Reversible brain-stem dysfunction following acute traumatic subdural hematoma

A clinical and electrophysiological study

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Of 366 consecutive patients with severe head injury, treated and managed by a uniform protocol, 61 (17%) were admitted with signs of severe brain-stem dysfunction. Forty-three of the 61 patients (70%) had surgical mass lesions and 30% had diffuse brain damage. Twelve of the 61 patients (20%) survived, but only six patients made a good to moderately disabled recovery. All six of these patients had a traumatic acute subdural hematoma (SDH). The records of the 20 comatose patients with an acute SDH and severe brain-stem dysfunction were reviewed to discover which factors contributed to functional recovery. The average survivor was operated on within 21 hours after injury and the nonsurvivors within 48 hours. Prompt surgical intervention and prudent control of postoperative intracranial pressure were major factors in preventing permanent brain-stem damage, with a significance of p < 0.05 and p < 0.02, respectively. Measurement of multimodality evoked potentials in the early postoperative period correctly distinguished between reversible and irreversible brain-stem dysfunction in six of seven patients.

KEY WORDS: subdural hematoma · brain stem · head injury · intracranial pressure · evoked potentials

In head-injured patients, the presence of a combination of three clinical signs (namely: 1) bilaterally impaired or absent oculocephalic/oculovestibular reflexes, 2) bilaterally absent pupillary light reflexes, and 3) decerebrate posture) is thought to indicate severe brain-stem dysfunction or damage, and as such has grave prognostic significance. Over 80% of patients with this constellation of three adverse neurological signs die following head injury. Those patients who have had these three adverse neurological signs and survive do not often make a functional recovery (good recovery or moderate disability): they either die or remain in a severely disabled or vegetative state.

A group of patients from the Medical College of Virginia head-injury series has made a functional recovery despite having this constellation of clinical signs indicating severe brain-stem dysfunction. Thus, either the presence of these three adverse neurological signs does not uniformly forecast a grave outcome, or brain-stem dysfunction can be reversible. We reviewed our clinical and electrophysiological experience with those head-injury patients who had the three adverse neurological signs and made a good to moderately disabled recovery to identify the factors that contributed to their favorable outcomes. Additionally, we hoped to gain insight into the phenomenon of reversible brain-stem dysfunction.

Clinical Material and Methods

Background for Case Selection

The patients evaluated for this retrospective study comprise a subset of the 366 consecutive cases of
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severe non-missile-related head injury admitted to the Division of Neurological Surgery, Medical College of Virginia, from December, 1972, through February, 1980. All patients included in this study were noted after resuscitation to be unresponsive to verbal command on initial neurosurgical evaluation, but were able to breathe spontaneously and had negative drug and toxic screens. All were older than 2 years. During this time interval, 61 of the 366 (16%) comatose patients were admitted with the three adverse neurological signs, indicating severe brain-stem dysfunction. They comprise the series of patients studied. All patients were managed similarly according to a protocol that has been described previously.2 Every patient with a hematoma that caused a midline brain shift greater than 5 mm was treated surgically. Rapid temporal craniectomy with partial evacuation of any clot (epidural or subdural) immediately preceded temporofrontoparietal craniotomy in all cases. Only hematomas in the subdural space with at least 5 mm thickness were categorized as an acute subdural hematoma (SDH). After removal of the SDH, any significant hemorrhagic contusions, especially in the anterior frontal and temporal regions, were identified and carefully evacuated.

Method of Analyzing Evoked Potentials

A complete study of the multimodality evoked potentials (MEP) consisted of visual, auditory, and somatosensory cortical evoked potentials (VEP, AEP, and SEP, respectively) together with auditory and somatosensory brain-stem evoked potentials. Electroretinograms, eighth nerve action potentials, and peripheral nerve action potentials were recorded when necessary to verify the integrity of the peripheral receptors of each of the above systems. The technical aspects of obtaining and analyzing MEP data in comatose head-injured patients have been described in earlier reports.14-16 Evoked potential studies were performed on Days 1, 4, 14, 90, 180, and 365 following injury, but only the data from the earliest study were used for this report. All studies were analyzed prospectively, the investigator having no knowledge of the patient's condition or ultimate outcome.16 The data were entered into a computer data bank following the completion of each study.

