origin found in normal individuals.

Serial observations were made in one patient over a thirteen-day period following an anoxic episode as he progressed from coma with evidence of preserved brain and brain-stem functions to a state in which the criteria of brain death were met. The evolution of abnormalities in the auditory brain-stem responses and their relationship to the clinical and EEG findings were particularly well documented (fig. 4 and Tables 3 and 4).

The patient was a 19-year-old man admitted on May 14, 1974, in coma. He had been a resident of a nursing home for a neurological disorder manifested by slurred speech and spasticity thought to be due to cerebral palsy. On the day of admission, he aspirated and became unconscious. He was intubated at the nursing home and artificially respired. In the hospital emergency room he was unresponsive, without spontaneous respirations and with a pulse of 30. The blood pH was 7·07. He was resuscitated with intravenous bicarbonate and adrenaline. Within thirty minutes the blood pressure was 110/60, the pulse was 100, and spontaneous respirations resumed several hours later. The clinical course was marked by generalized and myoclonic seizures on the first two hospital days. Thereafter there was a gradual decrease in spontaneous and evoked motor activities, cranial nerve functions and spontaneous respirations. The patient died on the fourteenth day after admission. The pertinent clinical features and the results of the EEGs and of auditory brain-stem and cortical evoked responses during admission are listed in Tables 3 and 4.

Auditory brain-stem responses to monaural left-sided click signals were of normal amplitude and latency on the fourth hospital day at a time the patient was in coma with preserved brain-stem reflexes. The EEG on the preceding day showed diffuse delta rhythms with only occasional spike activity. On the seventh hospital day, when the patient had lost pupillary, corneal and oculocephalic reflexes auditory brain-stem responses were abnormal with an increase in the latencies of all the components but a decrease in amplitude of only the IV–V complex. The extent of the latency shift increased progressively

TABLE 3. LATENCY AND AMPLITUDE OF AUDITORY BRAIN-STEM RESPONSE COMPONENTS FOLLOWING AN ANOXIC EPISODE

Latency (ms)	I	II	III	IV–V	VI	VII
Day 4	1·6	2·8	3·8	5·2	7·0	9·0
Day 7 am	1·8	3·5	4·9	7·5	—	—
pm	1·8	3·3	4·5	7·0	—	—
Day 8 am	1·9	3·5	4·9	8·0	—	—
pm	2·0	3·5	4·9	8·2	—	—
Day 10	2·2	—	—	—	—	—
Day 11	2·3	—	—	—	—	—
Amplitude (nV)						
Day 4	425	50	200	475	—	—
Day 7 am	550	50	150	150	—	—
pm	650	100	175	150	—	—
Day 8 am	500	100	150	75	—	—
pm	450	50	50	100	—	—
Day 10	250	—	—	—	—	—
Day 11	250	—	—	—	—	—

— Not detected. Click intensity, 65 dB HL.

TABLE 4. SUMMARY OF CLINICAL AND LABORATORY MEASURES FOLLOWING AN ANOXIC EPISODE

	Spontaneous respiration	*Corneal reflex*	*Pupillary light reflex*	*Oculocephalic reflex*	*Motor behaviour*	*EEG*	*Auditory brain-stem response*	*Cortical evoked response*
Day 1	−	+	+	+	Myoclonic and generalized seizures	NT	NT	NT
Day 2	+	−	+	+	Myoclonic and generalized seizures	Poly-spikes and suppression	NT	NT
Day 3	+	+	+	+	No seizures. Withdraws to pain	Delta	NT	NT
Day 4	+	+	+	+	Withdraws to pain	NT	Normal	Intact visual, only early components to auditory and somatosensory
Day 5	+	+	−	+	Decerebrate posturing	NT	NT	NT
Day 6	+	+/−	−	−	Decerebrate posture	Low voltage delta	NT	NT
Day 7	+	−	−	−	Decerebrate posture only to pain	Low voltage delta	Abnormal. Waves IV and V	Absent
Day 8	−	−	−	−	Decerebrate posture only to pain	Isoelectric	Components prolonged in latency Waves IV and V absent	Absent
Day 9	−	−	−	−	Decerebrate posture only to pain	Isoelectric	NT	NT
Day 10	−	−	−	−	No movement	Isoelectric	Only Wave I present	Absent
Day 11	−	−	−	−	No movement	NT	Only Wave I present	Absent
Day 12	−	−	−	−	No movement	NT	NT	NT

