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Auditory Brain Stem Response Spectral Content in Comatose Head-Injured Patients*

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ABSTRACT

Auditory brain stem response (ABR) spectral content was analyzed for 25 normal subjects and 70 comatose, severely head-injured subjects. The normal ABR spectrum was characterized by energy peaks in three main regions: 0 to 200 Hz, 500 to 600 Hz and 900 to 1000 Hz. Head-injured subjects, in contrast, showed less overall ABR spectral energy. Even head-injured subjects with normal ABR interwave latency values had reduced spectral energy in comparison to the normal group. Among these subjects, there were differences in spectral content for subjects with good neurologic outcome versus those who died within 2 weeks post injury. Finally, head-injured subjects with abnormal interwave latency intervals typically showed distinctive spectral patterns. This subject group demonstrated energy peaks in frequency regions which, for the other groups, were void of energy peaks. Rationale for further study of ABR spectral content is offered.

The auditory brain stem response (ABR) is a complex electrophysiologic waveform typically recorded in the time domain. Analysis of ABR data usually involves determination of the absolute latency and amplitude of major wave components, and often relative latency or amplitude measures such as the wave I–V latency interval or the wave V/I amplitude ratio [see Hall (1) for review]. By means of spectral analysis techniques, it is now also possible with commercially available instrumentation to calculate the frequency composition of an ABR waveform. That is, ABR amplitude is described in the frequency domain rather than the customary time domain. ABR spectral analysis is usually done with fast Fourier transformation which deconvolutes a complex waveform into its frequency components (2, 3).

There is a modest literature on the spectral content of the ABR in persons with normal peripheral and central nervous system (CNS) status (4–11). Reported data on the ABR spectrum are based on normative studies with a cumulative total of less than 20 subjects (5–10). There is general agreement that the ABR consists of a relatively high energy, low-frequency component (150 Hz and below), a component in the 500 to 600 Hz region, and a relatively high-frequency component in the 900 to 1100 Hz region, and that spectral energy is minimal above 2000 Hz. Less clear is the relationship of ABR spectral characteristics to the individual wave components recorded in the time domain, e.g., waves I, III, and V. Kevanishvili and Aphonchenko (7), for example, concluded from digital filtering and power spectral analysis techniques that the major energy contributions of the ABR waves were 400 to 1000 Hz for waves I and II, 100 to 900 Hz for wave III, and 100 to 500 Hz for waves IV through VI. Using similar techniques, Suzuki et al (10) supported these findings. Boston (5) studied ABR spectrum with digital filtering techniques. He attributed the high-frequency spectral component (900 to 1100 Hz) to the early ABR waves (I, II, III), the midfrequency component (around 500 Hz) to wave V, and the low-frequency energy to the slow wave upon which the ABR is superimposed. However, the relationship among ABR waves and spectral characteristics remains tenuous and somewhat controversial (6). There are, to our knowledge, no reports of ABR spectral amplitude for a series of patients with documented CNS pathology with ABR waveforms recorded reliably in the time domain.

The purpose of the present study was to assess the effect of acute CNS trauma on the spectral content of the ABR.

METHOD

ABR data were analyzed for 95 subjects. There were 25 normal-hearing subjects with no history of neurologic dysfunction and 70 acute, severely head-injured patients who were comatose at the time of ABR measurement. The head-injured subject group was subdivided on the basis of ABR findings and outcome. Twenty-five patients yielded an ABR with normal absolute and interwave latency values (within 2 standard deviations of our normative mean value) and had good long-term neurologic outcome, as defined by a grade VII or VIII on the Ranchos Los Amigos Hospital Scale (12) at 3 months postinjury.

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Twenty-seven head-injured patients also had a normal acute ABR by these criteria but died within 2 weeks after injury. The third head injury group consisted of 18 patients with ABR wave I–V latency abnormalities bilaterally. All subjects in this group died within 2 weeks after the injury. The distribution and characteristics of the subject groups are displayed in Table 1. Hearing sensitivity in the normal group was 20 dB HL or better or octave frequencies of 250 through 8000 Hz. Hearing status could not be assessed for the head-injured subjects, due to coma, but all showed a well-formed and reliably recorded ABR. As shown in Table 2, there were no significant differences in wave I latency or amplitude among subject groups.

