Somatosensory Evoked Potentials and Intracranial Pressure in Severe Head Injury

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Abstract: The purpose of this study was to explore the relationship between neurologic function, using a quantitative measurement of continuous somatosensory evoked potentials (SSEPs), and intracranial pressure (ICP) following traumatic brain injury. During a 6 year period, severely head-injured patients with a Glascow Coma Scale ≤ 8 who were not moribund were monitored with SSEPs and ICP measurements. SSEPs from each hemisphere and ICP were recorded hourly for each patient. Neurologic outcomes were scored using the Glasgow Outcome Scale at three months post injury. Although initial SSEP amplitude did not correlate well with outcome, final SSEP summed peak to peak amplitude from both hemispheres ($p = .0001$), the best hemisphere ($p = .0004$), and the worst hemisphere ($p = .0001$) correlated well with the Glasgow Outcome Scale groups. Of a total of 72 patients, 40 had deteriorating SSEPs and 32 had stable or improving SSEPs. Peak ICP values were not statistically different in these groups ($p = .6$). Among patients with deteriorating SSEPs, 52.5% lost the greatest proportion of hemispheric electrical activity prior to ICP elevation. In the remaining patients, the percent reduction of SSEP activity after peak ICP levels was not statistically different from the percent reduction in SSEP activity prior to the peak ICP levels ($p = .9$). This data suggests that in a select group of patients with severe head injury, ICP does not cause SSEP deterioration, but rather is the consequence of deterioration of brain function.

Résumé: Potentiels évoqués somesthésiques et pression intracrânienne dans les traumatismes crâniens sévères. Le but de cette étude était d'explorer, au moyen d'une mesure quantitative des potentiels évoqués somesthésiques (PESs) continus, la relation entre la fonction neurologique et la pression intracrânienne suite à un traumatisme crânien. Pendant une période de 6 ans, les traumatisés crâniens ayant un score ≤ 8 à l'échelle de coma Glasgow et qui n'étaient pas moribonds, ont été surveillés par enregistrement des PESs et mesure de la pression intracrânienne (PIC). Les PESs de chaque hémisphère et la PIC étaient enregistrés à chaque heure chez tous les patients. L'issue neurologique étaient évaluée selon l'échelle d'issue du coma Galsgow (EG) trois mois après le traumatisme. Bien que la corrélation de l'amplitude initiale des PESs avec l'issue finale n'était pas bonne, l'évaluation finale de la somme de l'amplitude des PESs d'un pic à l'autre des deux hémisphères ($p = .0001$), du meilleur hémisphère ($p = .0004$) et du pire hémisphère ($p = .0001$) avait une bonne corrélation avec les groupes EG. Sur un total de 72 patients, 40 avaient des PESs en détérioration et 32 avaient des PESs stables ou qui s'amélioraient. Les valeurs maximales de la PIC n'étaient pas statistiquement différentes parmi ces groupes ($p = .6$). Parmi les patients qui avaient des PESs qui se détérioraient, 52.5% avaient perdu la plus grande partie de l'activité électrique hémisphérique avant la hausse de la PIC. Chez les autres patients, le pourcentage de réduction de l'activité des PESs après l'obtention des niveaux maximums de la PIC n'était pas statistiquement différent du pourcentage de réduction de l'activité des PESs avant les niveaux maximums ($p = .9$). Ces données suggèrent que, dan un groupe choisi de patients qui ont subi un traumatisme crânien sévère, la PIC ne cause pas de détérioration des PESs mais qu'elle est la conséquence de la détérioration de la fonction cérébrale.

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Although previous work has clearly shown an association between peak levels of intracranial pressure (ICP) and adverse outcome,[1-3] a causal relationship between raised ICP and neurologic deterioration has never been unequivocally demonstrated. Non-randomized clinical trials of severely head injured patients have claimed that aggressive treatment of elevated ICP reduces the overall mortality and improves functional outcome several months post-injury.[4,5] These trials implied that levels of ICP > 15 - 20 mm Hg have adverse effects on patient outcomes. Unfortunately, because the trials did not have a true control group of patients in whom ICP was not treated aggressively, one cannot conclude that failure to control ICP at these levels

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results in poor outcome. Nevertheless, these studies have contributed to the current emphasis on ICP treatment in the management of head injury.

