Pressure-volume index in head injury

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The authors studied intracranial pressure (ICP) and intracranial compliance as defined by the pressure-volume index (PVI) in 34 severely head-injured patients with a Glasgow Coma Scale score of 8 or less. The objective of the research was to determine if there was a correlation between the pressure-volume status and subsequent increase in ICP. The PVI and ICP measurements were obtained serially, and the temporal course of the pressure-volume status and ICP was determined during the 5-day period following injury. Aggressiveness of ICP was quantified by a therapy intensity level scale. A clear relationship between the PVI measured soon after injury and subsequent development of ICP emerged. Following mechanical trauma the PVI is reduced, and the degree of reduction and extent of biomechanical recovery are closely related to outcome and development of raised ICP.

KEY WORDS • pressure-volume index • head injury • intracranial pressure • therapy intensity level

Since the introduction of intracranial pressure (ICP) measurement by Guillaume and Janny* and Lundberg,13 ICP monitoring has been a useful adjunct in the management of patients with brain injury. Clinical studies reported during the last decade have described the close correlation between intracranial hypertension and outcome.2,7,9,16,20,22 Other reports have emphasized the neurological deterioration resulting from secondary insult to the brain induced by depletion of volume-buffering capacity and development of high ICP. As a result of these studies, early aggressive treatment has been advocated by the majority of investigators.2,22 The importance of volume-pressure relationships in understanding the pathophysiology of raised ICP has been clearly demonstrated in the laboratory,10,12,26 and a number of clinical studies have focused on the correlation of brain compliance and ICP following traumatic brain injury.4,5,8,10,16,24,27,28 The alteration of biomechanical indices with trauma as well as the relationship of pressure-volume parameters to intracranial hypertension and eventual outcome have been explored in only a limited number of studies.8,24

This study describes the temporal course of the pressure-volume index (PVI) and ICP in adult patients with severe head injury. The objective was to characterize the biomechanical profile of the head-injured patient immediately upon stabilization in the neuroscience intensive care unit (ICU) and to follow the course of ICP and PVI during the 5 days postinjury. Having established this relationship, the concept that early detection of reduced neuraxis compliance can identify those patients at risk for development of raised ICP could be tested.

Clinical Material and Methods

Patient Assessment

This study population consisted of 34 severely head-injured patients (26 males and eight females) who were admitted to the ICU between 6 and 18 hours after injury (Table 1). The age range for the population was 14 to 81 years (mean ± standard deviation 33.4 ± 17.6 years, median 24 years). The mean Glasgow Coma Scale29 (GSC) score was 6.48 ± 2.64. In total, 576 manipulations during 199 studies (mean 3.46 per study) were performed. The ICP was measured by direct ventricular cannulation, and ICP strain-gauge pressures were referenced to the level of the external auditory meatus. Blood pressure was measured by cannulation of one of the radial arteries or the pedal artery, and
Pressure-volume index in head injury

TABLE 1
Clinical summary in 34 severely head-injured patients*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>time of NICU admission following injury (mean ± SD)</td>
<td>12 ± 6 hrs</td>
</tr>
<tr>
<td>sex (M:F)</td>
<td>26:8</td>
</tr>
<tr>
<td>age (yrs)</td>
<td>33.4 ± 17.6</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>24</td>
</tr>
<tr>
<td>range</td>
<td>14-81</td>
</tr>
<tr>
<td>Glasgow Coma Scale score</td>
<td>≤ 8</td>
</tr>
<tr>
<td>primary diagnosis</td>
<td></td>
</tr>
<tr>
<td>closed head injury</td>
<td>13</td>
</tr>
<tr>
<td>subdural hematoma</td>
<td>8</td>
</tr>
<tr>
<td>intracerebral hematoma</td>
<td>6</td>
</tr>
<tr>
<td>epidural hematoma</td>
<td>5</td>
</tr>
<tr>
<td>intraventricular hemorrhage</td>
<td>2</td>
</tr>
</tbody>
</table>

* NICU = neurosurgical intensive care unit; SD = standard deviation.

The ICP values reported in this study were obtained from the bedside ICU monitor and represent the highest mean ICP recorded by the nursing personnel during a 1-hour period. The average ICP over a 4-hour “block” was defined as the average of the four observations. The 4-hour “block” average and standard deviation for each individual patient were obtained and then averaged across patients to describe the temporal ICP course among patient groups.

