Intracranial pressure: to monitor or not to monitor?

A review of our experience with severe head injury

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The authors have analyzed their experience with intracranial pressure (ICP) monitoring in 207 patients over a 4-year period. Patients with either high-density or low-density lesions on computerized tomography (CT) at admission had a high incidence (53% to 63%) of intracranial hypertension (ICP persistently over 20 mm Hg). In contrast, patients with normal CT scans at admission had a relatively low incidence of ICP elevation (13%). Among these patients, three features were found to be strongly associated with the development of intracranial hypertension: 1) age over 40 years; 2) systolic blood pressure under 90 mm Hg; and 3) motor posturing — unilateral or bilateral. When two or more of these features were noted at admission, the incidence of intracranial hypertension was 60%, as compared to 4% when only one, or none, of these features were present. Thus, the patients at high risk for developing intracranial hypertension after severe head injury are those with abnormal CT scans at admission, and those with normal CT scans who demonstrate two or more of the above-mentioned adverse features. Based on these criteria, only 16% of this series of patients with normal CT scans would have qualified for monitoring.

In addition to the three clinical features noted above, multimodality evoked potential (MEP) studies were also found to be strong predictors of ICP elevation in the normal CT scan group, with a 75% incidence of intracranial hypertension in patients with disseminated deficits. There was no statistically significant correlation between the Glasgow Coma Scale score, eye movements, pupillary reaction, hypoxia, or anemia at admission and subsequent ICP elevation in the group with normal CT scans.

In this series, an intraventricular catheter was used as the sole monitoring device in 91% of the cases. In the remaining 9%, subarachnoid screws were employed, either alone, or upon failure of the ventriculostomy. While no mortality was directly ascribed to the monitoring process, there was a 7.7% complication rate (infection 6.3% + hemorrhage 1.4%). Eighty-five percent of the infections occurred in patients who had been monitored for 5 days or more, while no infections were noted in those monitored for less than 3 days. Used judiciously, this technique can be valuable in the monitoring and treatment of the brain-injured patient.

KEY WORDS □ head injury □ intracranial pressure monitoring □ computerized tomography □ ventriculostomy □ subarachnoid screw □ multimodality evoked potentials

The application of intracranial pressure (ICP) monitoring to the management of patients with both traumatic and nontraumatic neurological disorders has been described by several investigators. A distinction, however, must be made between the value of this technique as an “early warning system” or prognostic tool on the one hand, and a therapeutic modality on the other. While its value in the former roles has been clearly demonstrated, it has been more difficult to prove that the monitoring and control of ICP actually improves outcome. To complicate
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the issue further, debate still persists as to what constitutes intracranial hypertension. Most authors regard pressures below 10 mm Hg as being clearly normal, and initiate therapy if the ICP persists above 20 to 25 mm Hg. However, the cut-off point between "normal" and "elevated" remains somewhat ill-defined. As a result of these uncertainties coupled with the invasive nature of the technique, there has been some concern about its risk:benefit ratio. In the management of severe head injury, accepted practice spans the spectrum from uniform monitoring of all patients to complete avoidance of the technique.

As part of a prospective multidisciplinary investigation of traumatic brain dysfunction, we serially documented findings on clinical examination, ICP monitoring, computerized tomography (CT), and multimodality evoked potential (MEP) studies in a large group of uniformly treated patients with severe head injury. In this paper, we have analyzed data gathered over a 4-year period with the prime purpose of addressing the question: "Should ICP monitoring be undertaken routinely in all patients with severe head injury, or can certain patients be safely excluded on the basis of clinical, morphological, or electrophysiological data?"

Clinical Material and Methods

Patient Population

This analysis is based on 226 consecutive patients with severe head injury who were admitted to the neurosurgical service of the Medical College of Virginia Hospitals between April, 1976, and April, 1980. Criteria for admission to the study were inability to obey commands or to utter recognizable words following head injury, despite successful cardiopulmonary stabilization. The series did not include gunshot wounds of the head, or those who on admission to the emergency room were already apneic and fulfilled the criteria for brain death. Approximately half of the patients were admitted directly from the scene of their accident, while the other half were referred from other hospitals. A previous analysis of the series showed that 20% of the patients were seen by a neurosurgeon at our institution within 1 hour of injury, 35% within 2 hours, 70% within 6 hours, and all within 12 hours. The mean age was 31 years and the median 25 years. Patients ranged in age from 2 to 89 years. Of the 226 patients studied, 19 had insufficient ICP or CT data from admission to allow a valid analysis. The remaining 207 patients form the basis of this report.

