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Evoked Potentials in Head Injury and States of Increased Intracranial Pressure

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Summary: Recent clinical studies have addressed the utility of sensory evoked potentials (auditory, somatosensory, and visual) in head injury. The clinical significance of evoked potentials in head trauma, states of increased intracranial pressure, and post-traumatic syndrome is discussed. Emphasis is placed on up-to-date studies discussing evoked potential correlation with clinical findings, lesion localization, intracranial pressure, brain herniation, and prognosis. **Key Words:** Sensory evoked potentials—Head injury—Clinical findings—Intracranial hypertension—Prognostic indicator.

Electrophysiological monitoring has been advocated in intensive care settings as comparable to the clinical examination in reliable assessment of brain function. Evoked potential (EP) recording is safe, noninvasive, and may be performed at the neurotrauma bedside (generally requiring less than 1 h). In a situation in which adequate communication with the patient is impossible, EP monitoring may provide significant functional information that could not be known otherwise. EP recording has been shown to be a helpful clinical tool in the pediatric as well as the adult head-injured population (Sohmer et al., 1974; Nodar et al., 1980; Hecox et al., 1981; Hall et al., 1985). EPs have been commonly used not only as a diagnostic test but also as strong prognostic indicator (Greenberg et al., 1977*a,b,* 1981; Lindsay et al., 1981; Narayan et al., 1981; Hall and Mackey-Hargadine, 1984; Newlon and Greenberg, 1984; Karnaze et al., 1985; Cant et al., 1986).

Sensory EPS represent the neurophysiological responses of sensory pathways to appropriate sensory stimuli. These EPs can be classified according to the type of stimulation into somatosensory EPs (SEPs), auditory EPs (AEPs), and visual EPs (VEPs). EPs are also classified by time of occurrence after stimulation into short-, middle-, and long-latency potentials. Short-latency potentials include cochlear and brainstem (<10–

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TABLE 1. *Possible generator sources of BAEP components*

BAEP component	Generator source	Level
I	Auditory nerve	Cochlea-pontomedullary entry
II	Auditory nerve/cochlear nucleus	Pontomedullary
III	Olivary complex	Caudal pontine
IV	Lateral lemniscus	Rostral pontine/midbrain
V	Inferior colliculus	Midbrain
VI	Medial geniculate body	Thalamic
VII	Thalamocortical radiation	Thalamic/thalamocortical

15 ms) AEPs (BAEPs). The electrocochleogram (ECochG) reflects the electrical response of the cochlea and auditory nerve to acoustic stimulation. The BAEP is largely or completely generated by cochlear and brainstem auditory pathways. Short-latency potentials also include short-latency somatosensory potentials (SSEPs) (<25–50 ms according to the peripheral nerve being stimulated). On the other hand, middle- and long-latency potentials are considered cortically generated and called near-field potentials (SEPs, AEPs, and VEPs). Long-latency sensory EPs provide evidence for hemispheric function.

The possible generator sources of BAEPs and SSEPs are listed in Tables 1 and 2 (Lev and Sohmer, 1972; Sohmer et al., 1974; Buchwald and Huang, 1975; Starr and Achor, 1975; Cracco and Cracco, 1976; Starr and Hamilton, 1976; Thornton and Hawkes, 1976; Jones, 1977; Stockard and Rossiter, 1977; Chiappa et al., 1979; Hashimoto et al., 1979; Noel and Desmedt, 1980; Allison and Hume, 1981; Anziska and Cracco, 1981; Brown et al., 1981; Lueders et al., 1981; Lastimosa et al., 1982; Nakanishi et al., 1982; Chiappa, 1983; Lueders et al., 1983; Mauguière et al., 1983*b*; Hashimoto, 1984; Eisen and Aminoff, 1986; Chiappa, 1987). Less is known concerning the exact origin of middle- and long-latency potentials, often believed to reflect thalamocortical projections, primary cortical association areas, or secondary cortical association areas. VEPs are generated in the occipital cortex, and their components are thought to reflect electrical activity of the visual system (Creutzfeldt and Kuhnt, 1967; Monnier, 1974; Celesia, 1982; Halliday, 1982).

TABLE 2. *Possible generator sources of SEP components following median nerve stimulation at the wrist and posterior tibial nerve at the ankle*

SEP component (ms) (P/N)		
Median	Tibial	Generator source/level
9	—	Brachial plexus
11	18–20	Spinal cord entry
13	27	Dorsal column/dorsal column nuclei
14	30	Medial lemniscus/brainstem
18	32	Thalamus/thalamocortical radiations
22	36–38	Parietal somatosensory cortex

Short-latency potentials are very resistant to environmental and subject variations, whereas middle- and long-latency potentials show larger intersubject and intrasubject variability. The latter potentials are affected by state of consciousness or attentiveness of the subject, sedatives, barbiturates, and anesthetic agents (Cracco and Cracco, 1976; Hillyard et al., 1978; Sohmer et al., 1978; Chiappa et al., 1979; Halliday, 1982; Chiappa, 1983; Grundy, 1985). As a consequence short-latency potentials are more stable and have been more widely used in the intensive clinical setting with critically ill head-injured patients.

Latencies of the different peaks and interpeak latencies (IPLs) of the BAEP are more consistent and stable than waveform amplitudes in each individual under normal conditions (Coats and Martin, 1977; Coats, 1978; Sohmer et al., 1978). Latency and IPL changes may indicate more serious damage than amplitude changes (American Electroencephalographic Society, 1984). The I–V IPL and N13/N14–N20 latency reflect brainstem auditory or somatosensory central transmission time (CTT). IPL or CCT prolongation indicates pathological dysfunction of the central auditory system or somatosensory system and is considered a sensitive criterion for central (but not peripheral) conduction changes (Starr, 1976; Chiappa et al., 1980; Hume and Cant, 1981; Chiappa, 1983; Cant et al., 1986; Eggermont and Don, 1986; Stockard et al., 1986; Chiappa, 1987; Starr, 1987).

Delay in latency may indicate slowed conduction, whereas small amplitude may represent a decrease in the number of active neurons or, possibly, increased extracerebral resistance to recording the signals (Starr and Achor, 1975). Asynchrony of conduction may cause a variable decrease in amplitude, increase in latency, and waveform distortion. Pathological insults affecting a conducting neuronal pathway such as disruption, ischemia, or distortion/compression may alter EPs (Nagao et al., 1983, 1984; Grundy, 1985).

Integrity of peripheral sensory function should be assessed (when possible) before considering the EP results (Starr, 1976; Chiappa, 1983). This may be especially important in trauma. Peripheral assessment is generally performed by recording early components, verifying the adequacy of the sensory input. For example, appropriate ophthalmological examination and electroretinal testing is recommended in VEP testing. When recording BAEPs, ideally peripheral hearing should be verified with an audiogram. Middle ear impedance and pressure abnormalities may be found in comatose head-injured patients and may confuse the interpretation of BAEP findings, especially if absolute latencies, rather than IPLs, are considered (Hall et al., 1982, 1985). External or middle ear conductive hearing losses are perceived as a decreased stimulus intensity, and post-traumatic hemotympanum may complicate BAEP testing in head trauma. In SEP monitoring, slow peripheral nerve conduction velocities due to peripheral nerve injury, extremity length, or hypothermia may account for increased absolute latencies of SEP peaks (Cracco and Cracco, 1976; Jones, 1979; Chiappa et al., 1980; Allison and Hume, 1981; Anziska and Cracco, 1981; Newlon et al., 1982; Chiappa, 1983).