The PVI determinations were performed according to standard protocol and standard calculation from the response to both the withdrawal and the addition of CSF volume. Upon stabilization of ICP, the PVI based on withdrawal of volume (PVIw) was determined by removing 2 cc or less of CSF and observing the pressure response. For calculation of PVIw the baseline ICP prior to withdrawal (Po), the minimum pressure following withdrawal (Pm), and the volume (V) withdrawn were inserted in the equation PVIw = V/Log Po/Pm. The calculation of PVI from addition of volume (PVIA) was determined by replacing Pm by the peak pressure measured immediately following injection of volume (Pp) into the same equation. Care was taken to avoid waveform errors by selecting equivalent pressure points during the respiratory and cardiac fluctuations of ICP before and after bolus manipulation. Induction of pressure waves by volume perturbations was minimized by maintaining volume injection below 3 cc and warming the 0.9% sodium chloride to 37° prior to insertion. The actual volume injected was based upon the calculated PVIw from bolus withdrawal and the calculation of the injection volume limit (VIIM) necessary to insure an adequate perfusion pressure of 55 mm Hg. The maximum volume limit was computed from the equation VIIM = (PVIw) × Log (BP - 55)/Po where PVIw is the PVI calculated from the withdrawal sequence, BP the mean arterial pressure, and Po the mean baseline ICP. For example, a normal PVI of 25 ml, a normal ICP of 10 mm Hg, and an arterial pressure of 100 mm Hg result in a VIIM of 16.3 cc. A “tight” brain of 12.5 ml and an elevated ICP of 20 mm Hg restricted the possible injected volume to 4.4 cc. In all cases of tight brain (PVI < 15 ml), withdrawal PVI was determined first, and no more than 1 cc was added for calculation of PVIA.

Assessment of ICP Therapy Intensity Level

One major difficulty in correlating ICP profiles among patients is the complexity in ICP analysis introduced by therapy. Heretofore, the effects of therapy upon the ICP analysis have not been addressed. The need for such quantification becomes obvious when ICP control is achieved in one patient using mild hyperventilation and in another by means of barbiturates. The intracranial biomechanics in these two cases are not the same. Thus, the aggressiveness of therapy to treat the ICP becomes a factor that must be considered in analysis. Following the concept introduced by Lawrence Marshall, Chief of Neurosurgical Services at the University of California at San Diego, we have developed a method to grade ICP treatment according to the therapy intensity level (TIL). This technique of grading ICP treatment is routine in our ICU and is presently under study by the Traumatic Coma Data Bank of the National Institute of Neurological and Communicative Disorders and Stroke. The assessment for each therapeutic maneuver is based on a 15-point scale (Table 2).
For example, the use of sedatives (1 point), paralytic agents (1 point), and mild hyperventilation (1 point) accumulates a total TIL of 3 points for the hour. The use of intermittent ventricular drainage greater than four times within the same hour adds an additional 2 points to the index. A TIL of 7 or greater is an indication for mannitol administration. The maximum number of points that can be assessed without use of barbiturates is 12. A score of 15 is assigned when either bolus or continuous barbiturate infusion is used regardless of other therapy application. In the studies reported here, the specific therapies used for ICP management were documented each hour, and the TIL was calculated according to the therapeutic effort in the 4-hour “block” period.

Results

PVI Values Obtained from Bolus Addition Versus Withdrawal

Measurement of PVI by bolus addition was not always possible in patients with raised ICP and compromised compliance. On the basis that the logarithmic transformation of the pressure-volume curve is a straight line,15 it was theoretically possible to estimate the slope or PVI from volume reduction as well as volume removal. This concept was tested in patients where pressure was not excessive, thus permitting a direct comparison within each patient. Measurements of PVI on bolus withdrawal compared favorably with values obtained on bolus injection (Fig. 1). The linear regression of the PVI equaled the PVI on bolus injection (PVI(injection) = 2.65 ± 0.84 PVIw) in 92 manipulations (r = 0.87, p < 0.005). The PVI on bolus injection is slightly higher than that on bolus removal. At a normal PVI of 25 ml, the PVIw according to the regression formulas equals 23.6 ml. The close agreement of these methods suggests that both techniques are acceptable as measures of PVI in the head-injured patient.

Temporal Course of ICP, PVI, and Therapy Intensity Level

For purposes of analysis, patients were subdivided into three categories according to level of ICP: patients in whom ICP did not exceed 20 mm Hg (Group A); patients in whom ICP exceeded 20 mm Hg for periods of 10 minutes or more but was controllable (Group B); and patients whose ICP exceeded 20 mm Hg with subsequent development of uncontrolled raised levels despite aggressive therapy (Group C).