Management

Our protocol for the management of head-injured patients has been described previously. The first neurological assessment was performed after the patient's respiratory function and blood pressure had been stabilized in the emergency room. An emergency cranial CT scan was then obtained. Depending on the findings of this study, patients were taken either to the operating room for surgical evacuation of a mass lesion (more than 5 mm actual midline shift), or to the neurosurgical intensive care unit (ICU). All patients were paralyzed with pancuronium (Pavulon), and artificially ventilated. In the ICU, ICP was monitored continuously using a ventriculostomy catheter (91% of cases), or a subarachnoid screw (either alone, or in addition to a ventriculostomy) (9% of cases).

As part of the study, subsequent neurological evaluations were performed by a neurologist and/or a neurosurgeon on postinjury Days 1, 4, and 14, and in survivors at 3, 6, and 12 months. Follow-up CT scans and MEP studies were also scheduled routinely on these days. All patients received corticosteroids from the time of admission (dexamethasone, 4 mg every 6 hours; or methylprednisolone, 40 mg every 6 hours). In 20 patients, a trial of high-dose steroid therapy was carried out (methylprednisolone, 1000 mg/day), but no effect on ICP or outcome was found. These patients are therefore included with the rest of the group.

Computerized Tomography Categories

An EMI Mark 1 scanner with a 160 × 160 matrix was used until late 1977, when the unit was replaced with a 256 × 256-matrix Delta scanner.* Contrast-enhanced CT was usually performed. Patients were divided into four groups based on the presence or absence of abnormalities on the initial CT scan, the density of the lesions present, and the need for surgical decompression. The groups were patients with: 1) normal CT; 2) low-density lesions only; 3) nonsurgical high-density lesions (contusions or hematomas causing less than a 5 mm shift of the midline); or 4) surgical high-density lesions (epidural, subdural, or intracerebral hematomas causing a midline shift greater than 5 mm and requiring surgical decompression).

Intracranial Pressure Categories

All patients analyzed had ICP monitored, usually for 3 to 5 days, but longer if the ICP remained elevated. Average ICP measurements were recorded every hour. Elevated ICP was defined in this study as persistent elevation of the mean pressure above 20 mm Hg during the period of continuous monitoring. Pressures in the 21 to 40 mm Hg range were regarded as "moderately" elevated, while those in the range of 41 mm Hg and above constituted "severe" intracranial hypertension. When ICP remained above a mean of 25 mm Hg for more than 15 minutes, attempts were made to reduce it by hyperventilation to an arterial pCO2 of 20 mm Hg, by controlled cerebrospinal fluid drainage, or to undertake routine craniotomy (11% of cases).

* EMI Mark 1 scanner manufactured by EMI Tronics, Inc., 3605 Woodhead Drive, Northbrook, Illinois, and Delta scanner manufactured by Ohio-Nuclear, Inc., 29100 Aurora Road, Solon, Ohio.
TABLE 1
Outcome in different CT groups

<table>
<thead>
<tr>
<th>CT Group*</th>
<th>Cases</th>
<th>Outcome† (%)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Percent</td>
<td>G</td>
</tr>
<tr>
<td>normal</td>
<td>61</td>
<td>30</td>
<td>62</td>
</tr>
<tr>
<td>low-density lesions only</td>
<td>20</td>
<td>10</td>
<td>50</td>
</tr>
<tr>
<td>high-density nonsurgical lesions</td>
<td>40</td>
<td>19</td>
<td>40</td>
</tr>
<tr>
<td>high-density surgical lesions</td>
<td>86</td>
<td>41</td>
<td>26</td>
</tr>
<tr>
<td>total cases</td>
<td>207</td>
<td>100</td>
<td>42</td>
</tr>
</tbody>
</table>

* The correlation between computerized tomography (CT) findings and outcome is significant (p < 0.0001). The difference in outcomes associated with normal and abnormal (low- or high-density lesions) admission CT scans is also significant (p < 0.0001).
† G = good recovery; MD = moderately disabled; SD = severely disabled; V = vegetative.

(CSF) drainage against a positive pressure of 20 to 25 cm H₂O, or by a bolus of intravenous mannitol (0.5 to 1.0 gm/kg). Patients were divided into categories based on their ICP course: 1) those with normal ICP’s throughout; 2) those with ICP elevations amenable to therapy; and 3) those with ICP elevations refractory to therapy.

Multimodality Evoked Potential Categories

The technical aspects of obtaining and analyzing MEP data in comatose head-injured patients have been described in earlier reports. A complete MEP study consisted of visual (VER), auditory (AER), and somatosensory (SER) cortical evoked responses; together with early-latency (brain-stem) responses in the auditory and somatosensory systems. The MEP deficits were described as being either "focal" or "disseminated." Focal abnormalities were defined as unilateral, unimodal deficits. Thus, patients with abnormalities of only the SER, VER, or AER on one side were included in the focal abnormality category. The definition of disseminated abnormalities included bilateral or multimodal deficits in the brain stem, hemispheres, or both (global).