One of the difficulties in determining the relationship between neurologic function and ICP has been the absence of an objective, sensitive, and quantitative measure of brain function in comatose ventilated patients. Evoked potential measurement has been shown to be remarkably accurate in the prediction of outcome, both alone and when combined with clinical data.[6-13] Furthermore, it has been demonstrated that somatosensory evoked potentials (SSEPs) have a higher individual correlation with hemispheric function and neurologic outcome than other modes of evoked potential measurements such as visual evoked or auditory brainstem responses.[14-18] Presently, SSEPs can be recorded and analyzed in real time at the patient's bedside and compared to other physiological parameters. These characteristics make SSEPs very useful as a means of monitoring and as a predictive tool in the acute head injury period, particularly in those patients who are pharmacologically paralyzed. If SSEP traces can be objectively quantified during the ICP monitoring period, an accurate determination of the effect of ICP on neurologic function in the acute post-injury period can be obtained.

This paper will compare the changes in neurologic function, as measured by a quantitative measure of SSEP activity (peak-peak amplitude), to peak levels of ICP. The hypothesis of this work is that SSEP deterioration occurs prior to the period of maximal ICP in patients with severe head injury.

METHODS

SSEP Monitoring

From July 1987 to October 1992, selected patients with severe closed head injury (Glasgow Coma Scale (GCS) $\leq$ 8) admitted to the neurosurgical ICU were monitored with hourly SSEPs. Patients who were moribund with bilateral fixed and dilated pupils were excluded. SSEP monitoring was generally begun shortly after admission to the ICU and repeat clinical examination of the patient if the GCS remained below 8. Monitoring was discontinued after 5-6 days if ICP was stable and showed no increasing trends, later if ICP remained elevated, and sooner if the patient had evidence of electrical brain and brainstem silence with a consistent clinical examination (e.g., the development of an isoelectric EEG or of a P_{15} wave only on SSEP traces with the evolution of bilaterally fixed and dilated pupils).

The international 10 - 20 system was used to position the disk electrodes on the scalp in order to assure standard electrical recordings within and between subjects and to allow better comparison with other investigators. The median nerves were stimulated at the wrist with a voltage stimulus delivered by a pair of subdermal needle electrodes. One electrode was placed overlying the distal wrist crease and the other 1 cm proximal to it. The right and left median nerves were stimulated separately. Because all patients were pharmacologically paralyzed, it was not possible to use the twitch of the abductor pollicis brevis to gauge the adequacy of the stimulus. The presence of the P_{15} wave was used for this purpose. SSEPs were recorded from the contralateral C_3' and C_4' (central) electrodes referenced to linked ears. Each scalp electrode's impedance was carefully checked twice daily and maintained below 2 kilo-ohms. Any deviations were recorded in a log book kept at the patient's bedside.

The SSEP waveforms were generated from an average of 250 stimulus repetitions at a stimulus frequency of 2.1 Hz. High and low bandpass settings were 1 KHz and 3 Hz, respectively. The duration of the response recorded for each stimulus was 250 milliseconds. Each set of SSEP tests consisted of two repetitions recorded sequentially from both median nerves with a 15 second delay between repetitions. Approximately 25-30 waveforms were generated from each hemisphere daily. All information was stored on an IBM PC located at the patient's bedside.

After the monitoring period, SSEP traces for each patient were screened off-line to eliminate SSEP traces that contained artifact – typically due to muscle or movement artifact or from an increase in scalp electrode impedance resulting from drying of the electrode conducting gel. This was done by re-examining the clinical chart and log book for each patient and corroborating these periods with those of SSEP artifact. All traces were screened without any knowledge of patient outcome or of ICP trends during the period of monitoring. After screening, the SSEP traces were quantitatively assessed by a computer program by identifying peaks and summing peak to peak amplitude[19] in microvolts for a 250 millisecond waveform (starting at the P_{15} wave) for each hemisphere and for both hemispheres combined. The peak detection program has been described in detail in previous work.[20] In a patient with full SSEP activity, peaks that are usually identified by the program are the P_{15}, N_{20}, P_{29}, N_{35}, P_{50}, N_{70}, P_{100}, N_{150} and P_{175}. The latency of the latter peaks is approximate due to within and between patient variability.

ICP Recording

ICP was monitored with a subarachnoid bolt or an intraventricular catheter. These were placed in the operating room. ICP recording was begun on admission to the ICU. The nurse's manual "end-hour" recording was used to acquire ICP data.[21,22] The 24 hour data was reviewed to obtain a daily ICP maximum value for each patient. This data was stored in a head injury database file. From this information, the peak ICP level and the time of its occurrence were established for each patient during the monitoring period.

Patient Treatment Regimen

Patients were treated with a standard protocol including intravenous muscle relaxants, mechanical ventilation, osmotic diuresis and cerebrospinal fluid (CSF) drainage for elevations in ICP > 20 - 25 mm Hg. The pharmacological paralysis aided in preventing harmful elevations in ICP from occurring and produced cleaner SSEP traces, as movement has been shown to attenuate SSEP waveforms.[23]

Statistics

Data was analyzed in discrete points in time. Initial SSEPs were those obtained the first hour after monitoring was begun. Similarly, final SSEPs represent the last hour's SSEPs occurring at the termination of electrophysiological monitoring. SSEPs from the period prior to the time of peak ICP were obtained to determine the impact of ICP on SSEP activity.