The ICP of Group A patients ranged from 14.1 to 16.0 mm Hg measured on the 1st day postinjury (Fig. 2A center). The ICP remained within the range of 9.4 to 15.7 mm Hg during the 3-day period postinjury. The absence of raised ICP prompted removal of the ventricular catheters on the 4th day to prevent infection. This mild pressure elevation was associated with a PVI level of 20.7 ± 6.3 ml which represented a 20% reduction of PVI when compared to normal PVI values of 25 ml in man (Fig. 2A upper). The combination of mildly elevated ICP and mild reduction in compliance exhibited by this patient group was associated with TIL values of less than 3. Thus, no mannitol, aggressive hyperventilation, or drainage was required for ICP management of this group (Fig. 2A lower).

The average ICP for Group B patients equaled 18.75 ± 6.2 mm Hg on Day 1 and ranged from a mean of 20.57 ± 6.13 to 24.14 ± 5.11 mm Hg during the next 4 days postinjury (Fig. 2B center). The PVI measured on the 1st day of injury was moderately decreased to a level of 14.7 ± 4.24 ml (Fig. 2B upper). This represents a reduction of 41% from normal PVI values. The reduced compliance coupled with raised ICP was accompanied by an increased level of therapy. The TIL averaged 4.87 ± 2.2 on the 1st day, indicating that mannitol was required in a few instances. The average TIL did not exceed 5.13 ± 1.88 during the 5-day period, indicating that the management of this patient group consisted primarily of sedation, drainage, and mild hyperventilation with only occasional use of mannitol (Fig. 2B lower).

Patients who developed uncontrollable ICP (Group C) had the lowest PVI levels (Fig. 2C upper). The PVI levels in these patients were severely depressed and averaged 10.5 ± 3.7 ml during the first 24 hours following injury. There was only slight improvement in pressure-volume status during the subsequent 5-day period despite maximal therapy.

Relationship of Temporal Course of ICP and PVI to Outcome

Patients were grouped according to the Glasgow Outcome Scale6 measured at 6 months after discharge from the ICU setting. The three outcome groups used for purposes of analysis were: Group 1, patients with good recovery or moderate disability; Group 2, patients with...
Pressure-volume index in head injury

Fig. 2. The temporal course of intracranial pressure (ICP), pressure-volume index (PVI), and therapy intensity level (TIL) during the 5 days postinjury. Patients were subdivided on the basis of sustained ICP level (Group A: < 20 mm Hg; Group B: > 20 mm Hg, controllable; Group C: uncontrollable). Group A and B patients were associated with only a moderately reduced PVI and recovered toward a normal PVI level. Therapy in Groups A and B was also mild to moderate. Patients who developed a high ICP were associated with a low PVI and maximal TIL.

There were 17 patients who had good recovery or moderate disability (Group 1). The GCS score measured at admission for this group equaled 7.0 ± 1.7. Of the 17 patients, the ICP was maintained below 20 mm Hg in nine (53%). The ICP range of the group over the 5-day monitoring period varied from a low of 17.1 ± 5.7 mm Hg to a high of 25.3 ± 6.16 mm Hg (Fig. 3A center). The corresponding PVI of this group was decreased to a mean level of 16.8 ± 6.3 ml on Day 1; however, the PVI profile for the entire group shows a trend toward normal levels (Fig. 3A upper). By Day 5, the PVI averaged 20.9 ± 6.7 ml. The TIL for this group was relatively mild: during the 5-day period the mean level ranged from a low of 2.74 ± 1.23 to a high of 4.34 ± 1.34. Figure 3A lower shows that patients in Group 1 were managed with sedation, mild hyperventilation, and infrequent drainage. Mannitol was not required.

There were nine patients in the severely disabled or vegetative group (Group 2, Fig. 3B). The GCS score on admission was 5.7 ± 0.9. In seven of these nine patients ICP levels exceeded the 20 mm Hg; however, the ICP profile was not significantly different from that in Group 1. The mean ICP ranged from a low of 17.6 ± 4.7 mm Hg to a high of 23.8 ± 5.48 mm Hg over the 5-day period (Fig. 3B center). Despite the similarity in ICP profiles of outcome Groups 1 and 2, the aggressiveness of therapy necessary to manage the ICP in Group 2 was higher. The mean TIL levels in Group 2

severe disability or in a vegetative condition; and Group 3, patients who died. The corresponding temporal profiles of ICP, PVI, and TIL were analyzed for the respective groups in the same manner as the time course studies depicted in Fig. 2.