To enhance yield of information and display of EPs, serial recording with multichannel electrode arrays may be used to improve the visualization of certain peaks and verify multiple afferent pathways. Examples are the indifferent contralateral or noncephalic (neck) electrodes for waves IV and V of BAEP and SSEP waves P/N11–N18. Serial

EP recording is essential for early detection of insults and for demonstration of progression or regression of EP abnormalities. Repeated testing may be important for timely medical management or for monitoring the effectiveness of treatment (Hall et al., 1982, 1985). Measurement of latency intensity and amplitude intensity (input–output functions) of waves I, III, and V of BAEP and IPLs at multiple intensities may enhance the utility of BAEP in patients with audiological or neurological problems (Coats and Martin, 1977; Coats, 1978; Despland, 1987; Stone et al., 1987; Ghaly et al., 1988). An increase in stimulation rate may also increase the power of BAEP and VEP testing (Ommaya and Gennarelli, 1974, 1976; Coats and Martin, 1977; Stockard and Rossiter, 1977; Coats, 1978; Hecox et al., 1981; Pratt et al., 1981; Robinson and Rudge, 1982). Multimodality sensory EPs (MMEPs) or frequent testing generally requires a dedicated technician or team.

EP recording provides accurate information regarding functional integrity of the peripheral sensory system, brainstem, and hemispheres. Furthermore, significant changes in arterial oxygenation (P_aO_2), carbon dioxide (P_aCO_2) content, intracranial pressure (ICP), cerebral blood flow (CBF), cerebral perfusion pressure (CPP), and hypothermia may alter or obliterate EPs (Larson et al., 1966; Clague et al., 1973; Foit et al., 1980; Goitein et al., 1983; Nagao et al., 1984; Sohmer et al., 1984; Symon and Wang, 1984; Mackey-Hargadine et al., 1985). Changes often need to be extreme to markedly alter the short-latency EPs. The presumption is that early detection and correction of systemic or focal brain insults may improve the morbidity and mortality of head-injured patients. Prompt discovery of abnormal neuronal conducting systems could lead to timely diagnosis and treatment before irreversible damage takes place. Clinical–pathological correlates and the prognostic power of sensory EPs in head injury will be discussed below.

CLINICAL FINDINGS

An abnormal BAEP in comatose head-injured patients may indicate injury to the peripheral auditory apparatus (hemotympanum), eustacian tube dysfunction and middle ear pressure changes, temporal bone fracture, or brainstem abnormality (Greenberg et al, 1977*a,b*; Hall et al., 1982, 1985).

Clinical otological and BAEP findings were poorly correlated in acute head-injured patients (Aguilar et al., 1986). Otological disorders were reported in one-half of head trauma patients with a normal BAEP. By contrast, an abnormal BAEP due to brainstem insult can be seen in patients with a normal otological examination (Aguilar et al., 1986). Although no correlation was found between computed tomography (CT) findings and BAEP results, a temporal bone fracture could result in a loss of all BAEP waves beyond I or II. BAEP assessment, otological examination, and CT provided complementary information about the functional and structural status of the peripheral and central auditory system. Audiological follow-up and temporal bone CT analysis were recommended in patients with an abnormal BAEP and otological examination (Aguilar et al., 1986).

Although there is no linear correlation between the Glasgow Coma Scale (GCS) (Teasdale and Jennett, 1974) and EP findings, a higher incidence of EP abnormalities

occurred in patients with a low GCS score (Hall et al., 1984; Mackey-Hargadine and Hall, 1986). Patients with a GCS score of 8 or less are considered to have a severe head injury; and a GCS score of 9–13 indicates a more moderate injury. In a study of 111 traumatically head-injured patients, 52% of 95 patients with a GCS score of 3 or 4 had an abnormal BAEP recording, in contrast to only 19% of the 16 patients with a GCS score of 7 or greater (Mackey-Hargadine and Hall, 1986). EPs reflect sensory phenomena, whereas GCS scores include important prognostic motor functions. Nevertheless, the GCS score was found to correlate with SEP and VEP, but not with BAEP and AEP (Lindsay et al., 1981). Another study showed good agreement between a normal BAEP and a GCS score above 7, and there was also good agreement between an abnormal BAEP and a GCS score below 5. However, there was poorer correlation between BAEP changes and a GCS score of 5–7 (Brewer and Resnick, 1984).

Normal BAEP has been reported in patients with severe neurological dysfunction, including brainstem findings (Hall et al., 1984). Normalization of an abnormal BAEP was a good indicator for clinical improvement, and deterioration of the BAEP was seen in patients with clinical neurological deterioration (Nagao et al., 1982). Persistent EP abnormalities were seen in patients likely to remain severely disabled (Mjoen et al., 1983).

Brainstem dysfunction can be assumed when short-latency AEPs and SEPs are present but are severely abnormal. On the other hand, hemispheric dysfunction can be expected in patients with severely abnormal or absent VEPs, (long-latency) SEPs and/or AEPs (Seelig et al., 1981). The duration of coma, decerebration, and decortication were correlated with cerebral hemispheric dysfunction detected by cortical and subcortical (far-field) EPs (Greenberg et al., 1977*b*, 1981). Good agreement was present between intact brainstem reflexes and a normal BAEP, but not between an abnormal BAEP and brainstem reflexes (Brewer and Resnick, 1984).

Decerebration was associated with BAEP abnormalities including increased latency of all components and instability of the peaks. Recovery of these abnormalities was recorded later than recovery from decerebration (Klug, 1982). However, it is clear that decerebrate patients may show a normal BAEP, suggesting that posturing may relate to hemispheric dysfunction and not to brainstem dysfunction (Greenberg et al., 1977*b*; Uziel and Benezech, 1978; Karnaze et al., 1982). Another study showed no relationship among BAEP abnormalities, corneal reflex, and posturing abnormalities (Facco et al., 1985).

Absence of BAEP components, except wave I, was a common finding in flaccid patients, whereas a normal BAEP recording was more often found in patients with flexion reactivity (Uziel and Benezech, 1978). The majority of patients with bilateral dilated and fixed pupils showed abnormalities in or absence of waves V, IV, and III. Preservation of wave V correlated well with intact vertical eye movements, whereas preservation of wave III was correlated with intact horizontal eye movements. Unilateral pupillary abnormalities were associated with abnormalities in waves IV and V or with a normal BAEP (Uziel and Benezech, 1978). Significant association was found between the BAEP and oculocephalic reflex; an absence of the oculocephalic reflex for 8 h was associated with absence of the peaks beyond wave I (Karnaze et al., 1985).

BAEP changes were found to correlate with the clinical findings of transtentorial

(uncal) herniation and brainstem compression (Mackay et al., 1980; Tsubokawa et al., 1980; Nagao et al., 1982, 1984; Facco et al., 1985). Furthermore, in that setting, BAEP changes were recorded earlier than clinical changes (Ahmed, 1980; Mackay et al., 1980; Tsubokawa et al., 1980; Brewer and Resnick, 1984; Nagao et al., 1987). Changes in wave V amplitude were well correlated with pupillary signs during uncal herniation, which may reflect associated ischemia of the upper brainstem (Nagao et al., 1984, 1987).