The changes in the total amplitude of the SSEP from both hemispheres from the initiation of monitoring (first baseline) to the time of peak ICP, and from the time of peak ICP (second baseline) to the end of monitoring were calculated using the

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percentage change from baseline. Glasgow Outcome Scale (GOS)[24] scores at three months were determined for each patient and compared to SSEP activity. ANOVA and student t tests were used for statistical comparisons of SSEP data using the statistical program SAS (SAS Insitute, Box 8000, Cary, N.C., U.S.A. 27511-8000). Frequency data were analyzed using chi-square testing for a proportion. Data are reported as the Mean ± SEM.

Results

Clinical Features

A total of 72 patients between ages 15 - 77 years (37.4 ± 3.96 years) were monitored hourly with SSEPs and included in the analysis. Approximately 2/3 of the patients were referred from other hospitals and 1/3 received directly from the accident site. Of the 72 patients, 33 were diagnosed with a severe closed head injury without an operable mass lesion, 35 underwent craniotomy for evacuation of an operable mass lesion, and 4 had surgical debridement or elevation of skull fractures (Table 1). In 8 patients the ICP remained below 25 mm Hg at all times. In the remaining 64 patients, 33 had at least one ICP measurement that rose to levels between 25 - 40 mm Hg, and 31 had at least one ICP measurement that rose to levels above 40 mm Hg. Of the 31 patients who died, only 10 had ICP ≥ 40 mm Hg.

Clinical Outcome and SSEPs

At 3 months post injury, 17 patients were classified as having a good or moderate outcome (GM), 22 were severely disabled (S), and 33 were vegetative (n = 2) or dead (n = 31) (VD). The mean age of the patients was significantly different in the various outcome groups (GM = 31.53 ± 2.25 yrs, S = 32.5 ± 3.14 yrs, VD = 43.79 ± 3.23 yrs; p = .0094, F = 4.99, ANOVA).

Patients were monitored with SSEP recordings for an average of 5.86 days (range 1-13 days) with no significant difference in duration of monitoring between outcome groups (p = .13, Table 2). Furthermore, the mean start time of monitoring was 23.77 hours post-injury, again with no significant difference between outcome groups (p = .19, Table 2). Initial summed SSEP peak-peak amplitude responses from the best, worst, and both hemispheres combined were not statistically significant between outcome groups (p = .19, p = .11, p = .12, respectively). However, final SSEP responses for the best, worst and both hemispheres combined did show a significant difference between outcome groups (p = .0004, p = .0001, p = .0001, respectively, Figure 1a, b, c). For both hemispheres combined, the mean percent change in SSEP amplitude from initial baseline values was –9.9% in the GM group (p = .56), –20% in the S group (p = .13) and –53% in the VD group (p = .002).

ICP and SSEP Deterioration

ICP monitoring was carried out using a ventriculostomy in 58 patients and a subarachnoid bolt in 14. In the course of observing these patients, SSEPs appeared to deteriorate despite maintaining ICP below acceptable levels of 20 - 25 mm Hg. Furthermore, elevated levels of ICP sustained beyond 5 - 10 minutes were acted upon immediately with mild hyperventilation, osmotic diuresis and/or ventricular drainage. As such, prolonged periods of elevated ICP were very uncommon.

Table 1. Etiology of Head Injury from 1987-1992 in patients with SSEP monitoring.

Etiology	N
CHI	33
SDH	19
EDH	6
ICH	5
SDH/ICH	5
Skull #	4

CHI = Severe Closed Head Injury without an operable mass lesion. SDH = Subdural Hematoma. EDH = Extradural Hematoma. ICH = Operable Intracranial Hematoma. SDH/ICH = Subdural Hematoma and Operable Intracranial Hematoma. Skull # = Skull Fracture. All patients underwent surgery. Patients with SDH, EDH, ICH, SDH/ICH and Skull # underwent craniotomy as well as placement of ICP monitors, while patients with CHI only had placement of ICP monitors. There were 58 ventriculostomies and 14 subarachnoid bolts placed in total.

Table 2. SSEP Monitoring Characteristics in GOS Groups.

