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Hypo- and Hyperthermia in Clinical Auditory Brain Stem Response Measurement: Two Case Reports

James W. Hall III, Joan M. Bull, and Leslie H. Cronau

Department of Otolaryngology-Head and Neck Surgery [J.W.H.], Division of Hematology and Oncology, Department of Medicine [J.M.B.], and Department of Anesthesiology [L.H.C.], University of Texas Medical School, Houston, Texas

ABSTRACT

Two case reports are presented to highlight the important effects of body temperature in clinical auditory brain stem response (ABR) measurement. Case 1 is an 11 year old boy in coma secondary to severe head injury. High dose barbiturate therapy suppressed brain stem neurologic signs and the ABR was relied on as a monitor of CNS status. Hypothermia during this period of intensive care was a crucial factor for meaningful interpretation of ABR findings. The second case was a 26 year old male undergoing hyperthermic therapy for advanced cancer. As body temperature increased from 38 to 42 degrees Centigrade (107.6 degrees Fahrenheit), there was a systematic decrease in latency for waves III and V. An overall hyperthermia-related decrease in the wave I-V latency interval of 0.5 to 0.6 milliseconds was observed on two test dates. ABR results for these two cases are discussed in the context of basic knowledge on body temperature and auditory electrophysiology.

The effect of low body temperature (hypothermia) on auditory brain stem response (ABR) and other auditory evoked responses, especially the electrocochleogram (ECochG), has been extensively investigated for a variety of animal models, as indicated in Table 1. Clinically, most reports of ABR in hypothermia describe changes observed during open heart surgery (Table 2). Alterations in auditory electrophysiology related to low body temperature may be summarized as follows. In vitro depolarization in membrane potentials (a decrease) is recorded in supporting cells (Hensen's) of the organ of Corti (Santos-Sacchi, 1986). Cochlear microphonic (CM) amplitude is reversibly reduced, while CM latency shows little or no change (Butler, Konishi, & Fernandez, 1960; Brown, Nuttall, Masta & Lawrence, 1983; Coats, 1965; deBrey & Eggermont, 1978; Drescher, 1976; Fernandez, Singh & Perlman, 1958; Kahana, Rosenblith, & Galambos, 1950). Variable changes during hypothermia are found for the summating potential (Butler et al, 1960; Manley & Johnstone, 1974). Basilar membrane traveling wave transit time is increased (deBrey & Eggermont, 1978).

Lowered temperature also produces a reversible reduction in VIIIth nerve compound action potential (ECochG N1 component and ABR wave I) amplitude and a reversible increase of N1 (wave I) latency (Gulick & Cutt, 1961; Kahana et al, 1950). An initial effect of hypothermia may be the selective loss of auditory sensitivity for high-frequency signals, as estimated electrophysiologically (Brown et al, 1983; Manley & Johnstone, 1974). Synaptic trans-

Table 1. Summary of experimental studies on low body temperature (hypothermia) and auditory evoked responses (AERs)

Study, Year	AER*	Model
Butler et al, 1983	ECochG	Guinea pig
Britt et al, 1983	ABR	Rat
Cutt and Gulick, 1961	ECochG	Guinea pig
Christian, Brown, Smith, and Nuttall, 1983	ECochG	Guinea pig
Coats, 1965	ECochG	Cat
deBrey and Eggermont, 1978	ECochG	Guinea pig
Doyle and Fria, 1985	ABR	Monkey
Eggermont, 1974	ECochG	Guinea pig
Fernandez et al, 1958	ECochG	Guinea pig
Gold et al, 1985	ABR	Rat
Henry, 1980	ECochG	Mouse
Jones, Stoc[illegible] and [illegible]ner, 1980	ABR	Cat
Kaga, Takiguchi, Myokai, and Shiode, 1979	ABR	Cat
Kahana et al, 1950	ECochG	Hamster
Marsh, Yamane, and Potsic, 1984	ABR	Guinea pig
Manley and Johnstone, 1974	ECochG	Guinea pig, bat
Moffat and Capranica, 1976	ECochG	Toad (American)
Rossi and Britt, 1984	ABR	Cat
Santos-Sacchi, 1986	ECochG	Guinea pigs
Schorn, Lennon, and Bickford, 1977	ABR	Rat
Smolders and Klinke, 1977, 1982	ECochG	Cat, crocodile
Snider, Thomas, and Snider, 1982	ALR	Cat
Williston and Jewett, 1982	ABR	Rat

