

Central Somatosensory Conduction Time and Acoustic Brainstem Transmission Time in Post-Traumatic Coma

E. Rumpl, M. Prugger, F. Gerstenbrand, *W. Brunhuber, F. Badry, and *J. M. Hackl

*University Clinic of Neurology and *Anaesthesiology, University of Innsbruck, A-6020 Innsbruck, Austria*

Summary: Short-latency evoked potentials (SEPs) of the scalp and neck after median nerve stimulation and acoustic brainstem evoked potentials (BAEPs) were recorded in 85 patients in post-traumatic coma with clinical signs of brainstem impairment between days 2 and 6 after trauma. The central somatosensory conduction time (CCT), the amplitude ratio (AR) N20:N13, the interpeak latencies (IPL) I-III, III-V, I-V, and the ARs between waves I and V (I:V) and between wave I and the wave IV/V complex (I:IV/V) were calculated and related to the outcome of the patients. In cases of coma due to supratentorial lesions, CCT and ARs of SEPs were close to normal in patients with good outcome: CCT increased and ARs decreased with worsening of outcome. In cases of primary brainstem injury, a significant prolongation of CCT was also seen in patients with good recovery, whereas normal CCTs could be found in patients with severe disability and death outcome. In this case, unilateral absent scalp SEPs were frequently found. The IPLs I-III, III-V, I-V, and the ARs of BAEPs increased with worsening of outcome. Significant differences of IPL I-V and III-V (brainstem transmission time) were seen between patients with good recovery or moderate disability outcome and the patients with severe disability or death outcome. There was no difference in BAEPs between patients with primary brainstem lesion and patients with secondary brainstem lesion. Patients with bilateral absent SEPs and bilateral absent BAEPs not related to traumatic or preexisting hearing disorders died or survived severely disabled. Unilateral absence of scalp SEPs and unilateral absence of BAEPs were frequently found in patients who died or who had severe disability. Asymmetries in scalp SEPs appeared to be distributed equally to all outcome categories, but asymmetries in BAEPs increased with worsening of outcome too. In most of the patients who died or survived disabled, both SEPs and BAEPs were abnormal. **Key Words:** Central somatosensory conduction time—Acoustic brainstem transmission time—SEP—BAEP—Post-traumatic coma—Outcome.

The assessment of brain dysfunction in head-brain injuries may be provided by clinical examination (Plum and Posner, 1966; Gerstenbrand and Lücking, 1970). However, the clinical examination of post-traumatic comatose patients is severely impaired by sedative drugs, including barbiturates used frequently in the treatment of brain edema (Miller, 1979). Methods of assessing brain function that are not influenced by sedative drugs were found by the application of multimodality evoked potentials (Greenberg et al., 1977*a,b*, 1981; Lindsay et al., 1981; Newlon et al., 1982; Anderson et al., 1984). Short-latency somatosensory evoked potentials (SEPs) and brainstem auditory responses (BAEPs) were regarded to be of special clinical utility in comatose states (Uziel and Benezech, 1978; Seales et al., 1979; Goldie et al., 1981; Mjøen et al., 1983; Rumpl et al., 1983*b*; Yagi and Baba, 1983; Facco et al., 1985; Cant et al., 1986). Measurement of central somatosensory conduction time (CCT) was especially regarded as useful in predicting the outcome of post-traumatic coma (Hume and Cant, 1981; Rumpl et al., 1983*b*). Findings in primary brainstem-injured patients suggested a limited prognostic information of CCT and SEPs in these cases. Prolonged CCT, asymmetric SEPs, and absent SEPs were also found in patients with good outcome (Rumpl et al., 1983*b*). BAEPs had proved to be fair predictors of the overall outcome (Goldie et al., 1981; Facco et al., 1985) and were suggested to offer a diagnostic method for primary brainstem injury (Tsubokowa et al., 1980).

This study was undertaken to define the clinical utility of SEPs and BAEPs, especially of CCT and of interpeak latencies (IPLs) I-III, III-V, and I-V in diagnosis and prognosis of post-traumatic comatose states. Special attention was paid to patients who might have suffered from primary brainstem impact.

CASE MATERIAL AND METHODS

Normal data were obtained from 22 healthy persons (students, physicians, members of the nursing staff) at the intensive care unit in electrically unshielded rooms. The average age was 27 years, varying from 18 to 52 years.

Eighty-five patients in post-traumatic coma (average age, 25 years varying from 17 to 50 years) were studied at bedside from days 2 to 6 after head-brain injury. The evoked potential responses were recorded once in most cases, but in 18 cases two or more follow-up records were obtained. For statistical analysis only the first records were used.

All patients had severe brain injury and demonstrated signs of brainstem dysfunction. Eighty patients had closed head injuries; in five patients, first recordings were done after surgical evacuation of subdural or epidural hematoma. Most patients were sedated and curarized and sustained artificial respiration. Five patients were studied in brain death. Patients suffering from severe metabolic disturbances were excluded from the study. All patients underwent computed tomography (CT) examination on admission and immediately after surgical treatment, as well as frequent follow-up examinations.

The patients were classified neurologically in Stages 1-4 of the midbrain syndrome (MBS) according to Gerstenbrand and Lücking (1970) and in Stages 1 and 2 of the bulbar brain syndrome (BBS) described previously (Rumpl et al., 1983*a*). With regard to the terminology of Plum and Posner (1966), MBS 1 and MBS 2 correspond to the early diencephalic stage, MBS 3 to the late diencephalic stage, MBS 4 to the midbrain-upper

pons stage, BBS 1 to the lower pontine–upper medullary stage, and BBS 2 to the medullary stage. The stages usually characterize the well-known rostrocaudal deterioration in patients with supratentorial lesions and secondary brainstem involvement (McNealy and Plum, 1962; Plum and Posner, 1966). Lateralized neurological signs may be present, and they hint at the original hemispheric or brainstem lesions (Plum and Posner, 1966; Gerstenbrand and Rimpl, 1983). Neurological signs different from the expected rostrocaudal deterioration may be found in cases of primary brainstem injury. Most frequently, the relatively intact optomotor reactions are in contrast to decerebrate posture and respiratory abnormalities (Maciver et al., 1958). CT is diagnostic and can rule out a supratentorial lesion causing secondary brainstem dysfunction.

The outcome of the patients was assessed at least 6 months after the injury and classified according to Jennet and Bond (1975) into four categories—death, severe disability, moderate disability, and good recovery. The category of severe disability includes patients in an apallic (vegetative) state. The category of death only included patients who died from brain death within 2 weeks after the injury. This category excludes patients who remained severely disabled or were in an apallic state and later died from metabolic complications.

Square-wave pulses of 0.2-ms duration were applied at a rate of 5/s to the median nerve at wrist (at the elbow in rare cases) with a bipolar surface nerve stimulator. The responses were recorded with tin/lead disc electrodes placed over the seventh cervical vertebra and over the central area of the scalp contralateral to the stimulated wrist (C3 and C4, International 10–20 System). The reference electrode was placed over the midforebrain (Fpz). Two series of 512 responses were averaged. The difference in peak latency between N13 and N20, the central conduction time (CCT), and the amplitude ratios (ARs) between the peak of N20 and subsequent positivity and the peak of N13 and subsequent positivity were measured and calculated for each side. A clear difference in SEP components (CCT, side difference exceeding 0.9 ms, AR, waveform, number of positive/negative waves) was classified as asymmetry. Furthermore, the absence of SEP responses from one or both hemispheres was noted. Details of the method were given in a previous paper (Rimpl et al., 1983b).

Click signals (0.1-ms impulses of alternating polarity) were presented monaurally through an electromagnetically shielded TDH earphone at an intensity of 90 dB HL (in cases with absent wave I, 100 dB HL) at a rate of 10/s. The BAEPs were recorded bilaterally from tin/lead disc electrodes (vertex–mastoid) by means of Medelec MSC 6 equipment. Recording from the ipsilateral and contralateral ear was used as an aid in the identification of wave V (Stockard et al., 1978), because waves IV and V tend to be separated in contralateral derivation. The responses were amplified 10^5 , passed through a 150–3,200-Hz filter, and were averaged over a 12-ms time base. Two series of 1,024 clicks (2,048 clicks in selected cases) were averaged and recorded to ensure the reliability of identifying the evoked potentials components. Latencies were measured with the cursor, and measurements of amplitudes were made on photographs. To study the changing latencies and amplitudes of waves, clicks at decreasing intensity (60 dB, 40 dB, and 30 dB HL) were applied in a series of normal patients, as well as comatose patients. Calculations performed included the IPLs I–III, III–V, and I–V. The ARs I:V and I:IV/V were calculated. The amplitude of a wave was measured from its respective peak to the following negative trough. For the wave IV/V complex, the peak was taken

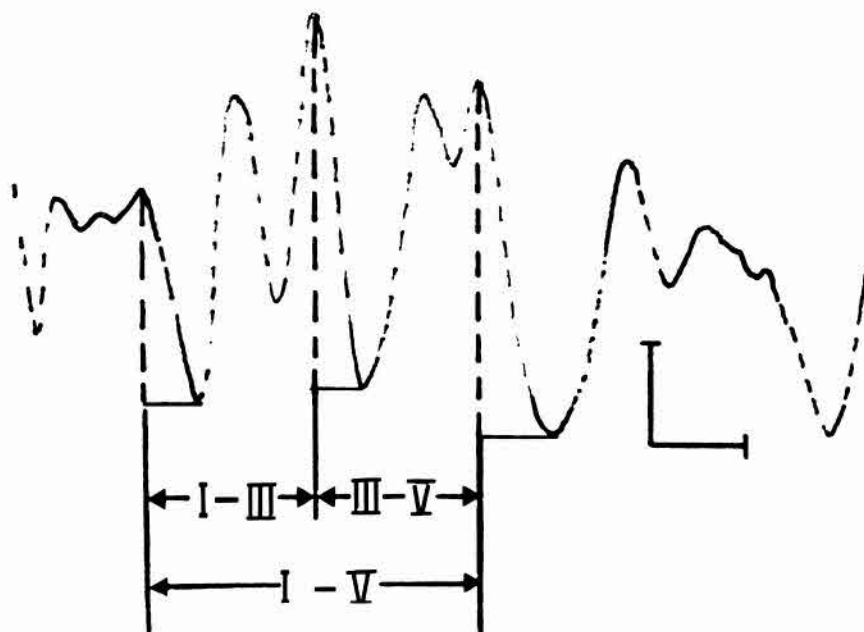


FIG. 1. BAEPs obtained from one ear in a normal subject showing the interpeak latencies (IPLs) I-III, III-V, and I-V, which are calculated I:V and I:IV/V (highest peak of IV/V complex). In this case, both ARs are the same, because wave V is the highest peak of the IV/V complex. The IPL III-V is termed acoustic brainstem transmission time (BTT). Calibration: 0.5 μ V; 1.2 ms/div.

from the highest point of the complex (wave IV or V; see Fig. 1). Recently, it was suggested that wave II might be generated in the extramedullary portion of the eighth nerve (Stockard et al., 1980; Goldie et al., 1981; Garg et al., 1982). There is general agreement that wave III is generated in the lower pons and that wave V is generated in the high pons or the low midbrain (Chiappa, 1983). Considering these sites of BAEP generators, acoustic brainstem transmission time (BTT) might be the appropriate term for IPL III-V. In cases in which wave I was absent but later waves (wave III and/or wave V) were present, the IPL III-V or the absolute latencies of wave V were used for interpretation. A clear difference in BAEP components (difference in IPLs latencies III-V and/or I-V of more than 0.2 ms, a difference of ARs of more than 50% after right and left stimulation, and the unilateral absence of one or more waves, with one wave always present) was classified as asymmetry.