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FIG. 3. The temporal course of intracranial pressure (ICP), pressure-volume index (PVI), and therapy intensity level (TIL) in each outcome group. A: Group 1 patients with good or moderate outcome were associated with a mildly reduced PVI that gradually return toward normal. The ICP was only mildly elevated and TIL was minimal. B: Group 2 patients with poor outcome (severe disability or vegetative state: sev/veg) exhibited a moderately reduced PVI that did not tend toward normal over the 5-day course. The ICP was not significantly different from that of Group 1; however, more aggressive TIL was required to manage the ICP. C: Group 3 patients who died had the lowest PVI and maximal ICP despite maximal TIL.

The relationship between PVI levels measured within 24 hours postinjury and subsequent course of the ICP is summarized in Table 3. Patients in whom ICP remained below 20 mm Hg were associated with mildly reduced PVI levels (mean 20.8 ± 4.17 ml), In contrast to this group, mean reduced PVI levels of 15.6 ± 4.45 ml were associated with patients in whom the ICP was elevated above 20 mm Hg but was manageable. The two patients presented with normal PVI and a normal ICP profile.

Statistical Analysis: Comparison Between Groups

Initial PVI Versus Subsequent ICP Course. The relationship between PVI levels measured within 24 hours postinjury and subsequent course of the ICP is summarized in Table 3. Patients in whom ICP remained below 20 mm Hg were associated with mildly reduced PVI levels (mean 20.8 ± 4.17 ml). In contrast to this group, mean reduced PVI levels of 15.6 ± 4.45 ml were associated with patients in whom the ICP was elevated above 20 mm Hg but was manageable. The two patients presented with normal PVI and a normal ICP profile.
Pressure-volume index in head injury

**TABLE 3**

<table>
<thead>
<tr>
<th>Outcome Group</th>
<th>ICP (mm Hg)</th>
<th>PVI (ml)</th>
<th>No. of Cases</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;20</td>
<td>20.8 ± 4.17</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>&gt; 20 controlled</td>
<td>15.6 ± 4.45</td>
<td>13</td>
<td>&lt;0.011†</td>
</tr>
<tr>
<td>3</td>
<td>&gt; 20 uncontrolled</td>
<td>10.1 ± 3.17</td>
<td>6</td>
<td>&lt;0.0004‡, &lt;0.0224</td>
</tr>
</tbody>
</table>

*Intracranial pressure (ICP) outcome is measured 5 days after admission. Pressure-volume index (PVI) is expressed as mean ± standard deviation. Outcome: Group 1 = good recovery or moderate disability; Group 2 = severe disability or vegetative state; Group 3 = died. Significance of difference: † p value compared to Group 1; ‡ p value compared to Group 2.

**TABLE 4**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>no. of cases</td>
<td>15</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>mean PVI ± SD</td>
<td>18.93 ± 5.27</td>
<td>16.80 ± 4.00</td>
<td>10.11 ± 4.31</td>
</tr>
<tr>
<td>p value compared to Group 1</td>
<td>NS</td>
<td>&lt; 0.0024</td>
<td></td>
</tr>
<tr>
<td>compared to Group 2</td>
<td></td>
<td></td>
<td>&lt; 0.011</td>
</tr>
</tbody>
</table>

*PVI = pressure-volume index; SD = standard deviation; NS = not significant. Outcome: Group 1 = good recovery or moderate disability; Group 2 = severe disability or vegetative state; Group 3 = died.

The 15- to 20-ml range of PVI reflects a moderately tight brain. The reduced level of PVI for this group was statistically significant (p < 0.011) compared to the level in patients with no ICP problems. Maximally reduced levels of PVI (10.1 ± 3.17 ml) were seen in patients who subsequently developed uncontrollable ICP. This reduction in PVI was statistically significant when compared to levels in patients who did not develop unmanageable ICP (p < 0.0004) and in patients in whom the ICP was elevated but controlled (p < 0.022).