BAEP changes can be seen in patients with brainstem ischemia before the onset of clinical deterioration and may correlate with clinical signs of increased ICP (Benna et al., 1982*b*; Nagata et al., 1984). Wave III was found to be a sensitive indicator for medullary failure and respiratory arrest (Nagata et al., 1984). Normalization of wave V latency was seen following the use of intravenous hypertonic solution in patients with high ICP due to supratentorial mass lesions. However, pupillary normalization was only seen in a limited number of patients (Nagao et al., 1987).

Intact pupillary responses and oculovestibular reflexes were associated with preservation of long- and middle-latency AEPs (Rosenberg et al., 1984). The presence of any cranial nerve reflexes was also associated with intact VEPs and SEPs, whereas absence of cranial nerve reflexes was associated with lost SEPs and VEPs (Trojaborg and Jorgensen, 1973).

SEP changes can also be seen with cerebral ischemia and hypoxia (Symon and Wang, 1984; Grundy, 1985; Mackey-Hargadine et al., 1986). Good agreement was observed between cortical activation produced either by peripheral nerve stimulation or by voluntary movement and regional CBF (Foit et al., 1980). Recovery of the evoked response was seen after correction of the causative vascular insult or systemic hypoxia (Larson et al., 1966; Branston et al., 1976; Grundy, 1985). SEP abnormalities were reported in trauma patients with hemiparesis or hemiplegia, and the chance of recovery was predicted from the severity of SEP abnormalities (Greenberg et al., 1977*b*; Anderson et al., 1984). Persistent asymmetry of the SEP was also reported in hemiplegic brain-injured patients (Greenberg et al., 1977*b*; Hume and Cant, 1981; Anderson et al., 1984).

Sensory EPs may successfully be used to assess patients under conditions in which the clinical neurological examination and other investigations may be misleading. For example, BAEP is a valuable test to assess patients in barbiturate coma at a time when EEG and neurological examinations are severely altered. Related conditions include chemically paralyzed patients and patients sedated to control respirations and intracranial pressure (Newlon et al., 1982; Newlon and Greenberg, 1984; Mackey-Hargadine and Hall, 1985).

EPs are less helpful regarding etiology of the lesion or pathological process. However, sensory EPs can certainly provide some diagnostic clues and substantiate suspicion of an intrinsic brainstem lesion or impending herniation. BAEP was reported to be of diagnostic importance in differentiating comatose patients with brainstem lesions from patients with metabolic or psychogenic disorders (Stockard and Rossiter, 1977; Hashimoto et al., 1979; Sohmer, 1983; Stockard et al., 1986). BAEP is expected to be abnormal if the underlying etiology of coma is a sizable brainstem lesion, but it is more likely to be normal if the underlying etiology is metabolic or psychogenic (Stockard and

Rossiter, 1977; Mackey-Hargadine and Hall, 1985; Stockard et al., 1986; Starr, 1987).

EP recording may also be useful in monitoring patients with altered mental status due to trauma-related changes, severe systemic metabolic problems, intoxications, and hypothermia. These conditions will also worsen EP abnormalities secondary to traumatic structural damage. Serial recordings have been recommended by many investigators to assess the patient's condition throughout the critical medical period and to provide an accurate evaluation of brain function at the time of recording. Delayed systemic or intracranial insults are common in severe head-injured patients, and serial recordings may be helpful (Greenberg et al., 1977*a, b*; Tsubokawa et al., 1980; Lindsay et al., 1981; Narayan et al., 1981; Newlon and Greenberg, 1984; Mackey-Hargadine and Hall, 1985; Ottaviani et al., 1986). At some point in the future, sensory EPs may become an even more effective guide in the treatment of post-traumatic coma.

Cortical and subcortical EPs can correlate with post-traumatic findings such as cognitive impairment, postconcussion syndrome, and behavioral disorders (Larson et al., 1973; Ommaya and Gennarelli, 1974; Rappaport et al., 1977; Benna et al., 1982*a*; Greenberg et al., 1982; Newlon et al., 1982; Gupta et al., 1986; Olbrich et al., 1986; Papanicolaou et al., 1986). Fifty-five patients with postconcussion syndrome underwent BAEP testing (Benna et al., 1982*a*), and 15 showed abnormal responses and particularly prolonged IPLs, whereas 9 had borderline abnormal responses. No correlation was found between BAEP changes and dizziness or caloric vestibular dysfunction. However, there was a good argument between BAEP improvement and clinical recovery (Benna et al., 1982*a*).

Latency of P300 wave of the AEP was considered to be a sensitive indicator of traumatic brain dysfunction in 18 patients (Olbrich et al., 1986). A strong correlation was found between P300 latency prolongation and neuropsychological measures, especially orientation and memory. A return to normal P300 values was associated with recoverly of cognitive impairment. In another study, P300 latency was thought to correlate with post-traumatic amnesia, and it normalized with the resolution of the amnesia (Papanicolaou et al., 1984).

VEP may also be a sensitive indicator for post-traumatic cognitive and interactive behavioral dysfunction (Bergamasco et al., 1966; Ommaya and Gennarelli, 1976; Bergström and Nystrom, 1970; Rappaport et al., 1977; Newlon et al., 1982; Gupta et al., 1986). The severity of cognitive impairment was correlated with alterations in pattern-shift (reversal) VEP (PSVEP) (Gupta et al., 1986). Of 33 head-injured patients tested with PSVEP, 50% of patients with severe cognitive dysfunction had abnormal PSVEP, 39% with moderate cognitive impairment had abnormal PSVEP, and only 11% with mild cognitive impairment had abnormal PSVEP. The mean of both P100 PSVEP latency difference and interocular P100 PSVEP latency difference was significantly abnormal in the head trauma group compared to the control group (Gupta et al., 1986). In a study of patients with symptomatic postconcussion syndrome, a mildly abnormal flash VEP was seen clearly at higher stimulation frequencies (Ommaya and Gennarelli, 1976). However, asymptomatic concussed patients had nearly normal VEP, which was mainly of small amplitude and was asymmetric. The ability to follow VEP at higher frequency was correlated with clinical recovery (Ommaya and Gennarelli, 1976).

LESION LOCALIZATION

BAEPs

BAEP findings have been reported in human pathological lesions and intraoperative brainstem recording in humans (Starr and Achor, 1975; Starr and Hamilton, 1976; Thornton and Hawkes, 1976; Hashimoto et al., 1979; Nodar et al., 1980; Scherg et al., 1984; Hammond et al., 1985; York, 1986). BAEP can provide reliable information concerning the site of the lesion as well as the degree of involvement of auditory pathways (Table 1). The BAEP peaks of waves I, III, and V are stable and often useful indicators for demonstrating auditory pathway integrity (Chiappa et al., 1979). Prolongation of both peak V latency and I–V IPL, at times reversible, was often found in patients with various brainstem pathological conditions (Starr, 1976; Thornton and Hawkes, 1976; Coats and Martin, 1977; Stone et al., 1983). On the other hand, all five peaks were intact in patients with cerebral or cerebellar lesions (Sohmer et al., 1974; Starr and Achor, 1975).