** ABR, auditory brain stem response; ALR, auditory late response; ECochG, electrocochleography.*

Table 2. Summary of clinical studies on low body temperature (hypothermia) and auditory evoked responses (AERs)

Study, Year	N	AER[a]	Recording Condition
Dorman, Britt, and Silverberg, 1981	1	ABR	Heart surgery
Kaga et al, 1979	12	ABR	Heart surgery
Markand, Warren, Moorthy, Stoelting, and King, 1984	16	ABR	Heart surgery
Kileny, Dobson, and Gelfand, 1983	12	AMR	Heart surgery
Marshall and Donchin, 1981	3	ABR	Circadian changes
Rosenblum, Ruth, and Gal, 1985	1	ABR	Aortic aneurysm surgery
Samra and Lilly, 1983	8	ABR	Heart surgery
Siu, Rossiter, Schorn, and Shattuck, 1977	15	ABR	Heart surgery
Stockard et al, 1978	10	ABR	Heart surgery

[a] *ABR, auditory brain stem response; AMR, auditory middle-latency response.*

Table 3. Summary of experimental and clinical studies on high body temperature (hyperthermia) and auditory evoked responses (AERs)

Study, Year	AER[a]	Model	Temperature Increase[b]
Experimental			
Barnett, 1980	ECochG	Cat	+8
Cutt and Gulick, 1961	ECochG	Guinea pig	+7
Gold et al, 1985	ABR	Rat	+5
Marsh et al, 1984	ABR	Guinea pig	+3
Clinical			
Bridger and Graham, 1985	ABR	Normal[c]	+1
Geraud et al, 1982	ABR	MS	+1
Phillips et al, 1983	ABR	MS	+1

[a] *ECochG, electrocochleography; ABR, auditory brain stem response.*
[b] *Increase in degrees Centigrade above baseline temperature (usually 37°C).*
[c] *Neurologic status of patient; MS, multiple sclerosis.*

mission is delayed and axonal conduction velocity is decreased (Benita & Conde, 1972; deJesus, Hausmanowa-Petrusewicz, & Barchi, 1973; Snyder, 1908). Consequently, ABR latencies are increased, especially for later versus earlier latency waves. With severe hypothermia (body temperature less than 14–20°C), the ABR disappears (Rosenblum, Ruth, & Gal, 1985).

Less well studied is the effect of hyperthermia (increased body temperature) on auditory evoked responses (Table 3). A handful of experimental studies have shown evidence of decreased latency and amplitude of ECochG N1 component and ABR waves with elevation of body temperature (Barnett, 1980; Cutt & Gulick, 1961; Gold, Cahani, Sohmer, Horowitz, & Shahar, 1985). Dubois, Coppola, Buchsbaum, and Lees (1981) reported decreased somatosensory evoked response latency as a function of temperature in man. Investigation of ABR and body temperature has been limited to observations in essentially normothermic subjects. Bridger and Graham (1985) recorded ABRs from 9 normal subjects while body temperature (measured under the tongue) was raised 1°C with a specially constructed heating blanket. Other studies (Geraud et al, 1982; Phillips et al, 1983) were likewise limited to very modest temperature increases (1° or less), and conducted in selected patients with neurological disease (multiple sclerosis). In this paper, we describe changes in serial ABR recordings for a head-injured patient during coma-related hypothermia and in a cancer patient undergoing whole body hyperthermia treatment.

Case 1

The patient was an 11 yr old male pedestrian who was struck by an automobile. He lost consciousness at the scene of the accident, and showed periods of apnea. Glasgow Coma Scale score (GCS) was 5, consistent with a severe head injury (Hall & Tucker, 1986). He was intubated, given emergency medical therapy (hyperventilation and maintenance intravenous fluids) and transported via helicopter to the hospital. Upon arrival, GCS was unchanged. Pupils were fixed and dilated. Computerized tomography (CT) revealed a large intracerebral hematoma on the left, and a left-to-right shift of the midline between cerebral hemispheres.