ABRs for all subjects were measured with commercially available equipment (Nicolet CA-1000). ABR data were stored on floppy disks (Nicolet DC-2000) for later latency, amplitude, and spectral analysis (Pathfinder II, Version 4.1 software). Normal subjects were tested in a quiet hospital room while resting in a supine position. Head-injured subjects were tested at bedside in an intensive care unit. Stimuli were clicks of 0.1 msec duration presented monaurally via TDH-39 earphones and MX-41/AR cushions at a rate of 21.1/sec. An ABR waveform was averaged from 2000 repetitions of the stimulus. Stimulus intensity level was 75 dB (re: normal click HL) for the normal group and 85 dB for the head-injured group. Analysis time period was 15 msec. Neural activity was detected with gold disk electrodes located on the high forehead (voltage positive), the earlobe ipsilateral to the stimulus (voltage negative), and the low forehead (ground). Interelectrode impedance was always less than 5000 ohms. The neural response was filtered before averaging at 30 to 3000 Hz (12 dB/octave). Following data collection, ABR spectral amplitude (in microvolts) was analyzed over the range of 65 to 2000 Hz. Resolution of frequency analysis was 65 Hz.

RESULTS

ABR latency and amplitude data for subject groups are compared in Table 2. As noted above, there were no group differences in wave I latency or amplitude. Likewise, all latency parameters were equivalent for the normal group and the two head-injury groups with a normal ABR. Waves III and V amplitudes, and the V/I amplitude ratio, were significantly reduced in all head-injured groups, in comparison to the normal group and, by subject group definition, the interwave latencies (I–III, III–V, I–V) were significantly prolonged in the final head-injury group.

A representative normal ABR time domain waveform and its spectrum are illustrated in Figure 1. Low-frequency (less than 200 Hz) energy predominates in the spectrum, but the characteristic normal spectral peaks in the 500 to 600 Hz and 900 to 1000 Hz regions are also observed. ABR time domain and spectral data are similarly illustrated for all subject groups in Figure 2. Each waveform is the grand average of individual waveforms for the first 10 consecutive subjects in the respective group. Spectral data for the groups are displayed in detail in Table 3. In comparison to normal findings, there is less energy in the low frequency region for all head-injury groups. Even the head-injured patients with normal ABR latencies and good outcome have significantly lower peak energy than the normal subjects. Both head injury subject groups with a normal ABR by latency parameters have the characteristic energy peak in the 500 Hz and 900 to 1000 Hz regions. However, for the head-injury group dying within 2 weeks, there is significantly less energy at 500 Hz, in comparison to the normal group and to the other head-injury group with equivalent ABR latency values. ABR spectral distribution for the final head injury group (I-V latency abnormality) is uniquely different. As seen in Figure 2, there appears to be an energy peak at approximately 400 Hz, and then a bi-peaked region of energy in the 700 to 1000 Hz range.

Case Report

A 21-year-old female was involved in a motor vehicle accident and sustained a severe closed head injury. She was aggressively treated immediately following the accident during helicopter transport to the Hermann Hospital emergency room. Upon arrival, Glasgow Coma Scale (GCS) score (13–15) was 6. Pupils were 2 mm in size and not reactive to light bilaterally. There was posturing to deep painful stimulation. No gag reflex was observed. Vital signs were stable. The patient underwent comput-

Table 1. Summary of characteristics for 95 subjects[a]

	Subject Group			
		Head injury		
		Normal ABR I–V latency		Abnormal ABR I–V latency
Characteristics	Normal	Good outcome	Death	Death
Male	14	14	13	12
Female	11	11	14	6
Total	25	25	27	18
Age (yr)				
Mean	29.7	26.2	26.5	32.9
(SD)	(6.2)	(7.6)	(8.9)	(14.2)
Glasgow Coma Score				
Mean		6.0	5.5	5.6
(SD)		(1.8)	(1.4)	(2.8)
Body temperature (°C)				
Mean		37.8	38.0	36.6[a]
(SD)		(0.66)	(0.70)	(1.72)

[a] *Difference between head injury group and other groups significant at 0.05 level of confidence.*

Table 2. Comparison of ABR latency and amplitude parameters among subject groups.