For statistical analysis the *t* test for paired or unpaired samples was used. To pool the data the values for the two sides were averaged. Normals were compared with each outcome category, and each outcome category was compared with the others. Only differences of high significance ($p < 0.01$, $p < 0.005$, $p < 0.001$) are presented in the results. Because it was also one aim of our paper to show which of the methods, SEP or BAEP, is less frequently affected by extracerebral (peripheral) lesions, the number of patients with brachial plexus lesions, cervical root trauma, and/or fractures of upper extremities was noted, as well as the number of patients with pre-existing deafness,

temporal bone fractures, and evidence of hemotympanum in the BAEP series. Furthermore, the number of patients who had technical problems (muscle artifacts) that prevented useful information was noted.

RESULTS

SEPs

The CCTs and ARs of normal persons, patients with good recovery, moderate and severe disability outcome, and brain death are listed in Table 1. There was an increase of CCT with worsening of outcome, showing highly significant differences ($p < 0.001$) between normals and patients who remained severely disabled or died, but also between normals and patients with good outcome. A less significant difference ($p < 0.01$) was between normals and the moderate disability outcome group. Significant differences in CCT ($p < 0.01$) were also found between patients with good recovery and severe disability or death outcome and between patients with moderate disability and death outcome. Highly significant differences ($p < 0.001$) were also noted between the ARs of normals and patients of all outcome categories.

In patients with secondary brainstem lesions (Table 2), the CCT appeared close to normal in the good outcome category. Highly significant differences ($p < 0.001$) were seen between the patients with good recovery and those who died. A further significant correlation ($p < 0.01$) appeared between good and severe disability outcome and between the moderate disability and the death outcome category. The ARs of patients later severely disabled differed significantly from normals ($p < 0.001$).

In cases with primary brainstem injury (Table 2) the CCT of patients with good outcome was strongly increased in comparison to normals ($p < 0.001$). No significant difference was found between primary brainstem-injured patients with good outcome and patients of the other outcome categories. The ARs were generally low in comparison to normals (Fig. 2, Fig. 3A and B) and showed a highly significant difference to normals in the good recovery, moderate disability, and brain death outcome categories ($p < 0.001$).

Asymmetry and absence of scalp SEPs in the different outcome categories are listed in Tables 1 and 2. Asymmetries were seen in the good outcome category as frequently as in the moderate disability and severe disability outcome groups. However, five of the eight patients with asymmetry and good outcome and four of the nine patients with asymmetry and moderate disability outcome were primary brainstem-injured patients. Distorted scalp SEPs on both hemispheres were found in six cases of the good outcome category (Fig. 3A), in five cases of the moderate disability outcome categories (Fig. 4), in seven cases of the severe disability, and in five cases of the brain death outcome category.

The number of asymmetries was relatively low in the death outcome category. However, 12 of these patients had absent SEPs on one or both hemispheres, and 3 patients showed markedly prolonged CCTs on both sides. Unilateral absence of scalp SEPs was not found in cases with good recovery, but it was found in three cases with moderate disability outcome. Bilateral absence of scalp SEPs was only seen in patients who survived severely disabled or died.

TABLE 1. SEP and BAEP findings in normals and in the different categories of all patients in post-traumatic coma

	SEP						BAEP						
	a	N	CCT	AR	Absent	Asymmetry	I-III	III-V, BTT	I-V	I:V	I:IV/V	Absent	Asymmetry
Normals	27	22	5.7	1.1			2.1	1.8	3.9	62	57		
SD	8		0.3	0.5			0.1	0.1	0.2	17	12		
Good outcome	25	22	6.7 ^c	0.5 ^c	0	8	2.1	1.9 ^c	4.1 ^a	94 ^c	77 ^a	4	8
SD	8		0.9	0.3			0.1	0.1	0.2	43	33		
Moderate disability	21	16	6.7 ^a	0.4 ^c	3	9	2.1	1.9	4.0	103 ^a	91 ^a	2	8
SD	4		1.0	0.3			0.1	0.1	0.2	53	41		
Severe disability	29	28	6.9 ^c	0.5 ^c	11	9	2.2	2.1 ^c	4.3 ^c	106 ^a	100 ^c	9	17
SD	13		0.9	0.4			0.2	0.2	0.3	41	37		
Death	24	20	7.7 ^c	0.5	12	5	2.3	2.1 ^b	4.4 ^c	127	110 ^a	13	13
SD	12		1.1	0.6			0.3	0.3	0.3	61	58		

Central somatosensory conduction time (CCT) increases and amplitude ratio (AR) N20:N13 decreases with worsening of outcome. Patients with good outcome also show an increase of CCT and decrease of AR. The interpeak latencies (IPLs) I-III, III-V, I-V, and the ARs I:V, I:IV/V increase with worsening of outcome, too. Important significant differences in comparison to normals are as follows: ^a p > 0.01; ^b p > 0.005; ^c p > 0.001. The values for two sides are averaged to pool the data. Furthermore, the number of absent or asymmetric scalp SEPs and BAEPs is listed. The number of absent SEPs and BAEPs increases with worsening of outcome. Asymmetries of SEPs distribute equally to all outcome categories. Asymmetries of BAEPs increase in lower outcome categories.

TABLE 2. CCT and AR of the different outcome categories in post-traumatic patients

	Primary brainstem injury					Secondary brainstem injury				
	N	CCT	AR	Absent	Asymmetry	N	CCT	AR	Absent	Asymmetry
Good outcome	11	7.2 ^a	0.4	0	5	11	6.3	0.7	0	3
SD		1.0	0.3				0.5	0.4		
Moderate disability	9	6.7	0.3	1	4	7	6.6	0.6	2	5
SD		1.1	0.2				1.0	0.4		
Severe disability	8	6.5	0.6	6	0	20	7.1	0.4	5	9
SD		0.7	0.5				0.9	0.3		
Death	1	5.4	0.5	1	0	19	8.0	0.5	12	5
SD							0.8	0.6		

Note the significant prolongation of CCT in cases with good outcome and primary brainstem injury in comparison to patients with good outcome and secondary brainstem involvement: $p < 0.001$. There is a clear increase of CCT and decrease of AR with worsening of outcome only in cases with secondary brainstem injury. Highly significant differences can be seen between patients with good recovery and severe disability outcome ($p < 0.01$). No absent scalp SEPs are found in cases with good recovery. Note the high number of absent SEPs in the severe disability outcome category after primary brainstem injury.

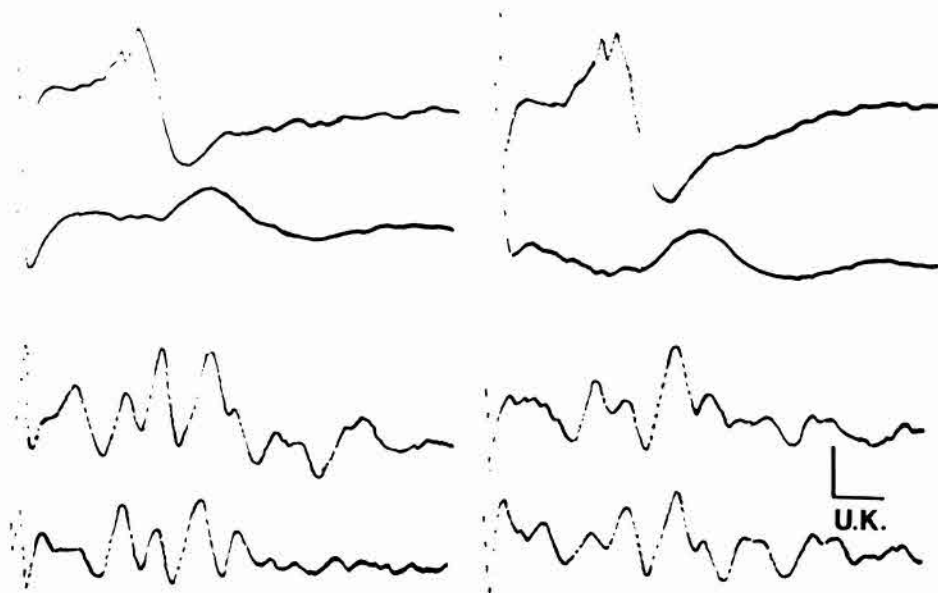


FIG. 2. Top: Cervical (upper channel) and scalp (lower channel) SEPs after right median nerve stimulation (left side) and after left median nerve stimulation (right side) in an 18-year-old female patient in midbrain syndrome Stage 2. Decerebrate posture is confined to the legs; this is a primary brainstem injury. CT was normal, and there was distorted N20 over both hemispheres, followed by poor positivity. A markedly prolonged CCT (7.5 ms on the left, 8.0 ms on the right) occurred. Calibration: 3.3 μ V; 5 ms/div. Bottom: BAEPs after right ear stimulation (left side) with response from ipsilateral derivation (upper channel) and contralateral derivation (lower channel), and BAEPs after left ear stimulation (right side) with ipsilateral derivation (lower channel) and contralateral derivation (upper channel). IPLs and ARs are within the range of normal. Bilateral recording was used for better identification of the wave IV/V complex. Abnormal SEPs contrast normal BAEPs, and outcome was good recovery. Calibration: 1.0 μ V; 1.2 ms/div.