**PVI and ICP Profiles Versus Outcome.** The comparison of ICP levels and profiles of patients with good outcome or moderate disability (Group 1) and severe disability or a vegetative state (Group 2) were significantly different (Fig. 3). Although ICP levels and profiles were similar, the TIL necessary to manage ICP was greater in Group 2 (Fig. 3B lower). As described earlier in this report, patients who died (Group 3) developed uncontrollable ICP despite maximal therapy.

The distribution of patient PVI's measured during the first 24 hours after admission and outcome is correlated in Fig. 4 and summarized in Table 4. The PVI levels of outcome Groups 1 and 2 evaluated 6 months postinjury were not significantly different from those on Day 1. However, PVI values measured during the first 24 hours postinjury in those patients who died (Group 3) were significantly reduced compared to Group 2 (<0.011) and Group 1 (p < 0.0024). Thus, a low PVI measured on Day 1 which is sustained or a decreasing PVI over time is associated with poor outcome.

Summarizing these findings, the level of PVI soon after admission identifies patients at risk for ICP elevation. Patients with a PVI of 20 ml or greater experience little difficulty with ICP, and therapy intensity is mild. Patients with PVI in the range of 15 to 20 ml have significant pressure elevations which are controlled with maximal therapy levels not exceeding a TIL of 12 (mannitol, hyperventilation, and drainage). The greater the reduction of PVI below this value, the greater the risk for developing uncontrollable ICP despite maximal therapy. Some patients with a low initial PVI respond to therapy and recover biomechanically. These patients can be identified by increased PVI within a 48- to 72-hour period. Patients with a sustained low PVI (<13 ml) are associated with poor outcome.

**Discussion**

One main objective of this study was to observe the biomechanical response of the brain to severe head injury.
injury. Our results show that, following mechanical trauma, brain compliance as assessed by the PVI is reduced and the degree of reduction and subsequent biomechanical recovery is closely related to outcome.

This study also demonstrates a clear relationship between the PVI measured soon after injury and subsequent development of ICP. Pressure-volume index values of 20 ml or above are associated with mild elevations of ICP. This represents a 20% reduction in PVI from a normal level of 25 ml, and it would appear on the basis of the unremarkable ICP course that this reduction is tolerated without complication and that the TIL for control of ICP is minimal. Reduction of the PVI level below 20 ml is associated with a significant ICP rise, and the intensity of therapy necessary to maintain ICP within tolerable limits is increased. The ICP course for PVI measurements above 13 ml can be controlled. From these studies, a PVI of 13 ml represents the critical PVI threshold for the head-injured patient, and patients with a PVI less than this critical value are at risk for developing uncontrollable ICP.

The "critical PVI threshold" of 13 ml observed in these studies is in close agreement with the findings of Tans and Poortvliet. These investigators studied the relationship of ICP and PVI in heterogeneous groups of 40 patients, of whom only three were head-injured. They concluded that volume-pressure relationship assessments were useful in head-injured patients who have a normal or elevated ICP without pressure waves. They found that, although a PVI of less than 18 ml should be considered pathological, 13 ml and 10 ml are the key PVI values. These results are very close to our findings. The mean PVI for patients who died from intracranial hypertension never exceeded 13.62 ml, and values in this range point to a dangerously tight brain. Experimental studies by Marmarou, et al., have shown that the distribution of PVI between cranial and spinal compartments equals 62% and 38%, respectively. Applying this distribution to man, the normal cerebral PVI component equals 17 ml while the spinal axis contributes 8 ml of volume-buffering capacity. From the studies presented in this report and the studies by other investigators, a measured PVI of 13 ml, of which 8 ml are comprised of spinal axis buffering, would indicate almost total exhaustion of cerebral volume-buffering capacity of the adult patient.

Normal PVI values in children are considerably less than adult levels. Shapiro, et al., have shown that the PVI in children is directly proportional to cranial volume and length of the spinal axis. They also observed that severely head-injured children do have reductions in their ideal PVI or neuraxis volume-buffering capacity. When the PVI was 50% of the predicted normal level, ICP management was usually unsuccessful. Accordingly, patients with 60% of the predicted normal PVI invariably experienced elevated ICP that required vigorous treatment (barbiturates, osmotherapy, and CSF drainage). According to their findings, PVI reductions of 50% to 60% of normal represent the limit for exhaustion of volume-buffering capacity, which coincides with estimates derived from experimental work by Marmarou, et al., and Löfgren and Zwetnow.

