Children with varied types of severe brain injuries require intensive care in the acute period after the injury. Severity of brain injury is typically defined on the basis of the neurological examination, and often graded with a relatively simple scale of neurological functioning, such as the Glasgow Coma Scale (GCS) (Figure 1 and references one, two, and three for detailed explanation). Patients with severe brain injury have GCS scores of 8 or less and are, by definition, comatose. That is, they are unconscious and have limited, or no, response to sensory stimulation. A patient with a GCS score of 4 to 5, for example, does not open eyes to command or even to painful stimulation (such as pinching), has only a simple and abnormal motor response, such as extension of the arms, to painful stimuli, and invariably does not vocalize, since a basic component of intensive care is intubation for mechanical ventilation (in an attempt to maintain adequate respiratory function artificially). Definition of the acute period after injury varies among institutions. However, a broad definition includes the first 2 to 3 weeks after hospital admission for a severe brain injury, when intensive care is required. The reader is referred to recent publications for general information on assessment and management of acute brain injury.1,3,4,5

We have accumulated experience with sensory-evoked response (SER) measurement of children whose etiologies for brain injury were quite diverse, as shown in Table 1. The most common type of injury is traumatic head injury.6,7 The actual incidents causing traumatic head injury have included motor vehicle accidents, particularly involving unrestrained children, automobile or auto-bicycle accidents, falls, and gunshot wounds, either accidental or self-inflicted. Whether or not patients with meningitis are initially evaluated in the pediatric intensive care unit (ICU) depends on the management protocol of the hospital attending staff. Although SER assessment of acute meningitis can contribute to description of neuropathology,8,9 we are often called on to evaluate postmeningitic hearing status after the acute phase.

Hypoxic-ischemic insults refer to injuries resulting in inadequate delivery of oxy-
Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Eye Opening</th>
<th>Verbal Response</th>
<th>Motor Response</th>
<th>Total Trauma Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score</td>
<td></td>
<td></td>
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<tr>
<td>4</td>
<td>4</td>
<td>4</td>
<td>1-16</td>
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<tr>
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</tr>
<tr>
<td>1</td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

Figure 1. Glasgow Coma Scale (GCS) for grading severity of brain injury and depth of coma.

TABLE 1. Summary of Clinical Experience with Sensory-Evoked Response Measurement, Including Auditory Brainstem Response in the Pediatric Intensive Care Unit

<table>
<thead>
<tr>
<th>Clinical Entity</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic head injury</td>
<td>102</td>
<td>43</td>
</tr>
<tr>
<td>Meningitis</td>
<td>62</td>
<td>25</td>
</tr>
<tr>
<td>Hypoxic-ischemic insult</td>
<td>23</td>
<td>10</td>
</tr>
<tr>
<td>Seizures</td>
<td>18</td>
<td>8</td>
</tr>
<tr>
<td>Hydrocephalus*</td>
<td>15</td>
<td>6</td>
</tr>
<tr>
<td>Other</td>
<td>17</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>257</td>
<td>100</td>
</tr>
</tbody>
</table>

*Excludes perinatal hydrocephalus

gen or blood to the brain. Causes include asphyxia in general, and near-drowning incidents, strangulation, cardiac arrest, severe respiratory distress, and, especially in neonates, intraventricular hemorrhage. Less common in children, but also included among hypoxic-ischemic insults, are stroke and intracranial bleeding secondary to ruptured aneurysm or arterial venous malformation. Finally, SEPs are sometimes useful in neurological evaluation of children with seizure disorders and often contribute to diagnosis and management in pediatric hydrocephalus. We reviewed the role of the auditory brainstem response (ABR) in hydrocephalic children, including neonates, in two recent publications.

In this article we describe the rationale and application of SEPs, primarily for the auditory modality, in evaluation of peripheral and central nervous system (CNS) status of acutely brain-injured children in the ICU.