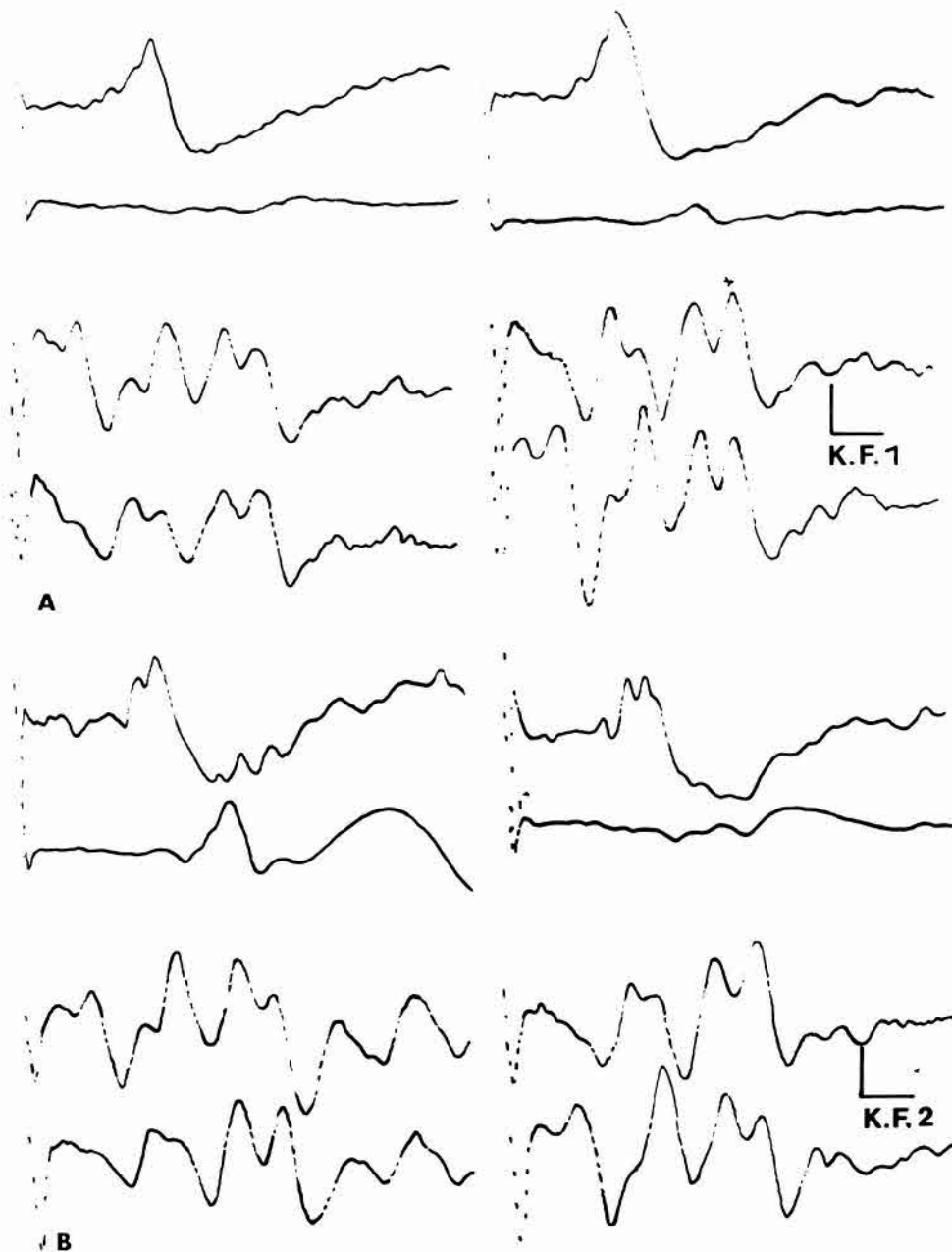


FIG. 3. **A:** Cervical and scalp SEPs after right and left median nerve stimulation in a 16-year-old male patient in midbrain syndrome Stage 4. There was decerebrate posture and secondary brainstem impairment. CT showed a right frontal area of contusion, small right frontal epidural hematoma, small scattered areas of contusions, and mild signs of tentorial herniation. Scalp SEPs were severely distorted, with a hardly detectable N20 over the left hemisphere. CCT was slightly prolonged to the left hemisphere but normal to the right hemisphere. BAEPs after right and left ear stimulation were within the range of normal. Calibration: 3.3 and 1 μ V; 5 and 1.2 ms. **B:** Cervical and scalp SEPs after right and left median nerve stimulation in the same patient (A) 8 days after the first test. There was significant improvement of the scalp response over the left hemisphere and a slightly better formed response over the right hemisphere. BAEPs were unchanged. At the time, clinical improvement was poor. There was recovery of the scalp SEPs, and the outcome was good recovery. Distribution of figures, derivations, and recording techniques are the same as in A and in Fig. 2.

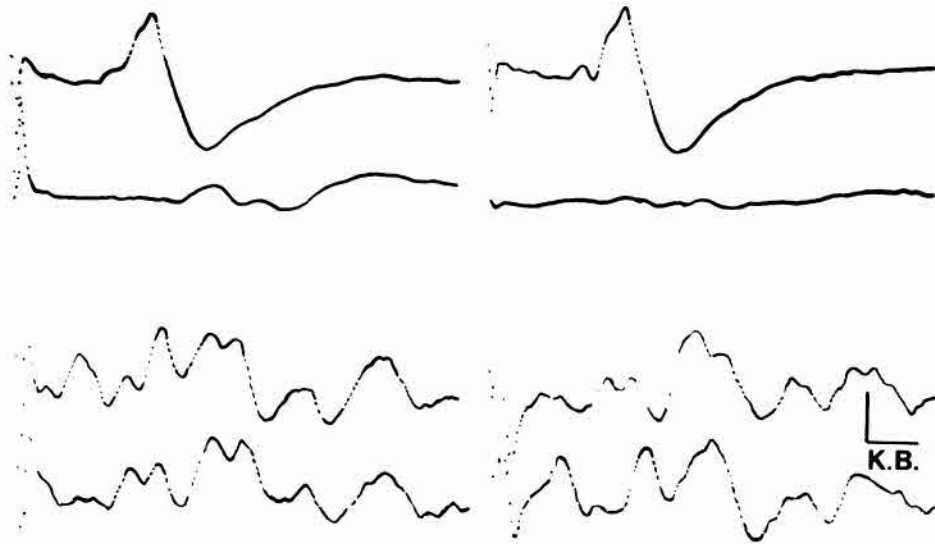


FIG. 4. Cervical scalp SEPs after right and left median nerve stimulation in a 22-year-old male patient in midbrain syndrome Stage 3 with lateralizing signs due to left pontine lesion. Decorticate posture was seen, and there was primary brainstem injury. CT showed a small area of contusion in the left frontal region; otherwise, findings were normal. Several distorted scalp SEPs more pronounced over the right hemisphere were seen; there were low ARs on both sides, and the CCT was extremely prolonged to right hemisphere (8.2 ms). BAEPs after right and left ear stimulation were normal; note the better performance of the IV/V complex in the contralateral derivation after left ear stimulation. Outcome was moderate disability. Distribution of figures, derivations, and recording techniques are the same as in Fig. 2.

BAEPs

The IPLs and ARs increased with worsening of outcome (see Table 1). Besides the increase of AR, it appeared that the amplitude of wave I became absolutely larger if waves IV or V were of small amplitude or were absent (Fig. 5). Significant differences in IPL I-V were found between patients with good recovery and severe disability or death outcome ($p < 0.01$) and between patients with moderate disability and severe disability outcome ($p < 0.005$) or moderate disability and death outcome ($p < 0.01$). BTT was strongly prolonged in patients with severe disability ($p < 0.005$) if compared with patients with good or moderate disability outcome. A significant difference ($p < 0.01$) was between the BTT of patients later moderately disabled and the patients who died.

The differences of IPLs and ARs between patients with primary and secondary brainstem lesions proved not to be significant in all outcome categories.

The IPL I-V exceeded 4.4 ms in 13 cases. Seven patients had bilateral prolongation of IPL I-V above this value. Four of these patients died; three remained severely disabled. Six patients had IPL I-V longer than 4.4 ms on one side. One patient of this group had a good recovery outcome. This patient suffered from primary brainstem injury. The other five patients died (one case) or survived severely disabled (four cases). Three of these patients were judged to suffer from primary brainstem impact.

The IPL III-V (BTT) was above 2.2 ms in 12 cases. Bilateral prolongation of BTT

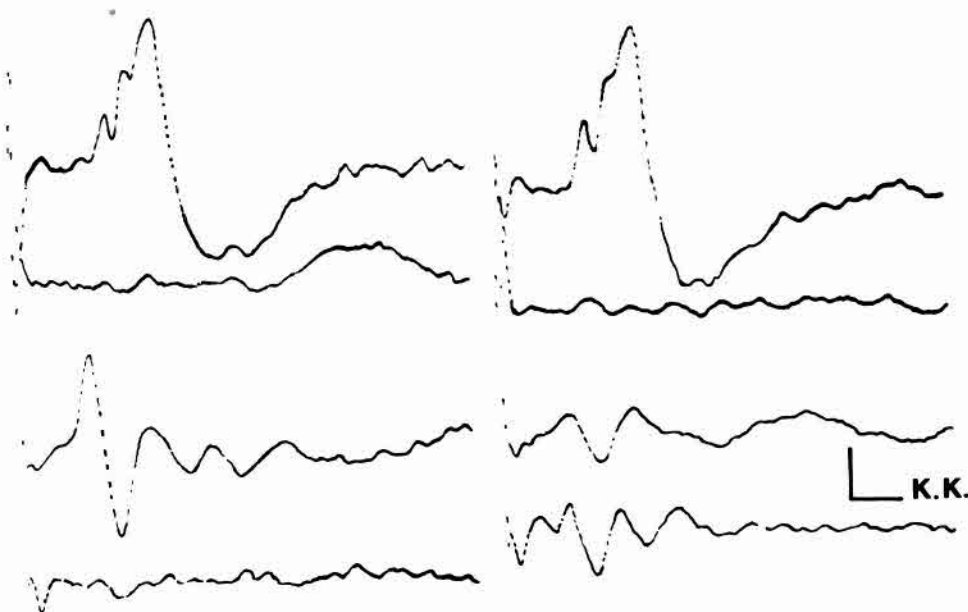


FIG. 5. Cervical and scalp SEPs after right and left median nerve stimulation in a 24-year-old male patient in midbrain syndrome Stage 4. Decerebrate posture and a secondary brainstem lesion were present. CT showed severe brain edema with large areas of contusion in the left and right temporoparietal regions, more pronounced on the left. N20 was absent over the right, with a poor response over the left hemisphere. BAEPs were recorded after right ear stimulation. There were a large-amplitude wave I and an absent wave V in the ipsilateral derivation, and no response in the contralateral derivation. After left ear stimulation, waves I to III were present, but later waves were absent. Outcome was brain death. Distribution of figures, derivations, and recording techniques are the same as in Fig. 2.

above this limit was seen in four cases. Three of these patients died, and one patient survived severely disabled. Eight patients had unilateral prolongation of BTT above 2.2 ms. Six of them had a severe disability outcome, one patient died, and one patient showed a moderate disability outcome. The last case was thought to suffer from primary brainstem injury.