Moreover, Shapiro and Marmarou found that a reduced PVI was an accurate predictor of impending intracranial hypertension; in subsequent work, these investigators reported that treatment of severely compromised neuraxis compliance in an anticipatory fashion prevented the development of intracranial hypertension (K Shapiro, A Fried, unpublished data, 1987). It is important to note that, in our studies, therapy was targeted toward ICP management and there was no direct attempt to modify the natural course of the PVI profile based on PVI calculations alone. This is clearly evidenced by the TIL scales which quantify therapy. The TIL profiles are closely linked to the ICP temporal course, and more aggressive therapy was coincident with ICP rise despite earlier reductions of PVI. Although the TIL simply quantifies the intensity of treatment and not the effectiveness of treatment, it is clear from the close correspondence of ICP and TIL profiles that the TIL is an effective means of describing the degree of effort used in ICP management.

The results of our studies and those of other investigators are in sharp contrast to investigations by Kosteljanetz. Kosteljanetz studied 16 head-injured patients (GCS scores 3 to 15) and concluded that PVI varied independent of clinical course, outcome, and ICP. The PVI was not studied in temporal sequence and the time of initial study varied from 0 to 8 days postinjury. However, if we compare the PVI values and ICP categories in six of his severely injured patients (GCS score of 8 or less) studied within 24 hours of injury, both the outcome and ICP profiles are in full agreement with the criteria described in this report. This emphasizes the importance of monitoring the temporal course of PVI.

With regard to methodological issues, we found (as others have observed) that PVI levels obtained from withdrawal of fluid compare favorably with bolus-derived PVI. This is particularly useful in patients with elevated pressure where even a small fluid addition may cause significant elevations in ICP. We also found the withdrawal method to be of practical use in the measurement of PVI soon after surgery and in patients with CSF leak. In both of these circumstances, the injection PVI may be artifactual and can be recognized by a rapid fall in ICP following addition of fluid. Large differences between PVI values obtained from injection and withdrawal signify errors in measurement and should be rechecked. When this occurs in our patients, we accept the withdrawal PVI as correct. It is also important to note that the PVI values reported herein did not involve the constant-term correction as proposed by other investigators. We conducted a study in which we compared the pressure offset in head-injured patients as measured by multiple injections to the simpler PVI equation. We found that the error for PVI values of greater than 10 ml is negligible, and as a practical tool
the simple calculation for determining PVI in patients is satisfactory.

The close relationship of PVI to outcome was unexpected. The temporal course of PVI associated with patients with good recovery indicates that the pressure-volume status following trauma was minimally affected and that biomechanical recovery was possible. With regard to the prognostic value of early PVI measures, the standard deviations relating to PVI measured during the first 24 hours and outcome are too large to make predictions on individual patients. However, when this series is taken as a group, the greater the initial swelling the poorer the outcome. The sustained reduction in buffering capacity over the 5-day monitoring period in patients with poor recovery is remarkable. This occurs despite aggressive pressure management. Although the numbers of patients are small, the early ICP levels of patients who subsequently developed uncontrollable ICP were variable and not significantly different from initial ICP levels in other outcome groups (Fig. 3). Studies have shown that it is possible for the brain to swell at a rate that would not result in appreciable elevation of ICP. This infers that ICP measurement alone may be insufficient as a measure of early brain swelling. In the acute stage, our data suggest that reduction in buffering capacity is better indicated by PVI than by ICP.

Why was pressure therapy ineffective in patients with low PVI? Our data indicate that the TIL increased proportionately with ICP, as might be expected. As described earlier, conventional pressure management in this series of patients addressed the control of ICP, not PVI. The observation that PVI is initially low and refractory to conventional ICP treatment is an important finding. It leads to the conclusion that, in a tight brain, small changes in volume can result in significant pressure reductions achieved by drainage, hyperventilation, or mannitol. One must infer from these data that ICP may be lowered in these circumstances but the effect is transient since the brain remains tight. Thus, with the return of small increments of blood volume or edema, pressure increases rapidly. This suggests that therapies be directed to increasing PVI as well as reducing ICP, as demonstrated by Shapiro and Marmarou.

In summary, it can be concluded that the PVI measured soon after injury and followed throughout the patient's course can be used as indicator of biomechanical recovery and pending ICP rise. Patients with a low PVI (independent of their ICP level) should be carefully monitored and PVI be determined frequently to track pressure-volume status. Our results provide further evidence that PVI values of 13 ml or less in the adult head-injured patient are dangerously low, and attempts to improve buffering capacity independent of normal ICP status should be considered.

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