Electrophysiological evidence of brain-stem dysfunction was considered present when both the auditory and somatosensory brain-stem potentials were severely abnormal or absent.14,16,17,30 If both the brain-stem and hemispheric responses were severely abnormal or absent, the patients' electrophysiological dysfunction was described as disseminated; a circumstance common in head-injured patients.16,30

Method of Statistical Analysis

The proportions presented in Table 3 were compared by Fisher's exact test because of the small sample sizes. The mean time intervals were compared using the Aspin-Welch t-test after normalizing logarithmic transformation was applied for the time interval. The mean time lapse from injury to surgery is expressed as mean ± the standard error (SE) of the mean. A significant difference was accepted when p < 0.05.

Results

Of the 366 cases with severe closed-head injury, 233 patients (61%) had a diffuse head injury without a mass, 82 patients (22%) had acute SDH, and 61 patients, (17%) had either an epidural or intracerebral contusion hematoma. Sixty-one patients (17%) were admitted with a combination of the three adverse neurological signs. Of these patients, 18 had a diffuse head injury, 23 had either an epidural or intracerebral hematoma, and 20 patients had an acute SDH (Table 1). Twelve of the 61 patients with the three adverse neurological signs survived; however, only six patients made a functional recovery (Table 2). Six other surviving patients did not make a functional recovery and were vegetative or severely disabled; three had diffuse head injury; two had intracerebral contusion-hematomas; and one had an epidural hematoma.

Because the six patients with the three adverse neurological signs who made a functional recovery all had an acute SDH, we compared them only to patients

<table>
<thead>
<tr>
<th>Type of Head Injury</th>
<th>Cases With TANS</th>
<th>Cases Without TANS</th>
</tr>
</thead>
<tbody>
<tr>
<td>mass lesions (operable cases)</td>
<td>43 70</td>
<td>100 33</td>
</tr>
<tr>
<td>acute subdural hematoma</td>
<td>20 33</td>
<td>62 20</td>
</tr>
<tr>
<td>epidural hematoma or intracerebral contusions</td>
<td>23 37</td>
<td>38 13</td>
</tr>
<tr>
<td>diffuse brain injury (nonoperable cases)</td>
<td>18 30</td>
<td>205 67</td>
</tr>
<tr>
<td>total</td>
<td>61 100</td>
<td>305 100</td>
</tr>
</tbody>
</table>
TABLE 2

Outcome of patients with TANS following head injury*

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Cases</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Percent</td>
</tr>
<tr>
<td>functional recovery (good recovery/moderate disability)</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>severe morbidity (severely disabled/vegetative)</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>deaths</td>
<td>49</td>
<td>80</td>
</tr>
<tr>
<td>total</td>
<td>61</td>
<td>100</td>
</tr>
</tbody>
</table>

* TANS = the three adverse neurological signs, see introduction.

who had both the three adverse signs and acute SDH (20 of 61 patients), instead of all patients with the three adverse signs (61 patients). By limiting our comparison to patients with acute SDH and the three adverse neurological signs (Table 3), we hoped to eliminate the pathophysiological variability between different subsets of head-injured patients.

Age and Sex

The mean age of the 20 patients with an acute SDH and the three adverse neurological signs was 32 years. Age was not significantly different between patients with functional recovery and those who died.

TABLE 3

Comparison of variables influencing acute SDH outcome with the presence of signs of severe brain-stem dysfunction*

<table>
<thead>
<tr>
<th>Feature</th>
<th>Death</th>
<th>Functional Recovery</th>
<th>Significance of Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>no. of cases</td>
<td>14</td>
<td>6</td>
<td>NS</td>
</tr>
<tr>
<td>age (yrs)</td>
<td>33</td>
<td>30</td>
<td>NS</td>
</tr>
<tr>
<td>sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>male</td>
<td>12</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>female</td>
<td>2</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>mode of injury</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>vehicular-related accident</td>
<td>9</td>
<td>4</td>
<td>NS</td>
</tr>
<tr>
<td>cerebral contusion-hematoma (surgically removed)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>present</td>
<td>10</td>
<td>5</td>
<td>NS</td>
</tr>
<tr>
<td>absent</td>
<td>4</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>postoperative ICP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 20 mm Hg</td>
<td>2</td>
<td>5</td>
<td>p &lt; 0.02</td>
</tr>
<tr>
<td>uncontrollable</td>
<td>9</td>
<td>0</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>time of injury to surgical decompression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>no. of cases†</td>
<td>12</td>
<td>5</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>mean time lapse ± SE (min)</td>
<td>273 ± 55</td>
<td>140.6 ± 23</td>
<td></td>
</tr>
</tbody>
</table>