EVALUATION OF PERIPHERAL AUDITORY DISEASE

Peripheral auditory deficits are common among children managed in the ICU, as summarized in Table 2. Evidence of at least minimal sensory integrity is essential for meaningful exploitation of evoked responses in evaluation of CNS status after severe brain injury. For example, a distinct and reliable ABR wave I component (eighth nerve response) is required before inferences can be made regarding brainstem functioning. Likewise, with median nerve somatosensory-evoked response (SSER) recordings an Erb's point component (arising from brachial plexus) assures that the peripheral sensory nerve is reasonably intact and permits evaluation of CNS conduction time. It is always important, therefore, to verify that severe peripheral deficits are not invalidating CNS assessment.

TABLE 2. Summary of Acute Clinical Entities Managed in the Intensive Care Unit with Possible Auditory Deficits

<table>
<thead>
<tr>
<th>Clinical Entity</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic head injury</td>
<td>Otologic disease in 67 percent of adults with severe head injury</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>Up to 25 percent of children have no measurable ABR</td>
</tr>
<tr>
<td>Meningitis</td>
<td>55 percent of children with bacterial meningitis have evidence of auditory deficit by ABR</td>
</tr>
<tr>
<td>Ototoxicity</td>
<td>More than 20 percent of severely burned children treated with ototoxic drugs in an ICU, develop serious permanent sensorineural auditory impairment</td>
</tr>
</tbody>
</table>

Case 1:

A 4-year-old child in the hospital for 3 weeks following a motor vehicle accident sustained a severe head injury. She was obtunded at admission, with no measurable auditory brainstem response. Over the next two months, the patient gradually improved and became fully alert, with minimal neurological sequelae. The auditory brainstem response remained non-measurable, although she was able to communicate through simple vocalizations.

Table 3 shows the correlation between the severity of head injury and the outcome of auditory brainstem response assessment.
lymphocytic meningitis. Given the well-known peripheral sequelae associated with the clinical entities displayed in Table 2, auditory-evoked responses, mainly the ABR, are often requested specifically to identify auditory sensitivity deficits, and determine, if possible, the type (conductive, sensorineural, mixed) and degree of loss. ABR test principles and practices commonly used for auditory sensitivity assessment with children in general are, with occasional modifications, appropriate for the brain-injured child. The following discussion will be limited to this application of the ABR.

We have emphasized elsewhere that ABR recording in an ICU environment, although not an impossible task, is often technically demanding. ABR measurement is particularly challenging for patients suspected of having peripheral deficits. Measurement problems in this population and techniques that we have found useful in solving the problems are summarized in Table 3. The objectives common to all ABR recordings in the ICU are confident identification of waves I, III, and V bilaterally at a relatively high-intensity level and, in the evaluation of peripheral function, documentation of a reliable wave V, minimally, for stimulus intensity levels close to actual auditory sensitivity in the 1000 to 4000 Hz region.

**CASE 1: TRAUMATIC HEAD INJURY**

A 4-year-old girl fell approximately 6 feet from a ladder onto sand and struck her head in the occipital region. Initially, she was obtunded, combative, and unresponsive to verbal commands. She lost consciousness briefly. There was a history of hydrocephalus since birth, with probable tracheostomy and questionable communication of the ventricles with the spinal canal. Her preinjury hearing was normal and speech-language development was age-appropriate. On transfer to the hospital emergency center via helicopter from an outlying hospital, physical examination showed dysconjugate gaze and pupils 1 mm in size bilaterally, round and unresponsive to light stimulation. CSF was draining from the left ear. The patient still did not respond to verbal commands. GCS (Fig. 1) was less than 8, confirming a severe head injury.

Emergency computed tomography (CT) scanning revealed a basilar skull fracture primarily on the right, right hematomyelia, pneumocephalus, fracture of the squamous portion of the left temporal bone, and a transverse fracture of the pterional ridge through the temporal bone. Moderate dilation of the lateral and third