Serial ABRs were recorded in 13 separate test sessions during the first 3 weeks postinjury. The patient was comatose on each occasion. Recordings were made at bedside in the pediatric intensive care unit (PICU) with a commercially available evoked response system (Nicolet CA-1000). Stimuli were clicks (0.1 msec duration) presented monaurally (right and left ears) via TDH-49 earphones at an intensity level of 85 dB NHL and a rate of 11.1 or 21.1/sec. The ABR was detected with EEG disc electrodes located on the forehead at the hairline (noninverting), earlobe ipsilateral to the stimulus (inverting) and nasion (ground). Analysis time was 15 msec and there were 512 data points within this period. Filters were set at 30 to 3000 Hz or 30 to 1500 Hz (slope of 12 dB/octave), with no notch filter.

Serial ABR latency data, and physiologic data, are summarized in Table 4. Representative ABR waveforms are illustrated in Figure 1. At the initial assessment (8 hr postinjury), a temperature reading was not available, but 4 hr later it was normal (36.8°C as measured rectally). Other physiologic data were also not yet available at the initial evoked response assessment. ABR latency values at this test session were well within normal limits (±2.5 SD above clinical normative mean values). Body temperature subsequently decreased. For five test sessions temperature was below 34°C (93.0°F). During this period, ABR absolute and interwave latency values markedly exceeded normal limits. Both wave I–III and III–V latency intervals were abnormally prolonged. With a return to normothermia (37°C and above), absolute and interwave ABR latencies for waves I, III, and V again fell within normal limits. This ABR pattern is illustrated in Figure 2, where wave I–V latency is plotted as a function of temperature.

At least three general physiologic factors must be considered in the interpretation of these ABR data. Blood gases can influence ABR findings (Gulick, 1958; Hecox & Cone, 1981; Rossini, Kula, House, & Cracco, 1982; Sohmer, Gafni, & Chisin, 1982). In this patient, there was no documented hypoxia (abnormally decreased arterial partial pressure for oxygen) or hypercapnia (elevated arterial partial pressure for carbon dioxide).

Table 4. Summary of serially recorded physiologic and auditory brain stem response (ABR) parameters for 11 yr old boy with acute head injury and hypothermia (case 1). ABR waveforms are illustrated in Figure 1

	Temperature in °C/F													
Parameter	C 36.8 F 98.2	34.2 93.6	34.2 93.6	33.9 93.0	32.8 91.0	35.4 95.7	33.3 91.9	32.2 90.0	32.7 90.9	37.2 98.9	37.4 99.3	37.4 99.3	37.5 99.5	r^a
Physiologic[b]														
PaO_2	NA	222	NA	145	163	154	174	201	169	NA	147	120	131	−0.67
$PaCO_2$	NA	23	NA	16	22	20	14	15	18	NA	20	21	24	0.56
MAP	NA	78	78	87	91	89	80	88	85	103	109	109	103	−0.81
ICP	NA	10	15	14	15	14	21	18	18	13	7	9	NA	0.77
CPP	NA	68	63	73	76	75	59	70	67	90	102	100	NA	−0.85
Barbiturates	No	No	Yes	Yes	Yes	Yes	Yes	No	No	No	No	No	No	
ABR latency (msec)														
I														
Right	1.80	2.22	2.16	2.34	2.40	2.22	2.46	2.37	2.46	2.04	2.10	1.74	1.92	−0.88
Left	1.68	2.16	2.10	2.10	2.22	2.04	2.22	2.25	2.22	1.86	1.92	1.92	1.98	−0.88
III														
Right	3.90	4.68	4.62	4.98	5.22	4.56	5.22	5.19	5.04	4.14	4.26	3.66	3.90	−0.95
Left	3.72	4.80	4.62	4.68	5.22	4.62	4.74	5.31	5.10	4.02	4.20	3.78	4.26	−0.92
V														
Right	5.88	7.20	7.14	7.26	7.86	7.02	8.04	7.89	7.62	6.66	6.42	5.52	5.64	−0.92
Left	5.64	6.84	6.78	7.14	7.74	6.90	7.68	7.71	7.38	6.00	6.06	6.24	6.06	−0.94
I–III														
Right	2.10	2.46	2.46	2.64	2.82	2.34	2.76	2.82	2.58	2.10	2.16	1.92	1.98	−0.96
Left	2.04	2.64	2.52	2.58	3.00	2.58	2.52	3.06	2.88	2.16	2.28	1.86	2.28	−0.90
III–V														
Right	1.98	2.52	2.52	2.28	2.64	2.46	2.82	2.70	2.58	2.52	2.16	1.86	1.74	−0.80
Left	1.92	2.04	2.16	2.46	2.52	2.28	2.94	2.40	2.28	1.98	1.86	2.46	1.80	−0.63
I–V														
Right	4.08	4.98	4.98	4.92	5.46	4.80	5.58	5.52	5.16	4.62	4.32	3.78	3.72	−0.93
Left	3.96	4.68	4.68	5.04	5.52	4.86	5.46	5.46	5.16	4.14	4.14	4.32	4.08	−0.92