Five patients were studied in brain death. They had totally absent BAEPs as well as bilateral absent scalp SEPs.

Four patients of the good outcome category had absent BAEPs, two of them on both sides (see Table 1). In one patient of this category, only waves III and V were present on one side. The patients with totally absent BAEPs had no recordable waves because of an unacceptable level of muscle artifacts. In one patient, there was pre-existing deafness in the left ear; another one had blood in the right middle ear (Fig. 6). No evidence of a traumatic peripheral hearing disorder could be demonstrated in the other patients of this group, either by clinical examination or by skull radiography.

One patient of the moderate disability outcome group had absent BAEPs on both sides. This patient suffered from hemotympanum on both sides. One patient had unilateral absent BAEPs; two patients demonstrated only wave III and IV/V complex on one side. The patient with unilateral absent BAEPs had ipsilateral temporal bone frac-

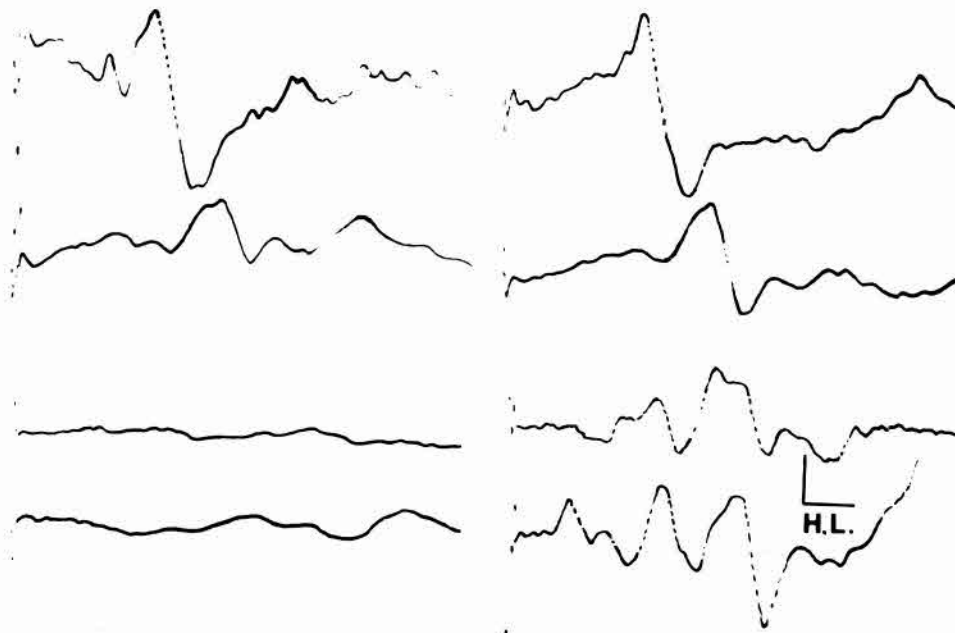


FIG. 6. Cervical and scalp SEPs after right and left median nerve stimulation in a 42-year-old female patient in midbrain syndrome Stage 2. Present were secondary brainstem injury and decorticate posture confined to the legs. CT demonstrated right frontal subdural hematoma and slight brain edema. The CCT was within the range of normal to both hemispheres, and N20 was of smaller amplitude in the left hemisphere. BAEPs were absent after right ear stimulation due to pre-existing deafness of the right ear. There was normal BAEPs after left ear stimulation and better performance of IV/V complex in the contralateral derivation. Outcome was good recovery. Distribution of figures, derivations, and recording techniques are the same as in Fig. 2.

ture. All other patients had normal clinical and skull radiographic findings. Technical problems in recording BAEPs was not noted in this group.

Patients with severe disability outcome demonstrated unilaterally absent BAEPs in five cases and totally absent BAEPs in four cases. Only waves III and V present on one side were found in two patients. Three patients demonstrated a singular wave V on one side with latencies of 6.0, 7.2, and 8.4 ms (Fig. 7), respectively. Two of the patients in this group had hemotympanum on both sides, and four patients had unilateral hemotympanum. Temporal bone fractures on skull radiography were seen in two cases.

In patients dying from brain death, totally absent BAEPs were found in seven patients. Six patients had unilateral absent BAEPs. In one patient, waves I to IV were present on the right and waves I to III on the left (see Fig. 5). Only wave I on both sides was found in one patient; another patient had wave I only on the left (Fig. 8). A unilateral singular wave V was found in three patients. One patient of this group had pre-existing deafness of both ears. A hemotympanum on one side was found in two cases. One patient had a temporal bone fracture, and two had multiple fractures of the base of the skull. Problems concerning an unacceptable level of noise or muscle artifacts in recording these patients were not noted.

Asymmetries in BAEPs were most frequently seen in the severe disability outcome

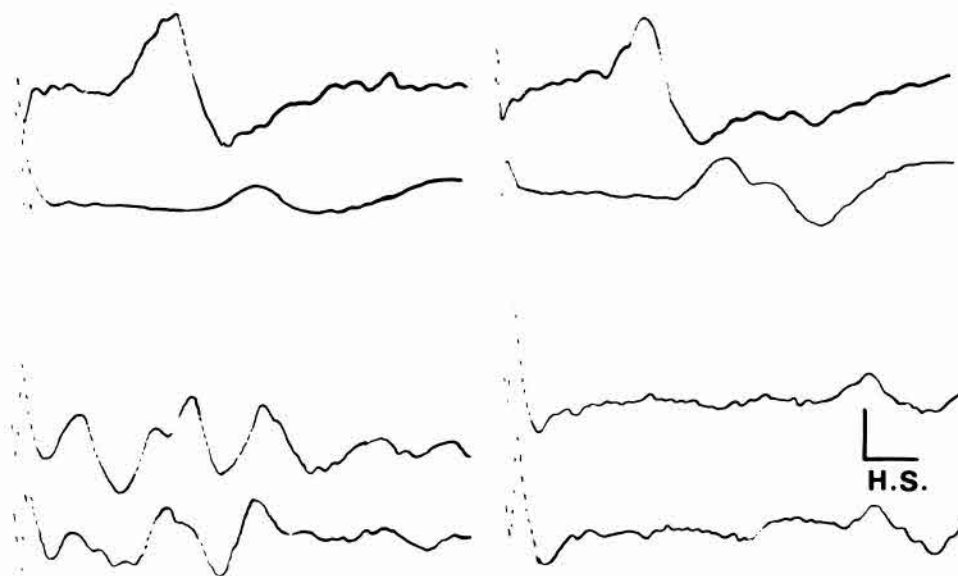


FIG. 7. Cervical and scalp SEPs after right and left median nerve stimulation in a 20-year-old male patient in midbrain syndrome Stage 4 with left lateralization signs. Decerebrate posture and secondary brainstem injury were present. CT showed severe brain edema and an intracerebral hematoma in left temporal region. The CCT prolonged to both hemispheres: 7.5 ms on the left, 8.0 ms on the right. Severely distorted small-amplitude scalp SEPs over the left hemisphere were seen. BAEPs after right ear stimulation demonstrated markedly prolonged IPL I-V (4.4 ms) but normal IPL III-V (1.9 ms). After left ear stimulation a strongly delayed singular wave at a 8.4-ms latency was demonstrated. Outcome was severe disability. Distribution of figures, derivations, and recording techniques are the same as in Fig. 2.

category (17 cases; see Table 1). Five of the six patients with asymmetry and good recovery and five of the eight patients with asymmetry and moderate disability outcome were classified as primary brainstem-injured patients. Asymmetries of BTT were seen in 13 patients. Two patients with asymmetric BTT had good recovery; one of them suffered from primary brainstem injury. The number of asymmetries in BTT increased in the moderate outcome group (6 cases) and severe disability outcome group (12 cases), and both groups included four patients with primary brainstem impact. There were only two asymmetries of BTT in the death category, but BAEPs were absent on one or both sides in 14 cases. Markedly prolonged BTT on both sides were seen in three cases developing brain death.