<table>
<thead>
<tr>
<th>Problem</th>
<th>Solutions</th>
</tr>
</thead>
<tbody>
<tr>
<td>No response, including absence of wave I</td>
<td>Perform listening check for adequacy of stimulus Use maximum stimulus intensity level Use bone-conduction stimulus verify proper electrode selection Use stimulus (such as 1000 Hz) Use insert transducers Extend filter settings Obtain otologic consultation</td>
</tr>
<tr>
<td>Response uninterpretable due to poor morphology</td>
<td>Decrease stimulus rate Use simultaneous multielectrode arrays (see references) Increase stimulus intensity level</td>
</tr>
<tr>
<td>Abnormal interspace latencies</td>
<td>Correct for temperature Correct for age Document medications Document physiological parameters</td>
</tr>
<tr>
<td>Excessive electrical artifact</td>
<td>Assess excellent ground electrode Use short (less than 2 feet) electrode wires Assess low and balanced electrode impedance Restrict filter settings After stimulus rate Repeat testing at later time</td>
</tr>
<tr>
<td>Excessive muscle artifact, including postauricular</td>
<td>Request sedatives for patient Decrease stimulus intensity Use alternative electrode arrays Restrict filter settings Increase number of averages Eliminate artifact rejection</td>
</tr>
</tbody>
</table>
ventricles was observed, with possible aque-ductal stenosis and hydrocephalus of the noncommunicating type.

Initial ABR assessment was carried out 1 day after the accident in the pediatric ICU. As illustrated in Figure 2, there was no ABR for air- or bone-conduction click stimuli presented at maximum intensity levels (95 dB hearing level [HL] referenced to adult behavioral click threshold for air conduction) and 55 dB for bone conduction. The same pattern was recorded in three subsequent ABR assessments during a 1-month period postinjury. Within this time, the patient regained consciousness, becoming alert and oriented, yet remained unresponsive to verbal commands. She developed left ear otitis media (Pseudomonas by culture), which was treated with antibiotics. Hydrocephalus was treated surgically (ventricular-peritoneal shunt).

Follow-up auditory evaluation and management are summarized in Figure 3.

Behavioral testing consistently failed to yield a response to maximum verbal and speech stimuli on the left ear. Initially (3 weeks through 3 months), there was a se vere-to-profound sensorineural auditory impairment on the right. Immittance audiometry indicated normal middle ear function bilaterally. Over the course of 9 months, pure-tone sensitivity gradually showed improvement. Recommendations for amplification and educational placement were made during the first week postinjury, and within 2 months a hearing aid was fitted to the right ear. Changes in amplification were made as required with improvement in auditory status. Aided hearing was adequate for communication (as shown in Figure 3) and the patient's speech and language remained intact.

Comment

With this patient, basilar skull and temporal bone fractures secondary to head injury resulted in serious auditory impairment. We have demonstrated recently, however, that ABR abnormalities are not invariably associated with temporal bone fractures. In fact, one-half of the patients (at least adults in the series reported) with CT-confirmed fracture had a clear and reliable ABR to air-conducted stimuli at high-intensity levels. This somewhat unexpected finding may, in part, be related to changing concepts regarding the nature of temporal bone fractures. That is, high-resolution CT scanning of the skull suggests that the traditional categorization of temporal bone fractures as tr

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When his mother
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...fractures as transverse versus longitudinal, and the subsequent patterns of cranial nerve and presumably auditory deficits, may not apply in many head-injured patients. Furthermore, by utilizing techniques already described (Table 3) and in recent publications, 8,11,12,23 we find that it is often possible to record an interpretable ABR from the majority of these patients. As an aside, ABR evidence of serious hearing impairment can have an impact on neurosurgical assessment and management as well. In this case, and others, 3,17-19 the GCS has undergone reinterpretation once the patient’s unresponsiveness to verbal commands could be attributed to a peripheral auditory versus neurological basis.

It is also important to keep in mind that serial ABR and behavioral audiometry is usually needed to define confidently long-term auditory status of the head-injured patient. In this case, hearing impairment was apparent and acute due to the trauma directly. However, auditory status clearly improved over time. Audiological management of patients with bilateral hearing loss, 3-5 (and 3-6) half of the patients, and parents (and caregivers) alike, have a clear and readily apparent stimuli at high-frequency stimulus levels. However, unexpected results related to changing patient’s condition and treatment. High-resolution images do not suggest that the patient had incurred damage to the temporal bone.