Outcome Categories

Outcomes of the patients were defined at 3, 6, and 12 months according to the following categories:

Good recovery (G): Complete neurological recovery, or minor deficits that do not prevent the patient from returning to his/her former level of function

Moderately disabled (MD): Deficits present that prevent normal function, but allow self-care

Severely disabled (SD): Marked deficits present that prevent self-care

Vegetative (V): No evidence of higher mental function

Dead (D).

Results

Outcome in Different CT Groups

A significantly higher proportion of good outcomes (62% G, 18% MD) was noted in patients with normal CT scans on admission as compared to the other groups (p < 0.0001) (Table 1). The outcomes recorded in the group with low-density lesions only (50% G, 10% MD), and those in the nonsurgical high-density lesion group (40% G, 20% MD) were similar, but showed a trend toward increasing morbidity associated with high-density lesions. As expected, those with high-density lesions requiring surgery had the smallest number of good recoveries (26% G, 13% MD). In the overall series of 207 patients, 42% made good recoveries, 15% were moderately disabled, 9% severely disabled or vegetative, and 34% died.

Prognostic Significance of Different ICP Courses

Elevation of ICP at any stage was associated with a significantly poorer outcome (27% G, 16% MD) as compared to patients with normal ICP courses (59% G, 18% MD) (p < 0.0001) (Table 2). Those with persistently elevated ICP's refractory to therapy almost always died. These findings are consistent

TABLE 2
Prognostic significance of different ICP courses

<table>
<thead>
<tr>
<th>ICP Course*</th>
<th>Cases</th>
<th>Outcome† (%)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Percent</td>
<td>G</td>
</tr>
<tr>
<td>normal throughout</td>
<td>111</td>
<td>54</td>
<td>59</td>
</tr>
<tr>
<td>elevated but reducible</td>
<td>75</td>
<td>36</td>
<td>27</td>
</tr>
<tr>
<td>elevated &amp; not reducible</td>
<td>21</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>total cases</td>
<td>207</td>
<td>100</td>
<td>42</td>
</tr>
</tbody>
</table>

* The correlation between intracranial pressure (ICP) course and outcome is significant (p < 0.0001). The difference in outcomes associated with normal and elevated ICP is also significant (p < 0.0001).
† G = good recovery; MD = moderately disabled; SD = severely disabled; V = vegetative.
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TABLE 3
Highest ICP recorded in different CT groups

<table>
<thead>
<tr>
<th>CT Groups*</th>
<th>No. of Cases</th>
<th>Highest ICP Levels (%)</th>
<th>≤ 20 (mm Hg)</th>
<th>21–40 (mm Hg)</th>
<th>41–60 (mm Hg)</th>
<th>≥ 61† (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>normal</td>
<td>61</td>
<td>87</td>
<td>11</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>low-density lesions only†</td>
<td>19</td>
<td>47</td>
<td>31</td>
<td>11</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>high-density nonsurgical lesions</td>
<td>40</td>
<td>43</td>
<td>40</td>
<td>10</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>high-density surgical lesions</td>
<td>86</td>
<td>37</td>
<td>36</td>
<td>13</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>total cases</td>
<td>206</td>
<td>54</td>
<td>29</td>
<td>9</td>
<td>8</td>
<td></td>
</tr>
</tbody>
</table>

* There is a significant correlation between computerized tomography (CT) features and intracranial pressure (ICP) profiles (p < 0.0001).
† In one patient in this group the highest recorded pressure was not available.

with our previous analyses in smaller groups of patients.28,31,33

ICP Elevations in Different CT Groups

Patients with normal CT scans at admission showed remarkably different ICP profiles as compared with those whose initial CT scan showed either low- or high-density lesions (p < 0.0001) (Table 3). In 53 of 61 patients (87%) with normal CT scans, the ICP never exceeded 20 mm Hg. The eight patients (13%) who did develop intracranial hypertension had other complicating factors that will be detailed later. However, even when an elevation did occur, the magnitude of the rise was usually moderate (20 to 40 mm Hg in seven of the eight), albeit with therapy. Thus, in 98% of patients with normal CT scans on admission, the ICP remained below 40 mm Hg.

In contrast, patients with low-density lesions had normal ICP courses in 47% of cases, and those with high-density lesions in only 43% and 37% of cases, respectively, in the nonsurgical and surgical groups. Thus, while the vast majority of patients with normal CT scans on admission have a normal ICP course, the same can be said of less than half the patients with abnormal CT scans. Furthermore, the degree of intracranial hypertension is more often severe in the latter groups (Table 3).