The combination of both tests (SEPs and BAEPs) showed that normal and abnormal responses in one of the tests were found frequently in the good and moderate disability outcome category, but both tests increasingly demonstrated abnormalities with worsening of outcome (Table 3). Using criteria requiring both sides to have a normal response for the test to be judged normal, five patients of the good outcome category had both tests normal. One patient each from the moderate disability category, severe disability category, and death outcome category had both tests normal. Using all criteria mentioned above to define abnormality (prolonged CCT, asymmetry of CCT, low AR, absence and asymmetry of scalp SEPs, prolonged IPLs including BTT, asymmetries of

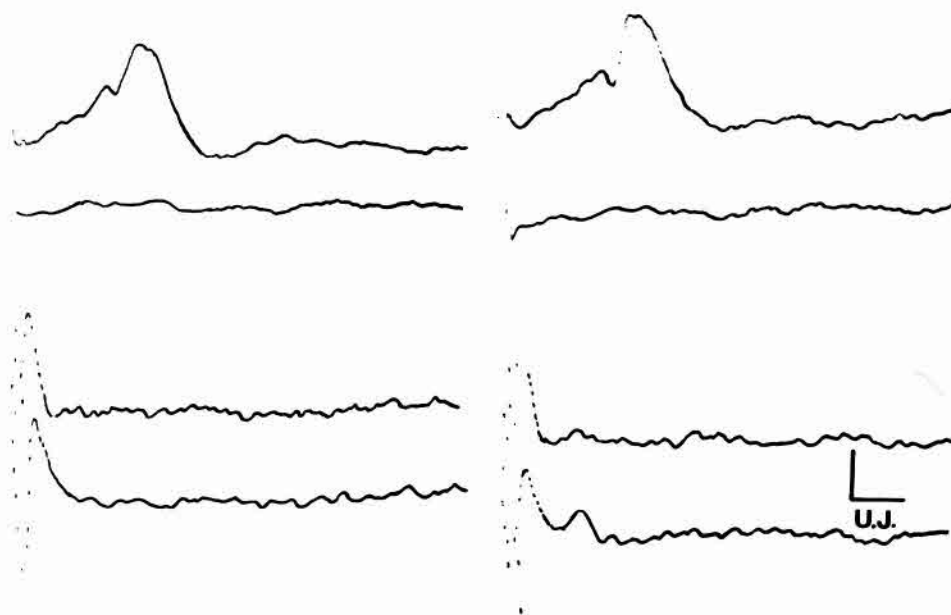


FIG. 8. Cervical and scalp SEPs after right and left median nerve stimulation in an 18-year-old male patient in bulbar brain syndrome Stage 2, flaccid coma, and with absent scalp SEPs over both hemispheres. BAEPs showed only wave I present after left ear stimulation. Outcome was brain death. Distribution of figures, derivations, and recording techniques are the same as in Fig. 2.

IPL I-V and BTT, high AR, absence and asymmetry of BAEPs) and requiring one side to have an abnormal response, five patients (23%) of the good outcome category had both tests abnormal. Eight patients (50%) of the moderate disability category had both tests abnormal. Eighteen patients (64%) of the severe disability category and 15 pa-

TABLE 3. Prognostic value of combination of SEPs and BAEPs related to the outcome categories

	N	Both tests normal	One test normal	Both tests abnormal	Not comparable
Combination of SEPs and BAEPs					
Good outcome	22	5	8	5	4
Moderate disability	16	1	4	8	3
Severe disability	28	1	4	18	6
Death	20	1	2	15	4

Both tests being abnormal is most frequently found in the low outcome categories (69%); both tests or one test being normal is more frequently seen in patients with good recovery or moderate disability outcome (47%) than in patients with severe disability or death outcome (16%). Prevention of comparison is mainly caused by damage to hearing mechanisms or muscle artifacts and thereby loss of BAEPs (14 of 17 cases).

tients (75%) of the death outcome category had both tests abnormal. A normal SEP finding was combined with an abnormal BAEP result in seven cases; five of them belonged to the good outcome category. One patient each was in the severe disability and in the death outcome category. An abnormal result of SEPs combined with normal BAEPs was seen in 11 cases. Three of them were in the good outcome category, four in the moderate disability category, three in the severe disability category, and one in the death outcome category. Bilateral absent scalp SEPs were not observed in patients with good or moderate disability outcome. In seven cases with severe disability outcome and absent N20 over both hemispheres, only one had absent BAEPs on both sides. In contrast, seven of the ten patients dying from brain death, in whom N20 was absent bilaterally, had totally absent BAEPs. Generally, the correlation between abnormal SEP and BAEP responses was poor in patients with good outcome, but the correlation between abnormal SEP and abnormal BAEP findings increased with worsening of outcome. No comparison of the tests could be drawn in 17 patients, because of the evidence of peripheral nerve lesions or technical problems. Extramedullary lesions of the auditory pathway or muscle artifacts prevented the ability to obtain reliable BAEPs in 14 cases (see Fig. 6). In only three cases was the reason for the defective comparison found in the absence of N13. In two cases, a unilateral brachial plexus lesion was responsible for the absence of one neck response. In one case, stimulation was impossible due to bone fractures of one upper extremity.

Follow-up studies were done in 18 patients. Four patients had good outcome (see Fig. 2A and B). Three of them showed bilateral improvement of CCT, whereas in one patient CCT slightly increased after right median nerve stimulation and decreased after left median nerve stimulation. In three patients, the IPLs were rather constant on both sides. In one patient, there was a general increase of IPLs on the right. ARs decreased in two cases and showed no remarkable changes in the others.

In the three cases with moderate disability outcome, the CCT generally decreased and the ARs of the SEPs increased. The IPLs including BTT showed no change in two cases. In one case, there was a slight increase of these latencies on the left. The ARs of the BAEPs were constant in two cases; in one case the ARs decreased.

The severe disability outcome group included nine patients. In two cases, scalp SEPs were absent and remained absent in two control examinations. In one patient, scalp SEPs were absent twice, but a poor unilateral response returned in two further controls. However, CCT increased in this case. In another case with both SEPs absent in the first examination, poor unilateral scalp SEPs appeared in a follow-up investigation. An increase of CCT on both sides was seen in one patient, whereas in the other four cases CCT decreased. ARs and waveforms of SEPs remained unchanged or showed only little improvement. The IPLs were stable in one case. In two cases, BAEPs appeared unchanged in one side, but a singular wave V could be detected on the other. In their first examination, all waves had been present on this side. One case showed all BAEPs on one side and an isolated wave V on the other. The follow-up record demonstrated a singular wave V on the side, where previously all waves had been present, and totally absent BAEPs on the other. One patient had unilateral absent BAEPs and prolonged IPLs on the other side. The follow-up recording revealed the appearance of waves III and V on one side but an increase of BTT on the other. IPLs increased on both sides in two cases; in one case, a symmetrical decrease of IPLs was

noted. In one case, BTT increased unilaterally. A return to normal IPLs was seen in one case. The ARs of BAEPs were unchanged in four cases. In two cases, AR increased unilaterally; in one case, AR was decreased unilaterally.

Two patients were studied in the death outcome category. In one patient, CCT was markedly prolonged after right and left median nerve stimulation. In the second recording, CCT was unchanged after right median nerve stimulation but decreased after left median nerve stimulation. The third record showed unchanged CCT after right median nerve stimulation but a decrease of CCT after left median nerve stimulation. The ARs generally decreased in the follow-up examinations. The IPLs I-V and the right BTT were markedly prolonged in the first recording. The IPLs I-V further increased in the second test but showed slight decrease in the third recording (Fig. 9A, B, and C). In the second case, CCT increased and ARs of the SEPs decreased on both sides. The first recording presented all BAEPs with normal IPLs including BTT. The second examination revealed bilateral late singular waves but absent earlier BAEP components.

Neurological Findings

Good Recovery Outcome Category

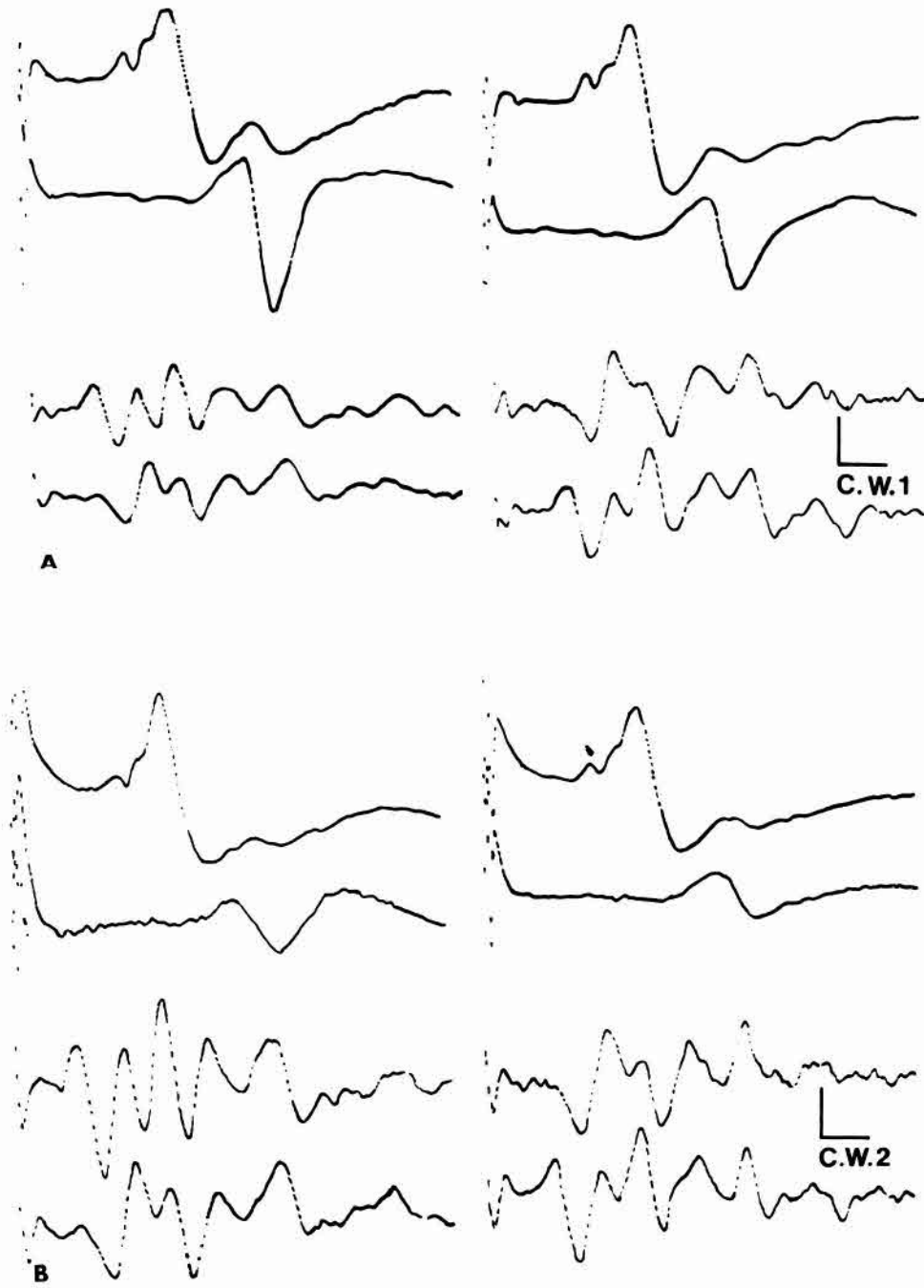
Fourteen of 22 patients in this category were classified in the early stages of midbrain syndrome (MBS 1 and MBS 2). Four patients belonged to MBS 4, and five patients belonged to MBS 3. Ten patients showed lateralizing neurological signs. Eleven patients were judged to suffer from primary brainstem injuries. This group included three patients in MBS 4. One patient with MBS 4 showed decerebrate posture after early surgical treatment of acute right-sided subdural hematoma. He had normal SEPs and BAEPs with a larger amplitude of N20 over the area of trepanation.