NT = not tested

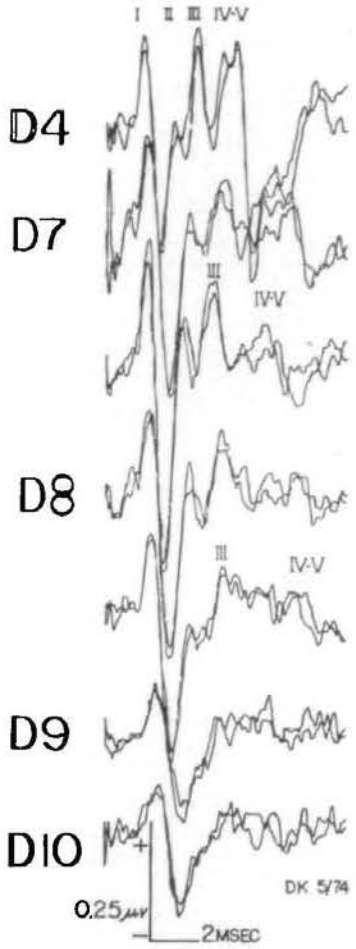

FIG. 4. Auditory brain-stem response to monaural clicks at 65 dB HL (*re* normal) in a patient following an anoxic episode. The designations on the left, D4, D7 etc., refer to the hospital day; on days 7 and 8 the two traces represent recordings taken in the morning and afternoon respectively. Note that on day 4 (D4) all the components were of normal amplitude and latency. Subsequently there was a decrease in the amplitude and prolongation of latency on the various components (*see* Table 3).

with each successive response component. Thus the latency difference between day 4 and day 7 was 0·2 ms for Wave I, 0·7 ms for Wave II, 0·9 ms for Wave III and 2·3 ms for the IV–V complex. The time difference between Waves I and the IV–V complex is a measure of central conduction along the auditory pathway. This increased from 3·6 ms on the fourth hospital day (normal range is 3·5–4·2 ms) to 5·2–5·7 ms on the seventh hospital day. On the eighth hospital day the response latencies were prolonged further and the IV–V complex was of extremely low amplitude. The EEG contained only low voltage delta activity. On the tenth and eleventh days, when the patient's clinical examination fulfilled the criteria of brain death, auditory brain-stem responses were restricted to Wave I. The EEGs were isoelectric.

Cortical evoked responses to light flash were present in normal form on the fourth hospital day. In contrast, auditory cortical responses consisted of only an early negative and positive component in the first 100 ms. Somatosensory cortical responses also consisted of only early positive and negative components in the first 75 ms. On subsequent days, cortical evoked responses were absent except for the very early components of visual evoked responses thought to be the reflection of the ERG.

In this patient the gradual decrement of auditory brain-stem responses paralleled the clinical deterioration of the patient to the state of brain death. There were 3 other patients in whom auditory brain-stem responses were monitored for several days after an anoxic episode leading eventually to a clinical state compatible with brain death. The response latencies listed in Table 5 show the phenomena of an increase in latency and a loss of components as the interval after anoxia lengthened.

TABLE 5. LATENCY OF AUDITORY BRAIN-STEM RESPONSE COMPONENTS (ms) FOLLOWING ANOXIC EPISODE

	Wave					
	I	II	III	IV–V	VI	VII
Patient 2						
Day 1	1·9	3·2	4·4	5·5	ND	ND
Day 5	1·9	3·3	4·3	6·5	ND	ND
Day 12	3·5	4·0	ND	8·5	ND	ND
Day 13*	ND	ND	ND	ND	ND	ND
Patient 3						
Day 1	1·6	2·6	3·5	5·2	ND	ND
Day 2	1·7	3·0	3·7	5·7	ND	ND
Day 4*	ND	ND	ND	ND	ND	ND
Patient 4						
Day 1	1·9	ND	ND	6·0	ND	ND
Day 2*	2·1	ND	ND	ND	ND	ND

ND Not detected. * Clinical criteria of brain death. Click intensity, 75 dB HL.

DISCUSSION

Auditory brain-stem responses are an objective measure of the functioning of the cochlea and central auditory pathway. The response components are thought to be the far-field reflection of electrical events generated deep within the brain at the various nuclei and tracts comprising the auditory pathway. These potentials spread passively through brain tissue and can be detected by scalp electrodes after sufficient amplification and computer processing. Far-field recording is also the basis of the electrocardiogram which employs electrodes on the chest wall and limbs remote from the heart to detect cardiac muscle potentials.