Comment

There are recent clinical reports of ABR in assessment of auditory and neurological status of infants secondary to hypoxic-ischemic insult or other acute encephalopathy. 24-28 ABR findings may range from a response that has normal latency and amplitude of major components, to decreased amplitude for wave V versus I and increased interwave latencies, to a wave I only, with no subsequent evidence of auditory brainstem activity. In our patient, clinical factors clearly put the child at risk for CNS dysfunction, and the physical examination, EEG, and VERs confirmed...
neurological deficits. ABR findings, however, were consistent with integrity of peripheral and central auditory function. In agreement with the experience of the investigators just cited, this case illustrates that a normal ABR in children with a hypoxic-ischemic pathophysiologic condition often implies favorable outcome. We hasten to point out, however, that principles of ABR interpretation for such nontraumatic brain injuries are in some respects distinctly different from those in traumatic head injury. This point will be discussed in greater detail in the next section. Our finding of peripheral auditory integrity in this child was particularly important for the multidisciplinary team of pediatricians, social workers, and physical therapists managing the patient, in view of the evidence of significant visual deficits.

EVOKED-RESPONSE PATTERNS IN CENTRAL NERVOUS SYSTEM DISEASE

The ABR provides electrophysiologic information about brainstem function that can complement clinical findings from the neurological examination (see Hall and Tucker for review). In combination with SSERs and VERs, ABR is a clinically feasible and diagnostically powerful means of assessing acute CNS status in brain-injured children. ABR can be applied with varied pediatric CNS conditions, including demyelinating and infectious diseases, hypoxic-ischemic encephalopathies, acute intracranial cerebrovascular accidents (such as intraventricular hemorrhage), and hydrocephalus. For each of these disorders, ABR abnormalities are useful in documenting the effect of basic brain pathophysiologic processes, such as ischemia, hypoxia, and increased intracranial pressure (ICP), on neuron functional status.

ABR measurement for the purpose of CNS evaluation is, of course, just as challenging as it is for peripheral auditory evaluation. The special measurement techniques that were summarized previously in Table 3 are therefore equally applicable. Two additional factors must be taken into account. One is patient age. Evoked responses for each modality (auditory, somatosensory, and visual) are immature at birth and show age-related changes associated with maturation and myelination of the CNS. These effects are particularly important to consider in interpretation of interwave latencies and amplitudes. A second factor is the relationship among ABR and SSERs versus VERs. Often, a single modality does not adequately describe CNS status. Obviously, SSER or VER measurements or both, are extremely useful in evaluation of CNS status for patients with severe peripheral otologic disease and no ABR, including wave absence of the wave I (eighth cranial nerve) component. However, the overall integrity of the nervous system may in some clinical entities be more accurately described with a battery of evoked response modalities, because of differences among modalities in anatomy and sensitivity to pathophysiology. For example, VERs are, as already noted, especially sensitive to brain dysfunction resulting from increased ICP in hydrocephalus. Evoked response findings for more than one modality are also often better than those for the ABR alone in estimating long-term outcome after brain injury. Finally, for confirmation of brain death with evoked responses, multimodality sensory stimulation provides valuable additional information on comprehensive CNS integrity, rather than the functioning of one portion of the CNS.
of clinical studies have confirmed the usefulness of ABR in children with acute brain injuries,\textsuperscript{2,10,11,18,25,26,28-29,32-33} We have identified three uses of ABR in this population: (1) monitoring with serial recordings during the acute period after injury, in an attempt at early detection of potentially reversible changes in CNS status; (2) documenting irreversible changes in CNS status, often in combination with other SERs, thereby estimating long-term neurological outcome; and (3) confirmation of brain death. Of these applications, monitoring is perhaps the most promising and clinically useful. We will, therefore, now review the clinical concepts important in understanding monitoring of the brain-injured patient.

Monitoring patient status is a fundamental and indispensable component, and the foremost challenge, of intensive care after brain injury. During this acute period, the patient is at risk for secondary injury (such as increased ICP, intracranial hemorrhage, hypoxia, and hypotension). Standard physiological parameters monitored include cardiac output, respiratory variables, body temperature, blood pressure, ICP, blood gases, and other blood chemistry data. The overall objective of neuromonitoring in particular is early identification of reversible CNS events that have the potential to result in adverse acute or long-term neurological outcome.