	Glasgow Outcome Scale Groups		
	GM	**S**	**VD**
N	17	22	33
Start Time *	32.70 ± 24.96	21.00 ± 10.85	21.01 ± 26.96
Range	6.50 - 89.00	7.00 - 21.00	5.50 - 152.00
Length of Monitoring **	6.24 ± 1.52	6.36 ± 1.87	5.33 ± 2.34
Range	4 - 10	4 - 11	1 - 13

Start time (hours post-injury). Length of Monitoring (days). Data reported as mean ± std dev. There is no statistical difference between start time* (F = 1.72, p = .19, ANOVA) or length of monitoring** (F = 2.07, p = .13, ANOVA) in the different outcome groups.

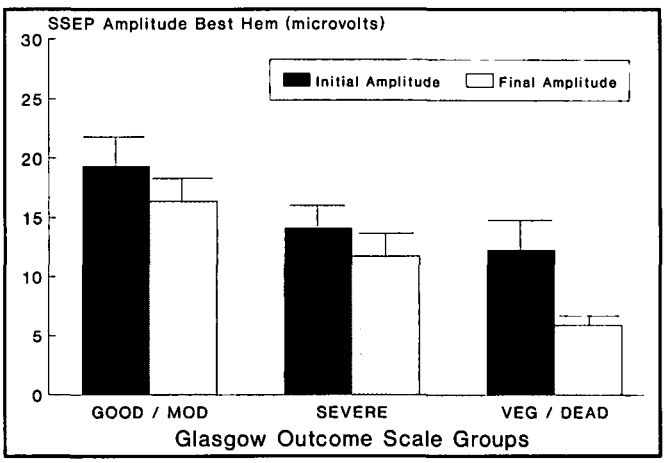

Figure 1a. *Summed peak to peak amplitude response in microvolts (best hemisphere) for various outcome groups (Means + SEM). Initial response is not significantly different (F = 1.71, p = .19 , ANOVA) whereas the final response is significantly different among outcome groups (F = 8.81, p = .0004, ANOVA).*

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Because elevated ICP is thought to result in global cerebral dysfunction, SSEP peak-peak amplitude for the right and left hemispheres were added together and used to determine the ICP effect on global cerebral electrophysiology. Based on a mean change in SSEP amplitude (for both hemispheres combined) from the beginning to the end of the monitoring period for the different outcome groups, SSEP deterioration was defined as a total SSEP amplitude decrease of at least 20% during the period of monitoring or the presence of the P_{15} wave only at the start of monitoring. The 20% measure for deterioration was used as a clinically relevant approximation, as this was the level of deterioration that separated good and moderate patients (GM) from those with a worse outcome (S or VD). According to these criteria, there were 40 patients with absent (only P_{15} waves, n = 3) or deteriorating SSEPs (SSEPs deteriorating by 20% or more, n = 37). These included 6 patients who had a good/moderate outcome, 10 who were severely disabled and 24 who were vegetative or dead at 3 months. There were 32 patients with stable (SSEPs deteriorating by less than 20%, n = 8) or improving SSEPs (SSEPs improving from initial baseline values, n = 24). In this group, there were 11 patients with a good/moderate outcome, 12 who were severely disabled and 9 who were vegetative or dead at 3 months. Therefore, only 28% of patients with stable or improving SSEPs were vegetative or dead at 3 months as compared to 60% of patients with deteriorating SSEPs (p = .02, Chi-Square = 7.7). The peak ICP values between the deteriorating and stable/improving SSEP groups were not statistically different (46.6 mm Hg vs. 43.7 mm Hg respectively, p = .6).

SSEP activity tended to decay in a gradual fashion that did not appear to be related to ICP (Figures 2, 3). Among the 40 patients with deteriorating SSEPs, 21 patients lost the greatest proportion of SSEP activity in the period preceding the day of occurrence of maximum ICP. In the remaining 19 patients with

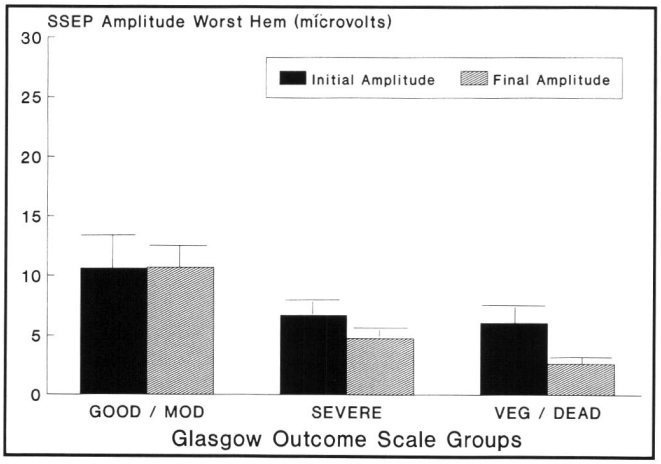

Figure 1b. *Summed peak to peak amplitude response in microvolts (worst hemisphere) for various outcome groups (Means + SEM). Initial response is not significantly different (F = 2.30, p = .11, ANOVA) whereas final response is significantly different among outcome groups (F = 16.47, p = .0001, ANOVA).*