Influence of ICP on Outcome in Different CT Groups

Elevation of ICP, either persistently, or at any stage, was associated with a poorer outcome in all CT categories (Table 4). Patients in all four groups showed lower proportions of good outcomes in the face of intracranial hypertension, with a 43%, 31%, 33%, and 27% drop in the percentage of good outcomes, respectively. Elevation of ICP did not seem to have an inordinately greater or lesser effect in any of the groups, although a statistically significant difference in outcome could be established only in the high-density surgical lesion group. This was due in part to the larger number of patients in this group.

Patients with Normal CT Scans Who Developed Intracranial Hypertension

As described earlier, eight of the 61 patients (13%) with normal CT scans on admission developed elevated ICP (Tables 3 and 4). The eventual outcome of these eight patients was poorer when compared to the other 53 patients in the normal CT group, although, because of the small number of patients in the elevated ICP group, a statistically significant difference could not be demonstrated (Table 4). We analyzed these patients in depth in order to ascertain features that

TABLE 4
The association of intracranial hypertension with poorer outcomes

<table>
<thead>
<tr>
<th>CT Group*</th>
<th>Intracranial Pressure</th>
<th>Cases</th>
<th>Outcome† (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.</td>
<td>Percent</td>
</tr>
<tr>
<td>normal</td>
<td>normal</td>
<td>53</td>
<td>87</td>
</tr>
<tr>
<td></td>
<td>elevated</td>
<td>8</td>
<td>13</td>
</tr>
<tr>
<td>low-density lesions only</td>
<td>normal</td>
<td>9</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>elevated</td>
<td>11</td>
<td>55</td>
</tr>
<tr>
<td>high-density nonsurgical lesions</td>
<td>normal</td>
<td>17</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>elevated</td>
<td>23</td>
<td>57</td>
</tr>
<tr>
<td>high-density surgical lesions</td>
<td>normal</td>
<td>32</td>
<td>37</td>
</tr>
<tr>
<td></td>
<td>elevated</td>
<td>54</td>
<td>63</td>
</tr>
<tr>
<td>total cases</td>
<td></td>
<td>207</td>
<td>42</td>
</tr>
</tbody>
</table>

* Although a difference in outcomes is apparent between those patients with normal and those with elevated intracranial pressures, a statistically significant difference could not be established in the first three computerized tomography (CT) groups (p = 0.11, 0.33, and 0.07, respectively). In the high-density surgical lesion group, however, the difference was significant (p = 0.002).
† G = good recovery; MD = moderate disability; SD = severely disabled; V = vegetative.
Table 5

Association of various factors with development of intracranial hypertension in patients with normal CT scans on admission*

<table>
<thead>
<tr>
<th>Factors</th>
<th>Data</th>
<th>No. of Cases</th>
<th>% Normal</th>
<th>% Elevated</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>blood pressure (systolic) (mm Hg)</td>
<td>over 90</td>
<td>50</td>
<td>94</td>
<td>6</td>
<td>0.003</td>
</tr>
<tr>
<td>MEP studies</td>
<td>normal</td>
<td>39</td>
<td>92</td>
<td>8</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>diss. deficits</td>
<td>4</td>
<td>25</td>
<td>75</td>
<td></td>
</tr>
<tr>
<td>motor posturing</td>
<td>none</td>
<td>40</td>
<td>95</td>
<td>5</td>
<td>0.030</td>
</tr>
<tr>
<td></td>
<td>unilateral</td>
<td>12</td>
<td>83</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td></td>
<td>bilateral</td>
<td>9</td>
<td>56</td>
<td>44</td>
<td></td>
</tr>
<tr>
<td>age (yrs)</td>
<td>0–20</td>
<td>28</td>
<td>96</td>
<td>4</td>
<td>0.071</td>
</tr>
<tr>
<td></td>
<td>21-40</td>
<td>23</td>
<td>83</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td></td>
<td>41+</td>
<td>10</td>
<td>70</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>GCS score</td>
<td>7 or over</td>
<td>23</td>
<td>92</td>
<td>8</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>6 or less</td>
<td>38</td>
<td>78</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>eye movements</td>
<td>normal</td>
<td>42</td>
<td>90</td>
<td>10</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>impaired unilat</td>
<td>7</td>
<td>71</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td></td>
<td>impaired bilat</td>
<td>12</td>
<td>83</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>pupillary reaction</td>
<td>normal</td>
<td>54</td>
<td>87</td>
<td>13</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>absent bilat</td>
<td>7</td>
<td>86</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>hypoxia</td>
<td>none</td>
<td>41</td>
<td>93</td>
<td>7</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>present</td>
<td>20</td>
<td>75</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>anemia</td>
<td>none</td>
<td>56</td>
<td>88</td>
<td>12</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>present</td>
<td>5</td>
<td>80</td>
<td>20</td>
<td></td>
</tr>
</tbody>
</table>