	Group			
		Head injury		
		Normal I–V latency	Abnormal I–V latency	
ABR Parameters	Normal	Good outcome	Death	Death
Latency (msec)	(n = 25)	(n = 25)	(n = 27)	(n = 18)
I				
Mean	1.81	1.75	1.72	1.80
(SD)	(0.58)	(0.32)	(0.26)	(0.19)
III				
Mean	3.84	3.82	3.77	4.33[a]
(SD)	(0.15)	(0.43)	(0.29)	(0.33)
V				
Mean	5.75	5.77	5.74	6.73[a]
(SD)	(0.15)	(0.39)	(0.35)	(0.46)
I–III				
Mean	2.15	2.12	2.05	2.53[a]
(SD)	(0.16)	(0.17)	(0.20)	(0.19)
III–V				
Mean	1.91	1.90	1.96	2.39[a]
(SD)	(0.13)	(0.14)	(0.20)	(0.54)
I–V				
Mean	4.05	4.02	4.01	4.92[a]
(SD)	(0.17)	(0.21)	(0.30)	(0.48)
Amplitude (μv)				
I				
Mean	0.26	0.30	0.28	0.25
(SD)	(0.15)	(0.19)	(0.16)	(0.16)
III				
Mean	0.24[b]	0.19	0.16	0.12[a]
(SD)	(0.08)	(0.09)	(0.10)	(0.06)
V				
Mean	0.50[b]	0.35	0.30	0.25
(SD)	(0.12)	(0.18)	(0.13)	(0.17)
V/I				
Mean	3.0[b]	1.64	1.64	1.52
(SD)	(2.8)	(1.24)	(1.53)	(1.42)
Vi–Vf				
(SD)	0.29	0.20	0.04[a]	0.10
Mean	(0.37)	(0.33)	(0.11)	(0.09)

[a] *Difference between head injury abnormality group and other groups significant at 0.05 level of confidence.*

[b] *Difference between normal group and head injury groups significant at 0.05 level of confidence.*

erized tomography (CT) scanning which revealed extensive subarachnoid hemorrhage around the brain stem, cerebellum, and in the Sylvian fissures (Fig. 3). There was also intraventricular hemorrhage within the lateral ventricles, and in the third and perhaps fourth ventricles. The patient was taken to the operating room for placement of an intracranial pressure (ICP) monitor (opening pressure 17 cm H20), and to the intensive care unit.

During the first 3 days postinjury, GCS remained 6. ICP was controlled adequately with chemical paralysis, sedation, and hyperosmolar therapy. Auditory evoked response assessment consistently yielded a repeatable ABR with normal wave I–V latencies, but an abnormally reduced wave V/I amplitude (Fig. 4, test 1). The spectrum of this ABR is shown in Figure 5 (test 1). Neurologic, physiologic, and ABR data are summarized chronologically in Table 4. Energy was noted in the 500 and 900 Hz regions on the right and also in the 700 Hz region. On the left, there was no evidence of spectral amplitude peaks above 500 Hz. We never observed an auditory middle-latency response. On the 5th day, repeat CT scanning (Fig. 3) showed enlargement of the lateral ventricles, suggesting obstructive hydrocephalus secondary to the intraventricular hemorrhage. The perimesencephalic cistern was not well visualized, in part due to artifact. On the previous day the ABR was characterized by apparent deterioration of waveform morphology, but interwave latency values were still within normal limits (Fig. 4, test 2). Overall, the ABR spectrum appeared unchanged, but less low frequency energy was apparent (Fig. 5). Later on the 5th day, ICP became elevated and unresponsive to previous therapy modalities, and barbiturate-induced coma was initiated.

On the 6th day, CT scanning showed progressive brain pathology (massive left middle cerebral artery distribution infarction and left-to-right shift). Compression of the perimesencephalic cistern suggested transtentorial herniation. The pupils had become fixed and dilated. There were no spontaneous respirations, and brain stem reflexes were not present, although this latter finding probably was due to barbiturate therapy. ICP was markedly elevated (70 mm Hg). Repeat ABR assessment (Fig. 4, test 3) showed a wave I–V latency abnormality on the right with an apparently normal I–V interval on the left although a wave III was not observed. Again, inspection of ABR spectrum revealed no distinct changes from previous data. The fourth and final ABR assessment (Fig. 4, test 4) at this time yielded no response on the left, and only a wave I and possible wave III component on the right. Even with these marked ABR waveform alterations on the right, we still observed spectral energy through and above 1000 Hz. There was, however, a sharp reduction in low-frequency energy, an unusual peak at 250 Hz, and less repeatability of later peaks. The spectrum of the left ear ABR (no response) was characterized only by low-frequency energy. Blood pressure was maintained with medication. Nuclear cerebral blood flow studies 6 hours later showed no cerebral circulation. The patient was declared brain dead.