* SDH = subdural hematoma; NS = not significant; SE = standard error; ICP = intracranial pressure.
† Onset of injury could not be determined in three patients.

Seventy-five percent of these patients were males, and 86% of the deaths occurred among males; whereas 50% of the functional recoveries were among females, and 60% of the females made a functional recovery. Because of the low number of females in this study, females did not have a significantly better outcome than males.

Mode of Injury

Vehicular-related accidents, consisting of automobile, motorcycle, and pedestrian accidents, occurred in approximately 65% of the patients with acute SDH and signs of severe brain-stem dysfunction. They occurred at the same frequency in all patient groups regardless of outcome.

Intracranial Pressure

Postoperative intracranial pressure (ICP) was continuously monitored in all patients for at least the first 3 days. The peak ICP was defined as the highest ICP recorded. Uncontrollable ICP was defined as greater than 60 mm Hg, sustained, and unaltered by ventricular cerebrospinal fluid drainage, mannitol, or hyperventilation. Outcome in patients with an acute SDH and the three adverse neurological signs was substantially influenced by ICP (Table 3). Only 14% of the patients with a fatal outcome had a peak ICP of less than 20 torr compared to 84% of those with a functional recovery (p < 0.001). Uncontrollable ICP was prevalent in 64% of those with the three adverse signs who died, and was never present in patients who recovered (p < 0.05).

Type of Intracranial Mass Lesions

Of the comatose head-injured patients with signs of severe brain-stem dysfunction, 70% had mass lesions amenable to surgery. Of the patients without the three adverse neurological signs, 67% had diffuse brain damage (no mass) (p < 0.001). Only 33% of head-injured patients who were admitted comatose but without signs of brain-stem dysfunction had operable mass lesions (Table 1).

All six head-injured patients with the three adverse neurological signs who made a functional recovery had acute SDH. Five of these patients had associated cerebral contusions; however, the presence or absence of a cerebral contusion did not statistically alter outcome (Table 3).

Multimodality Evoked Potentials

In seven of the 20 patients with the three adverse neurological signs and acute SDH, MEP were recorded within 48 hours of admission to the hospital
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and after surgical removal of the hematoma. Six of the seven patients had normal to mildly abnormal VEP, AEP, and SEP, as well as normal auditory and somatosensory brain-stem evoked potentials (Fig. 1). Five of the six patients (83%) with normal MEP made a functional recovery. The one patient with normal MEP who failed to have a good outcome had a delayed intracerebral contusion-hematoma with massive brain shift on the side opposite to the acute SDH; this occurred several days after the MEP study was performed, and the patient died. On the initial computerized tomography (CT) scan, this hemorrhage was not visible.

One of the seven patients with MEP recording had severely abnormal evoked potentials; this patient died. This patient’s brain-stem SEP was isoelectric except for the “P13” component, thought to be generated in the brain-stem medulla (Fig. 2). Somatosensory component N20, generated in the primary somatosensory cortex, was absent. Thus, SEP data indicated a lesion between the pontomedullary junction and the cortex. The first five positive waves of the auditory brain-stem potential were present, but all subsequent auditory wave peaks were absent (Fig. 2). This finding would place the lesion rostral to the pons. Furthermore, VEP were present but abnormal; the waveform was monophasic, having lost the complexity of a normal VEP, and indicated that cortical dysfunction was also present in this patient. Since a VEP was obtained, one can speculate that the brain stem was functional at the level of the lateral geniculate bodies. On the basis of MEP, data this patient’s central nervous system dysfunction or lesion resulting from head trauma was determined to be in the midbrain as well as the cortex.