[a] *r, Correlation.*

[b] *PaO_2 and $PaCO_2$, arterial pressure of oxygen and carbon dioxide; MAP, mean arterial pressure; ICP, intracranial pressure; CPP, cerebral perfusion pressure.*

ABR changes may also be related to reduced cerebral perfusion pressure, presumably secondary to brain stem ischemia (Goitein, Fainmesser, & Sohmer, 1983; Hall & Tucker, 1986). In this case, cerebral perfusion pressure was marginally depressed (less than 60 mm Hg) on only two of the test dates. [Note: cerebral perfusion pressure (CPP) is calculated by subtracting intracranial pressure (ICP) from mean arterial pressure (MAP).] It is reasonable to suspect that lowered CPP could have been a factor in the prolongation of ABR latencies. However, ABR changes were more consistently accompanied by fluctuations in temperature than CPP. For example, from the fourth to the fifth test session CPP actually increased slightly (from 73 to 76 mm Hg) while temperature decreased by a degree. The ABR I–V latency increase (approximately 0.5 msec from one test session to the next) appeared to correspond to the change in temperature. Close inspection of the relationship between ABR latency and CPP versus temperature for other test dates similarly showed a closer link with temperature than CPP.

A final physiologic factor to be considered for case 1 was medical therapy. The patient was treated with high-dose barbiturates during a portion of the ABR assessment period, which seriously suppressed CNS activity. Although barbiturate blood levels exceeded 30 μg/ml, there is ample experimental (Marsh, Frewen, Sutton, & Potsic, 1984) and clinical evidence (Hall, 1985) that ABR latencies are not seriously influenced by barbiturates. Thus, the changes in serial ABR findings for this patient appeared to be primarily temperature related.

Case 2

The patient was a 23 yr old male with widely metastatic melanoma, originating in the liver, who was undergoing whole body hyperthermia treatment for the cancer (Barlogie et al, 1979; Bull et al, 1978). ABRs were continuously recorded in the operating room during two separate treatment sessions, each of approximately 5 hr duration. A total of 41 replicated ABR waveforms were averaged during the first hyperthermic test session and 43 were averaged during the second test session. The stimulus and acquisition parameters were as described above for case 1, with two exceptions. Stimuli were delivered with an insert (Etymotic ear tip) ear cushion (versus standard audiometric earphone and cushion) and to only the left ear (versus each ear). On each date, the patient was anesthetized (nitrous oxide and sufentanyl) and chemically paralyzed (pancuronium) during the ABR assessment. Lidocaine was administered periodically (200 mg bolus at the start and then 150 mg/hr drip infusion). Whole body hyperthermia was achieved by means of a specially modified Clinitron bed.