SERs, including the ABR, have contributed in unique ways to neuromonitoring. Given recent advances in intensive care and increasing sophistication of techniques for evaluating CNS status, it is reasonable to ask why another monitoring modality is needed. The answer is that, unlike other monitoring modalities, SERs reflect the function of neurons in specific regions of the CNS and are, to a large extent, independent of the medical therapies typically used in acute brain injury.\textsuperscript{51} CT is a measure of structure, not function and is actually not a feasible monitor, since the scanning cannot be done at bedside and moving an acutely brain-injured patient to the scanner regularly (multiple times per day) is medically contraindicated. The general physiological parameters that are monitored (already listed) are invaluable in management of acute brain injury, but do not necessarily correspond directly to neuronal physiology and usually do not provide information on different parts of the CNS, such as brainstem versus temporal lobe of the cortex. By far the most common and essential neuromonitoring technique is the clinical neurological examination. Unfortunately, some of the medical therapies used with the majority of acutely brain-injured patients, such as neuromuscular blockers, sedatives, and high-dose barbiturates,\textsuperscript{12,13,5} suppress brainstem and cerebral neurological signs (such as corneal reflex, oculocephalic reflex, gross motor reflexes, and even the pupillary response to light). The neurological examination is especially difficult and limited in the brain-injured infant, whose CNS is immature.\textsuperscript{52}

The following two case reports illustrate applications of ABR in acute pediatric brain injury.

CASE 3: CORRELATION OF ABR WITH CLINICAL FINDINGS AND OTHER EVOKED RESPONSES

The patient was a 2-month-old female involved in a motor vehicle accident. She was riding in an unsecured car seat and was thrown about the car at impact. Admission CT scanning revealed a frontal hematoma extending into the left sylvian fissure region of the temporal lobe of the cortex, with temporal lobe edema. After emergency therapy and stabilization, she was transferred to the pediatric ICU.

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Initial evoked response evaluation was carried out within 10 hours after the accident. At the time, vital signs were reported as normal. Pupils were equal bilaterally and reactive to light. ICP, as indicated by a monitor inserted at the fontanelle, was 6 mmHg. As shown in Figure 5, VER and SSER peripheral components were present, but cortical components were not observed. Normally, there is a wave within the first 100 msec of the VER, indicating retinal and optic nerve integrity, and then a
large positive voltage wave after 100 msec, reflecting activity in the occipital lobe after the flash photic stimulation. The upper SSER waveform in Figure 5 has a distinct and reliable Erb's point component (N9) confirming peripheral (median nerve) integrity and an adequate stimulus. In the lower waveform, the expected negative-polarity component at approximately 20 msec, the thalamic-parietal cortex component, is clearly not present. Initially, an ABR was reliably recorded bilaterally, with interwave intervals within age-matched normal limits (top waveform in Figure 6).

In view of the markedly abnormal cortical SER findings, the attending physician removed and recalibrated the ICP monitor. On replacement of the monitor, ICP was 109 mmHg (for clinical purposes, an ICP greater than 20 mmHg is considered abnormal). A repeat CT scan showed white matter edema in the posterior fossa and extending into the midbrain (Fig. 7). The patient was managed aggressively for the next 3 days, but the SSER and VER cortical components were repeatedly absent and ABR interwave latencies became progressively and excessively prolonged. On day 4 after injury, there was no detectable ABR (bottom tracing in Fig. 6). An EEG at this time was isoelectric. Support systems were withdrawn and the patient died.

Comment

SERs may provide ICU medical personnel with information not available from conventional clinical data. In some cases, as
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Illustrated by this patient, the patterns of SER results leads to reappraisal of conventional data and important alterations in medical or surgical management strategies for the acute brain-injured child.23,10,11,12 No single clinical parameter will invariably be validly monitored in the ICU. Therefore, complementing clinical data with SERs strengthens the overall ongoing assessment of CNS status during the critical period after injury.