* Abbreviations: CT = computerized tomography; ICP = intracranial pressure; MEP = multimodality evoked potentials; diss. deficits = disseminated deficits present; GCS = Glasgow Coma Scale; NS = not statistically significant.

could be associated with the ICP elevations (Table 5). The nine factors studied at admission included age, blood pressure, hypoxia, anemia, Glasgow Coma Scale (GCS) score, motor posturing, eye movements, pupillary reaction, and MEP deficits. Of these, a statistically significant relationship was found in only four using the chi-square test: blood pressure, MEP deficits, motor posturing, and age (Table 5). Although certain trends were evident, a statistically significant association could not be demonstrated between the GCS score, eye movements, pupillary reactions, hypoxia, or anemia and subsequent ICP elevation in the normal CT group.

A remarkable association was noted between hypotension at admission and subsequent ICP elevation (Table 5). Only 6% of those patients with a systolic blood pressure over 90 mm Hg at admission developed intracranial hypertension, as compared to 45% of those with a systolic blood pressure of 90 mm Hg or less (p = 0.003). Motor posturing was also a strong indicator. In the absence of abnormal flexor (decorticate) or extensor (decerebrate) responses, only 5% of patients with normal CT scans developed ICP elevations. This figure rose to 17% in the presence of unilateral posturing, and to 44% when bilateral posturing was demonstrated at admission (p = 0.030).

Age was the third strong clinical indicator. Older patients with normal CT scans were more likely to develop intracranial hypertension. Only 4% of patients under 20 years of age who had normal CT scans developed elevated ICP. This figure rose to 17% in the 20 to 40 year age group, and to 30% in those over 40 years of age (p = 0.071). We found maximum predictive accuracy when 40 years was used as the critical age.

It is important to note that of the eight patients with normal CT scans who developed intracranial hypertension, only one did so less than 12 hours after injury. The onset of intracranial hypertension in these patients was noted at 4, 12, 20, 24, 24, 35, and 50 hours after the head injury, respectively, in seven patients. The eighth patient developed severe hyponatremia (Na 117 mEq/liter) and intracranial hypertension 7 days after the original injury. Follow-up CT scans showed diffuse bilateral low-density lesions. Incidentally, this was the only patient in this group to develop an ICP greater than 40 mm Hg. If she were excluded, the average time interval between injury and ICP elevation in these patients was found to be 24 hours.

MEP studies were noted to be strong predictors of ICP elevation in patients with normal CT scans. Only 8% of patients with normal MEP studies developed elevated ICP as compared to 75% of those with disseminated deficits (p = 0.003). It is apparent that these studies can serve as valuable predictors of intracranial hypertension in the presence of normal morphological studies.

Selecting Patients for Monitoring in the Normal CT Group

We found that ICP elevation could be accurately predicted 93% of the time in patients with normal CT scans with just four indicators (blood pressure, MEP data, motor posturing, and age), using the linear
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logistic regression model. If MEP data were excluded, the three clinical indicants performed almost as well, with a 92% prediction rate.

To further define the risk of intracranial hypertension in individual patients, we related the incidence of ICP elevation in the normal CT group to the clinical features in different combinations. As stated previously, the three critical factors associated with intracranial hypertension in the normal CT group were: 1) age over 40 years; 2) systolic blood pressure under 90 mm Hg; and 3) motor posturing — unilateral or bilateral. When a patient demonstrated two or more of these adverse features at admission, the incidence of intracranial hypertension was 60%, as compared to 4% when only one or none of these features were present (Table 6). In other words, 96% of patients with normal CT scans who demonstrate less than two of these adverse features will have a normal ICP course. These findings suggest that ICP monitoring need not be routinely performed in patients with normal CT scans unless they present with two or more of the adverse features noted above.

Based on these criteria, only 10 out of 61 (16%) of our patients with normal CT scans would have qualified for ICP monitoring. This figure corresponds fairly closely to the 13% incidence of ICP elevation in this group. Four of the 53 patients (8%) who in fact had normal CT courses would have been monitored, while six out of eight (75%) of those who developed intracranial hypertension would have qualified. One of the two patients who were not expected to develop intracranial hypertension by these criteria, but did, developed a delayed intracerebral hemorrhage. This patient did have disseminated deficits on MEP studies and would possibly have been monitored on that basis.

We did determine the incidence of intracranial hypertension associated with each of these three adverse indicants in the absence of the other two. Bilateral motor posturing was the only adverse feature present in five patients, and only one of these developed ICP elevation. Of the five patients aged over 40 years, and the three patients with a blood pressure of less than 90 mm Hg as the sole adverse feature, none developed intracranial hypertension. There appears to be an interaction of adverse factors in the genesis of intracranial hypertension.