Our information is incomplete as to the precise locus within the brain of the generators for the various components of the auditory brain-stem response. Some preliminary interpretations can be derived from (1) the effects of central nervous system lesions on auditory brain-stem responses in both man (Starr and Achor, 1975) and experimental animals (Lev and Sohmer, 1972; Buchwald and Huang, 1975) and (2) the temporal coincidence of electrical responses recorded from structures deep within the brain-stem with the components of the scalp derived

far-field auditory brain-stem response (Jewett and Williston, 1971). Wave I is believed to be due to eighth nerve activity since it is coincident in time with the initial negative component of the cochlear nerve response. Waves II and III persist after midbrain lesions indicating that their generators are located in the medullary portions of the auditory pathway. Waves IV and V are presumed to originate in the lateral lemniscus and/or inferior colliculus since these components are lost following midbrain lesions. The site of the generators of Waves VI and VII are uncertain.

The results from the present study indicate that auditory brain-stem responses can provide information as to the functional integrity of the auditory pathway in patients being examined for brain death. When the clinical criteria of brain death were fulfilled, auditory brain-stem response was either absent or consisted of just the initial component (Wave I). Moreover, when auditory brain-stem responses were measured serially in 4 patients following an acute anoxic episode, they provided graphic evidence of the gradual dissolution of brain-stem function in a rostrocaudal direction. In the period close to the anoxic episode, all components of the response were present and of normal amplitude and latency. Subsequently, the later components (IV and V) decreased in amplitude at a time when the early components (I, II and III) were normal. Finally, Wave I alone remained but was prolonged in latency and decreased in amplitude. The mechanisms permitting persistence of eighth nerve activity (Wave I) at a time when the other components of the auditory pathway are not functional may reflect different susceptibilities of receptor and central neurons to anoxic insult. There is a parallel finding in the visual system in which the components of the ERG are present at a time when the cortical components evoked by light flash are absent (Trojaborg and Jorgensen, 1973).

Neuropathological examination of the brain-stem auditory pathway was made on 3 of the patients. A detailed description of the changes encountered are to be published elsewhere as part of a study detailing the distribution of brain-stem pathology with abnormalities of auditory brain-stem responses (Starr and Hamilton, 1976). The findings from the 3 patients following anoxic insult included coagulative necrosis of the neuropil, swelling and pyknosis of neurons, and vacuolization of the grey and white matter in a patchy manner throughout the brain-stem, including portions of the fibre tracts and nuclei of the auditory pathway.

It is important to stress that auditory brain-stem responses may not be detected in patients who are alert without any clinical suspicion of brain death. Absence of response is characteristic of (1) *deaf* subjects (hearing loss greater than 75 dB) and of (2) patients whose face and neck are tense, resulting in muscle potentials that mask the detection of the auditory brain-stem responses. Moreover, in patients with demyelinating diseases of the brain-stem, brain-stem infarcts or

brain-stem tumours, Waves I and II may be the only components of the response detected (Starr and Achor, 1975). Though some of these latter patients were neurologically quite disabled and even in coma, their condition did not raise a suspicion of brain death. Thus, auditory brain-stem responses are relevant for the assessment of brain death *only* when the patient's neurological condition and acoustic function are also taken into consideration.

Auditory brain-stem responses have been of particular assistance in evaluating patients in whom a clinical question of 'brain death' was raised without adequate history as to the predisposing cause. In some of these patients coma was due to massive drug overdoses. Under this circumstance auditory brain-stem responses were normal at a time when the patients were without spontaneous respirations and when even oculocephalic, corneal and pupillary reflexes were absent (Starr and Achor, 1975). In our experience the EEG in these patients are not isoelectric, though isoelectric recordings have been reported previously in individuals with drug overdose and anoxia who subsequently recovered (Jorgensen, 1974). The finding of normal auditory brain-stem responses in this last group of patients immediately resolved the suspicion of 'brain death' in a favourable way.

Auditory brain-stem responses must be evaluated in the context of the patient's clinical examination, laboratory findings and EEG. Deafness, pre-existing brain-stem lesions or technical artifacts can result in the failure to detect auditory brain-stem responses or the definition of only the initial components. The judicious use of auditory brain-stem responses can provide additional information to the clinician attempting to resolve the question of brain death. The responses are an objective measure of one component of brain-stem function which can be applied at the bedside to promptly provide data on the functional integrity of the auditory pathway in its course through the brain-stem.

SUMMARY

Auditory brain-stem responses were measured by far-field recording techniques in 27 patients fulfilling the criteria of brain death. The responses were either absent or consisted of the presence of just the initial component (Wave I). Wave I, when present, was of normal amplitude but prolonged in latency. Four patients were followed over several days from a state of coma with evidence of preserved brain-stem and cerebral functions to a clinical state compatible with brain death. Auditory brain-stem responses were initially intact and then showed a decrease in amplitude and a prolongation of latency of the later components until finally Wave I was alone. Auditory brain-stem responses are an objective measure of one of the sensory pathways traversing the brain-stem and can be used to evaluate the functional states of the brain-stem in patients in whom the question of brain death has been raised.

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