This case also demonstrates that the initial ABR does not always adequately describe CNS status. In our experience, initial ABR interwave latency values are within normal limits for more than 70 percent of severely brain-injured patients, if the assessment is carried out within the first 48 hours. There may be widespread cerebral damage and dysfunction, as evident on CT scans and cortical SERs, in such patients. However, the normal initial ABR becomes a baseline neurophysiological index by which subsequent serious CNS deterioration can be documented serially. Finally, the progressive loss of a once-normal ABR is useful in determining the need for last-resort, aggressive management, prognosis of unfavorable outcome, or, when all therapeutic attempts are unsuccessful, timely confirmation of brain death. This latter application of ABR will be discussed in greater detail later. The progressive return of normal SERs when initial findings are abnormal, conversely, contributes information that is useful in planning rehabilitation. There are numerous articles describing the role of SERs in predicting neurological outcome among survivors of severe brain injury (see Hall and Tucker4,16 for review).

CASE 4: CONFIRMATION OF BRAIN DEATH

A 15-month-old male was brought to the emergency center by the parents after reportedly falling. The mother's incomplete recollection of the details of the accident, and old bruises on the child's body, suggested abuse. On admission, the patient was lethargic. Pupils were sluggishly reactive to light. Emergency CT scanning indicated a subdural hematoma in the right frontal region. After surgery for evacuation of the hematoma, he was transferred to the pediatric ICU.

Evoked responses, assessed within 24 hours after the injury, are illustrated in Figure 8. There was no evidence of cortical activity in the VER and SER waveforms (top two panels of the figure). Peripheral components were reliably observed for each modality, implying ocular and median nerve integrity. With left ear stimulation, ABR waves I and possibly II were recorded, providing evidence of sensorineural functioning, but subsequent brainstem components of the response were not present. On the left, there was no ABR. Multimodality SERs showed widespread and apparently fatal brain injury. Based on this neurophysiological evidence of CNS inactivity, plus unresponsiveness by neurological examination, the child was removed from life support 2 hours after testing was completed and died.

Comment

Clinical diagnosis of brain death has been the subject of considerable discussion and, indeed, controversy in recent years.11,15,35-38 In this diagnosis, it is important to differentiate among cerebral unresponsiveness, brainstem unresponsiveness,
and actual brain death. Inactivity of cerebrum, brainstem, and cerebellum are required for brain death. Cerebral inactivity, in the presence of brainstem integrity, is common in acute severely brain-injured patients. This pattern may result in prolonged vegetative state, but may also be due to metabolic coma or the effects of recreational drugs or CNS suppressant medical therapies and is not invariably associated with permanent inactivity or vegetative state. Brainstem inactivity is virtually always incompatible with unassisted life.

Many of the traditional criteria for definition of brain death required clinical observation of brainstem and cerebral unresponsiveness for an extended period of time, such as 12 or 24 hours. The time criterion was included to eliminate effectively the likelihood of a false-positive error in determination of brain death, that is, declaring a living person dead. In the era of organ transplantation, however, the time criterion is unacceptable. The EEG, which until recently was a confirmatory procedure in brain death diagnosis, is now not considered a clinically feasible and valid index of brain integrity in the ICU, for technical and neuroanatomical reasons.

Along with bedside nuclear cerebral blood flow techniques, SERs are assuming an important role in timely confirmation of brain death. The ABR is uniquely valuable in this regard, since it documents brainstem inactivity (versus cerebral inactivity), an essential component of the definition of brain death, and is independent of the therapies that invalidate the neurological responses.
nological examination, such as neuromuscular blockers (pancuronium bromide, metocurine), sedatives (morphine, haloperidol), and high-dose barbiturates (pentobarbital). The audiologist involved in ABR measurement for documentation of brainstem integrity of patients with potentially fatal injuries must keep in mind three important points. First, a peripheral (wave I) component must be reliably observed to rule out injury-related, or preexisting, auditory impairment as the basis for an absent ABR. The alleged lack of a wave I in many patients evaluated for brain death has been the source of criticism of the ABR as a confirmatory method. Most investigators, however, deferred SER measurement until days after injury, and when all other criteria for brain death had been met. In our experience, an ABR wave I is present in more than 95% of patients evaluated within the first 24 hours after injury, if techniques summarized previously in Table 3 are used. Typically, we record a normal ABR initially and then in some patients the response gradually deteriorates, eventually resulting in a wave I only, or no response. By virtue of his or her training, the audiologist is well-suited to verify peripheral integrity in the brain-injured patient.