Moderate Disability Outcome

Eight of 16 patients with moderate disability outcome demonstrated signs of early stages of midbrain syndrome (MBS 1 and MBS 2). Four patients were judged to be in MBS 4, and four patients belonged to MBS 3. Ten patients demonstrated lateralizing neurological signs. Ten patients fulfilled the criteria of primary brainstem injury. One patient with MBS 3 was tested after surgical treatment of left epidural hematoma. CCT was slightly increased to left hemisphere, but AR was strongly increased over the area of trepanation when compared with the right side.

Severe Disability Outcome Category

Eighteen of 28 patients in this category were classified as MBS 4; five patients were classified as MBS 3. Two patients showed signs of early uncal herniation. Lateralizing signs other than uncal herniation were seen in five patients. Two patients were in MBS 2 and one in BBS 1. Eight patients were thought to suffer from primary brainstem injury. Three patients were recorded after surgical treatment of epidural hematoma (two cases) or subdural hematoma (one case). In two of these cases scalp SEPs were absent on both hemispheres. One patient had prolonged CCT to the side of removed hematoma with amplitude of N20 still very small on the side of trepanation. In one case later



FIGS. 9A and B. See legend on facing page.

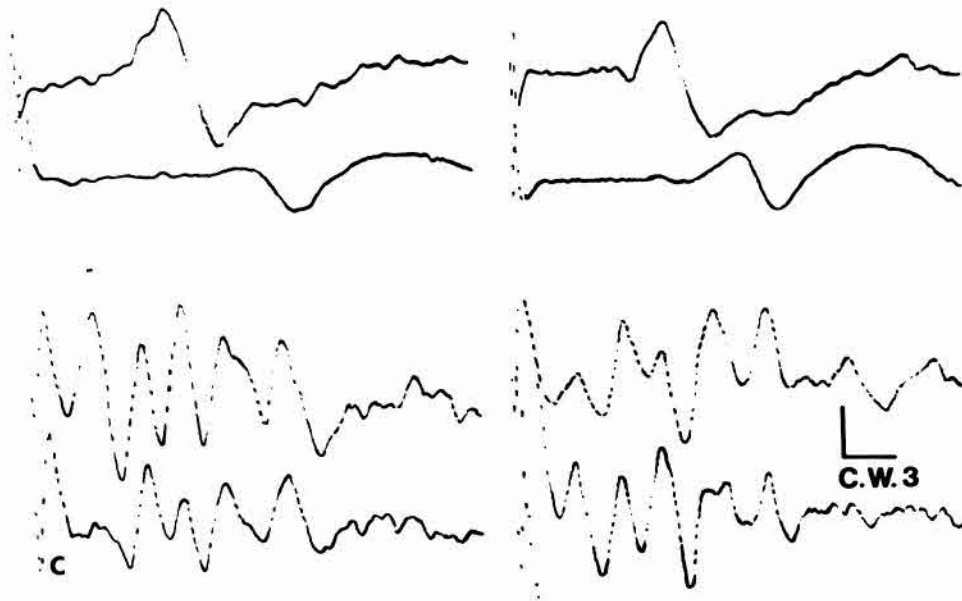


FIG. 9. A: Cervical and scalp SEPs after right and left median nerve stimulation in a 24-year-old male patient in midbrain syndrome Stage 4 with decerebrate posture and secondary brainstem injury. CT showed severe brain edema, large left temporoparietal intracerebral hematoma, and ischemic infarction in territory of posterior cerebral artery. There was tentorial herniation, and the CCT was prolonged to both hemispheres (7.8 ms, left; 8.2 ms, right). Normal waveform of scalp SEPs, but BAEPs showed extremely prolonged IPL I-V after right ear stimulation (4.6 ms) and after left ear (4.5 ms) stimulation. IPL III-V were markedly prolonged on the right (2.6 ms) and on the left (2.4 ms). **B:** Cervical and scalp SEPs from the same patient 1 day later show decreased amplitude of the scalp responses. The CCT to the left hemisphere was slightly decreased (7.5 ms) but was slightly increased to the right hemisphere (8.5 ms). BAEPs showed IPLs I-V increased after right ear stimulation (4.8 ms) as well as after left (4.7 ms) ear stimulation; there was recovery of IPL III-V on the right (2.1 ms). Bilateral increase of ARs was seen. **C:** Cervical and scalp SEPs of the same patient 10 days after the first test shows decreased amplitude of N20 over the left hemisphere. The CCT to the left hemisphere was unchanged (7.5 ms), but a clear decrease in the CCT to the right hemisphere was demonstrated (7.3 ms). BAEPs showed a decrease of IPL I-V after right ear stimulation (4.3 ms) but a constant finding after left ear (4.6 ms) stimulation. There was a further increase of ARs on the left, no change on the right. Outcome was brain death. Distribution of figures, derivations, and recording techniques are the same as in Fig. 2.

demonstrating the signs of a traumatic locked-in syndrome both SEPs and BAEPs were normal.

Death Category

Nine of the 20 patients who later were brain dead were in MBS 4. Three patients belonged to BBS 1 and seven patients belonged to BBS 2. Five of these patients fulfilled the clinical and EEG criteria of brain death. One patient showed signs of late uncal herniation. Only one patient was judged to suffer from primary brainstem impact.

CT Findings

Good Recovery Category (22 Cases)

CT was normal in eight cases, revealed slight brain edema in five cases, and showed moderate brain edema in two cases. Small local areas of contusion and small frontal or occipital hematomas were seen in six cases. Severe brain edema was found in one patient. Blood within the lateral ventricles was seen in one case. Small subdural or epidural hematomas were found in four cases. One patient with MBS 4, examined after surgical treatment of right-sided epidural hematoma, demonstrated a small subdural hematoma on the left side but otherwise had normal findings.

Moderate Disability Outcome Category (16 Cases)

CT was normal in four cases. A slight brain edema was seen in three cases, and a moderate brain edema was found in three cases. CT revealed severe brain edema in one case. Small areas of contusion and small intracerebral hematomas were seen in four cases. Small subdural hematomas were found in two cases. One patient showed an area of trepanation after surgical treatment of right epidural hematoma but otherwise had normal findings. Signs of tentorial herniation were found in one case.

Severe Disability Outcome Category (28 Patients)

CT demonstrated normal findings only in one case. Slight brain edema was seen in two cases, moderate brain edema was seen in three cases, and in eight cases CT showed severe brain edema. Large intracerebral hematomas were found in eight cases. Small subdural hematomas were seen in four; large epidural or subdural hematomas were seen in six cases. Three of these patients were surgically treated, but signs of brainstem compression were still present after surgery. Four patients showed CT evidence of tentorial herniation. Blood in the subarachnoid space was present in eight cases.

Death Outcome Category (20 Cases)

There were no normal CT findings in these patients. Brain edema was severe in 11 cases and moderate in 7 cases. Large intracerebral hematoma were found in five cases; epidural or subdural hematomas were found in five patients. Six cases demonstrated signs of tentorial herniation. In five cases, cerebral lesions were accompanied by traumatic subarachnoid hemorrhage.

DISCUSSION

In that critical period of treatment in which patients are sedated or paralyzed and artificially respirated, the use of evoked potential measurements is clearly of value to predict the outcome of patients, because at this moment clinical examination fails to give any useful information.

In our previous study, we emphasized that the absolute latencies of scalp SEPs in post-traumatic comatose patients did not differ from normals (Rumpl et al., 1983b). This finding confirmed the previous observation of Greenberg et al. (1977a), who

found absolute latencies of scalp SEPs similar to normals through the first 40 ms following stimulation in most brain-injured comatose patients. Because absolute (stimulus-to-peak) latencies of SEPs are affected by limb temperatures and peripheral neuropathies, clinical interpretation is based most useful on measurement of interwave latencies (Hume and Cant, 1978; Desmedt and Cheron, 1980; Eisen and Odusote, 1980; Chiappa and Ropper, 1982). Also the problem of how to ensure adequate stimulation of the median nerve when patients are paralyzed is solved by the simultaneous registration of the SEP from the neck. Some authors recorded peripheral nerve action potentials or neck SEP in order to verify the integrity of the peripheral receptors of somatosensory pathways (Goldie et al., 1981; Narayan et al., 1981, 1982; Anderson et al., 1984), but they did not use measurement of interwave latency for clinical interpretation.

Although only scalp SEPs were recorded in several studies, significant correlations between SEPs and outcome were noted (Greenberg et al., 1977*b*, 1981; De la Torre et al., 1978). A grading scheme, which used the presence or absence of waves to reflect the degree of deviation from normal, was developed by Greenberg et al. (1977*a*) and was frequently used in following studies (Greenberg et al., 1977*b*, 1981; Narayan et al., 1981, 1982). None of these included CCT to predict the outcome, but the number of SEP peaks and the outcome were found. However, it is not clear from these reports whether asymmetries in responses were noted and which response was used for further calculation. A more detailed study on SEPs was done by Lindsay et al. (1981), who found the highest correlation of outcome with the number of waves in response from the poorer hemisphere. A new grading scheme was introduced by Anderson et al. (1984). Their grade 2 record included prolongation of CCT, but also unilateral absence of the hemispheric positive deflection ("P22") with preserved N20. These authors found that unilateral or bilateral absence of SEPs strongly predicted an unfavorable outcome, but no further comment was given on CCT. Recently, Cant et al. (1986) presented a study in which they used CCT to grade SEP abnormality. Their grade 2 showed that "CCT always prolonged on one or both sides." These authors found no patients with good recovery in grade 2. Their observations suggested that no patient with persistent prolonged CCT recovered well.

The study of Hume and Cant (1981) showed that, in patients in post-traumatic coma, the CCT and the presence of SEPs over both hemispheres predicted the outcome. Thus, three-fourths of their patients showing normal CCT within 3.5 days of injury had good recovery, whereas consistent asymmetries of amplitude and CCT or long-term absence from one hemisphere predicted moderate or disability outcome. The absence of SEPs from both hemispheres was only noted in patients who died (Hume and Cant, 1981). Our previous observations (Rumpl et al., 1983*b*) generally confirmed the findings of CCT and AR close to normals in patients with good outcome and the increase of CCT and decrease of AR with worsening of outcome.