Comment. As illustrated by this case report, ABR spectral analysis may supplement the traditional analyses of wave component latency and amplitude parameters in the time domain. An ABR of normal latency was recorded bilaterally in a patient with severe brain pathology and neurologic dysfunction. This is not an uncommon finding in severe head injury (15–25). The spectrum of this ABR, however, differed from normal expecta-

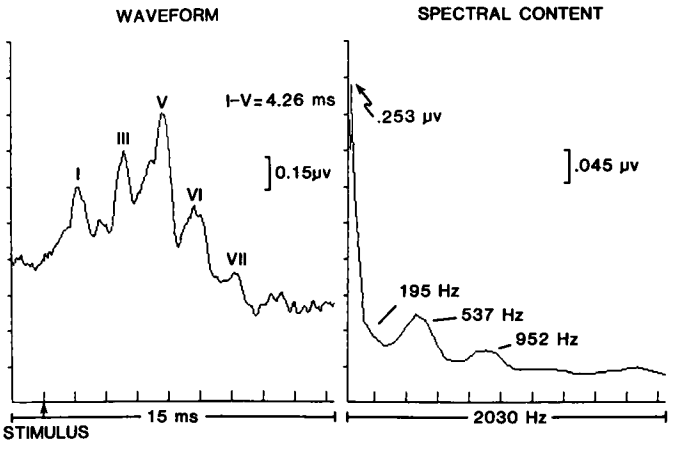

Figure 1. Auditory brain stem response (ABR) time domain and spectral waveforms for a normal 35-year-old subject. Note spectral peaks in three main energy regions: 0 to 200 Hz, 500 to 600 Hz, and 900 to 1000 Hz.

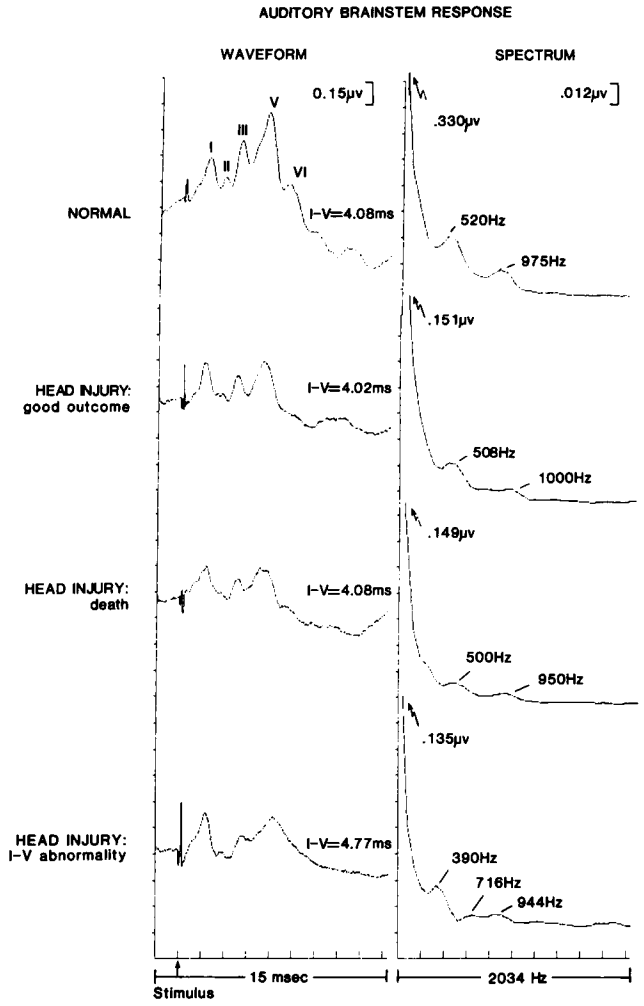

Figure 2. Grand average of ABR time domain and spectral waveforms for 10 consecutive subjects in four subject groups: normal, head-injured with normal ABR latency and good outcome, head-injured with normal ABR latency and death within 2 weeks post-injury, and head-injured with abnormal ABR interwave latency. See Tables 1 and 2 for descriptions of groups and ABR data.