In over 100 patients studied, abnormalities of each BAEP component were correlated with radiological and postmortem localization of brainstem lesions (Stockard and Rossiter, 1977). Alteration or loss of wave I was correlated with auditory nerve lesion, wave II with pontomedullary lesions, wave III with caudal pontine lesions, wave IV with rostral pontine or midbrain lesions, wave V with midbrain lesions, and wave VI with thalamic lesions (Starr and Achor, 1975; Starr, 1976; Starr and Hamilton, 1976; Thornton and Hawkes, 1976; Stockard and Rossiter, 1977; Uziel and Benezech, 1978; Nodar et al., 1980). Abnormal BAEP was found in all patients with pontine lesions (Hashimoto et al., 1979). Significant delay of mean latencies of waves III and V was observed, and, in particular, a prolonged latency of wave V was seen in midbrain lesions (Hashimoto et al., 1979).

Preservation of waves I and III with absence of wave V bilaterally was reported in two patients with rostral pontine tegmental hematomas (Brown et al., 1981). Lesions in the ventral pons that spare the dorsal tegmental auditory tracts (as in patients with "locked-in" syndrome) were associated with intact BAEP (Brown et al., 1981; Oh et al., 1981). The latter investigators reported a normal BAEP recording in patients with lateral medullary syndrome. Traumatic midbrain injury was associated with altered BAEP wave V, even before the lesion was visualized on CT (Ropper and Miller, 1985).

BAEP testing was done in two patients with unilateral gunshot wounds to the pons. In one case, BAEP was reported as normal, which indicated sparing of the adjacent superior olivary complex and lateral lemniscus (Clark et al., 1985). In the second case, ipsilateral BAEP showed loss of waves II and III, suggesting damage of the superior olivary complex (Boller and Jacobson, 1980). These investigators concluded that the clinical findings, neuroradiographical features, operative course, and evoked response data reflected the functional anatomy of the pons.

Asymmetries of bilaterally recorded BAEPs may refer to a unilateral brainstem lesion or to a lesion affecting the crossed auditory projection. Nevertheless, the abnormality of BAEP is usually ipsilateral to the brainstem lesion (Hashimoto et al., 1979; Oh et al., 1981). However, an abnormal BAEP recorded contralateral to the side of lesion was noted in some reports (Brown et al., 1981; Nagao et al., 1987). In summary, unilateral upper pontine or midbrain defects usually show abnormality (III–V IPL or

IV/V complex) on contralateral ear stimulation, whereas lateralized lesions from the eighth nerve to the caudal pons usually show abnormality ipsilateral to the ear stimulated (Starr and Hamilton, 1976; Chiappa, 1983; Starr, 1987).

BAEP was reported to provide more reliable early information regarding brainstem function than were neurological signs or CT findings (Starr and Achor, 1975; Starr and Hamilton, 1976; Hashimoto et al., 1979; Tsubokawa et al., 1980; Ropper and Miller, 1985; Stockard et al., 1986). Primary brainstem injury and hidden lesions can be diagnosed by BAEPs (Tsubokawa et al., 1980; Bricolo et al., 1983). Instrinsic traumatic brainstem lesions may be associated with increased latency of waves III–V and suppression of waves II–V, whereas extrinsic brainstem compression produced an increase in all latencies and interpeak latencies (Hashimoto et al., 1979). In one study, there was a poor correlation between BAEP findings and brainstem lesions as revealed by CT (Kjaer, 1980). Yet, many clinicians believe that serial BAEP recording is a useful measure to follow patients with brainstem lesions.

SEPs

SEP recording can be helpful in the clinical diagnosis of peripheral nerve or brachial plexus injury, spinal cord lesions, brainstem lesions, thalamic lesions, and cortical lesions (Jones, 1979; Noel and Desmedt, 1980; Anziska and Cracco, 1981; Glover et al., 1981). Avulsion injury of dorsal spinal roots was associated with intact P9 and absence of subsequent SEP peaks (Anziska and Cracco, 1981). Hemisection at the level of bulbospinal junction produced loss of SEP components beyond P/N13 (Mauguière et al., 1983*a*). P14 and N18 waves may still be seen in patients with thalamic, radiation, or cortical lesions (Anziska and Cracco, 1981).

Alteration of N13 (N14) or N18 has been seen in patients with brainstem lesions (Anziska and Cracco, 1981; Hashimoto, 1984), and N18 of SEP has been suggested for brainstem monitoring (Anziska and Cracco, 1981; Desmedt, 1986). The absence of N18 (N19) and a prolonged P/N13–N18 were noted in patients with pontine or midbrain lesions (Anziska and Cracco, 1980). In another study, the absence of SEP waves beyond P/N13 was seen in patients with pontine, midbrain, or total brain death (Chiappa, 1983).

Asymmetries of the SEP have been seen in patients with unilateral lesions affecting the sensory pathway (Giblin, 1964; Larson et al., 1966, 1973; Chiappa, 1983). Focal destructive hemispheric lesions may affect SEPs recorded over the affected lobe or side (Giblin, 1964; Liberson, 1966; Laget et al., 1967; Williamson, 1970). SEPs were also found to be helpful in patients with thalamic lesions (hemorrhage, infarction), particularly when CT failed to demonstrate the lesion (Mauguière et al., 1983*b*; Kudo and Yamadori, 1985). Ipsilateral delay in SEP latency and waveform changes (monophasic) were also reported in patients with compressive intracranial hematomas (Larson et al., 1973; Ommaya and Gennarelli, 1976). SEP abnormalities were correlated with the severity of these lesions (Giblin, 1964; Williamson et al., 1970). Several studies were done to correlate the type of intracranial insult and SEP findings. Compressive lesions, i.e., subdural hematomas, were associated with loss of waveform complexity and delayed latency. Ischemia was associated with amplitude suppression, whereas

TABLE 3. *Reported changes in auditory evoked potentials (AEP and BAEP) associated with elevated intracranial pressure and early herniation*

Depressed cortical AEP
Latency delay wave IV and V and amplitude depression
Latency delay wave III
Prolonged interpeak latencies I–V, III–V
Latency delay wave I

hematomas were associated with changes in waveform morphology (Larson et al., 1966, 1973; Baker et al., 1968).

Abnormal sensory EPs recorded in the postinjury period may relate to a reversible functional disturbance and not necessarily to an overt structural abnormality. Persistent EP abnormalities may point to irreversible peripheral or central nervous system damage (Lutschg et al., 1983; Rumpl et al., 1983). Brain or spinal cord edema may also contribute to EP abnormalities after injury. It should be remembered, however, that lesions sparing auditory or somatosensory pathways may be associated with normal BAEPs or SEPs, respectively (Anziska and Cracco, 1980; Brown et al., 1981; Oh et al., 1981).

VEPs and MMEPs

Visual pathway abnormalities may be detected by VEPs. Anterior visual pathway dysfunction and possibly posterior visual impairment can be diagnosed early using PSVEPs (Halliday et al., 1976). However, in our experience, orbital swelling from frontal or temporal impacts or swelling from surgery may not allow adequate visual stimulation in many severe head injuries.

MMEPs were reported to be useful in patients with acute traumatic intracranial hematomas (Greenberg et al., 1977*b*; Seelig et al., 1981). A positive correlation was found between the location of neuroanatomical lesions at autopsy or operation and the location of traumatic brain dysfunction detected by MMEPs. Occipital lesions were found to correlate well with VEP findings, parietal lobe lesions with SEPs, temporal lobe lesions with VEP and AEP, diencephalic lesions with SEP, and brainstem lesions with SSEP and BAEP. On the other hand, frontal lobe lesions failed to show correlation with MMEPs (Greenberg et al., 1977*b*).