Serial ABR and physiologic data for each test session are summarized in Table 5. Temperature is indicated in degrees Centigrade. Fahrenheit values are also shown for reference. Waveforms are illustrated in Figure 3. Temperature measured with an arterial probe is indicated in the Figure and in the top portion of Table 5. For comparison purposes, temperature values recorded simultaneously with rectal, tympanic membrane, and

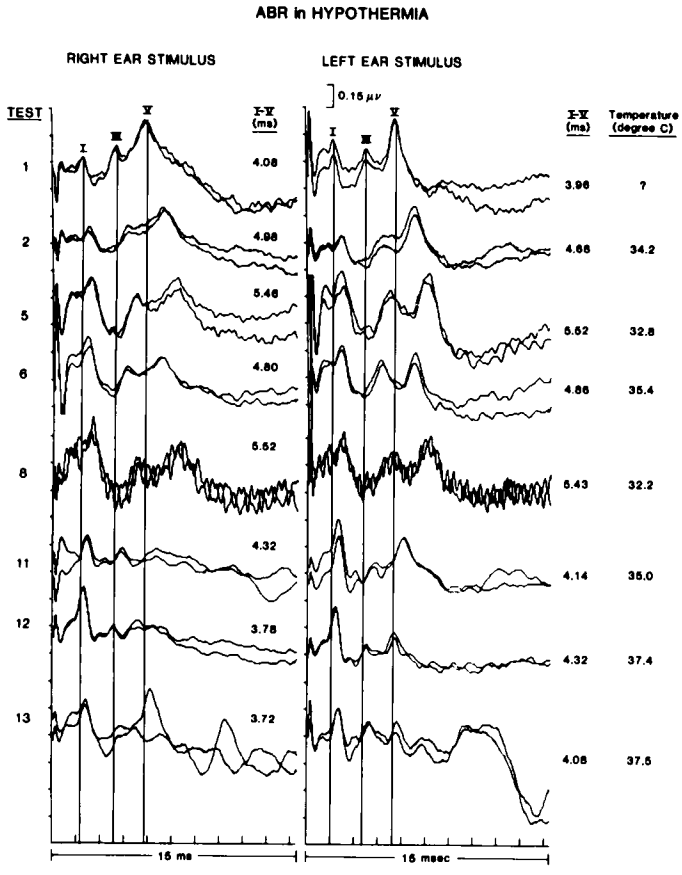

Figure 1. Serially recorded auditory brain stem response (ABR) waveforms for an 11 yr old with acute head injury and hypothermia (case 1). ABR and physiologic data are displayed in Table 4.

esophageal thermisters are also shown. There was close correspondence among these various temperature indices throughout each test session.

In the operating room setting, MAP was monitored constantly. Values at the time of ABR recordings are displayed in Table 5. Blood gases were only analyzed four times during treatment and are not indicated in the Table. However, PaO_2 levels were always greater than 120 mm Hg and O_2 saturation consistently exceeded 94%. $PaCO_2$ values were in the 36 to 45 mm Hg range. As temperature increased from approximately 38.5 to 42°C (101.3–107.6°F), wave I latency remained unchanged. Latency for wave III and wave V decreased systematically as temperature increased. The overall decrease in latency for wave V, and consequently the I–V latency interval, was on the order of 0.5 msec. Notably, the effects of hyperthermia on ABR latency were highly repeatable over the two test sessions. Hyperthermia can increase heart rate and, on occasion, lead to sinus tachycardia. Consequently, lidocaine, an antiarrythmic agent, is often administered during hyperthermia treatment. ABR latency may be affected by lidocaine (Ruth, Gal, DiFazio, & Moscicki, 1985; Worthington, Brookhauser, Mohiuddin, & Gorga, 1985) and, as noted above, case 2 received this drug during each treatment session. The reported effect of lidocaine on the ABR, however, is to increase interwave latencies while we consistently observed shortened latencies. The ABR changes in the present study, therefore, cannot be accounted for by the lidocaine, although it's possible that an even greater decrease in latencies might have been recorded without the lidocaine.

As evident from Figure 3, amplitude values for all wave components (not shown in Table 5) appeared to be unaffected by increased temperature. The ABR wave I–V latency interval during the first test session is shown in the context of our normative clinical data (mean value ± 2.5 SD for male and female subjects combined) in Figure 2. At the highest temperature, the I–V latency interval was at the lower limit of the combined-sex normative region, and exceeded the −2.5 SD limit for young male subjects.