tions. On subsequent assessments ABR latency increased on the right and remained stable on the left, yet the spectrum did not appear to change for either ear, except for a possible decrease in overall energy. With only a clear ABR wave I component, and poorly formed wave III for the right ear on the final test date, spectral analysis yielded an abnormal distribution of energy, although some energy was observed for frequencies above 1000 Hz. On the left, low frequency energy was apparent even though there was no ABR. This case suggests that in a comatose head-injured patient, an abnormal ABR spectrum may be generated from an ABR waveform with normal latency parameters. Changes in latency and spectrum may occur independently. A clear-cut relationship among ABR wave component presence versus absence and corresponding spectral waveform was not observed.

Table 3. Comparison of ABR spectral amplitude (μv) among subject groups

	Spectral Amplitude in μv by Group			
		Head injury		
		Normal I–V latency		Abnormal I–V latency
Frequency Region (Hz)	Normal	Good outcome	Death	Death
0–200				
Mean	0.287	0.192[a]	0.169[a]	0.162[a]
(SD)	(0.128)	(0.119)	(0.079)	(0.076)
201–400				
Mean	0.008	0.007	0.005	0.007
(SD)	(0.022)	(0.020)	(0.012)	(0.014)
401–600				
Mean	0.030	0.026	0.015[a,b]	0.013[a,b]
(SD)	(0.015)	(0.017)	(0.012)	(0.014)
601–800				
Mean	0.001	0.002	0.002	0.005
(SD)	(0.006)	(0.008)	(0.006)	(0.008)
801–1000				
Mean	0.012	0.007	0.008	0.006
(SD)	(0.010)	(0.010)	(0.008)	(0.008)
1001–1200				
Mean	0.005	0.007	0.005	0.001
(SD)	(0.008)	(0.009)	(0.007)	(0.003)
1201–1400				
Mean	0.001	0.002	0.001	0.003
(SD)	(0.003)	(0.004)	(0.002)	(0.006)
>1400				
Mean	0.003	0.002	0.002	0.003
(SD)	(0.004)	(0.003)	(0.003)	(0.006)

[a] *Difference between head injury and normal group significant at 0.05 level of confidence.*

[b] *Difference between two or three head injury groups significant at 0.05 level of confidence.*

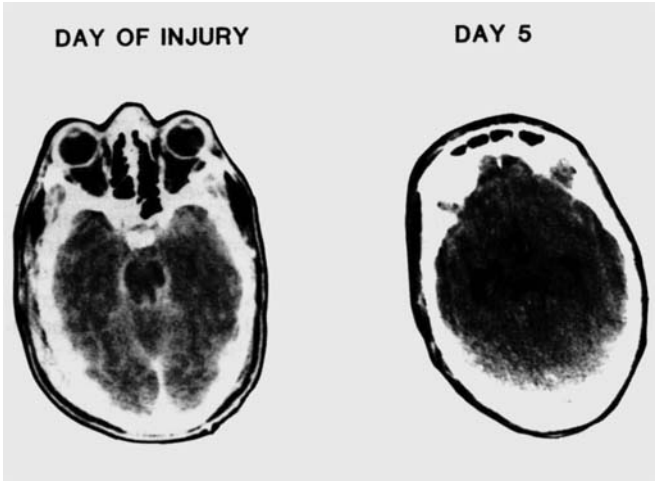

Figure 3. Computerized tomography (CT) scans for a 21-year-old female with severe closed head injury on the day of injury and on postinjury day 5 showing brain stem level pathology.