However, we also found prolonged CCT and asymmetric or absent SEPs in cases with good recovery. All these patients were judged to suffer from primary brainstem injuries. The current study confirmed these previous observations by demonstrating markedly prolonged CCT and asymmetric, severely distorted scalp SEPs in patients with good outcome. By separating the patients suffering from primary brainstem impact from those with secondary brainstem involvement, we could show that the general prolongation of CCT and the decrease of AR in patients with good recovery derived

from patients with primary brainstem injury. Singular patients with normal CCT could also be observed in the lower outcome categories, but most of them had unilateral absent scalp SEPs.

In contrast to our previous study (Rumpl et al., 1983*b*), in which we found bilateral absent SEPs in one case with good recovery, we did not see this phenomenon in the present study in the good outcome category or in the moderate disability outcome category. One explanation for this divergent finding might be seen in the varied time interval between the brain injury and the date of SEP recording. The majority of patients investigated in our previous study were recorded within the first 2 days after trauma, whereas the present study presents data mainly obtained after the second day of the trauma. Thus, the suppression or abolition of scalp SEPs might reflect a functional disturbance of neurons within the first 2 days after trauma, but prolonged absence or distortion of waves increasingly points to a structural lesion within the central somatosensory pathway. Therefore, one might assume that beyond the second day after brain injury, absent SEPs more strongly predict an unfavorable outcome. An exception to this rule in a case with primary brainstem injury was reported (Rumpl et al., 1983*b*). Despite full recovery, this female patient had unilateral absent scalp SEPs over 1 month. However, at the time a rather normal clinical finding in this patient contrasted to unilateral absence of scalp SEPs. A considerable difference of scalp SEPs was still noted 2 years after the first recording (Rumpl, 1985).

Although primary brainstem injury rarely exists in pure form (Mitchell and Adams, 1973), a primary brainstem lesion may be the principal cause of coma. Acute cervical hyperextension is the most important traumatogenic mechanism (Lindenberg and Freytag, 1970). Focal infarcts and hemorrhages occur in the distribution of perforating arteries in the medulla and the pons. These lesions are due to shearing of perforating arteries, which results from the differential motility of the brainstem and vessels (Pilz et al., 1982). Additionally, axial tension forces on the brainstem are leading to avulsions at the pontomedullary junction (Lindenberg and Freytag, 1970). In animal experiments, Gennarelli et al. (1981) reported avulsion of the brainstem at this level after extremely high acceleration of the head in the sagittal plane. Clinically, the appearance of neurological signs different from the expected rostral-caudal pattern (McNealy and Plum, 1962; Plum and Posner, 1966; Gerstenbrand and Lücking, 1970) may point to direct brainstem injury. CT is diagnostic and eliminates supratentorial lesions causing secondary brainstem dysfunction (Plum and Posner, 1980). Furthermore, the final outcome may be helpful for better distinction between primary and secondary brainstem lesions. Despite severe neurological findings, such as decerebrate posture immediately after the injury, the prognosis of these patients is generally good if the brainstem lesions are reversible. Because of the slight cortical impairment and good recovery of cognitive functions, cases with irreversible damage of the brainstem resemble a state described as traumatically induced locked-in syndrome (Britt et al., 1977). In cases without detectable lesions of the supratentorial regions in the CT scan, asymmetric or absent scalp SEPs, low ARs, prolonged CCT, and asymmetries in CCT may point to primary brainstem injury. As seen in our cases with good recovery, a single examination failed to give prognostic information. Repeated tests will show whether the lesion of the brainstem is reversible or not (Cant et al., 1986).

Unfortunately, the question of SEP pattern in primary brainstem injury was not dis-

cussed in previous papers (Greenberg et al., 1981; Narayan et al., 1982; Anderson et al., 1984; Cant et al., 1986). In cases with supratentorial lesions, our findings not only confirmed the results of Hume and Cant (1981), Lindsay et al. (1981), and Cant et al. (1986), but also our previous findings in patients with secondary brainstem impairment (Rumpl et al., 1983*b*). The present comparative study showed that BAEPs were a more significant prognostic aid than the SEPs. The IPLs I-III, especially III-IV (BTT) and I-V and the ARs I-V and I:IV/V, increased with worsening of outcome. Significant differences were not only found between normals and the different outcome categories, but also between patients in the good outcome or moderate disability outcome and patients with severe disability or death outcome.

In contrast, no significant difference was found if the BAEPs of patients with primary brainstem injury were compared with the BAEPs of patients with secondary brainstem lesions. Therefore, BAEPs appeared to be of little value in diagnosis of primary brainstem injury and did not support the hopeful suggestion of Tsubokowa et al. (1980) that BAEPs might be diagnostic in primary brainstem injuries. BAEPs confirmed the assessment of brain damage resulting from SEP findings in most cases with bad outcome, and discrepancies of normal SEPs combined with abnormal BAEPs and vice versa were more likely found in patients with moderate disability or good recovery outcome.

Looking specifically at BAEPs, our findings confirmed previous reports that alterations in IPLs and ARs might be related to the outcome (Uziel and Benezech, 1978; Seales et al., 1979; Tsubokowa et al., 1980; Karnaze et al., 1982; Yagi and Baba, 1983; Anderson et al., 1984). The IPL I-V was frequently used for grading BAEP abnormality and sometimes was termed central auditory conduction or transmission time (Seales et al., 1979; Klug, 1982; Anderson et al., 1984). Some authors did not define the time of prolongation (Greenberg et al., 1977*a*; Anderson et al., 1984; Cant et al., 1986), whereas other authors found the critical latency between survival (fair outcome in most cases) and those patients who died or remained severely disabled at about 4.4 ms (Karnaze et al., 1982; Yagi and Baba, 1983; Facco et al., 1985). A unilateral IPL I-V above this limit was only seen in one of our patients with good outcome. This patient was judged to be primary brainstem injured. All other patients with IPL I-V longer than 4.4 ms remained severely disabled or died. Therefore, our results confirm previous suggestions of a break point between reversible dysfunction and irreversible damage to the brainstem at this IPL.

The IPL III-V (BTT) was rarely mentioned at all (Uziel and Benezech, 1978; Karnaze et al., 1982; Klug, 1982) and was not used as a prognostic aid by these authors. Uziel and Benezech (1978) showed a significant increase of BTT in three of their patients but did not comment on the outcome of these cases. Klug (1982) only reported on patients who died and found a significant increase of BTT in comparison to normals. The critical limit, which may separate reversible from irreversible brainstem lesions, appeared at a latency of 2.2 ms, since only one patient with unilateral prolongation of BTT above this latency had a moderate disability outcome. Interestingly, this patient was judged to suffer from primary brainstem injury. All other patients with unilateral or bilateral prolongation of BTT above 2.2 ms remained severely disabled or died. The IPL I-V may strongly be impaired by lesions of the cochlear nerve until its entry to the brainstem. Therefore, we found acoustic BTT an appropriate term to define IPL III-V. Both IPL I-V and BTT were found to be of prognostic value, but BTT might be a more

accurate prognostic aid probably by exclusion of extramedullary lesions. We further found that the increase of ARs was closely related to the worsening of outcome. This increase of AR was not only due to small-amplitude IV/V complexes (Goldie et al., 1981), but also due to an absolutely large amplitude of wave I. The significant increase of ARs was also reported by Klug (1982) in his patients who died.

There is general agreement that the absence of BAEPs points to bad outcome. This was especially true in cases with preserved wave I and II and absent later waves, but also true in patients who had bilateral absent BAEPs not thought to be due to peripheral hearing loss (Uziel and Benezech, 1978; Tsubokowa et al., 1980; Goldie et al., 1981; Rosenberg et al., 1984; Ottaviani et al., 1986). The bilateral total loss of BAEPs was frequently found in brain death patients (Starr and Achor, 1975; Starr, 1976; Tsubokowa et al., 1980; Goldie et al., 1981; Klug, 1982). Our results confirmed these previous observations. Most of our patients who died and all patients studied in brain death had absent BAEPs, but also many of our patients who were later severely disabled had absent BAEPs at least on one side. This might partly result from peripheral injuries to the ears frequently found in severe head trauma. Conductive deafness following head injury is usually caused by blood in the middle ear. Interestingly, the absence of waves I and II accompanied by preserved waves III and V as well as the absence of waves I to IV with presence of a wave V was rarely mentioned in literature (Lindsay et al., 1981; Rohr et al., 1983; Cant et al., 1986). Lindsay et al. (1981) and Rohr et al. (1983) reported "significant" singular waves within the latency of wave V. Rohr et al. (1983) also found strongly prolonged large amplitude waves, which the authors classified as abnormal. However, no comment on the outcome of these patients was given. We observed a few patients with absent waves I and II, but rather normal waves III and V, in the good recovery, moderate disability, and severe disability outcome category. In contrast, a singular late wave, probably a wave V, which exceeded the normal latency of wave V in most cases, was only seen in patients with severe disability and death outcome.

Changes in click intensity produced marked changes in the absolute latency and amplitude of all BAEP waves (Jewett et al., 1970), but there was little change in IPLs (Terkildsen et al., 1973; Eggermont and Don, 1980). Auditory brainstem responses in normal subjects as a function of signal intensity showed a steady increase of latency and decrease of amplitude, as well as distortion of waves with decreasing intensity; finally only a wave V could be identified (Starr and Achor, 1975) and confirmed by our own observations in normals and comatose patients. Much of the BAEP behavior of diminished click intensity was the same as found in our patients with a singular late-latency wave. Damage to the conductive hearing mechanism (external canal obstruction, tympanic membrane perforation, hemotympanum) probably decreased click intensity and thereby might have been the cause for this BAEP pattern in our comatose patients. Other possible reasons were traumatic lesions of the cochlear nerve and, more frequently, an insufficiency of the cochlear blood supply deriving from a disturbed circulation of the basilar artery system. As seen in our patients also, this BAEP pattern reflected the severity of the head-brain injury. Although BAEPs appeared to yield more information about the patient's outcome than did SEPs, there are practical reasons for preferential use of SEPs (Cant et al., 1986). Damage to the ears is a frequent concomitant of head trauma, whereas damage to the upper limbs is a less frequent event. Damage to

hearing mechanism or muscle artifacts prevented the ability to obtain useful BAEP data in 14 cases, whereas only three patients had unilateral absent neck SEPs due to lesions of the upper limbs. To save time and cost, it appeared to be useful to start the evoked potential study with SEPs. In cases with bilateral normal scalp SEPs or bilateral absent scalp SEPs, BAEPs usually add no additional information. In cases with SEP findings of difficult interpretation, BAEPs are of clear additional value. For instance, in patients with primary brainstem injury and good outcome, normal BAEPs frequently contrasted the prolongation of CCT.