INTRACRANIAL PRESSURE AND HERNIATION

Several clinical studies have shown that measurements of BAEP waves I, III, IV, and V and the III–V and I–V IPLs may be sensitive indicators of increased ICP and transtentorial herniation (Table 3). Generally, very high ICP or actual brainstem shift has been required to produce these abnormalities. In addition, studies have suggested that VEPs may be abnormal in states of hydrocephalus and increased ICP.

BAEPs

Auditory sensitivity, in the face of raised intracranial tension, is known to deteriorate by about 30 dB and to return to normal following surgical decompression (Saxena et al., 1969; Barlas et al., 1983). This deterioration in hearing may reflect changes in perilymphatic pressure in response to raised ICP (Carlborg and Farmer, 1983). The most common EP changes detected with increased ICP and brain herniation have involved alteration in the AEPs.

In a recent BAEP study of 15 patients with supratentorial mass lesions, wave V absolute latency prolongation was noted 2–15 h before pupillary changes (Nagao et al., 1987). BAEP changes were marked when ICP approached 30 mm Hg. Following the lowering of ICP with intravenous glycerol, shortening or normalization of a prolonged wave V absolute latency was demonstrated in almost all patients with anisocoria, with or without clinical improvement of uncal herniation. Inconsistent III–V IPL prolongation was also noted (Nagao et al., 1987).

Wave V absolute latency prolongation and suppression of wave V amplitude as a result of high ICP and early signs of uncal herniation were also reported in another series of 12 patients with supratentorial mass lesions (Nagao et al., 1984). Efforts to lower ICP by glycerol administration were successful in normalizing wave V latency with or without clinical improvement (Nagao et al., 1984). Suppression of wave V and prolongation of I–V IPL were noted earlier than clinical deterioration in three patients with brainstem compression secondary to supratentorial lesions (Ahmed, 1980). Administration of steroids caused normalization of wave V latency in two of the three tumor patients (Ahmed, 1980). Wave V changes were found to correlate with rostrocaudal deterioration of brainstem function, and wave V recovery was delayed compared to the earlier BAEP components (Nagao et al., 1984; Nagata et al., 1984; Ghaly et al., 1988).

Another study was done in 15 patients with different causes of increased ICP (Nagao et al., 1983). Changes in latency and amplitude of wave V were seen earlier and at slightly raised ICP in patients with clinical signs of central and uncal herniation. In contrast, patients with diffuse intracranial hypertension without brainstem displacement did not show wave V changes even at a very high level of ICP (60–70 mm Hg) (Nagao et al., 1983).

BAEP abnormalities have been correlated with clinical brainstem functioning in both uncal and central herniation syndromes. Normalization occurred after evacuation of intracranial lesions. At the beginning of central herniation in a patient with acute obstructive hydrocephalus, there were no remarkable BAEP changes (Nagao et al., 1982). As the process of herniation progressed, delay in latency and suppression of wave V was seen, and waves VI and VII disappeared. An immediate shunt operation produced progressive recovery of the BAEP. Thus, BAEP correlated with neurological deterioration and the severity of downward herniation in this patient (Nagao et al., 1982). Prolongation of wave V absolute latency was seen earlier than amplitude suppression and accompanied rostrocaudal axial deterioration. An important conclusion of these studies was that physical shift of the brainstem itself and not the height of ICP was believed responsible for determining BAEP changes (Nagao et al., 1983).

The upper brainstem was reported to be more vulnerable than the lower brainstem

during clinical rostrocaudal deterioration (Jennett and Stern, 1966). Accordingly, BAEP abnormalities can be helpful in the detection of transtentorial herniation at different stages of progression. In a study of 20 patients with traumatic brainstem lesions, patients with I–V IPL (<4.48 ms) showed basal cistern obliteration with or without temporal horn dilatation of CT, whereas patients with a prolonged I–V IPL (>4.48 ms) or absence of all BAEP components beyond wave I showed more severe brainstem deformation on CT (Zuccarello et al., 1983). The presence of brainstem rotation or foreshortening on CT was found to correlate with prolongation of the I–V IPL (>4.48 ms) (Zuccarello et al., 1983; Facco et al., 1985). CT-verified compression of the basal cisterns about the brainstem may relate to a delay in III–V IPL. However, 45% of 59 patients with a normal BAEP had basal cistern compression (Mackey-Hargadine and Hall, 1986).

BAEP changes, including prolongation of III–V and I–V IPL, suppression of wave V, and absence of waves VI and VII, were reported in 8 of 20 patients with supratentorial tumors and clinical signs of increased ICP (Benna et al., 1982*b*). In a patient with a large supratentorial meningioma, BAEP wave latency was reported to be an early sign associated with high ICP (Skondras et al., 1986).

Forty-one patients with impending transtentorial herniation underwent binaural BAEPs recorded between Cz and the mastoid ipsilateral to a lateralized lesion (Nagata et al., 1984). Nonsurvivors showed prolongation of wave V absolute latency and I–V IPL. Prolongation of wave I latency was a consistent finding in those who died compared to those who survived. In the early third-nerve stage of transtentorial herniation, I–III and I–V IPLs were within normal limits. In the midbrain–upper pons stage, I–V IPL was increased and no patients survived. Finally, in the medullary stage, waves III and V were absent, and no patients survived. There was good general correlation between ICP and BAEP latencies over the course of transtentorial herniation. BAEP peaks disappeared as the ICP reached its highest level, and the reduction of ICP was associated with shortening of IPLs. Consequently, absolute BAEP latencies and IPLs were thought to be sensitive indicators in early detection of brainstem dysfunction (Nagata et al., 1984).

An elevated BAEP threshold was also noted in 70% of 40 hydrocephalic patients; the condition improved in some of the patients when the increased ICP was corrected (Kraus et al., 1984). The most common abnormal response in these latter patients was distortion of wave V. Wave V was broadened, and it lacked its characteristic following reverse slope (Kraus et al., 1984). A depression of wave V amplitude and prolongation of I–V IPL was observed in a hydrocephalic neonate who improved after shunting (Despland and Galambos, 1980).

In a BAEP study of 16 hydrocephalic babies, waves I and V showed prolonged latency and depressed amplitude (Edwards et al., 1985). The V/I ratio was significantly reduced. Elevated BAEP threshold was also noted. However, I–V IPL did not show marked changes. Of all BAEP abnormalities, reduction of wave V amplitude presented the most common abnormality. Improvement of BAEP was noted in some patients on followup testing (Edwards et al., 1985).

Some investigators concluded that there was no relationship between BAEP changes and ICP in humans (Keith et al., 1983; Karnaze et al., 1985). However, others have found that CPP was the crucial factor, rather than ICP (Goitein et al., 1983; Hall and

Mackey-Hargadine, 1984). Very high ICP values (64 mm Hg) did not affect BAEP recorded from children as long as the CPP was maintained above 30 mm Hg (Goitein et al., 1983). Recovery of BAEP abnormalities occurred as soon as the CPP was corrected (Goitein et al., 1983). Low CPP is believed to produce brainstem ischemia, which results in BAEP abnormalities. Moreover, in experimental studies, BAEP was found to be more resistant than EEG and SSEP to low CPP. Mildly low CPP values suppressed EEG and the late components of SEPs, but significant changes were not yet observed in BAEPs. Only at very low CPP did BAEPs show significant changes (Sohmer, 1983; Sohmer et al., 1984).