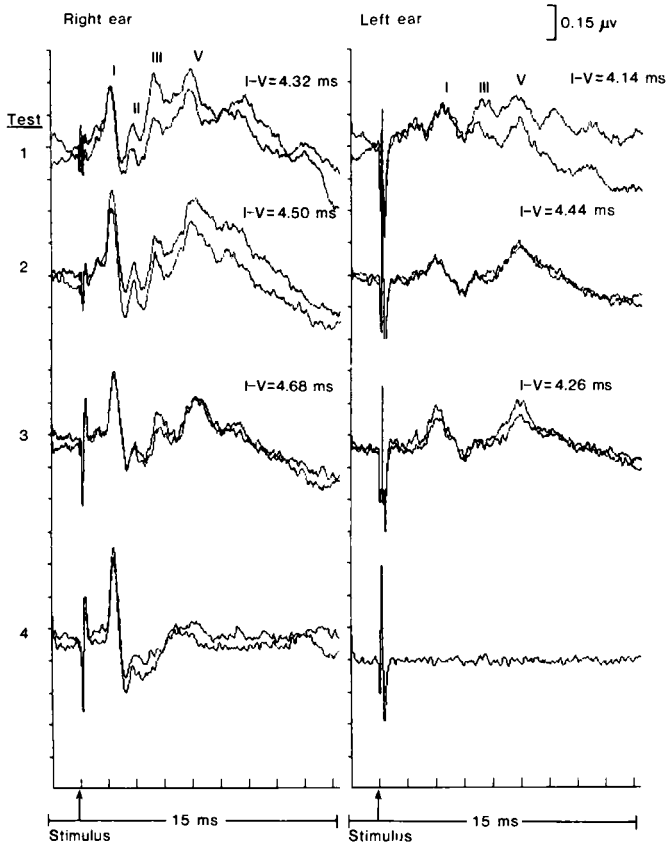

Figure 4. Serial ABR time domain waveforms for a 21-year-old female with severe closed head injury. See Table 3 for summary of physiologic and neurologic findings at test times.

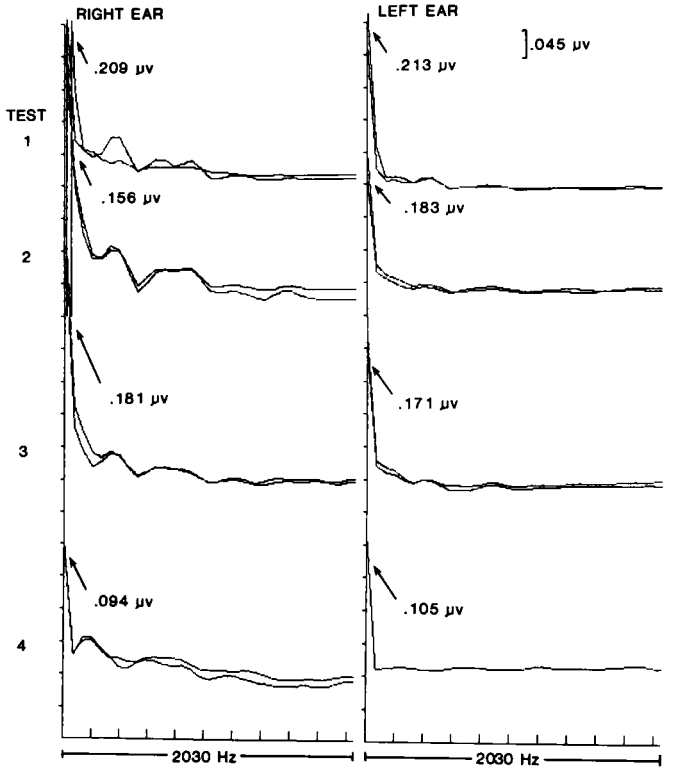

Figure 5. Serial ABR spectral waveforms for a 21-year-old female with severe closed head injury. ABR time domain waveforms are shown in Figure 4.

DISCUSSION

The normative ABR spectral findings described in the present paper are in good agreement with data reported by others (4–11). Spectral energy was invariably greatest within a low-frequency region (below 200 Hz). This appears to be a characteristic feature of the normal ABR spectrum (5–10). The majority of normal subjects also had spectral energy peaks in the vicinity of 500 and 900 Hz, although, consistent with the results of previous studies (5–10), the magnitude of spectral energy decreased as frequency increased. These normative spectral data must be viewed as specific to the test protocol and instrumentation used in the present study. Based on clinical experience and existing preliminary reports (4–11), we postulate that ABR spectral content is influenced by subject characteristics (age, sex, body temperature), stimulus factors (type, intensity, rate, monaural versus binaural presentation mode) and measurement conditions (electrode site, filter settings), as are conventional response parameters (latency and amplitude) in the time domain (1).

ABR spectral content for the severely head-injured subjects differed qualitatively and quantitatively from these normative data. Fewer head-injured subjects than normal subjects showed evidence of spectral energy peaks in the apparently normal regions (e.g., 400 to 600 Hz and 900 to 1100 Hz), and magnitude of energy in these regions

Table 4. Chronologic summary of physiologic, neurologic, and ABR data for a 21-year-old female with acute severe head injury.