COMMENTS

Although body temperature is regularly cited as a factor in ABR measurement (e.g., Hall, 1984; Marshall & Donchin, 1981; Stockard & Westmoreland, 1981), it is probably not necessary to document temperature routinely in ABR assessments for audiologic or neurologic purposes in generally healthy patients. Documentation of body temperature is required for meaningful and valid interpretation of ABR latency data recorded in seriously ill patients and in electrophysiologic monitoring of neurologic status. Examples of patients in this first category are those with infection accompanied by fever or those with hyperthermia caused by certain metabolic diseases, pharmacologic agents, or CNS pathology (Milton, 1982). Also included in this category are patients with acute illness at risk for

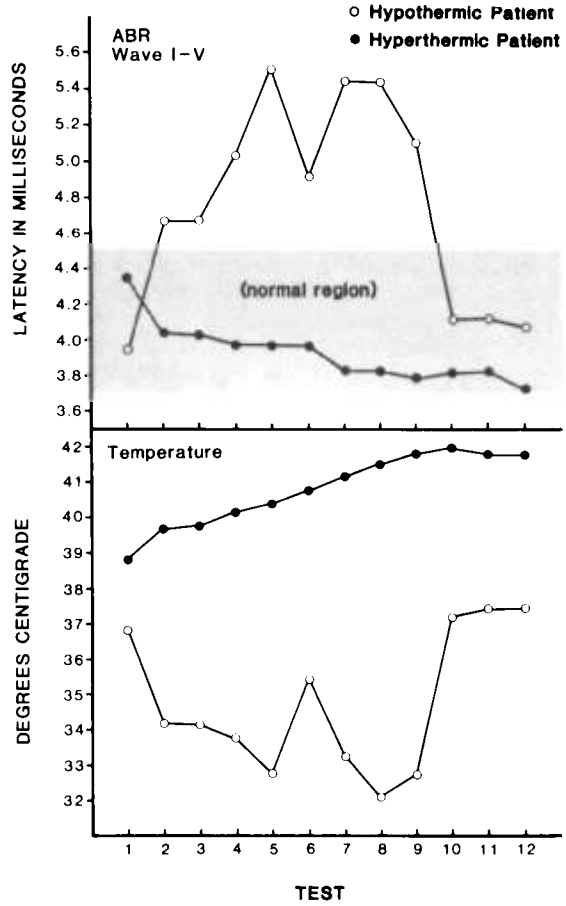

Figure 2. Auditory brain stem response (ABR) wave I–V latencies and body temperature for an 11 yr old boy during hypothermia (case 1) and a 26 yr male during hyperthermia (case 2).

Table 5. Summary of serially recorded physiologic and auditory brain stem response (ABR) parameters for 26 yr old male undergoing hyperthermic therapy for cancer (case 2). ABR data are for left ear stimulation. ABR waveforms are illustrated in Figure 3

	Temperature in °C/F*									
	C	38.5	39.0	39.5	40.0	40.5	41.0	41.5	42.0	42.0	
Parameter	F	101.3	102.2	103.1	104.0	104.9	105.8	106.7	107.6	107.6	*r*
Physiologic											
Rectal temperature (C)											
Test 1			38.7	39.4	40.0	NA	40.8	41.4	42.1	42.0	
Test 2			39.1	39.5	40.1	40.4	41.2	41.5	42.2	42.4	
Tympanic membrane temperature (C)											
Test 1			38.4	39.2	38.6	NA	40.7	41.3	41.6	41.5	
Test 2				39.5	40.0	40.5	41.2	41.4	41.8	41.5	
Nasal temperature (C)											
Test 1			38.7	39.5	40.2	NA	41.0	41.5	42.0	41.8	
Test 2			39.1	39.4	40.0	40.4	41.1	41.4	42.0	42.0	
Mean arterial pressure (mm Hg)											
Test 1			66	68	75	76	74	73	62	65	−0.21
Test 2		86	100	99	89	94	90	84	78	72	−0.71
ABR (latency in ms)											
I											
Test 1			1.29	1.32	1.32	1.29	1.29	1.29	1.32	1.38	−0.41
Test 2		1.32	1.36	1.32	1.35	1.26	1.26	1.29	1.32	1.32	−0.38
III											
Test 1			3.36	3.33	3.33	3.27	3.24	3.27	3.15	3.27	−0.83
Test 2		3.66	3.57	3.54	3.42	3.48	3.39	3.36	3.30	3.54	−0.74
V											
Test 1			5.61	5.34	5.28	5.25	5.25	5.10	5.01	5.10	−0.93
Test 2		5.67	5.67	5.49	5.40	5.37	5.29	5.25	5.13	5.16	−0.98
I–III											
Test 1			2.07	2.01	2.01	1.98	1.95	1.98	1.83	1.89	−0.92
Test 2		2.34	2.21	2.22	2.07	2.22	2.13	2.07	1.98	2.22	−0.65
III–V											
Test 1			2.25	2.01	1.95	1.98	2.01	1.89	1.83	1.62	−0.86
Test 2		2.01	2.10	1.95	1.98	1.89	1.90	1.89	1.83	1.62	−0.82
I–V											
Test 1			4.32	4.02	3.96	3.96	3.96	3.81	3.69	3.72	−0.93
Test 2		4.35	4.31	4.17	4.05	4.11	4.03	3.96	3.81	3.84	−0.97