It has been our experience that elevations of ICP in the clinical range frequently encountered, in the absence of brainstem shift, do not significantly alter routine BAEPs in patients with intracranial lesions. Yet laboratory and clinical evidence exists that cochlear function is affected by elevated ICP.

Only a few studies in humans are available correlating high ICP and SEP, or AEP. AEPs (middle- and long-latency AEPs) were abnormal in 6 of 15 hydrocephalic patients (deVlieger et al., 1981). P300 of AEP was significantly altered in a patient with marked hydrocephalus due to congenital aqueductal stenosis despite a nearly normal VEP, BAEP, and middle-latency auditory response (MLR) (Woods et al., 1987). SEP recording was normal in hydrocephalic patients despite abnormal VEP (McInnes, 1980).

VEPs

A number of studies in patients with hydrocephalus and increased ICP had shown definite VEP alterations that tended to normalize after cerebrospinal fluid diversion (Engel, 1975; Fichsel, 1976; Rossini et al., 1978; Ehle and Sklar, 1979; Sklar et al., 1979; McInnes, 1980; deVlieger et al., 1981; Onofrj et al., 1981; York et al., 1981; Guthkelch et al., 1982; Humphrey et al., 1982; McSherry and Walters, 1982; Guthkelch et al., 1984; York et al., 1984; Alani, 1985).

In patients with hydrocephalus or brain edema, a linear relationship was found between ICP levels (>300 mm H_2O; 39 mm Hg) and a latency shift of the N2 wave of flash VEPs (York et al., 1981, 1984). VEP changes included a delayed latency of P100, abnormal P100 waveform, asymmetries, and fatigability to increasing stimulus frequency (Sklar et al., 1979). A reduction of P100 latency was seen after shunting for hydrocephalus, and worsening of the VEP was associated with progression of hydrocephalus (Sklar et al., 1979).

PSVEP was found to be an excellent indicator of visual pathway dysfunction in patients with hydrocephalus (Alani, 1985). VEP abnormalities were consistently seen in infants with ventriculomegaly and a slow return of P100 to normal values after shunting (Guthkelch et al., 1982). Flash VEP was abnormal in 8 of 15 hydrocephalic patients, whereas AEP was abnormal in 6 patients. Four patients had abnormal VEPs and abnormal AEPs (deVlieger et al., 1981).

In 10 patients with hydrocephalus, six had abnormal VEP configuration and two had delayed latency (McInnes, 1980). Following ventriculoperitoneal shunting, normalization of VEP was only seen in those with abnormal VEP configuration (McInnes, 1980). Delayed latency of flash-evoked P100 was seen frequently in infants developing hydrocephalus before 56 weeks of age (Guthkelch et al., 1984). Significant pro-

TABLE 4. *Evoked potential findings associated with unfavorable outcome*

Consistent gross abnormalities
Multiple abnormalities
Abnormality(ies) detected in more than one modality
Absence of peak(s)
Prolonged interpeak latencies (central conduction time)
Reversed amplitude ratio

longation of P100 was also observed in macrocephalic infants with brain damage. Recovery of P100 after shunting was mostly seen in patients with normal mental status regardless of ventricular size. These investigators suggested that serial VEP recording might be helpful in assessing mental development of hydrocephalic infants (Guthkelch et al., 1984).

Additional studies showed VEP changes in patients with papilledema and benign intracranial hypertension (Hume and Cant, 1976; Kirkham and Coupland, 1981). Others believe that a poor correlation exists between papilledema, benign intracranial hypertension, and VEP (Rouher et al., 1969; Asselman et al., 1975; Babel et al., 1977; Halliday and Mushin, 1980; Halliday, 1982; Chiappa, 1983). Further confirmation of relative VEP changes due to hydrocephalus and increased ICP is needed, but this may well prove a fruitful area of investigation.

EPs AS A PROGNOSTIC SIGN

Serial clinical examination at times provides the most accurate prediction of outcome in severely head-injured patients. However, sensory EPs may enhance the accuracy of outcome prediction, particularly when clinical findings are unclear. BAEP in particular was found to be a reliable measure in patients who were in barbiturate coma or were therapeutically paralyzed, or when the clinical examination was not obtained (Newlon et al., 1982; Hall and Mackey-Hargadine, 1984; Mackey-Hargadine and Hall, 1985, 1986). EP results were believed by many authors to be more reliable than clinical findings, and no false pessimistic predictions were encountered (Narayan et al., 1981; Anderson et al., 1984; Karnaze et al., 1985).

Sensory EPs may even give an accurate prediction of the patient's outcome in the early postinjury period (Seales et al., 1979; Kaga et al., 1985; Ottaviani et al., 1986). Mechanical brain trauma and edema may cause EP changes recorded in the early postinjury period (Lutschg et al., 1983). These EP abnormalities may recover at a later time, emphasizing the importance of serial and delayed EP recording in predicting outcome.

Repeatedly, grossly abnormal sensory EPs, or abnormalities in more than one EP modality, usually indicate an unfavorable outcome, and consistently normal potentials often predict favorable outcome (Table 4). Mildly abnormal EPs may not be as helpful in prognostication. Serial monitoring is required for accurate prediction of outcome, and further EP changes may relate to deterioration or improvement in the patient's condition. Reversible EP abnormalities as a result of aggressive management may or may not indicate improvement in the ultimate outcome (Hall et al., 1984).

BAEPs

Although the type of sensory EPs used to evaluate head injury prognosis has varied, many investigators believe that BAEP is a useful prognostic indicator in head-injured patients (Seales et al., 1979; Tsubokawa et al., 1980; Hall et al., 1982; Ducati et al., 1983; Hall et al., 1983; Mjoen et al., 1983; Scarpino et al., 1983; Anderson et al., 1984; Facco et al., 1985; Papanicolaou et al., 1986). These investigators and others recommend the use of a combination of BAEPs and AEPs (middle- and long-latency auditory potentials) as predictors of outcome (Karnaze et al., 1982; Hall et al., 1983; Hall and Mackey-Hargadine, 1984; Rosenberg et al., 1984; Kaga et al., 1985; Karnaze et al., 1985; Ottaviani et al., 1986).

BAEP is a sensitive indicator of the functional integrity of the brainstem (Tsubokawa et al., 1980; Sohmer, 1983; Brewer and Resnick, 1984). Absence of all BAEP components or absence of all components beyond wave I or II usually indicates poor outcome and is often irreversible (Tsubokawa et al., 1980; Klug, 1982; Klug et al., 1983; Ducati et al., 1983; Hall et al., 1983; Lutschg et al., 1983; Mjoen et al., 1983; Scarpino et al., 1983; Sohmer, 1983; Yagi and Baba, 1983; Brewer and Resnick, 1984; Hall and Mackey-Hargadine, 1984; Rosenberg et al., 1984; Ottaviani et al., 1986).

Gross BAEP abnormalities were often seen in traumatized patients with an unfavorable outcome (Hall et al., 1982; Karnaze et al., 1982; Hall et al., 1983; Karnaze et al., 1985; Papanicolaou et al., 1986). Absence of wave IV, V, or all BAEP peaks carried a worse prognosis than simply a delay in latencies (Marcus and Stone, 1984). Marked exaggeration of wave I amplitude compared to other waves was seen in cases with severe brainstem dysfunction (Starr and Hamilton, 1976; Mjoen et al., 1983; Hall and Mackey-Hargadine, 1984; Hall et al., 1985). In another study, all patients with absent IV–V or earlier waves died (Ducati et al., 1983).