	Test (Postinjury day)					
Parameter	1 (2)		2 (4)		3 (6)	
Physiologic[a]						
Temperature (°F)	96.2		97.0		99.5	
MAP	98		85		123	
ICP (mm Hg)	8		4		78	
CPP	90		79		45	
PaO_2	104		111		191	
$PaCO_2$	23		42		16	
Neurologic						
Pupils						
Size (mm)	2		2		6	
Responsiveness[b]	±		±		–	
ABR						
Latency (msec)	Right	Left	Right	Left	Right	Left
I–III	2.34	2.10	2.46	2.04	1.86	–
III–V	1.98	2.04	2.22	2.16	–	–
I–V	4.32	4.14	4.68[c]	4.20	–	–
Amplitude (μV)						
I	0.39	0.16	0.48	0.16	0.66	–
III	0.10	0.13	0.09	0.09	–	–
V	0.17	0.16	0.11	0.11	–	–
V/I	0.43[c]	1.00	0.23[c]	0.69	–	–

[a] *°F, Degrees Fahrenheit; MAP, mean arterial pressure; ICP, intracranial pressure; CPP, cerebral perfusion pressure; PaO_2, arterial pressure oxygen; $PaCO_2$, arterial pressure carbon dioxide; mm Hg, mm mercury.*

[b] *+, Normal response; ±, sluggish; –, no response.*

[c] *Exceeds ±2.5 SD of normative mean value.*

was significantly decreased in comparison to normal expectations. This pattern of spectral findings was especially noteworthy in the two head-injury groups with well-formed and reliably recorded ABRs which, by selection criteria, had interwave latency values (I–III, III–V, I–V) that were within normal limits, and no CT evidence of brain stem pathology. The distribution of spectral energy in the head-injured subjects with abnormal interwave latency values was uniquely different. In addition to a general reduction in energy at all spectral regions, there were spectral energy peaks in frequency regions uncharacteristic of the normal group and the other two head injury groups, for example 200 to 400 Hz and 600 to 800 Hz.

Prognosis with ABR spectral data was not an objective of the present study. Curiously, however, there was a significant difference in the spectral content of the ABR for head-injured patients who survived with good outcome versus those who died within the first 2 weeks postinjury. These groups were not differentiated by other ABR parameters (latency or amplitude), nor severity of the injury as determined by the GCS score. More detailed analysis of clinical neurologic signs for the two groups was not carried out. It is possible that these neurologic data or other, multimodality evoked response data might also have differentiated these two head-injured groups.

The distinctive ABR spectral content of the head-injured subjects is, perhaps, related to coma. Recent clinical studies of compressed spectral array EEG have shown a reduction in higher frequency (θ or α frequency range of 4 to 13 Hz) energy in deep coma (26, 27), and a correlation of the reappearance of these frequencies with good recovery. All of the head-injured subjects in the present study had GCSs of 8 or less and were, by accepted definition (13), deeply comatose. Coma is, in part, related to decreased neural activity in the reticular formation (28). Thus, cortical evoked responses and the EEG are usually abnormal in coma and altered or abolished by therapeutic doses of CNS-depressants, such as barbiturates (13, 15–19, 29). The ABR, at least the latency parameter, is remarkably resistant to these influences (15–19, 29). The findings of the present study suggest that the spectral content of the ABR is a distinct response parameter, just as latency and amplitude parameters are independent indices of neural function that may be differentially effected by brain pathophysiology (30–33). It should be noted that, in the present study, the head-injured subjects showed significantly reduced ABR wave III and V amplitude. ABR wave component amplitude in the time domain and spectral amplitude are, probably, related and may have some common neurophysiologic bases not shared by the latency parameter.

Many clinical questions remain unanswered. Further investigations of ABR spectral characteristics in brain-injured populations are needed to determine whether or not spectral analysis will, in fact, contribute to neurodiagnosis. Definition of the relationship of ABR spectral content with outcome and physiologic parameters, such as blood gases, ICP, and cerebral perfusion pressure would be especially useful for clinical application of this ABR parameter in acute head injury. Clinical exploitation of this ABR spectral analysis in general requires a better understanding of the potential influences of subject characteristics, stimulus and measurement parameters, and drugs in normal populations, and the effects of peripheral auditory deficits.

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