Complications of ICP Monitoring

It has been our practice over the past 5 years to use intraventricular catheters whenever possible for ICP monitoring. We have found these to be reliable devices for pressure monitoring and useful for therapeutic CSF drainage when the ICP exceeds 20 mm Hg. However, when placement of an intraventricular catheter becomes difficult due to slit-like or displaced ventricles, we use a subarachnoid screw. In this series, a catheter was successfully used as the sole monitoring device in 91% of cases. In the remaining 9%, however, subarachnoid screws were employed, either alone, or upon failure of the ventriculostomy. Monitoring was continued, usually for at least 3 days. Thereafter, monitoring was discontinued, usually as soon as possible if the ICP appeared to have come under control.

The two most frequent complications of ICP monitoring are infection and intracranial hemorrhage.36 In this series of 207 patients, 22 (10.6%) developed ventriculitis with or without meningitis. However, in seven of these patients the infection could not be solely ascribed to the monitoring device because of associated complications (three had CSF otorrhea, one had rhinorrhea, two had compound skull fracture with dural tear, one had multiple craniotomies, and one had craniotomy wound infection). Two patients were excluded because no organism could be cultured from the ventricular CSF, although there was a pleocytosis suggestive of an infectious process. Thus, 13 of the 207 patients (6.3%) who were monitored developed ventriculitis with or without meningitis, that could be directly related to the monitoring device.

Of the 207 patients monitored, only 19 (9%) had subarachnoid screws placed. Of these, only one developed a device-related meningitis, giving an infection rate of 5.3% as compared to 6.4% for the ventriculostomy-monitored group. However, the number of screw-monitored patients in this series prevents us from making a definitive statement regarding the relative risk of these two techniques. In 1976, this center reported its experience with ICP monitoring using primarily the subarachnoid screw.36 In that series of 112 patients, only 10 were monitored with a ventriculostomy alone. The infection rate reported at that time was comparable to the present figures.

In reviewing the distribution of infections in the different CT groups, we found a device-related infection rate of 4.9% in the normal CT group, 0% in the low-density lesion group, 5% in the high-density nonsurgical lesion group, and 9.3% in the high-density surgical lesion group. Although multiple factors can contribute to the higher infection rate in the last group, we believe the most significant factor is the duration of monitoring. These patients have greater problems with intracranial hypertension, and tend to

**TABLE 6**

<table>
<thead>
<tr>
<th>No. of Adverse Signs†</th>
<th>Cases</th>
<th>ICP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 or 1</td>
<td>51</td>
<td>84</td>
</tr>
<tr>
<td>2 or 3</td>
<td>10</td>
<td>16</td>
</tr>
<tr>
<td>total cases</td>
<td>61</td>
<td>100</td>
</tr>
</tbody>
</table>

*ICP = intracranial pressure; CT = computerized tomography. Association statistically significant (p < 0.0001).
†Age over 40 years, systolic blood pressure < 90 mm Hg, and unilateral or bilateral motor posturing at admission.
be monitored for longer periods of time, thus increasing the chance of infection. In this series, 85% of patients who developed device-related infections had been monitored for 5 days or longer; the other 15% having been monitored for 4 days. No infections occurred in those who had been monitored for less than 3 days (p < 0.0001).

Intracranial hemorrhage secondary to ventriculostomy or screw placement did not prove to be a common problem. In this series of 207 patients, only one required surgery for a device-related hematoma. However, an intraventricular or intraparenchymal hematoma did develop in a total of three patients (1.4%). Patients at greater risk in this regard are those with disseminated intravascular coagulation with fibrinolysis or other coagulopathies, and those in whom technical difficulties necessitate multiple passes during catheter placement. As a general rule, we limited the number of passes permissible on each side to three.

Discussion

The earliest descriptions of ICP monitoring date back almost 100 years. Most significant advances in this area, however, have occurred over the last three decades. With technological refinements, the process of monitoring has become more elegant, reliable, and safe. Consequently, it is being increasingly applied in a variety of neurological disease states, especially traumatic coma. However, for the individual neurosurgeon faced with the question “To monitor or not to monitor?” no clear guidelines exist. With the advent of CT scanning, another variable has entered the reckoning. In this study, we have reviewed our experience over the past 4 years with a uniformly treated and fully documented group of patients with severe head injury. Our primary purpose in so doing was to identify trends in ICP in different CT groups, and, based on this information, to formulate specific guidelines for the application of this technique. In view of the fact that our patient population was predominantly adult (mean age 31 years, median 25 years), caution must be exercised in directly applying conclusions derived from this analysis to pediatric head injuries.