A normal to mildly abnormal BAEP was associated with favorable outcome (Karnaze et al., 1982; Hall et al., 1983; Mjoen et al., 1983; Sohmer, 1983; Karnaze et al., 1985; Ottaviani et al., 1986). Absence or delayed latency of wave V may also be reversible and has been observed with a favorable outcome (Tsubokawa et al., 1980; Lutschg et al., 1983; Gennarelli, 1987). Absence of wave V with subsequent stepwise loss of wave peaks IV and III at less than 12 h following injury was associated with an unfavorable outcome (Tsubokawa et al., 1980).

Timing of BAEP testing as well as repeated recordings were helpful to reflect the severity and progression of brainstem injury and to predict outcome (Tsubokawa et al., 1980). Patients with prolonged I–V IPLs were graded better than patients with absence of wave V (Mjoen et al., 1983; Ottaviani et al., 1986). Patients with a high IV–V/I amplitude ratio were more likely to have a favorable outcome (Scarpino et al., 1983). I–V IPL was found to be a sensitive predictor of outcome in severely head-injured patients (Facco et al., 1985). Favorable outcome was noted in patients with I–V IPL <4.48 ms compared with an unfavorable outcome in those with I–V IPL >4.48 ms (normal I–V IPL, $4.03 + 0.17$) (Facco et al., 1985). In 30 head trauma patients, there was a correlation between III–V IPL and poor outcome. Prolonged I–III IPL was associated with grave prognosis (Scarpino et al., 1983).

An abnormal BAEP recording was a better predictor of unfavorable outcome than a normal test was for predicting a favorable outcome (Anderson et al., 1984; Hall and

Mackey-Hargadine, 1984; Papanicolaou et al., 1984; Kaga et al., 1985; Cant et al., 1986; Mackey-Hargadine and Hall, 1986). It has been reported that patients with bilateral normal BAEP responses had the best chance of survival (Ducati et al., 1983). In one study it was estimated that the overall predictive accuracy of BAEP was 50% (Hall et al., 1982). By contrast, another BAEP study in post-traumatic patients showed an overall predictive accuracy of 77% or 91%, if deaths from extracranial causes were excluded (Brewer and Resnick, 1984).

BEAPs, MLRs, and AEPs

Patients with preservation of middle- and long-latency AEPs had a favorable outcome (Karnaze et al., 1982; Rosenberg et al., 1984; Kaga et al., 1985). These later auditory potentials could be significant measures of communicative/cognitive outcome in head-trauma patients. Favorable outcome was also observed in patients with intact long-, middle-, and short-latency potentials, compared to those with only short-latency (BAEP) responses (Karnaze et al., 1982; Rosenberg et al., 1984; Kaga et al., 1985). Normalization of BAEPs has been correlated with recovery from the postconcussion syndrome (Benna et al., 1982*a*).

BAEPs and MLRs (10–50 ms) may enhance accuracy of prognosis (Karnaze et al., 1982; Hall et al., 1983; Hall and Mackey-Hargadine, 1984; Rosenberg et al., 1984; Kaga et al., 1985; Mackey-Hargadine and Hall, 1986; Ottaviani et al., 1986). Consistently normal MLRs within 10 days postinjury was reported to indicate good neurological and communicative outcome, whereas consistently abnormal MLRs indicated poor outcome even with a normal BAEP (Hall and Mackey-Hargadine, 1984; Ottaviani et al., 1986). However, MLRs can be altered by medications and other factors. The outcome of patients with an increased BAEP I–V IPL was found to be related to MLR results and to subsequent improvement of auditory waveforms within the first 3 months after injury (Ottaviani et al., 1986).

Fifty-four acutely comatose patients were assessed with short- (BAEP), middle- (MLR), and long-latency AEPs within 72 h after admission (Kaga et al., 1985). Survival rate was 100% in patients with normal potentials, 91% in patients with only an absence of long-latency potentials, 60% in patients with only intact BAEPs, 10% in patients with abnormal BAEPs and absent subsequent potentials, and no survival of patients with absence of all AEPs. All patients with altered or obliterated BAEPs had absent subsequent potentials. These investigators deduced that a normal MLR was clearly a predictor of survival in comatose patients, whereas a normal BAEP was not a reliable predictor of survival. Abnormal or absent BAEP was a reliable predictor of death (Kaga et al., 1985). The addition of clinical findings to BAEP and AEP was found to predict the outcome more accurately and with no false pessimistic predictions (Karnaze et al., 1985).

The P300 (ms) long-latency AEP was strongly correlated with neuropsychological evaluation and was useful to confirm cognitive impairment and residual brain dysfunction (Larson et al., 1973; Ommaya and Gennarelli, 1974; Rappaport et al., 1977; Benna et al., 1982*a*; Greenberg and Ducker, 1982; Newlon et al., 1982; Gupta et al., 1986; Olbrich et al., 1986; Papanicolaou et al., 1986). The P300 was also found to correlate with post-traumatic amnesia (Papanicolaou et al., 1986).

SEPs

SEPs were thought to be a reliable predictor of favorable or unfavorable outcome from head injury (De La Torre et al., 1978; Hume and Cant, 1981; Prugger et al., 1983; Rumpl et al., 1983; Anderson et al., 1984; Mackey-Hargadine and Hall, 1985; Pfurtscheller et al., 1985; Cant et al., 1986; Mackey-Hargadine and Hall, 1986; Walser et al., 1986). It has recently been reported that SSEPs were more vulnerable to central nervous system trauma than BAEPs and VEPs (Anderson et al., 1984; Cant et al., 1986). In addition, normal BAEPs have been reported in patients with severely abnormal SSEPs (Cant et al., 1986).

Trauma patients with eight identifiable SEP wave peaks in the first 300 ms had a better outcome than those with only five peaks, whereas no recovery was reported in patients with only two primary SEP peaks (De La Torre et al., 1978). Absence of early components had a worse prognostic value than absence of later components only (provided no barbiturates or sedatives were given). The majority of patients with absence of all components or absence of all components beyond P15 died (Greenberg et al., 1977*a,b*; De La Torre et al., 1978; Lutschg et al., 1983; Rumpl et al., 1983; Pfurtscheller et al., 1985; Walser et al., 1986).

SSEPs have been correlated with outcome in 75–80% of head-trauma patients. Improvements of SSEPs was seen in patients with good recovery, whereas consistently abnormal SEPS were recorded in disabled patients (Hume and Cant, 1981).

The central conduction time (CCT) (CCT = N20 − N14) of SEPs has been correlated with outcome after head injury (Hume and Cant, 1981; Lutschg et al., 1983; Prugger et al., 1983; Rumpl et al., 1983; Cant et al., 1986; Walser et al., 1986). In addition, CCT was found to be more resistant to barbiturates than later SEP components (Lutschg et al., 1983; Marcus and Stone, 1984).