Second, temperature must always be noted before ABR findings are interpreted. It is conceivable that a markedly abnormal or even undetectable ABR may be found in deep hypothermia. Third, there is a distinction between the significance of ABR findings for traumatic head injury, in which brainstem dysfunction usually evolves secondary to increased ICP and supratentorial compression of cerebral blood vessels and brainstem structures, and hypoxic-ischemic insults. With traumatic injuries, an ABR characterized by only a wave I is strongly correlated with absence of cerebral blood flow and compatible with brain death. In patients, children or adults, with brain injury secondary to asphyxia, hypothermia, cardiac arrest, other hypoxic-ischemic causes, and demyelinating diseases, such as multiple sclerosis, the finding of only a wave I in ABR measurement is not necessarily a reflection of complete brainstem inactivity. Although this pattern is, of course, always evidence of brainstem disease and often implies poor prognosis, there are reports of patients surviving for prolonged periods of time, or even showing an apparently complete reversal of the abnormality with good neurological recovery.

ROLE OF THE AUDIOLOGIST IN THE PEDIATRIC INTENSIVE CARE UNIT

The hospital-based audiologist who is willing to assume clinical responsibility for recording and interpreting evoked responses for the auditory, somatosensory, and, perhaps, visual modalities can be a valued member of the pediatric ICU team. There are at least five prerequisites for a successful SER clinical service in the ICU: (1) expertise in evoked response recording, the ability to interpret data quickly and accurately, and the professional independence required to report findings to attending physicians; (2) a firm grasp of evoked response neuroanatomy and neurophysiology; (3) an understanding of the mechanics and pathophysiology of brain injury; (4) familiarity with terminology and techniques of conventional monitoring and management of acute brain injury; and (5) the ability to work well under a reasonable amount of time pressure and stress, and work closely with varied medical personnel. The reader is referred to a recent text for comprehensive and practical guidance on establishing a SER service in the ICU.

Acknowledgments

We thank Drs. Rolf Habersang, Jeff Cone, and Wayne Paulus of Texas Technical Health Science Center in Amarillo and Dr. Judy R. Mackey-Hargadine of the University of Texas Medical School in Houston for assistance in data collection. Preparation of this article was supported in part by the 1987 University of Mary.
REFERENCES

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ARTICLE FOUR
SELF-ASSESSMENT QUESTIONS

1. One indicator of severe head injury is:
   (a) a Glasgow Coma Scale score of greater than 12
   (b) hematomyranum
   (c) a Glasgow Coma Scale score of less than 8
   (d) a temporal bone fracture
   (e) admission to the pediatric intensive care unit

2. Which one of the following abbreviations refers to a radiologic procedure for evaluation of head injury:
   (a) PaO2
   (b) ABR
   (c) ICU
   (d) CT
   (e) PTA

3. Which one of the following criteria must be met for diagnosis of brain death:
   (a) inactivity of cerebrum, cerebellum, and brainstem
   (b) Glasgow Coma Scale score of 3
   (c) unresponsiveness to sensory stimulation
   (d) dependence on ventilatory support
   (e) need for cardiopulmonary resuscitation

4. Which set of clinical problems are associated with auditory deficits of patients in an pediatric intensive care:
   (a) otitis media and tympanic membrane perforation
   (b) otosclerosis and ossicular chain fixation
   (c) Meniere’s disease and endolymphatic hydrops
   (d) presbycusis and acoustic trauma
   (e) hydrocephalus and meningitis

5. Which set of pathophysiologies often cause secondary brain damage during the acute period following head injury:
   (a) increased intracranial pressure and hypoxia
   (b) temporal bone fracture and osseous chain disruption
   (c) heart attack and hypertension
   (d) cervical fracture and hemiparesis
   (e) hypothermia and hyperthermia