* *Recorded with an arterial thermister.*

hypothermia, including low birthweight infants (Stockard & Westmoreland, 1981) and persons in coma secondary to severe brain injury (Hall & Tucker, 1986).

Temperature is a particularly important parameter to consider in the interpretation of serially recorded ABR data, or sensory evoked responses in general, during CNS monitoring (Hall & Tucker, 1986; Kileny & McIntyre, 1985). The clinical objective of evoked response monitoring is early detection of deleterious changes in neurologic status secondary to dynamic pathophysiology (e.g., brain ischemia). These CNS changes are reflected by increases in the ABR, most often latency. Nonpathologic bases for altered ABR findings, including physiologic factors such as body temperature, must be ruled out before a change in neurologic status can be presumed.

ABR findings for case 1 were important in his acute medical management following severe head injury. High-dose barbiturates were employed as a last resort in preserving residual brain function and preventing further secondary brain injury (Hall & Tucker, 1986). This therapy modality, however, suppressed clinically observed brain stem signs and seriously complicated daily assessment of neurologic status. As evidence of this clinical problem, cerebral perfusion studies were twice conducted with this patient because impending brain death was suspected. The ABR, in this case, served as the only direct index of CNS integrity. Based in part on this information, an aggressive treatment approach was taken. Failure to account for the effects of temperature in interpreting ABR latency prolongations might have had dire implications for this patient's intensive care.

The findings for case 1 had, in addition to these clinical implications, some relevance to basic science. Selected alterations in auditory electrophysiology previously associated with hypothermia, such as increased wave I (ECochG N1) latency and increased synaptic transmission time and axonal conduction velocity as reflected in interwave latency prolongation, were observed for case 1. It is