Normal CCT and N20/N14 amplitude ratio (ratio between the peak of N20 and its subsequent positivity and the peak of N14 and its subsequent positivity) were found in trauma patients with good outcome (Rumpl et al., 1983). Prolonged CCT and decreased amplitude ratio were correlated with poor outcome. The most prolonged CCT was seen in patients who died. Asymmetries of SSEPs were seen in patients with moderate to severe disability (Rumpl et al., 1983). Early normalization of CCT and amplitude ratio was found in patients with a favorable outcome. It was reported that patients with primary brainstem injuries who had a good recovery might demonstrate asymmetric or absent SSEPs and increased CCT (Prugger et al., 1983; Rumpl et al., 1983).

In another study, bilateral absence of the N20–P23 complex was associated with poor outcome, and the majority of these patients met the clinical criteria of brain death (Marcus et al., 1984). The length of survival was correlated partly with the presence or absence of BAEPs and brainstem reflexes. Patients with only BAEP wave I or no BAEP response survived 8–46 days. Patients with unilateral delay in SSEP N14–N20 conduction had a favorable outcome compared to patients with bilateral N14–N20 delay (Marcus et al., 1980; Marcus and Stone, 1984).

Patients with unilateral or bilateral absence of N20 within the first 4 days had an unfavorable outcome (Cant et al., 1986). Preservation of N20/P23 implied a good prognosis, whereas loss of N20/P23 implied poor prognosis (Goldie et al., 1981).

Long-latency SEPs were correlated with the patient's clinical status and outcome

(Perot, 1976). Normal long-latency SEPs indicated favorable outcome, whereas missing or questionable long-latency SEPs was an unfavorable sign (provided no barbiturates or sedatives were given). A return of consciousness and improvement of neurological status correlated with return of the late SEP components (Perot, 1976). In another report, the appearance of long-latency SEPs during emergence from coma was compatible with a more optimistic outcome (Pfurtscheller et al., 1985).

VEPs

A study of VEPs in head-trauma patients showed that flash VEP was a good indicator of unfavorable outcome, but false pessimistic predictions were present (Anderson et al., 1984). A study of monocular PSVEPs was found to correlate with cognitive function in awake post-traumatic patients. P100 latency prolongation was the most common finding (Gupta et al., 1986).

In another study of brain-damaged patients, long-latency VEPs were reported to correlate with psychosocial disability (Rappaport et al., 1977). Mildly abnormal VEPs were found in patients with post-traumatic syndrome (Ommaya and Gennarelli, 1974, 1976; Rappaport et al., 1977; Gupta et al., 1986). Recovery was frequently underway when the VEP was able to follow a faster rate of stimulation (Ommaya and Gennarelli, 1976). Long-latency VEPs restricted to the occipital region did not correlate with clinical status or outcome (Pfurtscheller et al., 1985). However, severe hemispheric dysfunction was usually present if VEPs were preserved in the occipital region only. A gradual spreading of VEP over the scalp tended to indicate improvement in cerebral function (Pfurtscheller et al., 1985).

MMEPs

In one study of severely head-injured patients, MMEPs (AEPs, SEPs, and VEPs) were well correlated with outcome, whereas short-latency brainstem potentials alone did not correlate with outcome (Lindsay et al., 1981). The above investigators found that simply counting the number of identifiable waves present was an optimum method for analyzing the data. Summation of waves from all responses provided a useful index of brain dysfunction. The fewer the number of wave peaks, the poorer the outcome (Lindsay et al., 1981). Later cortical components of each modality correlated with the level of consciousness and depth of coma (Greenberg et al., 1977*a,b*; Newlon et al., 1982).

A combination of SEPs and BAEPs was a more reliable indicator of outcome than either EP alone (Lutschg et al., 1983). SSEP was found to be a powerful prognostic indicator in the early postinjury period, whereas BAEP was significantly correlated to later outcome (Greenberg et al., 1977*a,b*). A combination of SEP–BAEP (Lutschg et al., 1983; Cant et al., 1986) or long-latency SEP–VEP (Pfurtscheller et al., 1985) has also been suggested. MMEPs with a specific grading scale for AEPs, SEPs, and VEPs were thought to give the most accurate results. MMEP recording appears to be an excellent prognostic indicator as the functional integrity of three different sensory pathways is assessed (Greenberg et al., 1977*a,b*, 1981; Lindsay et al., 1981; Seelig et al., 1981; Newlon et al., 1982).

Graded MMEPs predicted outcome in 100 severely head-injured patients with approximately 100% accuracy, excluding patients who died from systemic causes (80% without exclusion) (Greenberg et al., 1981). Mildly abnormal MMEPs were predictive of good to moderate outcome in 81% of patients, whereas severe to absent MMEPs indicated a poor outcome in 76% of patients. A linear relationship was found between severity of MMEP abnormality and a less favorable outcome. Severely abnormal MMEPs (the last grade in the scale) was maximally predictive of poor outcome regardless of any other factors (Greenberg et al., 1981).

In patients with acute subdural hematoma, MMEP was a clear indicator not only for outcome prediction, but also for early detection of reversible and irreversible brainstem dysfunction (Seelig et al., 1981). Similar results were found using BAEP in patients with severe brain contusion and subdural hematoma (Tsubokawa et al., 1979). A large decompressive procedure was found ineffective in patients with absent BAEP peaks (Tsubokawa et al., 1979).

In 133 severely head-injured patients, MMEPs were the most reliable predictor for outcome (Narayan et al., 1981). The accuracy rate was 91%, and there were no false pessimistic predictions. When clinical and MMEP data were combined together, the accuracy rate became 89% with 4% false pessimistic predictions. By contrast, clinical data alone predicted outcome with 82% accuracy and 9% false pessimistic predictions (Narayan et al., 1981).

A follow-up study over a one-year post-injury period was carried out using MMEPs (Newlon et al., 1982). Patients with consistently normal MMEPs had good recovery, whereas patients with consistently absent potentials had poor outcome. Patients with stable or improving mildly abnormal MMEPs had favorable outcome despite complications. Patients with severe MMEP abnormalities that later improved had a favorable outcome, whereas those with persistent or deteriorating severe EP abnormalities had poor outcome. The changes found in serial MMEPs were better prognostic indicators than the presence or absence of medical complications. MMEP changes (deterioration or improvement) may at times precede changes in the patient's clinical status (Newlon et al., 1982).

CONCLUSIONS

EP recording is a useful tool for providing accurate information regarding the integrity of sensory input to the central nervous system in critically head-injured patients. This is especially important in unconscious patients given barbiturates or who are therapeutically paralyzed to control increased ICP.

Correlations exist between EPs and clinical findings such as the lower GCS, ocular abnormalities, rostrocaudal brainstem deterioration, and post-traumatic cognitive disorders. Sensory EPs are also valuable in terms of lesion localization. Short-latency potentials reflect brainstem function, whereas middle- and long-latency potentials assess hemispheric function. Localization of a brainstem lesion within several centimeters using short-latency EPs may be possible, but it must be remembered that EPs test the functional rather than anatomical integrity of a specific tract.

Experience with EP monitoring in patients demonstrating clinical herniation syndromes and increased ICP has shown significant clinical promise. In particular, AEPs

and VEPs appear helpful in these clinical situations. EP monitoring, especially MMEPs, have also proved to be an excellent predictor of outcome. In a number of studies, outcome prediction from EP analysis has been more effective with fewer false pessimistic results than the clinical examination.

The morbidity and mortality of post-traumatic coma remain high, and there is a great need for physiological monitoring in this challenging group of patients. More investigation remains to be done in head-injured patients as we find the sensory EP modalities that correlate best with neurological function and ultimate outcome.

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