Monitoring of ICP was never solely responsible for a fatal outcome in this series, but it did carry a morbidity rate of 7.7%, primarily due to infection (6.3%) and intracranial hemorrhage (1.4%). Although both complications are almost always reversible with appropriate therapy, they do adversely influence a patient’s recovery, and thus underscore a need for more definitive guidelines relating to the indications for this invasive technique. In other words, we need to determine the risk/benefit ratio of the procedure in different groups of head-injured patients, and to ensure that in our enthusiasm for providing our patients with every means of support, we are not, in fact, doing more harm than good in certain groups.

The present study indicates that only 27% of patients with reversible ICP elevation had a good outcome, as opposed to 59% of those with normal ICP courses (Table 2). Persistent ICP elevation refractory to treatment was almost always fatal. These data reaffirm our previous contention that intracranial hypertension adversely affects outcome and should be corrected as soon as possible. Although results from this and other centers indicate that aggressive therapy including ICP monitoring can improve outcome in severe head injury, these data do not directly prove that ICP monitoring per se improved outcome. Such proof is, however, difficult to establish. Nevertheless, there have been reports that support such an inference. Bowers and Marshall, in a study of 200 patients with severe head injury, noted that among 86 patients with a GCS score of 3 to 5, there was only a 39% mortality rate in patients who underwent monitoring with an effort to control ICP, as compared to a 62% mortality rate in those not monitored (p < 0.05). However, their decision to monitor or not to monitor ICP was somewhat arbitrary and not strictly randomized. Another study, by Saul and Ducker, reported a reduction in mortality in cases of severe head injury by initiating treatment for elevated ICP at 16 mm Hg instead of 20 to 25 mm Hg. However, two temporally independent (although comparable) series of patients were used in this study. Although these two studies do not conclusively prove that efforts to monitor and control ICP improve outcome in severe head injury, they do lend support to this hypothesis.

A recent study by Galbraith and Teasdale on patients with traumatic intracranial hematomas, in whom the surgeon was undecided about the need for surgery, indicates that all patients with ICP’s above 30 mm Hg eventually deteriorated and required surgery. This rarely happened in those patients with an ICP below 20 mm Hg. Patients in the 20 to 30 mm Hg range were about evenly divided between the surgical and nonsurgical groups. This study exemplifies the value of ICP monitoring as an early-warning system that allows appropriate measures to be taken before actual neurological deterioration occurs.

The main question that the present study addressed is whether or not CT scanning can identify a group of patients who could just as safely be managed without ICP monitoring. Our findings indicate that in severely head-injured patients with either low- or high-density lesions on CT scanning, intracranial hypertension occurs in 53% to 63%, respectively, with a significant proportion of the patients recording pressures in excess of 40 mm Hg (severe intracranial hypertension) (Tables 3 and 4). A strong case can be made for ICP monitoring in these patients. Patients with normal CT scans, however, tend to behave differently. In our experience, these patients showed a normal ICP course in 87% of cases (Table 3). Another 10% showed elevations in the 20 to 40 mm Hg range, while only 2% developed pressures in the 40 to 60 mm Hg range.
albeit with therapy. These findings are consistent with observations previously reported by us,4,25 and others, such as Haar, et al.,16,47 who noted that ICP remained below 20 mm Hg in 28 out of 29 patients with normal CT scans. In this context, reference must be made to the work of Fleischer, et al.,7 who monitored ICP in 40 patients with severe head injury but without angiographic evidence of mass lesion. A third of these patients demonstrated ICP elevations above 20 mm Hg. Angiography, however, does not always demonstrate small lesions, such as contusions, and it seems likely that several of those patients who developed intracranial hypertension would probably have been classified under the “low-density” or “high-density nonsurgical” lesion group if a CT scan had been available. It is also likely that, with continued improvements in the resolution and speed of CT scanners, more subtle lesions will be identified, thus making the “normal” CT scan group slightly smaller but “purer.”

With a complication rate from monitoring being 7.7% and the proportion of patients with intracranial hypertension in the normal CT scan group being about 13%, a case can be made for not routinely monitoring all patients with normal CT scans. However, the small proportion of patients who do develop intracranial hypertension could benefit from monitoring. How then can one identify the subgroup at greater risk? Our results indicate that ICP elevation in the normal CT group can be predicted at admission with up to 92% accuracy using just three pieces of basic information—blood pressure, age, and the motor response. A systolic blood pressure of less than 90 mm Hg, age over 40 years, and the presence of motor posturing were associated with an increased risk of developing intracranial hypertension. When two or more of these adverse features were present, the incidence of elevated ICP was around 60% as compared to just 4% when only one or none of these features were present. Ninety-six percent of patients with a normal CT scan and less than two of these adverse features had a normal ICP course. Therefore, in patients with a normal CT scan, ICP monitoring can be limited to those who on admission demonstrate at least two of the three adverse signs. In those patients not monitored initially, monitoring may be initiated later in their course should they deteriorate neurologically, or if they demonstrate delayed hematomas on follow-up CT scans.