Promptness of Surgical Decompression

Because of the time delay associated with cerebral angiography, from 1972 to mid-1975 we relied initially on emergency (twist-drill) air ventriculography for rapid determination of ventricular compression, shift, and ICP measurement and to evaluate the need for surgical intervention. Computerized tomography has substantially improved the rapid and accurate diagnosis of surgical mass lesions, ventricular size, and ventricular shift. Since mid-1975, virtually all of our head-injured patients have been evaluated by emergency CT. The promptness of surgical decompression has been measured by determining the time from injury to the time of surgical decompression (as recorded in the anesthesia record). Patients who made a functional recovery and had signs of severe brain-stem dysfunction underwent surgery an average of 140 minutes after head injury (Table 3), whereas patients who died underwent surgical decompression an average of 273 minutes following their injury (p < 0.05).

Discussion

Severe brain-stem dysfunction was reversible in our series. Of 366 cases of closed-head injury, 61 deeply comatose patients were admitted with a combination of neurological signs indicative of severe brain-stem dysfunction caused by either direct compression, ischemia, or primary damage of the brain stem. Although 12 patients survived (20%), only six (10%) made a functional recovery. All six patients who made a functional recovery were found at surgery to have an acute SDH, with 83% having an associated intracerebral contusion-hematoma. Our results suggest that patients presenting with bilateral absent pupillary

FIG. 1. Visual (VER), somatosensory (SER), somatosensory brain-stem (SBSR), and auditory brain-stem (ABSR) evoked potentials obtained postoperatively from a comatose head-injured patient whose clinical findings on admission included bilaterally impaired ocuulococulor/oculovestibular reflexes, bilaterally absent pupillary light reflexes, and decerebrate posturing. This patient’s evoked potentials are normal to mildly abnormal, and are consistent with a good functional recovery.
FIG. 2. Evoked potential data recorded from a patient with the three adverse neurological signs (see text). This patient had disseminated dysfunction of the central nervous system defined by the multimodality evoked potential study. An area of dysfunction/lesion was identified in the midbrain because: 1) the somatosensory brain-stem evoked potential was isoelectric except for the P15 component, and 2) the first five positive waves of the auditory brain-stem evoked potentials were recorded, but all subsequent auditory wave-peaks were absent (see text). Furthermore, the patient's visual cortical evoked potentials were present, although abnormal, indicating that: 1) the brain stem at the level of the lateral geniculate bodies was probably intact, and 2) cortical dysfunction was present (see text).

Clinical Recovery in Patients with Brain-Stem Dysfunction

Reversal of brain-stem dysfunction, manifested by impaired or absent oculocephalic/oculovestibular function, and decerebrate rigidity (the three adverse neurological signs) do poorly unless an acute SDH is present. However, the reversibility of brain-stem dysfunction in our group of patients with acute SDH was dependent on rapid diagnosis and prompt surgical evacuation of the mass as well as careful control of postoperative ICP. The patient's age and sex, the mode of injury, and the presence or absence of an associated cerebral contusion did not significantly influence reversibility of brain-stem dysfunction (Table 3). Evoked potential data, however, accurately forecast patient outcome, as they are a valuable indicant of the state of the brain stem.

Light reflexes, impaired or absent oculocephalic/oculovestibular function, and decerebrate rigidity (the three adverse neurological signs) do poorly unless an acute SDH is present. However, the reversibility of brain-stem dysfunction in our group of patients with acute SDH was dependent on rapid diagnosis and prompt surgical evacuation of the mass as well as careful control of postoperative ICP. The patient's age and sex, the mode of injury, and the presence or absence of an associated cerebral contusion did not significantly influence reversibility of brain-stem dysfunction (Table 3). Evoked potential data, however, accurately forecast patient outcome, as they are a valuable indicant of the state of the brain stem.
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### TABLE 4