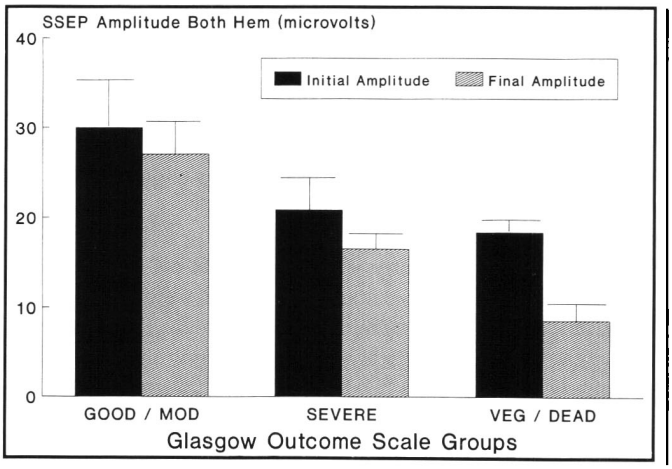

Figure 1c. *Summed peak to peak amplitude response (both hemispheres combined) in microvolts for SSEP traces at the initiation and at the end of monitoring for the various outcome groups (Means + SEM). Initial amplitude is not significantly different in these groups (F = 2.17, p = .12, ANOVA). Final amplitude response is significantly different in these groups (F = 12.52, p = .0001, ANOVA).*

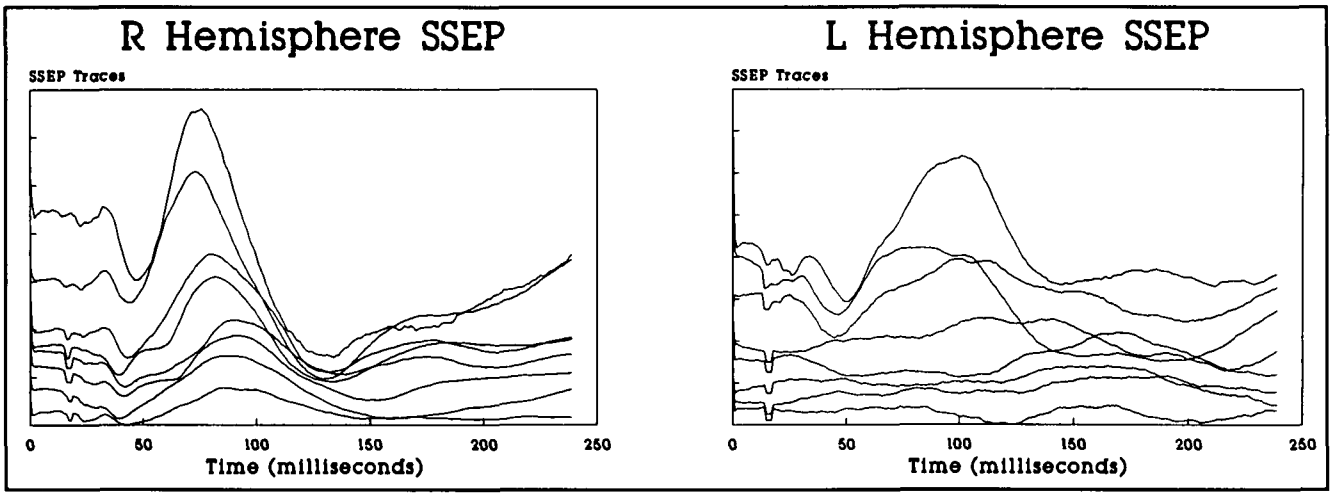

Figure 2. *R and L Hemisphere SSEP traces in a patient following evacuation of an acute L SDH and placement of a L ventriculostomy. Monitoring was begun upon admission to the ICU at 18 hrs post-injury. SSEP traces from top to bottom are those representative of those occurring at 18, 27, 41, 51, 65, 75, 91.5, and 100 hours post-injury.*