It was interesting to note that, among patients with normal CT scans, there was no statistically significant correlation between the GCS score, eye movements, pupillary reaction, anemia, or hypoxia on admission and the development of intracranial hypertension (Table 5). Of special interest is the finding that hypotension, but not hypoxia, was associated with subsequent ICP elevation. This finding confirms the observation of Senter, et al.,40 who found that an increased ICP was more likely to occur after a hypo-tensive rather than a hypoxic insult in patients with nontraumatic brain damage. The pathophysiology of ischemic brain injury is a somewhat controversial area, and several mechanisms have been implicated in its genesis. Two possible explanations for delayed ICP elevation are the development of brain edema and vascular engorgement. The pros and cons of each hypothesis have been discussed elsewhere.3,19,29,30

However, it seems probable that blood-brain barrier dysfunction, brain edema, impaired autoregulation, and increased cerebral blood volume can all contribute to the development of elevated ICP in the head-injured patient. In turn, intracranial hypertension, especially in the face of arterial hypotension, can effectively reduce cerebral perfusion pressure. Since the injured brain is less able to tolerate such stresses, it stands to reason that such insults should be strenuously avoided; failing which, every effort should be made to restore normalcy as soon as possible.19,27,28

Based on the guidelines established in this report, about 16% of all severely head-injured patients with normal CT scans on admission will qualify for ICP monitoring (assuming a patient population comparable to ours). The actual incidence of ICP elevation in this group is around 13%. Only two out of 61 patients (3%) in the normal CT category developed ICP elevation, a number contrary to expectation based on these criteria. As stated previously, one of these patients was found to have developed a delayed intracerebral hemorrhage. Since only one of eight patients who developed intracranial hypertension did so in less than 12 hours and since the average time taken for this to occur was 24 hours, it is recommended that all patients who are not monitored initially should routinely have a follow-up CT scan 12 to 24 hours after admission to rule out the development of a delayed lesion. In this context, it is of interest to note that Gudeman, et al.,13 in a study of delayed traumatic intracerebral hematomas, found that these lesions appeared within 48 hours in 11 of the 12 cases, and that seven out of the 11 patients monitored (64%) did manifest intracranial hypertension. However, only three of the 12 patients in that study had a normal CT scan at admission. In such patients, the appearance of a delayed traumatic intracerebral hematoma on follow-up CT scan may not be an absolute indication for ICP monitoring. Features to be taken into consideration in arriving at a decision would include the size of the lesion, mass effect, the patient’s neurological status, and the presence of a coagulopathy.

The value of MEP data as powerful prognostic indicators in severe head injury has been previously reported.19,27,30 This study reveals another potential application. In the normal CT scan group, only 8% of patients with normal MEP studies developed intracranial hypertension, as compared to 75% of those with disseminated deficits. It stands to reason that all patients with disseminated deficits on MEP studies...
should be monitored. Data from CT, ICP monitoring, and MEP studies can thus complement each other, and when interpreted within the context of the patient's clinical progress can form the basis for rational decisions regarding patient management.

Conclusions

1. A more selective approach may be adopted toward ICP monitoring in patients with severe head injury. Patients with abnormal CT scans at admission are at high risk for developing intracranial hypertension. Routine ICP monitoring may, therefore, be justified in these patients.

2. Patients with normal CT scans at admission, however, have a low incidence of intracranial hypertension, and should be monitored only when they demonstrate two or more of the following adverse features at admission: a) systolic blood pressure under 90 mm Hg; b) unilateral or bilateral motor posturing; and c) age over 40 years. If these criteria were applied to our patients with normal CT scans, only 16% would qualify for ICP monitoring. The presence of disseminated deficits on MEP studies can also serve as an indication for ICP monitoring in this group.

3. Patients with normal CT scans who are not monitored should routinely have a follow-up CT scan 12 to 24 hours after admission to rule out the presence of delayed lesions, ventricular enlargement, or midline shift. Scans should also be obtained if the patient deteriorates neurologically.

4. In view of the strong positive correlation between the duration of ICP monitoring and the infection rate noted in this and in previous studies, monitoring should be discontinued as early as possible after the first 3 days, taking into consideration the patient's clinical progress and ICP trend.

5. Used judiciously, this technique can be valuable in the monitoring and treatment of the brain-injured patient.

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