REFERENCES

- Anderson DC, Bundlie S, Rockswold GL. Multimodality evoked potentials in closed head trauma. *Arch Neurol* 1984;41:369-74.
- Britt RH, Herrick MK, Hamilton RD. Traumatic locked-in syndrome. *Ann Neurol* 1977;1:590-2.
- Cant BR, Hume AL, Judson JA, Shaw NA. The assessment of severe head injury by short-latency somatosensory and brainstem auditory evoked potentials. *Electroencephalogr Clin Neurophysiol* 1986;65:188-95.
- Chiappa KH. *Evoked potentials in clinical medicine*. New York: Raven Press, 1983:145-202.
- Chiappa KH, Ropper AH. Evoked potentials in clinical medicine (second of two parts). *N Engl J Med* 1982b;306:1205-10.
- De la Torre JC, Trimble JL, Beard RT, Hanlon K, Surgeon JW. Somatosensory evoked potentials for prognosis of coma in humans. *Exp Neurol* 1978;60:304-17.
- Desmedt JE, Cheron G. Central somatosensory conduction in man: neural generators and interpeak latencies of the far-field components recorded from neck and right or left scalp and earlobes. *Electroencephalogr Clin Neurophysiol* 1980;50:382-403.
- Eggermont JJ, Don M. Analysis of the click-evoked brainstem potentials in humans using high-pass noise masking. II. Effect of click intensity. *J Acoust Soc Am* 1980;68:1671-5.
- Eisen A, Odusote K. Central and peripheral conduction time in multiple sclerosis. *Electroencephalogr Clin Neurophysiol* 1980;48:253-65.
- Facco E, Martini A, Zuccarello M, Agnoletto M, Giron GP. Is the auditory brainstem response (ABR) effective in the assessment of posttraumatic coma? *Electroencephalogr Clin Neurophysiol* 1985;62:332-7.
- Garg BP, Markand ON, Bustion PF. Brainstem auditory evoked responses in hereditary motor-sensory neuropathy: site of origin of wave II. *Neurology* 1982;32:1017-9.
- Gennarelli TA, Adams HJ, Graham DI. Acceleration induced head injury in the monkey. I. The model, its mechanical and physiological correlates. *Acta Neuropathol (Berl)* 1981(suppl VII):23-5.
- Gerstenbrand F, Lücking CH. Die akuten traumatischen Hirnstammschäden. *Arch Psychiat Neurochir* 1970;213:264-81.
- Gerstenbrand F, Rimpl E. Das prolongierte Mittelhirnsyndrom traumatischer Genese. In: Neumärker KJ, ed. *Hirnstammläsionen*. Leipzig: S. Hirzel, 1983:236-48.
- Goldie WD, Chiappa KH, Young RR, Brooks EB. Brainstem auditory and short-latency somatosensory evoked responses in brain death. *Neurology* 1981;31:248-56.
- Greenberg RP, Mayer DJ, Becker DP, Miller JD. Evaluation of brain function in severe human head trauma with multimodality evoked potentials. Part 1. Evoked brain-injury potentials, methods and analysis. *J Neurosurg* 1977a;47:150-62.
- Greenberg RP, Newlon PG, Hyatt MS, Narayan RK, Becker DP. Prognostic implications of early multimodality evoked potentials in severely head-injured patients. A prospective study. *J Neurosurg* 1981;55:227-36.
- Greenberg RP, Becker DP, Miller JD, Mayer DJ. Evaluation in brain function in severe human head trauma with multimodality evoked potentials. Part 2. Localisation of brain dysfunction and correlation with posttraumatic neurological conditions. *J Neurosurg* 1977b;47:163-77.
- Hume AL, Cant BR. Central somatosensory conduction after head trauma. *Ann Neurol* 1981;10:411-9.
- Hume AL, Cant BR. Conduction time in central somatosensory pathways in man. *Electroencephalogr Clin Neurophysiol* 1978;45:361-75.
- Jennet B, Bond M. Assessment of outcome after severe brain damage. *Lancet* 1975;i:480-4.
- Jewett DL, Romano MN, Williston JS. Human auditory evoked potentials: possible brainstem components detected on the scalp. *Science* 1970;167:1517-8.

- Karnaze DS, Marshall LF, Mc Carthy CS, Klauber MR, Bickford RG. Localizing and prognostic value of auditory evoked responses in coma after closed head injury. *Neurology* 1982;32:299-302.
- Klug N. Brainstem auditory evoked potentials in syndromes of decerebration, the bulbar syndrome and central death. *J Neurol* 1982;227:219-28.
- Lindenberg R, Freytag E. Brainstem lesions characteristic of traumatic hyperextension of the head. *Arch Pathol* 1970;90:509-15.
- Lindsay KW, Carlin J, Kennedy I, Fry J, Mc Innes A, Teasdale GM. Evoked potentials in severe head injury—analysis and relation to outcome. *J Neurol Neurosurg Psychiatry* 1981;44:796-802.
- Maciver JN, Lassmann LP, Thomas CW, McLeod J. Treatment of severe head injury. *Lancet* 1958;ii:544-50.
- Mc Nealy DE, Plum F. Brainstem dysfunction with supratentorial mass lesions. *Arch Neurol* 1962;7:26-48.
- Miller JD. Barbiturates and raised intracranial pressure (editorial). *Ann Neurol* 1979;6:189-93.
- Mitchell DE, Adams JH. Primary focal impact damage to the brainstem in blunt head injury. Does it exist? *Lancet* 1973;ii:215-8.
- Mjoen S, Nordby HK, Torvik A. Auditory evoked brainstem responses (ABR) in coma due to severe head trauma. *Acta Otolaryngol* 1983;95:131-8.
- Narayan RK, Greenberg RP, Miller DJ, Enas GG, Choi SC, Kishore PRS, Selhorst JB, Lutz HA, Becker DP. Improved confidence of outcome prediction in severe head injury. A comparative analysis of the clinical examination, multimodality evoked potentials, CT scanning, and intracranial pressure. *J Neurosurg* 1981;54:751-62.
- Narayan RK, Kishore PRS, Becker DP, Ward JD, Enas GG, Greenberg RP, Da Silva AD, Lipper MH, Choi SC, Mayhall CG, Lutz HA, Young HF. Intracranial pressure: to monitor or not to monitor? A review of our experiences with severe head injury. *J Neurosurg* 1982;56:650-9.
- Newlon PG, Greenberg RP, Hyatt MS, Enas GG, Becker DP. The dynamics of neuronal dysfunction and recovery following severe head injury assessed with serial multimodality evoked potentials. *J Neurosurg* 1982;56:168-77.
- Ottaviani F, Almadori G, Calderazzo AB, Frenguelli A, Paludetti G. Auditory brainstem (ABRs) and middle latency auditory responses (MLRs) in the prognosis of severely head-injured patients. *Electroencephalogr Clin Neurophysiol* 1986;65:196-202.
- Pilz P, Strohecker J, Grobovschek M. Survival after ponto-medullary tear. *J Neurol Neurosurg Psychiatry* 1982;45:422-7.
- Plum F, Posner JB. *Diagnosis of stupor and coma*, 1st ed. Philadelphia: Davis, 1966:78-95.
- Plum F, Posner JB. *Diagnosis of stupor and coma*, 3rd ed. Philadelphia: Davis, 1980:73-4.
- Rohr W, Zschokke ST, Janzen RWC. Use of auditory evoked brainstem response (AEBR) in coma. *Electroencephalogr Clin Neurophysiol* 1983;56:160P.
- Rosenberg C, Wogensen K, Starr A. Auditory brainstem and middle- and long-latency evoked potentials in coma. *Arch Neurol* 1984;41:835-8.
- Rumpl E. Anwendung der SEP in der Intensivmedizin. *Akt Neurol* 1985;12:53-7.
- Rumpl E, Prugger M, Bauer G, Gerstenbrand F, Hackl JM, Pallua AK. Incidence and prognostic value of spindles in posttraumatic coma. *Electroencephalogr Clin Neurophysiol* 1983a;56:420-9.
- Rumpl E, Prugger M, Gerstenbrand F, Hackl JM, Pallua AK. Central somatosensory conduction time and short latency somatosensory evoked potentials in posttraumatic coma. *Electroencephalogr Clin Neurophysiol* 1983b;56:583-96.
- Seales DM, Rossiter VS, Weinstein ME. Brainstem auditory evoked responses in patients comatose as a result of blunt head trauma. *J Trauma* 1979;19:347-53.
- Starr A. Auditory brainstem response in brain death. *Brain* 1976;99:543-54.
- Starr A, Achor J. Auditory brainstem responses in neurological disease. *Arch Neurol* 1975;32:761-8.
- Stockard JJ, Stockard JE, Sharbrough FW. Nonpathologic factors influencing brainstem auditory evoked potentials. *Am J EEG Technol* 1978;18:177-209.
- Stockard JJ, Stockard JE, Sharbrough FW. Brainstem auditory evoked potentials in neurology: methodology, interpretation, clinical application. In: Aminoff MJ, ed. *Electrodiagnosis in clinical neurology*. New York: Churchill Livingstone, 1980:370-413.
- Terkildsen K, Osterhammel P, Huis in't Veld F. Electrocochleography in far-field technique. *Scand Audiol* 1973;2:141-8.
- Tsubokawa T, Nishimoto H, Yamamoto T, Kitamura M, Katayama Y, Moriyasu N. Assessment of brainstem damage by the auditory brainstem response in acute severe head injury. *J Neurol Neurosurg Psychiatry* 1980;43:1006-11.
- Uziel A, Benezech J. Auditory brainstem response in comatose patients: relationship with brainstem reflexes and levels of coma. *Electroencephalogr Clin Neurophysiol* 1978;45:515-24.
- Yagi T, Baba S. Evaluation of the brainstem function by the auditory brainstem response and the caloric vestibular reaction in comatose patient. *Arch Otorhinolaryngol* 1983;238:33-43.