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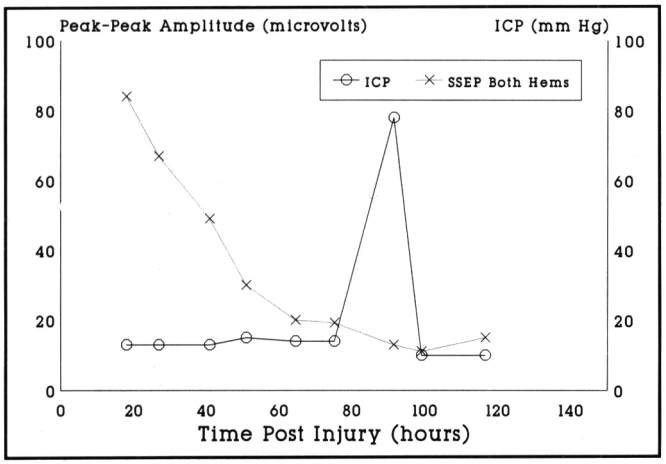

Figure 3. *ICP and summed SSEP peak to peak amplitude responses for both hemispheres in the patient represented in Figure 2. SSEP responses became attenuated much sooner than did ICP become unmanageable. This was typical of patients dying with uncontrollable ICP.*

deteriorating SSEPs, the mean change in SSEP activity before and after the occurrence of maximum ICP was not statistically significant (p = .9). Furthermore, in 9 of 10 patients dying with an ICP > 40 mm Hg, the evoked potentials became severely attenuated or absent prior to the occurrence of maximum ICP.

Discussion

Quantitative SSEPs

Early reports on severely head-injured adult patients provided a snapshot of one or two SSEP traces at some point in time during the post-injury period – usually several days after a patient's admission to the ICU and not consistent from patient to patient.[25] Human and experimental evidence, however, indicates that axonal damage following severe head injury is not a static process which is fixed within milliseconds of impact, but rather an evolving process.[26-33] As a result, one or two isolated measurements of an SSEP during the acute phase of brain injury may not be reflective of evolving axonal disruption. Some studies that have looked at frequently monitored SSEPs have focussed only on the short latency waveforms (< 50 milliseconds) and have found that initial SSEPs are highly correlated with outcome;[18,34] however, long latency SSEPs (e.g., the N_{70} peak) are a sensitive measure of cortical function and are useful to quantify functional and structural dysfunction.[25,35-39] In brain impairment, a stepwise loss of these peaks is well known despite the preserved N_{20} response.[25] Furthermore, the loss of long latency peaks does not occur in isolation, but has been reported to be accompanied by a diminution of amplitude and degradation of morphology of peaks occurring at earlier latencies.[35] Thus, the long-latency SSEP provides important clinical information on cortical integrity which is complementary to the short-latency SSEP responses.

SSEPs have generally been graded by observers by central conduction time or in a semi-qualitative fashion based on patterns of wave latencies and morphology, the latter increasing observer bias and contributing to possible inaccuracies. Quantitative measures for SSEPs do exist and have the advantage of not being influenced by observer knowledge of the patient's condition.

To circumvent some of the limitations of previous SSEP studies, a quantitative measure (peak-peak amplitude) for the analysis and interpretation of short- and long-latency SSEPs in a continuous fashion from the time of admission to the ICU was used in this report.

SSEPs and Outcome

Monitoring of SSEPs from the time of admission to the ICU when a patient is comatose (GCS $\leq$ 8) allows one to carefully monitor the critical cerebral hemispheric changes that occur in patients with severe head injury. Although some authors have successfully shown that the early SSEPs are highly predictive for outcome after cerebral trauma,[36] it appears from this analysis, that the outcome following head injury cannot be accurately predicted from the initial SSEP responses. This is substantiated by others who have found that if the cortical responses are near normal in the first 24 hours after trauma, then SSEPs should be repeated later in the monitoring period before an accurate prediction can be given.[40] In this study, the final peak-peak SSEP amplitude was a more reliable predictor of the patient's neurologic outcome than the initial peak-peak SSEP amplitude. The initial SSEP peak-peak amplitude measurement for both hemispheres combined was 30.00 $\pm$ 4.93 microvolts for the GM group, 20.85 $\pm$ 3.38 microvolts for the S group, and 18.36 $\pm$ 3.49 microvolts for the VD group (p = .12, F = 2.17, ANOVA) whereas the final SSEP peak-peak amplitude response was 27.05 $\pm$ 3.44 microvolts for the GM, 16.50 $\pm$ 2.76 microvolts for the S, and 8.58 $\pm$ 1.93 microvolts for the VD group (p = .0001, F = 12.52, ANOVA, See Figure 1c). This inability to differentiate between GOS groups initially was also evident when analyzing either the best or worst hemisphere responses (Figure 1a, b). Analysis of the last waveforms, usually occurring on the fifth or sixth days after head injury, revealed that the quantitative peak-peak amplitude measurements were significantly different among outcome groups (Figure 1a, b). Preliminary analyses of peak-peak amplitude responses over time in these patients indicate that the ability to accurately predict the outcome based on SSEP responses occurs between 48 - 72 hours after trauma.