**Relationship of mortality to adverse neurological signs in series of acute subdural hematomas**

<table>
<thead>
<tr>
<th>Authors, Year</th>
<th>Total Cases</th>
<th>Percent Mortality</th>
<th>Functional Recovery (%)</th>
<th>Decerebrate</th>
<th>Bilaterally Nonreactive Pupils</th>
<th>Decerebrate &amp; Bilaterally Nonreactive Pupils</th>
</tr>
</thead>
<tbody>
<tr>
<td>McLaurin &amp; Tutor, 1961</td>
<td>74</td>
<td>73</td>
<td>16</td>
<td>6</td>
<td>67</td>
<td>70</td>
</tr>
<tr>
<td>Gutterman &amp; Shenkin, 1970</td>
<td>14</td>
<td>65</td>
<td>35</td>
<td>14</td>
<td>65</td>
<td>62</td>
</tr>
<tr>
<td>Jamieson &amp; Yelland, 1972</td>
<td>207</td>
<td>63</td>
<td>14</td>
<td>28</td>
<td>68</td>
<td>46</td>
</tr>
<tr>
<td>Richards &amp; Hoff, 1974</td>
<td>100</td>
<td>75</td>
<td>14</td>
<td>34</td>
<td>74</td>
<td>48</td>
</tr>
<tr>
<td>Cooper, et al., 1976</td>
<td>50</td>
<td>90</td>
<td>4</td>
<td>55</td>
<td>78</td>
<td>46</td>
</tr>
<tr>
<td>Bricolo &amp; Turazzi, 1980</td>
<td>94</td>
<td>67</td>
<td>26</td>
<td>59</td>
<td>76</td>
<td>48</td>
</tr>
<tr>
<td>Seelig, et al., 1981</td>
<td>82</td>
<td>57</td>
<td>34</td>
<td>28</td>
<td>68</td>
<td>25</td>
</tr>
</tbody>
</table>

* Decerebrate or bilaterally nonreactive pupils.

Gutterman and Shenkin \(^{18}\) reviewed the case histories of 29 patients with posttraumatic decerebration and intracranial hematoma. Seven of 10 patients with acute epidural hematomas, five of 14 patients with acute SDH, and two of five patients with intracerebral hematoma survived. Unfortunately, pupillary reactivity and ocular motility were not reported for these patients. Cooper, et al., \(^{10}\) recommended against hemi- or craniectomy for acute SDH if the patient demonstrated signs of brain-stem dysfunction consisting of decerebration and/or pupillary abnormalities, because the authors had only recorded a 4% functional recovery rate in their 50 cases. Bricolo, et al., \(^{8}\) reported an extensive series, 800 patients, with decerebration following head injury, in which 149 (19%) deeply comatose patients with absent oculomotor function were found. Five of the 149 (3%) survived, but the nature of their intracranial pathology and their pupillary reactivity to light were not provided. In a personal communication, Bricolo and Turazzi \(^{7}\) described an 80% mortality among 46 patients with acute SDH who were decerebrate and had bilaterally unreactive pupillary light reflexes (Table 4).

Although our results and those of others appear discouraging, careful evaluation indicates that signs of brain-stem dysfunction are more than just occasionally reversible. Approximately one-third of patients with acute SDH and decerebration survived in our series and in the series of McLaurin and Tutor, \(^{29}\) Gutterman and Shenkin, \(^{18}\) and Richards and Hoff, \(^{34}\) but only 10% survived in Jamieson and Yelland's series \(^{20}\) (Table 4). In these patients, the presence of an additional adverse clinical sign, bilaterally unreactive pupils, increased the mortality rate to 76% to 85%, depending on the series. In our series and that reported by Bricolo and Turazzi \(^{7}\) there was an 80% mortality rate, whereas Jamieson and Yelland \(^{20}\) recorded a 95% mortality rate for those patients who had bilaterally unreactive pupils and decerebration. The greater the percentage of patients with acute SDH in any series of patients who had decerebrate posturing and/or absent pupillary light reflexes, the worse the outcome, as documented in the two series by Ransohoff and associates \(^{10,33}\).

Our experience and that of others indicates that patients harboring an acute SDH in the presence of severe brain-stem dysfunction have a better chance of a functional survival than those with other traumatic lesions. Compared to an epidural hematoma, the SDH is usually a relatively slower growing lesion of venous origin, and simply allows the brain a longer period of time to compensate for the expanding mass before uncontrollable ICP and irreversible brain-stem damage occur. By the time an extradural hematoma causes the three adverse neurological signs, the process is more often than not irreversible. We believe there exists a critical volume and rate of growth of that volume of hematoma and/or edematous necrotic brain for each patient which is required to cause brain-stem compromise, and that any larger volume invariably leads to irreversible damage, flaccidity, and death.