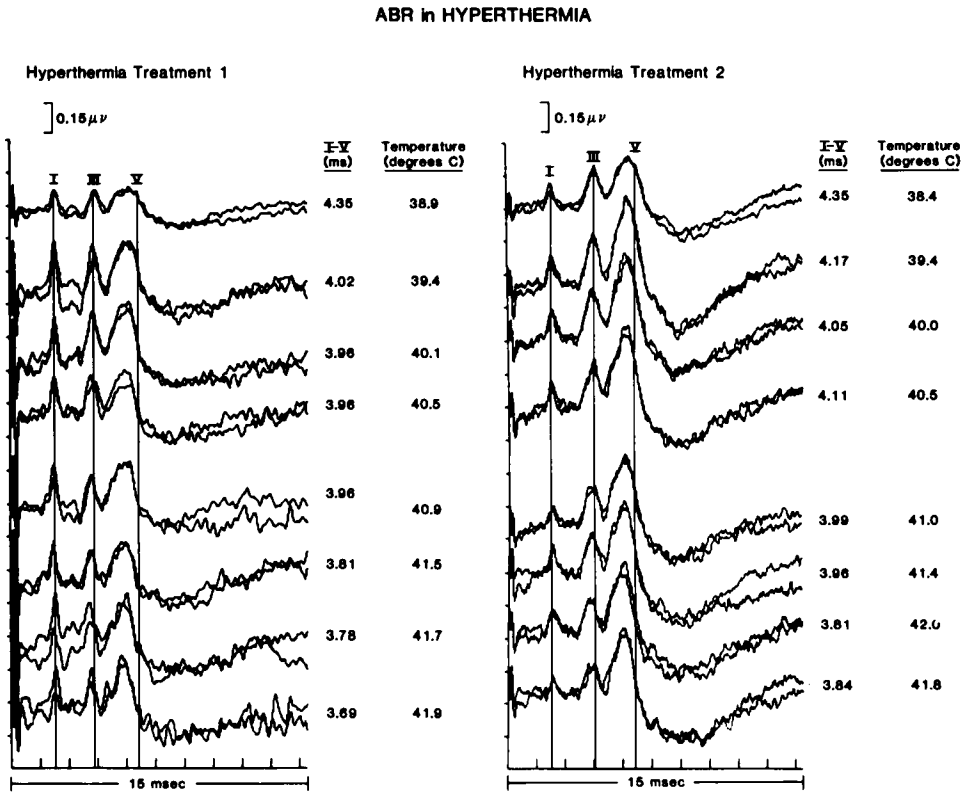

Figure 3. Auditory brain stem response (ABR) waveforms recorded from a 26 yr old male during two separate sessions of hyperthermia treatment for cancer (case 2). Waveforms on each test day are for left ear stimulation. ABR and physiologic data are displayed in Table 5.

important to mention at this point that during intraoperative (versus ICU monitoring) auditory electrophysiologic monitoring, particularly of ECochG, rectal temperature may be an inadequate index. If the middle ear cavity is exposed surgically, cochlear temperature can be significantly lower than temperature measured rectally (Nuttall & LaRouere, 1980).

Early clinical experiences with whole body hyperthermia therapy for advanced cancer suggested a mortality rate as high as 16%, and even more recent reports describe post-treatment CNS dysfunction as an infrequent complication (Barlogie et al, 1979). Our experimental investigations with rats showed that acute CNS dysfunction accompanying body temperature elevation (up to 43°C) can be reflected by ABR outcome. We are currently exploring the usefulness of auditory evoked responses as a clinical monitor of CNS status during hyperthermia therapy. The decreased I–V latency and unchanged wave amplitude reported for case 2 is representative of the expected and consistent effect of increased temperature on the ABR in man. It is likely that little or no decrease in latency during hyperthermia should be considered as strong evidence of brain stem dysfunction. Certainly, any latency increase in hyperthermia would be an ominous sign.

Guidelines exist for taking hypothermia into account in ABR interpretation (e.g., Britt, Lyons, Ryan, Saxer, & Rossi, 1983; Stockard, Sharbrough, & Tinker, 1978). Our clinical test protocol calls for a somewhat conservative correction factor for the wave I–V latency of 0.2 msec (200 μsec) for every degree of body temperature below average normal (37°C). Applying this correction to data for case 1 in the present paper resulted in ABR wave I–V latency values that were, on several occasions, at the upper limit of the normal region, but were never clearly abnormal bilaterally.

There are no published clinical guidelines for correction of ABR latency values in hyperthermia. We have consistently found a I–V latency decrease of 0.5 to 0.6 msec over the temperature range of 38 through 42°C in young male and female patients (8, to date) with no CNS pathology undergoing hyperthermia therapy. Based on this experience, we recommend a correction factor for the I–V latency interval of 0.15 msec for each degree of increased body temperature. We define increased temperature as the patient's value versus the average normal 37°C or, in serial ABR recording, the current temperature compared to previous temperature values for the patient.

In closing, the two cases described herein underscore the importance of documenting body temperature when recording ABRs serially. Patients with auditory brain stem integrity, yet unusually high or low body temperature, may have ABR latency values outside of the clinically normal region.

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Address reprint requests to James W. Hall III, Ph.D., Division of Hearing and Speech Sciences, Department of Otolaryngology, School of Medicine, Vanderbilt University, Nashville, TN 37232.

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