Many patients with a poor prognosis have a marked deterioration in SSEPs over time, even though initially they may have similar or identical SSEP responses as those patients who ultimately do well. The mean percent change of peak-peak amplitude for both hemispheres combined was –9.9% for the GM group, –20% for the S group, and –53% for the VD group. As a result, careful frequent monitoring and quantitative representation of SSEPs allows one to identify time dependent changes in global cerebral function (peak-peak amplitude information for both hemispheres combined) and to correlate these changes with other physiological parameters believed to lead to poor outcome.

Deterioration in SSEPs

As previously mentioned, neurologic damage after head injury may be a dynamic process that occurs after the initial insult. Impact damage to the cerebral parenchyma causes a change in the physical properties of the brain with the development of pathological markers of diffuse axonal injury. In the

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early stages after nonpenetrating head injury, stains for nerve processes reveal axonal retraction balls brought about by extrusion of axoplasm from anterograde axoplasmic flow.[41] These axonal retraction balls develop 12-18 hours after the occurrence of head injury.[32] Over time, a cellular reaction develops around these nerve fibers and forms microglial clusters.[27,32,41,42] These traumatically-induced axonal changes, which progress from the time of impact, and appear to be independent of systemic or cerebral physiology,[30,31] may actually explain why the SSEP responses also generally decay over time, even in patients with a favorable outcome. We speculate that SSEP deterioration may be the electrophysiological correlate of the observed axonal pathology.

Impact of 'Secondary Insults' on SSEP Deterioration

Deterioration in SSEP responses in patients with severe head injury has previously been documented by Newlon et al.[7] who noted a poor prognosis in patients whose multimodality evoked potentials deteriorated from one examination to another. The deterioration was attributed to the effects of 'secondary insults'[43] such as raised ICP. If ICP were related to neurologic deterioration, a clear association between ICP trends and SSEP trends would then be expected to occur – raised ICP would be expected to precede SSEP deterioration. We did not observe this in our study population. This lack of association between ICP and electrophysiological changes has also been documented by Garcia-Larrea et al.[44] who noted that acute rises in ICP > 40 mm Hg were not always followed by brainstem auditory evoked potential changes.

SSEP deterioration may be due to other 'secondary insults' not analyzed in this study or due to primary impact injury processes. The effect of intracranial 'secondary insults' such as cerebral ischemia and vasospasm need to be studied to identify their impact on electrophysiological activity in the acute phase post traumatic brain injury. Furthermore, extracranial 'secondary insults' such as fluid and electrolyte imbalance and episodes of hypotension also need to be considered with respect to their effect on SSEP activity in the brain traumatized individual.

When all other 'secondary insults' have been excluded as the major determinants of SSEP dysfunction, then the primary impact injury with development of the axonal changes as described above may be considered as the cause of neurologic deterioration. Presently, this concept remains speculative; however, present studies are ongoing to attempt to identify which physiological parameters may be responsible for the apparent deterioration in electrophysiology which occurs from the time of admission to the ICU.

ICP and Neurologic Deterioration

Elevated ICP is a common occurrence in patients with severe closed head injury. Miller et al.[1] found that 52% of patients who had intracranial masses evacuated had ICP > 20 mm Hg and that in patients without mass lesions, 33% had ICP > 20 mm Hg which required aggressive therapy at some point during the hospital admission. Marmarou et al.[3] found a substantially higher proportion (72%) of raised ICP in severely head injured patients, but patients with penetrating gunshot wounds were included in the analysis. More recently, Unterberg et al.[45] showed that ICP was elevated in 92.5% of patients after significant head trauma; however, patients who were too elderly or who had evidence of devastating brain injury were not monitored.

The present analysis revealed that ICP was elevated > 25 mm Hg in 64 of 72 patients (88.9%). This, one may argue, explains why most of our patients (76.4%) at three months post injury were severely disabled, vegetative or dead. This is consistent with other previously reported studies in head injury showing an association between raised ICP after head injury and poor outcome several months after injury.[1-3] However, while these studies show an association between raised ICP and neurologic outcome, none demonstrate causation, nor do they address the temporal relationship between ICP and neurologic decline in head injury.