In four of six patients who made a functional recovery, preoperative intraventricular hypertension was documented. Two of our patients did not have preoperative ventricular pressure measurements. Freedman \(^{19}\) also found preoperative intracranial hypertension by performing lumbar puncture in six patients with acute SDH, and four of these recovered. Hence, the pathogenesis of reversible brain-stem dysfunction must by in part based on conditions where dramatic and rapid improvement in cerebral perfusion pressure is possible by reduction of ICP prior to permanent brain-stem ischemia. The acute SDH is the lesion that has most commonly been associated with recovery of brain-stem function.

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Clearly, acute SDH and associated hemorrhagic intracerebral contusions should be promptly evacuated before flaccidity occurs, and postoperative ICP should be maintained at or below 20 mm Hg. Our results indicate that an average of no more than 24 hours should pass from the time of injury to surgical decompression, otherwise irreversible brain-stem damage can occur. Ten of the 11 patients in our overall series who were already flaccid on admission and had an acute SDH died in spite of prompt evacuation; the other flaccid patient remains severely disabled. Thus, therapy should be aimed at prompt intervention to prevent flaccidity and irreversible brain-stem compromise.

Evoked Potentials and Brain-Stem Dysfunction

We have shown that MEP data can accurately and reliably predict patient outcome from head injury in over 90% of cases. Furthermore, evoked potentials can help evaluate brain-stem and hemispheric function, as well as identify the location and extent of CNS lesions following severe head injury. Of particular relevance to this report is the clinical progress made recently with auditory and somatosensory brain-stem evoked potentials that allows noninvasive evaluation of brain-stem function. For example, coma caused by overdose of drugs (such as barbiturates, diazepam, glutethimide, amitryptiline) or metabolic conditions (such as diabetic ketoacidosis, anemia, hepatic failure, meningitis) can be distinguished by means of auditory brain-stem evoked potentials from coma caused by brain-stem damage. Starr and Achor reported that in some comatose patients with toxic or metabolic coma, the auditory brain-stem evoked potentials were normal when spontaneous respiration, cold caloric responses, and oculocephalic reflexes were absent or depressed. If the cortex is not irreversibly damaged, comatose patients with normal auditory brain-stem evoked potentials may make excellent recoveries. In a study of the relationship between brain-stem reflexes, levels of coma, and auditory brain-stem evoked potentials, Uziel and Benezech reported their results in 20 comatose patients, 75% of whom had head injury. They noted, as have others, that the first five wave-peaks of the AEP are thought to arise from a sequence of caudal-rostral-oriented generators in the auditory neural pathway. Three auditory brain-stem evoked potential wave-peaks are particularly robust; namely, Wave I, pontomedullary; Wave III, pontine; and Wave V, rostral pontine-collicular region. Flaccid patients generally had only Wave I present, while patients with bilaterally reactive pupillary dilatation had abnormalities of Wave I, II, or III. Decerebration was present in 11 patients. Eight of these patients had no abnormalities of their auditory brain-stem evoked potentials. The remaining three patients had only mild evoked potential abnormalities. Those patients in an apallic state also had no auditory brain-stem evoked potential changes, confirming the rostral location of lesions producing this clinical picture.

In the present report, the MEP data suggest that when auditory and somatosensory brain-stem evoked potentials are normal or only mildly abnormal in a patient with three adverse neurological signs, suggesting brain-stem dysfunction, one can confidently expect that the brain-stem dysfunction is reversible. On the other hand, severely abnormal brain-stem evoked potentials are harbingers of death or very poor recovery because they indicate irreversible brain-stem compromise.

As is illustrated by the clinical and electrophysiological data presented here, signs of brain-stem dysfunction in head-injured patients, especially those with acute SDH, do not inevitably forecast a grave outcome, as brain-stem compromise can sometimes be reversed. Prompt surgical intervention along with prudent control of postoperative ICP were of major import in preventing permanent brain-stem damage and death in a significant number of our patients with acute SDH.

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