In this study, it was not possible to demonstrate any significant differences in peak ICP values in patients with deteriorating or absent SSEPs and those with stable or improving cerebral electrical function. We chose to look at peak levels of ICP because of the previously demonstrated relationship between this parameter and outcome.[1] Unlike other analyses, however, the population was based on a slightly different patient pool. Previous investigators focussed on consecutively admitted, unselected head injured patients who were not obeying commands or worse[1,2,4,25,46] making their inclusion criteria substantially more liberal than the entry criteria for this series. Patients with milder head injury (GCS 8-10) would not have been monitored as aggressively with ICP and evoked potential recordings in our ICU unless they required aggressive ICP management. At the opposite end of the injury spectrum, patients who were moribund and not expected to survive beyond a few hours post injury were usually not studied. In our experience, in the few patients with unrecoverable brain injury who have been monitored, SSEP activity is absent from the initiation of monitoring to the onset of uncontrollable ICP. In this analysis, of the 40 patients with deteriorating SSEPs, the majority (52.5%) lost SSEP activity before reaching peak levels of ICP. In those patients whose SSEPs continued to deteriorate following peak ICP levels, there was no evidence to suggest that SSEP change following peak ICP was greater or occurred more rapidly than that occurring prior to peak ICP levels. This may indicate that in many cases ICP is simply a reflection of the volume of injured brain, or may occur independently of neurologic deterioration, possibly related to vascular pathology.

The fact that there is no universally accepted level of ICP for the treatment of raised ICP suggests in part that its treatment is based on empirically derived levels such as 20 - 25 mm Hg.[3,47] This is reinforced by studies showing that lowering ICP has a beneficial effect on prognosis.[4,5] This is contradicted by other studies that have shown that reasonable outcomes can be achieved in severely head injured patients when ICP is not monitored[48] suggesting that ICP may not be a critical determinant of outcome.

Impact on Clinical Management

The use of SSEPs, as well their quantitative interpretation, has made it clear that the treatment of ICP in the severely head injured patient must be individualized for each patient. Elevations in ICP may occur long after irreversible structural damage to the brain has occurred as indicated by severely attenuated SSEP responses (Figure 3). In the majority of cases where disappearance of SSEP activity occurs prior to large refractory elevations in ICP, it has been possible to avoid heroic measures such as craniectomy or barbiturate coma. In 2 cases where ICP became medically uncontrollable in the face of good SSEP responses, decompressive craniectomies were performed. At three months post-injury one patient was moderately disabled

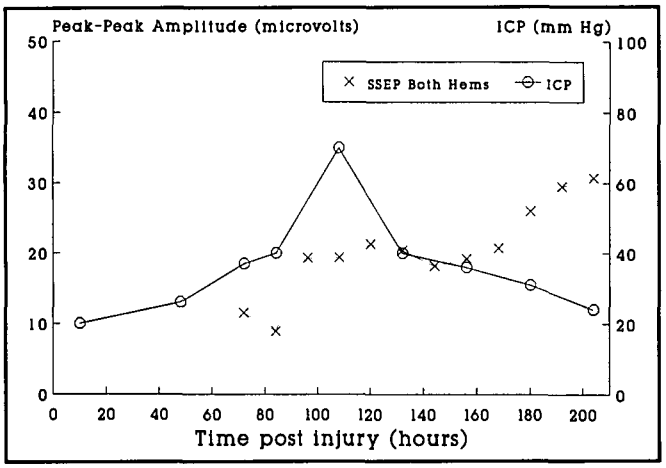

Figure 4. ICP and summed SSEP peak to peak amplitude responses for both hemispheres in a patient with an acute L extradural hematoma. The patient underwent a craniotomy for evacuation of the clot and placement of an intraventricular catheter. ICP monitoring was begun shortly after admission to the intensive care unit. SSEP monitoring was begun 70 hours post-injury when the patient's neurologic status deteriorated to a GCS < 8. ICP began to rise to uncontrollable levels. Because the patient had evidence of adequate or improving SSEP responses, bilateral decompressive craniectomies were performed. SSEP responses continued to improve post-operatively despite levels of ICP greater than 25 mm Hg.

and the second had a good outcome. In this latter case, intermittent elevations in ICP persisted but occurred simultaneously with improvement in SSEP activity (Figure 4). This suggests that SSEP changes may be independent of ICP until the point of uncal herniation, and that arbitrary levels of ICP such as 20 or 25 mm Hg may not adequately predict impending herniation or the need for urgent surgery. This is substantiated by studies showing that even if ICP remains low in patients with intracranial hematomas, management decisions based on ICP monitoring alone fail to predict the need for surgery in a significant proportion of patients.[49]

The results presented in this paper examine the relationship between ICP measurements and hourly SSEP activity, quantified by summing peak to peak amplitude over the duration of a 250 millisecond SSEP trace. Initial SSEP responses are not different between GOS groups, whereas final responses are significantly different. Patients with a poor outcome have greater deterioration in SSEP activity over the monitoring period (–9.9% in the GM as opposed to –20% in the S and –53% in the VD group). Our data show that peak levels of ICP are not causally related with progressive deterioration in SSEP activity.

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